WORLD
RHEUMATOLOGY
FORUM
SUMMIT 2026

RHEUMATOLOGY MASTERCLASSES

Myositis

14th - 15th March 2026

Psoriatic Arthritis

WRF SOUVENIR

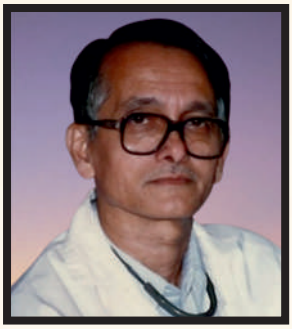
Mayfair Lake Resort, Raipur, Chhattisgarh

www.wrfsummit.com

TABLE OF CONTENTS

S. No	Topic	Page No.
1.	Message	2
2.	Award Message	17
3.	Faculty Spotlight	22
4.	Exploring Chhattisgarh	28
	Land of thirty six forts	30
	Seahorse of a Peninsula	36
	Crafts and Cravings	40
	Scout the unexplored	43
5.	Scientific Program Schedule	51
6.	Organising committee	55
7.	Challenging Cases (By Consultants)	57
8.	Abstracts	
	Platform	71
	Posters	81
9.	Rheumatology Patient Support Groups in India	211
10.	Rheumatologist corner	
	Unscripted Journey - Patient stories to inspire you	219
	Articular Arts - Extra - academic talent of our Rheumatology colleagues	234
11.	The Mini-Rheum Reporter	244
12.	Bridging conversation: Past WRF Highlights	265
13.	Crossword	270
14.	Event Sponsors	273

MESSAGE



Dr V R Joshi
Former HOD Medicine,
Rheumatologist and Director of Research
PD Hinduja National Hospital, Mumbai

It gives me great pleasure to pen this message for the World Rheumatology Forum, a non-profit organisation formed with the commendable goal of combining teaching and training under a single forum.

Dr Santhanam Sham is organising the sixth WRF summit at Raipur, which will surely be a catalyst for much-needed growth of rheumatology in the area. I compliment Dr Sham for holding the event there.

I am also happy to see the WRF becoming an important academic and training event of the rheumatology calendar, and I sincerely wish for this year's meeting to be a grand success.

Regards,
Dr VR Joshi

MESSAGE

Dr K M Mahendranth
MBBS, DTM & H (Liverpool, UK), F.R.C.P. (London, UK)
Master ACR, Master APLAR, BJD (Bone and Joint Decade) Ambassador
Consultant Rheumatologist,
Samarpan Rheumatology and Arthritis Centre, Bangalore3

I am very happy to know that "Rheumatology Masterclasses" organised by the World Rheumatology Forum will be held in Raipur this year. This year's masterclass will focus on Myositis and Psoriatic arthritis. These Masterclasses are attracting not only young Rheumatologists and trainees but also senior Rheumatologists. Real-world class leaders in their field are sharing their experience and enriching us. In regions where there is a shortage of Rheumatologists and public awareness of Rheumatology is inadequate, this summit will have a significant impact. I wish the summit a great success and congratulate the organisers for their efforts.

Best wishes
Dr KM Mahendranath

MESSAGE

Prof (Lt General) Ved Chaturvedi, MD, DM
Ganga Ram Institute of Postgraduate Medical
Education and Research
New Delhi, India

It gives me immense pleasure to extend my warm greetings to all delegates, faculty members, researchers, and industry partners participating in the World Rheumatology Forum (WRF) 2026, being hosted in the vibrant city of Raipur.

The World Rheumatology Forum has consistently served as a distinguished global platform for the exchange of knowledge, innovation, and clinical excellence in the field of rheumatology. WRF 2026 promises to further strengthen this legacy by bringing together eminent national and international experts to deliberate on emerging advances, evolving treatment paradigms, and future directions in rheumatologic care. Such scientific congregations play a pivotal role in translating research into practice and improving patient outcomes worldwide.

I commend the Organising Committee for their dedication and vision in curating a comprehensive scientific program while fostering collaboration, mentorship, and global partnerships. I am confident that WRF 2026 will not only enrich academic discourse but also inspire young clinicians and researchers to contribute meaningfully to this dynamic speciality.

I wish the conference every success and hope that all participants take back enriching academic experiences along with fond memories of Raipur's culture and hospitality.

With best wishes,
Prof. Ved Chaturvedi

MESSAGE

Prof. Dr G. Narsimulu
Past President, IRA
President API

World Rheumatology Conference 2026
Theme: Myositis and Psoriatic Arthritis

It gives me great pleasure to extend a warm welcome to all delegates, faculty members, researchers, and industry partners to the World Rheumatology Conference 2026, being held on 14th and 15th March 2026 in Raipur.

Rheumatology is a rapidly evolving speciality, and continuous academic exchange is essential to improve patient outcomes and advance clinical practice. The chosen theme of this conference — Myositis and Psoriatic Arthritis — is both timely and highly relevant. These complex, immune-mediated inflammatory disorders pose significant diagnostic and therapeutic challenges, and they demand a multidisciplinary and evidence-based approach to care.

This conference provides an excellent platform to discuss recent advances in pathophysiology, diagnostics, imaging, biomarkers, and evolving treatment strategies, including biologics and targeted therapies. The scientific program has been thoughtfully curated to encourage meaningful discussions, promote collaborative learning, and translate research insights into real-world clinical practice.

I am confident that the deliberations, panel discussions, workshops, and case-based sessions will enrich your knowledge and help refine your clinical decision-making. Equally important, this meeting offers valuable opportunities for networking, sharing experiences, and building long-lasting professional relationships across institutions and regions.

I congratulate the organizing committee for their dedication and meticulous planning in bringing together an esteemed group of national and international experts. Their efforts will undoubtedly make this conference a memorable and academically rewarding experience for all participants.

I encourage every delegate to actively participate, engage in discussions, and make the most of this scientific gathering. I wish the World Rheumatology Conference 2026 every success and hope that it serves as a catalyst for continued excellence in rheumatology education, research, and patient care.

With best wishes for a successful and inspiring conference.

Prof. Dr G. Narsimulu
MD, FICP, FIACM

Sr. Consultant-Rheumatologist at GVN Medical Centre
Past President, Indian Rheumatology Association
Former Dean ICP / API
Former Prof. & HOD Rheumatology, NIMS, HYD.
Former Chairman, Association of Physicians of India, Hyderabad chapter
Former Chairman of Association of Physicians of India –AP chapter
Former president of IACM (Indian Association of Clinical Medicine)
Former Governing Council member of Association of Physicians of India.
Faculty Council Member of API, India
Former Assistant Editor, Journal of Association of Physicians of India
Former Member of Syllabus Committee for Rheumatology, Medical Council of India
Former Chairman APICON 2016, Hyderabad.

MESSAGE

Dr (Prof) Alakendu Ghosh
Senior Consultant Rheumatologist
Institute of Neuroscience (I-NK), Kolkata

Dear Friends,

It is my great pleasure to welcome you to the WRF Master class sessions to be held at Raipur, where we unite to advance the frontiers of Rheumatology with a special focus on Myositis and Psoriatic arthritis. With world leaders in the specific fields around you, you will get the opportunities to enjoy the invaluable insights into managing these complex immune conditions - from novel biologics and JAKi to precision biomarkers and real-world trial outcomes. Together, let's forge the pathway to better outcomes for our patients.

Best Wishes
Dr Alakendu Ghosh

MESSAGE

Prof. Dr Shashank Gupta
Senior Consultant Physician
Ex-HOD, Department of Medicine
Pt JNM Medical College, Raipur

With great pride and optimism, I extend my warm welcome and greetings to all delegates, faculty, professionals, and students, both national and international, participating in this distinguished conference. I am honoured and excited to be associated with this wonderful gathering at Raipur.

We live in an era marked by rapid transformation and complex challenges. This conference marks profound significance as it serves not merely as a forum for presenting research but as a platform where ideas are questioned, refined, and transformed into solutions that shape the future. The spirit of real-world case discussions and masterly lectures from different faculty that defines this conference reflects our shared commitment to progress, responsibility, and lifelong learning. The discussions and interactions taking place here have the potential to inspire fresh perspectives, spark meaningful partnerships, and influence both thought and action far beyond the duration of this event. Each presentation and dialogue would contribute to a larger narrative of growth, innovation, and societal advancement.

I congratulate the organising committee, academic advisors, and volunteers for their vision, dedication, and tireless efforts. Their commitment has transformed an idea into a vibrant reality, providing participants with a platform that encourages excellence and intellectual courage. To all participants, I urge you to engage deeply, question boldly, and share generously. May this conference ignite new ideas, strengthen your resolve to contribute meaningfully to your fields, and inspire you to become catalysts of positive change.

I am confident that the insights gained and connections forged here will resonate long after the conference concludes. I wish the conference resounding success and hope it leaves each participant inspired, enriched, and empowered.

Prof. Dr Shashank Gupta
Senior Consultant Physician

MESSAGE

Dr Vinod Ravindran
MD, CCT-Rheumatology (UK), FRCP (Edin)
Adjunct Professor, Dept. of Medicine, Kasturba Medical College, MAHE, Manipal
Consultant Rheumatologist, Centre for Rheumatology, Calicut, Kerala
Co-Editor, *Rheumatology (Oxford)*
President-Elect, *Indian Rheumatology Association*

Dear Colleagues and Friends,

I welcome you to the World Rheumatology Forum (WRF) Summit 2026– Rheumatology Masterclasses, being held on 14th and 15th March at Raipur, Chhattisgarh.

I am happy that this much-anticipated biennial conference is taking place in Chhattisgarh, a unique state where I have spent much of my childhood. I am confident that this conference will strengthen the advocacy of Rheumatology in this part of our country by imparting knowledge and empowering the delegates through its renowned "*Masterclasses*" format.

This year, the themes of the conference are Myositis and Psoriatic Arthritis. The scientific committee led by Dr Sham S has put together an engaging scientific agenda with distinguished faculty from the UK and our own country. Presentations by the trainees and consultants showcase the breadth of rheumatology, highlighting its relevance and impact in making a difference to our patients' lives.

The organising team, led by Dr Arun Kumar Kedia, has worked tirelessly for the last several months to give you the most satisfying academic feast in a well-chosen venue.

I wish this conference a grand success.

Best wishes.

Dr Vinod Ravindran
President-Elect, *Indian Rheumatology Association*

MESSAGE

Dr Rajkiran D
Hon Secretary,
Indian Rheumatology Association (IRA)

It gives me immense pleasure to extend my warmest greetings to the organisers and participants of the **World Rheumatology Forum (WRF) Summit 2026**. As the Secretary of the **Indian Rheumatology Association (IRA)**, I am proud to see such high-calibre academic initiatives being hosted in Raipur.

The theme of this year's Masterclasses—**Myositis** and **Psoriatic Arthritis** addresses two of the most complex and rapidly evolving fields in our speciality. The emphasis on a multidisciplinary "Cross Talk" involving radiology, dermatology, and histopathology is particularly commendable, as it mirrors the collaborative approach required for the modern management of autoimmune diseases.

I am especially heartened by the summit's commitment to nurturing the next generation of rheumatologists. By providing a platform for trainees and young consultants to present abstracts and engage with international masters like **Dr Hector Chinoy** and **Dr William Tillett**, the WRF Summit is playing a vital role in advancing clinical excellence across India.

On behalf of the IRA, I congratulate the Organising Secretary, **Dr Arun Kumar Kedia**, the Scientific Chair, **Dr Sham S**, and the entire Raipur team for their tireless efforts in putting together this academic feast.

I wish the summit every success and trust that the insights gained here will translate into enhanced care for our patients.

Dr. Rajkiran Dudam
Hon Secretary,
Indian Rheumatology Association (IRA)

MESSAGE
From the Organizing Chair

Dr VP Pandey, MD, FRCP
Ex Dean and CEO, MGM Medical College, Indore
Ex Prof and HOD, Department of Medicine, MY hospital

Dear Esteemed Colleagues and Friends,

It is a distinct honor to invite you to the *World Rheumatology Forum (WRF) 2026* in Raipur, the vibrant capital of Chhattisgarh. This year, we aim to host a scientific extravaganza that perfectly balances rigorous academic updates with the tranquil hospitality of our region.

Our venue, the Mayfair Lake Resort, stands as a signature landmark of Raipur and will provide a memorable backdrop for our sessions. Beyond the conference, I encourage you to experience Chhattisgarh's lush forests and rich tribal heritage through planned pre- and post-conference tours.

Under the guidance of our Scientific Chair, Dr. S. Sham, and Organizing Secretary, Dr. Arun Kedia, we are dedicated to making this event a landmark in our professional journey. We invite you to join us with expanded time to fully immerse yourselves in the knowledge and culture of this beautiful state.

We look forward to welcoming you to Raipur for an experience that will stay with you for years to come.
Warm regards,

Dr V P Pandey
Organizing Chairperson, WRF 2026

MESSAGE

From the Organizing Secretary

Dr Arun Kumar Kedia
Senior Consultant, Director
Lifeworth Hospital, Raipur

It is with great joy, pride, and a deep sense of fulfilment that I welcome you to the World Rheumatology Forum Summit 2026, being hosted for the very first time in the beautiful city of Raipur, the capital of Chhattisgarh.

This conference holds special significance as it brings focused attention to Myositis and Psoriatic Arthritis—two complex, often challenging disease entities that continue to test our clinical acumen and inspire meaningful research. By centring our deliberations on these conditions, we hope to encourage deeper understanding, shared learning, and thoughtful dialogue that will ultimately translate into better patient care.

The true strength of this meeting lies in the coming together of an outstanding international and national faculty, experienced clinicians, researchers, and enthusiastic young rheumatologists. It is our earnest hope that the exchange of ideas here will spark new insights, foster collaborations, and open pathways for innovation in the diagnosis and management of inflammatory muscle and psoriatic diseases.

Hosting an international rheumatology conference in Raipur is a landmark moment for us. Alongside rich academic discussions, we hope you will take time to experience the warmth, culture, and hospitality that our city is known for, making your stay both intellectually enriching and personally memorable.

I extend my heartfelt gratitude to our faculty, organising committee members, sponsors, and all delegates whose support and commitment have made this conference a reality. Your presence truly gives life and meaning to this gathering.

I wish all participants a stimulating, rewarding, and memorable conference.

Dr Arun Kumar Kedia
Organising Secretary

MESSAGE
From the Joint Secretary

Dr Amit Dua
Consultant, Dua's clinic, Bilaspur and NH MMI Raipur.

It is with a deep sense of joy and gratitude that I write this forward for the souvenir of the "World Rheumatology Forum Summit 2026 – Rheumatology Masterclasses", being held at Mayfair Lake Resort, Raipur, on 14–15 March 2026.

This meeting, centred on myositis and psoriatic arthritis, brings together not just experts and trainees, but a community united by a shared purpose – to ease the burden of illness for our patients and their families. Every day in our clinics, we see individuals whose lives have been interrupted by pain, weakness, fatigue, disfigurement and uncertainty. Behind every CK value, every HRCT image and every swollen joint is a story of lost work, altered roles, and quiet resilience.

It is these stories that make our scientific pursuit meaningful and give heart to the countless hours we spend reading, discussing, and refining our approach to diagnosis and treatment.

The carefully crafted programme of this summit reflects that commitment. From state-of-the-art lectures on myositis and psoriatic arthritis, to cross-talks, multi-disciplinary workshops, radiology and neuropathology insights, case presentations and a fun rheumatology quiz, each session is designed to bridge knowledge and compassion, evidence and experience, science and the art of healing. I am especially inspired by the enthusiasm of our trainees and young consultants, whose cases and abstracts remind us that the future of rheumatology is in thoughtful and empathetic hands.

As Joint Secretary, I feel humbled to be part of a team that has worked tirelessly to shape this meeting – our patrons, senior advisors, organising and scientific committees, faculty, and numerous volunteers whose names may not always appear in print, but whose efforts are felt in every detail of the programme. I am equally thankful to each delegate who has travelled to Raipur, trusting that these two days will enrich their practice and, in turn, touch the lives of many patients they care for. I hope that when we look back at WRF Summit 2026, we will remember not only the lectures and discussions but also the sense of camaraderie, the informal conversations over tea, the moments when a difficult concept suddenly became clear, and the renewed resolve to stand by our patients through their most vulnerable times. May this souvenir capture those memories and serve as a gentle reminder of why we chose this path – to listen, to learn, and to heal.

With warm regards,

Dr. Amit Dua
Joint Secretary, WRF 2026

MESSAGE
From Scientific Chair

Dr S Sham
Consultant Rheumatologist
Kauvery Hospital (Alwarpet), Chennai.

Dear all,

It is my privilege to warmly welcome all participants to this scientific meeting dedicated to two major rheumatic diseases: **'Myositis' and 'Psoriatic Arthritis'**. These complex immune-mediated disorders, especially when accompanied by comorbidities and coexisting diseases, continue to challenge our clinical expertise and highlight the importance of advancing knowledge through rigorous academic engagement.

The scientific program has been carefully designed to foster both depth and breadth of learning through **"State-of-the-art lectures",** focused **"Masterclass sessions",** interactive **"Meet the Masters"** engagements, and interdisciplinary **"Cross-talks"** involving perspectives from dermatology, histopathology, and radiology over a day and a half. Such collaborative exchange reflects the multidisciplinary approach vital to contemporary rheumatology practice and research. The program also features young consultants presenting challenging cases that resonate with clinicians' daily practice. There is also a 'Fun-quiz' to lighten the mood. Additionally, more than 200 abstracts have been submitted by rheumatology and internal medicine trainees and consultants from across the country, showcasing their clinical work and promoting academic exchange.

I sincerely appreciate the distinguished faculty, members of various committees, and all participants, whose contributions are vital for the success of this academic endeavour. I also thank my co-chairs, Dr Kavitha MM, Dr Keethi Talari, and Dr Himanshu Pathak, for their support in curating the scientific programme and managing abstract submissions and evaluations.

I trust that the deliberations will be intellectually enriching and professionally rewarding for everyone involved.

Our motto shall be "Uniting expertise to decode complexity and deliver care."

Warm regards,

Dr S Sham
Scientific Chair, WRF 2026

MESSAGE
From Souvenir Team

Dr Madhuri H R
Sr Consultant
Star Hospitals, Nanakramguda
Hyderabad.

Dr Kushagra Gupta
Consultant Rheumatologist
Gupta Medical Centre
Hisar, Haryana

Dear Delegates
We welcome you to the World Rheumatology Forum 2026, at Raipur.

The theme for this issue is 'Psoriatic arthritis and Myositis'. As always, WRF conferences promise to be an academic extravaganza, with a focus on learning and knowledge sharing.

The scientific committee, expertly led by Dr Sham, has assembled a carefully curated panel of international faculty to share their expertise and experience. The scientific programme extends beyond conventional updates to address key interdisciplinary areas often considered the rheumatologist's Achilles' heel—namely dermatology, pathology, and imaging.

The organising team, skillfully led by Dr Arun Kedia, has worked tirelessly over the past several months to ensure the successful execution of a flawless conference.

Complementing the conference, this souvenir celebrates the rich cultural heritage of Chhattisgarh. In addition to conference highlights, we showcase the extra-academic talents of rheumatology colleagues from across India. We also present an overview of rheumatology patient support groups nationwide, offering resources that may benefit both patient care and clinical practice.

We invite you to explore our special section, “Mini-Rheum Reporter,” a delightful feature celebrating the creativity, imagination, and talent of the children of our rheumatology colleagues. This Gen Z–led section showcases their distinctive perspectives and emerging talents, and we are proud to highlight their achievements.

We wish you learn, enjoy and celebrate the camaraderie

Regards

Souvenir Team

SOUVENIR TEAM

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Hyderabad.

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SOUVENIR TEAM

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Dr Dheeraj Mittal
Rheumatologist & Clinical Immunologist Hathipole, Udaipur, Rajasthan

AWARD MESSAGE

Captain (Prof) Dr A N Chandrasekaran
Former Professor and Head
Department of Rheumatology, Madras Medical College
Former Director of Medical and Rural Health Services, Tamil Nadu
Past Editor-in-Chief, Indian Journal of Rheumatology
Recipient – Dr BC Roy Award & APLAR Master Award

It gives me great pleasure to extend my warm greetings to all delegates of the World Rheumatology Conference 2026. The coming together of clinicians, researchers, educators, and young minds from across the globe reflects not only the remarkable growth of our specialty, but also our shared commitment to advancing patient care through science, compassion, and collaboration.

Rheumatology has evolved tremendously over the decades—from an era of limited therapeutic options to one defined by precision medicine, translational research, and multidisciplinary care. Yet, at its core, our discipline continues to be anchored in careful clinical observation, thoughtful inquiry, and a deep understanding of patients' lived experiences. These principles remain as relevant today as ever.

I commend the organizing committee for their vision and tireless efforts in bringing together an outstanding scientific program. Conferences such as this provide an invaluable platform to exchange ideas, challenge existing paradigms, and inspire the next generation of rheumatologists. I am particularly encouraged by the active participation of young clinicians and researchers, whose curiosity and dedication will shape the future of our field. The award for the “Best case presentation by a young consultant” seeks to honour achievement while reaffirming the indispensable role of clinical research in shaping well-rounded physicians.

With best wishes for a stimulating and memorable meeting.
Dr A N Chandrasekaran

AWARD MESSAGE

Prof. Anand N. Malaviya,
Consultant Rheumatologist, 'A&R Clinic'
Visiting Sr. Consultant Rheumatologist,
ISIC Superspeciality Hospital, New Delhi.

It gives me great pleasure to extend my warm greetings to the organisers and participants of the WRF Summit 2026 in Raipur. Regional academic meetings, such as WRF, play a crucial role in nurturing a scientific temper, disseminating up-to-date knowledge, and strengthening the rheumatology community nationwide. I commend the organising team for their commitment to academic excellence and for creating a platform that brings together experienced clinicians, young rheumatologists, and trainees.

I am particularly touched by the continued recognition of the Prof. AN Malaviya Award for Best Poster Presentation, which was established to recognise and encourage trainees in Rheumatology and Internal Medicine. Such initiatives are vital; they not only reward merit but also instil confidence, curiosity, and a spirit of inquiry among young physicians early in their careers.

A Note to Young Trainees

Research is not merely about publishing papers; it is about asking the right clinical questions, observing carefully, and seeking honest answers that ultimately improve patient care. Good research often begins at the bedside, by noticing patterns, inconsistencies, or unmet needs in routine clinical practice. Even simple, well-conducted observational studies can be impactful when guided by clear thinking and methodological rigor.

When presenting your work—whether as a poster or an oral paper—remember that clarity outweighs complexity. Define your question clearly, explain why it matters, describe your methods transparently, and interpret your results thoughtfully while acknowledging limitations. Integrity in research and humility in interpretation are far more valuable than overstated conclusions.

Most importantly, remain curious, ethical, and persistent. Not every study will yield positive results, but every honest effort contributes to collective knowledge and sharpens your clinical reasoning. I hope the Prof. AN Malaviya Award serves not merely as a prize but as a reminder that thoughtful clinical research is integral to becoming a complete physician.

I wish the WRF Summit 2026 every success and hope it proves academically rewarding and personally enriching for all participants.

With best wishes,

Prof. A. N. Malaviya

MD, FRCP (Lond.), '*Master*'-ACR & APLAR, FACP, FICP, FAMS, FNASc,

({Retired} Head of the Department of Medicine, and Chief of Clinical Immunology and Rheumatology Services, All-India Institute of Medical Sciences, New Delhi)

AWARD MESSAGE

Prof VR Joshi
Former HOD Medicine, Rheumatologist
Direcor Of Research
PD Hinduja National Hospital
Mumbai

Dear Young Friends,
My compliments to you on participating in the WRF conference. I have been asked to address you on research—a challenging task, given the vastness of the subject and my own modest claims as a researcher. I will therefore approach it in a simple question-and-answer format.

Why do research?
Research is essential for progress. It benefits society and improves the quality of one's own work. It offers intellectual satisfaction and achievement, and while recognition may follow, it should never be the primary goal.

Who can do research?
The answer is simple: anyone. All clinicians are capable of meaningful research, even without sophisticated infrastructure.

What makes a good researcher?
Above all, curiosity. Leeuwenhoek, without formal medical training, built simple microscopes out of curiosity and discovered microbes, becoming the father of microbiology. The ability to observe and ask "why" is equally vital—Newton's falling apple remains a powerful metaphor. Questioning established dogma is another hallmark: Spallanzani disproved spontaneous generation using the simplest tools. Often, research begins with the urge to solve a problem, as Robert Koch did when he identified the cause of anthrax in a remote setting with minimal resources.

Perseverance is crucial. Paul Ehrlich tested 605 compounds before discovering Salvarsan for syphilis. Luck plays a role too, but only a prepared mind recognizes serendipity—Alexander Fleming identified the significance of penicillin because he had long been searching for antimicrobials.

Practical tips: Ask simple, practical questions; address one question at a time; respect folklore and traditional observations; remain alert to opportunities presented by new drugs and epidemics; and never dismiss unconventional ideas, as exemplified by Barry Marshall's work on *H. pylori*. Attend conferences to learn, share, and refine your work.

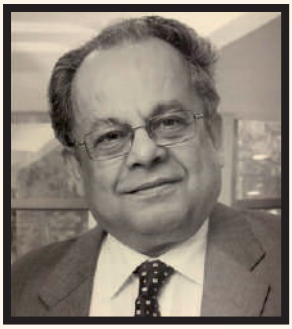
Finally, it is not enough to do research—it is a moral duty to publish both successes and failures.

Research begets more research and can become a lifelong companion in your rheumatology journey—one that constantly reshapes your understanding and practice. To quote Gelett Burgess, “If in the last few years you haven't discarded a major opinion or acquired a new one, check your pulse. You may be dead”.

The institution of this award in my name is received with gratitude and a deep sense of responsibility. Beyond being a prize, may this award serve as a lasting reminder that careful clinical inquiry lies at the heart of good doctoring.

With best wishes for a rich learning experience,
Dr V. R. Joshi

AWARD MESSAGE



Prof Sukumar Mukherjee
MD FRCP (Lond) FRCP (Edin) Hony D. Sc (CU)
Hony. D.Lett (Techno India Univ), FICP FSMF

Foreword

It gives me great pleasure to present the *World Rheumatology Forum Conference 2026*, being held in Raipur on 14–15 March 2026. This meeting brings together leading rheumatologists, researchers, and clinicians to exchange ideas, share experiences, and discuss the latest advances in the diagnosis and management of rheumatic diseases. The theme of this year's conference, **"Psoriatic Arthritis and Myositis,"** highlights two complex and evolving areas in rheumatology that continue to challenge clinicians while offering exciting opportunities for research and therapeutic innovation.

The scientific program has been carefully curated to address contemporary issues including early diagnosis, imaging, novel biomarkers, targeted therapies, and multidisciplinary management strategies. Through expert lectures, panel discussions, and interactive sessions, the conference aims to facilitate meaningful academic exchange and practical learning.

We extend our sincere appreciation to **Dr Arun Kedia**, Organising Secretary, whose dedication and meticulous planning have made this conference possible, and to **Dr Sham Santhanam**, Scientific Chair, for developing an outstanding academic program.

We hope this meeting fosters collaboration, inspires new research, and ultimately improves patient care in rheumatology. I wish the conference every success and an enriching experience for all participants.

Prof Sukumar Mukherjee

Ex-Prof and HOD of Medical College , Kolkata

FACULTY

Dr William Tillett
MBChB, BSc, PhD, FRCP
Clinical Lead, RNHRD, Royal United Hospitals Bath NHS Foundation Trust
Senior Lecturer, Department of Life Sciences and Centre for Therapeutic Innovation, University of Bath

Dr William Tillett is an Honorary Consultant Rheumatologist and Clinical Lead at the Royal National Hospital for Rheumatic Diseases (RNHRD), Royal United Hospitals Bath NHS Foundation Trust, and Senior Lecturer in the Department of Life Sciences and Centre for Therapeutic Innovation at the University of Bath. His work focuses on precision care in psoriatic arthritis—integrating clinical, radiographic, and patient-reported assessments to enable earlier detection and smarter treatment strategies.

He is Chief Investigator for a portfolio of clinical and commercial studies, including the Bath Psoriatic Arthritis Biobank, and has helped design and deliver multiple Phase III–IV drug-development and treatment-strategy trials. Dr Tillett **chairs the British Psoriatic Arthritis Consortium (BritPACT)**, serves on the GRAPPA steering committee, and chairs the peripheral-arthritis domain for the 2025 GRAPPA treatment-recommendations update. He **co-chaired the 2023 BSR PsA biologics guidelines, is European chair of the OMERACT PsA working group**, sits on the Journal of Rheumatology editorial board, and served on the EULAR task force to strengthen meaningful patient involvement. He is a medical advisor to several charities, including the Psoriasis Association and Psoriasis and Psoriatic Arthritis Association.

Take Home Message:

- Attendees will gain insights into recent advances in Psoriatic Arthritis treatment strategy, including the use of combination csDMARDS, early use of biologic therapy and the therapeutic pipeline
- Attendees will gain insights into the role of comorbidity and treatment selections in Psoriatic Arthritis. There will be a focus on obesity and the impact of non-pharmacological weight loss strategies.

Prof Hector Chinoy
Consultant Rheumatologist, Salford Royal
Professor of Rheumatology & Neuromuscular Disease,
University of Manchester

Professor Hector Chinoy is a Consultant Rheumatologist at Salford Royal and Professor of Rheumatology & Neuromuscular Disease at the University of Manchester. His clinical and research work focuses on autoimmune inflammatory myopathies, particularly the genetic and clinical determinants of disease subsets. He **leads the MRC-funded MYOPROSP prospective myositis cohort**, chairs the international MYONET registry, a global consortium that collects standardised clinical data, co-leads the UKMYONET network, and **co-led the recent British Society for Rheumatology myositis treatment guideline**. As of January 2026, he has 283 publications, 14,858 citations, and an h-index of 57.

KEY TAKEAWAY MESSAGES -

State-of-the-art Lecture 1: Therapeutic roadmap for Idiopathic Inflammatory Myositis

1. Stratify by serology and phenotype to help predict disease course and prognosis.
2. Act early and aggressively—avoid the "wait-and-see" approach; prompt immunosuppression from the outset improves steroid sparing and quality of life.

Masterclass I: Myositis-associated Interstitial Lung Disease

1. For ILD, adopt a specialist multidisciplinary team model (rheumatology + respiratory)
2. Ensure rapid assessment of anti-MDA5 status and ferritin to stratify RP-ILD risk.
3. Emerging therapies (anti-fibrotics, JAKi) are expanding the therapeutic arsenal, but robust M-ILD-specific trials remain an urgent need.

FACULTY

Dr Anita Mahadevan,
MD, FAMS
Professor & Head, Department of Neuropathology
Co-ordinator, Human Brain Tissue Repository (Brain Bank)
Officer in charge, Autoimmune Lab
National Institute of Mental Health & Neurosciences
Bangalore

Dr Anita Mahadevan is Professor and Head of the Department of Neuropathology at the National Institute of Mental Health and Neurosciences (NIMHANS), Bengaluru. She serves as the Coordinator of the Human Brain Bank and is the Officer-in-Charge of the Autoimmune Laboratory. With a passionate interest in autopsy-based neuropathology, her areas of expertise include neuroinfections, brain banking, autoimmune neurology, the biology of epilepsy, developmental neuropathology, and peripheral neuropathy. She has authored over 200 national and international publications and is a founder member of the Neuropathology Society of India.

Salient learning points:

Cross Talk 1: Interpreting Histopathology in Inflammatory Myositis

Despite advances in myositis-specific autoantibodies and imaging, muscle biopsy remains essential in the diagnostic pathway, particularly when non-invasive tests are negative or discordant with clinical suspicion. Myositis serology is highly specific (~94–99%) but has limited sensitivity (~40–60%), leaving a substantial proportion of patients seronegative.

Muscle biopsy provides direct tissue-level evidence of immune-mediated injury, including inflammatory infiltrates, necrotic fibres, perifascicular atrophy, and degenerative changes. It is critical for distinguishing dermatomyositis, immune-mediated necrotising myopathy, inclusion body myositis, and overlap myositis, and for excluding dystrophic, metabolic, and toxic mimics. In appropriate clinical contexts, biopsy sensitivity and specificity are typically ~70–85%.

Key take-home messages:

1. Muscle biopsy is indispensable in seronegative, atypical, or diagnostically discordant cases.
2. Histopathology provides subtype-defining and mechanistic insights beyond serology and MRI.
3. Ancillary techniques (MHC-I/II, MAC, and protein aggregation markers) significantly enhance diagnostic precision.

FACULTY

Dr Rajiv Sekhri
Senior Consultant
Fortis Noida, UP

Dr Rajiv Sekhri is a senior dermatologist and academician with a strong research focus, particularly in the field of autoimmune and immune-mediated dermatological disorders. His scholarly work has contributed to advancing the understanding of disease mechanisms, improving diagnostic precision, and optimising therapeutic strategies in complex skin diseases.

A major component of Dr Sekhri's research has centred on autoimmune dermatoses, encompassing their clinical spectrum, immunopathogenesis, and long-term management. His work reflects a sustained interest in translating immunological insights into practical, patient-centred care. Through clinical and translational research, he has examined treatment outcomes and evidence-based approaches to immunomodulatory therapies in chronic and relapsing conditions.

Dr Sekhri has authored over 50 peer-reviewed publications in national and international journals and has contributed scholarly articles and academic chapters that address both foundational concepts and emerging advances in dermatology. In addition to his research contributions, Dr Sekhri is actively involved in academic teaching and mentorship, fostering critical thinking and a research-driven approach among trainees. His academic philosophy emphasises the integration of clinical insight with systematic investigation, reinforcing the central role of research in advancing dermatological practice.

Salient learning points:
Psoriasis is a common dermatologic condition best handled by dermatologists. It has a varied skin presentation and needs special attention in the management of some of its severe variants. The clinical presentation provides clues to the diagnosis, based on characteristic clinical features.

Identifying and managing triggers:
Psoriasis occurs in 1-3% of the population. The disease is transmitted genetically. Exact triggers are not identifiable. Maybe streptococcal infection, stress, some environmental factor, physical trauma (scratching, sunburn, or surgery), drugs (lithium, beta blockers, anti-malarial), or alcohol consumption act as triggers.

Look for the comorbidities such as arthritis, Crohn's disease & ulcerative colitis, heart disease, diabetes, cancer, and hypertension for better management.

Key Take-home message:
It is desirable to take dermatology opinion when the diagnosis is not certain, or the disease is severe e.g. pustular / erythrodermic psoriasis.

FACULTY

Dr Khushboo Pilania Agrawal
MBBS, MD (Radiodiagnosis), FIVR
Founder & Consultant Radiologist
Izen Imaging and Interventions
Noida, India

Dr Khushboo Pilania Agrawal is a practising radiologist with focused subspecialty expertise in **musculoskeletal radiology and chest imaging**, with a particular academic and clinical interest in **connective tissue disease–associated interstitial lung disease (CTD-ILD) and inflammatory myopathies**. She is the Founder and Consultant Radiologist at Izen Imaging and Interventions, a comprehensive multimodality diagnostic centre in Noida, India, providing advanced MRI and CT imaging with an emphasis on protocol optimisation and multidisciplinary care.

She has been actively involved in ILD research through national registries and guideline committees and has authored and co-authored several peer-reviewed publications on **interstitial lung disease, inflammatory myopathies, and advanced MRI techniques**, including a dedicated review on the role of MRI in idiopathic inflammatory myopathies and multiple consensus and registry-based ILD publications.At this conference, Dr Pilania presents a **practical, pattern-based imaging approach** tailored for rheumatology practice.

From an HRCT perspective, she emphasises that CTD-ILD is often the **earliest or dominant disease manifestation**, sometimes preceding overt systemic or muscular symptoms. Structured HRCT interpretation—when combined with optimal acquisition techniques—allows meaningful differentiation between **active inflammation and irreversible fibrosis**, directly influencing treatment decisions, therapeutic responsiveness, and prognosis.

A major focus of her work is the evolving role of **muscle MRI in inflammatory myopathies**. Myositis MRI has emerged as a reasonably sensitive **imaging biomarker** for early disease detection, phenotyping, and **accurate localisation for biopsy**, often before clinical weakness becomes apparent. When performed using a **dedicated myositis protocol**, it adds substantial value in baseline assessment, treatment planning, and follow-up. While still on a learning curve, its role in routine rheumatology practice continues to expand, supported by growing evidence and clinical experience.

Dr Pilania strongly advocates that **early diagnosis remains the most critical modifiable factor** in improving outcomes in CTD-ILD and inflammatory myopathies, and that close collaboration between rheumatologists and radiologists is essential to achieve this goal.

Key Take-Home Messages for Rheumatologists (Radiology Perspective)

- CTD-ILD may precede systemic or muscular symptoms; **HRCT is often the first diagnostic clue**
- **NSIP and NSIP–OP overlap patterns** are strong imaging indicators of CTD and myositis-associated ILD, respectively.
- Distinguishing **inflammation from fibrosis on HRCT** directly impacts treatment strategy
- **Myositis MRI is a powerful tool for early diagnosis, phenotyping, and biopsy guidance**
- A **dedicated myositis MRI protocol** is essential—routine MRI is inadequate
- MRI plays a valuable role in **baseline assessment, prognostication, and follow-up**
- Early, protocol-driven imaging combined with multidisciplinary discussion **changes outcomes**

EXPLORING CHHATTISGARH

EXPLORING CHHATTISHGARH

"अरपा पैरी के धार, महानदी हे अपार
इन्द्रावती हा पखारे तोर पईया,
महूं पांव परौ तोर भुँइया
जय हो जय हो छत्तीसगढ़ मईया...."

Written and composed by **Dr. Narendra Dev Verma**, a noted Chhattisgarhi composer and literary figure. His song was officially adopted as the **state song of Chhattisgarh in November 2019** and is often sung at cultural and official events

These opening lines of *"Arpa Pairi Ke Dhar"* pay tribute to **Chhattisgarh's life-giving rivers** — the Arpa and Pairi — alongside the mighty Mahanadi and the nurturing Indravati. The song is a heartfelt **prayer to the land itself**, lovingly calling it *Chhattisgarh Maiyā* (Mother Chhattisgarh).

In just a few words, it captures the **richness of the soil, the generosity of the rivers, and the deep pride of its people**

- Dr Ashlesha Shukla , DM
Assistant Professor, DKS & PGI Research Center, Raipur (C.G)

THE LAND OF THIRTY-SIX FORTS

Dr. N.Kavya Devi
MBBS MD DM (Rheumatology- NIMS)
Consultant Rheumatologist, Anvitha Hospitals ,Andhra Pradesh

Long before it became a state, this land was shaped by its people, rivers, and forts. Dotted with **thirty-six strongholds**, each guarding villages, trade routes, and forests, the region came to be known as **Chhattisgarh — the land of thirty-six forts**.

A Century-Wise Journey Through Time

1. Prehistoric Period (Before 3rd Century BCE)

Archaeological evidence confirms that **human civilisation existed in Chhattisgarh thousands of years ago**. Stone tools and early habitation sites found across the region indicate continuous human presence long before recorded history.

Singhanpur revealing Stone Age tools and cave paintings, proving prehistoric human habitation

2. Epic Age & Early Historic Period (c. 1500 BCE – 3rd Century BCE)

Known in ancient texts as **Dakshin Kosala**, the region is mentioned in the *Ramayana* and *Mahabharata*. It is traditionally believed to be the **native land of Kausalya**, mother of Lord Rama, and a part of the forests where **Rama spent years of exile**.

Dakhin Kosala- The ancient identity of Chattisgarh

During this period, Dakshin Kosala evolved into a **spiritual confluence of Buddhism, Shaivism, and Vaishnavism**, laying the foundation for its plural religious identity.

3. 3rd Century BCE

The **Sitabengra Cave** in the Ramgarh Hills dates to this period and is regarded as one of the **earliest theatre-like structures (Natyasala)** in India, complete with carved seating. Local tradition associates the cave with **Sita's residence during exile**.

Sitabengra caves- an ancient Indian theatre

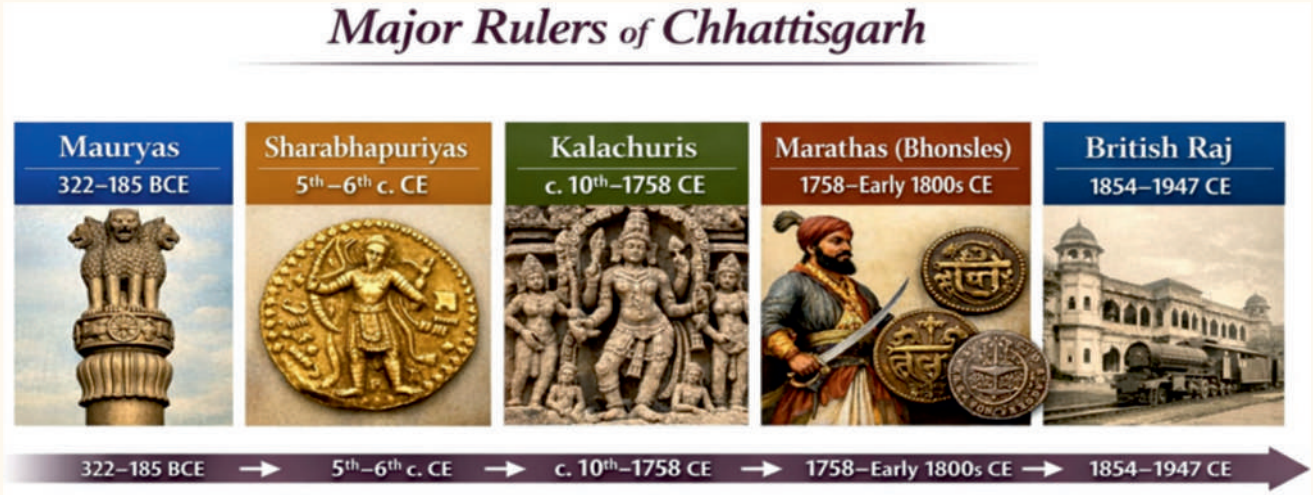
Nearby, the **Jogimara Caves** preserve one of the **earliest Brahmi inscriptions**, believed to record a personal love message between an artist and a dancer—offering rare insight into early art, theatre, and emotional expression.

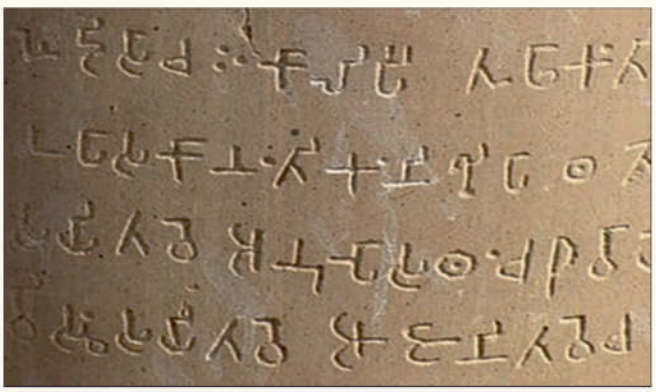
Jogimara caves- A rare window into artistic expression

4. 4th–6th Century CE | Sharabhapuriya Dynasty

The **Sharabhapuriya rulers** established Sirpur as their capital. Their reign is marked by **Sanskrit inscriptions in early Brahmi–Gupta script** and the issuance of **gold coins**, among the earliest in central India. These reflect a **well-organised agrarian economy and administrative system**.

Sirpur emerged as a major **Hindu, Buddhist, and Jain centre**, highlighting the region's religious inclusivity.





Sharabhapuriya inscription, - Early Sanskrit epigraphy of Dakshin Kosala

Sharabhapuriya gold coin - Evidence of early monetised economy

*The iconic **Laxman Temple at Sirpur**,*

*an outstanding brick temple dedicated to Lord Vishnu, was built by **Queen Vasata**, mother of King Mahashivagupta Balarjuna, in memory of her husband King Harshagupta. The temple remains a rare surviving example of early brick temple architecture.*

5. 9th–12th Century CE | Kalachuri Dynasty

The **Kalachuris**, the longest-ruling dynasty of the region, brought political stability and cultural prosperity. **Ratanpur** served as their administrative and military centre, emerging as a hub of governance and trade.

Kalachuris of Tripuri (10th-18th Century)

The Kalachuris of Tripuri *Capital at Ratanpur* *Temples and Forts Construction*

During this period, Chhattisgarh earned the title **"Kashi of the South"** due to extensive temple construction. Notable monuments include:

- **Bhoramdeo Temple**, dedicated to Lord Shiva, known for its Nagara-style architecture and sculptural richness, earning it the title *"Khajuraho of Chhattisgarh"*

- **Madwa Mahal**, believed to be a royal ceremonial or marriage pavilion

- **Mahamaya Temple**, a major Shakti Peeth symbolising the deep-rooted Shakta tradition of the region

"Whispers of ancient stone, where shiva resides in serenity"- Bhoramdeo temple

Madwa Mahal- a graceful symbol of Chhattisgarh's rich cultural and architectural heritage.

Mahamaya Temple, Ratanpur
– A sacred shakti peeth of Chhattisgarh

6. 18th Century | Maratha Rule

In the 18th century, Chhattisgarh came under the control of the Bhonsles of Nagpur. Maratha administration focused primarily on revenue and governance. **They built 36 forts in and around Chhattisgarh to guard their territory from the Mughals giving it the name Chhattisgarh.** Raipur developed into an important administrative and commercial center during this period.

Chhattisgarh is one of the few states that was never ruled by the Mughals As such most of the temples and ancient architecture can be found intact in this region

7. 19th–Early 20th Century | British Colonial Period

After the **Third Anglo-Maratha War (1818)** and the **Battle of Raipur**, Chhattisgarh became part of the **British-administered Central Provinces**. The British built **railways, administrative reforms, and new revenue systems**, improving the region's infrastructure.

The **demand for a separate Chhattisgarh state** began gaining momentum in the **1920s**, rooted in cultural and regional identity.

Raipur as the Maratha Hub *Battle of Raipur, 1817 - Clash of Empires* *Raipur Under British Rule*

8. 21st Century | Formation of the State (2000 CE)

After decades of political movements and negotiations, **Chhattisgarh was carved out of Madhya Pradesh on 1 November 2000**, becoming the **26th state of India**. This marked a historic transition toward regional self-governance and development

THE SEAHORSE OF THE PENINSULA

Dr Naman Jain,MD, DrNB
Consultant Rheumatologist, Ram krishna Care hospitals, Raipur, C.G

A Geographical profile of Chhattisgarh

Born at the turn of the millennium in the year 2000, Chhattisgarh was carved out of Madhya Pradesh to forge its own identity as the "**Mineral powerhouse"** and the **"Rice Bowl of India"**. With a geographical area of approximately 1,35,191 square kilometers, it constitutes roughly 4.1% of India's total landmass.

What immediately strikes the observer is the state's unique cartographic silhouette. Resembling a **Sea Horse (Hippocampus)**, Chhattisgarh stretches across the heart of the country, bridging the distinct cultures of the north and the south.

Feature	Details
Establishment	November 1, 2000 (Separated from MP)
Total Area	1,35,191 sq km (4.1% of India)
Shape	Sea Horse (Hippocampus)
Forest Cover	44.25% (≈59,820 km²)
Neighbors	7 States Longest Border: Odisha Shortest Border: Andhra Pradesh
Major Rock Types	Archean (Granite), Dharwar (Iron), Gondwana (Coal)
Major Rivers	Mahanadi, Shivnath, Indravati, Hasdeo

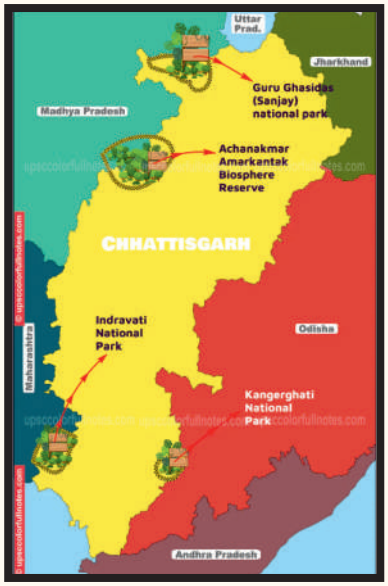
Location & Extent

- Situated on the **Peninsular Plateau**
- Tropic of Cancer (23°30' N) passes through the state
- The state is divided into three distinct agro-ecological zones:
    - The Northern Hills (Surguja region)
    - The fertile Central Plains (Mahanadi basin)
    - The Bastar Plateau in the south.

- **The central plains are so fertile for paddy cultivation that the state is widely nicknamed the "Rice Bowl of India".**

44% area of Chhattisgarh is covered by Forests making it one of the most distinct biodiverse regions in India.

Largest Green Cover



- **44.25% of area** (~59,820 km²) covered in dense forests
- Supports rich biodiversity and climate regulation
- 3 national parks
- The Achanakmar-Amarkantak Biosphere Reserve is the primary biosphere reserve in Chhattisgarh, established in 2005 and recognized by UNESCO in 2012. The reserve hosts over 1,500 plant species and endangered fauna, including the Bengal tiger, Indian wild dog, and sarus crane

Geology & Soil

- **Rock Systems:**
  - Archean (Granite, Mica, 50%)
  - Dharwar (Iron ore, Tin, Corundum, South)
  - Cuddapah (Limestone, Dolomite, Central)
  - Gondwana (Coal, North & East)
  - Deccan Trap (Bauxite, Black Soil)
  - Vindhyan Series

- **Soil Types:** Black, Red-Yellow, Red-Sandy, Laterite, Mixed

It is the only Indian state that produces Tin concentrates. It also holds massive reserves of coal, iron ore, and limestone, contributing significantly to India's steel and power production.

Rivers: The Lifelines

- **Mahanadi river**: The Mahanadi isn't just a river; it's the state's lifeline. Legend says the Mahanadi River was **created when the warrior sage Parashurama struck the earth with his axe**. Today, it is known as one of the **most active silt-depositing** rivers in the world. Along its banks lie holy sites like **Rajim** (the "Prayag of Chhattisgarh") and **Sirpur**, where ancient temples mirror the state's long geological and cultural history.
- **Shivnath river**: The Shivnath River holds a controversial place in history as the first river in India to have a portion of its water rights **sold to a private company** in 1998. It is also the longest tributary of the Mahanadi.
- **Indravati river**: Also known as the **'Ganga of Bastar'**, the Indravati River is the spiritual heart of southern Chhattisgarh. It is home to the **Chitrakote Falls**

CRAFTS AND CRAVINGS

Dr. Pranavi Vanga MD, DM
Consultant Rheumatologist at Kokilaben Dhirubhai Ambani Hospital, Indore, M.P

From the glint of **golden Kosa silk** to the fiery tang of **red ant chutney**, Chhattisgarh is a land where **tradition and creativity dance together**, and every artifact and dish tells a story of people deeply in tune with their land.

Famous & Exclusive Crafts of Chhattisgarh

1. Dhokra Metal Craft

- One of India's oldest surviving metal art traditions
- Uses the **lost-wax casting technique**
- Unique tribal motifs: deities, dancers, animals, rural scenes
- Almost entirely concentrated in Bastar and surrounding tribal regions

Dhokra metal craft depicting the tribal life style and culture

2. Bastar Tribal Art

- Sculptures, masks, and ritualistic objects in **iron, wood, and bell metal**
- Motifs inspired by **folklore, hunting, festivals, and forest spirits**
- Ritualistic significance makes it culturally exclusive

Bastar Metal Artwork

3. Kosa Silk (Golden Silk)

- Wild silk from **Antheraea mylitta silkworms** native to Chhattisgarh forests
- Hand-spun and handwoven in **Champa, Bastar, and Korba**
- Renowned for **natural golden sheen, durability, and rich texture**
- Symbol of sustainable textile heritage

Kosa silk sarees made from Tussar Silk

Tussar Silk Art Over Map of Chhattisgarh

4. Terracotta & Clay Work (Bastar & Raipur)

- Handmade pots, lamps, idols, and decorative items
- Earthy textures, natural tones
- Used in daily life and sacred rituals

Artist from Sarguga Making terracotta artworks

5. Bamboo & Cane Crafts

- Functional and decorative baskets, traps, and tools
- Handcrafted weaving style **unique to tribal regions**

Women making Bamboo cane products

Suntainable bamboo cane products

THE CULINARY SOUL OF CHHATTISGARH

A Journey Through Taste:

Chhattisgarh's cuisine is more than food—it's a **story of the land, its forests, and its people**. Every bite celebrates **tradition, generosity, and harmony with nature**, from a simple bowl of Bore Baasi to the golden sheen of Mahua sweets.

In Chhattisgarh, food is **simple, nourishing, and deeply tied to the land**.

Rice: The Heart of the Diet

Chhattisgarh consumes rice in countless ways: boiled, fermented, steamed, or ground into flour. It forms the **foundation of daily meals** and the soul of traditional dishes

Chhattisgarh is known as the Rice Bowl of India home to 23,000 different indigenous rice varieties.

Different Rice Delights

- **Chila:** Soft rice pancakes served with chutney or curd
- **Faraa:** Steamed rice dumplings, light and wholesome
- **Bafauri:** Protein-rich steamed chana dal balls, full of flavor
- **Angakar Roti:** Thick, traditional rice flatbread cooked on a clay griddle, eaten with chutneys or vegetable curries.
- **Bore Baasi:** Fermented rice soaked overnight, eaten cold with chutney or yogurt—an **age-old rural breakfast** perfect for hot summers

Various Rice delicacies staple in Chhattisgarh

Farra the regular breakfast

Traditional Bore baasi platter

Making Traditional Angaar roti

Angaar roti served with Dhaniya & Tamatar chutney - The Traditional way

Forest & Seasonal Flavors

- **Mahua flowers** used to make a local, aromatic alcoholic beverage
- **Tamarind and wild greens** add tang and nutrition

  Meals closely follow **agriculture and forest cycles**, connecting people to the land.

Mahua flowers picked up by lady

Making of Mahua the desi drink

Festive Sweets

- **Dehrori (**A soft, mildly sweet **fermented rice dessert**, often made with curd. Light, nourishing, and traditionally prepared during festivals or special occasions.
- **Thetri & Khurmi:** A **crisp, deep-fried sweet**, enjoyed during celebrations. Sweet, crunchy, and full of festive flavor, it is a staple treat in Chhattisgarh's culinary heritage

Dehori made often in Diwali

Thetri & Khurmi often made on occasion of Pora Festival

Chaprah: The Red Ant Chutney

A culinary icon of Chhattisgarh, Chaprah reflects **tribal knowledge and ecological wisdom**:

- Made with **red ants and their eggs**, blended with chilies, garlic, coriander, and local spices
- Sharp, tangy (citrus), fiery, and protein-rich
- Symbolizes **fearless culinary heritage** and sustainable living

Chaprah: The red ant Chutney is a culinary staple of Chhattisgarh. It tastes tangy and citrusy like lemongrass giving it a fresh feel

Raw ants

The process of making Chutney

The Final Product

Dr. Sujata Elizabeth Mathews
Professor, Medicine, ABVIMS, Dr.RML Hospital, Delhi
Dr Ashlesha Shukla, DM,
Assistant Professor, DKS & PGI Research Center, Raipur

Chhattisgarh Tourism: Full of Surprises

Tucked in the heart of India, Chhattisgarh reveals a world of **roaring waterfalls, sacred rivers, lush forests, vibrant tribal culture, historic temples, artistic heritage, and offbeat landscapes** — all waiting to be explored. Far from the usual tourist trail, this state blends **nature, spirituality, history, and culture** into one unforgettable journey

1. Cascading Waterfalls

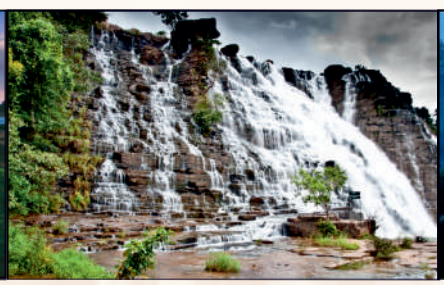
**Chitrakote waterfalls is the widest river in India.
It changes its character with seasons, from thundering brown in winters to blue streams in the winters**

- **Chitrakote Falls: Widest waterfall of India**, also known as the ***Niagara of India***, this horseshoe-shaped cascade creates rainbow-filled mist for mesmerising views. It changes character entirely with the seasons—from a thundering brown "Niagara" in the monsoon to multiple clear, blue streams in the winter.

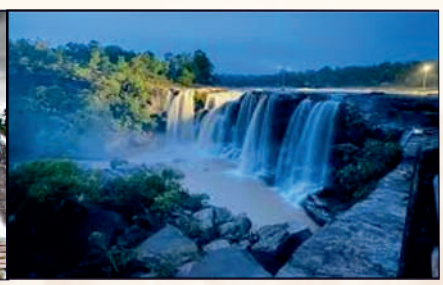
- **Tirathgarh & Jatmai Waterfalls:** Multi-tiered cascades nestled in forested valleys, ideal for monsoon visits and trekking.

- **Amritdhara Falls & Forest Streams:** Hidden gems for nature lovers..

Chitrakoot Waterfall
Jagdalpur



Tirathgarh Waterfall
Kanger Valley



Amritdhara Waterfall
Baikunthpur

2. Hills, Plateaus & Offbeat Landscapes

- **Mainpat** – "Mini Tibet" or the "Shimla of Chhattisgarh"- On the scenic **plateau of Mainpat**, the hills seem alive.

The "Dancing Mountains" get their name from rocks that vibrate or hum when walked upon, a mystical natural phenomenon.

Surrounded by lush forests and tribal villages, the area blends **nature, folklore, and geology**. Visitors can walk among the rocks, explore tribal culture, and witness one of Chhattisgarh's most intriguing landscapes.

Tiger Point at Mainpat

Dancing Mountains (Dalha Pahad), Mainpat

Madku Island (Bilaspur): A frog-shaped river island with ancient temples and Mandukya Rishi memorial

World's Largest Shivling Mountain Madheswar at Jashpur

Mandukya rishi memorial, Madku Island

Historical Site at Madky Island, Mungeli

3. Spiritual Sojourn: Temples & Pilgrimage Trails

- **Danteshwari Mandir & Bastar Dussehra – The Heart of Bastar**

Nestled in **Jagdalpur**, the **Danteshwari Mandir** is one of India's **18 Shakti Peethas**, sacred shrines dedicated to Goddess Shakti. Legend says it marks the spot where a part of **Sati Devi's body fell**, making it a powerful center of **spiritual energy and devotion**. Pilgrims from across the country visit to seek blessings, especially during **Bastar Dussehra**, a **75-day festival** that celebrates tribal traditions, rituals, and community life in all its grandeur.

Bastar Dussehra is one of the longest festivals lasting 75 days
Royal family still participates in the ceremonies at the temple

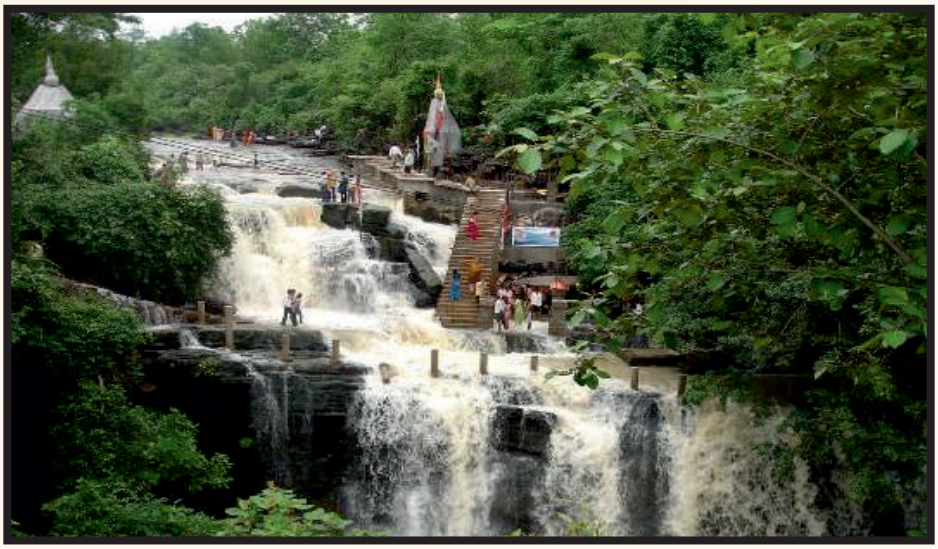
The festival itself is unique — unlike typical Dussehra celebrations, it is deeply rooted in Bastar's **tribal heritage**. Historically, the **King of Bastar** played a central role in organizing the festivities, overseeing rituals, and preserving customs. Even today, the royal family participates in ceremonies at the temple, symbolizing the **enduring bond between monarchy, religion, and tribal culture**

Dantesehwari Shaktipeeth, Dantewada

75 Days Longest Festival Bastar Dusshehra

- **Temple (Kawardha):** Often called the Khajuraho of Chhattisgarh for its exquisite stone carvings.

- **Jatmai Mata Temple:** Picturesque shrine beside natural cascades; vibrant during Navratri. at Raipur



- **Shivrinarayan:** A sacred town on the banks of the Mahanadi River, Shivrinarayan is steeped in mythology and tranquility. Believed to be the place where Lord Rama, Sita, and Lakshmana stayed during their exile, it is home to the ancient **Shivrinarayan Temple** and offers a serene confluence of history, spirituality, and riverside beauty.

Shivrinarayan Ghat on the banks of Mahanadi

Shivrinarayan Temple at Janjgir Champa

- **Rajim:** Known as the *"Prayag of Chhattisgarh"*, Rajim is famed for its **Triveni Sangam**, where the Mahanadi, Pairi, and Sondur rivers meet. The town is dotted with intricately carved temples, hosting vibrant festivals like the **Rajim Kumbh**, making it a hub of devotion, culture, and riverside charm.

- At the heart of Rajim stands the **Rajiv Lochan Temple**, an **8th-century Vishnu temple** known for its stunning stone carvings, intricately sculpted pillars, and historic charm. Built by the Nala dynasty, this ancient shrine draws thousands of devotees each year, who come to seek blessings and experience its **timeless architectural grandeur**

Rajiv Lochan Temple

Glimpse of Punni Mela,
Rajim Kumbh at Gariabandh

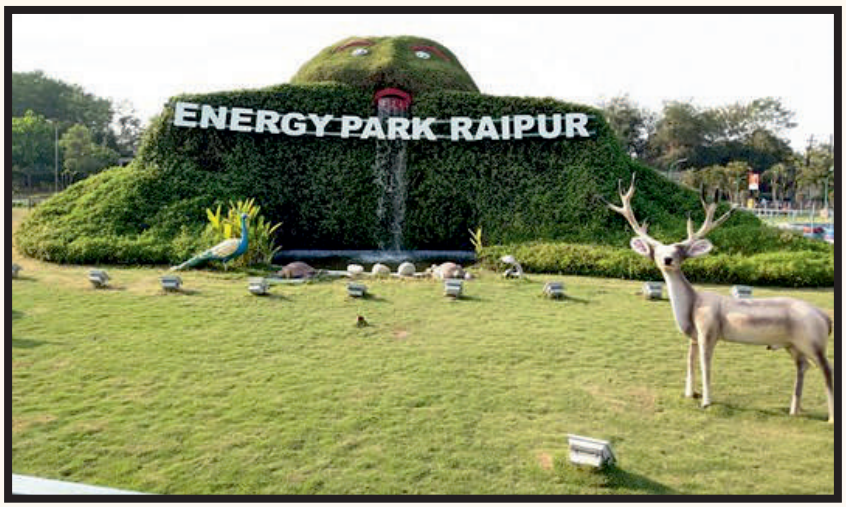
4. Artistic & Cultural Heritage

- **Indira Kala Sangeet Vishwavidyalaya (Khairagarh):** India's prestigious music and performing arts university, shaping classical arts and cultural research.

- **Purkhouti Muktangan (Naya Raipur):** Open-air cultural garden displaying life-size tribal art, folk traditions, and artifacts.

- **Museum & Cultural Hubs (Raipur):** Mahant Ghasidas Memorial Museum and Digital Tribal Museum showcase history, art, and anthropology.

- **Energy Park(Urja park):** A recreational science park in Raipur with boating and renewable energy exhibits at Raipur.



5. Living Traditions: A Homestay Story from Chhattisgarh

Under the **Government of Chhattisgarh's Homestay Programme 2025**, families in **Bastar and Surguja** now welcome travellers into their homes, giving the authentic tribal experience. They share everyday life through local cuisine, crafts, customs, and stories shaped by the forest.

Visitors live alongside their hosts — walking the land, cooking together, and learning traditions by doing, not observing.

HOMESTAYS REVOLUTION

Mahua Homestay
Kere, Jashpur

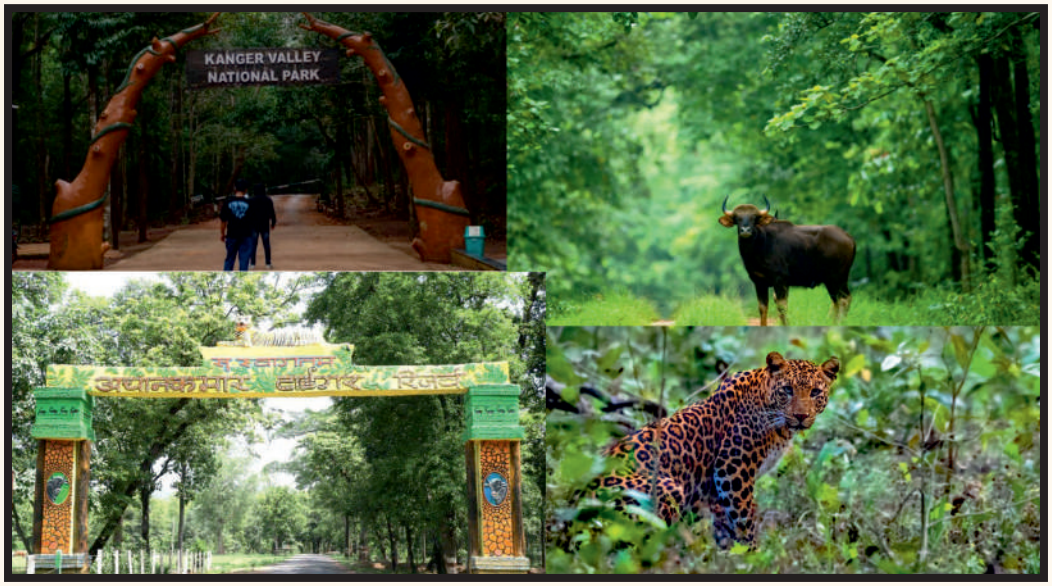
SonBhadra Homestay
Pendra (Janjgir-Champa)

Mystique Village Homestay
Chhorebodal, Bastar

Jhonjha Village Homestay
Gaurela, GPM District

6. Wildlife & National Parks

- **Kanger Ghati National Park:** Dense forests, limestone caves (Kotumsar), waterfalls; home to **bison, leopards, and the Hill Myna**.
- **Barnawapara Wildlife Sanctuary:** Jeep safaris through **sal and teak forests**; spot leopards, bison, and migratory birds.
- **Achanakmar Tiger Reserve:** Tropical deciduous forest with **tigers, leopards, and over 600 medicinal plant species**



7. Dhudmaras Village – UNWTO Best Tourism Village

In the heart of **Kanger Valley National Park**, Dhudmaras shines as a **UNWTO Best Tourism Village**, chosen from over 260 applications worldwide. Here, the **Dhurwa Tribe** welcomes visitors with kayaking, bamboo rafting, trekking, and traditional home stays. Forests, festivals, crafts, and vibrant tribal life come alive, showing how culture, nature, and community-led tourism coexist beautifully.

Dhudmaras, an ecotourism village was recognized as Best tourism village by UNWTO in 2024, famous for its homestays

SCIENTIFIC PROGRAMME

Day 1 – Saturday, 14th March 2026 | Session I

09:00 am - 10:00 am — **Abstract Presentation by MD Trainees**
Chairs : Mukhyaprana Prabhu (Manipal), Sunil Kumar Singh (Mumbai), Meghna Gavali (Hyderabad)
Judges : Ashish Badika (Indore), Ghanshyam Pangtey (New Delhi), Pratyusha Rajavarapu (Guntur)

10.00 am - 10.45 am — **State of the Art Lecture-1**
Chairs : Sukumar Mukherjee (Kolkata), KM Mahendranath (Bengaluru), URK Rao (Hyderabad), Jyotsna Oak (Mumbai)

10:00-10:30 am	**Therapeutic roadmap for Idiopathic Inflammatory Myositis** · Genetics, autoantibodies and clinical implications · Choosing between targeted DMARDs · Refractory muscle disease	**Hector Chinoy (UK)**
10:30-10:45 am	Q & A	

10.45 am - 11.30 am — **Challenging Cases 1**
Chairs : Krishnamurthy V (Chennai), Smruti Ramteke (Nagpur), Rajendra Varaprasad (Hyderabad)
Judges : Jyoti Ranjan Parida (Bhubaneshwar), Narendra Kumar Bagri (New Delhi), Spoorthy Kothapalli (Hyderabad)

10:45-11:10 am	**An Unyielding Enemy** **Wolf in Brown Clothing** **Serendipity and a therapeutic answer**	**Kavya Devi (Vijayawada)** **Sanket Shah (Ahmedabad)** **Pallavi Pimpale (Mumbai)**
11:10-11:30 am	Discussion and Q &A Discussants	**Hector Chinoy** **Anita Mahadevan**

11:30-12:00 pm — **Inauguration**
Tea-break

12.00 pm - 12.30 pm — **Cross Talk 1**
Chairs : Able Lawrence (Lucknow), Lalit Duggal (New Delhi), Manish Bavaliya (Rajkot)

12:00-12:20 pm	**Interpreting histopathology in inflammatory myositis** · Histopathologic spectrum · Differentiating features · Tips for sampling and transport	**Anita Mahadevan (India)**
12:20-12:30pm	Q & A	

12.30 pm - 1.15 pm — **Challenging Cases 2**
Chairs : Madhumita Priyadarshini Das (Guwahati), Paramjeet Singh (Haldwani), Mithun CB (Cochin)
Judges : Divya Agarwal (New Delhi), Lubna Khurshid (Srinagar), Nachiket Kulkarni (Pune)

12:30-12.55 pm	**Rare and scary** **Trigger, Mimic or Coincidence** **The face tells a different story**	**Sandeep Yadav (Mumbai)** **Shubha Bhalla (New Delhi)** **Shivraj Padiyar U (Mangalore)**
12.55-01:15pm	Discussion and Q &A Discussants	**Hector Chinoy** **Anita Mahadevan**

01:15-02:30pm — **Lunch and Poster Viewing**

SCIENTIFIC PROGRAMME

Day 1 – Saturday, 14th March 2026 | Session II

2.30 pm - 3.15 pm — **State of the Art Lecture-2**
Chairs : AN Malaviya (New Delhi), Porkodi R (Chennai), Alakendu Ghosh (Kolkata)

02:30-03:00 pm	**Selecting drugs to optimise outcomes in Psoriatic arthritis** • Treatment strategy • Differential efficacy of targeted DMARDs • Response to advanced lines of therapy	**William Tillett (UK)**
03:00-03:15 pm	Q & A	

3.15 pm - 4.00 pm — **Challenging Cases 3**
Chairs : Arul Raja Murugan (Chennai), Kavita Krishna (Pune), Varun Dhir (Chandigarh)
Judges : Ajit Nalawade (Pune), Aarti Sharma (New Delhi), Dantis Emmanuel (Alappuzha)

03:15-03:40 pm	**A Real-World Challenge** **The road to the Axis** **Ammunition running out, and a war to wage**	**Saranya C (Chennai)** **Sree Devi S (Trivandrum)** **Raghavendra H (Bangalore)**
03:40-04:00 pm	Discussion and Q &A Discussants	**William Tillett** **Rajiv Sekhri**

04:00-04:30 pm — **Tea and Poster Judging**

4.30 pm - 5.00 pm — **Cross Talk 2**
Chairs : Uma Kumar (New Delhi), Indrajit Agrawal (Gurugram), Shashank Akerkar (Mumbai)

04:30-04:50 pm	**Psoriasis – what a rheumatologist should know?** • Clues to early diagnosis of Psoriasis • Identifying and managing triggers • Prioritizing dermatology referral – when?	**Rajiv Sekhri (India)**
0.4:50-05.00 pm	Q & A	

5.00 pm - 5.45 pm — **Challenging Cases 4**
Chairs : Ravichandran R (Chennai), Gurmeet Singh (Jammu), Shivaprasad BN (Mysuru)
Judges : Amit Kansal (Ludhiana), Abraham Mohan (Kottayam), Natasha Negalur More (Mumbai)

05:00-05:25pm	**Overcoming a Herculean Task** **Takes two to tango** **Surviving the therapy, not the disease**	**Kasturi Hazarika (Guwahati)** **Nikhil Gupta (New Delhi)** **Vishnu Priya S (Salem)**
05:25-05:45pm	Discussion and Q &A Discussants	**William Tillett** **Rajiv Sekhri**

5.45 pm - 6.30 pm — **FUN QUIZ**
Chairs : Ajaz Kariem Khan (Srinagar), Kiran Seth (Noida), Vijaya Prasanna (Hyderabad)
Quiz Master : Sri Anusha Matam (Bengaluru), Devender Bairwa (Faridabad), Sushant Shinde (Mumbai), Niladri Bhowmick (Hyderabad)

SCIENTIFIC PROGRAMME

Day 2 – Sunday, 15th March 2026

8.00 am - 9.15 am — **Meet the Masters**

Hall A: Mimics, Malignancy and Imaging in Myositis

- Inflammatory vs non-inflammatory myopathies
- Risk stratification and screening for malignancy
- MRI in muscle disease

Faculty: Hector Chinoy, Khushboo Pilania

Chairs : Shefali K Sharma (Chandigarh), Raheesh Ravindran (Mumbai), Hema M (Chennai)
Coordinator : Meha Sharma (New Delhi), Phanikumar Devarasetti (Hyderababd)

Hall B: Clinical conundrums in Psoriatic arthritis and Psoriasis

- Suspecting and diagnosing axial psoriatic arthritis
- Smart use of imaging in psoriatic arthritis
- What is and what is not psoriasis?

Faculty: William Tillett, Rajiv Sekhri

Chairs : John Mathew (Vellore), PD Rath (New Delhi), Hemant Gopal (Bathinda)
Coordinator : Priyanka Kharbanda (New Delhi), Vikramraj Jain (Bengaluru)

9.30 am - 10.30 am — **Abstract Presentation by Rheumatology Trainees**
Chairs : K Shanmuganandam (Chennai), Uma Karjigi (Bengaluru), Balaji C (Chennai)
Judges : Raja N (Vellore), Chethana Dharmapalaiah (Bengaluru), Naval Mendiratta (Gurugram)

10.30 am - 11.00 am — **Master Class - 1**
Chairs : BG Dharmanand (Bengaluru), Ved Chaturvedi (New Delhi), Amita Agarwal (Hyderabad)

10:30-10:50 am — **Myositis-associated Interstitial lung disease** — **Hector Chinoy**

- Clinical phenotypes and classification challenges
- Autoantibody-guided prognostication
- Therapeutic strategies

10:50-11:00 am — Q & A

11:00-11:30 am — **Tea**

11.30 am - 12.00 pm — **Master Class - 2**
Chairs : Vikas Agarwal (Lucknow), Anupam Wakhlu (Lucknow), Sameer Gulati (New Delhi)

11:30-11:50am — **Comorbidities in Psoriatic Arthritis** — **William Tillett**

- Spectrum of comorbidities
- Screening strategies
- Therapeutic balancing

11:50-12:00pm — Q & A

SCIENTIFIC PROGRAMME

Day 2 – Sunday, 15th March 2026

12.00 pm - 12.30 pm	**Cross Talk 3** **Chairs :** Sundeep Upadhyaya (Noida), Kaushik Bhojani (Mumbai), Arun Hegde (Belgavi)	
12.00-12.20 pm	**HRCT in Myositis-associated Interstitial Lung Disease** · Decoding the reporting terminologies · Imaging patterns · Disease monitoring	**Khushboo Pilania (India)**
12.20-12.30 pm	Q & A	
12.30 pm - 1.00 pm	**Challenging Cases 5** **Chairs :** Bhakuni DS (Newdelhi), Ashit Syngle (Chandigarh), Shailee Chandak (Nagpur) **Judges :** Ajit Kumar Surin (Bhubaneshwar), Nagaraj S (Bengaluru), Shaloo Bhasin (New Delhi)	
12.30-12.50 pm	**Double trouble** **Deceptive Beginnings**	**Harikrishnan G (Kottayam)** **Rahul Bisaralli (Dharwad)**
12.50-1.00 pm	Discussion and Q &A Discussants	**Hector Chinoy** **Khushboo Pilania**
01:00-01:15 pm	**Valedictory and Awards**	
01:15pm	**Lunch**	

Poster Judges

Abhishek Patil, Abhra Chowdhury, Akhil Goel, Ankit Patwari, Anupama Swamy, ARK Naidu, Arun Tiwari, Ashwin M Nair, Daisy Doley, Deonis Xess, Divya Lala, Gautam Panjabi, Hemant Sharma, Jagbir Singh, Jithin Mathew, Joydeep Samanta, Juhi Dixit, Kishore Babu, Kunal Chandwar, Nagaprabhu VN, Paresh Jena, Piyush Jain, Princi Jain, Pulin Gupta, Radhakrishna B, Ravi Kumar, Raviraj Acharya, Rebecca Marak, Ritasman Baisya, Sabarinath M, Sachin Dhote, Sandeep Kharkar, Sanjay Dubey, Shailaja Sabnis, Saumya Ranjan Tripathi, Sundaram TG, Thilagavathi N, Varsha Bhatt, Venkatesh S Pai, Vidya S.

E-Newsletter Team

Abhilasha Manwatkar, Anisha Kwatra, Anoop C, Anushka Aggarwal, Ashish Baweja, Bindhyachal Gupta, Illiasul Ibad, Merwin Samuel, Nikhila GS, Rishabh Nanavati, Sasikala B, Sourabh Malviya, Srujana Arekal, Sumana B, Vishwaprakash Tiwari.

AWARDS

Prof. A N Chandrasekaran prize for
"Best case presentation by young consultant"

Prof. V R Joshi prize for
"Best abstract - Oral presentation by Rheumatology trainee"

Prof. Sukumar Mukherjee prize for
"Best abstract - Oral presentation by Medicine trainee"

Prof. A N Malaviya prize for
"Best abstract - Poster presentation" (Rheumatology and Medicine trainee each)

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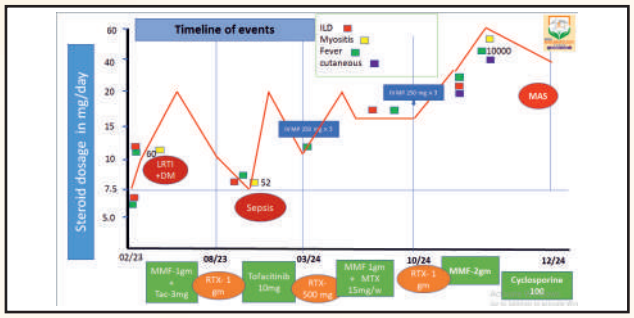
"An Unyielding Enemy"
Dr. N.Kavya Devi

Case Details

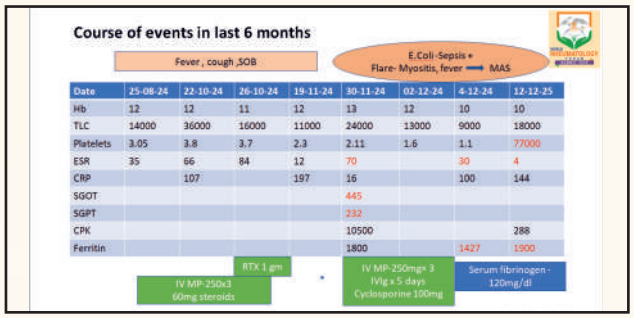
"An Unyielding Enemy"

Dr. N.Kavya Devi

MBBS, MD, DM (Rheumatology-NIMS)
Anvitha Arthritis Clinic
Vijayawada, Andhra Pradesh



55 Y/F - **No comorbidities** **Pastor by occupation** **Symptomatic since Feb, 2020**	**CBP- 11/17000/3.5** **CRP-60** **LFT / RFT / Virals - Negative**
• Intermittent fever spikes 100-102 F • Cutaneous rash- upper back, neck, hands, knees– s/o mechanics hands • Exertional shortness of breath with cough • Denies any proximal muscle weakness	HRCT chest – consolidation with GGO's in both lower lobes, right middle lobe and left lingular lobe Multiple reticulations and septal thickening in both lower lobes s/o ILD-organising pneumonia Biopsy – Mild expansion of interstitium by lymphocytes. Alveolar lumen showing foamy histiocytes s/o organising pneumonia. BAL- cultures sterile



Immuno profile	Key Clinical features
CPK - 1280 ANA - ELISA-- 80 (60) ANA profile – Ro 52/Ro 60 2+ ve Myositis Profile – PL-7 Ro 52 positive	**Baseline disease** Mechanics hands Myositis ILD- Organising Pneumonia type Fever **Outcome** – Refractory, Steroid dependence Macrophage activation syndrome
	Death

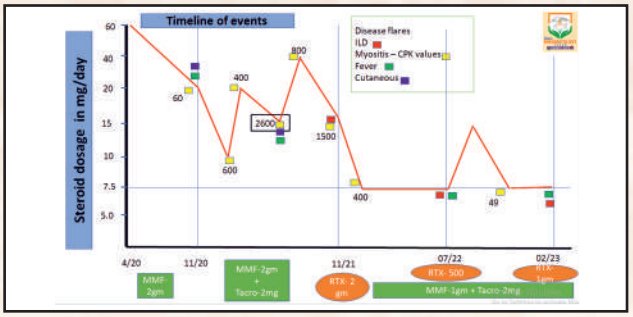
Why was this case so difficult?

- All 4 key clinical features – Cutaneous, ILD, Myositis, Fever being refractory.
- Steroid dependence for long time with associated complications despite prophylactic measures.
- Leucocytosis being occurring with every disease flare sometimes leading to difficulty in differentiating between infection and disease.
- Late onset of catastrophic MAS

WHY REFRACTORY?	Complications despite precautions
Could not maintain on a stable low-dose steroids despite many DMARDS	Received Pneumococcal / Influenza / Covid-19 vaccination schedules Cotrimoxazole prophylaxis Calcium + Vit-D supplements CT chest and abdomen done to r/o malignancy
MMF 2gm/day MMF 2 gms + Tacro 2 mg RTX 2 gm 6 monthly cycles MMF 1 gm + Tacro 2 mg Tofacitinib – 10mg MMF 1 gm + MTX 15mg/week Cyclosporine	1 Herpes zoster 1 Pneumonia 1 Sepsis with pneumonia plus Steroid induced Diabetes, Hypertension Cataract Shoulder fracture

Questions

- Any validated predictors that should alert us to a refractory or steroid-dependent course in anti-synthetase syndrome?
- How do we anticipate or recognize MAS in such cases? – clinical and/or by laboratory parameters?



Thank you

Serendipity and a therapeutic answer
Dr. Pallavi Pimpale

Serendipity and a therapeutic answer

Dr Pallavi Pimpale

Pediatric Rheumatologist, Mumbai
Special Interest in Autoinflammatory Diseases and Genetics

Fellowship in Pediatric Rheumatology (Mumbai)
Fellowship in Autoinflammatory Diseases (March 2020- January 2025) (NHGRI, NIH, USA)

Affiliations:
Pediatric Rheumatology and Autoinflammatory Clinic
Lilavati, Surya, and NH SRCC Children's Hospital
HBT Medical College/Dr. R N Cooper Municipal Hospital

KK, 17y, male, NCM

Initial presentation – age 6y
Facial swelling & rash – 10d
Pain & difficulty walking, sitting up – 10d

General examination:
Wt. 20kg (50th centile), Ht. 118cm (75th centile)
Vitals stable
Facial puffiness
Diffuse erythematous facial rash, Heliotrope rash +, Gottrons papules +
Livedoid rash upper & lower limbs
Thigh & calf tenderness

Systemic examination:
Respiratory, cardiovascular, abdominal examination – Normal
Central nervous system: oriented alert, head lag +, nasal twang, dysphagia
power bilateral UL & LL – 4/5, trunk 3/5

Nail Fold Capillaroscopy

Laboratory	
SGOT	156 U/L
SGPT	74 U/L
CPK	4830 U/L
LDH	320 U/L
ANA	2+
ANA blot	Ro 52, nucleosome +, Jo1 negative
Imaging	
MRI	inflammatory myopathy with mild muscular atrophy
EMG	myopathic pattern
Muscle biopsy	myositis

Criteria fulfilled
Diagnosis: Juvenile dermatomyositis

- GCOM 2019 Berlin
- Cost 60 tabs (Rs 64,000/ 711 USD/605 Euro)
- Informed consent and multiple discussions

- Tofacitinib started with Methotrexate, Colchicine, and oral steroids
- Myositis-specific antibodies – not tested

- Gradual improvement of muscle strength and healing of ulcers

Use of Tofacitinib in Refractory JDM
Paik J, Albayda J, Tiniakou E, Koenig A, Christopher-Stine L. Study of Tofacitinib in Refractory Dermatomyositis (STIR): An Open Label Pilot Study in Refractory Dermatomyositis - ACR Meeting Abstracts. Arthritis Rheum 2018;70:L02.

Age 6y – 17y (treated elsewhere)

Disease course:
- Recurrent **flares** on tapering steroids
- Episode of **mental slowing, irritability, nystagmus, delayed speech output** (MRI brain: mild cerebellar atrophy)
- **Myocarditis with AV block**, multiple premature complex
- **Ulcerations**
- **Calcinosis**
- **Bone infarcts** in the distal end of the femur

Treatment course:
- Multiple pulse therapy and oral steroids
- Methotrexate, Azathioprine, Cyclosporine, Hydroxychloroquine, Tacrolimus, Mycophenolate mofetil
- 3 cycles of Rituximab
- 6 cycles of Cyclophosphamide (500mg/m²)
- 14 cycles of IVIg (2g/kg)

Last visit (Feb 2022)
- CMAS = 52
- No skin ulcers
- New calcinosis with oozing

First visit to our clinic November 2019 (age 17y)

- Ulcerative lesions on the face, B/L arms, and back
- Redness of the nose and ear lobes
- Lipoatrophy arms
- Calcinosis right side of chest, right side of lower abdomen, and the pubic region
- CMAS on arrival- 11

Questions

- How should "treatment success" be defined in refractory JDM — biochemical, radiological, or functional?
- Is calcinosis reversible, or is stabilization a realistic goal?

Acknowledgments:

Case and Mentor:
Dr Raju Khubchandani

Mentor:
Dr Lisa Rider

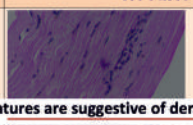
The Wolf in Myositis Clothing: A Diagnostic Tug of War
Dr. Sanket Shah

The Wolf in Myositis Clothing:
A Diagnostic Tug of War

Dr Sanket Shah (MD, DM)

Consultant Rheumatologist,
RheumaCARE
Ahmedabad, Gujarat

Next step was to Go ahead with Biopsy

Investigation	Results
Muscle Biopsy	

- Histomorphological features are suggestive of dermatomyositis,

REVIEWING
- Preserved muscle architecture
- No myofiber necrosis
- No inflammatory infiltrates
- No perifascicular atrophy

Image Courtesy:
Dr Chetna Bullar, MD Pathology
Histopathology Dept, Sterling Hospital, Ahmedabad

We as treating rheumatologist were not convinced with the reports

Clinical Presentation: History

63-year-old female with
- **Disease duration:** 3 months
- **Chief Complaints**
  - ☐ **Skin lesions:**
    - Initially lower limbs → upper limbs and trunk
    - Papulonodular, mild itching and burning sensation
  - ☐ **Muscle weakness with myalgia**
    - Progressive proximal
      - Lower limbs > upper limbs
      - Limiting daily activities
  - ☐ **Fever**
    - Intermittent moderate-grade
  - ☐ **Constitutional symptoms**
    - Fatigue, reduced appetite

Comorbidities
- ☐ **Autoimmune hepatitis** (5–6 years)
  - On low-dose prednisolone and azathioprine
- ☐ **Prediabetes,**
- ☐ **Hypertension**

Next step was to Go ahead with Biopsy

Investigation	Results
Skin Biopsy	underlying dermis and subcutaneous tissues. Subcutis reveals presence of mixed inflammatory infiltrate composed iof lymphocytes, neutrophils and collections of histiocytes. PAS-stained sections do not reveal any organisms.

- Histomorphological features are consistent with panniculitis

- Lobular panniculitis
- Dense atypical lymphoid infiltrate
- Rimming of adipocytes by lymphocytes
- Nuclear atypia and apoptotic debris

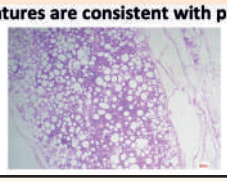


Image Courtesy:
Dr Chetna Bullar, MD Pathology
Histopathology Dept, Sterling Hospital, Ahmedabad

Clinical Presentation: Examination

Conscious, Cooperative, Oriented
Vital Signs
- Febrile on Palpation
- Blood pressure - 134/80 mm Hg
- Pulse rate - 108 bpm, regular in rhythm
- RR - 24/minute

General Examination
- **Pallor Present**
- **No** [illegible]

Head to Toe general examination:
Skin: Multiple ill-defined, deep-seated papulonodular lesions

Lesion on right thigh

- Neurological Examination
- Cardio-Respiratory
- Abdomen & Genito-Urinary
- Musculoskeletal Examination

Neurological Examination
Higher Mental Functions:
- Normal

Motor System:
- Proximal muscle weakness
- More marked in lower limbs
- MMT at 52/60

Reflexes:
- Deep tendon reflexes: +
- Planters: Bilateral Flexor

Sensory & Cerebellar: Normal

Cardio-Respiratory
- **CVS:** JVP not raised, S1, S2 heard with normal intensity, No added sounds
- **URT & LRT:** Normal.

Abdomen & Genito-Urinary
- No Hepatosplenomegaly,
- No free fluid

MSK
- Tender & swollen joints - 0/0

Clinical & Investigation Summary

63-year-old female with Disease duration: 3 months
- **Manifestations**
  - ☐ **Papulonodular Like lesions**
    - ☐ Biopsy suggestive of Panniculitis
  - ☐ **Proximal Weakness**
    - ☐ Raised muscle enzyme
    - ☐ Biopsy reported as Dermatomyositis
    - ☐ Anti Pm/Scl antibody: Borderline Positive
  - ☐ **Fever**
    - ☐ Negative Infection workup

Treatment received during hospital

Antibiotics -> No response

Steroids -> Very good response

So, from the intensivist team, Diagnosis of Inflammatory Myositis was considered
Steroid + Mycophenolate was started

Investigation at First Consultation

CBC & APR	REPORTS
Hemoglobin (g/dL)	9.8
MCV	84.6
WBC Count (cells/µL)	13020
N/L/E/M/B (%)	67/22/6/3/2
Platelets (cells/µL)	1,28,000
ESR at 1 hour	52
CRP (mg/L)	22.1

RFT & Electrolytes	REPORTS
Urea	23
Creatinine (mg/dL)	1.2
Sodium	132
Potassium	4
Calcium	8.8
Phosphorus	3.4
Magnesium	1.8
Uric Acid	5.7
Urine Microscopy	Normal

LFT	REPORTS
T. BIL	0.45
SGPT (U/L)	62
SGOT (U/L)	109
ALP	127
T. Protein	7.1
Albumin/Globulin	3/4.1
PT/INR	16.3/1.16
CPK Total	504
LDH (U/L)	709
S.Ferritin (ng/mL)	6300.4

Microbiology	
Urine c/s	No Growth
Blood c/s	No Growth
Viral marker	Negative

Investigation	Results
ANA	3+ Nuclear speckled pattern
ENA	Anti-Pm/Scl positive 1+

Reason For Reference

Correct Diagnosis was done with Immunohistochemistry

Immunohistochemistry :	
CD 3 (Clone - POLYCLONE)	- Positive (Atypical cells)
CD 4 (Clone - c8/144b)	- Positive (Atypical cells)
Ki-67 (Clone - MIB 1)	- 50-60%
CD 20 (Clone - L26)	- Negative
CD 30 (Clone - BER-H2)	- Negative
CD 4	- Negative
CD 56 (Clone - 123C3)	- Negative
CD 10 (Clone - 56C6)	- Negative
TDT (Clone - EP266)	- Negative
CD 68 (Clone - KP1)	- Negative

IHC findings are suggestive of Subcutaneous panniculitis - like T-cell lymphoma (SPTCL)

PET CT	No Significant Metabolic Uptake suggestive of malignancy in visceral organ
Bone Marrow Aspiration & Biopsy	Normal , No evidence of Malignant cells

Further Investigation

Investigation	Results
ECG	Sinus tachycardia
Echo	LVEF 60%, Concentric LVH, RVSP 41
USG abdomen	Mild Hepatomegaly No splenomegaly No free fluid
CT Thorax & abdomen	Subcutaneous Fat stranding suggestive of panniculitis

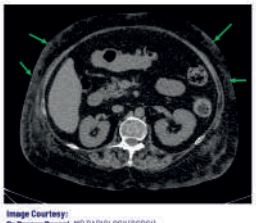


Image Courtesy:
Dr Pranav Devani, MD RADIOLOGY (SGPGI)
Consultant Radiologist, Sterling Hospital, Ahmedabad

Final Correct Diagnosis

Subcutaneous panniculitis-like T-cell lymphoma Presenting as a myositis mimic

Revised management
- Shifted to Hematology care-> Cyclosporine and steroids initiated

Outcome
- Afebrile, Improving skin lesions
- Ambulating independently with mild residual weakness

Learnings	• High CK, LDH, and weakness do not always indicate myositis • Panniculitis with fever warrants evaluation beyond CTD • Steroid responsiveness is not diagnostic • Muscle biopsy is essential when phenotype and labs mismatch • Re-biopsy and IHC are critical when clinical course is atypical

Rare and Scary
Dr. Sandeep Yadav

Rare and Scary

Dr Sandeep Yadav

Consultant
Rheumatology Department
PD Hinduja National Hospital and Medical Research Centre
Mumbai, Maharashtra

The Diagnostic Pivot (Neurophysiology)

Conduction Study (NCS)Motor: Prolonged distal latencies, slow conduction velocities, and absent F-responses in Median, Ulnar, Tibial, and Peroneal nerves Generalized motor demyelinating symmetric peripheral neuropathy suggestive of Guillain-Barré Syndrome

Dermatomyositis	GBS
Skin involvement • Gottrons papules • Periorbital rash Myositis • Respiratory muscle weakness • Proximal muscle weakness • Dysphagia	• Bilateral facial nerve palsy • Generalised areflexia • Absent gag reflex • Dysphagia • Respiratory muscle weakness • NCS: Demyelinating Neuropathy

Diagnosis : Dermatomyositis with GBS

History & Initial Clinical Presentation

19 years Male

1 week Aug

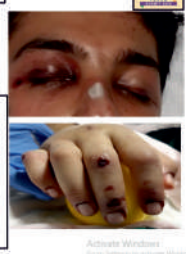
- Fever (high grade) / decreased appetite
- Rash- Knuckles ,chest and back non itchy erythematous
- Erythema over bilateral upper eye lids
- Difficulty in getting up from sitting position
- Bilateral thigh pain

Admission

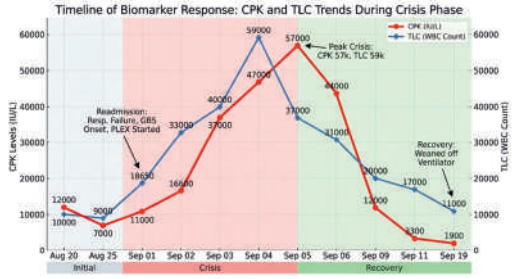
Day 1

Physical Examination
- Vitals: Stable.
- Cutaneous: Periorbital heliotrope rash (R>L)
- Erythematous papules over MCP/PIP joints (Gottron's papules)
- Rash over upper chest/back.

Neurological:
- Power: Upper Limbs 4/5, Lower Limbs 3/5 (Proximal weakness).
- Reflexes: All present; no distal weakness.
- No cranial nerve or respiratory muscle involvement.



Timeline of Biomarker Response: CPK and TLC Trends During Crisis Phase



Initial Evaluation & Workup

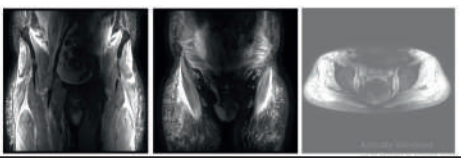
Admission 1

3rd week Aug

- Laboratory Findings
  - **Enzymes:** CPK: 12,000 IU/L , LDH: 12,000 , SGOT: 1193, SGPT: 262
  - **Renal/Urine:** Urine Protein 3+; UPCR 1.50
  - **Autoimmune Profile:** ANA by IF negative; PR3/MPO negative;

Haemoglobin	16.30
MCV	77
WBC	10,000
DLC	N81/L8/M11
PLATELETS	108
CREATININE	0.8
C3	57
Serum protein	1-4.60 A – 2.0 G- 2.6

Radiology (MRI Bilateral Thigh)
Diffuse STIR hyperintensity in anterior and medial compartments and pelvic muscles. Suggesting inflammatory myositis



Outcome

Progress by Day 19-
- Respiratory: Patient weaned off ventilator; maintained on T-piece with tracheostomy.
- Neurological: Facial muscle weakness improved.
- Biochemical: CPK normalized significantly to 1,900 IU/L.
- Renal function normalized (Creatinine 0.5).
- General: Limb edema and muscle tenderness subsided

Follow up 3 months

- Prednisolone 5 mg/day
- MTX 20 mg S/C per week
- Tracheostomy Closure
- Labs normal Fully functional

Follow up 3 years

Off treatment no reoccurrence

Initial Treatment & Response

Admission 21 to 25 Aug

Diagnosis : Inflammatory Myositis

Treated with Pulse Solumedrol (500mg x 3 days) followed by oral Prednisolone (60mg/day) and Methotrexate (15mg/week).

Response Discharged - Day 5

- Fever subsided, rash improved
- CPK dropped to 7,000,
- power improved (UL 4+/5, LL 4-/5).

- Denied renal/ Muscle biopsy
- Myositis Antibody panel negative

Discussion & Literature Review

Neuro myositis :
- A term introduced by Senator in 1893 to describe concomitant PNS and muscle involvement.
- Rarity: While Myasthenia Gravis overlaps with myositis (often thymoma-associated), the overlap of GBS and Dermatomyositis is exceptionally rare.

Pathogenesis:
- Unclear mechanisms.
- Proposed theories include: Vasculitic neuropathy (VEGF overproduction/ischemia).
- Molecular mimicry targeting both myelin and muscle antigens.

Literature:
Only ~12 cases of neuropathy with myositis reported; predominantly axonal neuropathy, unlike the demyelinating pattern in this case.

Literature Overlap		NUMBER OF CASES
AIDP AND CIDP	Myasthenia Gravis	6
Myasthenia Gravis	Polymyositis	16
Myasthenia Gravis	Dermatomyositis	10
DM	GBS	NONE
Myositis	Neuropathy	12

Acute Decompensation

Readmission (1 Week Post-Discharge)- 1st Sep

Emergency
- Generalized weakness and body ache
- Dysphagia
- Respiratory distress.

- Examination
  - Higher functions normal/Ocular muscles Normal
  - Bilateral ptosis with bilateral facial nerve palsy , absent gag reflex with generalized absence of reflexes.
  - Examination Bilateral proximal muscle weakness power Lower limb 2/5 bilaterally ,Bilateral upper limb 3/5
  - No distal muscle weakness no bowel bladder involvement , higher functions normal, ocular muscles normal.
  - Respiratory failure WITH TACHYPNEA requiring intubation and mechanical ventilation

Blood Cultures negative /PCT Normal

	1/SEP	2/SEP	3/SEP	4/ SEP	5/ SEP	6 / SEP	7/SEP
HB	16.3	17.0	16.0	14.1	9.8	7.7	9.8
Retic count 4.82%/RF 34%/Comb test negative/Stool for occult blood neg ANA negative DS DNA neg /C3 79 /Lu Plus anticoagulant and APLA negative							
TLC	10600	11600	10,300	9400	6700	11000	10000
DLC	89/7	93/3		N95/L 1			
PLAT	52000	24000	20000	48000	68000	58000	68000
CREAT		1.1	1.1			1.3	1.4
CRP	31.7		40				
CPK	12000	10,817		7700		47000	
LDH	1234	1305					
URINE	ALB TRACE	UPCR 6.3					

Questions

- Do we view "Neuromyositis" as a distinct entity or a coincidental overlap of two autoimmune catastrophes?
- To Biopsy or Not: We relied on MRI and NCS for diagnosis. In such acute, unstable overlap cases, would a muscle or nerve biopsy have changed our management, or is empirical "rescue" therapy (PLEX/IVIG) the only practical path?

The Face Tells a different Story
Dr. Shivraj Padiyar

The Face Tells a Different Story

Dr Shivraj Padiyar, MD, DM Rheumatology (CMC Vellore)
Professor, Dept. of Clinical Immunology and Rheumatology
Kasturba Medical College, Mangalore

EVALUATION IN 3RD HOSPITAL

MRI Face (July 2021)- Multifocal edema in the muscles of head and neck suggestive of inflammatory myositis
Bulky right masseter- non enhancing necrosis
MRI neck (August 2021): edema involving the multiple muscles of the face and neck (Right > Left)
Bilateral parotid and submandibular gland enlargement
Significant resolution of right masseter necrosis, cervical lymphadenopathy

Aug 2021

USG guided biopsy of masseter: Chronic inflammatory process with necrosis

Aspiration of pus from masseter muscle: Klebsiella Pneumonia

USG guided biopsy of Right submandibular node: Reactive suppurative lymphadenitis

Repeat muscle biopsy (Buccinator and masseter)- Features of inflammatory myositis

Treated initially with combination of antibiotics and subsequently with steroids.

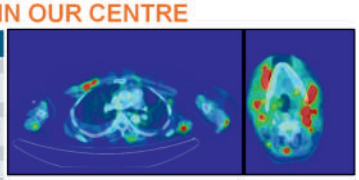
- **47 year old female, housewife, from Bihar, presented to CMC Vellore**
- Symptomatic since 1 year (September 2020) before presentation to us
- Buccal mucosal ulcers , painful and recurrent
- Swelling over the right side of face , gradually progressive, initially soft and painless progressing to firm and painful in 6 months
- Since last 6 months – swelling of left side of the face of similar nature
- Since 3 months , c/o fever with Chills, evening rise, without other localisation
- Since last 1 month, swelling has progressed to bilateral over part of the face with difficulty in opening the eyes
- Reddish rash over the neck region for 10 days

NEGATIVE HISTORY

No history of weight loss, alopecia, inflammatory joint pains , Raynaud's, skin tightness, muscle weakness, redness of eyes, GERD, abdominal pain, frothy urine

EVALUATION IN OUR CENTRE

Investigations	Results
Hemoglobin	11.5 g/dL
TLC	3400 (N-76, L-15, Myelo-3)
Platelets	65000 cells/mm³
LFT	0.43/0.23/5.7/3.5/127/83/44
LDH	860 U/L
Creatinine	0.6 mg/dl
Calcium	8.2
CPK	240 U/L
CRP	1.12 mg/L
Ferritin	3600
Myositis profile	Negative
INR/APTT	0.95/32



- Metabolically active uptake of muscles of mastication and salivary glands
- Nodular areas of thickening in intercostal muscles / biceps /paraspinal muscles (max SUV 36)
- Few enlarged level 1nodes, max SUV 6
- Bilateral pleural effusion – Right > left, metabolically inactive

- **On Examination**
- Stable Vitals, No pallor/ Icterus/ Cyanosis/Clubbing/LN/edema
- Hard swelling over both sides of the face, mildly tender, with subcutaneous edema
- Ecchymotic rashes over the face and neck region
- Right-sided buccal mucosal ulcer 2x3 cm, multiple small ulcerations on both sides

SUMMARISING: A middle aged lady with progressive hard swelling around the bilateral face and neck region over 1 year, with non healing buccal mucosa ulcers with multiple biopsies showing inflammatory process and features of myositis.

- Open biopsy was done
- From tail of parotid , Level II lymph nodes, and right buccal mucosa

High grade Non Hodgkins Lymphoma (Histological features more suggestive of Extranodal NK T cell lymphoma)- negative for Gamma Delta Clonality

CD3: Positive, CD30: Positive, CD8: Positive, CD56: Positive
TIA-1: Positive, Granzyme B: Positive, EBV LMP1: Negative
CD4: Negative, CD20: Negative, ALK: Negative
EMA: Negative, MIB-1 (Ki-67): ~80%
Bone marrow biopsy : No abnormal cells

FINAL DIAGNOSIS: HIGH GRADE NHL, NK T CELL LYMPHOMA, STAGE III, With no bone marrow involvement

- She was started on CVP regimen – Cyclophosphamide, Vincristine, Prednisolone
- She underwent tracheostomy in order to restore the airway in view of stridor
- She developed febrile neutropenia in ICU– covered with antibiotics
- Developed sudden respiratory distress- ? Septic ARDS
- Progressive DIC and later succumbed to her illness within a span of 1 week

OUTSIDE EVALUATION

DEC 2020

- Painful oral ulcers over the buccal mucosa
- Swelling over the right side of face over the parotid and retromandibular region
- Progressive pain and induration over the swelling

- CECT face: Right submandibular gland enlargement with heterogenous contrast enhancement, periglandular fat stranding
- Multiple cervical LN +
- Treated with antibiotics- No relief

MAY 2021

- FNAC: Acute on chronic sialadenitis, treated conservatively with analgesics and Chlorhexidine mouth washes.
- Repeat Imaging (May 2021) CE-MRI: Infective /Inflammatory process involving the right parotid, submandibular space, masticator space.
- Biopsy from the right cheek- Inflammatory masticatory muscle myositis

- ANA (IF)- Speckled 1+
- ANA profile- Jo-1 1+
- IgG4- 0.93
- ACE-22
- CPK-69
- Treated with low dose steroids- partial response

QUESTIONS TO EXPERTS

- How common is focal myositis? Does a myositis mimic often get mislabelled as focal myositis?
- What are the clues, clinically and in muscle biopsies, that make one suspect a myositis mimic?

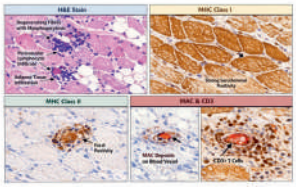
Trigger, MIMIC or Coincidence?
Dr. Shubha Bhalla

TRIGGER, MIMIC OR COINCIDENCE?

Dr. Shubha Bhalla

Consultant Rheumatologist
DEPARTMENT OF RHEUMATOLOGY
FORTIS HOSPITAL, MANESAR, GURGAON
HARYANA

Histopathology of muscle biopsy



- Maintained fascicular architecture
- Mild variation in muscle fibre size
- Occasional focus of perivascular lymphocytic collection in perimysium
- Few degenerated fibres with myophagocytosis
- On IHC- upregulation of MCH class I and II and occasional CD3 +cell seen around endomysium
- MGT stain doesn't reveal ragged fibres
- F/S/O Dermatomyositis

Muscle biopsy done from right vastus lateralis

HISTORY

A 61 years old female k/c/o Disseminated TB (brain tuberculoma and left sided pleural effusion on ATT from last 8 months, presented with

- Progressive photosensitive skin rash involving the face, neck and upper back for the past 1 month
- Progressive weakness B/L upper and lower limbs (proximal>distal) from last 10 days
- Difficulty in swallowing solids and liquids
- Progressively increasing generalised swelling
- No h/o fever prior to onset of weakness, no h/o Raynaud's phenomenon, SOB, palpitation

DIFFERENTIAL DIAGNOSIS	MANAGEMENT CHALLENGES
➢Para-infectious dermatomyositis	➢High disease activity
➢Anti-SRP associated dermatomyositis - ? Seronegative	➢Ongoing disseminated TB
➢Overlap myositis	➢Limited immunosuppressive options
➢Drug-induced dermatomyositis	
➢Tuberculosis-associated inflammatory myopathy	

On examination

➢At presentation:

➢*General examination*

Conscious , oriented to time place and person

Severe pallor

Anasarca+

Normotensive -140/80, P.R-95/min, SpO2- 99% on RA

Skin
- Heliotrope rash,
- malar rash involving the nasolabial folds+
- Shawl SIGN +
- V- NECK SIGN +

➢*Systemic examination*

CHEST – B/L SCATTERED CREPITATION+

CVS- S1S2+, NO MURMUR

P/A –Soft, non-tender, no palpable HSM, FF

Neuromuscular examination

Muscle weakness (Proximal>distal)

MMT-8 SCORE on day 1- 73/150

All DTR- intact, No sensory or cranial nerve involvement

On 3rd day

Anasarca worsened , **MMT-8 score 36/150**

Management

- She was given i.v pulse steroid 1 g for 3 days with electrolyte and blood sugar monitoring- inadequate response
- 2 units PRBC with adequate fluid management and diuresis
- Continued Anti-tubercular treatment –Risk- benefits explained
- AED-levitracetam and other supportive treatment
- IVIG 2g/kg over 5 days
- RTX was deferred due to disseminated TB
- Injectable methotrexate 25 mg s/c weekly was started with oral prednisolone 1 mg/kg body weight

WORKUP

	07/09/2021	11/9/2021	23/9/2021
CBC	HB-7.3 TLC-12380 DLC(85/06) P/C-4.36	HB-9.4 TLC-13080 P/C-3.53	HB-12.9 TLC-12700 DLC(86/09) P/C-2.92
SGOT/SGPT	340/53	296/62	170/54
LDH/CPK TOTAL	1096/886	984/794	819/438
ALB/GLOBULIN	2.0/3.0		2.4/2.3
Na+/potassium	133/3.6		
Urea/Creatinine	14/0.3		16/0.4
CRP/ESR	21.3/84		12.8/60
URINE R/M	ALB+, KETONES+, GRANULAR CAST+	Urine ketones –ve	ALB-ve, PUS CELLS-2-4, RBCs- NIL, NO CASTS
URINE P:C ratio		C3/C4-103/28	0.78

TSH-1.43
VIRAL MARKERS WERE NEGATIVE

ANA – negative by EIA
MAA - ANTI RIB-P- Strong positive
U1 nRNP/Sm- positive

MSA- anti-SRP+

EMG-Compatible with myopathic pattern

2D echo- No RWMA LVEF- 50%

ON FOLLOW UP

After 1 month she could walk with support

Anasarca disappeared but skin rashes persisted

MMT-8 score – 92/150

18/11/2021	22/06/2022
HB-10.2 TLC-7100 P/C-1.89	Hb-11.1 TLC-8040 P/C- 1.6
SGOT-42.3 SGPT-47.5	42 45
LDH-703 CPK(T)159	579.5 124
ALB-3.2 GLOB-2.1	3.8 2.4
CRP-2.6	3.45
ESR-40	30
Cr	0.77

IMAGING

Inflammatory myopathy – rapidly progressive
Dermatomyositis skin rash
Dysphagia
Anasarca
In background of active disseminated TB infection

HRCT CHEST

Confluent irregular fibrocalcific lesion

Tractional bronchiectasis in apico posterior segment of B/L upper lobes , rt middle lobe and lingular segment of left upper lobe

Small crescentric hypodensity along the posterior aspect of the left upper hemithorax s/o organised pleural effusion (in resolving stage)

CT-WHOLE ABDOMEN-

Calcific granulomatous lesions in the liver with small fluid in the pelvic retroperitoneal space

QUESTIONS

➢ Can tuberculosis act as a trigger for inflammatory myositis or just an association?

➢ Does this patient classify as Overlap myositis, Seronegative Dermatomyositis, or Anti-SRP positive dermatomyositis?

Thank you for your patient listening.

A Real-World Challenge!
Dr. C.Saranya

A real-world challenge!

Dr. C.Saranya,M.D.,D.M.,
Professor of Rheumatology
Saveetha Medical College & Hospital, Chennai.
Consultant Rheumatologist
O.M. Child health and Joint pain Superspeciality centre, Chennai

Management

- T. Etoricoxib 90mg sos
- T. Pantoprazole 40mg od
- T. Methotrexate 15mg/week
- T. Folic acid 5mg/wk
- T. Cilnidipine 10mg od
- T. Atorvastatin 20mg hs
- T. Thyroxine 100mcg od
- Inj. Adalimumab 40mg sc every 2 weeks

After 4 doses of Inj. Adalimumab

SKIN FLARE

Due to flare of skin lesions, switched to
✓Inj. Secukinumab 150mg sc – loading doses weekly (5 doses) followed by maintenance dose every 4 weeks for 1 year followed by
✓Inj. Secukinumab 150mg sc every 8 weeks

CURRENT MEDICATIONS
T. Methotrexate 15mg/week
T. Folic acid 5mg/wk
T. Cilnidipine 10mg od
T. Atorvastatin 20mg hs
T. Thyroxine 100mcg od
Inj. Secukinumab 150mg sc every 8 weeks

Case History

PRESENTING COMPLAINTS

A 56 year male presented with

➢Multiple joint pain with swelling, involving small joints of both hands and feet – 2 weeks

➢Increase in psoriatic lesions over both hands and legs – 2 weeks

PAST HISTORY

➢Known Psoriasis vulgaris for 8 years on topical medications and T. Methotrexate 10mg/wk for 1 year.

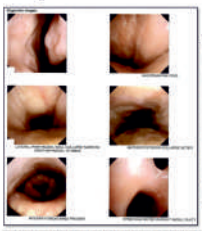
➢H/o intolerance to Tab Apremilast.

➢Known hypothyroid on Thyroxine 100mcg/day for the past 20 years

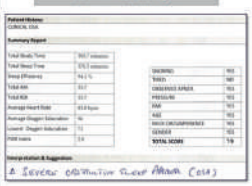
PERSONAL HISTORY
Not a smoker/ alcoholic

FAMILY HISTORY : nil significant

Course of the illness



Polysomnography report



BMI
Weight : 95.8 kg
Height : 163cm
BMI : 36.1 kg/m2
(Obese Class II)

Obstructive sleep apnoea (circumferential collapse) with right nasal cavity synechiae

Treatment: Auto CPAP - 6hrs/day , Weight reduction

Examination

GENERAL EXAMINATION:
•Weight : 88 kg
•Height : 163cm
•BMI : 33.1 kg/m2
(Obese Class II)
•Waist circumference: 108cm
•Hip circumference : 105cm
•Waist to hip ratio: 1.03

VITAL SIGNS:
BP - 130/100mmHg
PR-76/min

MUSCULOSKELETAL EXAMINATION

Enthesitis - 0
Dactylitis - 0

SKIN : Multiple erythematous plaques seen over dorsal aspect of both hands, palmar aspect of left hand, extensor aspect of both knees, legs, dorsum of both feet.
NAILS: Multiple pits, dystrophy +
SCALP: Psoriasis

Follow up

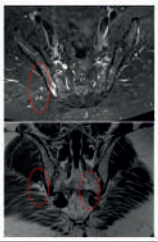
- Psoriatic arthritis – Polyarthritis, axial (b/l sacroiliitis)
- Psoriasis Vulgaris -With Nail changes, scalp psoriasis
- Co-morbids
  - Obesity-class III
  - Hypothyroidism
  - Hyperlipidemia
  - Hypertension
  - Prediabetes
  - MAFLD
  - Obstructive sleep apnea syndrome

ESR: 12mm/hr; CRP : 2.4mg/L (<10mg/L)
Uric acid : 6.4mg/dl
LFT: SGOT-98, SGPT- 104 IU/l
Lipid profile
T.Chol- 174, TGL- 92, LDL-94, HDL- 66, non HDL-108, VLDL-18.4, chol/HDL-2.6
Weight : 78 kg; Height : 163cm
BMI : 29.4 kg/m2 (obese class I)
Waist to hip ratio: 0.96

Treatment
✓AutoCPAP – 6hrs/day,
✓Weight reduction
✓T. Cilnidipine 10mg od
✓T. Atorvastatin 20mg hs
✓T. Thyroxine 100mcg od
✓Inj. Secukinumab 150mg sc every 8 weeks
Tab Methotrexate was stopped

Investigations

CBC: 12.7gm/7800cells/cu.mm/ 3.07 lakhs/cu.mm	ESR- 48mm/hr CRP- 3.3 (<0.8mg/dl)
LFT: AST 28, ALT 49 RFT : urea 28, S.Cr-0.9, UA-7.6 Urine routine -normal	HLA B27– negative Vit D - 44.34 ng/ml
HbA1C: 6.2, FBS-98mg/dl, PPBS-168mg/dl Lipid profile T. Chol- 212, TGL- 93, LDL- 145, HDL- 48 Thyroid profile : TSH-5.8 ECG, 2D ECHO - normal Viral markers : negative Mantoux – negative	IMAGING USG abdomen : Grade II fatty liver MRI SI joint with spine screening: Marrow edema is seen along iliac and sacral aspects of both SI joint (L>R), erosions seen along both SI joint (L>R), s/o Acute on chronic asymmetrical sacroiliitis (L>R)



Follow up

PAST 2 MONTHS
C/o increase in frequency of semisolid stools

MGE opinion
Colonoscopy : mild erythema in caecum

HPE : colonic mucosa with preserved crypt architecture and mild increase in lymphocytes and plasma cells in lamina propria, no granulomas or dysplasia or cryptitis, s/o non-specific colitis

What next?

NSAIDS	• Hepatotoxic, Hypertensive, CV risk
MTX	• Hepatotoxic, MAFLD
TOFACITINIB	• Hepatotoxic, Dyslipidemia, weight gain, CV risk
TNFi	• Paradoxical reactions
IL-17A	• Risk of IBD

Diagnosis

- ✓ Psoriatic arthritis - Polyarticular, Axial (b/l sacroiliitis)
- ✓ Psoriasis Vulgaris - With nail changes, scalp psoriasis
- ✓ Co-morbids
  - Obesity-class II
  - Hypothyroidism
  - Hyperlipidemia
  - Hypertension
  - Prediabetes
  - MAFLD

Questions

➢ What could be the cause of colitis? Should we consider stopping Inj. Secukinumab when non-specific colitis is present?

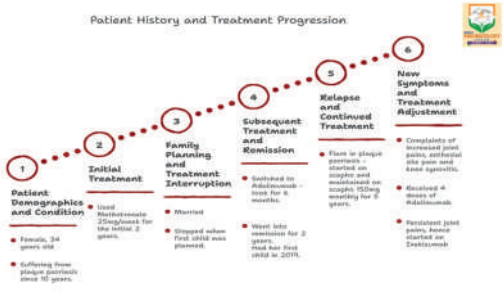
➢Role of newer weight-loss drugs in clinical practice in patients with PsA?

Ammunition is Running out, and a War to Wage
Dr. Raghavendra H

Ammunition is running out, and a war to wage.

Dr Raghavendra H

Consultant Rheumatologist
Manipal Hospital Yelahanka & Hebbal
Bengaluru, Karnataka



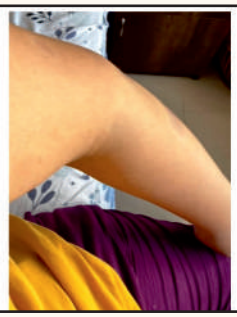
Scalp psoriasis

Plaque Psoriasis lesion over the thigh

Medications used till date

- MTX 25mg/wk
- ADALIMUMAB initially 12 doses and repeated 4 doses recently.
- Secukinumab 150mg monthly for 5 years.
- Apremilast+ MTX 15mg/wk.
- Ixekizumab + MTX 15mg/wk.

Left elbow arthritis with enthesitis



Present issues:

- Flare in plaque psoriasis and left elbow and left knee enthesitis and arthritis of the 2nd toe.
- Did not respond to Adalimumab 40mg biweekly, which was previously effective and actually worsened the plaque psoriasis.
- Now started on Ixekizumab 160mg subcutaneous stat, followed by 80mg, for a total of 5 doses till now.
- Arthritis persisted; therefore, methotrexate was initiated at 10mg/week and subsequently increased to 15mg/week.
- Skin lesions and arthritis are improving.
- Discussed Guselkumab with her, but she is not currently willing.
- Plans to conceive in the near future.

Left knee arthritis

Left 2nd toe dactylitis

Questions

- Was it a good choice to use Ixekizumab, given that the patient had already used Secukinumab? And what would be the next best choice?
- How to proceed about choice of drugs, considering her plans for pregnancy?

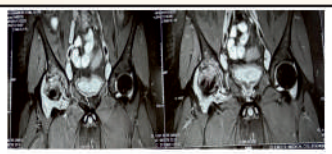
The Road To The Axis
Dr. Sreedevi S

THE ROAD TO THE AXIS

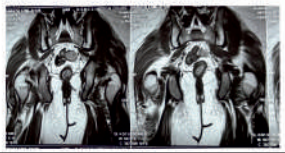
Dr Sreedevi S

ASSISTANT PROFESSOR IN RHEUMATOLOGY
THIRUVANATHAPURAM GOVT MEDICAL COLLEGE
THIRUVANANTHAPURAM
KERALA

MRI Hips



- Right hip Moderate hip joint effusion with distension of the capsule, diffuse synovial thickening, and cortical erosions in the femoral head
- Left hip mild effusion, mild synovial thickening with enhancement



T1W-MRI SI JOINT

Bilateral fatty metaplasia, erosions, Right joint pseudo-widening, irregular joint borders bilaterally

His Story- 17 yrs/Male - Duration of 9 months

3 months	6 months	After 1 month of ATT	After 4 months of ATT
• Oligoarthritis- Left hip ,Right hip ,Rt knee • Difficulty walking • Left posterior part of heel pain and swelling? Achilles tendonitis	• Treated as TB arthritis • No improvement in arthritis • Referred to our center	• Polyarthritis of small and large joints of both hand and feet	• Plaque lesions palms,soles,scalp • Low back ache 1ith EMS>30min-2 months • Bed bound-1 months

c/o radiating neck pain, with no weakness of limbs 3 months into treatment –MRI CERVICAL SPINE TAKEN

MCRAES LINE-ODONTOID TOUCHING IT

CHAMBERLAIN LINE-ODONTOID TIP 5.5 mm ABOVE IT

BASILAR IMPRESSION

Dens irregular ,sclerotic with expansion of tip. Endplate irregularities and sclerosis AA,AO joints

Examination

- Patient conscious oriented
- Vitals Stable
- BMI-25.6
- Skin-Acanthosis Nigricans, Scaly plaque lesions-scalp, palms, soles Psoriasis
- Nail-All hand nails-subungual hyperkeratosis, pitting
- MSK-Pain, swelling-Bilateral Elbow,wrist,2-5 PIP&DIP
  - Bilateral MCP squeeze+
  - Both-Hip-restriction of all movements Rt>Lt, Thomas Test + Rt Hip
  - Pain ,swelling –B/L Knee, Ankle, midtarsal joints
  - Left Achilles Tendonitis
  - Fabers couldn't be done

PSORIATIC ARTHRITIS

Treatment

- @Admission-Polyarticular arthritis + Axial-T MTX 10 mg hiked to Inj 20 mg s/c weekly once +Etoricoxib /Ibuprofen

(couldn't hike methotrexate as he had transaminitis with Fibroscan showing LSM-8 Kpa)

- @ 1 monthBASDAI-7
  ASDAS 4.1
  DAPSA->13
  - ✓ Added Inj **Adalimumab 40 mg sc bi-monthly** * 6 doses (3 month
  - ✓ High ds activity Axial + Peripheral
  - ✓ Physiotherapy
- @4 months BASDAI-6
  ASDAS 3.9
  DAPSA-10
  - ✓ Cervical involvement-Physiotherapy avoided
  - ✓ Financial constraints
  - ✓ No 50% reduction BASDAI/ASDAS CRP change <1.1
  - ✓ ?TNFi Non response
  - ✓ Replaced with **Etanacerpt 50 mg weekly once** *8 doses+ Inj MTX 20 MG

Investigations

Hb	10	Ferritin	1000
TC	15140cu mm	RF	negative
ESR	95mm/hr	Anti CCP	Negative
CRP	31mg/dl	ANA(IF)	NEGATIVE
Uric acid	3.1 mg/l	HLAB27	NEGATIVE
RBS	200 mg	Chol/LDL/TGS	198/102/146
Sgot/pt	45/54 IU	Hba1c	9
Na/K	132/4.5 meq	Ca/P	9.2/3.4

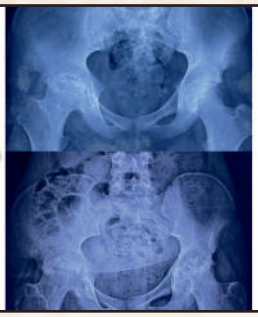
Treatment continued

- @6 months BASDAI 2
  ASDAS CRP 1
  DAPSA 2
  - Financial constraints
    BASDAI <4/ASDAS CRP<2.1 (Remission for 6 months
    Etanacerpt made once in 2 week *12 doses along with Mtx
- @1 year BASDAI 6
  ASDAS CRP 3.1
  DAPSA 13
  - Disease active on tapering biologic
    Family not willing to continue biologicals due to financial constraints
    Added Tofacitinib 5 mg od hiked to bd along with Mtx
- @1.5 YRS BASDAI <4
  ASDAS CRP <2.1
  DAPSA 2
  MDA>5/7
  BASMI 8
  - Continues **MTX 15 mg oral**
    Tofacitinib 5 mg OD
    NSAID SOS

XRAY PELVIS WITH B/L HIPS

28.12.23

- RT SI joint irregularities - ?pseudo widening ,sclerosis BILATERAL GRADE 3 SACROILIEITIS
- RT HIP –head deformed, cortical erosions, joint space narrowing, acetabular sclerosis and erosions- RT HIP ARTHRITIS



24.04.24

- SI joints obscured
- BILATERAL HIP ARTHRITIS

Questions

- Should we screen for axial disease in PsA, even in the absence of symptoms?
- Is axial PsA a distinct phenotype or part of the Axial SpA spectrum — and should management strategies differ accordingly?

Overcoming a Herclean Task
Dr. Kasturi Hazarika

Overcoming a Herculean Task

Dr Kasturi Hazarika

Consultant
Department of Clinical Immunology and Rheumatology
Apollo Excelcare Hospital
Guwahati, Assam

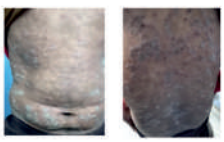
August 2021

DAPSA SCORE - 48

- Skin: • Disease Flare • PASI – 30.2/
- Joint: • Peripheral: SJC/TJC 18/20 • Axial : BASDAI 6.3

Secukinumab increased to 300mg/month- subSEQUENT BD → AFTER 2 MONTHS → PASI 29.2 TJC/SJC 12/12 DAPSA - 41 → Secukinumab Changed to Tofacitinib 5mg BD → (At the end of 3 months - VAS- 0 x UCAIC - 0) → Tofacitinib dose increased to 10 mg BD

- 2015- 17 year old male with history of inflammatory back pain and asymmetric polyarthritis (large and small)
- FABER's positive/ High inflammatory markers with x ray suggestive of sacroiliitis; HLA B27positive
- SPA(A+P)--> Started on NSAIDS/SSZ--> LOST TO FOLLOW UP



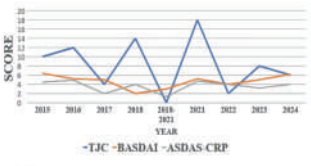
- 2018-Recurrence of inflammatory back pain/neck and back stiffness/ heel pain
- Plaque-type psoriatic skin lesions over the feet hands scalp/pitting of finger nails
- Psoriatic arthritis, PASI-29/ DAPSA-46/HAPSI-16/100

SKIN &ARTICULAR FLARE- INFLIXIMAB 5MG/KG → AFTER 3 MONTHS-->IR-->INFLIXIMAB INCREASED TO 10MG/KG AND TACROLIMUS ADDED- PASI –4 ; 1 TJ / SJ 2 → POST SURGERY-SKIN & ARTICULAR FLARE-PASI-22.2/BASDAI-5.4/SJC/TJC-12/12-INFLIXIMAB GIVEN AT 10MG/KG --> PASI 20/BASDAI-5/SJC/TJC-8/8 → INFLIXIMAB CONTINUED ALONG WITH PREDNISOLONE 10MG-ON TAPERING DISEASE FLARED(TOTAL DOSE-14)-PASI 28.4/BASDAI-5.1/DAPSA 36 → SWITCHED TO 50mg GOLIMUMAB --> 1Month later --> PASI 20/BASDAI - 4

- 2016-low dose prednisolone along with methotrexate 10 mg / week along with topical therapy for skin lesions as advised by the dermatologist
- 2017- gradually methotrexate was increased upto 25mg/week, Leflunomide was added, peripheral joint pain improved, PASI-14, persistent neck pain and back pain, seemed mechanical
- 2018-Flare of symptoms, TJC-14/SJC-14,PASI-20.4/ Started on injection Secukinumab 150mg s.c once monthly(without loading dose) along with injection methotrexate 25mg /week

AFTER 1 MONTH OF GOLIMUMAB

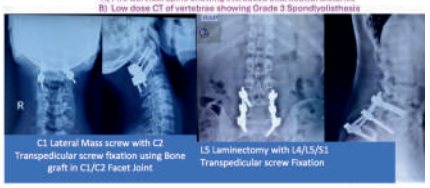
after 3 months	ISSUES PENDING
PASI- 2.9 BASDAI-2 ASDASCRP-1.3	NECK PAIN - DTR increased
DAPSA-8	LOW BACK ACHE - Radicular symptoms



Pending issues

A) MRI Cervical spine showing increased atlantoaxial distance
B) Low dose CT of vertebrae showing Grade 3 Spondylolisthesis

SOLUTION



Questions

- How long do we wait for a response to medication to call it truly refractory?
- What is the risk vs benefit ratio for the usage of biologics or small molecules at higher dosages in refractory PsA?

Takes Two to Tango
Dr. Nikhil Gupta

Takes two to tango

Dr Nikhil Gupta

Senior Consultant Rheumatologist
Centre for Arthritis and Rheumatological diseases, New Delhi

TNF Inhibitor Switch (June 2022 – Early 2023)

- **Switched to Methotrexate + Leflunomide + Golimumab**
- **Golimumab 50 mg once monthly**
- **Outcome:**
  - **Joints:** Complete remission over 3 months
  - **Skin:**
    - PASI improvement ~90%
    - Complete remission with topical therapy
- **Sustained control** until mid-2023

PATIENT PROFILE

- **Age / Sex:** 50-year-old male
- **Diagnosis:** Psoriatic arthritis (peripheral predominant)
- **Disease duration:** Since 2012
- **Follow-up:** Since 2017
- **Comorbidities:**
  - Obesity – BMI 31 Kg/m2
  - Type 2 diabetes mellitus (diagnosed April 2023; controlled on oral agents)
- **Joint involvement:**
  - Bilateral MCP, PIP, DIP, wrist
  - Elbows
  - Knee, feet
  - Dactylitis – left hand thumb
- **Skin disease:**
  - Severe plaque psoriasis, including nails
  - **Body surface area involvement >90%**
  - Baseline **PASI >90**

Disease Flare and Dual-Target Challenges (Mid-2023–early 2025)

- **April 2023:** New-onset diabetes mellitus
- **July 2023:**
  - Severe **psoriasis flare, including nail psoriasis**
  - Joints remained quiescent
  - Golimumab interval extended to **every 45 days**
- **Switched to Methotrexate + Apremilast + golimumab**
  - No response after 80 days
- **Switched to Methotrexate + Secukinumab re-introduced (300 mg loading)**
  - **Skin:** PASI ↓ ~70%, nail psoriasis partial response
  - **Joints:** Severe flare → patient bed-bound
- **Switched to Methotrexate + Golimumab + short-course steroids**
  - Joint symptoms resolved
  - Skin worsened
- **Alternate-month Golimumab and Secukinumab (6 months) + Methotrexate**
  - **Post-secukinumab:** Joint flares requiring steroids, PASI score ↓ ~70%
  - **Post-golimumab:** Partial skin response but complete joint response

Initial Treatment and Early Course (2017–2019)

- **Methotrexate + Leflunomide + short course of steroids**
  - Good control of **skin, joints, nail psoriasis, and dactylitis for ~2 years**
- **2019:**
  - Secondary loss of response
  - Severe flare of **both joints, skin and nail psoriasis**
- **Baricitinib 4 mg/day added**
  - **Partial response** in joints, skin and nail psoriasis, over 6 months
  - Persistent disease activity
  - Required **prednisolone 10 mg/day**

IL-23 Strategy and Targeted Combination (2025)

- **April 2025:** Guselkumab loading dose
  - **Skin:** PASI 90 → **100% clearance including nail psoriasis**
  - **Joints:** Partial response
- **June 2025:** Golimumab added (1 month post-loading)
  - **Joint remission achieved (100%)**
- **Maintenance strategy:**
  - Alternating **guselkumab and golimumab** every months
  - November 2025: Guselkumab
  - December 2025: Golimumab
- **Current Status (2026)**

Skin: Complete remission (PASI 100) including nail psoriasis
Joints: Complete remission
Steroid-free

First Biologic Phase (2020)

- **Switched to Methotrexate + Leflunomide + Etanercept**
- **Response:**
  - **Joints:**
    - Rapid improvement
    - ~80% remission within 3 months
  - **Skin:**
    - Partial response
    - PASI improvement ~50% at 6 months
- **Limitation:** Persistent moderate-to-severe psoriasis

Questions

➢How should clinicians justify dual biologic use in D2T PSA — should this remain a rescue strategy, or could it become the standard of care in the future?

➢In a patient exhibiting clear pathway discordance—TNF-responsive joints but IL-23–responsive skin—how do you view the role of planned dual biologic therapy versus rapid cycling of monotherapy?

IL-17 Inhibition Phase (2021–Mid-2022)

- **Switched to Methotrexate + Leflunomide + Secukinumab**
- **Secukinumab 300 mg loading → 300 mg monthly**
- **Skin response:**
  - PASI improvement **80–90% including nail psoriasis**
  - Relapse **~25 days after maintenance dose**
- **Joint response:**
  - **No meaningful improvement**
  - Required continuous **prednisolone 10 mg/day**
- **Conclusion:** Skin-dominant response with joint failure

Note: Clinical images shall be added

Surviving the Therapy not The Disease
Dr. Vishnupriya S

Surviving the therapy, not the disease

Dr. VISHNUPRIYA S
MD(Gen med) DM (Clinical Immunology and Rheumatology)
SKS Hospital & Postgraduate Medical Institute, Salem

INVESTIGATIONS

Sr cortisol –low
Sr ACTH-low
Sr. calcium-N
Intact PTH-normal
DEXA scan –osteoporosis (neck of femur); osteopenia wrist and spine

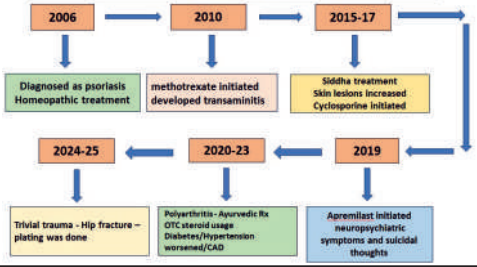
History

46/M
IT Professional
Married
Two children
Psoriasis for 20 yrs
Polyarthritis for 5 yrs
T2DM/SHT/CAD for 3yrs

DISEASE ACTIVITY SCORES

- **DAPSA -83(High disease activity)**
- **NAPSI-35(Moderate disease activity)**
- **PASI -41.8(Severe psoriasis)**

Clinical Course



TREATMENT PLAN

Disease specific	Comorbidities
Cutaneous Psoriasis Nail psoriasis Dactylitis Enthesitis Without axial symptoms No history of IBD/uveitis MTX induced transaminitis Apremilast induced neuropsychiatric symptoms Mixopathy treatment	T2DM SHT CAD Secondary adrenal insufficiency Depression Osteoporosis

Clinical Examination

Skin psoriasis
Eye-normal
Nail –pitting /dystrophy

GAIT-NORMAL
Neck/Hip ROM-Normal
FABER-negative
Chest expansion –normal
Pain and swelling over IP Thumb and DIP little finger
Dactylitis little finger
Bilateral Achillies enthesitis
TJC-15,SJC-12

TREATMENT AND FOLLOW-UP

- ☐ Inj.Secukinumab 300 mg once weekly for 5 weeks and then monthly once
- ☐ Inj.Teriparatide 20mcg daily
- ☐ T.Hydrocortisone 10 mg 1-0-1-0

- ☐ Skin psoriasis improved
- ☐ Dactylitis and arthritis persisting

INVESTIGATIONS

CBC RFT/LFT-N CRP-40 ESR-55mm/hr Viral markers-neg IGRA-NEG TSH-N	RFT/LFT-N FECAL CALPROTECTIN NEGATIVE	ECHO-RWMA EF-40% NO PHT CT CHEST-NORMAL	RF/Anti CCP/HLA B27-neg GI SCOPY-N MRI SPINE-N

QUESTIONS

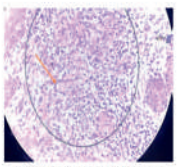
- Treatment modalities to reduce arthritis and dactylitis in this patient, with multiple comorbidities?
- Cardiovascular risk in psoriatic arthritis compared to RA and the use of TNFi in patients with reduced ejection fraction in practice?

Double Trouble
Dr. Harikrishnan G

Double Trouble

HARIKRISHNAN G
ASSISTANT PROFESSOR RHEUMATOLOGY
GOVT MEDICAL COLLEGE KOTTAYAM

Ulcer edge Biopsy



well defined granuloma with Langhan's giant cell

Dx: disseminated TB (lung, skin, and bone), Anti-synthetase syndrome

Rx: MMF stopped, ATT started

59 /F; k/c/o HTN, Pulm. TB -33 yrs ago, ATT x 6 months

Sept 2024

DOE x 6m

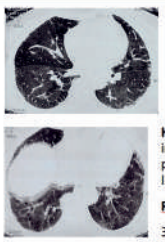
Intermittent fever x 1 m

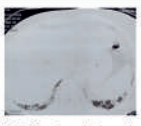
wt loss +

Evaluated at Pulmonary medicine dept

TB workup (-)ve

FVC: 67%, FEV1: 69%,FEV1 ratio: 0.89





HRCT Chest: subpleural interstitial changes b/l lungs, post TB changes, early NSIP ILD

Rx: Tab.methylprednisolone 32mg/d

Oct 2024

Rheumatology workup for CTD-ILD

h/o mechanic's hand +

MMT8: 80/80

ANA IF: 1+ nuclear dots

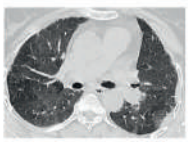
Myositis profile: anti EJ+++,anti Ro52+++

December 2025

Completed 6 months of Anti-tuberculous Rx

On prednisolone 7.5mg/d

Ulcer healed, no features of active anti-synthetase syndrome



Repeat HRCT Thorax: no significant progression of ILD

Oct 2024

Dx: anti-synthetase syndrome anti EJ +, NSIP-ILD

Rx: MMF 2g/d, prednisolone 40mg (0.5mg/kg)

Feb 2025

DOE

on prednisolone 10mg/d

MMF 2g/d

Serial PFT: FVC-70% (3%)

May 2025

Non-healing ulcer- Rt ankle x 2 m

unable to bear weight, intermittent fever

Take-home message

- In IIM, keep a high index of suspicion for infections, especially in atypical presentations and in patients on immunosuppressants
- Concomitant treatment of IIM and TB can be challenging
- Treatment decisions should be made on a case to case basis

D/ds considered

- **Venous ulcer:** site of ulcer ✓

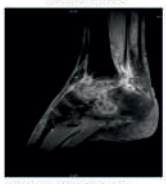
  no varicose vein/venous doppler normal ✗
- **Infection:** immunosuppressed, fever ✓
- **Anti-synthetase syndrome related**/disease activity- fever, ✓ not responding to multiple courses of antibiotics, cultures sterile
- **Malignant ulcer:** old age, immunosuppression ✓

Questions for the panel

- What are the indicators of infection radiologically in a patient with pre-existing ILD?
- What are the components to be looked for radiologically, to comment on improvement or worsening of ILD?
- In a patient with active TB and worsening IIM-ILD, how would you balance antimicrobial therapy with immunosuppression?

June 2025

MRI Rt ankle



diffuse marrow edema and enhancement in talus and subarticular portion of calcaneum, ankle jt effusion, synovial thickening and enhancement

HRCT Thorax

Multiple centrilobular nodules with tree in bud appearance

Deceptive Beginnings: A Rapidly Progressive ILD Culminating in Early Mortlity

Dr. Rahul Bisaralli

Deceptive Beginnings : A Rapidly Progressive ILD culminating in Early Mortality

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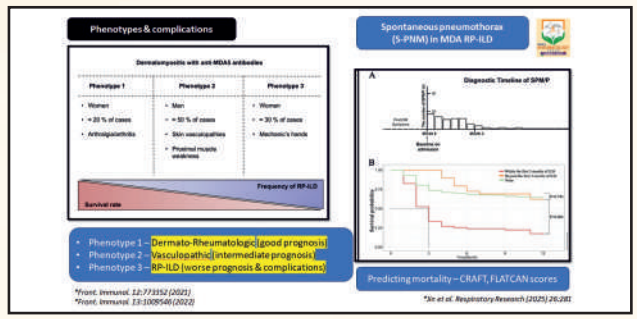

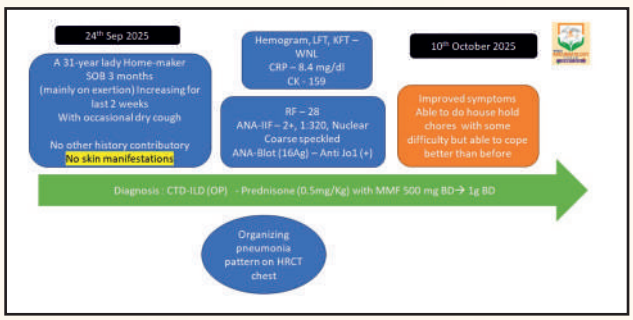

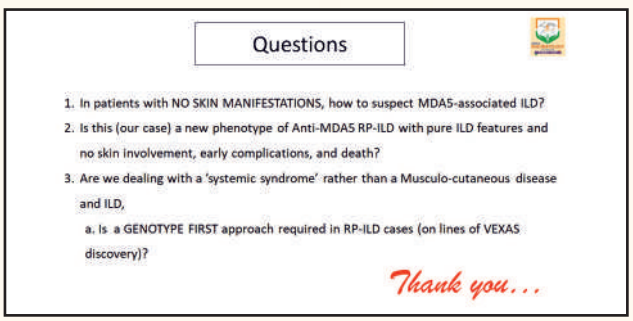

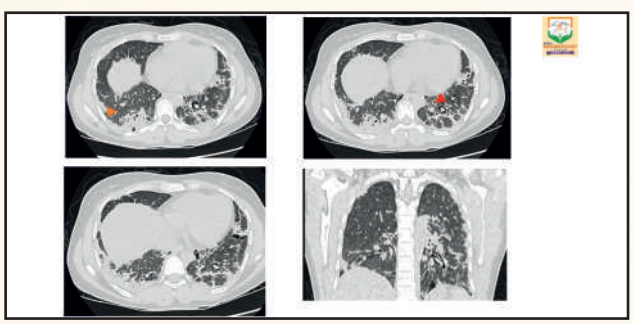

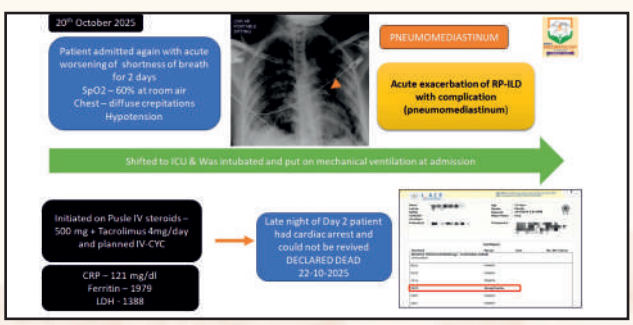

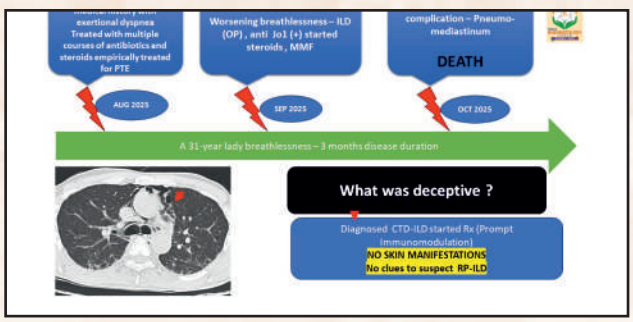

PLATFORM PRESENTATIONS BY RHEUMATOLOGY TRAINEES

ORR1: A Wolf in disguise: Insights beyond the Autoimmune Lens

Nanditha S
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Introduction: Subcutaneous panniculitis-like T-cell lymphoma (SPTCL) is a rare primary cutaneous lymphoma accounting for <1% of non-Hodgkin lymphomas, predominantly affecting middle-aged adults with a slight female preponderance. Patients usually present with multiple painless subcutaneous nodules or plaques over the trunk and extremities, frequently leading to misdiagnosis as benign panniculitis.

Case Report: A 50-year-old lady presented with a 3-month history of intermittent high-grade fever, inflammatory polyarthritis and indurated skin lesions over cheeks and upper limbs. She had been previously treated as undifferentiated connective tissue disease due to positive RF, anti-CCP, ANA, and weak PL-7 antibodies with chronic leukopenia and panniculitis-like lesions. During the current admission she had persistent fever with bicytopenia, hyperferritinemia, hypofibrinogenemia, and transaminitis (H-score 174), consistent with MAS. FDG PET CT showed FDG avid lesions in her cheeks, chest wall, abdomen and upper limbs with soft tissue thickening encasing superior mesenteric vessels. Deep skin biopsy from cheek and abdomen showed lobular panniculitis with rimming of adipocytes by atypical lymphoid cells, fat necrosis, and dermal mucin. IHC revealed CD3+, CD4+/CD8+ T cells with focal TCRβF1 positivity, TIA-1 and granzyme B positive, and negative CD30/CD56, confirming SPTCL. The clinical course was complicated by septic shock due to Stenotrophomonas maltophilia. Despite high-dose corticosteroids and intensive supportive care, the patient succumbed to her illness.

Conclusion: SPTCL can masquerade as autoimmune disease with misleading autoantibodies. Presence of panniculitis with cytopenias should prompt early deep biopsy with IHC. MAS is a complication of SPTCL which clinicians should be on a watch out for that can critically worsen outcome, underscoring the need for rapid diagnosis and institution of lymphoma-directed therapy.

ORR2: Recurrent Fever, Progressive Polyarthritis and Persistent Coronary Aneurysms- A Pediatric Inflammatory Enigma

Dhiliprajan K, P.S.Arul Rajamurugan, S Ramesh, M.Sabarinath, R.Ishwarya
Institute of Rheumatology, Madras Medical College, Chennai

Introduction: Kawasaki disease is an acute childhood vasculitis and leading cause of acquired pediatric heart disease, whereas juvenile idiopathic arthritis is a chronic inflammatory arthritis. Their overlap is rare and challenging. We describe a child with recurrent KD-like episodes evolving into chronic

polyarthritis with persistent coronary aneurysms, refractory to conventional therapy and requiring biologic

Case Report: A 7-year-old girl, born of a third-degree consanguineous marriage, had a 3-year history of inflammatory polyarthritis. At 4 years, she presented with fever, conjunctivitis, polymorphic mucocutaneous rash, elevated acute-phase reactants, and a coronary artery aneurysm, and was diagnosed with Kawasaki disease (KD). She was treated with IVIG, steroids, and aspirin. Six months later she developed recurrent fever, polyarthritis, high APR and ferritin, and worsening saccular coronary aneurysms, requiring repeat IVIG, escalated steroids, aspirin, and acitrom. One year later, recurrence with arthritis, leukocytosis and thrombocytosis prompted revision to SOJIA–KD overlap, and methotrexate was started. Arthritis flared with steroid tapering and coronary aneurysms persisted. RF, ACPA, and ANA were negative. She later developed alopecia and progressive deforming arthritis, leading to initiation of infliximab. Whole-exome sequencing showed a pathogenic DEPDC5 variant without clinical correlation. She is currently maintained on infliximab, methotrexate, steroids, and supportive therapy.

Conclusion: This case underscores the diagnostic challenge of Kawasaki disease–JIA overlap with persistent coronary aneurysms and chronic arthritis. Recurrent inflammation and steroid dependence required escalation to biologic therapy. Early recognition and targeted immunosuppression are essential to reduce long-term cardiac and joint complications

ORR3: The Monoclonal Mimic of Myositis

Pragya, Eshan Taneja, Naina Mehta, Neeraj Jain, Lalit Duggal
Sir Ganga Ram Hospital, New Delhi

Case Report: A 57-year-old previously healthy man presented with acute right thigh pain, difficulty rising from sitting, and subsequent ecchymosis, later involving the contralateral thigh. MRI demonstrated bilateral proximal muscle edema. He reported weight loss and fatigue, with examination revealing proximal lower-limb weakness and subtle blanchable erythema. Laboratory evaluation showed deranged coagulation (prolonged PT/aPTT), normal muscle enzymes, and mildly elevated inflammatory markers. Autoimmune testing revealed ANA, Ro-52, and anti-SAE positivity, prompting treatment for presumed inflammatory myositis. However, further evaluation revealed acquired factor X deficiency. Work-up for plasma cell dyscrasia demonstrated elevated lambda free light chains and 5% clonal plasma cells on bone marrow examination. Cardiac imaging showed features of infiltrative cardiomyopathy, while fat pad biopsy was negative for amyloidosis. Despite <10% plasma cells, the presence of multisystem involvement established a diagnosis of monoclonal gammopathy of clinical significance (MGCS). The patient was initiated on daratumumab-based therapy with clinical improvement.

ORR4: Late-Onset Pompe Disease Mimicking Inflammatory Myositis: A Diagnostic Pitfall

Chandrashekara MN, Kirti Gupta, Shankar Naidu, Susheel Kumar, Shefali Sharma Khanna, Varun Dhir, Aman Sharma, Sanjay Jain

Dept of Clinical Immunology and Rheumatology, PGIMER, Chandigarh

Introduction: Pompe disease is a rare autosomal recessive metabolic myopathy caused by lysosomal acid alpha-glucosidase deficiency, leading to glycogen accumulation in skeletal muscle. Late-onset Pompe disease presents with progressive limb-girdle weakness and elevated muscle enzymes, often mimicking autoimmune inflammatory myositis and resulting in diagnostic delay.

Case Report: A 35-year-old woman presented with progressive proximal muscle weakness for one year. Initial evaluation elsewhere showed elevated creatine kinase, positive antinuclear antibody, borderline myositis antibodies (Mi-2 alfa 1+, Anti Ro 52 1+), myogenic electromyography, and a muscle biopsy interpreted as polymyositis. She was treated with high-dose glucocorticoids and cyclophosphamide without clinical improvement. Weakness progressed, leading to wheelchair dependence and respiratory distress. At our centre, she had severe neck, truncal, and limb-girdle weakness with preserved reflexes and no connective tissue disease features. Despite pulse methylprednisolone and rituximab, she developed type 2 respiratory failure. Detailed reassessment revealed subtle weakness since adolescence, scapular winging, scoliosis, fractures, and asymmetric septal hypertrophy in 2D Echo. Thigh MRI showed predominant fatty muscle replacement (Figure 1). Repeat myositis antibodies were negative. Repeat muscle biopsy demonstrated glycogen storage disease (figure 2), and genetic testing confirmed pathogenic GAA variants, establishing late-onset Pompe disease. The patient succumbed to hospital-acquired pneumonia.

Conclusion: Late-onset Pompe disease may mimic inflammatory myopathy, and low-titer autoantibodies or biopsy inflammation can mislead clinicians. Absence of connective tissue disease features, poor response to immunosuppression, fatty muscle replacement on MRI, and multisystem involvement should prompt evaluation for metabolic myopathies. Early genetic confirmation enables timely enzyme replacement therapy.

Fig 1

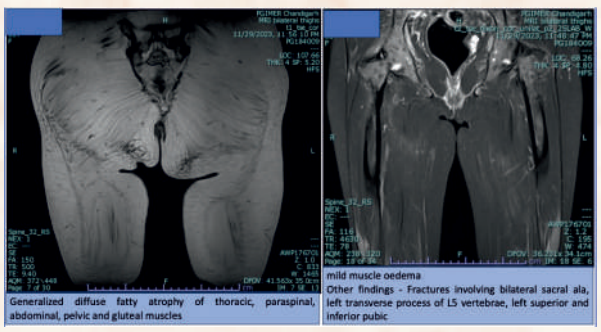
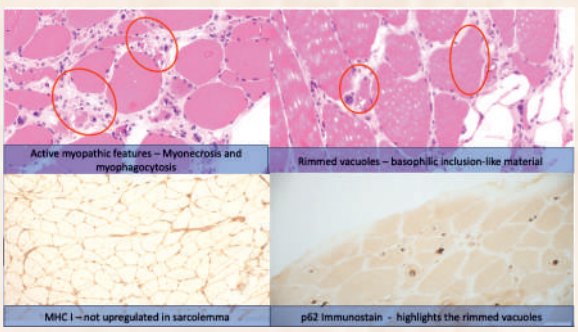


Fig 2



ORR5: Refractory Sweet-like neutrophilic dermatosis with chondritis, periorbital edema and inflammatory arthritis unveiling VEXAS syndrome in an elderly male

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Dept of Rheumatology and Immunology, Max Superspeciality Hospital, Saket, New Delhi

Introduction: VEXAS syndrome is an adult-onset autoinflammatory disorder caused by somatic UBA1 mutations and characterized by heterogeneous, steroid-dependent inflammatory manifestations. Negative autoimmune serology and absence of fever, as seen in this case, may delay diagnosis, underscoring the importance of recognizing characteristic clinical clusters and pursuing timely genomic evaluation.

Case Report: An 84-year-old male presented with recurrent painful erythematous plaques and nodules involving the scalp, face, neck, and lower limbs. Skin biopsy confirmed Sweet-like neutrophilic dermatosis. He had poor and unsustained response to corticosteroids, colchicine, and dapsone, with relapse on tapering. Autoimmune workup including ANA, ENA, ANCA, rheumatoid factor, anti-CCP, complements, and serum protein electrophoresis was negative. He subsequently developed urinary retention with weight loss; MRI prostate showed PI-RADS v2–III changes, while FDG PET-CT excluded malignancy. The disease course was complicated by recurrent external ear chondritis, inflammatory arthritis involving bilateral shoulders and ankles with early morning stiffness, and bilateral periorbital edema. Symptoms were steroid responsive but steroid dependent. There was no history of fever, oral ulcers, respiratory, cardiac, or haematological involvement. Given refractory multisystem inflammation, clinical exome sequencing identified a pathogenic somatic UBA1 mutation, confirming VEXAS syndrome. Treatment with Janus kinase (JAK) inhibitors with steroid taper resulted in sustained clinical remission.

Conclusion: VEXAS syndrome should be suspected in elderly males with refractory Sweet-like neutrophilic dermatosis, inflammatory arthritis, periorbital edema, weight loss, ear chondritis, and steroid-dependent multisystem inflammation. Early genomic confirmation enables targeted therapy and improved outcomes.

ORM1: Pancreatitis-Panniculitis-Polyarthritis Syndrome: A Diagnostic Masquerader of Septic Arthritis

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Department of Medicine, Shri Balaji Institute of Medical Science, Mowa, Raipur

Introduction: Pancreatitis-panniculitis-polyarthritis (PPP) syndrome is a rare extra-pancreatic manifestation of pancreatic disease, with only 30 cases formally reported worldwide. It is frequently under-recognized, often mimicking rheumatologic or septic conditions and leading to diagnostic delay.

Case Report: We report a 34-year-old chronic alcoholic who presented with dull aching abdominal pain for 1.5 months, followed by acute onset severe polyarticular pain and bilateral lower limb swelling for one week. Initial examination revealed tense, tender swelling of both legs, raising suspicion of compartment syndrome. Subsequent evaluation showed inflammatory arthritis involving the second and third metacarpophalangeal and interphalangeal joints of the left hand, right elbow, and bilateral ankles, along with multiple tender subcutaneous nodules over the legs suggestive of panniculitis (Figure 1). Marked leukocytosis and systemic inflammation led to a provisional diagnosis of septic arthritis. However, markedly elevated serum amylase and lipase levels (>50-fold) prompted diagnostic reconsideration. Serial investigations demonstrated parallel clinical and biochemical improvement with declining inflammatory markers. Correlation of clinical, biochemical, radiological, and histopathological findings confirmed the diagnosis of pancreatitis–panniculitis–polyarthritis (PPP) syndrome (Table 1).

Conclusion: PPP syndrome should be considered in patients presenting with unexplained polyarthritis and panniculitis, particularly in those with alcohol use or abdominal symptoms. Early recognition facilitates appropriate management by targeting pancreatic pathology.

TABLE 1: Serial laboratory parameters during hospital stay

Laboratry Parameters	Total Leukocytes count (cells/mm^3)	Hemoglobin (gm/dl)	Platelet count (cells/m^3)	S. amylase (U/L)	S. Lipase (U/L)	S. albumin (gm/dl)	S. Procalcitonin
Baseline	17500	9.8	4.5×10^5	4275	5252	2.37	>50.00
Day 6	36100	8.9	5.22×10^5	6121	11961	2.41	
Day 10	23200	8.7	5.96×10^5			2.24	0.20
Day 13	14600	6.7	4.52×10^5	1730	3028	1.66	
Day 25	12900	9.5	5.35×10^5	1151	2074	2.39	
Day 30	9900	10.2	4.12×10^5				

Figure 1

Figure 1 : 1A. Polyarthritis with involvement of 1st and 2nd Metacarpophalangeal Joints, Proximal and Distal interphalangeal joints of 2nd and 3rd phalanges of left hand . **1B.** Joint inflammation of right elbow joint.

Figure 2: Multiple red to violaceous indurated lesions seen over the anterolateral aspects of both legs; Erythema Nodosum (EN)

Figure 3. 3A. Histopathological section of subcutaneous tissue showing septal panniculitis characterized by thickened fibrous septae with lymphohistiocytic infiltrate and relative preservation of fat lobules. **3B.** Sections show lobular panniculitis with prominent fat necrosis characterized by loss of adipocyte outlines and associated inflammatory infiltrate.

ORM2: The Ascites That Wasn't Cirrhosis: A Rare Encounter with TAFRO Syndrome Revealed by Multimodal Investigations

Mohamed Yasin, Chandrasekar S
Department of Medicine, Stanley Medical College, Chennai

Introduction: Not all ascites is cirrhosis. In India, TAFRO syndrome—an aggressive variant of idiopathic multicentric Castleman disease characterized by thrombocytopenia, anasarca, fever, reticulin fibrosis, and organomegaly—remains exceedingly rare, with only one indexed case reported. We report a diagnostically delayed case emphasizing early suspicion and timely histopathologic confirmation.

Case Report: A 45-year-old woman with long-standing rheumatoid arthritis and previously treated pulmonary tuberculosis presented with progressive abdominal distension, pedal edema, and exertional dyspnea of three months' duration. Examination revealed tense ascites, hyperpigmented skin, and inguinal lymphadenopathy. Laboratory evaluation showed bicytopenia with anemia and thrombocytopenia and elevated inflammatory markers, with negative viral serology and autoimmune markers. Imaging demonstrated massive ascites, hepatosplenomegaly, and lymphadenopathy, while ascitic fluid revealed low SAAG, high protein with negative tuberculosis testing. FDG PET-CT showed

metabolically active supra- and infradiaphragmatic lymphadenopathy with splenic and skeletal involvement. Bone marrow biopsy revealed reticulin fibrosis, and excisional lymph node biopsy confirmed multicentric Castleman disease with negative HHV-8 serology confirming idiopathic disease. A diagnosis of idiopathic multicentric Castleman disease with TAFRO syndrome was established. Treatment with pulse methylprednisolone, rituximab, and mycophenolate led to resolution of cytopenias, ascites, and organomegaly with clinical improvement.

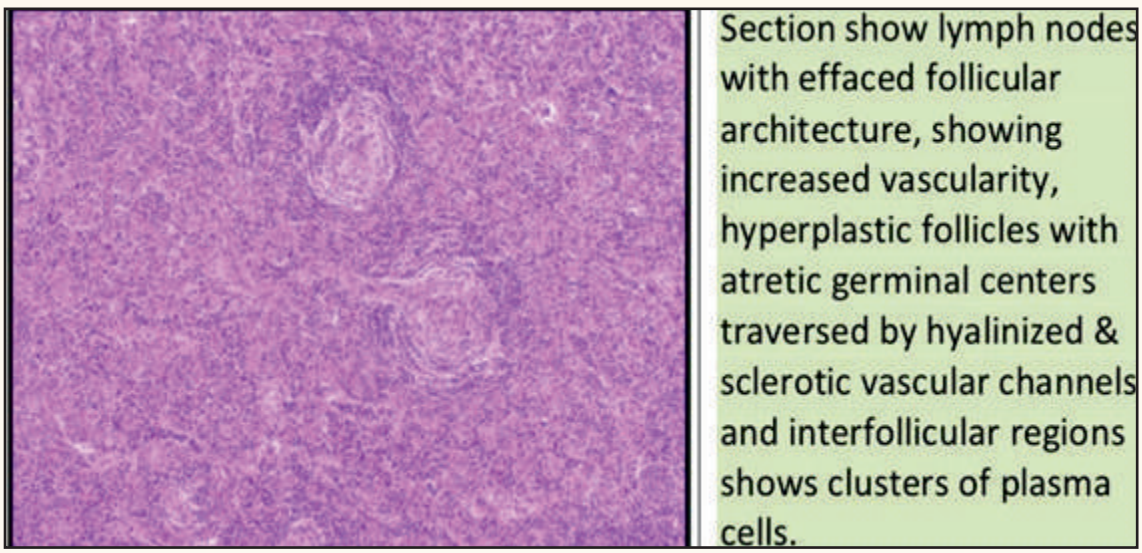
Conclusion: TAFRO syndrome is a rare, life-threatening manifestation of idiopathic multicentric Castleman disease that may mimic chronic liver disease and delay diagnosis. This case highlights the importance of early clinical suspicion and timely histopathologic evaluation, as prompt immunosuppressive therapy can reverse systemic manifestations, improve outcomes, and reduce morbidity and mortality.

Figure 1

INVESTIGATIONS	
▪ Initial investigations revealed Bicytopenia(Anemia, Thrombocytopenia)	
TC 9,600, RBC 3.24 HB 5.9, HCT 22.8, PLT 52,000	S. Iron – 34 µg/dlS. Vit B12 – 528 pg/mlCRP – +ve
Urea – 27, Creat 0.9, Na 135, K – 4.5 TB 1.0, DB 0.3, OT 16, TP 5.5, S. Alb 3.8,PT 18.4, INR 1.4	Peripheral Smear – Dimorphic anemia (Normocytic normochromic with microcytic hypochromic)with Thrombocytopenia
HBsAg, Anti HCV, HIV – NON-REACTIVE	ECHO – No RWMA, Normal LVSF, EF 60%, No MR/TR
CECT Abdomen – Massive ascites, hepatosplenomegaly,and enlarged left inguinal nodes	USG Abdomen – Coarse liver echoes, ascites and splenomegaly
▪ A provisional diagnosis of volume overload state secondary to decompensated chronic parenchymal liver disease (DCLD) made.	
ASCITIC FLUID ANALYSIS (**Low SAAG High Protein** levels)	Alb 2.9, Protein 4.1, ADA – 4 U/L **CBNAAT – MTB not detected**
PV Doppler, Fibroscan, OGDscopy	Normal study
ANA, Anti CCP – NEGATIVE	S. amylese, lipase - 31/22 U/L
INGUINAL LYMPH NODE EXCISION BIOPSY	**Reactive follicular hyperplasia**
BONE MARROW BIOPSY	Hypercellular marrow with trilineage hematopoiesiswith reticulin-grade 2 marrow fibrosiswith no increase in blast/lymphomatous infiltration
▪ Despite treatment for DCLD, the patient developed fever and symptoms worsened.	

FURTHER WORKUP:	
FDG PET-CT Scan of Whole Body	**Lymphoproliferative disorder involving supra and infra diaphragmatic lymph nodal groups,** spleen, skeletal system
Repeat Excision Biopsy from the Axillary Lymph Node	**Mixed hyaline vascular and plasma cell variant of Castleman diseasePossibility of multicentric Castleman disease (MCD-TAFRO)**
HHV-8 Testing	**NEGATIVE**
Capillary Electrophoresis	**No monoclonal band seen, Normal**
▪ The findings, along with thrombocytopenia, anemia, fever, reticulin fibrosis in bone marrow and organomegaly, aligned with a diagnosis of **TAFRO syndrome.**	

Figure 2



ORM3: A Triple Threat: Antiphospholipid Antibody Syndrome with Acute Ischemic Stroke, Myocardial Infarction, and Severe Aortic Stenosis

Monesh Gupta
Department of General Medicine, MGM Medical College

Introduction: Antiphospholipid antibody syndrome is a systemic autoimmune thrombophilia with varied clinical manifestations. We report a young woman with recurrent pregnancy loss presenting with acute ischemic stroke and myocardial infarction in the setting of severe aortic stenosis, illustrating a rare and severe multisystem presentation of APS.

Case Report: A 28-year-old right-handed woman with a history of recurrent spontaneous abortions presented with sudden-onset right upper limb weakness and sensory loss, maximal at onset with partial spontaneous improvement. There was no involvement of cranial nerves, lower limbs, or bladder and

bowel function. Neurological examination suggested a cortical localization. Cardiovascular examination revealed a harsh grade IV ejection systolic murmur at the right second intercostal space with carotid radiation and a palpable thrill. Brain MRI demonstrated multiple acute cortical infarcts involving bilateral posterior frontal, precentral, and parieto-occipital regions, along with an old right parieto-occipital infarct. Electrocardiography showed features of an anteroseptal myocardial infarction. Transthoracic echocardiography revealed a thickened, severely stenotic aortic valve with anteroseptal hypokinesia and a reduced left ventricular ejection fraction of 35%. Immunological evaluation revealed triple-positive antiphospholipid antibodies, confirming the diagnosis of antiphospholipid antibody syndrome. The patient was managed with anticoagulation and multidisciplinary care.

Conclusion: This case highlights the aggressive multisystem involvement of antiphospholipid antibody syndrome in young patients. Early recognition of atypical cardiac and neurological manifestations is essential to prevent irreversible morbidity. A high index of suspicion and prompt multidisciplinary management can significantly improve outcomes in this potentially catastrophic autoimmune thrombophilia.

ORM4: Autoimmunity Striking the Gut and the Spine: A Rare Presentation Defying Boundaries

Rohan Ghosh
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Introduction: Systemic lupus erythematosus is a multisystem autoimmune disorder with diverse organ involvement, while ulcerative colitis is an immune-mediated gastrointestinal disease. Their coexistence is rare (~0.4%). Neuropsychiatric lupus is common, but cauda equina syndrome is exceptionally rare in SLE, making concurrent gastrointestinal and neurological presentations diagnostically challenging.

Case Report: A 31-year-old woman presented with fever, altered sensorium, inflammatory polyarthritis, photosensitive rash, oral ulcers, and frothy urine. Examination showed pallor, tachycardia, lupus skin lesions, and depressed consciousness. Investigations revealed pancytopenia, hyponatremia, sub-nephrotic proteinuria, high anti-dsDNA, anti-ribosomal P positivity, and low complements; renal biopsy showed class I lupus nephritis. She was diagnosed with multisystem SLE and improved after hypertonic saline, pulse steroids, and cyclophosphamide. She later developed acute diarrhea and neurogenic bladder–bowel dysfunction. Fecal calprotectin was elevated; colonoscopy with biopsy confirmed ulcerative colitis with superadded CMV infection, responding to mesalamine, topical steroids, and antivirals. Persistent neurological deficits prompted evaluation, revealing bilateral S1 radiculopathy and MRI evidence of sacral arachnoiditis causing cauda equina syndrome. CSF showed mild protein elevation without infection, supporting inflammatory lupus myelitis. Continued cyclophosphamide led to clinical and radiological recovery, with restoration of bladder and bowel function on maintenance immunosuppression.

Conclusion: This case demonstrates rare coexistence of SLE and ulcerative colitis complicated by CMV colitis and lupus-related cauda equina syndrome. Careful differentiation from infectious or lupus enteritis is essential. Prompt, targeted immunosuppression can reverse even severe neurological involvement and improve outcomes.

ORM5: The Gut - Skin Autoimmune Nexus: A Rare Overlap of Cronkhite - Canada Syndrome and Systemic Sclerosis

Thandrothu Prudvi Raj, Sushama Jotkar, Y. V. S. Srikar
D. Y. Patil Medical College, Kolhapur, Maharashtra

Introduction: Cronkhite–Canada syndrome is a rare, nonhereditary disorder characterized by diffuse gastrointestinal polyposis and ectodermal changes such as alopecia, nail dystrophy, hyperpigmentation. It presents with diarrhea, malnutrition, electrolyte imbalance, increased gastrointestinal cancer risk and has been reported in association with autoimmune conditions including systemic sclerosis.

Case Report: A 42-year-old multiparous woman presented with six months of chronic non-bloody diarrhea, 15-kg unintentional weight loss, fatigue, weakness, and postural giddiness. She developed progressive ectodermal changes, including near-total alopecia, diffuse hyperpigmentation, and dystrophy of fingernails and toenails, with no relevant family history or substance use. She later presented with acute abdominal pain and vomiting; imaging revealed ileocecal intussusception. Exploratory laparotomy was performed, and right hemicolectomy revealed multiple intestinal polyps. Upper gastrointestinal endoscopy demonstrated gastroduodenal polyposis with esophageal sparing. Autoimmune evaluation showed weakly positive ANA and positive CENP-A/B antibodies, and skin biopsy demonstrated dermal collagen thickening consistent with systemic sclerosis. The patient improved with nutritional rehabilitation, electrolyte correction and corticosteroid therapy, establishing a diagnosis of Cronkhite–Canada syndrome associated with systemic sclerosis.

Conclusion: This case emphasizes considering Cronkhite–Canada syndrome in patients with chronic diarrhea and ectodermal changes. Recognizing its multisystem involvement, including rare associations with autoimmune disorders like systemic sclerosis, enables early diagnosis, multidisciplinary care, and timely intervention, potentially improving outcomes in this rare but serious condition.

Figure 1:

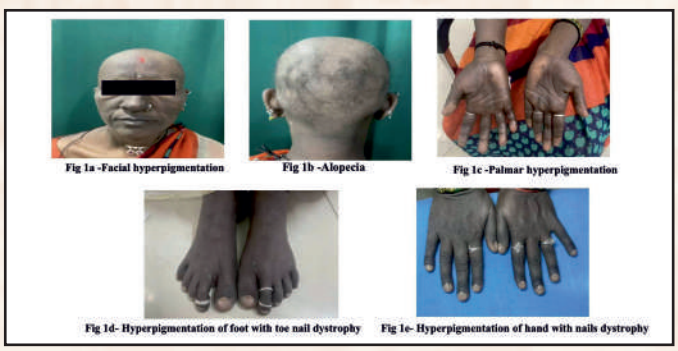
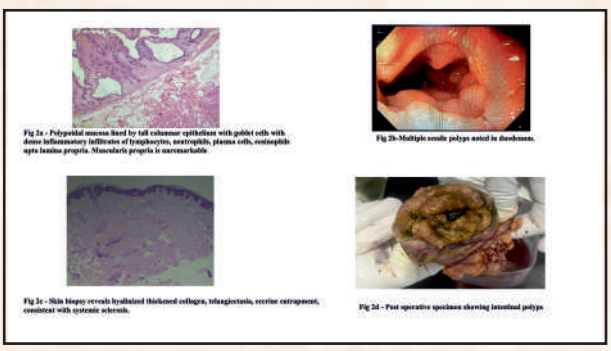


Figure 2:



POSTER PRESENTATIONS BY GENERAL MEDICINE TRAINEES

POM01: The Pancreatic Masquerade: Deciphering Two Distinct Presentations of SLE-Associated Pancreatitis

Aayush Mishra, Krishna Murari Pathak, Sanjay Dubey, V P Pandey, A D Bhatnagar
Department of Medicine, MGM Medical College, Indore

Introduction: Systemic lupus erythematosus (SLE) is a multisystem autoimmune disorder where pancreatitis is a rare but severe complication, affecting 0.7%–4% of patients globally. In India, incidence of SLE associated pancreatitis is around 1.5% among SLE patients, primarily females aged 20–40. Though infrequent, its high morbidity and mortality necessitates early recognition during systemic flares.

Case Report: This case series highlights two distinct cases of acute pancreatitis in the context of SLE. 1) The first case involves a 25-year-old female who presented with acute abdominal pain and was diagnosed with acute pancreatitis. She had no prior history of autoimmune disease. Investigations ruled out common causes of pancreatitis such as infection, metabolic disturbances, drug reactions, endocrine, or vascular conditions. Further immunological work-up revealed high titers of anti-nuclear antibodies (ANA) and anti-dsDNA, leading to a diagnosis of SLE. Her clinical course was complicated by the subsequent development of lupus nephritis stage 3 on follow-up. 2) The second case pertains to a 35-year-old female with a known history of SLE, primarily manifesting with cutaneous involvement. She presented with acute pancreatitis as a flare of her underlying disease. Comprehensive evaluation excluded other etiologies, including infectious, metabolic, drug-related, or vascular causes. Immunological markers confirmed the exacerbation of SLE. Aggressive management with corticosteroids and immunosuppressive therapy led to clinical improvement.

Conclusion: Both cases highlight the diagnostic challenges of SLE-associated pancreatitis. High clinical suspicion is vital for unexplained pancreatitis in young females in absence of common triggers. Timely recognition and aggressive management are crucial to improve outcomes and monitor for further complications, such as lupus nephritis, through long-term follow-up.

Fig 1:

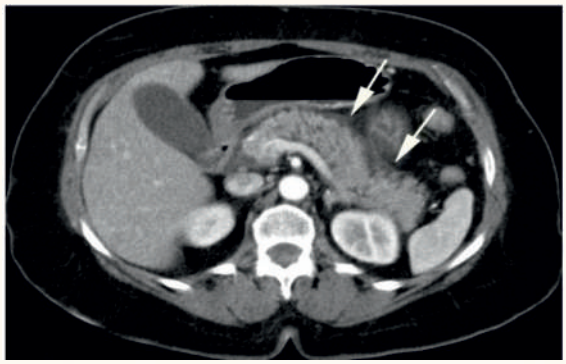
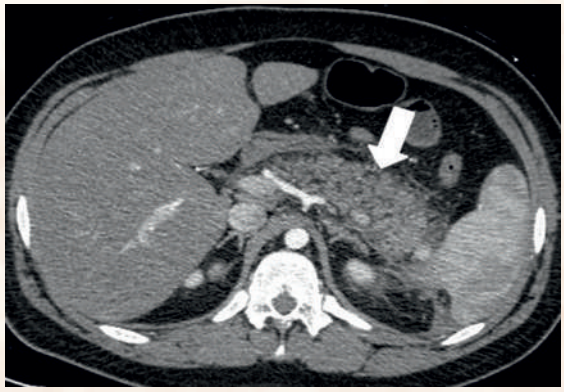


Fig 2:



POM02: Beyond Myopathy: Dermatomyositis

as a Paraneoplastic Clue to Neuroendocrine Lung Malignancy

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Case Report: A 42-year female with no comorbidities presented with complaints of severe muscle ache, weakness of bilateral lower limbs, difficulty in swallowing, nasal regurgitation along with rashes all over the body for 5-6 days and fever for 4 days. Clinical examination revealed poor swallow, dusky red rash over the upper eyelids, papules over the PIP joints. Investigations showed normal CBC, electrolytes, NCV; raised LDH, ferritin, ESR. CPK was markedly raised. ANA, ANCA, Anti-CCP were non-reactive. PET-CT revealed mildly avid subpleural nodule in the posterior segment of right lower lung (11*7mm); large FDG avid poorly enhancing subcarinal nodal mass, ill-defined areas of central necrosis (38*26mm) indenting right pulmonary artery; suggesting primary lung malignancy. Patchy areas of increased FDG uptake in skeletal muscles and areas of subcutaneous fat stranding throughout the body were noted. EBUS TBNA from station 7 mediastinal LN showed metastatic small cell neuroendocrine carcinoma. IHC was synaptophysin and chromogranin positive. Diagnosis of paraneoplastic dermatomyositis was confirmed in this case due to symmetrical proximal muscle weakness, dermatological findings, elevated CPK and supporting PET-CT finding. She was administered pulse IV Methylprednisolone alongside broad spectrum antibiotic coverage followed by Dexamethasone, Etoposide and Carboplatin. She is currently being followed up in OPD, receiving chemotherapy and has received significant improvement of the muscle pain and weakness.

Conclusion: Any case of new onset disease with phenotypic resemblance to dermatomyositis but apparently too aggressive or atypical course should raise suspicion for some primary underlying etiology.

Fig.1 Cutaneous features in the patient

POM03: Severe Non-Compressive Peripheral Neuropathy in Primary Sjogren's Syndrome: A Case of Diagnostic Dilemma

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Introduction: Sjogren's syndrome causes neuropathy in 15% of cases, predominantly manifesting as axonal polyneuropathy or ganglionopathy. Primary demyelinating pathology is rare (<5%). We present a unique case of clinico-pathological dissociation where biopsy-proven demyelination contradicted electrophysiologically-proven axonal neuropathy, offering a critical learning opportunity regarding pseudo-demyelination

Case Report: A patient with ANA-positive SS presented with severe, non-compressive peripheral neuropathy. Nerve Conduction Studies (NCS) confirmed a length-dependent axonal sensorimotor neuropathy. However, sural nerve biopsy uniquely revealed demyelinating disease without vasculitis. This discordance is highly atypical for primary SS. We identified this as "secondary segmental demyelination" resulting from severe axonal loss, rather than a coincidental CIDP overlap. The patient was managed with Rituximab and DMARDs. This case teaches that histologic demyelination in SS is not always diagnostic of a primary myelinopathy. Differentiating secondary changes from true demyelination is crucial, as it supports treating the underlying vasculitic or axonal driver rather than switching unnecessarily to pure demyelinating protocols.

Conclusion: This case highlights that "demyelinating" biopsy findings in Sjogren's can be misleading artifacts of severe axonopathy. We learned that electrophysiology remains the gold standard for phenotyping. Recognizing this "pseudo-demyelination" prevents misdiagnosis and validates the continued use of B-cell depletion (Rituximab) over primary demyelinating therapies.

POM04: Macrophage Activation Syndrome Triggered by Dengue Fever in Newly Diagnosed Mixed Connective Tissue Disease: A Rare Case Report

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Introduction: Macrophage Activation Syndrome (MAS) is a life-threatening hyperinflammatory condition. Although it is common in systemic juvenile idiopathic arthritis and systemic lupus erythematosus, but rare in Mixed Connective Tissue Disease (MCTD). Infection is a well-recognized trigger.

Case Report: A 23-year-old married female presented with polyarthralgia with deformities for 1.5 years, Raynaud's phenomenon, distal sclerodactyly, microstomia, fever (103°F), generalized weakness, and petechial rash. Examination revealed severe pallor and moderate hepatosplenomegaly. Hemogram showed pancytopenia without abnormal cells. Laboratory evaluation demonstrated elevated transaminases (ALT 135 U/L, AST 120 U/L), hyperferritinemia (1450 ng/mL), hypertriglyceridemia (312 mg/dL), hypofibrinogenemia (153 mg/dL), and raised CRP with normal ESR. Dengue NS1 antigen and IgM were positive. Bone marrow examination confirmed hemophagocytosis. She fulfilled 6/8 HLH/MAS diagnostic criteria. Autoimmune workup revealed ANA 4+ (speckled pattern) with high titers of anti-U1 RNP (100.09; reference <20), anti-dsDNA, and anti-nucleosome antibodies. Clinical features along with serology satisfied Alarcón-Segovia and Kahn criteria for MCTD. A final diagnosis of MAS triggered by dengue infection in newly diagnosed MCTD was made. The patient was treated with intravenous corticosteroids, broad-spectrum antibiotics, and supportive care, showing significant clinical improvement.

Conclusion: MAS in adult MCTD is a rare but fatal complication. Infection can trigger early recognition, prompt evaluation for HLH, and aggressive immunosuppressive therapy are critical to improving outcomes. A high index of suspicion is essential in patients with autoimmune disease presenting with fever and cytopenias.

POM05: When Purpura Opens the Gateway: A Vascular Journey Through Bone and Heart in ANCA-negative Small and Medium Vessel Vasculitis.

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Introduction: Polyarteritis nodosa is commonly known to have systemic involvement in the form of renal, mesenteric and testicular involvement. It involves the cardiac and musculoskeletal systems less commonly. Immune triggers by infections apart from hepatitis B and hepatitis C can lead to relapse of cutaneous PAN.

Case Report: A 42 year-old male presented with constitutional symptoms like malaise, intermittent fever followed by palpable purpura surrounding painful multiple, erythematous nodules with necrotic centre, round to oval in shape over bilateral lower legs, majorly on anterior aspect than posterior, predominantly over pretibial region and around ankles associated with erythema, swelling and pain and with a blood pressure of 150/100mmHg. He had a past history of myocardial infarction 10 years ago at the age of 32 and was medically managed. Three years later, he had developed similar lesions on bilateral lower limb and was treated with oral steroids and remission was achieved in 15 days. A year later, he had pain in bilateral hips, MRI was suggestive of bilateral femoral head AVN with effusion.He had COVID-19 infection and ARDS and needed ventilator support for 8 days after which he had relapse of multiple erythematous nodular ulcerations and purpua in bilateral lower limbs. Biopsy was performed which was suggestive of bullous leucocytoclastic vasculitis. 2d echo was suggestive of IHD, LVEF 45%, hypokinetic anterolateral wall. ESR and CRP levels elevated. No evidence of Autoimmune disease, renal involvement, pulmonary involvement, central nervous system involvement and ANCA status was negative.Hepatitis B and hepatitis C status was negative.

Conclusion: This case illustrates relapsing immune-complex mediated small-vessel vasculitis with medium vessel multisystem involvement involving cardiovascular and musculoskeletal system.Myocardial infarction and hypertension in young should raise a suspicion of systemic vasculitis. COVID-19–related immune dysregulation likely acted as a trigger for disease relapse. Avascular necrosis appears to result from combined effects of vasculitic ischemia and prior corticosteroid exposure. Early recognition of systemic involvement is essential for optimal management and prevention of long-term complications of vasculitis.

Fig 1:

POM06: A rare case of co-occurrence of ANCA-associated vasculitis (AAV) and Inflammatory bowel disease (IBD)

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Introduction: Immune-mediated inflammatory disorders may occasionally coexist, complicating diagnostic evaluation. While ANCA positivity in inflammatory bowel disease is often atypical,

PR3-ANCA is strongly associated with systemic vasculitis. The occurrence of PR3-ANCA–associated vasculitis in Crohn's disease is uncommon.

Case Report: A 55-year-old male with systemic hypertension presented with low-grade fever, left-sided colicky abdominal pain, inflammatory arthritis, bilateral ocular redness, and recurrent oral ulcers. Examination revealed scleritis and abdominal tenderness. Initial investigations showed elevated ESR and CRP, and urinalysis revealed active sediment with 3+ hematuria. Abdominal CT imaging showed bilateral renal cortical hypoenhancement and short-segment luminal narrowing in the rectosigmoid junction. Colonoscopy revealed aphthous pancolitis with ulcerative terminal ileitis; on biopsy, active ileitis with cryptitis and crypt abscess. Serology revealed strongly positive Anti-PR3 ANCA and IgA-ASCA, with other autoantibodies negative and normal complements. A possibility of Overlap syndrome with ANCA-associated vasculitis and IBD was considered. He was started on IV Methylprednisolone pulse therapy. Following this, he was treated with Rituximab as induction therapy, and the patient showed rapid improvement in ocular, articular and abdominal symptoms. Later, considering the inflammatory bowel disease component, methotrexate was also added along with steroids and rituximab.

Conclusion: This case highlights a rare overlap of PR3-ANCA–associated vasculitis with inflammatory bowel disease. Early recognition and prompt immunosuppression can result in rapid clinical improvement and favourable outcomes.

POM07: Antibody–Defined Clinical Spectrum and Therapeutic Patterns in Dermatomyositis: A 12-Patient Case Series

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Introduction: Dermatomyositis is an inflammatory myopathy characterized by cutaneous features and variable systemic involvement. We present a case series highlighting distinct antibody–phenotype correlations and differences in therapeutic escalation. Our aim was to describe the clinical spectrum and management patterns in our cohort.

Case Report: We reviewed 12 patients (age 17–83 years) with female predominance (F:M = 10:2) and a mean age of 43.7 years. Four were anti-MDA5 positive; two had interstitial lung disease, including one juvenile dermatomyositis case. Anti-MDA5–associated ILD required mycophenolate with escalation to rituximab in one case. One anti-SAE/Ro52–positive patient had refractory steroid-dependent cutaneous disease. Four patients had active myositis, three were clinically amyopathic, and five had mild muscle involvement. Extensive calcinosis was observed in two patients with anti–TIF1-γ and anti-Mi2 positivity. Anti–PM-Scl positivity defined one overlap in inflammatory myositis phenotype. Malignancy was detected in two cases. Five patients required advanced immunomodulation, including rituximab (n=3), tacrolimus (n=1), and JAK inhibitor therapy (n=1).

Conclusion: This case series highlights the broad clinical and immunological spectrum of dermatomyositis, ranging from amyopathic and cutaneous-predominant disease to interstitial lung disease and malignancy-associated presentations.

Antibody profiling aided risk assessment and influenced monitoring and therapeutic escalation.

POM08: Lupus Nephritis with Concurrent IgA Nephropathy: Importance of Renal Biopsy in Overlap Glomerulopathy

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Introduction: Systemic lupus erythematosus (SLE) frequently involves the kidney, most often as immune complex–mediated lupus nephritis (LN). Non-lupus glomerulopathies such as IgA nephropathy (IgAN) can rarely coexist with SLE and modify prognosis and treatment decisions; their renal biopsy plays a very important role in such cases.

Case Report: A 28-year-old woman with 2 months of fever, inflammatory polyarthralgia,suggesting an evolving connective tissue disorder. Investigations-normocytic normochromic anemia (Hb 9.4 g/dL), serum creatinine 1.9–2.2 mg/dL, active urinary sediment with proteinuria and granular casts, and hypocomplementemia (low C3, normal C4). Serology revealed ANA positivity, high anti-dsDNA, SSA/Ro and Ro-52 antibodies, positive antiphospholipid antibodies,raised inflammatory markers,fulfilling EULAR/ACR criteria for SLE with renal involvement. Renal biopsy demonstrated diffuse proliferative (class IV) lupus nephritis with concurrent mesangial IgA-dominant deposits consistent with IgA nephropathy, confirming overlap glomerulopathy. She was treated with corticosteroids, mycophenolate mofetil, hydroxychloroquine, achieving stabilization of renal function and proteinuria. Biopsy was crucial, as low C3 and active SLE serology suggested classic LN, but dominant IgA deposits indicated coexistent IgAN; this overlap may represent dual pathology or an IgA-predominant LN variant, often relatively indolent but with possible progression to ESRD, with complement consumption reflecting lupus nephritis than isolated IgAN.

Conclusion: In SLE with renal involvement, renal biopsy is indispensable not only for Lupus Nephritis class assignment but also to exclude coexisting non-lupus nephropathies such as IgA Nephropathy. Identifying Lupus Nephritis–IgA Nephropathy overlap helps refine prognosis as coexistence of IgA nephropathy may have a more indolent course.

POM09: Beyond Morphea: Progressive Hemibody Atrophy in Deep Linear Scleroderma (Parry-Romberg Spectrum)

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Introduction: Deep linear scleroderma is a rare localized autoimmune disorder of childhood that may extend beyond skin to involve subcutaneous tissue, muscle, bone, and nerves. Progressive hemibody atrophy represents the severe end of the Parry–Romberg spectrum, reported in <1–2% of cases.

Case Report: A 15-year-old girl with progressive thinning of one limb and persistent skin lesions was assessed through clinical, neurological, radiological, and histopathological evaluations. Tests included autoimmune serology, muscle enzymes, imaging, nerve conduction studies, and

a deep skin-muscle biopsy to confirm the diagnosis and rule out systemic sclerosis, neuromuscular, and neurocutaneous disorders. The patient presented with unilateral hyperpigmented, thickened skin lesions on the left side of her face, trunk, and limb, along with hemifacial and limb atrophy, contractures, limb shortening, calcinosis cutis, and trouble walking. A neurological exam showed sensory loss and axonal peroneal neuropathy. Autoimmune markers, muscle enzymes, and imaging results were normal. Histopathology revealed thick collagen in the dermis with perivascular lymphocytic infiltration, confirming deep linear scleroderma. The presence of hemifacial atrophy supported the diagnosis of linear scleroderma with Parry-Romberg spectrum involvement.

Conclusion: This case showcases a rare, severe presentation of the Parry-Romberg spectrum related to deep linear scleroderma. Early detection and immunosuppressive treatment are crucial to stop progression and prevent permanent deformity and disability in affected children.

Fig.1

POM10: Primary Sjögren's disease presenting without sicca symptoms with marked splenomegaly and pancytopenia

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Introduction: Primary Sjögren's disease is an autoimmune disorder classically presenting with sicca symptoms. However, extra-glandular involvement may predominate and delay diagnosis. We report an atypical presentation of primary Sjögren's disease without sicca symptoms presenting with marked splenomegaly and pancytopenia.

Case Report: A 20-year-old woman presented with four months of symmetrical polyarthralgia involving large joints, low-grade fever, fatigue, and generalized weakness. There was no history of dry eyes, dry mouth, photosensitivity, oral ulcers, Raynaud's phenomenon, or alopecia. Examination revealed pallor and marked splenomegaly without lymphadenopathy. Investigations showed pancytopenia, deranged liver enzymes, hypergammaglobulinemia, and positive antinuclear antibody (1:80, speckled pattern). Anti-Ro (SSA) and Anti-La (SSB) antibodies were strongly positive, while rheumatoid factor, anti-CCP, anti-dsDNA, anti-Sm antibodies, and viral markers were negative. Imaging demonstrated splenomegaly with normal liver echotexture and no portal hypertension. Schirmer's test and tear film break-up time were normal. Lip biopsy revealed focal lymphoplasmacytic infiltrates with a focus score greater than one, confirming primary Sjögren's disease.

Conclusion: Primary Sjögren's disease may

rarely present without sicca symptoms and manifest predominantly with extra-glandular involvement. Clinicians should consider this diagnosis in patients with unexplained cytopenias and splenomegaly. Lip biopsy plays a pivotal role in establishing diagnosis when classical clinical features are absent.

POM11: Enigmatic Arthritis in an Elderly Male: A Diagnostic Journey

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Introduction: Adult-onset Still's disease (AOSD) is a rare systemic autoinflammatory disorder that may mimic severe infection. Elderly-onset presentations are uncommon and often atypical, leading to diagnostic delay. Recognition is crucial when culture-negative systemic inflammation and hyperferritinemia coexist.

Case Report: A 75-year-old previously healthy male presented with 2 weeks of high-grade polyarthritis, fever, and rapid functional decline. Initial evaluation suggested seronegative inflammatory arthritis; however, purulent-appearing synovial fluid led to treatment for presumed septic arthritis. Despite broad-spectrum antibiotics and synovectomy, the patient developed multisystem involvement including high-degree atrioventricular block, pleuro-pericardial effusion, hepatic dysfunction, and proteinuria. Extensive infectious, autoimmune, and malignant workup was negative. Laboratory evaluation revealed marked neutrophilic leukocytosis, anemia, thrombocytosis, and hyperferritinemia. Pulse corticosteroids resulted in dramatic clinical improvement, but relapse occurred during tapering, requiring intravenous immunoglobulin therapy with sustained response. Renal biopsy showed no immune complex-mediated glomerulonephritis, supporting systemic inflammatory etiology. Based on clinical, laboratory, and therapeutic response, elderly-onset AOSD was diagnosed
.

Conclusion: Elderly-onset AOSD can closely mimic severe sepsis. Hyperferritinemia, sterile inflammatory synovial fluid, and steroid responsiveness are key diagnostic clues. Early recognition enables timely immunosuppression and prevents unnecessary prolonged antimicrobial therapy

POM12: Idiopathic Inflammatory Myositis Mimicking Guillain–Barré Syndrome: A Diagnostic Pitfall

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Introduction: Idiopathic Inflammatory Myositis sometimes can closely resemble GBS, particularly in cases presenting with acute or subacute weakness and areflexia. A thorough clinical evaluation, supported by electrodiagnostic tests, serum biomarkers, MRI, and muscle biopsy, is essential for an accurate diagnosis. Timely differentiation is critical to ensure appropriate management.

Case Report: A 30-year-old male presented with 15 days of insidious, progressive ascending quadriparesis, rendering him bedridden. Examination revealed flaccid paralysis (power 0/5 in all limbs), hypotonia, areflexia with preserved ankle jerks, and mute plantar responses. A preceding episode of dysentery

raised suspicion of Guillain–Barré syndrome. Nerve conduction studies showed severe symmetrical pure motor axonal neuropathy, supporting the initial diagnosis, and IVIG was planned. However, routine investigations revealed progressively rising transaminases with AST predominance. Given AST abundance in skeletal muscle and preserved ankle reflexes, a myopathic process was suspected. Serum CPK was markedly elevated (35,391 U/L) with high urine myoglobin levels. Myositis panel revealed Mi-2B and Ro-52 antibody positivity. MRI whole-body STIR imaging demonstrated diffuse symmetrical muscle hyperintensities consistent with inflammatory myositis. The diagnosis was revised to idiopathic inflammatory myositis. The patient developed respiratory muscle weakness requiring ventilatory support. Following IVIG and steroids, muscle power improved and he was successfully weaned off ventilation.

Conclusion: Idiopathic inflammatory myositis can rarely masquerade as Guillain–Barré syndrome, particularly in acute presentations with areflexic weakness. Disproportionate AST elevation, markedly raised CPK, preserved reflexes, and supportive MRI and antibody profiles are key diagnostic clues. Early recognition prevents misdiagnosis and ensures appropriate, life-saving therapy.

POM13: Macrophage Activation Syndrome as a Life-Threatening Complication of Adult-Onset Still's Disease: A Case Report

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Introduction: Adult-Onset Still's Disease is a rare systemic autoinflammatory disorder diagnosed largely by exclusion. Although frequently responsive to corticosteroids, it can be complicated by macrophage activation syndrome, a severe hyperinflammatory syndrome. Early clinical suspicion, timely diagnosis, and prompt immunosuppressive therapy are essential to reduce morbidity and mortality.

Case Report: A 20-year-old woman with no comorbidities presented with intermittent high-grade fever for 1–2 years, associated with polyarthritis, morning stiffness, and an evanescent salmon-colored rash coinciding with febrile episodes. Examination revealed pallor, generalized tender lymphadenopathy, and splenomegaly. Investigations showed severe anemia (Hb 4.6 g/dL), neutrophilic leukocytosis, markedly elevated ferritin (>10,000 ng/mL), high CRP and ESR, hypertriglyceridemia, and low fibrinogen. Extensive infectious, malignant, and autoimmune workup was negative apart from ANA positivity by IFA. Imaging demonstrated generalized lymphadenopathy and splenomegaly, with initial lymph node biopsies showing reactive changes. Based on fulfillment of Yamaguchi criteria, Adult-Onset Still's Disease was diagnosed, with partial steroid response. Steroid taper precipitated relapse, and repeat lymph node biopsy revealed hemophagocytosis, confirming Secondary HLH/ MAS (HScore 270; >99% probability). Treatment with high-dose dexamethasone, hydroxychloroquine, and methotrexate led to clinical and biochemical improvement.

Conclusion: Macrophage activation syndrome (MAS) should be strongly suspected in patients with AOSD presenting with persistent or worsening systemic symptoms, extreme hyperferritinemia, cytopenias, and multi-organ

involvement. Early recognition and timely initiation of immunosuppressive therapy, including corticosteroids and DMARDs, are essential to improve outcomes and prevent life threatening complications.

POM14: Digital Gangrene Without Recognised Raynaud's: Unmasking Early Systemic Sclerosis

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Introduction: Digital gangrene in young patients without atherosclerotic risk factors should raise suspicion of autoimmune microvascular disease. Limited cutaneous systemic sclerosis typically presents with Raynaud's phenomenon and rarely with critical ischemia at onset. This atypical presentation of multifocal digital gangrene without Raynaud's, emphasizes the need for early recognition and treatment.

Case Report: A 39-year-old female presented with dry gangrene of the left middle finger with autoamputation of proximal interphalangeal joint, gangrene of the right index finger, and left great toe. There was no history of skin tightening, rashes, difficulty in swallowing or prior history suggestive of Raynaud's phenomenon. No history of diabetes, hypertension, smoking, or known connective tissue disease. Systemic examination was unremarkable. Mouth opening was normal. Relevant investigations included ESR 80 mm/hr, CRP 104 mg/L, and strong CENP-B positivity. Other antibodies were negative. Lipid profile was normal. HRCT thorax was normal. Doppler studies showed reduced distal arterial flow in the upper limbs and normal triphasic flow in the lower limbs. Nail fold capillaroscopy could not be done. The patient was managed with vasodilators (calcium channel blocker and prostacyclin), antiplatelet agents, and local wound care. Progression of gangrene was arrested following initiation of therapy, with no new ischemic areas developing during follow-up.

Conclusion: Digital gangrene without preceding Raynaud's phenomenon may represent an early and rare manifestation of systemic sclerosis. Screening for systemic sclerosis–specific antibodies should be considered in all cases of unexplained digital ischemia. Early diagnosis and prompt initiation of vasodilator therapy is essential to prevent further vascular compromise and tissue loss.

Fig.1

POM15: Silent Storms: A Case Report on the Catastrophic Nature of Antiphospholipid Syndrome

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Introduction: Catastrophic antiphospholipid syndrome (CAPS) is a rare, life-threatening variant of antiphospholipid syndrome characterized by rapidly progressive widespread thrombosis leading to multiorgan failure. We report a case highlighting its fulminant presentation and diagnostic challenge.

Case Report: A 30-year-old male presented with bilateral lower limb swelling following trivial trauma, acute dyspnea, and sudden onset altered sensorium. On examination, he was hypotensive with tachycardia, raised jugular venous pressure, icterus, and depressed consciousness. Laboratory investigations revealed anemia, thrombocytopenia, leukocytosis, and deranged liver and renal function tests, initially suggestive of sepsis with multiorgan dysfunction. Imaging confirmed bilateral deep vein thrombosis and pulmonary thromboembolism. Although disseminated intravascular coagulation was initially suspected, further evaluation showed high titers of anticardiolipin antibodies and lupus anticoagulant with negative ANA and ANCA, establishing the diagnosis of catastrophic antiphospholipid syndrome. The patient was managed with systemic thrombolysis, therapeutic anticoagulation, broad-spectrum antibiotics, plasma exchange followed by intravenous immunoglobulin, and surgical debridement. He showed marked clinical improvement and was discharged on lifelong anticoagulation. Repeat antiphospholipid antibody testing after three months confirmed persistent positivity.

Conclusion: CAPS should be suspected in patients presenting with acute multiorgan dysfunction, thrombocytopenia, and widespread thrombosis mimicking sepsis or DIC. Early recognition and aggressive multimodal therapy are critical for improving survival.

POM17: When Blue Fingers Tell a Bigger Story: Limited Systemic Sclerosis with Digital Gangrene and Interstitial Lung disease

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Introduction: Limited cutaneous systemic sclerosis is characterized by skin involvement distal to the elbows and knees with facial involvement and is classically associated with Raynaud phenomenon, digital ischemia, pulmonary arterial hypertension, and gastrointestinal involvement. Early pulmonary manifestations may be misinterpreted as infectious pathology in endemic regions.

Case Report: We report a case of a 32-year-old female farmer who presented with progressive hyperpigmentation and tightening of skin over the face and hands for three years, dry cough for one year, and acute onset breathlessness for ten days. She had a history of Raynaud phenomenon for two years, complicated by friction ulcers, and gangrene of distal fingers. She had previously received empirical ATT based on radiological findings, with no clinical improvement. Clinical examination revealed skin involvement limited to the face and areas distal to the elbows, consistent with lcSSc. Nailfold dermoscopy demonstrated microhemorrhages and capillary dropouts (capillary washouts), suggestive of an active scleroderma microangiopathy. Respiratory examination showed reduced chest expansion with bilateral basal crepitations. PFT revealed a restrictive pattern, and HRCT thorax showed bibasal infiltrates, more prominent on the left, suggestive of an NSIP pattern of interstitial lung disease. ECG findings of low voltage QRS complexes with P pulmonale raised suspicion of

pulmonary arterial hypertension. Laboratory evaluation revealed a raised ESR and strong positive ANA speckled pattern and 1:3200 titre.

Conclusion: This case underscores the importance of considering systemic sclerosis in young females presenting with Raynaud phenomenon, skin tightening, and unexplained pulmonary infiltrates. Early recognition can prevent misdiagnosis and irreversible organ damage.

POM18: Autoimmune Convergence in liver - A Case of PBC-AIH Overlap

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Introduction: Primary biliary cirrhosis is a chronic autoimmune cholestatic liver disease which is frequently associated with Sjogren syndrome. Autoimmune hepatitis overlap along with it represents a subset with combined hepatic and cholestatic features posing d and therapeutic challenge. We present a similar case that showed response to combined immunosuppressive and supportive therapy.

Case Report: A middle-aged woman presented with long standing dyspepsia, anorexia and unintentional weight loss. There was no history of alcohol intake, drug use and viral markers were also non-reactive. There was no evidence of chronic liver disease as well. On evaluation liver enzymes were elevated with a mixed pattern: AST 229 U/L, ALT 250 U/L and ALP 516 U/L. Autoimmune serology revealed strongly positive ANA (3+, mixed pattern, titer 1:1000), strong anti-Ro 52 positivity (3+++) and positive anti mitochondrial antibody (AMA-M2). Based on the biochemical and serological profile, a diagnosis of primary biliary cirrhosis-autoimmune hepatitis overlap syndrome was made. The patient was treated with Urso deoxycholate and systemic steroids following which she demonstrated significant biochemical improvement and symptomatic relief.

Conclusion: Strong ANA positivity with marked transaminitis suggests an autoimmune hepatitis component while AMA-M2 positivity point towards an autoimmune cholestatic disease and Ro52 suggests a suspicion of associated Sjogren syndrome thus pointing towards an AIH-PBC overlap.

POM19: Severe Sensorimotor Polyneuropathy as an Early and Severe Manifestation of Active Systemic Lupus Erythematosus with Class V Lupus Nephritis: A Rare Neuro-Renal Presentation

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Introduction: Peripheral nervous system involvement in systemic lupus erythematosus (SLE) is uncommon and often under-recognized. While distal symmetric neuropathy is most frequently reported, acute severe sensorimotor polyneuropathy presenting early in the disease course is rare. Such neurological involvement may reflect high systemic disease activity and can coexist with immune-mediated renal involvement.

Case Report: A young woman presented with rapidly progressive, symmetric quadriparesis

positive, with borderline anticardiolipin antibody, supporting secondary Antiphospholipid Syndrome.

Conclusion: This case demonstrates the silent temporal evolution of systemic autoimmunity, where early antiphospholipid-related obstetric morbidity and Raynaud's phenomenon preceded the development of a systemic sclerosis–predominant Mixed Connective Tissue Disease phenotype. It emphasizes need for early recognition of subtle autoimmune signals to enable timely intervention and prevent long-term organ damage

POM21: Anti-CCP-Positive Psoriatic Arthritis -Arthritis Mutilans with Nail Psoriasis Coexisting with Secondary Sjogren's Disease - A Very Rare Overlap

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Introduction: Psoriatic arthritis exhibits heterogeneous clinical phenotypes, with arthritis mutilans representing its most destructive form. Anti-CCP positivity and coexisting primary Sjögren's syndrome are uncommon in PsA, posing diagnostic challenges and important implications for disease classification and management

Case Report: A 44-year-old female presented in a bedridden state with severe pain, swelling, and progressive deformities involving bilateral knees, wrists, and small joints of both upper and lower limbs. Examination revealed symmetric polyarthritis with marked deformities of hands and feet. There was a prior history of psoriasis, and nail examination demonstrated nail pitting and dystrophy. Radiographs showed severe osteolysis and deformities consistent with arthritis mutilans. Rheumatoid factor and anti-CCP antibodies were positive. She also reported xerostomia, prompting further evaluation. Schirmer's tests I and II were positive. ANA by indirect immunofluorescence was 2+, and ANA profile revealed high titers of anti-SSA/Ro52, anti-SSA/Ro60, and anti-SSB antibodies. Based on clinical, radiological, and serological findings, a diagnosis of anti-CCP–positive psoriatic arthritis with arthritis mutilans and coexisting primary Sjögren's syndrome was established.

Conclusion: This case underscores extreme autoimmune heterogeneity, demonstrating the coexistence of anti-CCP–positive psoriatic arthritis with arthritis mutilans and primary Sjögren's syndrome, reflecting pathogenic overlap between distinct HLA-driven immune pathways and challenging traditional boundaries between inflammatory arthritides and connective tissue diseases.

POM22: Anti-Synthetase Syndrome: Unmasking an Elusive Culprit in a Fever of Unknown Origin.

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Introduction: Autoimmune diseases in male patients are a rare entity, even more so when autoimmune inflammatory myopathies are suspected. Fever of unknown origin with systemic inflammatory features can further obscure diagnoses, especially when the presentation is highly atypical.

predominantly involving the lower limbs, accompanied by severe burning neuropathic pain and autonomic dysfunction manifesting as bowel and bladder involvement. Neurological examination revealed marked distal weakness with initially preserved reflexes. Nerve conduction studies demonstrated severe, symmetric sensorimotor polyneuropathy with mixed axonal and demyelinating features, predominantly affecting the lower limbs, with low-amplitude responses and absent motor and sensory potentials. Immunological evaluation revealed strongly positive antinuclear antibodies with high-titer anti–double-stranded DNA antibodies and hypocomplementemia. Concurrent renal involvement was evident, with nephrotic-range proteinuria and active urinary sediment. Renal biopsy confirmed Class V lupus nephritis. High disease activity was supported by elevated SLEDAI-2K scores and inflammatory markers. Infectious, metabolic, nutritional, toxic, and drug-induced causes of acute polyneuropathy were systematically excluded. The temporal association of severe neuropathy with active lupus nephritis suggested a shared immune-mediated pathophysiology involving small-vessel vasculitis and immune complex deposition affecting both peripheral nerves and renal glomeruli.

Conclusion: Severe sensorimotor polyneuropathy can rarely be an early and aggressive manifestation of active SLE and may occur concurrently with lupus nephritis as part of a neuro-renal immune-mediated disease spectrum. Recognition of this association is crucial, as prompt diagnosis and early initiation of immunosuppressive therapy may significantly improve neurological recovery and renal outcomes.

POM20: MCTD WITH SECONDARY APS: A DECADE OF SILENT AUTOIMMUNE EVOLUTION - A CASE REPORT

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Introduction: Mixed Connective Tissue Disease (MCTD) is a systemic autoimmune disorder characterized by overlapping clinical features and anti-U1 RNP positivity. Its onset is often insidious, with early manifestations remaining unrecognized for years. We present a case demonstrating the silent temporal evolution of autoimmunity, culminating in MCTD with secondary Antiphospholipid Syndrome.

Case Report: A 40-year-old woman presented with a two-month history of headache, giddiness, and recurrent sudden falls without loss of consciousness, seizures, or focal neurological deficits. Neurological examination was normal except for orthostatic hypotension, and MRI brain with MR venography revealed no intracranial pathology. A detailed history revealed a decade-long evolution of systemic symptoms, beginning with chronic fatigue and generalized weakness. Her obstetric history (P5L2A3) included three consecutive intrauterine fetal losses beyond 10 weeks, suggesting an antiphospholipid-mediated process. Over subsequent years, she developed progressive dysphagia, suggestive of esophageal dysmotility, and long-standing Raynaud's phenomenon. Examination revealed puffy hands, early sclerodactyly, and digital pitting scars, consistent with a systemic sclerosis–predominant Mixed Connective Tissue Disease phenotype. Autoimmune evaluation showed strongly positive ANA with a speckled pattern and high-titer anti-U1 RNP and anti-Sm/RNP antibodies, confirming MCTD. Lupus anticoagulant was

Case Report: A 27-year-old male, chronic alcoholic, smoker, presented with complaints of intermittent moderate-grade (101 degree Fahrenheit) fever for 1.5 months, severe weakness, night sweats, weight loss, and transient non-pruritic maculopapular rash with long-standing mild inflammatory small-joint pain in MCP and PIP joints. Examination revealed hepatosplenomegaly, proximal > distal muscle weakness, cervical and right axillary lymphadenopathy. Labs showed pancytopenia, hyperferritinemia (3494 ng/mL), transaminitis, low CRP, raised LDH and triglyceride levels, microalbuminuria and autoimmune hemolytic anemia. Infective and malignant causes including tuberculosis, subacute bacterial endocarditis, HIV, STIs and lymphoma were ruled out. Bone marrow aspiration showed no evidence of Macrophage Activating Syndrome. Autoimmune work-up revealed ANA positivity (3+ fine speckled, cytoplasmic) with low complement levels, elevated CPK, and a strongly positive myositis panel (Mi-2, Ku, Jo-1, SRP, PL-7, PL-12, OJ). HRCT thorax showed GGOs without an established ILD. A diagnosis of Anti-synthetase syndrome was made. Treatment with pulse dose corticosteroids and hydroxychloroquine caused remission.

Conclusion: This case highlights that autoimmune diseases like anti-synthetase syndrome are an under-recognized cause of prolonged fever even in males and can present with only systemic inflammation and cytopenia. Early consideration of autoimmune etiologies in PUO and timely immunosuppression can be lifesaving and may prevent the development of interstitial lung disease.

POM23: A Fatal Neurological Complication in Male Lupus Nephritis

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Introduction: Posterior reversible encephalopathy syndrome is an uncommon but life-threatening neurological complication in lupus nephritis, usually triggered by severe hypertension, renal dysfunction, and active disease. Early recognition is critical, as delayed diagnosis may lead to irreversible neurological injury and mortality.

Case Report: A 49-year-old man with lupus nephritis presented with generalized edema, nephrotic-range proteinuria, severe hypertension, and acute kidney injury with active serology. Despite immunosuppressive therapy and blood pressure control, he developed sudden seizures and altered sensorium. Magnetic resonance imaging revealed bilateral parieto-occipital hyperintensities consistent with posterior reversible encephalopathy syndrome. His neurological status progressively worsened with recurrent seizures and refractory encephalopathy. He subsequently developed multiorgan dysfunction including worsening renal failure and hemodynamic instability requiring intensive care. Despite aggressive supportive management, he deteriorated and succumbed to complications. The clinical course suggested severe lupus activity with hypertensive crisis precipitating posterior reversible encephalopathy syndrome leading to fatal outcome.

Conclusion: Posterior reversible encephalopathy syndrome should be suspected in lupus nephritis patients with seizures or altered consciousness. Prompt blood pressure control and early neuroimaging are essential. Delay may convert a

potentially reversible condition into a fatal neurological complication.

Fig.1

POM24: Cerebral Venous Thrombosis as the Presenting Manifestation of Undifferentiated Connective Tissue Disease (UCTD) / early evolving SLE in a Young Female: A Case Report

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Introduction: Cerebral venous thrombosis (CVT) is presence of blood clot in dural venous sinuses, cerebral veins or both. It's in 0.5% patients presenting as stroke rare but potentially life-threatening neurological emergency. CVT as an initial manifestation of UCTD / SLE is rare with only 0.3 – 0.5% incidence in SLE. Early recognition of autoimmune etiology is important in CVT as it's of prognostic importance.

Case Report: A 24-year-old female presented with sudden-onset right upper limb numbness, focal seizures with secondary generalisation and weakness of right upper limb, associated with headache for 4 days. Neurological examination revealed right upper limb power of 2/5 with otherwise normal neurological findings. MRI Brain demonstrated a large venous hemorrhagic infarct in the left superior frontal region with thrombosis of the superior sagittal sinus and few superficial cortical veins confirming CVT. Workup revealed strongly positive ANA by IFA (3+) with elevated RNP 68kd/A/C antibody. She had a significant history of thrombocytopenia and anemia a year ago. Other autoimmune, infectious and prothrombotic markers were unremarkable. She was managed with anticoagulation (LMWH for 5 days followed by rivaroxaban), antiepileptics, steroids and hydroxychloroquine. The patient was discharged stable and showed significant neurological improvement on follow up with physiotherapy, immunosuppressants and anticoagulation. Based on clinical presentation and serology, a diagnosis of UCTD / early evolving SLE presenting as CVT was made.

Conclusion: This case highlights that CVT as a neurological emergency may be the initial dominant manifestation of UCTD / SLE. Unprovoked CVT should prompt autoimmune evaluation especially in young females. CVT in SLE may be caused by a variety of pathogenic factors such as vasculitis and APS. Early immunosuppression alongside anticoagulation improves the prognosis of lupus with CVT.

POM25: Primary Sjögren's Disease Presenting as Longitudinally Extensive Transverse Myelitis without Sicca Symptoms

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Introduction: Primary Sjögren's disease is a multisystem autoimmune disorder classically presenting with sicca symptoms. Neurological involvement may rarely precede glandular features. We report an unusual presentation of Sjögren's disease manifesting as longitudinally extensive transverse myelitis without sicca symptoms.

Case Report: A 35-year-old woman presented with acute onset progressive weakness of both lower limbs, sensory loss up to the T4 level, and bladder–bowel dysfunction. Neurological examination revealed paraparesis with absent deep tendon reflexes and bilateral extensor plantar responses. MRI spine with brain and orbits showed longitudinally extensive transverse myelitis from D2–D6 without evidence of optic neuritis. CSF analysis revealed mild lymphocytic pleocytosis with raised protein; infectious workup was negative. Serum NMO-IgG (AQP4) was positive. ANA was positive with high titers of anti-Ro (SSA) and anti-La (SSB) antibodies. Despite absence of sicca symptoms and normal Schirmer's and TBUT (tear film breakup time) tests, lip biopsy demonstrated extensive chronic focal lymphoplasmacytic infiltrate around glands and ducts with focus score >1, confirming primary Sjögren's disease. The patient was treated with intravenous methylprednisolone followed by rituximab, resulting in significant neurological improvement.

Conclusion: Primary Sjögren's disease can present with severe neurological manifestations such as longitudinally extensive transverse myelitis without sicca symptoms. High clinical suspicion and lip biopsy are crucial for diagnosis. Early immunosuppression can lead to favorable outcomes.

POM26: Hypokalemic Quadriparesis as the Initial Presentation of Distal Renal Tubular Acidosis in a Newly Diagnosed Sjogren's Syndrome

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Introduction: Primary Sjögren's syndrome (pSS) is a systemic autoimmune disorder primarily affecting exocrine glands. Severe hypokalemia secondary to dRTA can rarely present as acute flaccid paralysis, often mimicking primary neuromuscular disorders and posing a diagnostic challenge.

Case Report: A 40-year-old woman with no comorbidities presented with insidious, progressive, symmetric flaccid quadriparesis causing difficulty in rising from bed. There was no history of fever, sensory symptoms, cranial nerve involvement, bowel or bladder disturbance, drug intake, or similar past episodes. She reported long-standing xerostomia with dysphagia and dry eyes. Examination revealed hypotonia with power 2/5 in all limbs, diminished deep tendon reflexes, and flexor plantar responses; sensory and cranial nerve examinations were normal. Investigations showed normal anion gap metabolic acidosis (pH 7.2, HCO_3^- 10.2 mmol/L) with severe hypokalemia (<2.6 mEq/L), inappropriately alkaline urine (pH 6.5), and renal potassium wasting, consistent with distal renal tubular acidosis. Nerve conduction studies were normal. Schirmer's test showed severe dryness.

Autoimmune evaluation revealed ANA positivity with anti-SSA and anti-SSB antibodies. Salivary gland ultrasonography and minor salivary gland biopsy confirmed primary Sjögren's syndrome, establishing distal RTA as the cause of hypokalemic paralysis.

Conclusion: Hypokalemic paralysis as a rare but reversible initial manifestation of primary Sjögren's syndrome. In patients with acute flaccid paralysis and normal anion gap metabolic acidosis, autoimmune etiologies should be actively considered, even in the absence of prominent sicca symptoms. Early diagnosis and prompt correction of electrolyte and acid–base disturbances are lifesaving and prevent recurrence.

POM27: Nephrotic-Range Proteinuria as the Initial Manifestation of Primary Sjögren's Syndrome: A Rare Glomerular Presentation

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Introduction: Primary Sjögren's syndrome is a systemic autoimmune disease predominantly affecting exocrine glands, with renal involvement occurring in a minority of patients. Renal disease is usually tubulointerstitial; glomerular involvement causing nephrotic-range proteinuria, especially without sicca symptoms, is rare and diagnostically challenging.

Case Report: A patient presented with generalized edema and nephrotic-range proteinuria without complaints of dry mouth, dry eyes, parotid swelling, arthralgia, or constitutional symptoms. Routine evaluation for common causes of nephrotic syndrome was non-contributory. Autoimmune work-up revealed positive anti-Ro (SSA) antibodies. Schirmer's test was negative. Minor salivary gland (labial lip) biopsy demonstrated focal lymphocytic sialadenitis with a focus score of 2, confirming primary Sjögren's syndrome. Renal biopsy showed glomerular pathology consistent with immune-mediated glomerular involvement rather than tubulointerstitial nephritis. Based on clinicopathological correlation, a diagnosis of primary Sjögren's syndrome presenting with glomerular disease and nephrotic syndrome was established. This case represents an unusual renal manifestation of Sjögren's syndrome occurring in the absence of sicca symptoms.

Conclusion: Primary Sjögren's syndrome should be considered in the differential diagnosis of unexplained nephrotic syndrome, even in the absence of sicca features. Early recognition of this rare glomerular manifestation is crucial, as timely diagnosis and appropriate immunosuppressive treatment may improve renal outcomes.

POM28: Psoriatic Arthropathy Beyond Joints: Proteinuria Signaling Secondary Amyloidosis

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Introduction: Psoriatic arthritis is a chronic inflammatory arthropathy with systemic involvement. Although extra-articular manifestations are recognized, secondary AA amyloidosis involving the kidneys is a rare (<1%) but serious complication. Renal involvement may be subtle and often remains undetected until significant proteinuria develops.

Case Report: A 42 year-old male with long-standing psoriatic arthritis and established joint deformities presented with progressive bilateral pedal edema without any known comorbidities, no addictions, and no prior renal disease. Laboratory evaluation revealed urinary protein +3 on dipstick with nephrotic-range proteinuria (urine ACR 5197mg/g). Renal function was preserved with serum creatinine 1.0 mg/dL and blood urea 35 mg/dL. CBC showed hemoglobin 11.1 g/dL. Liver function tests were within normal limits (AST/ALT 20/36 IU/L). Total serum protein was 6.5 g/dL with hypoalbuminemia (albumin 2.7g/dL). Viral markers and Anti-CCP was also Negative. Ultrasonography of the abdomen demonstrated bilaterally enlarged kidneys, measuring 11 × 6 cm and 11.5 × 6.1 cm, respectively. Cardiac evaluation showed Grade I diastolic dysfunction on echocardiography. Given the chronic inflammatory background and nephrotic-range proteinuria, a renal biopsy was performed, which confirmed AA-type secondary amyloidosis, establishing the diagnosis of renal amyloidosis secondary to psoriatic arthritis.

Conclusion: This case highlights secondary renal amyloidosis as a rare (<1%) but important complication of long-standing psoriatic arthritis, presenting with pedal edema and nephrotic-range proteinuria despite preserved renal function. Routine renal surveillance and early biopsy in PsA patients with unexplained proteinuria may enable timely intervention and improved outcomes

POM29: Immunological Discordance in Secondary Evans Syndrome: Systemic Lupus Erythematosus with Fluctuating Antiphospholipid Antibodies and Refractory Thrombocytopenia

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Introduction: Evans syndrome is a rare autoimmune cytopenic disorder with higher relapse rates and mortality when secondary to systemic lupus erythematosus (SLE). Heterogeneous autoantibody expression, fluctuating antiphospholipid antibodies, and selective cytopenia refractoriness remain poorly understood and pose major therapeutic challenges.

Case Report: A lady presented with menorrhagia, mucocutaneous bleeding, and severe anemia. Laboratory evaluation showed hemoglobin 3 g/dL, platelet count 5,000/cumm, reticulocytosis, strongly positive direct antiglobulin test, high-titer ANA (homogeneous pattern), hypocomplementemia, and lupus anticoagulant positivity. Bone marrow examination demonstrated erythroid hyperplasia. She fulfilled EULAR/ACR criteria for SLE (score 15) with secondary Evans syndrome. Initial treatment with high-dose corticosteroids resulted in hematological remission. Following treatment discontinuation, the patient developed multiple relapses characterized by isolated immune thrombocytopenia, while other lupus parameters remained controlled. Progressive steroid refractoriness necessitated intravenous immunoglobulin therapy. Intermittent antiphospholipid antibody positivity and persistent thrombocytopenia despite global disease improvement raised unresolved diagnostic and therapeutic concerns.

Conclusion: This case highlights the

immunological heterogeneity of secondary Evans syndrome in SLE. Selective persistence of thrombocytopenia, fluctuating antiphospholipid antibodies, and steroid resistance underscore unresolved pathophysiological mechanisms and emphasize the need for individualized, escalation-based treatment strategies.

POM30: Arthritis Mutilans in Psoriatic Arthritis with Autoimmune Serological Overlap: When Immune Dysregulation Becomes Destructive

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Introduction: Psoriatic arthritis affects 20–30% of patients with psoriasis, but arthritis mutilans occurs in less than 5%, leading to severe osteolysis, digital telescoping, and permanent disability. Anti-CCP positivity, seen in only 5–10% of PsA cases, is associated with aggressive erosive disease, while overlap conditions such as secondary Sjögren's syndrome further complicate diagnosis and management.

Case Report: A 36-year-old woman with long-standing psoriasis vulgaris presented with a 2.5-year history of progressive inflammatory polyarthritis involving both small and large joints, accompanied by prolonged morning stiffness and severe functional impairment. On examination, she had marked joint deformities, including digital shortening, telescoping of fingers, swan-neck deformities, and features consistent with classical arthritis mutilans, along with extensive psoriatic plaques and nail dystrophy. She also reported sicca symptoms, including dryness of eyes and mouth. Laboratory investigations during disease exacerbation revealed significantly elevated inflammatory markers (CRP 113 mg/L). Immunological workup showed positive rheumatoid factor, anti-CCP antibodies, a homogeneous ANA pattern, and elevated anti-Ro52 and anti-Ro60 antibodies. Schirmer's test was positive, supporting a diagnosis of secondary Sjögren's syndrome. X-ray of bilateral hands showed a pencil-in-cup appearance, while MRI of the sacroiliac joints demonstrated preserved joint spaces without evidence of active sacroiliitis. A labial salivary gland biopsy was performed, and histopathological confirmation is awaited.

Conclusion: This case highlights the rare coexistence of arthritis mutilans, rheumatoid arthritis–like autoantibody positivity, and secondary Sjögren's syndrome in psoriatic arthritis, reflecting profound immune dysregulation and catastrophic joint damage. Early diagnosis and timely, aggressive targeted immunomodulatory therapy are crucial to prevent irreversible disability and improve long-term outcomes.

POM31: The Paradox Within: Autoimmune Cytopenia Unveiling a Silent Thrombus

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Introduction: The simultaneous occurrence of autoimmune cytopenias and life-threatening thrombosis presents a diagnostic paradox. We report an intriguing case where evolving hematologic and neurological manifestations led to the revelation of a rare autoimmune triad.

Case Report: A 29-year-old female presented

with insidious onset of generalised weakness for two months, unaccompanied by any organ-specific symptoms. Examination revealed pallor and hepatosplenomegaly. Laboratory evaluation showed severe anaemia (Hb 3.9 g/dL) and thrombocytopenia (45,000/µL), raising suspicion of a bone marrow disorder. Before the planned biopsy, she developed a sudden severe headache. MRI brain revealed a right cerebellar haemorrhage, prompting urgent evaluation for an underlying coagulopathy. The direct Coombs test was strongly positive and ANA titre was 1:1280 (homogeneous).The working diagnosis shifted toward autoimmune cytopenia, and high-dose methylprednisolone was initiated. However, MR venography subsequently revealed superior sagittal sinus thrombosis, an unexpected finding in a profoundly thrombocytopenic patient. She developed blurring of vision with fundoscopy revealing grade 4 papilledema with flame-shaped haemorrhage. Further work-up disclosed strongly positive antiphospholipid antibodies, confirming a diagnosis of Probable Catastrophic Antiphospholipid Antibody Syndrome.

Conclusion: This case highlights the need for vigilance when cytopenia coexists with neurological events. Evans syndrome unveiling APLA Syndrome through CVST presentation is exceedingly uncommon. Prompt recognition of this rare autoimmune interplay and early institution of immunosuppression with anticoagulation can be lifesaving.

Fig.1

POM32: Rheumatoid arthritis associated with Hashimoto Thyroiditis: A case report

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Introduction: Rheumatoid arthritis (RA) is a chronic systemic autoimmune inflammatory disorder primarily affecting synovial joints, while Hashimoto thyroiditis (HT) is the most common cause of autoimmune hypothyroidism. The coexistence of multiple autoimmune diseases in a single patient reflects shared immunogenetic mechanisms and immune dysregulation.

Case Report: I report a case of 41 year old female with a two year history of rheumatoid arthritis who was on regular treatment with methotrexate, hydroxychloroquine, and iguratimod . 6 month back, she presented with increasing pain and stiffness for which she showed to many physicians and her therapy was escalated without much improvement. She also complained of fatigue, weight gain, hair loss, and irregular menses. Physical examination revealed bradycardia, non-pitting pedal edema, and a mildly diffuse goitre. Laboratory evaluation showed a markedly elevated thyroid-stimulating hormone level (TSH: 96.5 mIU/L) along with elevated anti–thyroid peroxidase antibodies, confirming the diagnosis of Hashimoto thyroiditis. The patient was started on levothyroxine therapy, following which a marked improvement in systemic and joint symptoms was observed on follow-up.

Conclusion: This case highlights the importance of screening for autoimmune thyroid disease in patients with rheumatoid arthritis, as early identification and appropriate management can improve overall outcomes and quality of life.

Recognition of such autoimmune overlap syndromes is essential for a comprehensive, multidisciplinary approach to patient care.

POM33: Vanishing Vision and the Pulseless Clue: Takayasu Arteritis Presenting as Sudden Monocular Vision Loss in an Adolescent Male

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Introduction: Takayasu arteritis is a rare large-vessel vasculitis with a strong female predominance, making diagnosis in males uncommon and often delayed. Ocular involvement as an initial manifestation is rare. We report an adolescent male presenting with sudden monocular vision loss, underscoring the need to suspect large-vessel vasculitis in such atypical presentations.

Case Report: A 17-year-old previously healthy male presented with sudden, painless loss of vision in the right eye. Visual acuity was reduced to hand movements on the right and normal on the left. Ophthalmic examination, including anterior segment and fundus evaluation, was normal, with no relative afferent pupillary defect. Systemic examination revealed right-sided carotid tenderness, an audible bruit over the right carotid artery, and absence of the right radial pulse, with inter-arm blood pressure asymmetry. Laboratory evaluation showed markedly elevated inflammatory markers (erythrocyte sedimentation rate 90 mm/hr, C-reactive protein 38 mg/L). Computed tomography angiography demonstrated concentric wall thickening and luminal narrowing of the aortic arch branches, including the right common carotid artery, consistent with Takayasu arteritis, Numano type IIa. The visual loss was attributed to ocular hypoperfusion secondary to carotid involvement. The patient was diagnosed with active Takayasu arteritis and initiated on systemic immunosuppressive therapy.

Conclusion: Takayasu arteritis should be considered in young patients presenting with unexplained monocular vision loss, particularly when vascular signs are present. Early recognition through careful clinical examination and vascular imaging is essential, as prompt immunosuppressive therapy may prevent irreversible ischemic complications.

POM34: Lost Antibodies, Persistent Disease: A Refractory Anti–PL-7 Myositis

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Introduction: Anti–PL-7 antibody–associated inflammatory myositis is most commonly reported in association with interstitial lung disease, while muscle involvement is often mild. In contrast, this patient demonstrated no lung involvement but severe, metabolically active myositis with persistently elevated CK levels and diffuse PET-CT uptake, despite escalation to biologic therapy.

Case Report: A 68-year-old woman with inflammatory myositis and prior anti–PL-7 antibody positivity, on oral corticosteroids and azathioprine for two years, presented with a relapse marked by progressive proximal muscle weakness and myalgia of the upper and lower limbs for two months. She had comorbid type 2 diabetes mellitus and hypertension. Serum creatine kinase (CK) was markedly elevated (4,407 U/L). Whole-body PET-CT demonstrated diffuse fluorodeoxyglucose uptake in limb and shoulder girdle muscles, consistent with active

myositis. Repeat autoimmune testing during relapse was negative for anti–PL-7. Despite intravenous corticosteroids and rituximab, she failed to achieve remission at four months, with persistently elevated CK levels, indicating a steroid-dependent, treatment-refractory course. (Table 1. Timeline of clinical course, investigations, and treatment). Loss of detectable anti–PL-7 antibodies during relapse may reflect immune modulation or assay limitations rather than true disease inactivity, emphasizing the need to interpret serology in clinical context.

Conclusion: Anti–PL-7 myositis can present as a relapsing, myositis-dominant, steroid-dependent disease even without lung involvement. Serology alone is unreliable; integrated assessment is essential for monitoring disease activity and guiding development of targeted therapies.

POM35: When Lupus Strikes the Gut: A Rare Case of SLE Associated Intussusception

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Introduction: Intussusception, the telescoping of one intestinal segment into another, is common in children and usually idiopathic. In adults, it typically results from a pathological lead point such as a tumour, polyp, Meckel's diverticulum, or postoperative adhesions. In SLE, altered motility may contribute. Delayed recognition can cause significant morbidity if untreated.

Case Report: A 50-year-old woman with no prior comorbidities presented with 8 months of photosensitive pruritic scaly lesions, 6 months of inflammatory polyarthritis, and 7 days of dark red blood–mixed stools, along with alopecia, Raynaud's phenomenon, and oral ulcers. Investigations revealed pancytopenia, low C3/C4, elevated ESR and CRP, and ANA positivity (homogeneous, 1:1280) with antihistone and antinucleosome antibodies, suggestive of active SLE. She was initially treated as lupus enteritis after excluding infections and received pulse corticosteroids. Subsequently, she developed acute colicky right iliac fossa pain, abdominal fullness, constipation, followed by large-volume bloody stools. Abdominal ultrasound showed a 9 × 8 cm right iliac fossa mass, and CT enterography confirmed intussusception of the distal ileum into the ascending colon. In the setting of active SLE, mesenteric vasculitis was considered the likely cause. She was managed conservatively with close monitoring and immunosuppressive therapy.

Conclusion: Mesenteric vasculitis is a rare but life-threatening complication of SLE with high mortality. Early recognition through clinical vigilance and imaging enables timely initiation of high-dose corticosteroids and immunosuppressive therapy, preventing bowel ischemia, necrosis, and emergency surgery. A high index of suspicion is essential, even when classical lupus features are subtle or absent.

Fig.1

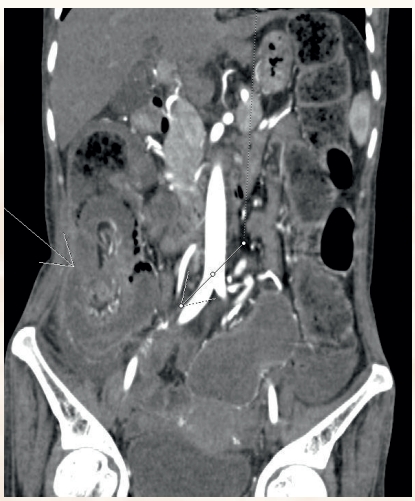
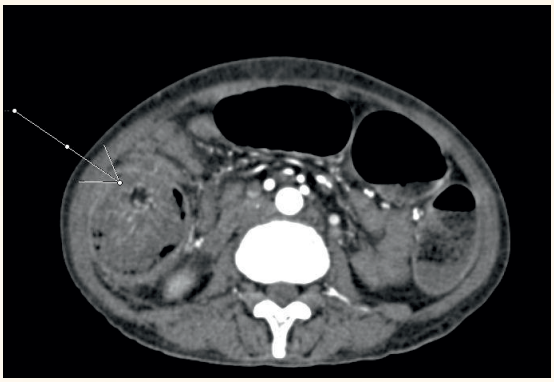


Fig 2.



POM36: When Still's Is Not Still: Progression from Macrophage activation syndrome to AA Amyloidosis

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Introduction: Adult-onset Still's disease (AOSD) is a systemic autoinflammatory disorder characterized by high-grade quotidian fever, arthritis, rash, and hyperferritinemia. Macrophage activation syndrome (MAS) is a severe hyperinflammatory complication. Persistent uncontrolled inflammation may result in secondary AA amyloidosis, an uncommon but serious cause of renal morbidity in young patients.

Case Report: A 21-year-old male presented with high-grade quotidian fever, inflammatory polyarthritis, evanescent rash, and sore throat. Laboratory evaluation showed anemia, neutrophilic leukocytosis, elevated CRP, and markedly raised ferritin (3,208 ng/mL). Bone marrow examination demonstrated hemophagocytosis. Extensive infectious and autoimmune workup was negative. Suspected macrophage activation syndrome (MAS) secondary to evolving adult-onset Still's disease (AOSD) was considered. He responded to pulse corticosteroids but was subsequently lost to follow-up. Within 1.5 years, he presented again with high-grade quotidian fever, inflammatory polyarthritis, and anasarca. Investigations revealed ferritin 1,292 ng/mL, severe hypoalbuminemia (1.5 g/dL), and nephrotic-range proteinuria. Infectious and autoimmune causes were excluded. Renal biopsy showed Congo red–positive deposits with apple-green birefringence under polarized light, confirming secondary AA amyloidosis. The clinical course suggested progression of inadequately controlled AOSD from hyperinflammatory flare to biopsy-proven renal amyloidosis.

Conclusion: Suspected MAS may be the initial manifestation of adult-onset Still's disease. Inadequate long-term inflammatory control, particularly after loss to follow-up, can lead to early secondary AA amyloidosis. This case underscores the importance of sustained disease-modifying therapy and strict monitoring to prevent irreversible organ damage in systemic autoinflammatory disorders.

POM37: Sjögren's Syndrome–Associated Sensory Neuronopathy Presenting as Progressive Sensory Ataxia: A Case Report

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Introduction: Profound sensory ataxia with preserved motor strength presents a diagnostic challenge, with etiologies ranging from paraneoplastic syndromes, autoimmune diseases, toxins, infections, and drug-induced ganglionopathy to idiopathic causes. Among these, primary Sjögren's syndrome is an under-

recognized but treatable cause of sensory neuronopathy requiring clinical suspicion and early immunomodulation.

Case Report: A 64-year-old woman presented with four years of progressive, asymmetric paresthesias evolving into profound sensory ataxia, rendering her dependent for ambulation. Examination revealed loss of joint position and vibration sense in all limbs, global areflexia, pseudoathetosis, and positive Romberg sign, with preserved motor strength. Nerve conduction studies showed non-recordable sensory nerve action potentials with normal motor responses, consistent with pure sensory neuronopathy. Somatosensory evoked potentials demonstrated prolonged central conduction times, suggesting dorsal column involvement. Autoimmune workup revealed anti-SSA (Ro52) positivity, Schirmer's test <3 mm, elevated IgG, and anti-TPO positivity, fulfilling criteria for primary Sjögren's syndrome with high disease activity (ESSDAI 16). Other metabolic, infectious, and paraneoplastic causes were excluded. She received intravenous immunoglobulin followed by rituximab and corticosteroids, with stabilization of symptoms. This case highlights a classic but frequently overlooked presentation of Sjögren's-associated ganglionopathy.

Conclusion: This case emphasizes that progressive ataxia without weakness is not always degenerative. Careful clinical localization, supported by electrophysiology, can uncover immune-mediated dorsal root ganglion disease. Recognizing Sjögren's-associated sensory neuronopathy early allows targeted immunotherapy, preventing irreversible disability and significantly altering long-term functional outcomes.

POM39: Silence in the Muscles, Persistence in Fever - Until the Lungs Spoke

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Introduction: Antisynthetase syndrome is a rare autoimmune disorder associated with anti–aminoacyl tRNA synthetase antibodies and diverse clinical manifestations. The anti-PL-12 subtype is uncommon and often presents with lung-predominant disease and minimal muscle involvement, frequently leading to diagnostic delay, especially when fever is the dominant feature.

Case Report: A 52-year-old woman presented with intermittent fever for two years without localizing symptoms. Extensive evaluation for pyrexia of unknown origin, including haematological, biochemical, microbiological, and bone marrow investigations, was unremarkable. Autoimmune screening, including antinuclear antibody testing was negative, and muscle enzymes, including creatine kinase and CK-MB, were within normal limits. Empirical courses of corticosteroids failed to produce sustained clinical improvement. High-resolution computed tomography of the chest revealed features consistent with interstitial lung disease. In view of unexplained ILD with systemic symptoms and absence of an alternative diagnosis, a myositis-specific antibody panel was performed, which demonstrated strong positivity for anti-PL-12 antibody. A diagnosis of antisynthetase syndrome with lung-dominant involvement was thus established. The case highlights the diagnostic challenge posed by steroid-resistant fever and isolated ILD in the absence of classical muscle manifestations.

Conclusion: Anti-PL-12–positive antisynthetase syndrome should be considered in patients with pyrexia of unknown origin and unexplained interstitial lung disease, even when muscle enzymes and ANA are normal. Early recognition is essential, as the disease often shows poor steroid response and requires steroid sparing immunosuppression.

POM40: A Rare Convergence: Antiphospholipid Antibody Syndrome with Deficiencies in Protein C and S, Presenting as Budd - Chiari Syndrome

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Introduction: Budd-Chiari syndrome (BCS) is an uncommon hepatic vascular disorder characterized by obstruction of hepatic venous outflow tract, leading to hepatic congestion, portal hypertension. This condition is strongly linked to hypercoagulable states, both inherited and acquired. APLA is an autoimmune prothrombotic condition associated with recurrent arterial and venous thrombosis

Case Report: We report a case of a female patient who initially presented with features suggestive of chronic liver disease. Standard evaluation for typical causes of liver dysfunction was unrevealing. Imaging with Doppler ultrasound and contrast enhanced studies confirmed hepatic venous outflow obstruction consistent with Budd chiari syndrome. The thrombophilia panel revealed high titres of antiphospholipid antibodies with concomitant protein C and S deficiencies. No evidence of cirrhosis, malignancy or extrahepatic liver pathology was found. The patient was managed with long term anticoagulation.

Discussion: BCS secondary to APLA is well documented but rare, often presenting subacutely or chronically with insidious onset and non-specific laboratory abnormalities, leading to diagnostic delay. Case reports indicates that hypercoagulability is a frequent underlying cause of BCS and recommends thorough thrombophilia screening in atypical presentation.

Conclusion: This case highlights the need for heightened rheumatologic vigilance for autoimmune mediated hypercoagulable disorders in patients with unexplained chronic liver disease with portal hypertension. Prompt identification of APLA and associated thrombophilia factors such as protein C and S deficiency can facilitate timely anticoagulation and improve outcomes

POM41: Anemia and Renal Failure in an Elderly Woman: Not Always CKD

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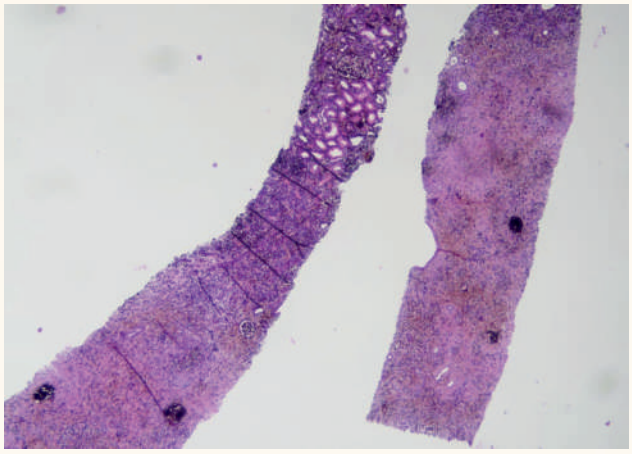
Introduction: We present a rare MPO-positive granulomatous vasculitis case in a 67-year-old woman. GPA involves upper/lower respiratory tracts and kidneys but can present only with renal involvement, mimicking multiple myeloma. This case shows atypical MPO-positive GPA with granulomatous interstitial nephritis and constitutional symptoms.

Case Report: A 67-year-old housewife, had 4 months of anorexia, weight loss, fatigue, NYHA II dyspnea, and 1 week dry cough and comorbidities of hypertension, hypothyroidism, T2DM. Examination showed pallor, pedal edema,

pulmonary systolic murmur. Investigations were significant for anemia (Hb 6.7 g/dL), leukocytosis (17,000/cumm, neutrophilia+), ESR 113 mm/hr, CRP 125 mg/L, Creatinine 3.62 mg/dL, hyperglobulinemia 6.58 g/dL. Further investigations revealed polyclonal gammopathy, episcleritis, sinusitis/ILD on CT, p-ANCA+/MPO+ (67 U/mL), and renal biopsy demonstrated necrotizing granulomatous nephritis.

Conclusion: Timely vasculitis workup in renal failure with hyperglobulinemia enabled GPA diagnosis and immunosuppression response.

Fig.1



POM42: Hypokalemic Periodic Paralysis as an Initial Presentation of Primary Sjögren's Syndrome Without Sicca Symptoms

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Introduction: Primary Sjögren's syndrome is a systemic autoimmune disorder primarily affecting exocrine glands. Extra-glandular manifestations may precede sicca symptoms and include renal involvement such as distal renal tubular acidosis, which can rarely present with severe hypokalemia and acute flaccid paralysis.

Case Report: A 50-year-old woman presented with acute onset quadriparesis upon awakening in the morning. There was no history of fever, sensory symptoms, bowel or bladder involvement, or prior similar episodes. Neurological examination revealed flaccid paralysis with power 1/5 in all four limbs, hypotonia, absent deep tendon reflexes, mute plantar responses, and preserved sensation. Laboratory investigations showed severe hypokalemia (1.6 mEq/L) with normal anion gap metabolic acidosis (pH 7.317, HCO_3^- 14.2 mmol/L), inappropriately high urine pH (6.5), positive urinary anion gap, and elevated urinary potassium excretion, suggestive of distal renal tubular acidosis. Autoimmune evaluation revealed positive ANA with anti-Ro and anti-La antibodies. Objective tests for sicca symptoms were normal. Ultrasound of salivary glands showed hypoechoic nodules, and minor salivary gland biopsy demonstrated lymphoplasmacytic infiltration with a focus score >1, confirming primary Sjögren's syndrome. The patient was treated with intravenous potassium chloride followed by oral potassium citrate, resulting in rapid neurological recovery. She was started on oral prednisolone followed by tapering dose.

Conclusion: Primary Sjögren's syndrome should be considered in patients with unexplained hypokalemic paralysis and distal renal tubular acidosis, even in the absence of sicca symptoms, to enable early diagnosis and targeted management.

POM43: VEXAS Syndrome Masquerading as Systemic Autoimmune Disease - A Rare Case

Report

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Introduction: VEXAS syndrome is a recently described adult-onset autoinflammatory disorder caused by somatic mutations in the UBA1 gene. It presents with refractory systemic inflammation and hematologic abnormalities and is frequently misdiagnosed as autoimmune or vasculitic disease.

Case Report: A 58-year-old male presented with inflammatory arthritis of small joints of the hands for two months,along with painful redness of the left eye with decreased vision and tender erythematous skin lesions over the chest, back and shoulders for three days. He had prior self-resolving episodes of painful red eye 6 months and 1 year back, and history of two ischemic strokes with hemiparesis in past 4 years. Examination revealed anterior uveitis, erythematous papular rash and inflammatory arthritis. Investigations showed macrocytic anemia, leukopenia and thrombocytopenia with elevated inflammatory markers and negative ANA profile,cANCA and pANCA. Skin biopsy revealed neutrophilic dermatosis. Bone marrow examination showed hypercellularity with multilineage dysplasia and prominent cytoplasmic vacuolization of myeloid and erythroid precursors. In view of a clinical picture with bone marrow suggesting MDS, VEXAS syndrome was kept as a possibility .Next-generation sequencing identified a somatic UBA1 (Met41) mutation, confirming VEXAS syndrome . The patient showed rapid clinical improvement with corticosteroids.

Conclusion: VEXAS syndrome should be considered in older males with unexplained systemic inflammation, cytopenias and steroid-responsive disease. Recognition of this entity is crucial to avoid diagnostic delay and guide appropriate immunosuppressive and hematologic management.

POM44: When Lupus Meets the Spine: A Rare Case of SLE with Ankylosing Spondylitis

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Introduction: Systemic lupus erythematosus(SLE) and ankylosing spondylitis(AS) are chronic autoimmune diseases with distinct immunopathogenesis, clinical features, and genetic associations. Their coexistence is rare, particularly in young female patients, and poses diagnostic and therapeutic challenges.

Case Report: A 26-year-old female presented with a 6 months history of intermittent fever, photosensitivity, oral ulcers, inflammatory polyarthritis, and fatigue. Examination revealed malar rash and non-erosive arthritis involving small joints of the hands. Laboratory evaluation showed anemia, leukopenia, elevated ESR, positive ANA, anti-dsDNA antibodies, and low complement levels, fulfilling the criteria for SLE. She was started on corticosteroids and hydroxychloroquine with partial improvement. During follow-up, the patient developed persistent inflammatory low back pain with morning stiffness lasting more than one hour, not responding adequately to standard SLE therapy. Further evaluation revealed restricted spinal

mobility, positive FABER test and MRI of the sacroiliac joints showing bilateral sacroiliitis. HLA-B27 testing was positive, leading to a diagnosis of AS.

Conclusion: The coexistence of SLE and AS is uncommon but should be considered in patients presenting with overlapping clinical features of connective tissue disease and inflammatory spondyloarthritis. Early recognition is essential to optimize management and prevent disease-related complications. This case highlights the importance of a multidisciplinary approach and individualized treatment.

POM45: Muscle's Blues & Neoplastic Hues- A Case of Paraneoplastic Polymyositis

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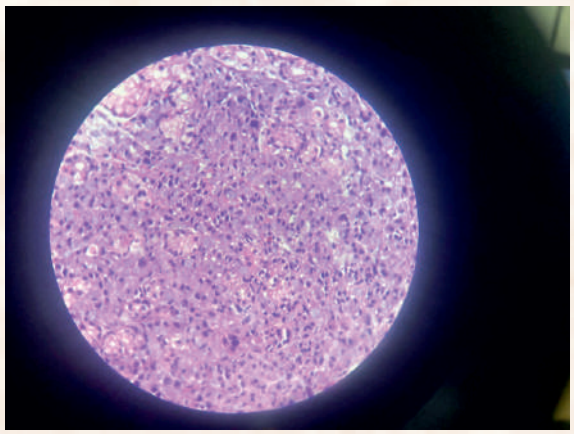
Introduction: A 49-year non-morbid male presented with recurrent vomiting and progressive abdominal distension for 3months, along with unintentional weight loss and multiple enlarged lymph nodes. Preceding these, he had a history of shoulder pain and proximal limbs weakness which had been managed as suspected polymyositis with oral steroids.

Case Report: Clinical findings included tachycardia/tachypnoea, pallor, distended abdomen with ileus; multiple, fixed, hard nodes in neck/axilla/groin. Investigations showed anaemia needing transfusions, neutrophilic leucocytosis, hyperuricaemia, hyperphosphataemia, hyperkalaemia, prerenal-AKI; raised ALP/CRP/CPK/ferritin. Cultures/LDH/amylase/lipase/procalcitonin/ANA were negative. Empirical antibiotics, electrolytes, TPN and hydration were undertaken. Whole Body PET/CT showed diffuse stomach wall thickening, FDG-avid nodes above and below diaphragm with bone and bladder lesions-likely metastases; FDG avid focally calcified lesions in muscles-sequelae of polymyositis. Endoscopic biopsy from gastric mucosa showed signet ring adenocarcinoma. Immunohistochemistry on core cervical node biopsy= CK7:Positive, CK20:Positive, SATB2:Positive, CDX2:Positive, TTF1:Negative, PSA:Negative-metastatic poorly differentiated adenocarcinoma possibly from lower GI primary. Considering advanced metastatic disease with poor general condition, palliative care was selected with focus on pain management, nutrition and symptomatic relief.

Conclusion: Initial presentation of muscle pain and weakness with the radiographic evidence was concluded to be paraneoplastic polymyositis as a heralding feature of the aggressive malignancy, and managed with low dose steroids. New-onset polymyositis that is atypical or aggressive in course should raise suspicion for underlying malignancy.

Fig.1



POM46: Retroperitoneal Fibrosis with Vasculitic Rash: A Rare Presentation of IgG4-Related Disease in a Young Female

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Introduction: IgG4 related disease is a systemic immune mediated fibroinflammatory disorder with diverse organ involvement. Retroperitoneal fibrosis is a recognized manifestation but its association with vasculitic skin rash in young females is rare and diagnostically challenging.

Case Report: A 24-year old female with history of low grade fever for one year, palpable purpuric rash over limbs for one year and episodic abdominal pain for one month. She presented to the emergency with abdominal pain refractory to intravenous analgesics. Ultrasonography revealed soft tissue thickening encasing the abdominal aorta, superior mesenteric artery and right renal artery. CECT abdomen suggestive of retroperitoneal fibrosis and mild right sided hydroureteronephrosis. Laboratory evaluation showed normal CBC, LFT, RFT, elevated ESR and CRP, negative ANA and ANCA profile. Serum C3 and C4 titres were 39 mg/dl and 8 mg/dl respectively. Serum IgG4 level was 500 mg/dL. Skin biopsy demonstrated leukocytoclastic vasculitis. Retroperitoneal Biopsy was not feasible due to inaccessibility of the mass. A diagnosis of probable IgG4-related disease was made and corticosteroid therapy was initiated resulting in complete resolution of rash and abdominal pain.

Conclusion: This case highlights a rare presentation of IgG4-related disease in a young female with retroperitoneal fibrosis and vasculitic rash. In the absence of histopathological confirmation, characteristic imaging findings, elevated serum IgG4 levels, low tires of complements, exclusion of mimickers and excellent steroid responsiveness support the diagnosis.

POM47: Expanding Clinical Spectrum of Anti-Synthetase Syndrome: A Three-Case Series Highlighting Phenotypic Variability and Diagnostic Challenges

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Introduction: Anti-Synthetase-Syndrome (ASS) is a heterogeneous autoimmune inflammatory myopathy characterized by anti–aminoacyl tRNA synthetase antibodies, most commonly anti-Jo1, and classically associated with myositis, interstitial lung disease (ILD), inflammatory arthritis, and cutaneous features. Clinical presentation varies widely depending on the antibody subtype and organ predominance, often leading to delayed diagnosis.

Case Report: We describe three patients with distinct ASS phenotypes. Case 1: A middle-aged female with prior ILD developed progressive proximal muscle weakness and inflammatory polyarthralgia. Laboratory evaluation showed elevated CPK (588 U/L) and strong anti–Jo-1 positivity. MRI confirmed inflammatory myositis. Case 2: A 48-year-old woman presented with severe proximal weakness (3/5), dysphagia, and hypophonic speech. CPK peaked at 6640 U/L. Anti–Jo-1 antibody was strongly positive, and CT chest revealed ILD. She showed minimal

response to steroids and azathioprine but improved markedly following rituximab therapy. Case 3: A 63-year-old male presented with chronic seronegative small-joint inflammatory arthritis and proximal weakness. CK was mildly elevated, and myositis antibody testing revealed anti-PL12 positivity. No pulmonary involvement was detected. All patients fulfilled criteria for ASS based on clinical features and antibody profiling. Phenotypes ranged from ILD-dominant and severe bulbar myositis to arthritis-predominant disease without lung involvement. Treatment included corticosteroids and immunosuppressive agents, with biologic therapy required in refractory disease.

Conclusion: This series underscores the protean manifestations of ASS ranging from ILD-dominant and bulbar myopathy–severe phenotypes to arthritis-predominant presentations without pulmonary involvement. Antibody profiling plays a pivotal role in early diagnosis, especially in patients with unexplained ILD, refractory myopathy, or seronegative polyarthritis.

Fig.1

POM48: Unprovoked Deep Vein Thrombosis as the Initial Manifestation of Primary Sjögren's Syndrome with Antiphospholipid Antibodies

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Introduction: Primary Sjögren's syndrome is an autoimmune disorder that may be associated with antiphospholipid antibodies. However, clinically significant thrombosis as the initial manifestation is uncommon. Recognition of this presentation is important, as venous thrombosis may precede classical sicca symptoms and delay diagnosis.

Case Report: A 35-year-old female with no prior systemic illness, obstetric morbidity, trauma, recent surgery, or drug exposure presented with one month of progressive left lower limb pain and swelling. Examination revealed non-pitting edema and erythema. Doppler ultrasonography showed deep vein thrombosis involving the left common femoral, superficial femoral, and external iliac veins. MRI pelvis confirmed venous thrombosis without compressive pathology. Routine hematological and biochemical tests were normal. Thrombophilia workup revealed positive anticardiolipin IgG (179) and beta-2 glycoprotein I antibodies, while protein C, protein S, antithrombin III levels, activated protein C resistance, factor V Leiden mutation, and homocysteine were normal. Autoimmune evaluation showed positive ANA, anti-SSA, and anti-SSB antibodies. Schirmer's test was abnormal, and a minor salivary gland biopsy demonstrated lymphocytic sialadenitis with a focus score greater than three, confirming primary Sjögren's syndrome. The patient improved with

anticoagulation

Conclusion: Although antiphospholipid antibodies may be present in a subset of patients with primary Sjögren's syndrome, thrombosis as the initial presentation is uncommon. This case emphasizes the need to evaluate young patients with unprovoked venous thrombosis for underlying autoimmune disorders to enable timely management.

POM49: Eosinophilic Granulomatosis with Polyangiitis presenting as a stroke chameleon

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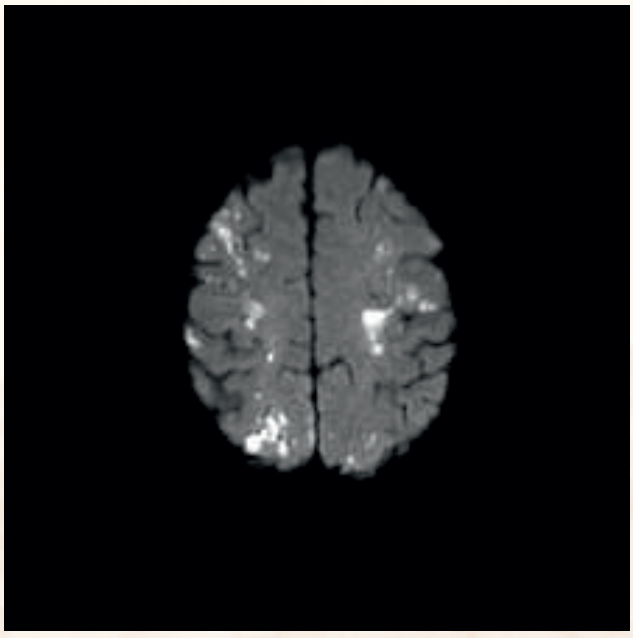
Introduction: Eosinophilic Granulomatosis with Polyangiitis (EGPA) is a rare ANCA-associated vasculitis characterised by asthma, eosinophilia, and multisystem involvement. Neurological and cardiac manifestations are uncommon and may mimic acute vascular events, leading to delayed diagnosis and potentially severe morbidity

Case Report: A middle-aged woman with a background of asthma, allergic rhinitis, recurrent nasal polyps and lichen sclerosus presented with acute central chest pain associated with diaphoresis and vomiting. Initial investigations revealed raised troponin T and marked eosinophilia, with electrocardiographic changes consistent with non-ST elevation myocardial infarction. She was initiated on standard acute coronary syndrome therapy. Three days later, she developed acute right upper limb weakness followed by visual disturbance and progressive quadriparesis. Neuroimaging demonstrated multiple acute bilateral cerebral and cerebellar infarctions. Vasculitis screening showed positive p-ANCA with elevated myeloperoxidase antibodies, and eosinophil count exceeded 1×10^9/L. Based on multisystem involvement and fulfilment of the 2022 ACR/EULAR classification criteria, a diagnosis of eosinophilic granulomatosis with polyangiitis was made. She was treated with high-dose corticosteroids and intravenous cyclophosphamide, resulting in significant neurological recovery with minimal residual deficit on follow-up

Conclusion: Eosinophilic Granulomatosis with Polyangiitis can present as a stroke mimic with concurrent cardiac involvement. Early recognition of eosinophilia and systemic features is essential. Prompt immunosuppressive therapy can result in significant neurological recovery and prevent irreversible organ damage in this rare but potentially life-threatening condition.

Fig.1



POM50: When Hyperinflammation Meets Capillary Leak: A Rare Case of Macrophage Activation Syndrome with Secondary

Systemic Capillary Leak Syndrome in a Patient of Rheumatoid Arthritis with Sjogren Overlap

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Introduction: Macrophage activation syndrome in association with secondary systemic capillary leak syndrome is rare, life threatening hyperinflammatory condition. Their coexistence is very uncommon and challenging in both diagnostic and therapeutic aspects, requiring strong clinical suspicion along with a multidisciplinary approach.

Case Report: We report a 55-year-old lady with background of Type II Diabetes, longstanding untreated rheumatoid arthritis and hypothyroidism, presenting in emergency with fever, body ache, anorexia, generalised body swelling with shock. The shock was only responding transiently to IV fluids and blood pressure was stabilized after starting steroids. Meanwhile she developed an infected baker's cyst which was drained surgically and the patient became afebrile, improving clinically. Routine labs were normal except hypoalbuminemia and raised CRP. The RA factor was more than 150, ANA was strongly positive with SSA 3+ and Rho 52 2+ suggestive of overlapping rheumatoid arthritis with secondary Sjogren's. Later she developed new onset high grade fever and severe pancytopenia, detailed work up excluded sepsis and DIC. Her fever responded to steroids but she was shifted back to ICU as despite resolution of infection, she had recurrent pattern of fluid responsive hypotension, marked third spacing, polyserositis, hypoalbuminemia without proteinuria. Treatment with IV albumin, pulse steroid and IVIG resulted in transient hemodynamic improvement.

Conclusion: This case underscores the rare coexistence of MAS and secondary SCLS in a scenario of uncontrolled autoimmune disease. Role of early recognition of hyperinflammatory syndromes in patients with refractory shock and capillary leak even in presence of apparent infection is crucial. Prompt immunomodulatory therapy may be lifesaving, although outcomes remain guarded.

POM51: Rheumatoid arthritis at the maternal interface: expected immunology, unexpected disease

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Introduction: Pregnancy and postpartum period are well known to impact the clinical course of rheumatoid arthritis. While many patients experience improvement during pregnancy, disease flares and new onset RA frequently occurs after delivery. Diversity in serological status, disease behaviour possess treatment and diagnostic challenges.

Case Report: We describe three clinically distinct presentations of RA in intra and peripartum period. Case 1: A patient developed a new onset RA flare soon after delivery, so much so that she was unable to hold her baby. Serology revealed strongly positive anti-CCP >500. Case 2: Another patient exhibited classical features of RA but was seronegative. He was successfully managed with conservative DMARDs and corticosteroids, illustrating that Sero negativity does not exclude active inflammatory disease

requiring treatment. Case 3: The third patient, diagnosed case of RA had worsening of RA symptoms during pregnancy contrary to typical disease course. Challenge was in the treatment as weapons (pregnancy safe drugs) are limited during pregnancy. The patient was managed with high dose steroids and conventional DMARDs.

Conclusion: These cases emphasize the heterogeneous immunological and clinical behaviour of RA during pregnancy and the postpartum period. These cases highlight the importance of individualized therapy and further research in management and diagnosis as pregnancy and lactation limit the available treatment modalities.

POM52: Dynamic spectrum of Rheumatologic Manifestations in a case of Psoriatic SpA

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Introduction: SpA encompasses conditions such as ankylosing spondylitis, psoriatic arthritis, reactive arthritis, enteropathic arthritis, and undifferentiated SpA with overlapping features of backache, arthritis, extra-articular manifestations (Uveitis, IBD), sacroiliitis and HLA-B27 positivity.

Case Report: A 52-year-old female presented to the ophthalmology department with complaints of left eye pain and blurring of vision for 1 month, she was diagnosed with Uveitis and treated with topical steroids. This was followed by symptoms of inflammatory back pain, EMS and enthesopathy. She was evaluated for SpA with MRI-SI joint and HLA B-27 (both positive). She was treated as axial SpA with NSAIDs, Salfasalazine, parenteral Depomedrol (weekly); her symptoms improved. She stopped medications after a few months and lost follow-up. She took NSAIDs when needed and was doing fine for 4 years when she developed insidious onset right knee swelling with pain associated with low-backache. She received 4 doses of Etanercept, which led to development of fever, diarrhea and abdominal pain, no improvement in knee synovitis. Her CECT/PET-CT showed terminal ileal-jejunal thickening and mesenteric nodes. Intestinal TB versus IBD was suspected, her colonoscopy revealed deep longitudinal ulcers with cobblestone appearance; biopsy was negative for tuberculosis. Diagnosis of Crohn's disease flare secondary to Etanercept was made and the patient was treated with mesalamine. Her symptoms improved over the next 6 months.

Conclusion: Spondyloarthropathies may present as continuum: the full manifestation of SpA may develop over years. Etanercept should be avoided in patients with Uveitis and IBD symptoms.

POM53: Gout in disguise: Asymmetric Sacroiliac involvement with peripheral crystal deposition

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Introduction: Inflammatory back pain with acute polyarticular involvement necessitates differentiation from infective and crystal-induced arthritides. Tuberculous arthritis and polyarticular gout are important mimickers of seronegative spondyloarthritis. Careful clinical phenotyping combined with advanced imaging is essential to identify the dominant disease process

and guide appropriate management.

Case Report: A 45-year-old male presented with a one-year history of inflammatory low back pain with morning stiffness, nocturnal symptoms, and improvement with activity, followed by episodic swelling with discharge from the left ring finger and acute onset symmetrical inflammatory polyarthritis involving bilateral ankles, feet, small joints of the hands, and subsequently the right knee. Examination revealed antalgic gait, ankle synovitis, positive modified Schober's test, unilateral sacroiliac tenderness, and acute dactylitis of the right fourth digit. Tuberculous arthritis was considered due to erosive changes on foot radiographs, however absence of constitutional symptoms, lack of progressive monoarticular destruction, and rapid steroid responsiveness made infection unlikely. Dual-energy computed tomography demonstrated monosodium urate crystal deposition in the ankles and hands, supporting coexistent polyarticular gout, however this did not account for axial inflammatory back pain and unilateral sacroiliitis confirmed on MRI. Non-steroidal anti-inflammatory drugs provided limited benefit, while systemic corticosteroids resulted in rapid clinical improvement.

Conclusion: This case highlights the diagnostic complexity of inflammatory arthritis with overlapping features of seronegative spondyloarthritis and polyarticular gout. Advanced imaging including DECT and MRI is crucial to distinguish coexisting pathologies, exclude infection, and identify the predominant inflammatory process for targeted therapy.

Fig.1

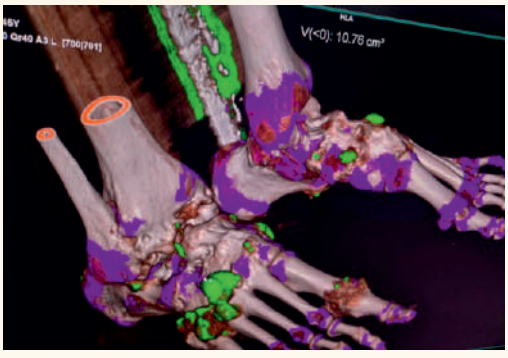
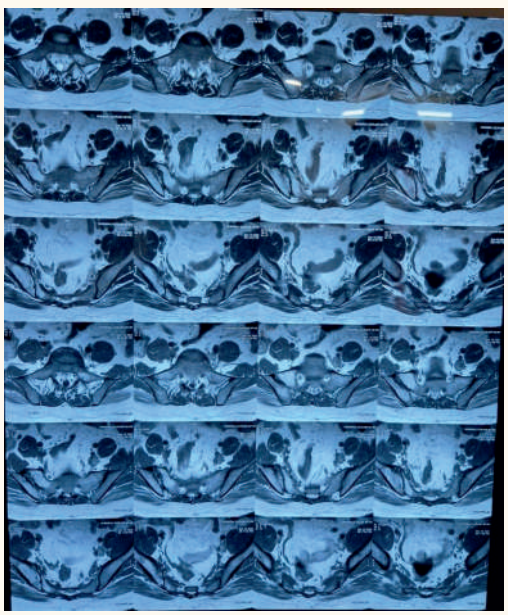


Fig 2.



POM54: Myositis overlap or a mixed bag of antibodies: A diagnostic conundrum

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Introduction: The spectrum of idiopathic inflammatory myopathies (IIM) is widening with distinct phenotypes and myositis-specific autoantibodies (MSAs). In clinical practice, combinations of different antibodies with overlap features are uncommon. Such overlapping antibody profiles may produce atypical or mixed clinical presentations, creating diagnostic and therapeutic challenges.

Case Report: A 65-year-old hypertensive

woman presented with a long-standing photosensitive rash, inflammatory polyarthritis, progressive dyspnea, dysphagia, and proximal muscle weakness. She later developed generalized edema, pustular and bullous skin lesions, hoarseness of voice, and cough. Examination revealed hyperkeratotic fissured finger lesions, pressure sores, bilateral lower-zone lung consolidation, and right upper-limb cellulitis, along with proximal-predominant quadriparesis. Investigations showed markedly elevated creatine phosphokinase (>7600 U/L) with raised AST and LDH. Leukocyte count, ESR, CRP, and serum procalcitonin were elevated. A myositis antibody panel was positive for anti-SAE-1(+++), anti-Ro-52(+++), and anti-PL-12(+). CT thorax showed ground-glass opacities with possible organizing pneumonia and consolidation. She was treated with moderate-dose systemic corticosteroids, antibiotics, and intravenous immunoglobulin, considering disease flare with concomitant infection. During the course, she developed a hospital-acquired infection due to carbapenem-resistant Acinetobacter baumannii with sepsis and multiorgan dysfunction, and ultimately succumbed to her illness.

Conclusion: Anti-SAE antibodies are linked to prominent cutaneous manifestations and dysphagia in dermatomyositis. In contrast, antisynthetase syndrome (anti-PL12) is frequently associated with interstitial lung disease and minimal or absent myositis. Myositis-specific autoantibodies aid phenotypic stratification and prognostication; early recognition and timely intervention may improve outcomes.

Fig.1

POM55: To Suppress or to Treat? SLE-Associated LETM Complicated by Disseminated Tuberculosis in an Adolescent

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Introduction: Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease with diverse neurological manifestations. Longitudinally extensive transverse myelitis (LETM) is a rare but catastrophic presentation. In tuberculosis-endemic regions, coexistence of active tuberculosis poses a major diagnostic and therapeutic challenge while initiating immunosuppression.

Case Report: A 16-year-old girl presented with a 2-month history of insidious, progressive maculopapular eruptions over scalp, face, trunk and extremities with nasolabial fold sparing, intermittent low-grade fever, alopecia, oral ulcers with polyarthralgia, anorexia and weight loss. She developed acute onset bilateral lower-limb weakness, sensory loss and bowel–bladder

dysfunction over 3 days. Examination revealed pallor, hypoxia (SpO_2 90% on room air), bilateral coarse crepitations, abdominal distension, flaccid paraplegia with power 0/5, absent sensations below T8 and mute plantar responses. Investigations showed bicytopenia, elevated ESR and CRP, hypocomplementemia, ANA positivity with anti-dsDNA, anti-Sm/RNP and anti-SSA antibodies. MRI spine demonstrated longitudinally extensive transverse myelitis. HRCT chest revealed tree-in-bud opacities; pleural fluid ADA was elevated, consistent with pulmonary and extrapulmonary tuberculosis. A therapeutic dilemma arose regarding immunosuppression in the presence of active tuberculosis.

Conclusion: This case highlights the complexity of managing neuro-SLE in tuberculosis-endemic settings. Early recognition of LETM, prompt initiation of immunosuppression alongside antitubercular therapy, and a balanced risk–benefit approach are crucial to prevent irreversible neurological deficits and ensure optimal outcomes.

POM56: A Difficult Case of Undiagnosed Mitochondrial Encephalomyopathy in an Adolescent

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Case Report: A 20-year-old male presented with recurrent episode of tonic-clonic movements of all 4-limbs for 3-years and hearing and visual impairment with cognitive decline for 3-months. The patient was apparently well 3-years back when he developed 8-10 episodes of GTCS, he was started on anti-epileptics, but seizure recurred despite being on medication. One year later he had GTCS followed by left sided hemiparesis and right facial deviation. The patient was admitted in tertiary care centre in Rohtak, Haryana, and extensively investigated. MRI Brain revealed subacute infarct in right temporo-parieto-occipital region, while MR-angiography was WNL. Within a month his hemiparesis improved completely. Follow-up CECT-head after 1-year revealed bilateral hypodensities. The patient presented to our centre with progressive visual and hearing loss for 3 months. Parents had to speak in ears to talk to him and also required physical assistance for daily activity. Considering seizures, stroke/SLE with visual and hearing impairment differential of vasculitis, genetic syndrome and DADA2 were considered. Ophthalmic examination revealed bilateral optic-atrophy. His neuroimaging was repeated and discussed, considering complete recovery from stroke, infarct in watershed area, and appearance of new lesion, MELAS was suspected. CPK was normal, Serum and CSF lactate was markedly elevated, MRS revealed lactate peak which confirmed MELAS.

Conclusion: Mitochondrial encephalomyopathy, lactic acidosis, and stroke like episodes-SLE's (MELAS) is a rare inherited multi-system mitochondrial disorder caused predominantly by the A3243G point mutation in tRNA. Suspect MELAS when SLE's do not conform to vascular territories. Associated with recurrent seizures, vision loss and SNHL.

Fig.1

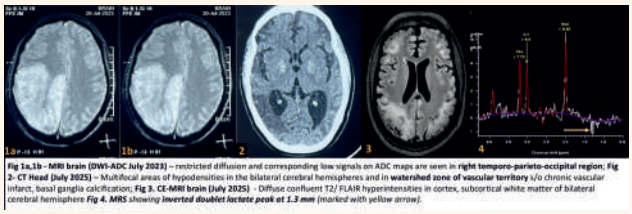

Fig 1a,1b - MRI brain (DWI-ADC July 2023) – restricted diffusion and corresponding low signals on ADC maps are seen in **right temporo-parieto-occipital region**; Fig 2- CT Head (July 2025) – Multifocal areas of hypodensities in the bilateral cerebral hemispheres and in **watershed zone of vascular territory** s/o chronic vascular infarct, basal ganglia calcification; **Fig 3. CE-MRI brain (July 2025)** - Diffuse confluent T2/ FLAIR hyperintensities in cortex, subcortical white matter of bilateral cerebral hemisphere **Fig 4. MRS** showing ***inverted doublet lactate peak at 1.3 mm*** (marked with yellow arrow).

POM57: Anti–Jo-1 Positive Dermatomyositis Mimicking Stiff-Person Syndrome: An Unusual Stiffness-Dominant Presentation

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Introduction: Dermatomyositis is an idiopathic inflammatory myopathy with varied clinical presentations. Rarely, it may initially present with predominant muscle stiffness, mimicking neurological conditions such as stiff-person syndrome, resulting in diagnostic delay. Early recognition of such atypical presentations is important for timely management.

Case Report: A 25-year-old male presented with progressive axial and proximal limb muscle stiffness associated with painful spasms, leading to difficulty in ambulation and rising from a seated position. Initial clinical features were suggestive of stiff-person syndrome. There was no sensory loss, sphincter disturbance, or autonomic dysfunction. Further evaluation revealed symmetrical proximal muscle weakness with subtle cutaneous manifestations. Serum muscle enzymes were markedly elevated. Electromyography showed a myopathic pattern. Myositis-specific autoantibody testing was positive for anti–jo-1 antibodies, supporting the diagnosis of dermatomyositis. Muscle biopsy was not performed as the patient declined the procedure despite detailed counseling. The diagnosis was made based on clinical features, serology, and supportive investigations. The patient was treated with systemic corticosteroids followed by immunosuppressive therapy, resulting in significant clinical improvement.

Conclusion: This case highlights a rare stiffness-dominant presentation of anti–jo-1 positive dermatomyositis mimicking stiff-person syndrome. Awareness of such atypical manifestations and the use of myositis-specific autoantibodies can aid early diagnosis and appropriate immunosuppressive treatment.

POM58: Dermatomyositis Sine Dermatitis - Not so Common Not So Rare Inflammatory Myopathy

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Introduction: Dermatomyositis (DM) is an idiopathic inflammatory myopathy characterized by proximal muscle weakness and skin manifestations. DM-sine myositis/ clinically amyopathic DM has characteristic skin finding without clinically significant muscle disease. DM-sine dermatitis has muscle involvement without skin lesions and is often misdiagnosed as Polymyositis (PM).

Case Report : A 22-year-old female presented with weakness in both upper limbs since 3 months, weakness in both lower limbs since 2 months, difficulty in swallowing since 20 days, more for solids than liquids. There was no history of rash, Gottron's papules, shawl rash, heliotrope rash, or sensory symptoms and no history of statins/fibrates intake. On examination, she had Symmetrical proximal muscle weakness with Power (MRC) of 3/5 (proximal) and 4/5 (distal)

muscles. Deep tendon reflexes were preserved. Lab investigations showed a raised Creatine phosphokinase (CPK)-[8201 U/L], elevated AST-[732 IU/L] , ALT-[137 IU/] and ESR-[38 mm/hr], ANA (IF) was Negative with NXP2 positive Myositis-specific-antibodies. EMG revealed short-duration, low-amplitude MUAPs pattern in bilateral vastus-lateralis. Muscle biopsy demonstrated perifascicular atrophy, perivascular inflammatory infiltrates with capillary involvement. The final diagnosis was Dermatomyositis Sine Dermatitis. She was given 3days of i.v methylprednisolone and intravenous immunoglobulin-(IVIG) 2gram/kg over 5 days along with Physiotherapy and rehabilitation, followed by oral Prednisolone 40mg daily and Mycophenolate Mofetil 500mg twice daily.

Conclusion: DM-sine dermatitis is under-recognized, Skin manifestations may be completely absent, Muscle biopsy being the gold standard, Malignancy screening is mandatory, early treatment improves outcomes. Diagnosis requires clinicopathological correlation and awareness prevents misdiagnosis and treatment delay.

Fig.1

Muscle biopsy (left vastus lateralis) - Perifascicular atrophy- blue arrow Perivascular inflammatory infiltrates- green arrow

POM59: Neurological Storm in a Patient of Systemic Lupus Erythematosus

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Introduction: Neuropsychiatric involvement in SLE (NPSLE) includes a broad spectrum of neurologic and psychiatric manifestation. Due to the rarity of lupus myelitis and seizure disorders, their diagnosis and management present significant clinical challenges.

Case Report: A 16-year-old female with a 2-year history of systemic lupus erythematosus (SLE), on hydroxychloroquine and methotrexate, presented with acute bilateral lower-limb weakness of one-day duration. Clinically, the weakness was of pure motor type, associated with bladder involvement, with an intact sensory system. MRI of the spine was grossly normal. Cerebrospinal fluid analysis was performed to rule out infectious etiologies, so a clinical diagnosis of lupus myelitis was made. Initial pulse methylprednisolone showed no response, after which subsequent cyclophosphamide led to significant neurological improvement within one week. After the second cyclophosphamide dose, the patient developed a perinephric abscess requiring percutaneous drainage, leading to discontinuation of cyclophosphamide. She remained stable for six months but later developed recurrent headaches followed by generalized tonic–clonic seizures. Brain MRI was normal. She was managed with multiple antiepileptics along with repeat pulse methylprednisolone and cyclophosphamide.

Conclusion: This case highlights lupus myelitis and recurrent NPSLE as diagnostic and therapeutic challenges. Clinical judgment

remains crucial, and aggressive immunosuppression must be carefully balanced against serious infectious complications.

POM60: The clinical spectrum of Thrombotic Thrombocytopenic Purpura: The role of Early Plasma Exchange and Biological therapy in Refractory Disease

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Introduction: Thrombotic Thrombocytopenic Purpura (TTP) is a life-threatening thrombotic microangiopathy caused by severe ADAMTS13 deficiency (<10%) that leads to accumulation of ultra-large von Willebrand factor multimers, resulting in uncontrolled platelet aggregation and microthrombi formation, and in MAHA, thrombocytopenia, and variable organ ischemia, mostly neurologic, cardiac, or renal involvement.

Case Report: Case 1: A 46-year-old female presented with fever, slurred speech, and an acute right parietal infarct. Labs revealed microangiopathic hemolytic anemia (DCT positive), thrombocytopenia, and ADAMTS13 activity <10% (inhibitor negative). Despite strong SS-B and borderline SS-A positivity, she lacked clinical autoimmune features and achieved remission with daily plasma exchange (PLEX) and pulse dose of corticosteroids. Despite a positive DCT, negative inhibitor antibodies, and a clinical absence of autoimmune features, the sequence of events triggered by infection points towards primary TTP. Case 2: A 36-year-old female with SLE on immunosuppression presented with fever, menorrhagia, confusion and seizures. Labs confirmed TTP secondary to SLE with low ADAMTS13 and positive inhibitor antibodies, and MRI showed multiple parenchymal infarcts. Despite initiating standard PLEX and steroids, she had persistent thrombocytopenia, seizures and a drop in GCS. This refractory condition necessitated initiating Rituximab and Eculizumab to achieve clinical stabilisation and platelet recovery.

Conclusion: These cases highlight the clinical spectrum of TTP. Primary cases often respond to PLEX and steroids. Secondary SLE-associated TTP can be refractory and may require early escalation to biologic therapies such as Rituximab. While PLEX remains the gold standard, early recognition via the PLASMIC score and targeted biological therapies are essential for managing refractory TTP and improving survival.

POM61: Charting the Unseen: Insights into Non-Giant Cell Large Vessel Vasculitis

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Introduction: Large vessel vasculitis (LVV) is traditionally categorized into Giant cell arteritis and Takayasu's arteritis. However, involvement of the aorta and its major branches can be a rare manifestation or overlap feature in other rheumatological spectra, including ANCA-associated vasculitis and spondyloarthritis, often mimicking malignancy, or presenting as subclinical imaging findings.

Case Report: Case 1: A 63-year-old female presenting with weight loss and anorexia, whose CECT and PET CT showed an irregular, hypermetabolic mass in the left upper lobe with features highly suspicious for lung carcinoma,

with incidental features of large vessel vasculitis and 50% stenosis. Biopsy of the lung mass confirmed necrotising granulomatous inflammation and vasculitis with giant cells and fibrinoid necrosis, without malignancy or infection. Laboratory investigations showed elevated ESR and a positive Anti-MPO (by ELISA), supporting a diagnosis of Atypical GPA presenting as a pseudotumor with large-vessel vasculitis. Case 2: A 39-year-old female presented with chronic back and shoulder pain, anterior uveitis and bilateral mixed hearing loss. Despite an MRI of the Sacroiliac joints showing typical findings of active sacroiliitis with HLA-B27 positivity suggestive of spondyloarthritis, systemic involvement (weight loss, low-grade fever) warranted PET-CT, which revealed active large-vessel vasculitis. HLA-B51 and all other autoimmune workups were negative. Hence, she was started on oral glucocorticoids and JAK inhibitors to tackle both LVV and spondyloarthritis.

Conclusion: The phenotypic spectrum of Large vessel vasculitis can extend beyond traditional classifications. The first case demonstrates the diagnostic necessity of histopathology to differentiate GPA-associated pseudotumors from malignancy, even in the presence of LVV and second case underscores the importance of a high index of suspicion for vasculitis in SpA patients presenting with systemic features.

POM62: From Fingertip to Lung: Vasculitic Digital gangrene with NSIP- interstitial Lung disease in mixed connective tissue disease vasculitis

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Introduction: Mixed connective tissue disease (MCTD) is a rare systemic autoimmune disorder characterized by overlapping clinical features of systemic lupus erythematosus, systemic sclerosis, Rheumatoid arthritis and polymyositis, in association with high titers of anti-U1 ribonucleoprotein antibodies. Raynauld's phenomenon- most common presentation. Pulmonary hypertension is the most common cause of death.

Case Report: A 42-year-old female presented with blackish discolouration of left index finger tip for 2 months, which was insidious in onset, gradually progressive. No history of trauma, fever, dyspnoea. There was no loss of sensations and distal pulsations. She had h/o similar gangrene in right second toe three months ago. She was a known hypertensive, diabetic, hypothyroid patient for 7 yrs.The ANA profile showed RNP - strongly positive, Anti Ds- DNA, Beta-2 glycoprotein were negative. Lupus anticoagulant, C3, C4 were normal. PFT showed a restrictive pattern with low DLCO. Arterial Doppler of the left upper limb and right lower limb showed no stenosis or occlusion. HRCT chest showed ground glass opacities in bilateral lung parenchyma in posterior basal segments of bilateral lower lobes - suggestive of NSIP pattern. Pulse methylprednisolone therapy was given for 3 days. The patient was initiated on sildenafil, calcium channel blockers, along with Inj. Cyclophosphamide 500 mg once a month for six months. She was started on oral steroids at 0.5 mg /kg and the dose was tapered gradually, she responded.

Conclusion: Mixed connective tissue disease can present with atypical and severe manifestations, including vasculitic digital gangrene and

interstitial lung disease. Concomitant interstitial lung disease further signifies systemic involvement. Prompt recognition and early institution of immunosuppressive therapy are essential to limit disease progression, reduce morbidity due to pulmonary artery hypertension.

POM63: The Rash That Reached the Nerves: Dermatomyositis with Vasculitic Neuropathy

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Introduction: Dermatomyositis is an idiopathic inflammatory myopathy characterized by proximal muscle weakness and pathognomonic cutaneous manifestations; in rare cases, severe cutaneous vasculopathy and systemic vascular involvement may occur, presenting with complications such as digital ulceration and mononeuritis multiplex, reflecting small-vessel vasculitic overlap.

Case Report: A 27-year-old female initially presented with a pea-sized raised skin lesion; biopsy showed tumid lupus erythematosus, which improved with treatment. She later developed diffuse hair fall leading to non-scarring alopecia. Over the next three months, she experienced progressive proximal lower-limb weakness with hip tenderness, bilateral hand pain, and violaceous rash that progressed to painful ulcerative lesions over the metacarpophalangeal joints and separate similar lesions over the elbows. Examination revealed symmetric shoulder and hip girdle weakness with ulcerated Gottron-like lesions. Serum creatine kinase was mildly elevated and ANA was negative. Nerve conduction studies performed for persistent hand pain revealed mononeuritis multiplex suggestive of vasculitic neuropathy. Imaging showed no interstitial lung disease, and malignancy screening was negative. A diagnosis of dermatomyositis with vasculitic overlap was made. She was treated with high-dose corticosteroids and immunosuppressive therapy with gradual clinical improvement.

Conclusion: This case reminds us that dermatomyositis is not always skin deep or muscle bound. When rashes ulcerate and nerves protest, vasculitic overlap should be suspected. Early recognition of neurological clues and timely immunosuppression can prevent lasting deficits, urging clinicians to look beyond the obvious in seemingly classic dermatomyositis presentations.

Fig.1

POM64: Systemic lupus erythematosus presenting as subacute intestinal obstruction and seizures - a rare case report

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Introduction: SLE is a multisystem autoimmune disease with diverse clinical manifestations.

Although gastrointestinal involvement is common, severe presentations such as subacute intestinal obstruction are rare and diagnostically challenging. We report a rare case of SLE presenting as subacute intestinal obstruction, highlighting the importance of early recognition and immunosuppressive management.

Case Report: A 25-year-old woman presented with complaints of abdominal pain, recurrent non-bilious vomiting, diarrhea followed by constipation (last 4 days), and low-grade fever since one month. Examination revealed pallor, edema, ascites, pleural effusion, and hypotension. Abdominal imaging showed diffuse bowel wall edema with proximal dilatation and no mechanical transition point, consistent with sub-acute intestinal obstruction. Laboratory evaluation demonstrated anemia, thrombocytopenia, hypocomplementemia, markedly elevated anti–double-stranded DNA antibodies, and positive ANA with anti-Ro52 and anti–ribosomal P antibodies. Urinalysis revealed hematuria with nephrotic-range proteinuria, suggestive of lupus nephritis. Pleural and ascitic fluid analyses supported lupus serositis.in hospital, she developed seizures, CNS involvement of SLE was suspected, MRI brain demonstrated bilateral periventricular and subcortical white matter hyperintensities with microhemorrhages, consistent with SLE vasculopathy and neuropsychiatric systemic lupus erythematosus. The patient received pulse corticosteroids followed by mycophenolate mofetil and oral steroids, resulting in clinical improvement.

Conclusion: Systemic lupus erythematosus may rarely present as subacute intestinal obstruction, mimicking a surgical emergency. A high index of suspicion is essential in young patients with unexplained gastrointestinal obstruction and multisystem involvement. Early autoimmune evaluation and timely immunosuppressive therapy can prevent unnecessary surgery and improve outcomes.

POM65: Expect the Unexpected: Misleading Autoantibodies Complicating the Diagnosis of Polymyositis

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Introduction: Polymyositis is an idiopathic inflammatory myopathy characterized by clinical, laboratory investigations and supportive EMG, HPE findings. Anti SS-A and Anti SS-B are classically associated with Sjogren's Syndrome. However their presence in patients with myositis without clinical features suggestive of Sjogrens , poses a diagnostic dilemma.

Case Report: A 30-year-old woman presented with a 7-month history of progressive symmetrical proximal and distal muscle weakness associated with myalgia and inflammatory polyarthritis involving small and large joints in an additive pattern. Examination revealed proximal muscle tenderness, reduced muscle strength, petechial rashes, and bilateral pitting pedal edema. Antinuclear antibody testing was positive. Extractable nuclear antigen profile demonstrated anti-SSA (Ro) and anti-SSB (La) positivity. Despite serological findings, the patient had no sicca symptoms and lacked clinical evidence of Sjögren's syndrome. The coexistence of inflammatory myopathy, arthritis, and isolated anti-SSA/SSB positivity without glandular involvement posed a diagnostic dilemma, raising

the possibility of an overlap connective tissue disease. This case underscores the potential for autoantibodies to precede or occur independently of classical clinical syndromes. It highlights the importance of correlating serology with clinical features and maintaining vigilant longitudinal follow-up in patients with evolving systemic autoimmune disease.

Conclusion: Serological findings may precede or occur without typical clinical manifestations, and reliance on antibody profiles alone can result in diagnostic misinterpretation without careful clinical correlation.

POM66: Mixed Connective Tissue Disease with Interstitial Lung Disease with Unusual Cause of Respiratory failure

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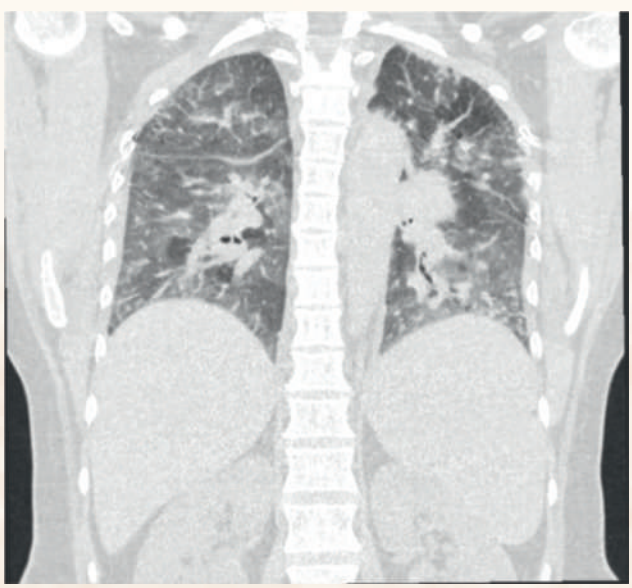
Introduction: Mixed connective tissue disease is a systemic autoimmune condition frequently associated with interstitial lung disease. Respiratory compromise is usually attributed to pulmonary involvement; however, overlapping neuromuscular disorders such as myasthenia gravis may contribute significantly and remain underrecognized.

Case Report: A 43-year-old woman initially presented with fatiguable limb weakness, bilateral ptosis and bulbar symptoms. She was diagnosed with acetylcholine receptor antibody–positive myasthenia gravis based on clinical features and a decremental response on repetitive nerve stimulation. Extended autoimmune evaluation demonstrated nRNP/Sm, Ro-52, and Scl-70 positivity, supporting a diagnosis of early mixed connective tissue disease. She responded well to intravenous immunoglobulin and immunosuppressive therapy. One year later, she developed recurrent generalised weakness and progressive exertional dyspnea. Imaging revealed progression of NSIP-pattern ILD with new infective changes. Despite significant pulmonary involvement, relapse of myasthenia gravis was identified as a major contributor to respiratory failure. Treatment with rescue IVIG, corticosteroids, and escalation of mycophenolate mofetil led to substantial clinical recovery.

Conclusion: Respiratory deterioration in mixed connective tissue disease is often attributed to progressive interstitial lung disease; however, this case demonstrates that neuromuscular relapse can be an important and reversible contributor. Awareness of myasthenia gravis exacerbation in patients with autoimmune overlap syndromes is essential to prevent delays in diagnosis and optimize therapeutic outcomes.

Fig.1



POM67: Neuropsychiatric Lupus Mimicking Tubercular Meningitis: A Diagnostic Dilemma

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Introduction: Neuropsychiatric systemic lupus erythematosus (NPSLE) presents with a wide spectrum of neurological and psychiatric manifestations and can closely mimic chronic central nervous system infections. In tuberculosis-endemic regions, this overlap often leads to misdiagnosis, delayed immunosuppression, and potentially irreversible neurological damage.

Case Report: A 30-year-old female presented with a 5–6 year history of symmetrical inflammatory polyarthralgia and a past history of healed hyperpigmented rash. Over six months, she developed recurrent episodes of headache, vomiting, dizziness, diplopia, and intermittent altered sensorium, leading to multiple hospital admissions where she was repeatedly diagnosed and treated as tubercular meningitis with antitubercular therapy and steroids, resulting in only transient improvement. One month prior to admission, she developed behavioral abnormalities with preserved memory, followed by sudden-onset right-sided hemiparesis, slurring of speech, gaze deviation, and altered sensorium. Neurological examination suggested multifocal CNS involvement. Fundus examination revealed disc edema with periarterial sheathing. Autoimmune workup was suggestive of systemic lupus erythematosus. Neuroimaging demonstrated multifocal vasculitic infarcts, confirming a diagnosis of neuropsychiatric SLE with CNS vasculitis.

Conclusion: Neuropsychiatric SLE should be strongly considered in patients with recurrent aseptic meningitis-like presentations, fluctuating neurological deficits, and multisystem involvement, especially in tuberculosis-endemic areas. Early diagnosis and prompt immunosuppressive therapy are crucial to prevent permanent neurological sequelae and improve patient outcomes.

POM68: Fever, Ferritin, and Failure to Improve: Think MAS—A Case of Systemic-Onset JIA with Infection-Triggered Secondary HLH

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Introduction: Systemic-onset Juvenile Idiopathic Arthritis (sJIA) is an autoinflammatory disorder characterized by fever, rash, and systemic inflammation. Macrophage activation syndrome, a severe form of secondary hemophagocytic lymphohistiocytosis (HLH), is a potentially fatal complication, frequently precipitated by infections and requiring early recognition and prompt management.

Case Report: A 15-year-old female, a known case of systemic-onset Juvenile Idiopathic Arthritis, presented with high-grade fever for 15 days(103°F), accompanied by a salmon-colored evanescent maculopapular rash over trunk and face. She developed acute onset painful facial swelling with puffiness, dysphagia, drooling of saliva, and breathlessness over five days. Examination revealed diffuse tender facial edema, generalized lymphadenopathy, and splenomegaly. Laboratory evaluation showed severe anemia (hemoglobin 4 g/dL),

thrombocytopenia (platelets 26,000/mm³), elevated transaminases (SGOT 100 U/L, SGPT 66 U/L), hypoproteinemia, raised inflammatory markers (CRP 36.7 mg/L), hyperferritinemia (>3000 ng/mL), and elevated procalcitonin (50 ng/mL), triglyceride-132 mg/dL. PRINTO criteria for MAS in JIA was fulfilled based on fever, hyperferritinemia, cytopenias, elevated liver enzymes, and systemic inflammation. Blood cultures grew Pseudomonas aeruginosa, confirming infection-triggered secondary HLH. The patient was treated with intravenous broad-spectrum antibiotics, high-dose corticosteroids, and immunosuppressive therapy, with subsequent clinical and biochemical improvement.

Conclusion: This case illustrates the complexity of diagnosing macrophage activation syndrome in sJIA, particularly when triggered by severe bacterial infection. Early application of PRINTO criteria and timely initiation of combined antimicrobial and immunosuppressive therapy are essential to improve outcomes.

Fig.1

POM69: Atypical Systemic Sarcoidosis Unmasked Following Severe Sepsis: A Diagnostic Challenge.

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Introduction: Sarcoidosis is a systemic inflammatory disease of unknown etiology characterized by non-necrotizing granulomatous inflammation, most commonly involving the lungs. Late-onset sarcoidosis with predominant extrapulmonary manifestations is uncommon and may present a diagnostic challenge, particularly when masked by severe infection and systemic inflammation.

Case Report: 67-year-old man with T2DM,HTN,CKD disease presented with severe sepsis due to MDR Klebsiella pneumoniae pneumonia, complicated by candidemia and multiorgan dysfunction. He initially stabilised after prolonged culture-directed antimicrobial and antifungal therapy but later developed persistent systemic inflammation with recurrent high-grade fever, inflammatory polyarthritis, and cardiopulmonary symptoms. Laboratory evaluation showed markedly elevated inflammatory markers (ESR 120 mm/hr, CRP 220 mg/L). Microbiological evaluation including sputum testing for tuberculosis, along with vasculitis and autoimmune work-up, was unremarkable. Whole-body PET–CT was performed, demonstrating metabolically active bilateral hilar and mediastinal lymphadenopathy, inflammatory pericardial thickening with mild effusion, bilateral shoulder synovitis, and reactive splenic and bone marrow uptake, without pulmonary parenchymal disease or malignancy. A

diagnosis of atypical extrapulmonary systemic sarcoidosis was suspected and Serum ACE was sent and was elevated. IGg4 was normal.The patient was initiated on oral corticosteroids ,azathioprine and on follow up showed resolution of inflammatory markers.

Conclusion: Late-onset sarcoidosis may present with predominant extrapulmonary involvement and severe inflammatory responses, particularly following severe sepsis. Persistent inflammation despite infection control should prompt consideration of alternative inflammatory etiologies. FDG PET–CT is a valuable tool for identifying multisystem involvement and guiding diagnosis and management in complex cases.

POM70: Disease progression versus opportunistic infections- An important clinical dilemma in ILD patients

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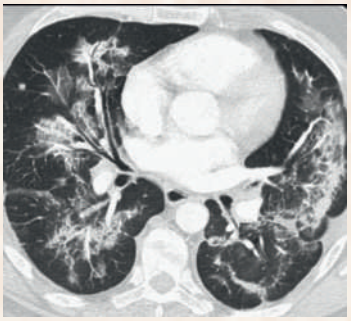
Introduction: Anti-synthetase syndrome is an autoimmune inflammatory myopathy associated with anti–aminoacyl tRNA synthetase antibodies (most commonly anti-Jo-1) and systemic manifestations such as interstitial lung disease (ILD), arthritis, Raynaud phenomenon, and "mechanic's hands." ILD frequently necessitates prolonged immunosuppression. Distinguishing ILD progression from superimposed infection is important

Case Report: A patient with anti Jo-1 positive anti-synthetase syndrome with fibrotic ILD -NSIP pattern and subclinical myositis, on mycophenolate mofetil and prednisolone, presented to the OPD in November 2025 with progressive dyspnea. Pulmonary function tests showed worsening, while HRCT thorax demonstrated fibrotic ILD–NSIP pattern. In view of symptomatic worsening, she received the first dose of rituximab. Twelve days later, she re-presented with worsening breathlessness, was tachypneic and hypoxemic (SpO_2 86% on room air), and developed type II respiratory failure ($PaCO_2$ 70 mmHg), requiring intubation. She was treated as community-acquired pneumonia with piperacillin–tazobactam, doxycycline, oseltamivir, and steroids; blood cultures grew Klebsiella pneumoniae sensitive to piperacillin–tazobactam. She received IVIG for ILD but remained ventilator dependent. Endotracheal aspirate BioFire was positive for CMV and parainfluenza; PCP pneumonia was excluded. Blood CMV PCR was negative, but endotracheal aspirate CMV PCR (~16,000 copies) supported localized CMV pneumonitis. Repeat CECT thorax showed stable ILD with new ground-glass opacities. Valganciclovir 900 mg BD, steroids led to improvement.

Conclusion: Opportunistic infections complicate immunosuppressed patients, particularly CMV and Pneumocystis. CMV pneumonitis in anti-synthetase syndrome, especially after rituximab, is difficult to diagnose. Disease may be confined to the lower respiratory tract with negative blood PCR despite high airway viral load. Early suspicion, diagnostics, imaging review, and prompt antiviral therapy improve outcomes.

Fig.1



POM71: From Menorrhagia to Renal: An Unusual Presentation of Mixed Connective Tissue Disease with Class V Lupus Nephritis Overlap With Isolated Lupus Anticoagulant Positivity

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Introduction: Mixed connective tissue disease is an autoimmune overlap syndrome characterized by anti-U1-snRNP positivity. Significant renal involvement with isolated lupus anticoagulant positivity is uncommon and poses diagnostic and therapeutic challenges.

Case Report: A 21-year-old female presented with menorrhagia, photosensitive rash, periorbital puffiness and bilateral pedal edema, evaluation revealed proteinuria (25 g/day), prolonged aPTT and lupus anticoagulant positivity. Autoimmune profile showed ANA 1:1280 and anti-U1-snRNP strongly positive, imaging excluded thrombosis, ultrasound-guided renal biopsy confirmed class v lupus nephritis, pulse methylprednisolone followed by oral steroids and immunosuppressive therapy initiated.

Conclusion: Overlap connective tissue disease can present with membranous lupus nephritis and isolated lupus anticoagulant positivity. A structured diagnostic approach and early biopsy-guided therapy are crucial to prevent renal progression and thrombotic events.

POM72: Beyond Sicca – Systemic Extraglandular Manifestations Revealing Primary Sjogren's Syndrome – A Case Series

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Introduction: Primary Sjögren's syndrome is a systemic autoimmune disorder classically associated with sicca symptoms. However, extraglandular manifestations may precede glandular involvement and significantly influence disease activity, management, and prognosis. Early recognition of systemic presentations is therefore crucial for timely diagnosis and appropriate immunomodulatory therapy.

Case Report: We report a four-case series highlighting varied extraglandular manifestations of primary Sjögren's syndrome. Case 1 was a middle-aged female presenting with acute paraparesis and bowel–bladder involvement; MRI spine revealed longitudinally extensive transverse myelitis, with AQP4 and MOG negativity, anti-SSA positivity, and confirmatory labial biopsy. Case 2 was a young female with petechiae and recurrent epistaxis; investigations showed immune thrombocytopenia with positive anti-SSA/SSB antibodies and Schirmer's test, managed with steroids and rituximab. Case 3 involved a young female with palpable purpura; ANA showed fine speckled pattern and skin biopsy confirmed leukocytoclastic vasculitis secondary to Sjögren's syndrome. Case 4 was a young male presenting with acute quadriparesis due to hypokalemic metabolic alkalosis; urinary electrolyte analysis supported Bartter syndrome secondary to Sjögren's syndrome. All cases responded to organ-specific supportive therapy and immunosuppression.

Conclusion: Primary Sjögren's syndrome may initially manifest with diverse systemic

involvement affecting neurological, hematological, cutaneous, and renal systems. Awareness of such presentations enables early diagnosis and appropriate management. Routine screening for extraglandular features is essential to reduce morbidity and improve long-term outcomes.

Fig.1

POM73: Systemic Lupus Erythematosus Presenting with UIP-Pattern Interstitial Lung Disease and Severe Acute Cutaneous Lupus — A Diagnostic Evolution

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Introduction: Interstitial lung disease with a usual interstitial pneumonia pattern is rare in systemic lupus erythematosus and may mimic rheumatoid arthritis–associated ILD. Low-titer ANA with a negative profile can delay diagnosis. Predominant pleuropulmonary and evolving cutaneous features underscore the heterogeneous, dynamic nature of SLE and the importance of longitudinal clinical evaluation.

Case Report: We report a 44-year-old woman who presented with an 8-month history of bilateral shoulder pain, progressive exertional dyspnea, persistent dry cough, and reduced exercise tolerance. Contrast-enhanced CT of the chest demonstrated symmetrically distributed interstitial lung disease with tubular traction bronchiectasis, consistent with a usual interstitial pneumonia (UIP) pattern. Three months later, she developed recurrent oral ulcers, non-scarring alopecia, and intermittent fever. Antinuclear antibody by immunofluorescence was low-positive (2+), with a negative ANA profile and low complement C3 levels. Rheumatoid factor was positive, whereas anti-cyclic citrullinated peptide antibodies were negative, raising initial suspicion of rheumatoid arthritis–associated ILD. Subsequently, she developed a malar rash and photosensitive lesions over sun-exposed areas, which rapidly progressed to acute cutaneous lupus with erosions and desquamation. Skin biopsy confirmed acute cutaneous lupus erythematosus. This case highlights the diagnostic complexity of evolving connective tissue disease presenting with UIP-pattern ILD and underscores the importance of immunological reassessment.

Conclusion: This case demonstrates that systemic lupus erythematosus may initially manifest with a usual interstitial pneumonia–pattern interstitial lung disease, preceding classical systemic and cutaneous features. Vigilant longitudinal assessment and early recognition of evolving autoimmune manifestations are essential to facilitate timely immunosuppressive therapy and prevent irreversible organ damage.

POM74: Systemic Lupus Erythematosus Presenting with UIP-Pattern Interstitial Lung Disease and Severe Acute Cutaneous Lupus — A Diagnostic Evolution

Sonalika Sharad Rathore, Sanjay Dubey, Vaibhav Yadav, A D Bhatnagar
Department of Medicine, MY Hospital and MGMMC Indore

Introduction: Interstitial lung disease with a usual interstitial pneumonia pattern is rare in systemic lupus erythematosus and may mimic rheumatoid arthritis–associated ILD. Low-titer ANA with a negative profile can delay diagnosis. Predominant pleuropulmonary and evolving cutaneous features underscore the heterogeneous, dynamic nature of SLE and the importance of longitudinal clinical evaluation.

Case Report: We report a 44-year-old woman who presented with an 8-month history of bilateral shoulder pain, progressive exertional dyspnea, persistent dry cough, and reduced exercise tolerance. Contrast-enhanced CT of the chest demonstrated symmetrically distributed interstitial lung disease with tubular traction bronchiectasis, consistent with a usual interstitial pneumonia (UIP) pattern. Three months later, she developed recurrent oral ulcers, non-scarring alopecia, and intermittent fever. Antinuclear antibody by immunofluorescence was low-positive (2+), with a negative ANA profile and low complement C3 levels. Rheumatoid factor was positive, whereas anti-cyclic citrullinated peptide antibodies were negative, raising initial suspicion of rheumatoid arthritis–associated ILD. Subsequently, she developed a malar rash and photosensitive lesions over sun-exposed areas, which rapidly progressed to acute cutaneous lupus with erosions and desquamation. Skin biopsy confirmed acute cutaneous lupus erythematosus. This case highlights the diagnostic complexity of evolving connective tissue disease presenting with UIP-pattern ILD and underscores the importance of longitudinal immunological reassessment.

Conclusion: This case demonstrates that systemic lupus erythematosus may initially manifest with a usual interstitial pneumonia–pattern interstitial lung disease, preceding classical systemic and cutaneous features. Vigilant longitudinal assessment and early recognition of evolving autoimmune manifestations are essential to facilitate timely immunosuppressive therapy and prevent irreversible organ damage.

POM75: Isolated Interstitial Lung Disease Unmasking Antisynthetase Syndrome: A Diagnostic Challenge

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Introduction: Antisynthetase syndrome (ASS) is a rare, chronic autoimmune disorder,characterized by Interstitial lung disease (ILD), non erosive arthritis, inflammatory myositis, Raynaud's phenomenon, mechanics hands in the setting of antibodies against amino acyl-transfer ribonucleic acid synthetases, with anti Jo-1 antibody being the most common.

Case Report: A 58 year old male with no previous comorbidities presented with complaints of exertional dyspnea,non-productive cough,easy fatigability and anorexia for 1 month.There was no history of fever,chest pain,palpitations,joint pain,skin rash or myalgia.On physical examination , patient was afebrile,oxygen saturation of 94%(on room air) and blood pressure of 110/70 mmHg.Auscultation of chest revealed basal end inspiratory crepitations. Cardiovascular,

abdominal and neurological system examinations were unremarkable. Routine investigations showed:Hb 11.1 g/dl,WBC 7,380 /cmm (N78,L15), PLT 3.4 L,,Bilirubin(total/direct) 0.955/0.22, albumin 3.18,,total protein 6.96,AST 162 ,ALT 45.9 ,ALP 58.2 ,serum calcium 8.8, ESR 46 ,CRP 42.42 CPK 196,LDH 320 ,triple serology negative,ABG suggestive of type 1 respiratory failure, ECG and Echocardiogram was normal,USG W/A normal, chest xray showed Bilateral reticular opacities. HRCT thorax showed diffuse interstitial fibrosis of both lungs suggestive of probable fibrotic NSIP pattern.On further evaluation to determine the underlying cause of ILD, myositis profile came out as strongly positive for anti Jo-1 antibody.

Conclusion: Antisynthetase syndrome's clinical manifestations can take many different forms and aren't always evident when the disease first manifests itself. The relevance of suspecting ASS in individuals presenting with unexplained ILD is emphasized in this case. Interstitial lung disease is a frequent occurrence and is associated with a bad prognosis during the course of antisynthetase syndrome.

Fig.2

POM76: Interesting case of sjogren syndrome associated inflammatory myositis

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Introduction: Inflammatory myopathies associated with connective tissue diseases pose significant diagnostic challenges due to overlapping clinical features and heterogeneous immunological profiles. Sjögren's syndrome–associated myositis is an uncommon entity and often requires a high index of suspicion.

Case Report: We report a 47-year-old female who presented with a 3-month history of insidious, progressive, symmetrical proximal more than distal weakness involving both upper and lower limbs, associated with neck flexor weakness and myalgia. There was no sensory, cranial nerve, bulbar, or sphincter involvement. She had significant weight loss, generalized muscle wasting, xerostomia, xerophthalmia, and Raynaud's phenomenon. Examination revealed hypotonia with proximal muscle weakness and preserved reflexes. Laboratory evaluation showed markedly elevated CPK (>4267 U/L), LDH, and transaminases. Autoimmune workup revealed ANA positivity (1:640, cytoplasmic speckled pattern) with positive anti-SSA (Ro52 and Ro60) antibodies; myositis-specific antibodies were negative. EMG and imaging supported a myopathic process. Muscle biopsy could not be performed due to lack of consent.

Conclusion: Based on clinical, biochemical, and immunological findings, a diagnosis of Sjögren's syndrome–associated inflammatory myositis was made. The patient showed clinical improvement with immunosuppressive therapy, highlighting the importance of considering overlap myositis in patients with systemic autoimmune features.

POM77: When Ku Antibodies Unmask an Inflammatory Myopathy: A Rare Diagnostic Clue

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Introduction: Anti-Ku antibody–associated inflammatory myopathy is a rare autoimmune muscle disorder, most often described in overlap syndromes involving systemic sclerosis or lupus, and infrequently reported as an isolated myositis. Owing to its rarity and heterogeneous presentation, diagnosis is often delayed or overlooked. Recognition of this entity is important, as it represents distinct immunologic subset.

Case Report: A 32-year-old female presented with progressive weakness of all four limbs for two weeks, predominantly involving proximal muscles. She reported difficulty in rising from a sitting position and combing her hair, along with associated myalgia. There was no history of trauma, drug intake, thyroid dysfunction, or systemic illness. On examination, muscle power was graded 4/5 in bilateral hip flexors, extensors, abductors, and adductors, as well as elbow flexors and extensors. Muscle tone was normal. Deep tendon reflexes were diminished, and plantar responses were bilaterally flexor. Sensory examination and cranial nerve assessment were unremarkable. Laboratory investigations revealed a markedly elevated serum creatine phosphokinase (CPK) level of 4355 IU/L. Electromyography demonstrated features consistent with proximal myopathy. Myositis antibody panel showed isolated anti-Ku antibody positivity. Muscle biopsy revealed inflammatory myopathy characterized by inflammatory cell infiltrates, confirming the diagnosis.

Conclusion: This case describes a rare anti-Ku antibody–positive inflammatory myopathy presenting with subacute proximal muscle weakness without connective tissue disease overlap. Recognition of this entity using serology, electromyography, and muscle biopsy is crucial for early diagnosis. Reporting such cases improves awareness of this uncommon subset of inflammatory myopathies.

POM78: Not all UIP is IPF: A tale of Amyopathic Inflammatory Myositis

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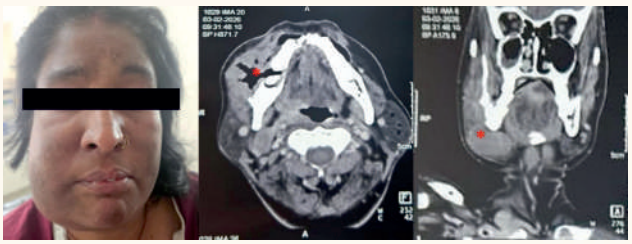
Introduction: Idiopathic pulmonary fibrosis (IPF) is a progressive fibrotic disease of the lung with unsatisfactory management options. However, some forms of interstitial lung disease (ILD) are being diagnosed as ILD with autoimmune features or CTD-ILD. Here, we describe a case of ILD that was managed as IPF but was later diagnosed as amyopathic inflammatory myositis, specifically anti-synthetase syndrome (ASS).

Case Report: A 47-year-old non-smoking man presented with 18 months of slowly progressive exertional dyspnoea and cough. He had been managed as IPF-UIP by a pulmonologist. On further assessment, he did not have Raynaud's, arthritis, rash, fever, or muscle weakness. Examination revealed grade III clubbing and bibasal Velcro crackles. CRP was elevated at 43.88 mg/L; Rheumatoid factor, anti-CCP, ANA, and ENA were negative. CPK and LDH levels were normal. Spirometry showed a restrictive pattern (FVC 1.57 L, 45% predicted). HRCT thorax demonstrated diffuse lower-lobe honeycombing with traction bronchiectasis and

ground-glass opacities, consistent with predominant UIP. He was tested positive for anti-PL-12 after extensive workup. He was treated as ASS with corticosteroids and pulse cyclophosphamide. However, after three cycles, he developed an acute ILD exacerbation with radiological progression. Rituximab was initiated, followed by MMF. He improved significantly both clinically and radiologically (FVC 1.92 L, 52% predicted). He is currently on MMF with antifibrotic therapy and remains in a stable, non-progressive phase.

Conclusion: All UIP-predominant ILD is not IPF. All idiopathic ILD should be evaluated for features of CTD or inflammatory myositis. Inflammatory myositis such as ASyS can present as isolated ILD with normal muscle enzymes, and myositis-specific antibodies can aid in diagnosis.

Fig.1



POM79: Late-Onset Malignancy in Anti-Mi-2 Positive Dermatomyositis: A Case Report

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Introduction: Dermatomyositis (DM) carries a 5–6-fold increased malignancy risk, highest within the first year after diagnosis. While myositis-specific antibodies such as anti-TIF1γ and NXP2 have higher risk, anti-Mi-2 positivity is associated with a favourable prognosis and low malignancy risk, with some studies reporting no risk. We report a late malignancy presentation in anti-Mi-2 positive DM.

Case Report: A 38-year-old female presented in November 2023 with subacute proximal muscle weakness and polyarthralgia. Investigations revealed CPK 2000 U/L, LDH 1807 U/L, AST/ALT 429/145 U/L, ANA positivity, and strong anti-Mi-2 antibody positivity. She was treated with glucocorticoids and IVIG, discharged on methotrexate, and subsequently received rituximab in January 2024 for refractory weakness. Malignancy screening at that time was negative. She remained well until June 2024, when she discontinued medications. She re-presented in January 2025 with disease flare (CPK 8998 U/L) and was treated with steroids, rituximab, and mycophenolate mofetil along with methotrexate, with good response. After again defaulting follow-up and stopping treatment, she presented in January 2026 with progressive right jaw swelling and proximal weakness. CPK was 13,119 U/L. She is currently receiving steroids and IVIG. CT showed tumour in the gingivobuccal sulcus eroding the mandible. Biopsy report is awaited.

Conclusion: This case highlights that malignancy risk in dermatomyositis may persist beyond the first year, even in anti-Mi-2 positive patients. Recurrent disease flares and interrupted treatment may delay malignancy detection. This shows the need for strict follow up, and increased watchfulness for malignancy even in Mi2+ cases.

POM80: Hypokalemic Quadriparesis as Initial Presentation of Primary Sjögren's Syndrome: A Case Report from Odisha, India

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Introduction: Primary Sjögren's syndrome is a rare systemic autoimmune disease (prevalence of 0.5-1%) predominantly affecting females (9:1). Extra-glandular involvement occurs in 30–40% of PSS patients, with renal involvement in 16–17%, and with distal RTA in 4.7–9%. We report a young female with acute onset quadriparesis due to severe hypokalemia as the initial manifestation in of primary Sjögren's syndrome.

Case Report: A 30-year-old female presented with acute onset flaccid quadriparesis of 3 days duration. The weakness was progressive, predominantly proximal and No cranial nerve involvement. Examination revealed MRC grade 2/5 power in all limbs with decreased tone in all limbs, preserved reflexes and flexor plantar responses and no sensory involvement with normal bowel and bladder movement. Laboratory evaluation showed severe hypokalemia (sr K+ 1.3 mmol/L). Arterial blood gas demonstrated normal anion gap metabolic acidosis with respiratory compensation (pH 7.37). Serum sodium, calcium, magnesium, renal and liver function tests were normal. Nerve conduction study excluded axonal neuropathy. The combination of hypokalemia and metabolic acidosis suggested distal renal tubular acidosis. Autoimmune work-up revealed strongly positive ANA with anti-Ro (SSA) and anti-La (SSB) antibodies. On further history, the patient reported intermittent dry eyes and dental caries with dry mouth. A diagnosis of primary Sjögren's syndrome presenting as distal renal tubular acidosis with hypokalemic quadriparesis was established.

Conclusion: Primary Sjögren's syndrome may initially present with life-threatening hypokalemic paralysis due to distal renal tubular acidosis before classical sicca symptoms appear. Early recognition of metabolic acidosis with hypokalemia and targeted autoimmune evaluation is crucial, as prompt correction is lifesaving and prevents diagnostic delay in young patients with unexplained quadriparesis.

POM81: The Myth of Amyopathy: Painful Inflammatory Myositis in Anti–MDA5 Dermatomyositis

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Introduction: Anti-MDA5 antibody–associated dermatomyositis is traditionally regarded as a form of clinically amyopathic dermatomyositis with minimal muscle involvement, normal creatine kinase levels, and predominant cutaneous and pulmonary manifestations. Presence of muscle pain or inflammatory myositis represents an atypical presentation and may lead to diagnostic delay if unrecognized.

Case Report: A 33-year-old male presented with a 3-month history of progressive, symmetrical proximal muscle weakness associated with significant muscle pain, difficulty in ambulation, overhead activities, and rising from a seated position. He also reported inflammatory back pain, polyarthralgia, intermittent fever, facial and thigh rashes, and painful ulcers over both elbows

and the right little finger. Examination revealed reduced proximal muscle power (3/5 in thigh flexion and adduction) with preserved distal strength and reflexes. Despite clinically evident myopathy and muscle tenderness, serum creatine kinase was normal (140 U/L). Autoimmune workup showed positivity for anti-MDA5 and anti-Ro52 antibodies, with negative ANA by IFA and normal complement levels. MRI of bilateral thighs demonstrated patchy STIR hyperintensities suggestive of active inflammatory myositis. NCCT chest revealed bilateral lower-lobe consolidations, ground-glass opacities, and fibroparenchymal changes indicating early interstitial lung disease. After excluding infectious, metabolic, and neuromuscular junction disorders, a diagnosis of anti-MDA5–associated dermatomyositis with painful inflammatory myositis was made.

Conclusion: This case underscores that anti–MDA5 dermatomyositis is not uniformly amyopathic. Painful inflammatory myositis with preserved creatine kinase levels represents an underrecognized phenotype. Reliance solely on muscle enzyme levels may be misleading; comprehensive antibody profiling and muscle MRI are essential for timely diagnosis and management, particularly given risk of pulmonary involvement.

POM82: Paediatric Systemic Lupus Erythematosus presenting as acute pancreatitis and hypertriglyceridemia

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Introduction: Systemic lupus erythematosus is a multisystem autoimmune disorder with diverse clinical manifestations. Gastrointestinal involvement is uncommon, and acute pancreatitis as the initial presentation—especially in pediatric patients—is rare. We report a case of pediatric-onset SLE presenting with acute pancreatitis, posing a diagnostic challenge.

Case Report: A 13-year-old girl with no prior comorbidities presented with severe epigastric pain radiating to the back, worsened with food intake and relieved on bending forward. She had a two-month history of low-grade evening fever, myalgia, and polyarthralgia. Examination revealed pallor, synovitis, and epigastric tenderness with guarding and reduced bowel sounds. Investigations showed severe anemia, thrombocytopenia, markedly elevated serum amylase and lipase, and hypertriglyceridemia, with normal liver and renal function. Immunological evaluation revealed high-titer ANA, low complement levels, positive anti-dsDNA, anti-Sm, anti-U1-snRNP, and a positive direct Coombs test. Contrast-enhanced CT abdomen demonstrated a bulky pancreas with peripancreatic fluid collections, consistent with moderate acute pancreatitis (CT severity index 6/10). After exclusion of common etiologies, a diagnosis of SLE-related acute pancreatitis was made. The patient was managed conservatively for pancreatitis and initiated on corticosteroids and rituximab, with gradual clinical improvement.

Conclusion: Lupus pancreatitis is the second most common cause of SLE-related acute abdomen. This atypical and life-threatening presentation of pSLE encouraged us to report this case so that awareness can be created and early management may be ensured

POM83: When Infections Ignite Autoimmunity: A Case Series of Macrophage Activation Syndrome

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Introduction: Macrophage Activation Syndrome (MAS) is a life-threatening hyperinflammatory state associated with autoimmune diseases and infections. Its heterogeneous presentation often delays diagnosis. We present a case series highlighting diverse autoimmune backgrounds and infectious triggers, emphasizing the importance of early recognition and prompt immunosuppressive therapy.

Case Report: This case series includes three patients with autoimmune diseases who developed MAS secondary to infections. The first was a patient with polymyositis presenting with prolonged high-grade fever, hepatosplenomegaly, lymphadenopathy, and pancytopenia, triggered by enteric fever. The second case involved a young female with newly diagnosed systemic lupus erythematosus who presented with fever, mucocutaneous ulcers, cytopenias, severe transaminitis, and extreme hyperferritinemia, precipitated by hepatitis C infection. The third case was a patient with mixed connective tissue disease presenting with fever, petechial rash, Raynaud's phenomenon, and hepatosplenomegaly following dengue infection. All patients demonstrated hallmark laboratory features of MAS including cytopenias, hyperferritinemia, hypertriglyceridemia, and hypofibrinogenemia, with bone marrow evidence of hemophagocytosis in selected cases. Each fulfilled diagnostic criteria for MAS/secondary HLH. Management included prompt initiation of high-dose corticosteroids along with targeted antimicrobial therapy and supportive care, resulting in favorable clinical and hematological outcomes.

Conclusion: MAS represents a common inflammatory endpoint in diverse autoimmune diseases when triggered by infections. High clinical suspicion, early application of diagnostic criteria, and simultaneous treatment of both immune dysregulation and underlying infection are critical to improving survival and outcomes.

Fig.1

POM84: When Pneumonia isn't Pneumonia: A Case of Rapidly Progressive ILD Revealing Anti Jo-1 Dermatomyositis

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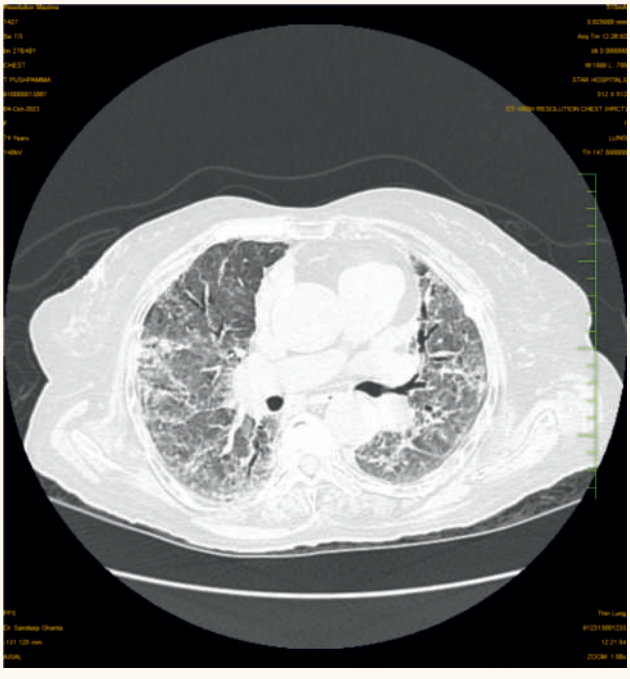
Introduction: Anti Jo-1 Dermatomyositis is an autoimmune inflammatory disorder that is characterised by proximal muscle weakness. Extra muscular manifestations, particularly

pulmonary involvement might dominate the underlying diagnosis, delaying appropriate immunosuppressive treatment leading to substantial morbidity and mortality.

Case Report: A 74-year-old woman presented with two months of progressive exertional dyspnoea and productive cough, with four weeks of worsening proximal upper and lower limb weakness. She was initially treated for presumed pneumonia after hypoxaemia and abnormal Chest Xray findings and was referred for further care. On arrival, she was tachypnoeic, hypoxic (SpO_2 85%) and bedbound from severe weakness. Investigations showed leucocytosis (30,000/mm^3), elevated C-reactive protein (18.6 mg/dL) and bilateral mid- and lower-zone opacities on Chest Xray. Despite broad-spectrum antibiotics and non-invasive ventilation, symptoms persisted. Refractory hypoxaemia, facial pigmentation, and progressive proximal weakness prompted myositis antibody testing, revealing anti–Jo-1 and anti–Ro-52 positivity consistent with dermatomyositis-associated ILD. After infection was excluded, pulse-dose corticosteroids were initiated. HRCT confirmed ILD without consolidation. She received intravenous cyclophosphamide followed by rituximab which led to marked improvement in oxygenation and muscle strength, regaining independent ambulation at discharge with home oxygen after appropriate vaccinations.

Conclusion: Anti–Jo-1 positive dermatomyositis may present primarily with interstitial lung disease frequently leading to hospitalization and can often be misdiagnosed as pneumonia. Anti–Jo-1 antibodies strongly predict ILD risk. Prompt antibody testing and immunosuppressive escalation can reduce respiratory symptoms and restore functional mobility, even in patients with marked impairment.

Fig.1



POM85: Large-Vessel Vasculitis Unmasked by Bedside Examination: A Case of Takayasu Arteritis

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Introduction: Takayasu arteritis is a rare, chronic, large-vessel vasculitis predominantly affecting young and middle-aged women. It involves granulomatous inflammation of the aorta and its major branches, resulting in vessel wall thickening, stenosis or occlusion. Clinical manifestations are diverse and nonspecific, often leading to delayed diagnosis. Bedside examination may provide crucial diagnostic clues.

Case Report: A case study of a 42-year-old female presenting with acute febrile illness,

burning micturition, and a syncopal episode. Systematic clinical evaluation, detailed systemic examination, and radiological imaging were performed. Differential diagnoses including urinary tract infection with septicemia, thoracic outlet syndrome, and subclavian artery atherosclerosis were considered and excluded. On examination, right upper limb pulses were absent with a marked inter-arm blood pressure difference (right arm systolic 60 mmHg vs. left arm 98/70 mmHg). A right subclavian bruit was audible. CT aortogram and angiography revealed multifocal narrowing and wall thickening of the right subclavian and left axillary arteries, consistent with large-vessel vasculitis. Based on clinical and radiological correlation, Takayasu arteritis was diagnosed.

Conclusion: Routine bedside checks (pulse, inter-arm BP) are vital in syncope or shock. In women with bruits, or BP asymmetry, suspect Takayasu arteritis—early imaging and immunosuppression prevent irreversible vascular damage.

POM86: Beyond Erosions: A Rare Osteophytic Pattern in Chronic Seropositive Rheumatoid Arthritis with Secondary Osteoarthritis

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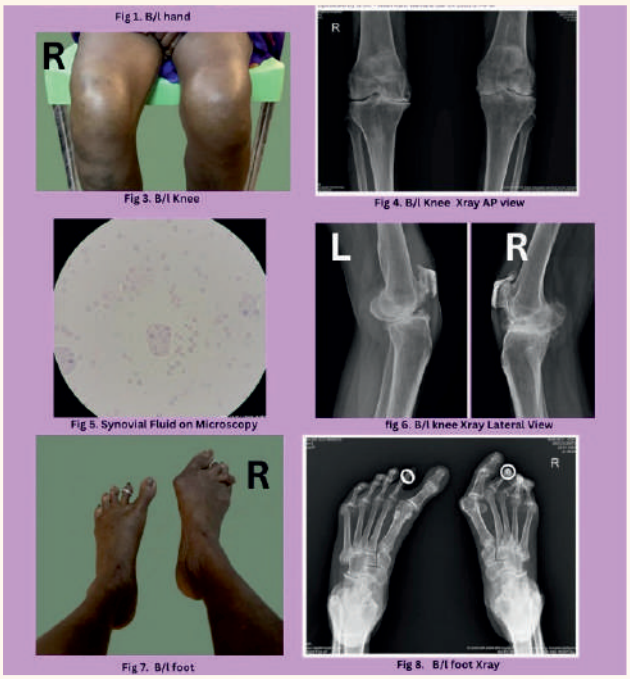
Introduction: Rheumatoid arthritis (RA) is a chronic inflammatory arthropathy characterized by symmetric polyarthritis and progressive joint damage. Typical radiographic findings include erosions and joint space narrowing. The presence of hooked osteophyte-like deformities is uncommon in RA and more frequently associated with metabolic or degenerative arthropathies.

Case Report: A 55-year-old female, a known case of seropositive RA for six years with poor treatment adherence, presented with a two-week history of acute flare involving multiple small and large joints, associated with swelling, tenderness, and early morning stiffness. Examination revealed synovitis of bilateral wrists, right 1st–5th MCP joints, bilateral knee crepitus with swelling, boutonnière deformities of toes, and overriding of the right third toe. Laboratory evaluation showed severe microcytic hypochromic anemia (Hb 5.5 g/dL), leukocytosis, thrombocytosis, low serum iron, vitamin D deficiency, and markedly elevated rheumatoid factor and anti-CCP antibodies, with normal inflammatory markers. Radiographs of hands demonstrated peculiar hooked osteophyte-like formations, while knee radiographs showed severe osteoarthritis. Such radiographic patterns are classically described in hemochromatosis and CPPD arthropathy and are rarely reported in RA, making this an unusual diagnostic overlap.

Conclusion: This case highlights a rare radiographic presentation of long-standing seropositive RA mimicking metabolic arthropathies. Recognition of such atypical imaging findings is crucial to avoid misdiagnosis and underscores the importance of sustained disease control to prevent irreversible joint deformities.

Fig.2



POM87: The Perfect Autoimmune Storm: A Rare Overlap of MDA5-Positive Dermatomyositis and Systemic Lupus Erythematosus

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Introduction: Overlap syndromes constitute 10–20% of idiopathic inflammatory myopathies, while dermatomyositis–systemic lupus erythematosus (SLE) overlap occurs in less than 5%. Anti–melanoma differentiation–associated gene 5 (MDA5)–positive dermatomyositis represents a high-risk subset associated with aggressive multisystem involvement, pulmonary vascular complications, and increased early mortality.

Case Report: A 20-year-old male presented with a 5-month history of intermittent fever, inflammatory symmetric polyarthritis, progressive proximal muscle weakness, jaundice, and weight loss. Examination revealed pallor, icterus, bilateral pedal edema, and proximal myopathy. Investigations showed pancytopenia, hyperbilirubinemia, positive direct Coombs test, and elevated inflammatory markers. Autoimmune profiling demonstrated anti-MDA5 (++), anti-Ro52 positivity, borderline anti-SRP, and ANA positivity with anti-histone and anti-nucleosome antibodies, confirming an overlap syndrome of dermatomyositis and systemic lupus erythematosus. Echocardiography revealed moderate pulmonary arterial hypertension. During hospitalization, the patient developed generalized tonic–clonic seizures consistent with neuropsychiatric lupus. He received high-dose corticosteroids, followed by oral steroids and hydroxychloroquine, with planned rituximab and mycophenolate mofetil. The course was complicated by Escherichia coli bacteremia and Enterococcus faecium urinary tract infection, treated successfully with culture-guided antibiotics, leading to clinical improvement and stable discharge.

Conclusion: This case highlights a rare and high-risk autoimmune overlap syndrome characterized by multisystem involvement, severe infections, and significant morbidity. Early recognition, comprehensive immunological evaluation, vigilant infection surveillance, and individualized immunosuppressive therapy, including biologics, are essential to optimize outcomes in such complex presentations.

POM88: Plasma Cytokine Profiling Reveals a CSF1/CSF2-Dominant Inflammatory Signature Distinguishing Flare from Remission in Indian SLE Patient

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Introduction: Managing Systemic Lupus Erythematosus (SLE) in India is hindered by unpredictable exacerbations and a lack of ethnicity-specific markers. Mapping the systemic proteomic architecture in plasma is essential for transitioning from reactive treatment to proactive, precision-based prediction of disease activity.

Methods: In a multi-centre study (KMC Manipal &AIIMS New Delhi), 48 SLE patients were stratified into Flare (n=24) and Remission (n=24) groups. Activity was mapped using the Easy-BILAG index. Molecular profiling utilized Olink Proximity Extension Assay (PEA) technology. Normalized Protein Expression scores were correlated with organ involvement via t-tests. Biological connectivity was modelled through STRING-based protein-protein interaction (PPI) networks to visualize synergistic inflammatory axes.

Results: Quantitative mapping revealed a distinct inflammatory architecture distinguishing flares from remission, with significant upregulations in IL-33, CCL-2, and CSF1 (all FC: 2.0). Functional analysis showed enrichment for monocyte/myeloid leukocyte chemotaxis and cytokine-receptor interactions. Notably, CSF2 (GM-CSF) was identified as a novel translational hit (FC: 1.41) integrated within this migratory signalling hub, extending beyond previously known lupus signatures. STRING-based modelling confirmed these proteins form a high-density synergistic axis driving systemic recruitment of inflammatory cells during clinical exacerbation.

Conclusion: By integrating Easy-BILAG stratification with high-precision proteomics,this study decodes molecular drivers of SLE flares.The CSF1/CSF2-dominant signature establishes a primary driver of myeloid-mediated inflammation, providing a rigorous foundation for non-invasive liquid biopsy panels and personalized immunotherapy.

Keywords: SLE, Cytokines, Flare, Proteomics, Biomarker

POM89: Rowell Syndrome Masquerading as DRESS: A Diagnostic Challenge

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Introduction: Rowell syndrome is a rare overlap syndrome characterized by systemic lupus erythematosus (SLE) with erythema multiforme (EM)–like lesions and characteristic immunological abnormalities. Due to its clinical resemblance, it is often misdiagnosed as a drug reaction with eosinophilia and systemic symptoms (DRESS).

Methods: To highlight Rowell syndrome as an important differential diagnosis in patients

presenting with EM-like rash suspected to be DRESS, particularly when drug chronology is inconsistent. A detailed clinical evaluation including drug history, dermatological assessment, autoimmune work-up, skin biopsy, and renal biopsy was performed in a patient referred with suspected DRESS syndrome.

Results: The patient presented with a generalized skin rash and was referred as a case of DRESS (RegiSCAR score 2), suspected to be secondary to allopurinol. However, she had been on allopurinol for one year, making drug hypersensitivity unlikely. Skin biopsy was suggestive of erythema multiforme. She had low-grade undocumented fever, alopecia, and anemia due to autoimmune hemolytic anemia. Immunological evaluation revealed positive antinuclear antibody and anti–double-stranded DNA antibody, confirming SLE. Renal evaluation showed significant proteinuria (urine protein–creatinine ratio ~2; 24-hour urinary protein 1800 mg/day), and kidney biopsy demonstrated lupus nephritis class IV. The rash responded to pulse corticosteroid therapy, following which two doses of rituximab were administered.

Conclusion: Rowell syndrome should be considered in patients with EM-like lesions mimicking DRESS. Early recognition prevents misdiagnosis and allows timely immunosuppressive therapy.

POM90: Efficacy and Safety of Rituximab Therapy among SLE Patients: A Retrospective Hospital-Based Study

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Introduction: Systemic lupus erythematosus is a chronic, multisystem autoimmune disease characterized by autoantibody production and a relapsing–remitting disease course. B-cell hyperactivity plays a central role in its pathogenesis, providing a rationale for B-cell–targeted therapies such as rituximab as off-label status in SLE

Methods: This retrospective observational study included adult SLE patients treated with rituximab between 2020 and 2025 at a tertiary care centre. Data were obtained from electronic medical records and patients were followed up until the date of analysis. Disease activity was assessed using the Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2K), and treatment response was categorized as complete remission, partial remission (≥50% improvement in SLEDAI-2K), or no response, based on 2021 DORIS criteria.

Results: A total of 36 patients with active SLE received rituximab therapy. Thirty-five patients were administered two 1 g doses 15 days apart,

while one patient received a single 1 g dose. The cohort was predominantly female (94%), with a median age of 32.4 years and median follow-up of 6–9 months. Lupus nephritis was the most common manifestation (n=24), followed by thrombocytopenia (n=8), cutaneous lupus (n=3), and neuropsychiatric lupus (n=1). Most patients achieved partial or complete responses, with higher complete remission rates in nephritis and thrombocytopenia. Mean SLEDAI-2K scores and serological markers improved significantly ($p<0.001$). Adverse events were mild and manageable.

Conclusion: Rituximab demonstrated favorable efficacy and an acceptable safety profile in selected patients with active SLE, leading to significant clinical, serological, and disease activity improvements across diverse manifestations. However, larger prospective, controlled studies are required to clearly define its optimal role

POM91: Prevalence of Metabolic Syndrome in Patients with Osteoarthritis: A Cross-Sectional Study from a Tertiary Care Centre in North India

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Introduction: Osteoarthritis (OA) is traditionally considered a degenerative "wear-and-tear" joint disease. However, emerging evidence suggests a strong association between OA and metabolic syndrome (MetS), indicating a possible metabolic phenotype of OA. Limited Indian data exist regarding the prevalence of MetS among OA patients.

Methods: This hospital-based cross-sectional observational study was conducted in the Dept of Medicine and Orthopaedics at ABVIMS & Dr. RML Hospital, New Delhi (February 2024–June 2025). A total of 93 adult patients (>18 years) fulfilling the 2010 ACR criteria for OA were included. Metabolic syndrome was diagnosed using NCEP ATP III criteria modified for Asian Indians. Clinical history, anthropometric measurements, blood pressure, fasting glucose, and lipid profile were recorded. Statistical analysis was performed using SPSS; $p<0.05$ was considered significant.

Results: The mean age of participants was 48.71 ± 16.31 years, with nearly equal gender distribution. The prevalence of metabolic syndrome among OA patients was 83.87% (n=78). Hypertension (70.97%), hypertriglyceridemia (81.72%), and low HDL (60.22%) were the most common metabolic abnormalities. MetS was significantly associated with age distribution ($p=0.035$) and female gender ($p=0.01$). On univariate logistic regression, male gender was associated with lower odds of MetS (OR 0.Osteoarthritis, Metabolic Syndrome, NCEP ATP III, Prevalence, Cardiometabolic Risk193; $p=0.016$).

Conclusion: A remarkably high prevalence of metabolic syndrome was observed among osteoarthritis patients in this study. These findings support the concept of a metabolic phenotype of OA and emphasize the need for routine metabolic screening in OA patients to enable comprehensive management and reduce long-term cardiovascular risk

POM92: A Cross-Sectional Study for the Incidence of Dyslipidemia in Rheumatoid Arthritis Patients in a Tertiary Care Hospital

in Eastern India

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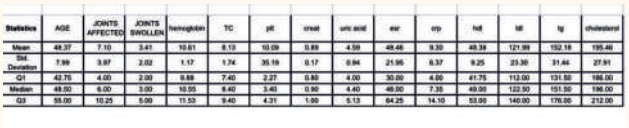
Introduction: Rheumatoid arthritis confers heightened cardiovascular risk through inflammation-mediated lipid abnormalities. Evidence from northeastern India is limited; this study evaluates the incidence, patterns of dyslipidaemia, and their association with inflammatory markers among patients with rheumatoid arthritis.

Methods: This hospital-based observational study included 82 patients with rheumatoid arthritis diagnosed using standard classification criteria at a tertiary care centre in northeastern India. Clinical and laboratory evaluation included ESR, CRP, haemoglobin, complete blood counts, and fasting lipid profile. Data were analysed using descriptive statistics. Correlation between inflammatory markers and lipid parameters was assessed using Pearson's correlation test, with $p < 0.05$ considered statistically significant.

Results: Among the 82 patients with rheumatoid arthritis, dyslipidaemia was highly prevalent. The most common abnormality was reduced HDL cholesterol, followed by elevated LDL cholesterol, triglycerides, and total cholesterol. ESR and CRP were elevated in most patients, reflecting moderate to high disease activity. Correlation analysis revealed a significant negative association between ESR and HDL cholesterol, and significant positive correlations between ESR, LDL cholesterol, triglycerides, and total cholesterol. Similar trends were observed with CRP. These findings suggest persistent systemic inflammation drives an atherogenic lipid profile, increasing cardiovascular risk in RA.

Conclusion: Dyslipidaemia is highly prevalent in rheumatoid arthritis and closely associated with systemic inflammation. Persistent inflammatory activity promotes an atherogenic lipid profile, heightening cardiovascular risk. Routine assessment of ESR alongside lipid profile can aid early identification and risk stratification, especially in resource-limited settings.

Fig.1



Statistics	AGE	JOINTS AFFECTED	JOINTS SWOLLEN	hemoglobin	TC	pH	creat	uric acid	esr	crp	hdl	ldl	tg	cholesterol
Mean	48.37	7.10	3.41	10.61	8.13	10.09	0.88	4.58	48.46	9.30	48.38	121.99	152.18	195.46
Std. Deviation	7.99	3.97	2.02	1.17	1.74	35.19	0.17	0.94	21.96	6.37	9.25	23.30	31.44	27.91
Q1	42.75	4.00	2.00	9.88	7.40	2.27	0.80	4.00	30.00	4.00	41.75	112.00	131.50	186.00
Median	48.50	6.00	3.00	10.55	8.40	3.40	0.90	4.40	46.00	7.30	49.00	122.50	151.50	196.00
Q3	55.00	10.25	5.00	11.53	9.40	4.31	1.00	5.13	64.25	14.10	53.00	140.00	176.00	212.00

POM93: Analysis of Epidemiology and Diagnosis of Isolated Non-Infectious Uveitis

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Introduction: Uveitis is inflammation of the uveal tract, predominantly affecting younger individuals. Although infectious and non-infectious forms share risk factors, their etiologies and management differ, hence, to avoid confounding epidemiologic data and risk factors this review focuses on non-infectious uveitis.

Methods: This is a retrospective study of patients presenting with isolated uveitis, where data was collected for anatomical type of uveitis, laterality, and the duration since presentation, for studying the epidemiology and diagnosis of uveitis. We have included patients above age 18yrs diagnosed with isolated non-infectious uveitis and excluded

all infectious/traumatic causes of uveitis.

Results: 103 patients with isolated non-infectious uveitis were evaluated; 43 had unilateral, 59 bilateral, and 1 alternating involvement. Acute, chronic, recurrent, and subacute disease occurred in 37, 36, 29, and 1 patient, respectively. Anterior uveitis predominated (n=66), next panuveitis (n=22), intermediate (n=13), and posterior (n=2). Anterior uveitis was idiopathic (n=32), HLA-B27+ (n=14), ACE-elevated (n=8), HLA-B5+ (n=1), autoimmune (ANA n=3, RF n=4, anti-Ro/La n=3), and Behçet's (n=1). Panuveitis included idiopathic (n=7), HLA-B27+ (n=2), ACE-elevated (n=8), RF+ (n=1), Behçet's (n=1), and VKH (n=3). Intermediate uveitis was idiopathic (n=9), ACE-elevated (n=2), HLA-B5+ (n=1), or ANA+ (n=1); posterior uveitis included one idiopathic and one elevated-ACE.

Conclusion: Anterior uveitis subtype predominated, with idiopathic, HLA-B27 positivity, and elevated serum ACE the most common etiology. The heterogeneous etiologies underscore the need for a multidisciplinary approach to identify systemic associations and guide management.

POM94: Gender Difference in Clinical Profile of Patients with Psoriatic Arthritis in a Tertiary Care Centre from South India.

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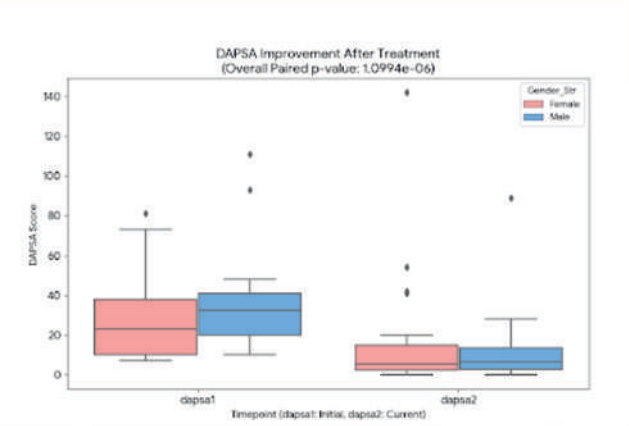
Introduction: Only scarce data on clinical and epidemiological features of PSA from the Indian subcontinent is available, especially from South India. It is perhaps underdiagnosed as other inflammatory arthritis. The study aims to diagnose and initiate optimal treatment, thereby preventing damage.

Methods: This is a cross-sectional, observational study that collected information on patients who fulfilled CASPAR Criteria for PSA from the Rheumatology Department in the Hospital. Information on epidemiological parameters, patterns of joint and domain involvement was collected. DAPSA before and after treatment were assessed. MDA, PSAID and HAQ-DI were calculated to know the quality of life. Statistical analysis on Male and Female differences between various parameters were looked into.

Results: Among 50 patients (M:F - 2:3) mean age was 34.9+ 15.73 in Females and 49.99 + 18.64 in Males which was statistically significant . Psoriasis was followed by arthritis in the majority but simultaneous onset of both were higher in males but not statistically significant. Of the patterns oligoarthritis was slightly higher in males(M-50%, F-40%). Very high DAPSA at presentation was more with males (60%). Women have a slightly higher mean HAQ-DI score (10% vs 5%). Females had a slightly higher mean impact score (1.52 vs 1.22). Statistically significant positive correlation was found between PASI and DAPSA ($p < 0.01$) in both genders.

Conclusion: Though females developed PSA at an early age, males had higher disease activity at presentation. Severity of psoriasis is proportional to the severity of arthritis, hence close follow up of these psoriasis patients for articular symptoms is mandatory for early institution of therapy to prevent damage.

Fig.1



POM96: Macrophage Activation Syndrome Triggered by Dengue Fever in a Newly Diagnosed Mixed Connective Tissue Disease

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Introduction: Hemophagocytic Lymphohistiocytosis(HLH), an aberrant hyperinflammatory immune response driven by T cells and associated with potentially fatal cytokine storm. Secondary HLH may be induced by autoimmune disorders, known as Macrophage Activation Syndrome (MAS). Mixed connective tissue disease (MCTD) is a systemic rheumatic disease with clinical features of SLE, SSc, RA, Polymyositis, dermatomyositis.

Case Report: A 23-year-old married female presented with c/o polyarthralgia a/w swelling & deformities since 1.5years, pain,numbness in finger tips while washing utensils since 8months, fever, generalized weakness,generalized petechial rash for 15 days. She has no h/o alopecia,photosensitivity,malar rash. On examination, she was febrile(103F),tacypnoeic,BP=114/78mmHg & had pallor and moderate hepatosplenomegaly. She had pursed lip, puckered mouth with microstomia, sclerodactyly distal to MCPJ, Raynaud's phenomenon. The hemogram showed pancytopenia. The patient had mild transaminitis. Dengue NS1 Antigen,IgM was positive.She had raised CRP levels,normal ESR and raised ferritin levels(1450ng/ml) which raised the suspicion of HLH/MAS triggered by infection in setting of underlying rheumatological disease which was further evident by low fibrinogen, high triglyceride and Bone marrow showing hemophagocytosis. Autoimmune workup revealed ANA 4+ speckled pattern with high titres of RNP, Nucleosome,dsDNA.Clinical and autoimmune profile suggested MCTD.Further U1RNP came positive satisfying diagnostic criteria of MCTD.

Conclusion: MAS in adult with MCTD is an uncommon presentation with very few case reports published in scientific literature. In our case,the patient satisfied 6/8 criteria of HLH/MAS and the criteria for MCTD (Alacron Segovia,Kahn). Her H score was 249 suggesting 99.3% probability of HLH-MAS. The patient was managed as per protocol to which she showed good response.

POM97: Levofloxacin-Induced Non-Erosive Arthritis in a Young Female with Abdominal Tuberculosis: A Case Report.

Introduction: We describe a 22-year-old woman with abdominal tuberculosis who developed insidious, non-erosive inflammatory arthritis of both knees and ankles after four weeks of levofloxacin in an MDR-TB regimen. Inflammatory markers were normal. Symptoms resolved on drug withdrawal and recurred on rechallenge, implicating levofloxacin. Early recognition prevents misdiagnosis and

unnecessary investigations.

Case Report: A 22-year-old female presented with a three-month history of abdominal pain, weight loss, and intermittent low-grade fever. Diagnostic evaluation, including contrast imaging , established abdominal tuberculosis. Subsequent drug-susceptibility testing demonstrated resistance to first-line anti-tubercular drugs, and a multidrug-resistant tuberculosis (MDR-TB) regimen was initiated as per national guidelines. The regimen included levofloxacin 750 mg once daily, along with ethionamide, cycloserine, and other second-line agents, which were initially well tolerated. After four weeks of therapy, she developed insidious-onset, progressive pain and swelling of both knees and ankles, with inflammatory features including 30–40 minutes of morning stiffness and functional limitation. There was no history of trauma or infection. Examination showed mild effusion and restricted movements without systemic features. Laboratory parameters, including ESR, CRP, and autoantibodies, were normal, and imaging revealed no erosions. Levofloxacin withdrawal led to complete symptom resolution within 14 days. Re-challenge resulted in symptom recurrence, confirming levofloxacin-induced non

Conclusion: Levofloxacin can rarely induce reversible non-erosive inflammatory arthritis, even in young patients. A high index of suspicion is necessary when patients on fluoroquinolones develop acute or subacute polyarthritis. Prompt drug withdrawal leads to rapid symptom resolution, preventing unnecessary diagnostic workup and morbidity.

POM98: Autoimmune Etiologies in Chronic Non-Healing Ulcers: A 5-Year Retrospective Analysis from a diabetic foot surgery unit of a Tertiary Care Centre

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Introduction: Chronic non-healing ulcers that fail to respond to standard wound care may reflect underlying autoimmune pathology. Prevalence and outcomes data for vasculitic and immune-mediated ulcers from low and middle-income countries are limited.

Methods: The objective was to evaluate prevalence, clinical profile, and treatment response of autoimmune versus non-autoimmune non-healing ulcers. A retrospective study conducted between 2018 to 2023, including 394 patients with chronic unhealed ulcers despite vascular intervention and wound care. We excluded uncontrolled diabetes, malignancies, arterial disease, burn wounds, tuberculous ulcers, and osteomyelitis. Autoimmune etiology was established through multidisciplinary discussion, biopsy, histopathology and direct immunofluorescence.

Results: Autoimmune etiology was identified in

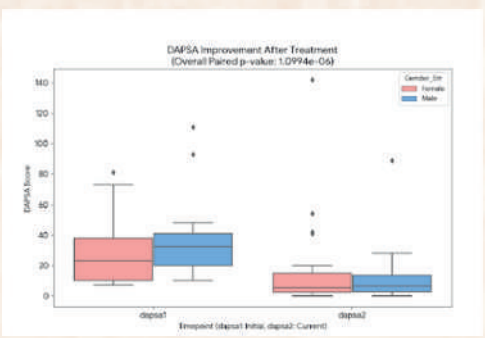
175 patients (44.4%). Vasculitic ulcers constituted predominant autoimmune subtypes, with others being livedo reticularis, panniculitis, and pyoderma gangrenosum. Autoimmune ulcers were associated with prolonged disease duration, disproportionately higher pain and failure of conventional wound care. Complete healing occurred in 70.3% of autoimmune ulcers compared to 81.7% of non-autoimmune ulcers (p = 0.041), indicating a significantly poorer prognosis in autoimmune related chronic ulcers. Targeted immunomodulatory therapy was associated with improved healing rates among autoimmune ulcers.

Conclusion: Nearly half of chronic non-healing ulcers were attributable to autoimmune etiologies, underscoring a substantial under-recognized burden. Early biopsy, prompt recognition of autoimmune pathology, and rheumatology involvement with immunomodulatory therapy can significantly improve healing and reduce morbidity.

Fig.1

Pyoderma gangrenosum on presentation (L) and healing wound after starting immunomodulation therapy (R)

Fig 2



POM99: A Case of Anti-NXP2 - Associated Proximal Myopathy in a Long-Standing Primary Sjögren's Syndrome

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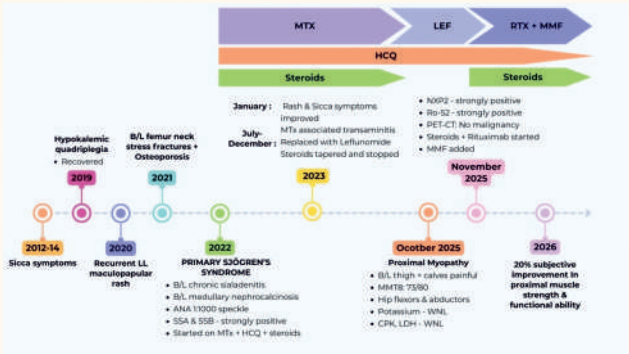
Introduction: Inflammatory myopathy is a rare extraglandular manifestation of primary Sjögren's syndrome (pSS), with limited literature and a reported prevalence of 1–3%. Anti-NXP2 antibodies are typically linked to dermatomyositis, but their significance in pSS-associated myopathy remains unclear. We report a diagnostically challenging case highlighting this uncertainty.

Case Report: We present a diagnostically challenging case of a 55-year woman with long standing pSS associated with chronic sialadenitis, distal renal tubular acidosis, nephrocalcinosis, osteoporotic fractures and past history of hypokalemic paralysis who has now developed proximal myopathy. She presented with complaints of subacute, progressive, symmetrical proximal lower-limb weakness associated with pain and tenderness in both thighs and calves. Within 4 weeks she developed functional limitation in rising from a seated position. Muscle enzymes including creatine phosphokinase and lactate dehydrogenase were not elevated. A myositis antibody panel revealed anti-NXP2 positivity with strong anti-Ro52 positivity. FDG-PET imaging demonstrated no metabolically active skeletal muscle inflammation, while

confirming known Sjögren-related salivary gland changes and bilateral medullary nephrocalcinosis. No evidence of underlying malignancy was noted. She was treated with oral glucocorticoids and rituximab which has prevented further progression of symptoms and 20% subjective improvement.

Conclusion: This case highlights the diagnostic challenges of inflammatory myopathy in pSS, particularly when muscle enzymes and imaging are unremarkable but myositis specific antibodies are positive. Recognition of such atypical presentations is essential to avoid diagnostic delays and to prompt appropriate therapy.

Fig.1



POM100: Quadriparesis as a presenting feature of Sjogren's syndrome

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Introduction: Sjögren's syndrome is an autoimmune disorder primarily affecting exocrine glands, but extraglandular involvement may occasionally precede or overshadow sicca symptoms, leading to diagnostic challenges.

Case Report: A 22-year-old female presented with acute-onset, rapidly progressive pure motor quadriparesis evolving over 24 hours, without sensory, cranial nerve, or autonomic involvement. Neurological examination revealed flaccid quadriparesis with power of 1/5 in all four limbs. Laboratory evaluation demonstrated severe hypokalemia (serum potassium 1.55 mmol/L), hyperchloremia, and normal anion gap metabolic acidosis with preserved renal function. Urinary indices revealed inappropriate renal potassium wasting, consistent with distal renal tubular acidosis (dRTA). The patient showed dramatic clinical improvement following intravenous potassium supplementation, with complete recovery of motor strength within 24 hours. In the absence of drug exposure or systemic illness, further evaluation was undertaken. Detailed history elicited intermittent ocular dryness, and Schirmer's test was positive. Immunological workup revealed strong anti-SS-A (Ro) antibody positivity with negative ANA, establishing the diagnosis of Sjögren's syndrome.

Conclusion: This case underscores a rare and atypical presentation of Sjögren's syndrome manifesting as hypokalemic paralysis secondary to distal renal tubular acidosis, without prominent sicca symptoms. Early recognition of renal involvement in autoimmune disorders is crucial, as timely correction of electrolyte abnormalities results in complete neurological recovery. This report emphasizes the need to consider Sjögren's syndrome in young patients presenting with unexplained hypokalemic paralysis.

POM101: Alopecia, an Early Clue to Multisystem Autoimmune Disease Presenting With Cardiac and Renal Failure: A Case Report

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Introduction: Alopecia can be an early and often overlooked manifestation of systemic autoimmune diseases. When associated with multiorgan involvement, including cardiac, renal, and hematological abnormalities, it may serve as an important clinical clue to an underlying systemic inflammatory disorder. Such presentations pose significant diagnostic challenges and require a comprehensive, multidisciplinary evaluation.

Case Report: We describe a patient who presented with alopecia as an initial complaint and was subsequently found to have multisystem involvement. The case highlights the importance of recognizing dermatological manifestations as potential markers of serious systemic disease and emphasizes the need for early diagnostic consideration of autoimmune etiologies. A patient presented with complaints of progressive alopecia for four months, with a prior history of jaundice. The patient later developed symptoms suggestive of heart failure, including dyspnea, abdominal distension, and pedal edema. Clinical examination revealed raised jugular venous pressure and ascites. Laboratory investigations showed severe anemia, with peripheral smear suggestive of microcytic hypochromic anemia. Inflammatory markers were elevated, with an erythrocyte sedimentation rate of 150 mm/hr and C-reactive protein of 6 mg/dL. Thyroid function tests were within normal limits. Renal function tests were suggestive of acute kidney injury. Urine routine microscopy revealed 12 pus cells per high-power field, granular casts, and 20–25 red blood cells per high-power field, indicating active urinary sediment. Autoimmune workup showed a positive antinuclear antibody (ANA). The constellation of alopecia, cardiac failure, renal involvement, anemia, serological positivity, and systemic inflammation suggested an underlying systemic autoimmune disorder with multiorgan involvement.

Conclusion: This case illustrates that alopecia may be an early manifestation of systemic autoimmune disease and should not be considered a benign, isolated finding when accompanied by systemic features. Early recognition of multisystem involvement and prompt evaluation for autoimmune etiologies are essential to prevent disease progression and organ damage.

PC01: Back to Basics: An "Old" Biologic Achieving Remission in Refractory Psoriatic Arthritis

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Introduction: Psoriatic arthritis is a clinically heterogeneous inflammatory disease with evolving phenotypes and variable treatment response. Despite expanding targeted biologic options, optimal sequencing in multi-biologic refractory disease remains unclear. We report sustained remission with infliximab, underscoring the enduring role of TNF-α inhibition in refractory PsA.

Case Report: A 35-year-old female with a 15-year history of chronic plaque psoriasis, initially skin-predominant, developed progressive nail and peripheral joint involvement over time. She presented with persistent pain, swelling, and deformity of the left fifth proximal interphalangeal joint for one year, consistent with psoriatic arthritis. Examination revealed dactylitis of the right 3rd toe and active synovitis of the left fifth PIP joint and wrist (Figure 1). She had prior exposure to adalimumab for cutaneous disease. Methotrexate was initiated following onset of arthritis but discontinued due to intolerance. Sequential therapy with secukinumab and guselkumab had resulted in inadequate clinical response. Despite exposure to IL-17, TNF-α, and IL-23 inhibitors, disease activity persisted. In view of refractory disease, infliximab therapy was initiated, following which the patient achieved clinical remission that was sustained on follow-up.

Conclusion: In multi-biologic refractory psoriatic arthritis with evolving clinical phenotype, established biologics such as infliximab remain relevant and may induce remission when newer targeted agents fail.

Fig.1

Pc02: When Psoriatic Arthritis Takes an Unexpected Turn: Vasculitis With Peripheral Neuropathy After Sequential Targeted Therapy

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Introduction: Psoriasis is a chronic, multisystem inflammatory disease with diverse clinical domains and a prolonged disease course. Patients may experience multiple therapeutic transitions and unexpected clinical manifestations over time. We report a case highlighting such diagnostic twists and turns in a patient with long-standing psoriatic arthritis complicated by systemic vasculitis.

Case Report: A 70-year-old female with a 10-year history of scalp psoriasis and inflammatory arthritis progressed to arthritis mutilans. She had received tofacitinib for five years with partial response. One year prior to presentation, she developed coronary artery disease, following which tofacitinib was discontinued and adalimumab was initiated. She subsequently presented with a painful, non-healing lower limb ulcer with sensory symptoms suggestive of peripheral neuropathy. Examination revealed ulceration with surrounding purpura and distal sensory loss. Inflammatory markers were elevated, and skin biopsy showed features of vasculitis. Despite discontinuation of adalimumab, the ulcer persisted. Mycophenolate mofetil was initiated with local wound care, leading to gradual ulcer healing and symptomatic improvement. This case highlights vasculitis with peripheral neuropathy in long-standing psoriatic arthritis. Differentiating drug-induced from coexisting systemic or psoriasis-associated vasculitis was challenging. Both TNF and JAK inhibitors have been linked to paradoxical immune-mediated events. Mycophenolate mofetil provided effective control of vasculitic manifestations.

Conclusion: Coexisting or drug-induced vasculitis can complicate long-standing psoriatic arthritis in patients exposed to multiple biologic and targeted therapies. A broader clinical perspective is crucial to recognize these diagnostic challenges and ensure timely and appropriate management of such complex cases.

Fig.1

Pc03: Anti Jo 1 Syndrome with Severe Myositis

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Introduction : Anti-Jo-1 syndrome is idiopathic inflammatory myopathies characterized by autoantibodies against histidyl-tRNA synthetase, leading to muscle inflammation, interstitial lung disease (ILD), arthritis, and systemic features. These specific Autoantibodies myositis-specific autoantibodies (MSAs)) are clinically useful biomarkers to help the diagnosis, predicting and prognosticating certain clinical manifestations of polymyositis/dermatomyositis.

Case Report: A 50-year-old female presented with severe and progressive proximal muscle weakness for 1 month. There was no skin rash (Gottron's papules or Heliotrope rash), ILD, arthritis, mechanic's hands or fever. There was anemia and raised acute phase reactants. CPK and LDH were significantly raised. ANA and anti Jo 1 were highly raised. Histopathology showed preserved fascicular architecture. There were polyfocal inflammatory infiltrates in the perivascular location in the endomysium and perimysium. There was perifascicular atrophy. Immunohistochemistry showed MxA- positivity in the perifascicular atrophic fibres (sarcoplasmic) and capillaries, p62 showed granular positivity in the necrotic fibres which were suggestive of dermatomyositis. While awaiting biopsy report, she was started on intravenous methylprednisolone 750 mg with MMF for 3 days but she progressed to pharyngeal and respiratory muscle weakness needing mechanical ventilation. She was given intravenous immunoglobulin which improved the

muscle weakness and she survived.

Conclusion: Myositis is the most common presentation of anti Jo-1 syndrome though it is not usually severe unlike anti SRP, anti NXP 2 diseases. Next common feature is ILD. Early diagnosis, multidisciplinary management, and immunomodulatory therapy improve outcomes and reduce complications and mortality.

PC04: Granulomatosis with Polyangiitis (GPA) Presenting as Fever and Otitis Media in an Adolescent: A Diagnostic Challenge

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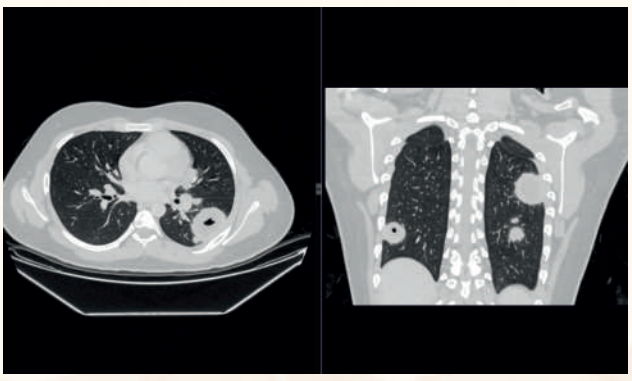
Introduction: Granulomatosis with polyangiitis (GPA) is a rare, multisystem necrotizing vasculitis. While several case series have been reported, the disease remains uncommon in the adolescent and pediatric population. We describe a case of biopsy-proven GPA presenting after a relatively short clinical history.

Case Report: A 15-year-old male presented with a short history of high-grade continuous fever associated with dry cough . There was no history of dyspnea, hemoptysis, urinary or abdominal symptoms, or contact with tuberculosis. Notably, he had developed right ear pain a few days prior to the onset of fever. On examination, the patient was febrile (38°C). Initial investigations revealed leukocytosis with elevated inflammatory markers. Chest radiography showed multiple cavitary nodules in both lung fields, which were confirmed on CT chest. A CT of the paranasal sinuses demonstrated otomastoiditis. A lung nodule biopsy revealed granulomatous necrotizing inflammation. Cytoplasmic antineutrophil cytoplasmic antibody (C-ANCA) levels were markedly elevated (146 AU/mL), confirming the diagnosis of GPA.

Discussion: This case is notable as the predominant presentation was fever, with otitis media being the only early clinical clue suggestive of vasculitis. Prompt recognition and early lung biopsy in this patient enabled definitive diagnosis and timely treatment.

Conclusion: Granulomatosis with polyangiitis is a small-vessel vasculitis characterised with upper, lower airway and kidney involvement. In tuberculosis-endemic regions, fever associated with cavitary lung lesions poses a significant diagnostic challenge. This case highlights the importance of considering GPA in adolescents presenting with fever, upper airway and lung involvement.

Fig.1



PC05: Dermatomyositis: Unusual presentation of JAK 3 mutation

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Introduction: Dermatomyositis (DM) is an

idiopathic inflammatory myositis characterised by muscle weakness and skin rash with a prominent type-I interferon (IFN-I) signature. The JAK-STAT pathway mediates IFN signalling. Germline mutations in the JAK family usually cause immunodeficiency. We report three cases of Dermatomyositis phenotype with novel JAK3 mutations, expanding the genotype-phenotype spectrum.

Case Report: We report three patients with dermatomyositis-like phenotype harbouring pathogenic JAK3 mutations, highlighting a novel immune–myositis overlap. Case 1 was a 4-year-old girl with classical juvenile dermatomyositis (JDM) features and proximal weakness (CMAS 24/52), elevated CK, and discordant myositis antibodies. Whole exome sequencing (WES) revealed a homozygous JAK3 exon 12 mutation (p.Gly297Ala). Case 2 was a 9-year-old boy with typical cutaneous features, myopathy on EMG/MRI, normal muscle enzymes, and Mi-2β positivity; WES showed compound heterozygous JAK3 mutations affecting splicing and protein structure. Case 3 was a 25-year-old woman with dermatomyositis features (skin rash and proximal muscle weakness), early-onset diabetes, normal muscle enzymes, and negative myositis antibodies, with a homozygous JAK3 exon 20 mutation (p.Arg918Pro). All patients had a consistent immunological pattern of reduced T cells with increased B cells, without clinical immunodeficiency. All showed clinical improvement with standard immunosuppression. These cases expand the phenotypic spectrum of JAK3 mutations beyond classical SCID and suggest a possible role in inflammatory myopathy pathogenesis.

Conclusion: We present three cases of a novel JAK3 mutation presenting as a clinical phenotype of Dermatomyositis with T-cell lymphopenia, without significant risk of infection. To further confirm pathogenicity, we plan to test for single-cell RNA sequencing, interferon signature and phosphorylation of pSTAT5 to demonstrate functional validation of defective JAK3 signaling.

PC07: Histiocytic Sarcoma Presenting as Anti-PL12 Positive Antisynthetase Syndrome: A Novel Paraneoplastic Association

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Introduction: Antisynthetase Syndrome (ASyS) typically presents with triad of myositis, arthritis, and interstitial lung disease (ILD). Anti-PL-12 variant is highly specific for severe ILD (90-100%). Malignancy is rarely associated with ASyS. We report a unique case of PL-12 positive ASyS presenting as a paraneoplastic manifestation of Histiocytic Sarcoma without lung involvement.

Case Report: A 53-year-old female presented with a one-year history of erythematous generalized rashes, inflammatory polyarthritis, fever, reduced appetite, weight loss, and difficulty in getting up from squatting for six months. Examination revealed erythematous plaque-like rashes involving the face, trunk, and the limbs; a MMT-8 score of 140/150. Routine labs were normal, muscle enzymes revealed normal CPK, but elevated SGOT. ANA was positive (1:1000, cytoplasmic); myositis profile confirmed strong PL-12 positivity. MRI of the thighs confirmed active myositis. CECT abdomen revealed a 7.1 x 6.8 x 6.8 cm retroperitoneal mass and remarkably

no ILD. Histopathological examination of the mass demonstrated - sheets of epithelioid histiocytes with hyperchromatic nucleus with brisk mitotic activity and moderate eosinophilic cytoplasm. Immunohistochemistry was strongly positive for CD4, CD68, and CD163, confirming a diagnosis of Histiocytic Sarcoma.

Conclusion: This case challenges two clinical paradigms: the nearly universal association of PL-12 with ILD and the low malignancy risk attributed to ASyS. Clinicians should maintain high suspicion for occult malignancy in ASyS, even with "low-risk" antibodies like PL-12, especially when B-symptoms are present or classic features like ILD are absent.

Fig.1

FIGURE-1: Erythematous plaque like rash involving the knuckles, small joints of hand, interphalangeal area (A), erythematous malar rash involving the nasolabial folds (B), erythematous plaque like rash involving both the upper limbs (C).

PC08: Spontaneous Pneumomediastinum in Anti-MDA5 Dermatomyositis Without RP-ILD: A Case Series

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Introduction: Anti–melanoma differentiation-associated gene 5 (MDA5) dermatomyositis (DM) is commonly linked to rapidly progressive interstitial lung disease (RP-ILD). Spontaneous pneumomediastinum is considered a poor prognostic marker, usually associated with RP-ILD. Its significance outside this setting is unclear. We describe three anti-MDA5 DM patients who developed spontaneous pneumomediastinum without RP

Case Report: Three patients with anti-MDA5 dermatomyositis developed spontaneous pneumomediastinum without rapidly progressive ILD. A 38-year-old woman with predominant cutaneous disease, mild myositis, and stable NSIP–organizing pneumonia–pattern ILD developed pneumomediastinum without respiratory compromise and stabilized with high-dose glucocorticoids and cyclophosphamide despite concurrent infections. A 22-year-old man with severe cutaneous ulcerations, arthritis, and mild ILD presented with pneumomediastinum complicated by pneumothorax and extensive subcutaneous emphysema. In the absence of RP-ILD, he improved with pulse steroids and combination immunosuppression including tacrolimus, tofacitinib, and rituximab. A 52-year-old woman with severe myositis and bulbar involvement requiring monthly IVIG had chronic stable ILD complicated by pneumomediastinum that resolved on follow-up imaging. Her relapsing multisystem disease was managed with cyclophosphamide, maintenance tacrolimus and mycophenolate mofetil, and rituximab for refractory myositis.

Conclusion: Spontaneous pneumomediastinum may occur in anti-MDA5 dermatomyositis even in the absence of rapidly progressive ILD and does not invariably indicate a poor prognosis. Recognition of this atypical presentation is important, as outcomes may be favourable with timely and individualized immunosuppressive

therapy

PC09: A Rare Triad: Macrophage Activation Syndrome in Isolated Neuro-Behçet's–Sjögren's Disease

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Introduction: Macrophage activation syndrome (MAS) is HLH secondary to autoimmune diseases. Association of MAS with Sjogren is rare. Association of MAS with Behçet's is rare. This case represents one of the very few reported cases of macrophage activation syndrome in Behçet's disease and an exceedingly rare coexistence with Sjögren's syndrome.

Case Report: A 35-year female presented with sudden-onset altered sensorium, headache, and vomiting, with a history of fever for 2 months. Examination showed bilateral plantar extensors, no neck stiffness, mild splenomegaly. Past history of herpes zoster present. Neuroimaging revealed hemorrhagic & ischemic lesions involving the thalamus, corpus callosum, cortical and subcortical regions, along with pachymeningeal enhancement suggesting vasculitis. Laboratory evaluation & declining ESR showed features of macrophage activation syndrome (MAS). Extensive evaluation for MAS including tropical fever, blood cultures, viral serology (EBV, CMV), ANA, Brucella, IgG4, CSF, and bone marrow examination, was unremarkable. Anti Ro-52 was 3+, HLA B51 PCR positive and VZV IgG positive. Lip biopsy showed focal chronic lymphocytic sialadenitis (focus score 1). Based on clinical, radiological, and immunological findings, a diagnosis of MAS secondary to isolated Neuro-Behçet's disease possibly triggered by VZV with coexisting Sjögren's disease was made. The patient was treated with Pulse steroids, IVIg, Heparin & oral Cyclosporin.

Conclusion: To our knowledge, this represents the second reported case of macrophage activation syndrome in isolated Neuro-Behçet's disease. Varicella-zoster virus has been hypothesized as a potential trigger for Neuro-Behçet's. Awareness of this association is crucial, as early recognition and prompt immunosuppressive therapy may be life-saving. Neuro-Behçet's may rarely coexist with Sjögren's disease.

PC10: Combination of immunosuppressive agents (ISA) in Inclusion Body Myositis (IBM)

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Introduction: IBM is a rare form of inflammatory myositis. Evidence for a use of ISA in IBM is weak or even controversial

Case Report: We report a case of a 69-year-old female who presented to us with bilateral lower limb weakness, altered coordination in hands and feet, difficulty in swallowing. As per history and medical examination, IBM was suspected. MRI of both thighs was advised which showed intra-muscular oedema with fatty infiltration in anterior and medical compartment muscles of thigh.

Myositis profile was borderline positive for Mi-2 alfa. ESR was 36, CRP, RF, ANA by IF were normal. Muscle biopsy showed intra-cytoplasmic vacuoles positive for p62 on immunohistochemistry. Total CPK was 976 U/ML. The patient was started on methotrexate 15 mg once a week with mycophenolate mofetil along with glucocorticoids 10 mg/day. She improved gradually and total CPK came down to 123 U/L. Physiotherapy was carried out concomitantly. Her IBM-FRS (functional rating scale) was 23 initially, which gradually improved to 26 and in last visit in February, 2026, it was 28 out of 40.

Conclusion: Combination ISA therapy can be helpful in improving and/or maintaining muscle function or at least, not degrading the muscle function.

PC11: Intracranial Aneurysms Preceding Mucocutaneous Features in Behcet's Disease: A Diagnostic and Therapeutic Challenge

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Introduction: Behcet's disease is a chronic, multisystem vasculitis, most commonly presenting with mucocutaneous and ocular manifestations. Vascular disease is seen in 15-50% of patients, with infrequent arterial involvement (3-5%). Intracranial aneurysms are a distinctly rare manifestation of Behcet's disease.

Case Report: A 45-year-old man presented with headache and recurrent syncopal episodes. He had two prior episodes of right lower motor neuron facial palsy treated with steroids and a history of superficial thrombophlebitis in both upper limbs. Examination showed acneiform lesions over the trunk. Blood tests, including inflammatory markers, ANA and ANCA, were normal. Echocardiography was unremarkable. CT angiography revealed a saccular aneurysm of the left anterior cerebral artery and hypoplastic right vertebral artery. Differential diagnoses included Behçet's disease, DADA2 and polyarteritis nodosa. PET scan and whole exome sequencing were unrevealing. Pathergy test was positive, and biopsy showed lymphocytic folliculitis. He later developed an oral aphthous ulcer, fulfilling criteria for Behçet's disease. He was treated with pulse methylprednisolone, oral steroids and cyclophosphamide. Persistent headache led to repeat imaging, which showed two intracranial aneurysms. As there was high risk of rupture owing to the large size of aneurysms and bifurcation sites, he underwent endovascular coiling of the aneurysm successfully, after which symptoms resolved.

Conclusion: Behcet's disease can present with neurovascular manifestations, with mucocutaneous symptoms appearing late. Intracranial aneurysms are rare in Behcet's disease and they carry high risk of rupture. Immunosuppression alone may not be sufficient and early endovascular/surgical intervention should be considered if high-risk morphology of aneurysms. The Pathergy test remains valuable in ambiguous cases.

PC12: Pseudo-Lupus: A Rare Confluence of Pancreatitis-Associated Thrombotic Micronagiopathy and Triple P Syndrome

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Introduction: Pancreatitis-panniculitis-polyarthritis (PPP) syndrome is a rare systemic manifestation of pancreatic disease caused by the release of pancreatic enzymes into the circulation, leading to fat necrosis at distant sites. It can masquerade as systemic autoimmune diseases. Pancreatitis-induced thrombotic microangiopathy (TMA) is another rare complication of pancreatitis endothelial injury and compleme

Case Report: A 17-year-old girl presented with abdominal pain, vomiting, and fever for 15 days and was diagnosed with acute pancreatitis with cholelithiasis. During hospitalization, she developed acute polyarthritis involving the wrists, ankles, knees, and several MCP and PIP joints, along with erythematous nodular lesions near right knee and left lateral malleolus. Labs showed a rapid fall in hemoglobin from 13.8 to 7.7 g/dL, thrombocytopenia (70,000/µL), elevated LDH (1114 U/L), rising creatinine (2.96 mg/dL), schistocytes on peripheral smear, proteinuria, and microscopic hematuria, raising suspicion of lupus-related thrombotic microangiopathy. However, ANA was negative and complement levels were normal. MRI of ankles demonstrated marrow signal abnormalities with periarticular edema, without synovitis or erosions, consistent with pancreatic enzyme-mediated bone marrow fat necrosis. Cytology of a skin lesion suggested panniculitis. A diagnosis of pancreatitis, panniculitis, and polyarthritis (PPP syndrome) was made. With conservative management alone, the patient showed spontaneous resolution of laboratory and musculoskeletal findings as pancreatitis improved, conforming PPP syndrome.

Conclusion: Pancreatitis can cause multisystem inflammatory manifestations, including TMA and PPP syndrome, closely mimicking autoimmune and vasculitic disorders. Management should prioritize treatment of the underlying pancreatic disease. True autoimmune disease and primary TMA rarely resolves spontaneously without targeted therapy. therapy

PC13: Vascular Ehlers-Danlos Syndrome: A Rare Mimicker of Medium-Vessel Vasculitis

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Introduction: Vascular Ehlers-Danlos syndrome (vEDS) is a rare connective tissue disorder caused by mutations in the COL3A1 gene and is characterized by arterial fragility, spontaneous rupture, and tissue laxity. Medium-vessel vasculitides such as polyarteritis nodosa can mimic its presentation, posing a diagnostic challenge.

Case Report: A 50-year-old man presented with abdominal pain and vomiting. He had a significant past history of spontaneous rupture of a left ulnar artery pseudoaneurysm requiring surgical repair, and a left tendoachilles rupture after trivial trauma. He also reported exertional dyspnea, intermittent productive cough for nearly ten years, and easy bruisability. CT scan of the chest and abdomen showed extensive centrilobular and paraseptal emphysema in a nonsmoker, along with multifocal fusiform aneurysmal dilatations of the left hepatic, splenic, middle colic, and bilateral renal arteries with peripancreatic hematoma. Subsegmental pulmonary artery thrombosis and thrombosis of the left branch of the middle colic artery were also

noted. Differential diagnoses included medium-vessel vasculitis, antiphospholipid syndrome, and non-inflammatory vasculopathies. Laboratory tests, including inflammatory markers, were unremarkable. Viral serology, ANA, and antiphospholipid antibodies were negative. A detailed family history revealed intracranial hemorrhage in his sister and keratoconus in his son. He fulfilled the Villefranche criteria and was diagnosed with vascular Ehlers–Danlos syndrome.

Conclusion: Unexplained arterial aneurysms or spontaneous vascular ruptures in adults should raise suspicion for vascular Ehlers-Danlos syndrome, even in the absence of classic skin or joint findings. Subtle connective tissue signs and family history—such as tendon rupture, easy bruising, or premature vascular events—are key clues to differentiate vEDS from other non-inflammatory vasculopathies.

PC14: Fulminant Rhabdomyolysis and Bulbar Involvement in Antibody-Negative Juvenile Dermatomyositis: A Rare Life-Threatening Presentation

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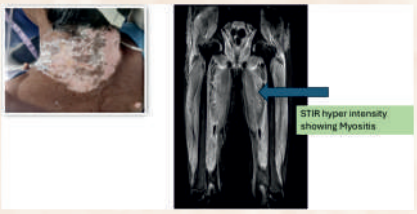
Introduction: Juvenile dermatomyositis (JDM) is a rare autoimmune inflammatory myopathy in children; fulminant rhabdomyolysis and bulbar muscle involvement are exceptionally rare and potentially life-threatening manifestations.

Case Report: We describe a severe presentation of juvenile dermatomyositis in an 11-year-old boy with symmetrical proximal muscle weakness, heliotrope rash, shawl sign, intermittent fever, and markedly elevated muscle enzymes (CK 8,207 U/L, LDH 1,042 U/L, AST 654 U/L). Myositis-specific autoantibodies were negative. Pulmonary function testing showed moderate restriction, while CT thorax excluded interstitial lung disease. Given high disease activity, he was treated with intravenous methylprednisolone pulses followed by oral corticosteroids and methotrexate, resulting in partial improvement. Shortly after discharge, he developed dysphonia, nasal regurgitation, severe myalgia, and neck pain, consistent with bulbar involvement. Despite early IVIg, he progressed to fulminant rhabdomyolysis (peak CK 40,802 U/L), MRI-confirmed myositis, and severe hyponatremia (Na^+ 119 mmol/L) complicated by seizures. He required pediatric intensive care, mechanical ventilation, and escalation of immunosuppression with cyclophosphamide and repeat IVIg, followed by gradual recovery and sustained clinical remission.

Conclusion: Antibody-negative juvenile dermatomyositis accounts for nearly 20–30% of cases, reflecting significant disease heterogeneity. This case highlights fulminant rhabdomyolysis as a rare but critical manifestation of severe immune-mediated muscle injury in antibody-negative JDM, emphasizing the need for phenotype-driven diagnosis and early aggressive immunosuppression to improve outcomes.

Fig.1



Pc15: Co-existence of autoimmunity and autoinflammation-Anti-synthetase Syndrome in a patient with Spondyloarthritis

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Introduction: Co-existence of autoimmunity and autoinflammation is rare but can occur as rheumatic diseases have a variable spectrum of manifestations.

Case Report: A 65-year-aged male with late onset spondyloarthritis diagnosed six months ago who has been taking tofacitinib 5mg BID since five months reported with cough, fatigue, low-grade fever, significant weight loss and pain in knees, and hips. Physical examination revealed fissured skin over both thumb, tender knees without effusion and normal muscle power in all extremities although diffuse tenderness noted in thighs. Whole body PET/CT excluded malignancy, however features of Interstitial Lung Disease (NSIP) and inflammatory arthritis in knees were seen. No significant FDG uptake in muscles noted. Myositis profile revealed anti-NXP2 and anti-PL-12 autoantibodies positivity, CPK levels were 226. MR imaging of pelvis revealed active inflammation in pelvic musculature along with active sacroiliitis. His inflammatory back ache was relatively less symptomatic. He improved with prednisolone and rituximab.

Conclusion: Treatment challenges can occur when autoimmunity and autoinflammation co-exist. In such situations, organ, and life-threatening features needs to be given therapeutic importance.

PC16 : Vogt–Koyanagi–Harada Syndrome: A Frequently Overlooked Cause of Vision-Threatening Uveitis

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Introduction: Vogt–Koyanagi–Harada (VKH) syndrome accounts for approximately 7–20% of non-infectious uveitis in Asian and Middle Eastern population and is a rare multisystem autoimmune inflammatory disorder directed against melanocyte-associated antigens and commonly presents with bilateral granulomatous uveitis, serous retinal detachment and variable neurologic and auditory manifestations.

Case Report: A 35-year-old woman presented with a one-month history of progressive, painless vision loss in both eyes, preceded by a week of severe headaches, vomiting, and hearing impairment. She had been managed elsewhere for macular edema with topical nepafenac, but her symptoms still significantly worsened. On presentation to our institute, the patient was vitally stable, and a comprehensive neurological examination revealed no focal deficits or abnormality. Ocular evaluation showed bilateral granulomatous panuveitis with characteristic mutton-fat keratic precipitates and posterior synechiae. Fundus examination revealed extensive serous retinal detachments in both eyes. Based on the ocular findings, her systemic

symptoms, and a pure tone audiometry report confirming bilateral sensorineural hearing loss (SNHL), a diagnosis of Vogt-Koyanagi-Harada (VKH) syndrome was made. The patient was managed with a three-day pulse of intravenous methylprednisolone, followed by a slow-tapering regimen of oral prednisolone. To ensure long-term inflammatory control and for a steroid-sparing effect, oral azathioprine was also initiated. She improved symptomatically with reduction in serous retinal detachment.

Conclusion: This case emphasizes the importance of early recognition of bilateral granulomatous panuveitis, serous retinal detachment and audiovestibular symptoms for the timely diagnosis of Vogt–Koyanagi–Harada disease. A high index of suspicion and prompt initiation of aggressive systemic immunosuppression at presentation are crucial to achieve sustained inflammatory control.

Fig.1

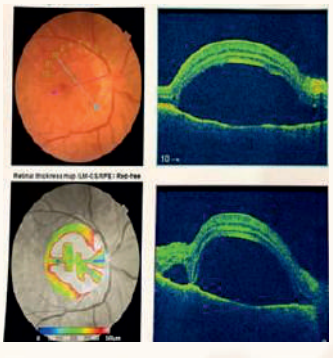
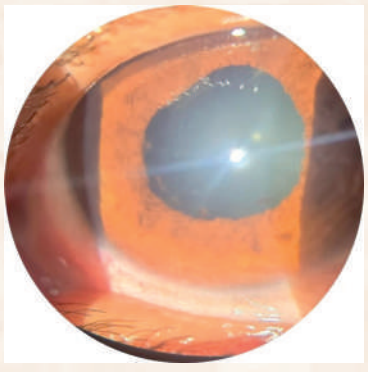


Fig. 2



PC17: A Rare Case of Sjogren's Syndrome Presenting with Extraglandular Manifestations.

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Introduction: Sjogren's Syndrome is a true multisystem disorder generally presenting as classical sicca symptoms signifying glandular involvement. Although it can present with various systemic involvement without sicca symptoms. We are presenting a case of Sjogren's syndrome which did not present with classical sicca symptoms but rather presented with renal involvement.

Case Report: A 50-year-old female presented to the emergency department with complaints of weakness in all four limbs. Investigations are suggestive of RTA and hypokalemic paralysis. Further investigations showed ANA and Ro La positivity without any sicca symptoms on objective evaluation as well. Lip biopsy and ultrasound parotid gland showed evidence of Sjogren's Syndrome.

Conclusion: Sjogren's Syndrome can present without sicca symptoms with renal involvement.

PC18: Anti --MDA 5 Positive Dermatomyositis with Rapidly Progressive Respiratory Failure: A Missed Opportunity or an Inevitable Outcome?

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Introduction: Anti – MDA 5 positive dermatomyositis is a distinct subset of myositis associated with high mortality and mortality,

predominantly due to rapidly progressive interstitial lung disease (ILD). The disease course may be aggressive even in absence of ILD. Optimal choice of immunosuppression strategies remains uncertain in such presentations.

Case Report: A 40-year-old male presented with six months of fever, joints pain, weakness and loss of appetite. Examination revealed proximal muscle weakness (power 3/5), periorbital edema with heliotrope rash, Gottron's erythema over dorsal hand and papules as shown in the figure and symmetric small joint polyarthritis. Investigations showed elevated muscle enzymes (serum CPK 839 U/L, LDH 739 U/L, AST 174 U/L). The HRCT chest showed no ILD. He was diagnosed with dermatomyositis and treated with intravenous methylprednisolone pulses for 3 days followed by oral prednisolone 1mg/kg/day. Myositis profile revealed strongly positive anti MDA-5 antibodies. In view of predominant arthritis with no ILD, Methotrexate 15 mg weekly once and Tacrolimus 0.5 mg twice daily was initiated and was discharged. Two weeks later he presented with rapidly progressive respiratory failure. (CT chest showed new bilateral patchy ground glass opacities with consolidation). Despite high dose steroids and broad-spectrum antibiotics, he succumbed within 24 hours.

Conclusion: This case highlights the unpredictable and fulminant course of anti MDA-5 positive dermatomyositis. Absence of ILD at presentation does not prevent the risk of rapid pulmonary deterioration. Early aggressive immunosuppression is warranted even in seemingly limited disease.

Fig.1

PC19: Beyond Behçet's: Recurrent Oral Ulcers as the First Clue to Pemphigus Vulgaris

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Introduction: Recurrent oral aphthous ulcers are frequently encountered in rheumatology practice and are commonly attributed to Behçet's disease, nutritional deficiencies, or drug-related causes. Pemphigus vulgaris (PV), an IgG-mediated autoimmune blistering disease, may initially present with isolated oral ulceration, closely mimicking benign or inflammatory conditions and resulting in diagnostic delay.

Case Report: We report three patients presenting

with recurrent, painful oral ulcerations refractory to standard therapy. Infective, nutritional, and systemic inflammatory causes were excluded. Detailed mucocutaneous examination was performed, followed by histopathological evaluation, direct immunofluorescence, and anti-desmoglein antibody testing.Three patients (age range 42–65 years) presented with recurrent oral erosions involving the buccal mucosa, tongue, gingiva, and, in one case, laryngeal structures. Initial investigations and biopsies were non-specific, leading to diagnostic delay. Disease progression with diffuse mucositis, involvement of keratinized oral sites, and subsequent cutaneous lesions prompted repeat evaluation. Histopathology demonstrated suprabasal acantholysis with intercellular IgG deposition on direct immunofluorescence and positive anti-desmoglein 3 antibodies, confirming pemphigus vulgaris. Treatment with systemic corticosteroids and rituximab resulted in marked clinical improvement.

Conclusion: Pemphigus vulgaris can initially masquerade as recurrent aphthous ulceration, mimicking common rheumatologic differentials such as Behçet's disease. Persistent, treatment-resistant oral ulcers—particularly with diffuse mucositis or keratinized mucosal involvement—should prompt evaluation for autoimmune blistering diseases. Early diagnosis is essential to reduce morbidity and prevent disease progression.

PC20: Waxing and Waning Myonecrosis: A Genomic Key to a Rare Disorder

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Introduction: Necrotizing myositis is a rare aggressive muscle disorder that often mimics infectious or autoimmune pathology. Genetic causes are seldom identified, making diagnosis complex

Case Report: A 49-year-old male presented with recurrent exertional thigh and calf pain, swelling, and transient fever for six months. MRI showed waxing and waning multifocal myonecrosis, while labs revealed elevated ESR, CRP, CPK (300–5046), and LDH, with normal liver enzymes and negative autoimmune/infectious panels. EMG/NCV indicated sensory motor axonal neuropathy but no myopathy. Genetic testing identified a heterozygous likely pathogenic PGAM2 variant, consistent with Glycogen Storage Disease X, a rare metabolic myopathy. The episodic nature, exertional trigger, and resolution with rest distinguished it from inflammatory myositis. Management remains supportive—rest during episodes, analgesics, hydration, multivitamins, and avoidance of strenuous exertion. Genetic counseling is advised.

Conclusion: The case illustrates the diagnostic challenges of necrotizing myositis, where infectious and autoimmune causes were initially suspected. It underscores the importance of genomic testing in unexplained atypical recurrent myonecrosis.

PC21: Parvovirus B19–Like Febrile Anaemia and Polyarthritis Following TNF-α Inhibitor Initiation in HLA-B27 Positive Spondyloarthropathy: A Diagnostic Challenge

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Introduction: Biological agents like TNF-α antagonists are potent treatment options for treating spondyloarthropathy, but there is risk of infectious complications and haematological complications. While bacterial and tubercular infections are well-documented, viral infections like Varicella, reactivation of Hepatitis B also seen with TNF-α antagonists. Clinical case reports, such as instances of reactivation or occult infection associated with Varicella virus, hepatitis B virus (HBV) and Parvovirus B 19 are present.

Case Report: Objectives To describe a case of pyrexia of unknown origin with anaemia and large joint synovitis occurring after adalimumab therapy. Methods The patient underwent comprehensive evaluation including clinical examination, laboratory investigations, synovial fluid analysis, imaging, microbiological studies, and bone marrow histopathology to determine the aetiology of fever and anaemia.

Conclusion: Results A 33-year-old HLA-B27 positive male with persistent low back pain and multiple joint pain of inflammatory type for 3 years, MRI lumbar spine inferred as sacroiliitis so diagnosed with spondyloarthropathy developed high-grade intermittent fever (102°F) with chills and synovitis shortly following his initial adalimumab dose and had received intraarticular steroid to knee a day prior. Patient

Fig.1

Category	Investigation	Result / Trend
Hematology	Hb	12.1 → 9.3 g/dL (Anemia)
	TLC / Platelets	11,500 / 5.63 lakh (↑)
	CRP / ESR	161 mg/L / 29 mm/hr
Iron profile	Ferritin	836–990 ng/mL (↑)
	Serum Iron	27.9 (Low)
Infection	Cultures	All negative
	Brucella IgG	Positive (11.88)
Synovial Fluid	Cytology	Paucicellular, neutrophil predominant
Imaging	CT/HRCT	Normal
	MRI SI + Hip	Inflammatory involvement
	PET-CT	Diffuse marrow FDG uptake
Bone Marrow	Findings	Erythroid hypoplasia, M:E 8:1
Virology	Parvovirus B19 PCR	Detected – high viral load

Pc22: Comparison of Lateral DXA with AP DXA in Predicting Low Bone Density in Patients with Ankylosing Spondylitis (AS)

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Introduction: Routine AP DXA scan underscores the prevalence of osteoporosis in AS hence we planned this study to compare Lateral DXA with AP DXA in AS

Methods: It was a cross sectional observational study done in 80 cases with AS. Lateral DXA and AP DXA was done along with biochemical tests

Results: The mean age of 80 cases was 31.1 ± 9.7 years and BMI was 23.7 ± 3.25 mg/m2. Average BASDAI was 4.27 ± 1.93 and 52.5 % patients had low disease activity. Schober's test was positive in 55% of patients. The mean mSASSS was 9.30 ± 12.98 and correlated with lateral and not AP DXA (rho= - 0.65, p <0.001). 78% of cases had X-ray abnormalities in the lumbar spine. BMD by lateral LS DXA was found to be 0.69 ± 0.13 g/cm2 as compared to 0.88 ± 0.12 g/cm2 by AP DXA (P < 0.001). Low BMD by lateral DXA was found to be associated with age, duration of disease, EMS, disease activity, ESR, CRP, mSASSS and X-ray hip findings. All cases with erosions or squaring of vertebra had low BMD by lateral DXA but only

58% by AP DXA

Conclusion: AP DXA grossly under estimates bones loss in AS and should be supported by lateral DXA in accurate management of these individuals

PC23: Descriptive Study of Clinical Features, Autoantibody Profile, and Treatment Patterns in Overlap Myositis: A Retrospective Analysis

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Introduction: Idiopathic inflammatory myopathies include overlap myositis (OM), a heterogeneous, under-recognized subset associated with connective tissue diseases. This study characterizes the clinical spectrum, serological features, extramuscular involvement, biopsy findings, and treatment patterns of OM in a tertiary care cohort

Methods: We conducted a retrospective longitudinal analysis of patients with overlap myositis (OM) at NIMS, included within the MYOIN cohort. OM was defined by fulfillment of Bohan and Peter criteria for idiopathic inflammatory myopathies along with classification criteria for a concomitant connective tissue disease—systemic lupus erythematosus, systemic sclerosis, mixed connective tissue disease, rheumatoid arthritis, or primary Sjögren's syndrome. Demographic, clinical, laboratory, serological, histopathological, and treatment data were analyzed.

Results: 64 patients were included (94% female), with mean age 37 ± 11 years and median disease duration 6 years. Associated CTDs were SLE (n=34), SSc (n=17), mixed connective tissue disease (n=10), and Sjögren's syndrome (n=1). Arthritis (60.9%), constitutional symptoms (68.7%), and myalgias (67%) predominated. Interstitial lung disease occurred in 15%, bulbar involvement in 9%, and pericarditis in 3%. Mean creatine phosphokinase was 1229 U/L, although 10% had normal values. Myositis-specific antibodies were detected in 44 patients, most commonly anti-Mi2 and anti-PL7, while anti-Ro52 and anti-Ku were frequent myositis-associated antibodies. Muscle biopsy was done in 13 (20%) which demonstrated predominantly non-specific inflammatory changes.

Conclusion: OM represents a clinically heterogeneous IIM subset strongly associated with SLE and SSc. Predominant arthritis, frequent anti-Ro52 positivity, and non-specific biopsy findings highlight diagnostic complexity. Larger comparative studies are essential to refine classification and improve clinical stratification.

PC25: Association Between Haematological Indices and Measure of Disease Activity In Patients with Rheumatoid Arthritis (RA)

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Introduction: Disease activity in RA is conventionally assessed using composite indices such as DAS28-CRP and CDAI. However, their

routine application may be limited in resource-constrained settings. Haematological indices derived from CBC, including NLR, PLR, MPV, and SII are inexpensive markers of systemic inflammation .

Methods: The study aimed to evaluate the association of NLR, PLR, MPV, and SII with disease activity assessed by DAS28-CRP and CDAI; to assess their relationship with inflammatory markers C-reactive protein CRP & ESR. A cross-sectional analytical study was conducted from May 2024 to November 2025 at AIIMS Nagpur. Adult patients with RA diagnosed as per 2010 ACR/EULAR criteria or previously diagnosed and on treatment were included in study.

Results: A total of 204 patients were enrolled, including 177 females (87%) and 27 males (13%), with a mean age of 49.96 ± 12.75 years. Most patients had moderate disease activity by DAS28-CRP (59.8%) and CDAI (54.4%). MPV demonstrated a significant progressive increase across disease activity categories by both scoring systems ($p < 0.0001$). MPV and SII showed consistent associations with DAS28-CRP and CDAI. SII correlated positively with CRP, while MPV showed the strongest association with ESR. MPV demonstrated good discriminatory performance for predicting ACR/EULAR remission (AUC 0.799).

Conclusion: MPV and SII are significantly associated with RA disease activity and inflammatory burden. MPV emerged as the most consistent and clinically useful hematological marker, supporting its role as a cost-effective adjunct for disease monitoring.

PC26: Demyelinating syndrome in systemic lupus erythematoses

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Introduction: Systemic Lupus Erythematosus (SLE) with acute myelitis or optic neuritis is rare and causes significant morbidity. Data on these conditions remain limited. This study aimed to evaluate the clinical profile and outcomes of patients presenting with demyelinating syndromes (transverse myelitis or optic neuritis) in SLE.

Methods: A retrospective chart review was conducted, identifying patients diagnosed with SLE and myelitis or optic neuritis at our centre between 2000 and 2023. SLE was diagnosed using ACR-EULAR 2019 criteria. The Expanded Disability Status Scale (EDSS) was used to assess disability levels. Data were analysed using descriptive statistics.

Results: In a cohort of 25 female patients (mean age 27.04 ± 9.69 years) with SLE and demyelination, 24% presented with demyelination as the initial manifestation. Mean SLEDAI was 17.72 ± 6.88. Acute severe symptoms peaked within 24 hours in 64%, including motor deficit (88%), sensory involvement (88%), urinary retention (72%), and optic neuritis (16%). All had abnormal MRI, with thoracic cord lesions in 88% and longitudinally extensive myelitis in 53%. ANA was positive in all; 76% had low complements, and 12% were AQP4-positive. Most (92%) received pulse methylprednisolone, and 68% cyclophosphamide. Severe disability (EDSS >5) decreased from 72% at baseline to 12% at 6 months. Three relapsed, one died from sepsis, and

no optic atrophy occurred within one year.

Conclusion: Demyelinating syndromes in SLE primarily affect young females and correlate with high disease activity. Early immunosuppressive therapy leads to significant improvement, though some patients retain residual disability, underscoring the need for prompt intervention.

Fig.1

Table 1: Patient Demographics and Clinical Characteristics

Characteristic	Value
Number of patients	25
Sex	100% (25/25) female
Age (years, mean ± SD)	27.04 ± 9.69
SLEDAI (mean ± SD)	17.72 ± 6.88
Demyelination as initial SLE manifestation	24% (6/25)
Time to nadir ≤ 24 hours	64% (16/25)
Motor deficit	88% (22/25)
Sensory level involvement	88% (22/25)
Urinary retention	72% (18/25)
Optic neuritis	16% (4/25)
Thoracic cord involvement (MRI)	88% (22/25)
Longitudinally extensive myelitis (MRI)	53% (13/25)
ANA positive	100% (25/25)
Low complements	76% (19/25)
Aquaporin-4 antibody positive	12% (3/25)

Fig. 2

Table 1: Treatment and Outcomes

Treatment/Outcome	Value
Pulse methylprednisolone	92% (23/25)
Cyclophosphamide	68% (17/25)
Severe disability at baseline (EDSS > 5)	72% (18/25)
Severe disability at 6 months (EDSS > 5)	12% (3/25)
Relapse after treatment	12% (3/25)
Mortality (sepsis)	4% (1/25)
Optic atrophy within 1 year (optic neuritis patients)	0% (0/4)

PC27: Assessment of Hepatic Stiffness in patients with Rheumatoid Arthritis on long term Methotrexate therapy

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Introduction: Methotrexate remains the anchor DMARD in rheumatoid arthritis. However, concerns regarding methotrexate-associated hepatotoxicity continue to influence long-term therapy decisions. Non-invasive assessment of hepatic fibrosis using elastography offers a potential alternative to liver biopsy.

Methods: This cross-sectional observational study was conducted at a tertiary care centre in North India. Adult patients fulfilling the 2010 ACR/EULAR criteria for RA and receiving uninterrupted methotrexate therapy for ≥36 months were included. Patients with obesity , alcohol use, diabetes mellitus, viral hepatitis, or other chronic liver diseases were excluded. Hepatic stiffness was assessed using 2D shear-wave elastography. Clinical, biochemical, and disease activity parameters were analysed for association with hepatic stiffness.

Results: The median duration of methotrexate therapy was 60 months, with a mean cumulative dose of 3796 ± 2184 mg. The mean liver stiffness value was 6.0 ± 2.11 kPa, with 21.8% of patients demonstrating stiffness values more than 6 kPa. No statistically significant association was observed between hepatic stiffness and cumulative methotrexate dose, duration of therapy, liver enzymes, disease activity indices, or inflammatory markers.

Conclusion: Long-term low-dose methotrexate therapy in rheumatoid arthritis is not associated with a significant increase in hepatic stiffness. 2D shear-wave elastography appears to be a useful non-invasive modality for hepatic monitoring, supporting the continued safe use of methotrexate in appropriately selected patients.

PC28: Descriptive study of clinical features autoantibody profile and histopathological features in a cohort of IMNM

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Introduction: Immune-mediated necrotizing myopathy (IMNM) is an autoimmune myopathy characterized by subacute onset, rapidly progressive proximal weakness, and muscle biopsy showing myofiber necrosis and regeneration with minimal inflammation. It is commonly associated with anti-HMGCR and anti-SRP antibodies.

Methods: This cross-sectional observational study was conducted at a tertiary care center in Hyderabad over one year. Newly diagnosed patients fulfilling the 2017 ACR/EULAR criteria for idiopathic inflammatory myopathies and subclassified as IMNM were included. Clinical data, muscle enzymes, antinuclear antibodies, and myositis-specific antibodies were recorded. Muscle biopsy cryostat sections underwent routine histopathology and IHC for MHC-I and type I interferon–inducible markers.

Results: Eighteen patients were analyzed. The mean age was 44 ± 17.3 years, and 50% were female. Bulbar involvement was present in 28%. The median creatine phosphokinase level was 7,817 U/L, and the mean MMT-8 score was 35.5 ± 8.6. Four patients were anti-SRP positive, two anti-HMGCR positive, and 12 were seronegative.Muscle biopsy showed universal necrosis, with degeneration and regeneration in 72%.Perivascular Inflammation was seen in 3 patients, perifascicular atrophy in one. ISG-15 and RIG-I positivity were each observed in 2 patients, while MxA was negative in all. MHC-I overexpression was present in 13 patients. During follow-up, 2 patients died, 3 improved with tapering immunosuppression, and 4 remained stable on maintenance therapy.

Conclusion: IMNM demonstrated heterogeneous pathological features with variable interferon pathway activation. Early diagnosis and comprehensive biopsy evaluation may guide prognosis and optimize immunosuppressive management

PC29: Rheumatic Disorders of Hand and Shoulder in Patients of Type 2 Diabetes Mellitus and Their Correlation with Microvascular Complications

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Introduction: Diabetes mellitus (DM) is known to cause several chronic systemic complications. But what often remains neglected and undernoticed are the various musculoskeletal manifestations which lead to considerable morbidity and functional disability, significantly compromising the patient's quality of life.

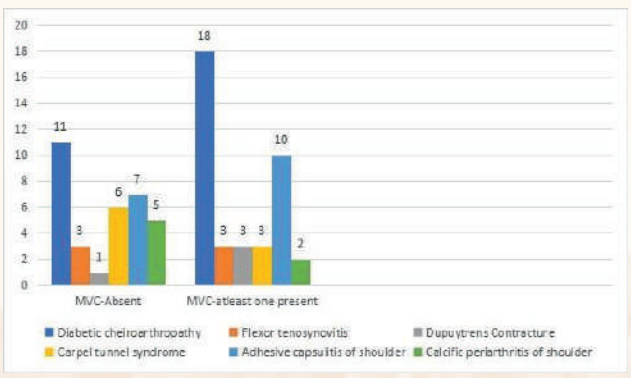
Methods: A single-center cross-sectional study was done after enrolling 100 patients of type 2 DM who were subjected to detailed history taking and underwent systemic clinical, biochemical, and radiological evaluations as indicated

Results: There was increased prevalence of rheumatic disorders of hand and shoulder in 63% patients with significant association with age,

duration of diabetes, and poor glycemic control. Diabetic cheiroarthropathy was the most common disorder seen and was found in 29% patients. Other lesions were adhesive capsulitis of shoulder (17%), carpal tunnel syndrome (CTS) (9%), calcific periarthritis of shoulder (7%), flexor tenosynovitis (6%), and Dupuytren's contracture (4%). There was no case of reflex sympathetic dystrophy . There was increased prevalence of microvascular complications in patients with hand and shoulder disorders, but the increase was not statistically significant. Patients with diabetic cheiroarthropathy had statistically significant correlation with microvascular complication

Conclusion: Better understanding of burden of hand and shoulder disorders in diabetic patients may create awareness regarding prompt diagnosis and subsequent management. However, larger studies are required to consolidate the above findings which may lead to early diagnosis, appropriate treatment, and improved quality of life.

Fig.1



PC30: Effectiveness of Hand Orthotic Intervention in Reducing Pain and Deformity in Chronic Inflammatory Arthritis: A Retrospective Analysis

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Introduction: Hand involvement in chronic inflammatory arthritis causes persistent pain, progressive deformity, and functional disability. Despite adequate pharmacological control, mechanical pain and deformity-related impairment often persist, highlighting the need for effective rehabilitation interventions such as hand orthoses.

Methods: A retrospective analysis of 42 patients with chronic inflammatory arthritis prescribed customized hand orthoses under a PMR-led rehabilitation program was performed. Clinical records were reviewed for demographics, deformity type, and orthosis used. Pain (VAS) and functional disability (HAQ-DI) were compared at baseline and 12-week follow-up.

Results: A significant reduction in pain intensity was observed following hand orthotic use (mean VAS reduction 2.0 ± 0.8; $p<0.001$). Functional disability also improved significantly, with a reduction in HAQ-DI scores ($p<0.01$). Most patients demonstrated stabilization or subjective improvement in deformity-related symptoms, including improved hand alignment and grip stability during daily activities. No major adverse events were reported.

Conclusion: Hand orthotic intervention is a safe and effective rehabilitation strategy for reducing pain and deformity-related functional limitation in chronic inflammatory arthritis. Early integration of PMR-guided hand orthoses into routine rheumatologic care may significantly improve disability-focused outcomes.

PC31: Early Musculoskeletal Symptom

Evaluation in the Indian Setting: A First-Contact Perspective on Myositis and Psoriatic Arthritis Mimics

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Introduction: Early musculoskeletal symptoms in India often arise from nutritional, metabolic, environmental, and lifestyle factors rather than established autoimmune disease. A pragmatic, risk-based first-contact approach may improve early management, reduce over-investigation, and enable appropriate referral for psoriasis.

Methods: A practice-based observational framework was developed for first-contact evaluation of patients presenting with arthralgia and early arthritis-like symptoms. Structured risk stratification across nutritional, environmental, metabolic, and genetic domains was applied in routine internal medicine outpatient practice. The framework was refined iteratively through recurring clinical patterns and illustrated using representative clinical vignettes, with outcomes assessed qualitatively based on symptom response, investigation requirements, and referral.

Results: Application of structured risk stratification frequently identified non-inflammatory and potentially reversible contributors to musculoskeletal symptoms, including vitamin D and B12 deficiency, metabolic syndrome, gastrointestinal malabsorption, lifestyle-related hyperuricemia, occupational biomechanical stress, and chronic dermatological or infectious conditions. Early emphasis on nutritional correction, lifestyle modification, ergonomic interventions, and patient education was associated with symptomatic improvement in a substantial proportion of patients, while allowing selective investigation and timely referral for suspected inflammatory disease. This approach reduced repeated testing, treatment escalation, and therapeutic switching across medical systems.

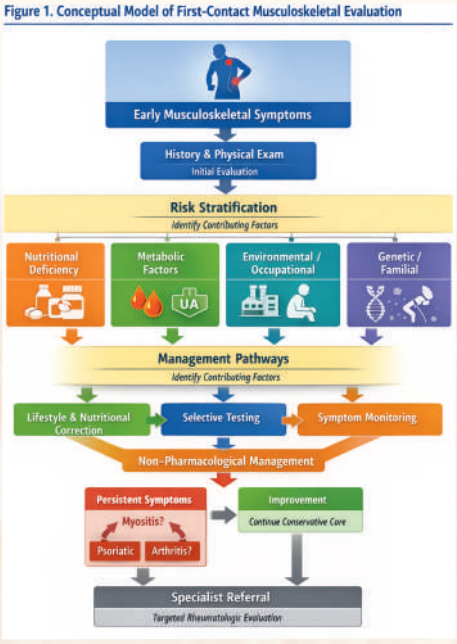
Conclusion: Early evaluation of musculoskeletal symptoms in an Indian setting requires a context-aware, risk-based approach that complements existing rheumatology guidelines.Incorporating nutritional, metabolic, environmental, and lifestyle assessment at the first-contact level can help distinguish mimics of myositis and psoriasis

Fig.1

Risk-Factor–Based Early Evaluation Framework for Musculoskeletal Symptoms

Domain	Key Factors Assessed	Clinical Implication
Nutritional	• BMI, protein intake, • vitamin D/B12 status, anemia	Deficiency-related arthralgia, myalgia, early OA
Environmental / Occupational	• Air pollution, repetitive joint use, posture, ergonomics	Mechanical pain, back pain, enthesopathy
Metabolic	• Diabetes, dyslipidemia, obesity, hyperuricemia, thyroid disease	Gout, metabolic arthropathy
Genetic / Familial	• Family history of autoimmune disease	Risk awareness, not sole diagnostic anchor

Fig. 2



Pc32: Peripheral and Extra-musculoskeletal Manifestations in Adult Patients of Spondyloarthritides

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Introduction: The primary aim of the study was to investigate the peripheral and extra-musculoskeletal manifestations in adult patients diagnosed with spondyloarthritides (SpA), focusing on the frequency of these manifestations and their association with specific types of SpA.

Methods: This cross-sectional, observational study was conducted at a tertiary care hospital in New Delhi, including 70 adult patients with SpA. Subjects were selected based on the 2009 ASAS classification criteria for SpA. Clinical assessments included demographic data, disease history, treatment details, and comorbid conditions. The Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) was used to evaluate disease activity, and patients were screened for extra-musculoskeletal manifestations, including enthesitis, dactylitis, uveitis, psoriasis and IBD.

Results: Among the 70 participants, 57.14% exhibited extra-musculoskeletal manifestations, with enthesitis (24.29%) being the most prevalent. There were significant associations between specific SpA subtypes and the presence of extra-articular manifestations, such as psoriasis and inflammatory bowel disease. Patients with peripheral SpA showed a higher incidence of psoriasis, while those with axial SpA were more likely to develop uveitis.

Conclusion: The findings highlight that extra-musculoskeletal manifestations are common in patients with SpA, particularly enthesitis. The study emphasizes the need for a comprehensive clinical approach in managing SpA, taking into account the full spectrum of peripheral and extra-articular features to improve patient outcomes.

PC33: Association of Epicardial Fat Thickness with Carotid Intimal media Thickness in Predicting Cardiovascular Risk in Patients with Systemic Lupus Erythematosus

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Introduction: SLE is an autoimmune disease which considered as high risk for cardiovascular disease (CVD) Premature CVD has emerged as a major cause of morbidity and mortality in SLE. We tried to compare Epicardial fat thickness (EFT) with carotid intimal media thickness (CIMT) in predicting cardiovascular risk in patients with SLE

Methods: A cross-sectional observational study done in 60 cases with SLE and 60 controls.2-D Echocardiography along with pulse wave Doppler was performed in all cases and controls and EFT was measured. EFT >5mmwas considered abnormal on ECHO. Measurement of C-IMT was done by B-mode ultrasound and (CIMT >0.6 mm was considered abnormal)

Results: The prevalence of cardiovascular disease (by CIMT) in patients with SLE was 56.67% and 10% in controls. prevalence of CVD by EFT in patients with SLE was 51.67% and the mean EFT of cases was 4.89 mm+ -1.1, which was

significantly higher as compared to 3.91mm + -0.68 (p<.001) in controls. The mean Average CIMT in case was 0.62+-0.14mm which was significantly higher as compare to controls 0.5+-0.09mm (p <.000)1. The mean EFT in controls with high CIMT >0.6MM was 4.17+-0.77 which was higher as compared to 3.88+-0.67MM in controls with normal CIMT

Conclusion: EFT in patients with SLE was found to be significantly higher than control. In patients with SLE, EFT >5.2mm had high sensitivity and specificity (85.3% & 92.3% respectively) for predicting cardiovascular risk in patients with SLE.

PC34: Clinical study of the correlation of obesity with inflammation and quality of life in OA, RA, SPA and PSA patients treated with Tirzepatide

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Introduction: Obesity increases inflammation and mechanical load in rheumatic diseases. This study evaluates Tirzepatide's impact on quality of life (RAPID 3) and inflammatory markers (ESR, CRP). DMARDS "dose-sparing" potential of Tirzepatide in rheumatic diseases needs to be explored.

Methods: 45 obese patients (BMI ≥ 30) with OA, RA, SpA, or PsA were treated with Tirzepatide doses (2.5mg–7.5mg). Primary endpoints included RAPID 3 scores and inflammatory markers (ESR, CRP). Data was analyzed using Linear Mixed Models in SPSS to analyse longitudinal trends. Categorical quadrant analysis identified "Dissociation Groups" (clinical improvement despite minimal weight loss). Binary Logistic Regression was utilized to standardize the effect of DMARDS in RAPID 3 improvement.

Results: 45 obese patients OA(37.8%), RA(24.4%), SpA(26.7%) and PsA(11.1%) were treated with Tirzepatide doses (2.5mg–7.5mg). Tirzepatide significantly reduced weight (97.9 to 91.4 kg, p<0.001) and BMI (38.5 to 37.4 kg/m2, p<0.001) within 8-12 weeks. RAPID 3 scores improved from 4.41 to 2.91 (p=0.0002). CRP 13.4mg/l dropped to 7.8 mg/l at 7.5 mg [Table 1]. Binary logistic regression analysis identified dose titration (OR 3.42) as the strongest predictor of QOL improvement. 26.7% of patients successfully tapered DMARDs/biologics without flare. A "dissociation group" (15.8%) achieved significant pain improvement despite minimal weight loss (<5% BMI reduction), suggesting benefits beyond weight loss [Figure 1].

Conclusion: Tirzepatide titration significantly improves quality of life in obese patients. The benefits could be weight reduction, reduction in inflammation and tapering of DMARDS.

PC35: Concurrent Psoriatic Arthritis and Multiple Myeloma – Therapeutic Challenges in a Resource-Limited Setting

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Introduction: Psoriatic arthritis involves IL-17/IL-23 axis dysregulation, while multiple myeloma features monoclonal plasma cell proliferation causing bone lesions, renal

impairment, and immunosuppression.Their coexistence is rare, though psoriasis/PsA ranks among common autoimmune comorbidities in MM registries

Case Report: The patient, diagnosed with PsA at age 36, developed MM at ≈43, confirmed by bone marrow plasmacytosis, bone pain, and reduced eGFR. Induction with VCD/CyBorD achieved near-complete remission and psoriatic lesion regression. Maintenance includes intermittent bortezomib ± dexamethasone; ASCT not performed (latest follow-up December 2025).(4,5). Post-dexamethasone withdrawal, severe PsA flare occurred with arthropathy, enthesitis, and joint damage(3).Challenges: unvaccinated status, cumulative immunosuppression, renal impairment, and financial limits. Secukinumab was preferred for PsA efficacy and reassuring malignancy profile (preclinical anti-MM potential) but unaffordable. Renal-adjusted tofacitinib provided symptom control despite theoretical JAK-STAT concerns in MM.(5,6) Vaccinations (COVID-19, VZV, PCV-13, PPSV-23) recommended; multidisciplinary monitoring ongoing.

Conclusion: This rare PsA-MM overlap underscores real-world constraints. While secukinumab aligns ideally with pathophysiology and potential MM benefits, cost favored tofacitinib. Pragmatic renal-adjusted dosing yielded acceptable outcomes. Multidisciplinary follow-up is essential for monitoring complications .

PC36: A Rare Case of Antibodies negative Myasthenia gravis

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Introduction: Myasthenia gravis (MG) is a chronic autoimmune disorder where antibodies disrupt communication at the neuromuscular junction. This case explores a middle-aged woman presenting with episodic dysarthria and lower limb weakness, highlighting the diagnostic challenge of AChR-negative presentations and the importance of identifying specific antibody subtypes to guide effective treatment

Case Report: This case describes a 42-year-old woman presenting with fatigable dysarthria and proximal lower limb weakness. Her symptoms follow a classic diurnal pattern, worsening with exertion and improving with rest. Clinical examination revealed a "vocal fade" during counting and ptosis (eyelid drooping) elicited by sustained upward gaze. The diagnosis of Myasthenia Gravis (MG) was confirmed despite a negative AChR antibody test. Instead, the patient tested positive for MuSK antibodies, which frequently present with prominent bulbar (speech and swallowing) symptoms. The Ice Pack Test and Repetitive Nerve Stimulation provided immediate supportive evidence of a neuromuscular junction disorder, while a CT scan ruled out thymoma. Differential diagnoses like Multiple Sclerosis and LEMS were excluded due to normal reflexes and the absence of sensory deficits. The patient was managed with corticosteroids, a common first-line therapy, resulting in significant functional recovery and stabilization of her speech.

Conclusion: This case describes a 42-year-old woman presenting with fatigable dysarthria and proximal lower limb weakness. Her symptoms follow a classic diurnal pattern, worsening with exertion and improving with rest. Clinical

examination revealed a "vocal fade" during counting and ptosis (eyelid drooping) elicited by sustained upward gaze.

POM38: A case of Mechanic's hand with Hypokalemic paralysis: Probable Antisynthetase or Sjogren's!

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Introduction: Distal RTA (dRTA) with hypokalemia is a known complication of Sjogren's disease. Antisynthetase syndrome (ASyS) often presents with inflammatory myositis or cutaneous manifestations or ILD. We present a case of ASyS that presented with features of proximal myopathy secondary to hypokalemia but was diagnosed as Probable ASyS after clinical examination.

Case Report : A 55-year-female presented with a 4-month history of gradually progressive proximal muscle weakness in all 4 limb, intermittent small-joint arthralgia, painless recurrent oral ulcers, alopecia, and a violaceous rash over the knuckles for the past 5–6 months. No other significant history. Examination showed Gottron's papules, and fissuring over the palms suggestive of mechanic's hands, hypotonia, normals DTRs & sensation. Investigations showed ESR 66 mm/hr, severe hypokalemia (1.86 mEq/L), total CPK 1192 U/L, non-anion gap metabolic acidosis on ABG suggesting distal RTA. ANA IIF 1:100, Anti-Ro and anti-La antibodies positive. Anti-dsDNA, C3, C4, RA, ACPA, MSA negative, Lip biopsy was normal. A diagnosis of probable Anti-synthetase syndrome with distal RTA causing hypokalemic paralysis was made. The patient responded successfully to treatment with oral potassium citrate, IV MPS pulse followed by oral prednisolone @1mg/kg with tapering & Methotrexate. After 2 months of follow up, her cutaneous manifestations also resolved.

Conclusion: Inclusion of Anti-Ro52 antibodies in the EULAR-ACR Classification criteria for ASyS facilitates the diagnosis of Probable ASyS. Our patient had a rare clinical presentation but thorough clinical examination led us to diagnose it as Probable ASyS rather than Sjogren's disease. The differentiation is clinically relevant as it helps guide planning immunosuppression.

PC24: Challenging the Exclusivity of Myositis Antibodies: A Clinical Study

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Introduction: Myositis specific antibodies (MSAs) are traditionally considered mutually exclusive within individual patients. This study aimed to evaluate the coexistence of multiple MSAs in a diverse cohort of patients diagnosed with idiopathic inflammatory myopathies (IIM).

Methods: We retrospectively analyzed 103 IIM patients using commercial immunoblot assays. We calculated the prevalence of "multipositivity" (coexistence of ≥2 MSAs) using two thresholds: low-stringency (any positivity ≥1+) versus high-stringency (strong positivity ≥2+). Clinical phenotypes were correlated with antibody profiles

Results: Among 103 IIM patients, juvenile dermatomyositis was most common (28.2%),followed by dermatomyositis (22.3%), polymyositis (14.6%), clinically amyopathic dermatomyositis(12.6%), antisynthetase syndrome(10.7%), overlap myositis(9.7%), and inclusion body myositis (1.9%). At the low threshold (≥1+), 50.5% of patients exhibited multipositivity. However, restricting analysis to strong positives (≥2+) significantly reduced the multipositivity rate to 21.4%. The most frequent high-titer combination was Mi-2α+Mi-2β (32.6%), representing epitope redundancy rather than distinct autoimmunity. True distinct overlap (e.g., Mi-2α+PL-7) was less frequent (13.4%). Among multipositives, 67.3% had 2 antibodies. In cases of double positivity, the clinical phenotype aligned with the dominant, high-titre MSA.

Conclusion: While commercial immunoblots often report multiple antibodies, high-titer distinct MSA coexistence is rare. The sharp reduction in multipositivity at higher titers suggests significant false-positive rates at 1+ levels. Clinicians must interpret signal intensity to avoid diagnostic errors in "multipositive" reports.

POR01: The Werewolf in the Clinic: Pseudo-enthesitis as the Deceptive Face of Disseminated Tuberculous Pyomyositis in Systemic Sclerosis.

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Introduction: In patients with AIRDs the synergy of disease-mediated immune dysfunction and immunosuppressive therapy significantly increases vulnerability to opportunistic infections. Distinguishing such infections from autoimmune flares remains a diagnostic dilemma, often complicated by atypical clinical presentations. We describe one such deceptive clinical sequence in systemic sclerosis

Case Report: A 40-year-old gentleman with systemic sclerosis and ILD(Anti-Scl-70+)—initially managed with Methotrexate, Prednisolone, SAAZ, and MMF at an outside facility—presented four months later to our hospital with recurrence of fever, disabling joint pain, generalised weakness and a significant weight loss (16kg). Clinical evaluation revealed pallor, axillary lymphadenopathy, multiple entheseal and adjoining muscle tenderness. Investigations revealed anaemia, neutrophilic leukocytosis, hypercalcemia, and elevated inflammatory markers. Further workup revealed PTH-independent hypercalcemia (Table 1). PET-CT demonstrated entheseal uptake and disseminated skeletal muscle collections (image1), with CBNAAT confirming M. tuberculosis in both sputum and pus. Following the initiation of ATT and NSAIDs, the patient developed drug-induced liver injury (DILI), necessitating a modified ATT regimen with the gradual reintroduction of Rifampicin. He had significant clinical improvement at his one-month follow-up, he developed acute diarrhoea followed by rapid, refractory shock and succumbed to the illness within two days.

Conclusion: This case highlights "pseudo-enthesitis" as a deceptive manifestation of tuberculosis (TB). Although the patient's periarticular pain and weakness clinically mimicked a systemic sclerosis flare with enthesopathy which is a rare manifestation, MSUS ruled out synovitis, and PET-CT revealed

diffuse uptake across entheses and skeletal muscles. These findings identify TB as mimic of this manifestation

POR02: Between the Lines of Lysis: The Diagnostic Dilemma of an Immune vs. Drug-Induced Necrotizing Myopathy

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Introduction : Differentiating between immune-mediated necrotizing myopathy (IMNM) and drug-induced myotoxicity presents a significant diagnostic challenge, particularly in the context of statin therapy. This report details a rare and critical presentation of fulminant myopathy, underscoring the difficulty in achieving a timely and precise diagnosis and initiating appropriate management

Case Report: A 55-year-old male with CAD, HTN, and T2DM, on long-term rosuvastatin (40 mg) and dual antiplatelet (Aspirin+ Clopidogrel), underwent PTCA three weeks before the current symptom onset. Post-procedure, he was initiated on ticagrelor and aspirin while continuing rosuvastatin (40 mg). He presented to us with generalised body aches, progressive muscle weakness, and reduced urine output. Labs confirmed profound rhabdomyolysis. He had anuric AKI requiring multiple sessions of dialysis. Despite withholding the statin, muscle enzymes rose, and weakness progressed to involve the neck muscles. Anti-HMG-CoA reductase and anti-SRP antibodies were both negative. MRI thigh showed only a few areas of enhancement in the right adductor magnus. To resolve diagnostic uncertainty and guide treatment, a muscle biopsy was performed. The course was complicated by refractory hypocalcemia and adynamic bowel obstruction; the patient succumbed to sepsis on Day 10. Biopsy results, reported later, showed extensive fibre necrosis with minimal inflammation, favouring Immune-Mediated Necrotizing Myopathy (IMNM).

Conclusion: While rosuvastatin-induced rhabdomyolysis is rare, interactions with drugs such as ticagrelor or diuretics, etc., elevate the risk. Differentiating toxic rhabdomyolysis from acute IMNM remains a diagnostic challenge, particularly when imaging, serology, and histopathology are discordant. Such ambiguity significantly complicates the management of these life-threatening conditions.

POR03: Spectrum of Rare Neurological Manifestations in Sjögren's Disease: A Five-Case Series

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Introduction: Neurological involvement in Sjögren's disease is heterogeneous, under-recognised, and often misattributed. Presentations may involve the optic nerve, spinal cord, peripheral nerves, corticospinal pathways, or movement circuits. We report five cases illustrating rare neuro-Sjögren phenotypes and apply a structured attribution approach to support diagnosis and treatment decisions.

Case Report: Five patients with objectively confirmed Sjögren's disease presented with distinct neurological phenotypes. Case 1 developed acute bilateral optic neuropathy with marked visual recovery following corticosteroids

and B-cell depletion. Case 2 had relapsing optic neuritis with thoracic myelitis and tumefactive demyelination requiring escalation with mycophenolate and intravenous immunoglobulin, achieving clinical stabilization. Case 3 presented with vasculitic mononeuritis multiplex manifesting as wrist and foot drop, demonstrating significant motor recovery after immunosuppression. Case 4 exhibited a motor neuron disease-like syndrome with corticospinal tract involvement and persistent deficits despite cyclophosphamide and rituximab. Case 5 developed blepharospasm and oromandibular dystonia with negative malignancy work-up, managed as neuro-Sjögren with modest symptomatic improvement. All patients fulfilled objective glandular criteria and underwent systematic exclusion of structural, infectious, ANCA-associated, cryoglobulinemic, and paraneoplastic mimics prior to phenotype-directed immunotherapy.

Conclusion: Sjögren's disease can present with diverse, rare neurological syndromes spanning optic neuropathy, demyelinating disease, vasculitic neuropathy, motor neuron disease–like phenotype, and movement disorders. Early recognition with structured mimic exclusion enables phenotype-directed immunotherapy, preserving vision and function and identifying refractory patterns requiring supportive care.

POR04: Black Digits and Dilated Coronaries in a Young Infant: A Diagnostic Challenge of Atypical Kawasaki Disease

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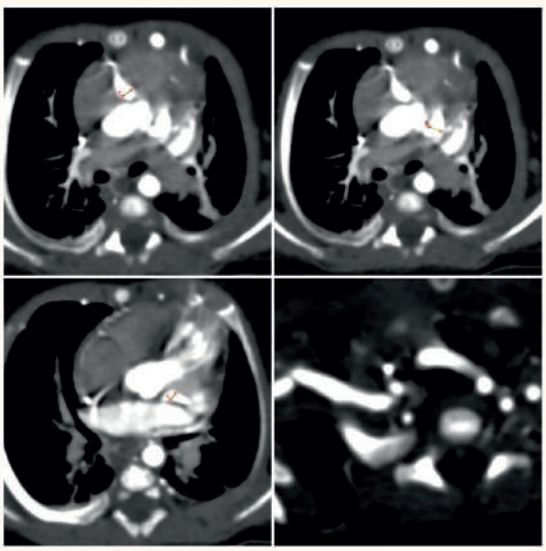
Introduction: Kawasaki disease is an acute systemic vasculitis of childhood with variable clinical presentation. In young infants, absence of classical mucocutaneous features can obscure the diagnosis and delay treatment. We report a rare presentation of atypical Kawasaki disease complicated by peripheral gangrene and giant coronary artery aneurysms

Case Report: A 45-day-old female baby, first born of nonconsanguinous marriage, presented with bluish discoloration progressing to gangrene of bilateral hands and feet following an episode of febrile illness. There were no classical mucocutaneous features of Kawasaki disease. On examination baby was pale, no icterus, no lymphadenopathy, no organomegaly, absent peripheral pulses in both upper, lower limbs, had presence of dry gangrene in both hands and feet. Investigations showed anemia, neutrophilic leukocytosis, hyponatremia, elevated inflammatory markers, negative infective workup, antiphospholipid antibody, autoimmune workup. Echocardiography and CT angiography demonstrated multiple giant coronary vessel aneurysms and other medium-vessel aneurysms. Whole-exome sequencing was negative. The child responded to IVIG, pulse steroids, anticoagulation and antibiotics. Differentials initially considered were atypical Kawasaki, MVV(PAN/DADA2) and Septic vasculitis. Finally based on clinical scenario, investigative workup, diagnosis of Atypical Kawasaki disease made. No new gangrene observed and discharged with tapering dose of steroids, aspirin, supportive measures. The baby was followed up and did not develop new gangrene

Conclusion: This case underscores the protean manifestations of atypical Kawasaki disease in young infants, where severe coronary and

peripheral vascular involvement may occur without classical signs. Early recognition, prompt cardiac imaging, and aggressive immunosuppressive therapy are essential to halt disease progression and prevent irreversible ischemic complications



POR05: Tubercular myositis masquerading as myositis flare in Antisynthetase syndrome patients: A report of five patients

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Introduction: Antisynthetase syndrome is an autoimmune inflammatory myopathy characterized by myositis, arthritis, Raynaud phenomenon, mechanic's hands, fever, and interstitial lung disease. Patients receiving immunosuppressive therapy are vulnerable to opportunistic infections. We report five rare cases of tubercular myositis in antisynthetase syndrome.

Case Report: Five patients (four females and one male), aged 33–61 years, were diagnosed with ASyS, based on the presence of a constellation of symptoms including fever, arthritis, Raynaud's phenomenon, myositis, ILD, and presence of antisynthetase antibodies (Table 1). All patients received systemic immunosuppressive therapy with initial clinical improvement. On follow up (one month to several years), patients presented with progressive muscle weakness, associated with fever and weight loss. A possibility of disease flare was considered and evaluation was performed. Radiological investigations (MRI and FDG PET) showed presence of patchy to diffuse myositis along with presence of lymphadenopathy, lung nodules and meningitis. Possibility of infective etiology was considered and muscle biopsies/FNAC were obtained in all the patients. GeneXpert from the muscle tissue was positive for mycobacterium tuberculosis with rifampicin sensitivity in all the five patients and TB culture was positive in two patients,. All patients were initiated on four drug anti-tubercular therapy with initial clinical improvement. On follow up two patients - improved, one had recurrent flares of ASyS & two patients died.

Conclusion: The association between antisynthetase syndrome and tubercular myositis has not been previously reported. Potent immunosuppressive therapy may predispose to this infection, although other susceptibility factors such as Mendelian susceptibility to mycobacterial diseases were not evaluated. Tubercular myositis mimicked disease flare in all patients, highlighting the need for high clinical suspicion.

POR06: Beyond Autoimmunity: A Diagnostic Deception

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Introduction: Primary Sjögren disease is a chronic autoimmune disorder characterized by sicca symptoms and systemic manifestations. Lymphomas may closely mimic its clinical and serological features, leading to diagnostic confusion. Herein, we present an unique case of T-Cell lymphoma which presented with features of systemic autoimmune rheumatic disease

Case Report: A 45 year old lady presented with fever, skin rashes over bilateral legs , sicca symptoms and weight loss for 1 year. She had past history of purpura over both legs and skin biopsy showed leukocytoclastic vasculitis. On examination she had bilateral parotid enlargement, livedoid rash over bilateral legs. Investigation revealed mild anemia, objective sicca, DCT positivity, ANA profile showing SS-A and PCNA positivity with a negative APS workup and viral markers. Lip biopsy showed lymphocytic infiltration. She was diagnosed as Primary Sjogren disease and treated with steroids and azathioprine. She responded well with steroids, but during follow-up she developed on and off cytopenias which was thought to be due to azathioprine. After 6 months, she was admitted in view of recurrent fever with generalized lymphadenopathy for 1 month. Infective workup were negative. PET-CT demonstrated features of lymphoproliferative disorder. Subsequent lymph node biopsy and IHC revealed T-Cell lymphoma.

Conclusion: T-cell lymphoma can rarely imitate primary Sjögren disease, presenting with sicca features, vasculitis, and autoantibody positivity. Recurrent fever, cytopenias, lymphadenopathy, or unexplained relapses should prompt consideration of lymphoma and early tissue biopsy to avoid diagnostic delay and guide appropriate therapy.

POR07: Recurrent HyperCPKemia in the Setting of Hypereosinophilia: A Diagnostic Enigma

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Introduction: Eosinophilia associated with skeletal muscle involvement is an uncommon and often under-recognized clinical entity. While inflammatory myopathies are classically characterized by proximal muscle weakness and elevated creatine phosphokinase (CPK), the coexistence of marked eosinophilia, recurrent rhabdomyolysis, and renal dysfunction poses a significant diagnostic challenge.

Case Report: A middle aged 43-year-old gentleman with two previous admissions in another hospital with sudden onset bilateral lower limb weakness, haematuria, vomiting and

abdominal pain, when investigated his CPK levels were>20,000. He had also developed acute kidney injury and was treated with hydration and symptomatic management. On evaluation, he had eosinophilia with raised AEC, raised serum IgE levels, UPCR -3.76, but refused renal biopsy, underwent bone marrow biopsy which revealed increase in eosinophils and its precursors. PET CT showed increased FDG uptake in the skeletal muscles? inflammatory myositis. Patient's eosinophilia panel, MPN Panel, myositis panel, MPO-PR3 ANCA, ANA Profile and PNH work up was negative. Muscle biopsy was suggestive of inflammatory myopathy with prominent myonecrosis? immune mediated necrotising myopathy. He was treated with steroids and improved however on follow up he had persistent raised CPK levels with mild fatigue without limb weakness.

Conclusion: Muscle disease is a rare but recognized manifestation of HES. The favourable response to corticosteroids further supports an eosinophil-driven inflammatory process. Persistent hyperCPKemia suggests ongoing subclinical muscle injury, emphasizing requirement of monitoring and steroid-sparing immunosuppression. This case underscores the importance of considering HES-associated myositis after ruling out myositis.

POR08: An Unexpected Culprit: Secondary IgA Nephropathy in Autoimmune Rheumatic Diseases

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Introduction: IgA nephropathy (IgAN) has been well described in Rheumatoid Arthritis (RA) and Spondyloarthritis. Its Occurrence in systemic sclerosis (SSc) and idiopathic inflammatory myopathies (IIM) is rarely reported. Herein, we describe four cases of IgAN occurring in Autoimmune Rheumatic Diseases (AIRD).

Case Report: This single-centre case series included adult patients of AIRD diagnosed with IgA nephropathy between December 2023 - 2025. Following details were extracted: demographics, disease characteristics, laboratory parameters, treatment details. Results: Median age was 61.5 years (45–75), with female predominance (3:1). Autoimmune diseases included RA (n=2), SSc (n=1), and IIM (n=1). Median disease duration at IgAN diagnosis was 39 months (3–192). Majority had sub-nephrotic range proteinuria except one. Renal dysfunction was observed in two. Clinical features, treatment, and outcome details as per Table 1 and renal histopathology abnormalities as per figure 1. Management included ACEi/ARBs and immunosuppressive therapy tailored to underlying disease. At median follow-up (FU) duration of 4 months (1-6), two are in active FU, one lost to FU and one succumbed to illness.

Conclusion: IgAN is an uncommon secondary renal manifestation in AIRD. It can occur either early or late in the disease course. Presentations may vary from asymptomatic urinary abnormality (sub-nephrotic to nephrotic range proteinuria) to renal dysfunction. Recognition of this association is crucial, as it may alter the outcome. This highlights the importance of urine examination at initial assessment\

POR09: Beyond Rash and Eosinophilia: DRESS Presenting as Myositis

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Introduction: Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome is a severe hypersensitivity reaction characterized by fever, rash, eosinophilia, and multiorgan involvement. Myositis is a rare but potentially debilitating manifestation.

Case Report: A 36-year-old female, on alternative medications for small joint pain, developed high-grade fever, loose stools, erythematous rash with desquamation, oral ulcers, facial puffiness, and limb swelling. She later experienced proximal muscle weakness, jaundice, and dark-colored urine. Examination revealed deep icterus, pedal edema, hepatomegaly, bilateral basal crepitations, and tender thighs with reduced muscle strength (power: 3/5, MMT-8 score: 48/70). Laboratory findings showed leukocytosis (TLC: 22,000/mm³), eosinophilia, elevated liver enzymes (SGOT/PT: 1732/1367 U/L), hyperbilirubinemia (T.B: 7.4 mg/dL), and raised inflammatory markers (ESR: 70 mm/hr, CRP: 120 mg/L). MRI of the thighs showed STIR hyperintensities suggestive of myositis. Infectious and autoimmune workups were negative, while a skin biopsy revealed interface dermatitis, consistent with a drug-induced reaction. Elevated absolute eosinophil count (640/mm³) and total IgE (2720 IU/mL) supported a diagnosis of DRESS syndrome. The patient was started on intravenous methylprednisolone (1 g pulse), followed by oral prednisolone (1 mg/kg/day). Within two weeks, her muscle strength returned to normal, and liver function test

Conclusion: This case highlights an atypical presentation of DRESS syndrome with myositis and liver dysfunction. Early recognition and prompt corticosteroid therapy are crucial for improving outcomes. Clinicians should consider DRESS in patients with multiorgan involvement, even without an obvious drug exposure history.

POR10: Inflammatory Myositis–Myasthenia Gravis Overlap: A Case Series

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Introduction: Myositis–MG overlap is rare but clinically significant. Steroid-induced worsening, bulbar symptoms, and respiratory involvement are key clues suggesting coexisting MG, even when electrophysiological studies are negative. We are presenting 2 cases.

Case Report: The first case was a 17-year-old girl who presented with four weeks of history, diagnosed as dermatomyositis. She received oral prednisolone (1 mg/kg/day) and weekly methotrexate. Four weeks later, she developed nasal regurgitation and breathlessness requiring intravenous immunoglobulin, with subsequent clinical improvement. Given deterioration on high-dose steroids and bulbar symptoms, myositis–MG overlap was suspected. AChR antibodies were positive, while repetitive nerve stimulation was negative. Thymoma was excluded. She was treated with rituximab gradual steroid taper; tacrolimus was added later for mild disease flare. The second case was a 29-year-old woman with progressive limb weakness for one year, diagnosed with inflammatory myositis (positive anti-SSA/Ro52, anti-U1-snRNP antibodies) supportive muscle biopsy and MRI, and elevated creatine kinase (2,359 IU/L). Following intravenous methylprednisolone, she developed worsening weakness and nasal regurgitation. AChR antibodies were positive,

RNS was negative, and PET imaging normal. She improved with steroid dose reduction and was started on mycophenolate mofetil.

Conclusion: Early recognition and multidisciplinary management are essential to optimize outcomes, particularly when patients worsen after immunosuppressive therapy.

POR11: Rheumatoid Arthritis Presenting as Focal Myositis: A Rare Case

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Introduction: Focal myositis is a localized inflammatory pseudotumor of skeletal muscle. In RA, immune-mediated inflammation and vasculitic muscle injury may contribute to pathogenesis. MRI helps localize disease, while biopsy confirms diagnosis and excludes malignancy.

Case Report: A 73-year-old male with known Rheumatoid arthritis on conventional disease modifying drugs presented with acute onset pain and swelling of the left calf with no synovitis. There was no history of trauma, fever, or systemic infection. CPK total was mildly raised with raised inflammatory markers. MRI lower limb showed focal muscle enlargement with diffuse T2/STIR hyperintense signal involving calf musculature, suggestive of inflammatory myositis. No abscess, collection, or mass lesion was identified. Findings favored focal inflammatory myopathy. On histopathology, a trucut biopsy from left medial gastrocnemius showed inflammatory myositis with myonecrosis and focal vasculitis, with no evidence of granuloma or malignancy. Microscopy demonstrated skeletal muscle infiltrated by neutrophils, lymphocytes, plasma cells, and histiocytes, along with areas of myofiber necrosis and fibrinoid necrosis of vessel walls. Patient improved after escalation of dose of methotrexate and short course of steroids within weeks of therapy.

Conclusion: Focal myositis with vasculitic changes is a rare extra-articular manifestation of RA. Early recognition prevents misdiagnosis and enables prompt immunosuppressive treatment with good outcomes.

POR12: When Joints Mislead and Arteries Reveal

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Introduction: Polymyalgia rheumatica (PMR) and giant cell arteritis (GCA) represent a clinical spectrum of large-vessel inflammation in older adults. Atypical musculoskeletal presentations can obscure early recognition, particularly when imaging findings are non-classical. Early identification is critical, as delayed diagnosis may result in irreversible ischemic complications, including permanent visual loss.

Case Report: A 68-year-old woman presented with atypical musculoskeletal pain involving the left shoulder, back, hip girdle, and knee, accompanied by markedly elevated ESR and CRP. Late-onset spondyloarthritis was initially suspected despite HLA-B27 negativity. FDG-PET/CT demonstrated a unilateral knee effusion without classical polymyalgia rheumatica-type periarticular bursitis. She showed partial improvement with glucocorticoids and sulfasalazine, supporting the preliminary working diagnosis. However, several weeks later, she developed new-onset headache, scalp

tenderness, jaw claudication, and transient visual blurring. These red-flag symptoms prompted urgent vascular imaging and temporal artery biopsy, which confirmed giant cell arteritis. In retrospect, the disproportionately elevated inflammatory markers, despite atypical imaging findings, represented an early clue toward an evolving PMR–GCA spectrum rather than isolated spondyloarthritis.

Conclusion: Atypical musculoskeletal symptoms with markedly elevated inflammatory markers in elderly patients should prompt consideration of the PMR–GCA spectrum, even when imaging lacks classical features. Close clinical surveillance is essential to identify evolving cranial vasculitis early and prevent irreversible ischemic complications.

POR13: Arthritis, Cytopenia, and Splenomegaly: Autoimmunity or Malignancy?

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Introduction: Rheumatoid arthritis is a systemic inflammatory disorder that may be complicated by cytopenias and splenomegaly, classically described as Felty syndrome. However, chronic immune stimulation also predisposes patients to lymphoproliferative malignancies, which may closely mimic rheumatic disease activity and create significant diagnostic uncertainty in clinical practice.

Case Report: A 33-year-old woman with a three-year history of seropositive inflammatory polyarthritis involving the MCP, PIP, and wrist joints (DIP spared) presented with fatigue, bluish discoloration of both feet, and progressive abdominal distension. She subsequently developed melena and hematemesis. Examination revealed pallor, petechiae, pedal edema, hepatosplenomegaly, and ascites. Laboratory evaluation demonstrated anemia, elevated LDH, hyperferritinemia, polyclonal hypergammaglobulinemia, and markedly elevated rheumatoid factor. ANA was 2+ homogeneous with centromere positivity. Imaging showed hepatosplenomegaly, ascites, omental thickening, and non-necrotic lymphadenopathy. In the context of long-standing rheumatoid arthritis with cytopenias and splenomegaly, Felty syndrome was considered the working diagnosis. However, persistent lymphadenopathy and constitutional features raised concern for an underlying lymphoproliferative disorder. Lymph node biopsy was advised but deferred by the patient, and immunosuppressive therapy was initiated.

Conclusion: Hematologic malignancies remain important mimickers of systemic rheumatic disease. In patients with rheumatoid arthritis and unexplained cytopenias, organomegaly, or lymphadenopathy, lymphoma must be actively excluded. Careful evaluation is essential, as management strategies differ substantially and delayed diagnosis may adversely affect prognosis.

POR14: From Eosinophilia to Infarcts: A Multiorgan Vasculitic Evolution

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Introduction: ANCA-associated vasculitis may present with overlapping allergic, eosinophilic, and systemic inflammatory features. In patients with asthma and marked eosinophilia, progression to multiorgan vasculitic involvement can be rapid and severe. Early recognition is essential, as delayed escalation of therapy may

result in irreversible neurological or renal damage.

Case Report: A 58-year-old man with hypertension and bronchial asthma presented with dry cough, fever, anorexia, and marked peripheral eosinophilia (55%). He subsequently developed cognitive decline. Evaluation revealed proteinuria with active urinary sediment, renal parenchymal disease on ultrasound, and multiple small cerebral infarcts on MRI. p-ANCA was markedly elevated. He was treated with intravenous methylprednisolone followed by oral glucocorticoids and azathioprine, with partial improvement. Shortly after discharge, he re-presented with generalized weakness, distal digit weakness, ankle swelling, and bilateral plantar and dorsiflexion weakness consistent with mononeuritis multiplex. The clinical relapse with progressive neurological involvement suggested active systemic vasculitis, and therapy was escalated to cyclophosphamide for induction of remission.

Conclusion: Asthma with hypereosinophilia and evolving multiorgan dysfunction should raise suspicion for systemic ANCA-associated vasculitis. Early identification and timely escalation to aggressive immunosuppression are crucial to prevent permanent neurological and renal sequelae. Careful monitoring is essential, as relapse may occur despite initial therapy

POR15: A case of Anti-Ku overlap myositis presenting with diffuse alveolar hemorrhage

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Introduction: The presence of anti-Ku antibodies identifies a distinct subset of overlap syndromes associated with severe myositis, myocarditis, and pulmonary involvement. Early recognition is essential due to its rapidly progressive and life-threatening course. We present a case of anti-Ku–positive SLE–myositis overlap syndrome in a young woman with fulminant multisystem involvement.

Case Report: A 22-year-old lady presented with cervical lymphadenopathy and fever followed by rapidly progressive oculopharyngeal and axial muscle weakness,. Lymph node biopsy revealed reactive follicular hyperplasia with immunoblastic proliferation During hospital stay, she developed myocarditis and later DAH requiring mechanical ventilation . Her condition was further complicated by ventilator-associated pneumonia and line-related sepsis with multidrug-resistant organisms. Laboratory findings showed markedly elevated CPK (65,000 U/L) and LDH (2100 U/L), ANA 4+ speckled , anti-Sm and anti-RNP positivity, and low complements (C3: 26, C4: <6 mg/dL). Anti-Ku antibody was strongly positive. Imaging revealed extensive lymphadenopathy, while GeneXpert, EBV ISH, and bone marrow biopsy ruled out infection or malignancy. Echocardiogram showed severe LV dysfunction consistent with myocarditis. She was treated with pulse methylprednisolone, followed by IVIG and seven sessions of plasmapheresis . Despite multiple complications,she had a good biochemical and clinical response . After a prolonged 100-day ICU stay, she was successfully weaned off ventilatory support.

Conclusion: This case illustrates an anti-Ku–positive SLE–myositis overlap with fulminant myocarditis and DAH, complicated by critical illness neuropathy and recurrent sepsis. Prompt immunomodulation with steroids, IVIG,

PLEX with meticulous infection control and rehabilitation, led to gradual recovery. Recognition of anti-Ku syndromes is crucial for early diagnosis and aggressive management to improve outcome

POR16: From VEXAS-Mimic to Neutrophilic Leukemia: The Spectrum of Clonal Hematopoiesis in Rheumatology

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Introduction: Clonal hematopoiesis (CH) refers to the expansion of hematopoietic stem cell clones bearing somatic driver mutations, often in genes like ASXL1, TET2, or DNMT3A. CH is increasingly recognized as a driver of systemic inflammation beyond hematologic disease. We report a case in which CH-associated inflammation presented as large-vessel vasculitis and ultimately revealed chronic neutrophilic leukemia

Case Report: 56-year-old man presented with pyrexia of unknown origin, marked neutrophilic leukocytosis, thrombocytosis, macrocytic anemia, elevated inflammatory markers, and hepatosplenomegaly. PET-CT revealed FDG avidity in the right common carotid artery and aorta, suggesting large-vessel vasculitis. Infectious and autoimmune workups were negative. Bone marrow biopsy revealed hypercellularity without dysplasia; flow cytometry and cytogenetics were normal. Given macrocytosis and the inflammatory phenotype, VEXAS syndrome was initially considered. However, genomic sequencing identified pathogenic ASXL1, SETBP1, and CSF3R mutations. While ASXL1 is associated with CH-driven inflammation, the CSF3R mutation is pathognomonic for Chronic Neutrophilic Leukemia (CNL). The constellation of persistent neutrophilic leukocytosis, granulocytic marrow proliferation, and CSF3R positivity fulfilled WHO criteria for CNL. The vascular uptake likely reflected CH-driven cytokine dysregulation rather than primary autoimmune vasculitis.

Conclusion: This case underscores the importance of considering CH–associated inflammation in patients presenting with vasculitis-like or otherwise unexplained inflammatory syndromes, particularly when accompanied by hematologic abnormalities. In such selected cases, genomic testing can aid in distinguishing clonal inflammatory disorders from primary autoimmune disease and help guide appropriate management.

POR17: A Diagnostic Conundrum of Arthritis, Myositis, and Cutaneous Nodules

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Introduction: The coexistence of arthritis, myositis, and skin nodules presents a diagnostic challenge in Rheumatology.

Case Report: A 40-year-old female with no prior comorbidities presented with a one-year history of inflammatory polyarthritis involving bilateral ankles, knees, wrists, MCPs, and PIPs, associated with myalgias. Over the preceding six months, she developed mild truncal weakness, Raynaud's phenomenon without ulcers or gangrene, and non-tender erythematous nodules over fingertips, nail cuticles, and elbows. Examination revealed synovitis in multiple joints, restricted shoulder movements, diffuse goitre without thyrotoxicosis

and pearly white periungual nodules (coral bead sign). Laboratory evaluation showed hemoglobin 11.4 g/dl, TLC 7700/mm³, platelets 4.34 lakhs/mm³, ALT 103 IU/L, AST 113 IU/L, creatinine 0.42 mg/dl, LDH 454 U/L, and CK 3078 U/L. CRP was 4.73 mg/dl and ESR 57 mm. RF and anti-CCP were positive; ANA was 3+ speckled. X-ray hands showed marginal erosions without joint space narrowing. Myositis-specific antibodies were negative. Skin biopsy revealed dermal histiocytic infiltrates with multinucleated giant cells, positive for CD68 and vimentin, confirming multicentric reticulohistiocytosis. ^{18}F-FDG PET/CT demonstrated inflammatory uptake in multiple joints and muscle groups.

Conclusion: MRH should be considered in patients presenting with arthritis and skin nodules MRH may exhibit myositis and multiple autoantibody positivity Histopathology and immunohistochemistry are crucial for diagnosis

POR18: The Vasculitis Masquerade

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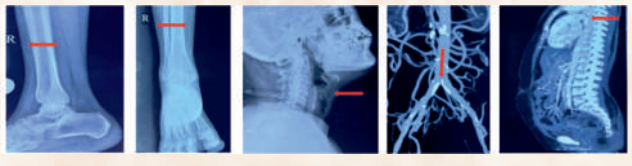
Introduction: Vascular calcification is a pathological process involving calcium phosphate deposition in arterial walls, leading to vessel stiffening and impaired blood flow. Common in aging, diabetes, and chronic kidney disease, it mimics atherosclerosis and vasculitis. Recognizing these rare conditions is critical to avoid unnecessary immunosuppression and optimize treatment.

Case Report: A 52-year-old postmenopausal manual labourer presented with one year of upper and lower limb claudication and three months of right-eye visual blurring. Examination showed absent peripheral pulses except carotids, non-recordable blood pressure, and dental caries; systemic examination was otherwise normal. Ophthalmology revealed a right superior temporal branch retinal vein occlusion. Routine labs, inflammatory markers, autoimmune profile, and capillaroscopy were normal. CT panaortography demonstrated diffuse large-vessel wall calcification with luminal narrowing, while PET-CT showed no FDG uptake. Radiographs and neck imaging confirmed widespread vascular and soft-tissue calcification. Metabolic evaluation showed hyperphosphataemia with normal calcium, PTH, alkaline phosphatase, and vitamin D, reduced urinary phosphate excretion, and normal TMP/GFR. Genetic testing identified a pathogenic GALNT3 mutation, establishing the diagnosis of familial tumoral calcinosis mimicking large-vessel vasculitis.

Conclusion: GALNT3–related hyperphosphataemic calcification can closely mimic large-vessel vasculitis clinically and radiologically. Normal inflammatory markers, absent FDG uptake, and dense calcification should prompt metabolic evaluation, as early recognition prevents misdiagnosis, avoids unnecessary immunosuppression, and enables targeted phosphate-lowering therapy.



POR19: Case Series of SRP1 antibody associated Immune Mediated Necrotizing Myositis

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Introduction: SRP1 (Signal Recognition Peptide) antibodies are rarely found in Immune-mediated Inflammatory Myositis (IIM) and show a strong predilection for the development of necrotizing myositis. It is characterized by rapidly progressive proximal muscle weakness and markedly elevated creatine kinase levels, often exhibits poor response to conventional therapy and requires aggressive immunosuppression.

Case Report: Case 1 A 46-year-old gentleman presented with a 4-month history of subacute onset of proximal muscle weakness, dysphagia, truncal muscle weakness and myalgias, with evidence of myocarditis. Myositis panel was strongly positive for SRP and was diagnosed as IMNM. Extensive malignancy workup was unremarkable. He received pulse steroids and Rituximab. At the 3-month follow-up, he was doing well with improved muscle power. Case 2 A 39-year-old lady presented with subacute onset of proximal muscle weakness and myalgias of 3 months duration and dyspnea on exertion. She had elevated muscle enzymes with MRI suggestive of asymmetric hyperintensity suggestive of muscle oedema in the flexor compartment of bilateral thighs. Myositis profile showed anti-SRP positivity. She was treated elsewhere with pulse steroids and plasmapheresis, but had worsening disease with bilateral lower limb weakness and myocarditis. For which she received IVIG followed by Rituximab. With these measures, she had improvement in her myalgias and there was improvement in muscle weakness.

Conclusion: SRP 1 associated IIM is characterized by a relatively sudden onset of severe pure motor quadriparesis with an increased prevalence of dysphagia and subacute carditis. These 2 cases highlight the seminal features of SRP1-associated IMNM, the knowledge of which leads to expedited clinical diagnosis and timely initiation of appropriate immunotherapy

POR20: Myositis or Mimic? An Intriguing Diagnostic Puzzle

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Introduction: Diabetic myonecrosis is a rare complication of long-standing diabetes, typically presenting with acute thigh pain and normal or mildly elevated creatine kinase levels. However, bilateral symmetrical involvement with associated subcutaneous and intrafascial edema is unusual, posing a significant diagnostic challenge and mimicking inflammatory myopathies.

Case Report: A 50-year-old male with long-standing uncontrolled diabetes mellitus presented with acute-onset severe bilateral thigh pain associated with difficulty rising from a sitting position and climbing stairs. Muscle involvement was bilaterally symmetrical, predominantly affecting the thighs and pelvic girdle, with sparing of the shoulder girdle. He denied fever, rash, sensory symptoms, or cranial nerve involvement. Examination revealed marked thigh tenderness with proximal lower-limb weakness (power 3/5) and preserved deep tendon reflexes; pain was disproportionate to the degree of weakness. Laboratory evaluation showed normal creatine phosphokinase with elevated inflammatory markers and anemia. Autoimmune workup, including antinuclear antibodies and myositis panel, as well as viral markers, was negative. MRI of the thighs demonstrated diffuse symmetric STIR hyperintensities involving the quadriceps

and adductor muscles with associated subcutaneous and intrafascial edema, without abscesses or fatty infiltration. Muscle biopsy revealed myofiber necrosis with edema and neutrophilic infiltrates predominantly in the perimysium, with absence of lymphocytic inflammation or vasculitis.

Conclusion: This case highlights an atypical symmetrical presentation of diabetic myonecrosis and underscores the importance of MRI and histopathology in establishing the diagnosis in patients with normal creatine phosphokinase and other muscle enzymes.

POR21: Beyond Rheumatic Fever: Chorea, Arthritis, Myositis, and Hypermobility in a Child

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Introduction: Children presenting with recurrent chorea and inflammatory musculoskeletal features pose significant diagnostic challenges in pediatric rheumatology. Neurological manifestations, inflammatory arthritis, myositis, and genetic connective tissue disorders may overlap or evolve over time. This case highlights an evolving phenotype, underscoring the importance of longitudinal follow-up.

Case Report: We describe a 13-year-old male with delayed developmental milestones who presented with recurrent inflammatory, neurological, and musculoskeletal manifestations over three years, posing diagnostic uncertainty. The illness began with a clavicular abscess followed by migratory knee arthritis and elevated inflammatory markers. 2 weeks later he developed fever and chorea , raised acute phase reactants, and elevated antistreptolysin-O titre, diagnosed as acute rheumatic fever. Chorea was refractory, requiring corticosteroids, antiepileptics, and intravenous immunoglobulin, with remission on penicillin prophylaxis. After two years, he re-presented with fever, asymmetric arthritis, muscle weakness (asymmetric), and recurrent chorea. Despite elevated inflammatory markers, streptococcal serology, autoimmune markers, and myositis workup were negative. MRI showed patchy STIR hyperintensities (asymmetric). He improved with methotrexate and corticosteroids. Subsequently, generalized joint hypermobility (Beighton score 7/9) and cutaneous hypopigmentation were noted. Whole-exome sequencing revealed a heterozygous for TNXB mutation consistent with Ehler Danlos syndrome classic like 1.

Conclusion: This case illustrates the diagnostic complexity of evolving pediatric presentations with overlapping inflammatory, neurological, and genetic features. Recurrent reassessment and longitudinal follow-up were crucial in identifying a unifying connective tissue disorder, emphasizing the need to reconsider initial diagnoses when clinical phenotypes change over time.

POR22: The rare occurrence of pulmonary artery hypertension in as case of Anti-MDA5 Positive Dermatomyositis: A Case Report

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Introduction: (MDA5) antibody–positive dermatomyositis represents a distinct clinical subset characterized by prominent cutaneous manifestations, minimal muscle involvement, and frequent association with interstitial lung

disease (ILD). Pulmonary arterial hypertension (PAH) is an uncommon but severe complication, likely reflecting underlying immune-mediated vasculopathy.

Case Report: A 30-year-old male presented with constitutional symptoms, arthritis, proximal myalgia, and Gottron's papules of 1.5 months duration. Evaluation revealed normal CK, markedly elevated serum ferritin, LDH. ANA was positive with cytoplasmic staining. MSA revealed 3+ anti-MDA5 antibody. During follow-up, the patient developed worsening dyspnea, progressing to MMRC grade 3 over a period of 2 months The patient was treated with pulse methylprednisolone followed by 1mg/kg prednisolone and tofacitinib, leading to stabilization of ILD and systemic inflammation over the next 6 months and prednisolone could be tapered to 5mg/day. During follow-up, the patient developed new-onset fatigue on exertion. HRCT thorax revealed marked reduction in severity of ILD. However, pulmonary trunk was dilated. CTPA did not reveal any thrombus. 2D echocardiography revealed severe PAH (TR gradient 64 mmHg). APLA was negative. The marked decrease in severity of ILD suggested that PAH was not secondary to ILD. The patient did not consent for right heart catheterization studies. PAH was attributed to dermatomyositis-related pulmonary vasculopathy. Mycophenolate mofetil and tadalafil were initiated .

Conclusion: This case with PAH despite improvement in ILD highlights the necessity of cardiopulmonary surveillance in anti-MDA5 associated inflammatory myositis.

POR23: An interesting case of refractory inflammatory polyarthritis with mediastinal adenopathy

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Introduction: The management of refractory seronegative inflammatory polyarthritis is often challenging. Rheumatoid arthritis, psoriatic arthritis, and connective tissue diseases are the usual causes. When associated with mediastinal adenopathy, tuberculosis, sarcoidosis, and lymphoma should be considered. Here we present a similar case which turned out to be MRH.

Case Report: A 55-year-old gentleman, known case of HOCM and Diabetes had intermittent low-grade fever, cough, breathlessness and unintentional weight loss of 3 months duration. Subsequently he had progressive polyarthritis. Extensive autoimmune workup was done and was inconclusive. FDG-PET scan was done which revealed FDG avid mediastinal lymphadenopathy and inflammatory arthritis in large joints. An EUS guided lymph node biopsy revealed giant cells and histiocytes with no evidence of malignancy and TB. A diagnosis of sarcoidosis was considered and started on steroids and hydroxychloroquine. In view of persistent arthritis multiple lines of treatment including methotrexate, mycophenolate mofetil, TNF inhibitors were tried but with no significant improvement. During follow up he developed papulonodular lesions over fingers, face, ears and scalp. Histopathological exam of the nodule confirmed a diagnosis as Multicentric reticulohistiocytosis.

Conclusion: Multicentric reticulohistiocytosis is a rare disorder and is an important mimic of sarcoidosis and seronegative inflammatory arthritis. This case highlights the need for

diagnostic vigilance in patients with inflammatory polyarthritis who fail to respond to conventional treatment and biologics.

POR24: A Breathless Enigma: A Case Series of Atypical ILD in Antisynthetase Syndrome

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Introduction: Antisynthetase syndrome is an autoimmune disease characterized by anti–tRNA synthetase antibodies and a triad of ILD, myositis, and arthritis. NSIP and OP are common ILD patterns, but atypical forms like AIP, LIP, and cystic disease with pneumothorax may occur. Early diagnosis and prompt immunosuppression improve outcomes, especially in rapidly progressive ILD.

Case Report: Three cases of antisynthetase syndrome (ASS) with atypical ILD patterns were observed. Case 1: A 44-year-old male presented with fever, cough, myalgia, polyarthralgia, and rapidly progressive respiratory failure requiring mechanical ventilation. CT chest showed an acute interstitial pneumonia (AIP) pattern. Anti–Jo-1 and anti–Ro-52 antibodies were strongly positive. He improved with pulse steroids and cyclophosphamide, later maintained on tacrolimus. Case 2: A 17-year-old female had recurrent pneumothorax with subpleural cysts and HRCT suggestive of lymphocytic interstitial pneumonia (LIP). Anti–PL-12 and anti–Ro-52 antibodies were positive. She responded well to corticosteroids and mycophenolate. Case 3: A 31-year-old female developed rapidly progressive dyspnea with inflammatory polyarthritis and HRCT showing bilateral alveolar opacities . Anti–Jo-1 and anti–Ro-52 positivity confirmed ASS. Diagnosed with rapidly progressive ILD. She improved with steroids and cyclophosphamide, later stabilized on mycophenolate after flare management.

Conclusion: Early and accurate recognition of ASS is critical, especially in patients with ILD of unclear etiology, as targeted immunosuppression can dramatically alter outcomes. The presence of antisynthetase antibodies, even in the absence of overt myositis, should prompt clinicians to consider ASS in the differential diagnosis of ILD, particularly when accompanied by systemic or autoimmune features.

POR25: Dual Immune Complex Nephropathy in Sjogren's Syndrome: Coexistence of Lupus Nephritis and Cryoglobulinemia

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Introduction: Primary Sjogren's syndrome rarely evolves into complex glomerular disease. While tubulointerstitial nephritis is common, the simultaneous emergence of Class IV lupus nephritis and cryoglobulinemic vasculitis is exceptional. This case illustrates the diagnostic challenge of evolving autoimmune overlaps and the efficacy of a combined, mechanism-driven approach in refractory multisystem disease

Case Report: A 53-year-old woman with a 10-year history of primary Sjogren's syndrome presented with lower-limb edema and persistent fever. Early manifestations included keratoconjunctivitis sicca, xerostomia with total dentition loss, and parotid enlargement. Over the subsequent decade, she developed hypokalemic

paralysis (secondary to renal tubular acidosis), inflammatory polyarthralgia and a pathological femoral neck fracture complicated by infection. From 2023, she experienced recurrent palpable purpura and proteinuria refractory to glucocorticoids, methotrexate, sulfasalazine, and mycophenolate mofetil. Renal biopsy demonstrated overlapping immune complex–mediated glomerulonephritis with features of Class IV lupus nephritis and cryoglobulinemic vasculitis, marking a rare evolution toward a complex overlap phenotype. Due to refractory multisystem activity, she received rituximab, cyclophosphamide, and tapering glucocorticoids. At 8-month follow-up, she achieved complete renal remission and resolved vasculitis, remaining stable on maintenance mycophenolate mofetil and rituximab.

Conclusion: Biopsy and electron microscopy were pivotal in identifying dual pathology, guiding a strategy where treatments conflict: rituximab for cryoglobulinemia versus cyclophosphamide for lupus nephritis. This mechanism-driven combination underscores the necessity of early biopsy for accurate phenotyping and managing complex, evolving autoimmune overlap syndromes.

POR26: A Diagnostic Pitfall: Vasculitic Myopathy in Polyarteritis Nodosa with Normal Creatine Kinase

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Introduction: Polyarteritis nodosa (PAN) is a necrotizing medium-vessel vasculitis with heterogeneous clinical manifestations. Musculoskeletal involvement is frequent but often underrecognized. Muscle pain in PAN is typically ischemic rather than myonecrotic, resulting in normal creatine kinase levels and diagnostic confusion with primary inflammatory myopathies, delaying diagnosis and appropriate treatment.

Case Report: A 31-year-old male presented with recurrent severe myalgia for one year, initially involving the calves and later extending to the palms and elbows, causing functional limitation. He had a prior inferior-wall myocardial infarction treated with percutaneous coronary intervention and recurrent left temporal swelling previously surgically incised. There were no cutaneous, respiratory, or neurological symptoms. Examination showed normal muscle bulk and strength, preserved peripheral pulses, and no features of inflammatory myopathy. Inflammatory markers were markedly elevated with persistently normal creatine kinase levels. Autoimmune and myositis antibody panels were negative. MRI showed multifocal T2/STIR hyperintensities without necrosis. Muscle biopsy revealed perivascular lymphocytic inflammation without myofiber necrosis. FDG PET–CT demonstrated diffuse muscular uptake with vascular involvement, confirming systemic polyarteritis nodosa.

Conclusion: Severe myalgia with normal creatine kinase represents a critical diagnostic pitfall in vasculitis. Recognition of ischemic muscle involvement in polyarteritis nodosa, supported by multimodal imaging and histopathology, is essential for timely diagnosis, appropriate immunosuppressive therapy, and prevention of disease progression and irreversible organ damage.

POR27: When the Eye Signals a Storm: Lupus Choroidopathy as a Marker of Fulminant SLE

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Introduction: Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease with heterogeneous manifestations. Ocular involvement is uncommon, and lupus choroidopathy is exceedingly rare, occurring in less than 1% of patients. It is considered a marker of severe systemic disease activity and may precede life-threatening complications.

Case Report: A 22-year-old female presented with fever, generalized lymphadenopathy, pancytopenia, and proteinuria. Immunological evaluation revealed high-titer ANA, anti-dsDNA positivity, and hypocomplementemia, confirming active SLE. She improved with pulse methylprednisolone and was discharged on hydroxy chloroquine /Mycophenolate mofetil and prednisolone. Within 24 hours, she developed sudden painless visual loss. Optical coherence tomography showed multiple neurosensory retinal and pigment epithelial detachments, consistent with lupus choroidopathy. During hospitalization, she deteriorated with diffuse alveolar hemorrhage requiring intensive care. Treatment with repeat pulse steroids and intravenous cyclophosphamide resulted in systemic stabilization and complete resolution of choroidal lesions on follow-up imaging.

Conclusion: This case underscores lupus choroidopathy as a vision-threatening manifestation and a surrogate marker of severe systemic disease, emphasizing the importance of early recognition and aggressive immunosuppressive therapy.

POR28: Oral Squamous Cell Carcinoma in a Patient with Psoriatic Arthritis on Long-Term

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Introduction: Psoriasis and psoriatic arthritis (PsA) are chronic immune-mediated inflammatory disorders associated with systemic immune dysregulation. Population-based studies have demonstrated an increased risk of certain malignancies in patients with psoriasis, including head and neck cancers, though site-specific data and well-documented clinical cases remain limited.

Case Report: Psoriasis and psoriatic arthritis (PsA) are chronic immune-mediated inflammatory disorders associated with systemic immune dysregulation. Epidemiologic studies suggest a modestly increased malignancy risk in psoriasis, though site-specific clinical documentation remains limited. We report a middle-aged male with PsA treated with disease-modifying antirheumatic drugs for approximately three years who presented with recent oral mucosal changes. Evaluation at a tertiary cancer center revealed an ulcerative lesion of the right buccal mucosa with adjacent muscular infiltration on imaging. Incisional biopsy confirmed squamous cell carcinoma.

Conclusion: This case emphasizes the importance of careful mucosal examination and malignancy surveillance in patients with chronic immune-mediated diseases receiving long-term DMARD therapy. Early detection and

multidisciplinary management can result in favorable outcomes.

POR29: The Antibody-Negative Enigma: A case of Refractory Dermatomyositis

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Introduction: Dermatomyositis (DM) is an idiopathic inflammatory myopathy that most commonly presents with progressive, symmetric, proximal muscle weakness and a group of characteristic cutaneous findings. The usual treatment of DM was based on steroids, methotrexate, and azathioprine. Intravenous immunoglobulin (IVIG), rituximab (RTX), or other biological treatments recently emerged as steroid-sparing agents.

Case Report: We hereby present the case of a young male who presented with characteristic cutaneous and musculoskeletal manifestations of 2 months duration. Laboratory evaluation revealed markedly elevated muscle enzymes (CPK 16,800U/L; LDH 3096/L) and transaminitis (OT/PT 1747/1328 U/L) . He was managed with pulse methylprednisolone 1000mg/day for 3 days with minimal clinical and biochemical improvement. Extensive immunological workup including ANA by IIF, ANA profile and 16-Antigen Myositis blot were negative. Alternative causes of myopathy were ruled out. A PET-CT was consistent with widespread active myositis. The patient had progressive disease with new onset respiratory and bulbar weakness. He was started on IVIG as well as Rituximab. Progressive respiratory failure however required intubation and mechanical ventilation followed by elective tracheostomy. Muscle enzymes continued to rise with a zenith CPK of 55,000 U/L. In view of relentless refractory involvement, he was given rescue therapy with Plasmapheresis with significant clinical and biochemical improvement.

Conclusion: The case highlights the rapidity of the disease and Possibility of ANA negativity as a potential poor prognostic factor. The use of Plasmapheresis needs to be explored in more research trials as a potential therapy for patients refractory to standard of care.

POR30: Beyond Negative Screening: Delayed Opportunistic Infections After Anti-TNF Therapy

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Introduction: Biologic therapies, particularly Tumor necrosis factor (TNF) inhibitors, are widely used in the treatment of inflammatory arthritis and are known to increase susceptibility to infections. Most reported infections occur during active therapy. Reports of serious opportunistic infections presenting years after treatment discontinuation remain limited.

Case Report: A 62-year-old male with seronegative inflammatory polyarthritis of four years was treated with Methotrexate followed by Adalimumab (40mg every 2 weeks for 6 months), with clinical improvement. Baseline tuberculosis screening, including QuantiFERON-TB Gold, Mantoux test and chest radiograph was negative. Three years after discontinuation of Adalimumab, he developed vesicular lesions over the left forehead and scalp, diagnosed as herpes zoster ophthalmicus, started on oral Valcyclovir. He also

reported low-grade fever, abdominal pain and intermittent diarrhea for one month. Contrast-enhanced CT abdomen showed multiple mesenteric and retroperitoneal lymph nodes. CT-guided lymph node biopsy demonstrated acid-fast bacilli on Ziehl-Neelsen staining, consistent with abdominal tuberculosis. On repeated history, he reported pulmonary tuberculosis treated 15 years earlier and chronic hepatitis B infection on Tenofovir. Two days later, he presented with altered sensorium and right-sided hemiparesis. Neuroimaging revealed a left intraparenchymal hemorrhage, and cerebrospinal fluid PCR was positive for Varicella-zoster virus, confirming VZV-associated CNS vasculitis.

Conclusion: This case highlights the potential for delayed opportunistic infections following anti-TNF therapy, even after treatment discontinuation. Patients with prior tuberculosis remain at risk of reactivation despite negative screening and warrant prophylactic anti-tubercular therapy as per NTEP/WHO recommendations. Herpes zoster should prompt vigilance for serious CNS complications, including vasculitis

POR31: Arteriopathies Masquerading as Vasculitis

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Case Report: Vasculitis mimics remain an important cause of diagnostic error and unwarranted immunosuppression. We describe two illustrative cases. A 62-year-old man with unilateral neck pain and focal carotid tenderness, had MRI evidence of peri-carotid soft-tissue inflammation, but negative PET scan during an asymptomatic phase, leading to a diagnosis of transient perivascular inflammation of the carotid artery (TIPIC syndrome). The second case involved a 58-year-old hepatitis B–positive hypertensive man presenting with abdominal angina. Despite initial suspicion of polyarteritis nodosa or DADA2, normal inflammatory markers, negative vasculitis workup, and CT angiography showing multifocal visceral arterial lesions with splenic infarction supported a diagnosis of segmental arterial mediolysis. These cases underscore the critical role of clinico-radiologic correlation in identifying vasculitis mimics.

POR32: Clinical profile and treatment outcomes of adult anti-NXP2 positive myositis patients- a single center series

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Introduction : Anti-NXP2+myositis represents a distinct subtype of IIM, predominantly described in the juvenile population. Data on adult anti-NXP2+IIM is limited due to low prevalence. We describe the clinical profile and outcomes of adult anti-NXP2 patients at our tertiary care center between January'2023 and December'2025.

Case Report: Six anti-NXP2+IIM patients were identified. All were females, with a median age at presentation of 49.5years. Four (66.67%) presented with dermatomyositis, one with polymyositis, and one with clinically amyopathic disease. Neck flexor weakness was present in four (80%); Concomitant distal weakness, bulbar and

respiratory muscle weakness occurred in two each (33.33%). Limb edema was observed in three (50%), and two (33.33%) developed rhabdomyolysis. Extra-muscular manifestations included skin rash in four (66.67%), gastrointestinal vasculitis and polyarthritis in two each (33.33%), fever, subclinical myocarditis and pleural effusion in one patient each. None had calcinosis or ILD. Initial therapy consisted of pulse steroids+ IVIg in three, pulse steroids alone in one, and oral steroids in two patients. Steroid sparing agents included rituximab and methotrexate (two each), cyclophosphamide and azathioprine (one each). Five patients improved, while one succumbed to hospital-acquired infections. One relapsed later and succumbed to refractory GI vasculitis and sepsis. The remaining four are in remission, with no malignancy detected during a median follow-up of 17 months.

Conclusion: Adult anti-NXP2+IIM demonstrates a heterogeneous clinical spectrum, ranging from amyopathic disease to life-threatening myositis. Gastrointestinal vasculitis is associated with poor prognosis.

POR33: Podocytopathy in Anti-Mi-2 Dermatomyositis: A newly emerging renal manifestation

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Introduction: Mi-2 beta is critical for renal progenitor cell proliferation and differentiation. Anti-Mi-2 dermatomyositis with podocytopathy lacks documentation, suggesting rarity or underreporting and emphasizing the need for research. Here, we describe two cases of podocytopathy in anti-Mi-2 positive dermatomyositis presenting as minimal change disease—an association not previously documented.

Case Report: Case 1: A 25-year-old man had six months of hyperpigmented knuckle lesions, misdiagnosed as contact dermatitis. Over three weeks, he developed thigh pain, proximal weakness, and difficulty climbing stairs. Examination revealed a heliotrope rash and Gottron's papules, with proximal and distal weakness. CK was 6,633 U/L; 24-hour urine protein was 1,615 mg/day. Immunoblot revealed strongly positive anti-Mi-2β. Renal biopsy (LM/IF) suggested minimal change disease, and electron microscopy confirmed podocytopathy. CT chest/abdomen excluded malignancy. He received high-dose parenteral steroids plus MMF, with clinical improvement and resolution of proteinuria. Case 2: A 52-year-old man presented with facial hyperpigmentation, progressive proximal weakness, and later dysphagia. He has a history of treated tuberculosis and vitiligo in the past. CK was 7,452 U/L; proteinuria was 1,150 mg/day. Anti-Mi-2β was positive. Renal biopsy showed minimal change disease with podocytopathy on electron microscopy. PET-CT was negative for malignancy. Pulse methylprednisolone and IVIG led to improved strength and normalized proteinuria.

Conclusion: This case report highlights

podocytopathy as an emerging underdiagnosed renal manifestation of anti-Mi-2 dermatomyositis. Further studies are needed to explore prevalence, pathogenesis, and optimal management of this potential complication.

POR34: Ulcerative Cutaneous Lesions in Anti-Mi-2 Dermatomyositis: A Phenotypic Overlap with Vasculopathic Dermatomyositis

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Introduction: Anti–Mi-2 dermatomyositis is typically associated with prominent muscle involvement, classic cutaneous features, and a favorable prognosis. Ulcerative skin lesions are uncommon and more frequently described in anti-MDA5 dermatomyositis. This case demonstrates that anti–Mi-2 dermatomyositis may rarely present with vasculopathic skin manifestations.

Case Report: 50-year-old woman with hypothyroidism presented with a 3-month history of progressive myalgias involving both upper and lower limbs, history s/o proximal muscle weakness (FC-3). She also developed painful rashes over the knuckles that later ulcerated, along with erythematous rashes over the face and bilateral palms. Examination revealed ulcerated Gottron's lesions over the dorsal surface of the second and third metacarpophalangeal joints, erythematous rash over the face and palms, and significant proximal muscle weakness of both upper and lower limbs. Based on ulcerated Gottron's lesions, palmar erythema, and proximal myopathy, anti-MDA5 dermatomyositis was initially considered. Myositis antibody testing demonstrated strong positivity for anti–Mi-2 (α and β) antibodies.Patient was treated with prednisolone and methotrexate, resulting in rapid improvement in muscle strength. However, cutaneous ulcers showed only partial response. Considering a suspected vasculopathic component, tadalafil was added, following which significant healing of ulcerative lesions was observed.(Sequential images provided-Fig1)

Conclusion: Ulcerative skin lesions are not exclusive to anti-MDA5 dermatomyositis and may occur in anti–Mi-2–positive patients. While muscle disease responds well to conventional immunosuppression, persistent cutaneous ulceration may require early vasodilator therapy, emphasizing the importance of recognizing phenotypic overlap.

POR35: Beyond Classic Myositis: Anti-Mi-2α Positivity Presenting as Amyopathic Dermatomyositis

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Introduction: Anti-Mi-2 antibodies are classically associated with dermatomyositis characterized by prominent muscle involvement and favorable prognosis. Amyopathic dermatomyositis (ADM) with anti-Mi-2α positivity is rarely described.

Case Report : We report a 56-year-old female with a 5-year history of inflammatory polyarthritis and Raynaud's phenomenon, who developed progressive dyspnea.She had new onset violaceous erythematous macular rash over b/l palms.Evaluation revealed interstitial lung disease on HRCT and pulmonary arterial hypertension on echocardiography. Skin biopsy was s/o necrotising vasculitis. Myositis-specific antibody panel showed strong positivity for anti-Mi-2α and b (3+). Muscle enzymes and clinical examination did not reveal myopathy, fulfilling

criteria for amyopathic dermatomyositis.Patient was started on steroids and MMF with good improvement in her symptoms.

Conclusion: This case highlights that anti-Mi-2α antibodies can be associated with an amyopathic dermatomyositis phenotype and significant extramuscular involvement, expanding the known clinical spectrum of anti-Mi-2–associated disease

POR36: A Diagnostic Paradox in Autoimmune Myopathy

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Introduction: This case highlights diagnostic challenges in inflammatory myopathies when histopathology, serology, and treatment response are discordant. Here we report a diagnostically challenging case of anti-HMGCR–associated necrotizing autoimmune myopathy presenting with severe inflammatory changes on muscle biopsy, highlighting limitations of histopathology alone and the critical role of serologic testing.

Case Report: 29-year-old woman presented with 3-year history of gradually progressive, symmetric proximal muscle weakness involving both upper and lower limbs. She had markedly elevated creatine kinase level(7441 IU/L). MRI thighs showed patchy T2/STIR hyperintensities in multiple muscle groups with hamstring atrophy, suggesting active inflammatory myositis with chronic changes. Initial evaluation with commercial myositis LIA (16 antigens) was negative. Muscle biopsy showed prominent perivascular lymphocytic infiltrates, diffuse sarcolemmal MHC-I overexpression s/o Dermatomyositis(Images provided-Fig1). She received multiple immunosuppressive therapies, including rituximab, methotrexate, mycophenolate, azathioprine and tofacitinib without improvement. Given clinical course and persistently high enzymes, IMNM was reconsidered despite the inflammatory biopsy pattern. Repeat serology using extended myositis LIA (18 antigen) with anti HMGCR, revealed strong anti-HMGCR antibody positivity. Diagnosis was revised to anti-HMGCR associated IMNM with atypical inflammatory histopathological presentation. The patient is currently being initiated on IVIg therapy, given its proven efficacy in anti-HMGR myopathy.

Conclusion: This case highlights the limitations of histopathology alone and emphasizes the need for an integrated clinico-serologic-pathologic approach and importance LIA containing 18 antigens for accurate diagnosis and optimal management of autoimmune myopathies.

POR37: A Challenging Case of Multisystem Dermatomyositis with Recurrent Granulomatous Complications

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Introduction: Dermatomyositis management in tuberculosis-endemic regions presents unique challenges, balancing immunosuppression against infection risk. We present a decade-long case with multiple myositis-specific antibodies, recurrent tuberculosis, and unexplained lytic bone lesions, highlighting diagnostic and therapeutic dilemmas in complex autoimmune disease.

Case Report: 2014: A 40-year-old male was diagnosed as dermatomyositis based on proximal myopathy, Shawl-sign, ILD, elevated CPK, and

myopathic EMG. MSA: Mi2a (3+), SAE1(1+), and OJ (1+). Initial response to steroids and methotrexate was good. 2015: The patient developed hemorrhagic pleural effusion that resolved with Cat-I ATT. He experienced intermittent disease flares on DMARDs (methotrexate, azathioprine, mycophenolate). 2023: He developed polyarthritis, and worsening of muscle weakness. After negative malignancy screening, rituximab resulted in resolution of arthritis. However, muscle weakness persisted. 2024: Biopsy from new-onset tenosynovitis affecting right palm and distal forearm revealed granulomatous inflammation that resolved with 9 months of Cat-I ATT. Nov' 2025: He presented with a discharging sinus over manubrium sterni. USG showed a collection in the left SC joint. HRCT-thorax incidentally revealed multiple lytic lesions in bilateral humerus, left clavicle, scapula, and manubrium. PET-CT screening was negative for primary malignancy. M-band, free-light-chain assay and bone-marrow examination was negative for multiple myeloma. Gram-staining, AFB, KOH-smears, bacterial culture.

Conclusion: This decade-long case demonstrates how intermittent episodes of extra-pulmonary tuberculosis has challenged the management of this case of multisystemic dermatomyositis. The current presentation with unexplained lytic lesions necessitates comprehensive evaluation including extended microbiological and thorough malignancy workup before resuming DMARDs.

POR38: Unmasking Sjögren's: Acute Liver Failure as a First Clue to Autoimmune Hepatitis

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Introduction: Primary Sjögren's syndrome (pSS) rarely overlaps with autoimmune hepatitis (AIH), with prevalence estimates of 1–4% in cohort studies. AIH presenting as acute liver failure (ALF) in undiagnosed pSS is exceptionally rare.

Case Report: A 33-year-old woman presented with grade-2 hepatic encephalopathy, jaundice, ascites, and coagulopathy, fulfilling ALF criteria. Initial management addressed spontaneous bacterial peritonitis and hyperglycemia (new-onset Type-1 diabetes, low C-peptide). Workup revealed high-titer ANA (speckled), anti-Ro/SSA positivity, objective xerophthalmia (Schirmer's test <5 mm bilaterally), high IgG and portal hypertension (low-risk varices). See Table 1 for details. Liver biopsy showed interface hepatitis with lymphoplasmacytic infiltrate and submassive necrosis but no fibrosis, confirming Type-1 AIH. Despite cirrhosis features clinically, biopsy indicated acute severe AIH atop early chronic disease within probable pSS (ACR/EULAR criteria met). Prednisone 0.5 mg/kg was initiated with glycemic control, planning azathioprine addition; transplant referral was declined due to resource constraints. AASLD/EASL guidelines advocate high-dose steroids with urgent transplantation for AIH-ALF, but modified dosing balanced infection and metabolic risks in this immunocompromised, socioeconomically challenged patient.

Conclusion: This represents the first reported case of AIH-ALF in occult pSS. Shared autoimmune pathways likely underpin coexistent Type-1 diabetes, supported by recent multiomic data. Early autoimmune screening in pSS hepatic

decompensation is critical. AIH-ALF unmasks occult pSS in rare instances, and tailored immunosuppression offers pragmatic management in centres without transplant access.

POR39: Atypical presentation of Neuropathy in a Young Woman: Lessons from a Challenging Case

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Introduction: Giant Axonal Neuropathy is an autosomal-dominant neurodegenerative disorder due to mutation in DCAF8 gene, that is located on chromosome 1q23. It is characterized by axonal neuropathy, muscular weakness, and atrophy during the second decade of life, as well as cardiomyopathy in severe cases.

Case Report: A 31-year-old woman without comorbidities or significant family history presented with progressive bilateral lower limb proximal muscle weakness associated with muscle pain, stiffness, and easy fatigability since 2014. She denied fever, weight loss, rash, diplopia, shortness of breath, or dysphagia. Evaluation revealed elevated creatine phosphokinase, negative myositis panel, and low-titre ANA positivity (ANA IF 1:100, nuclear speckled). Muscle biopsy showed few ragged red fibers, scattered fine vacuoles, and several COX-deficient fibers (30%), leading to an initial diagnosis of mitochondrial myopathy. She was treated with Coenzyme Q and lost to follow-up. In 2025, she developed numbness with allodynia involving both feet and legs up to the knees. Nerve conduction studies of the lower limbs showed absent SNAPs, consistent with sensory neuropathy. Routine blood workup, thyroid profile, blood glucose, serum protein electrophoresis, ACPA, ANCA, and anti-SSA were negative. Enhanced whole-exome sequencing with mitochondrial genome analysis identified a heterozygous DCAF8 gene mutation, exon 4 c.197G>A variant, confirming autosomal dominant giant axonal neuropathy type 2.

Conclusion: Muscle weakness can be the Initial presentation of neuropathy. In our case, a long standing indolent course of muscle weakness with fatiguability with delayed frank peripheral neuropathy pointing towards hereditary neuropathy. Therefore, strong clinical acumen and genetic analysis are the cornerstone to diagnose hereditary neuropathy.

POR40: The Great Mimicker Revisited: IgG4-Related Disease Presenting as a Pulmonary Pseudotumor

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Introduction: Pulmonary hyalinizing granuloma (PHG) is a rare benign condition characterized by singular or multiple fibrosclerotic lung lesions of unknown etiology. IgG4-related disease has been identified as a potential cause of PHG and we present a case supporting IgG4 disease as an etiology of PHG.

Case Report: A 41-year-old woman with no comorbidities initially presented in 2022 with arthralgia and sicca symptoms. Evaluation revealed ANA positivity (1:320, fine speckled) with negative ENA, normal complements, negative rheumatoid factor, and negative Schirmer's test. She was treated as undifferentiated connective tissue disease with

hydroxychloroquine and was lost to follow-up. In 2024, she re-presented with chest discomfort, elevated inflammatory markers (ESR 89 mm/1st hour, CRP 17 mg/L), and proteinuria. Chest radiograph showed subtle left lower zone opacities. HRCT thorax revealed a 3.7 × 3 cm irregular left lower lobe lesion encasing a subsegmental bronchus, with pulmonary nodules and ground-glass opacities. CT-guided lung biopsy demonstrated pulmonary hyalinizing granuloma. Immunohistochemistry revealed >40 IgG4-positive plasma cells per high-power field with an IgG4/IgG ratio of 0.8, and serum IgG4 levels were elevated (158 mg/dL). Renal evaluation showed proteinuria with preserved renal function, consistent with membranous glomerulonephritis with positive PLA2R and IgG4 on renal biopsy. A diagnosis of definite IgG4-related disease was established & treated with Rituximab.

Conclusion: While pulmonary hyalinizing granuloma (PHG) is often asymptomatic and considered benign, this case highlights that IgG4-related disease as the etiology of PHG can lead to substantial disease progression and goals in treatment should be in line with IgG4 RD targeted treatment.

POR41: Unmasking an Inborn Error of Immunity: Evans Syndrome and Lymphoid Interstitial Pneumonitis as Initial Clues

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Introduction: Common variable immunodeficiency is the most prevalent clinically significant adult primary immunodeficiency, characterized by heterogeneous manifestations. Alongside recurrent infections, non-infectious complications such as autoimmunity are frequent. This protean presentation often complicates diagnosis, leading to delays, under-treatment, and increased morbidity and mortality.

Case Report: A 42-year-old woman presented in September 2017 with giddiness and weakness. She had five prior miscarriages. Since March 2017, she had anemia with thrombocytopenia (hemoglobin 7 g/dL, platelets 95,000/mm^3). She denied fever, weight loss, bleeding, tuberculosis, joint pain, rash, sicca symptoms, or oral and genital ulcers. Evaluation showed microcytic hypochromic anemia with anisopoikilocytosis, reticulocyte count 15%, LDH 824 IU/mL, positive direct Coombs' test, unconjugated hyperbilirubinemia, and normal iron profile and vitamin B12, consistent with autoimmune hemolytic anemia. Abdominal ultrasonography revealed splenomegaly (18.5 cm). Autoimmune workup, including APLA panel, was negative. She received dexamethasone 40 mg/day for four days followed by maintenance steroids, with improvement in anemia and thrombocytopenia; azathioprine was added for suboptimal response and danazol refractoriness. In 2023, she developed a cough. HRCT thorax showed fibrobronchiectatic changes with lobar consolidation and mediastinal and retroperitoneal lymphadenopathy. PET-CT revealed a spiculated right upper-lobe lesion (2.1 × 1.2 cm), nodal enlargement (largest subcarinal 2.9 × 2.8 cm). Lymph node b

Conclusion: Common Variable Immunodeficiency (CVID) with Evan's syndrome and Lymphoid Interstitial Pneumonitis (LIP) represent a complex, multi-systemic case of CVID-associated immune dysregulation. Genetic analysis can open up the root disease entity behind

CVID.

POR42: Paraneoplastic seropositivity mimicking Rheumatoid Arthritis: Hip monoarthritis as initial presentation of occult lung adenocarcinoma

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Introduction: Rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibodies are hallmark markers of rheumatoid arthritis (RA), although seropositivity is not absolutely disease-specific. Paraneoplastic arthritis may rarely mimic seropositive RA, and recognition can be particularly challenging in tuberculosis (TB) endemic regions where presumptive TB diagnoses and TB-associated reactive arth

Case Report: A 60-year-old man presented with a 45-day history of productive cough and weight loss and was diagnosed with sputum-negative pulmonary tuberculosis on clinico-radiological grounds, for which four-drug antitubercular therapy was initiated. Two weeks later, he developed severe right hip pain followed by knee and ankle pain, progressive in nature, worse at night, associated with morning stiffness, and poorly responsive to NSAIDs. Examination revealed marked tenderness over the right anterior superior iliac spine, positive FABER test, and restricted internal rotation of the right hip, without sacroiliac tenderness. Knees and ankles were tender without swelling. Differentials included Poncet's arthritis, psoas abscess, and rheumatoid arthritis. Laboratory tests showed elevated ESR (63 mm/h) and CRP (25.6 mg/L) with normal counts, biochemistry, and alkaline phosphatase. RF and anti-CCP were markedly positive; HLA-B27 was negative. Pain severity and focal nocturnal symptoms prompted imaging. MRI pelvis showed multiple lytic lesions suggestive of metastases. PET-CT revealed an FDG-avid left lung nodule with widespread skeletal deposits. Biopsy confirmed lung adenocarcinoma. The patient was

Conclusion: Positive RF and anti-CCP antibodies do not exclude paraneoplastic arthritis, which may precede the diagnosis of solid malignancy. Serology–phenotype discordance, nocturnal pain, poor response to NSAIDs, weight loss and focal bone pain should prompt evaluation beyond inflammatory arthritis. Serum ALP is typically elevated in predominantly osteoblastic lesions, while it may remain normal or on

POR43: When Cellulitis refused to heal: Tuberculosis masquerading In Anti-synthetase Syndrome

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Introduction: Antisynthetase syndrome is a distinct autoimmune entity within idiopathic inflammatory myopathies, characterized by myositis, inflammatory arthritis, ILD, Raynaud's phenomenon, mechanic's hands, and antisynthetase antibodies, most commonly anti–Jo-1. Immunosuppression remains the cornerstone of treatment; however, infections pose a major challenge, especially in tuberculosis endemic areas.

Case Report: 50-year-old male with one-year history of symmetrical inflammatory polyarthritis was initially treated as rheumatoid arthritis based

on positive RA factor & anti-CCP antibodies. He developed painful nodular skin lesions, with histopathology suggestive of panniculitis. Subsequently, he developed progressive dyspnea, and HRCT chest showed bilateral GGOs, raising the possibility of infection vs ILD (NSIP pattern). Multiple antibiotic courses resulted in only transient improvement. On presentation to our center, he had progressive proximal muscle weakness and recurrent episodes of erythematous, indurated, painful cellulitis involving multiple limbs, refractory to broad-spectrum antibiotics. Considering multisystem involvement, autoimmune evaluation revealed anti–Jo-1 positivity, confirming ASS. In view of suspected infection & active systemic disease, IVIG was initiated along with antibiotics. Given antibiotic-refractory cellulitis, evaluation for infections was performed, confirming M. tuberculosis. ATT was initiated while IVIG was continued as bridging immunomodulatory therapy. Gradually, there was resolution of cellulitis, improvement in muscle strength & clinical improvement.

Conclusion: This case highlights the diagnostic challenge of tuberculosis masquerading as recurrent cellulitis in ASS. Early recognition of autoimmune overlap features and aggressive infection screening are critical to guide therapy and improve outcomes in TB-endemic settings.

POR44: Joints That Mislead: A Genetic Twist In Suspected Juvenile Arthritis

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Introduction: Progressive pseudorheumatoid dysplasia (PPD) is a rare inherited skeletal dysplasia affecting articular cartilage, causing progressive, painless joint stiffness and enlargement. Onset occurs in early childhood, involving large joints and the spine, leading to contractures, gait abnormalities, spinal deformities, and frequent misdiagnosis as juvenile idiopathic arthritis.

Case Report: A 12-year-old boy with a body mass index of 14.8 kg/m² presented with gradually progressive deformity and swelling involving the knees, wrists, proximal and distal interphalangeal joints, and metatarsophalangeal joints. There was no history of joint pain, early morning stiffness, low back pain, fever, rash, or other constitutional symptoms. Clinical examination revealed symmetrical joint enlargement with deformities and restricted range of motion. Laboratory investigations showed normal erythrocyte sedimentation rate and C-reactive protein levels, with negative rheumatoid factor, anti-CCP antibodies, and antinuclear antibodies. Radiographic evaluation demonstrated epiphyseal enlargement and metaphyseal irregularities suggestive of skeletal dysplasia. Based on the clinical and radiological findings, genomic sequencing was performed, which revealed a homozygous mutation in the CCN6 (WISP3) gene, confirming the diagnosis of Progressive Pseudorheumatoid Dysplasia.

Conclusion: Early recognition of PPD is crucial to avoid misdiagnosis and inappropriate immunosuppressive therapy. A high index of suspicion, supported by characteristic imaging findings and confirmed by genetic analysis, allows timely diagnosis. Management remains supportive, focusing on physiotherapy, orthopedic care, and long-term rehabilitation to preserve function and quality of life.

POR45: New Clues from Old Tests: Hemogram Derived Inflammatory Indices in Systemic Lupus Erythematosus

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Introduction: Systemic lupus erythematosus (SLE) is a heterogeneous autoimmune disease needing accurate activity assessment. Hemogram-derived indices—NLR, PLR, and SII—are inexpensive, accessible markers of inflammation, yet their precise clinical utility and correlation with SLE activity remain inadequately established.

Methods: This cross-sectional analytical study included 121 patients with SLE fulfilling ACR/EULAR 2019 classification criteria. hemogram parameters, SLEDAI scores were recorded at a single clinical encounter. Inflammatory indices were calculated using standard formulas. Due to non-normal data distribution, associations with SLEDAI were evaluated using Spearman rank correlation, and comparisons between renal and non-renal groups were performed using Mann–Whitney U test. Receiver operating characteristic (ROC) curve analysis assessed the discriminatory performance

Results: The study included 121 SLE patients (95.9% female; median age 33 years), with renal involvement in 38%. Median NLR, PLR, SII, and SLEDAI were 2.46, 157.34, 545.45, and 6, respectively. PLR showed a modest correlation with SLEDAI (ρ=0.202). NLR and SII were significantly higher in renal SLE, while PLR trended upward. UPCR was markedly elevated in renal cases. ROC analysis showed modest discrimination for renal involvement, with AUCs of 0.624 (NLR) and 0.607 (SII), both offering reasonable specificity.

Conclusion: Hemogram-derived indices show differing utility in SLE: PLR reflects overall activity, whereas NLR and SII associate with renal involvement. Despite modest accuracy, they offer low-cost adjunctive markers for disease and renal assessment. Prospective, longitudinal validation remains essential.

POR46: Impact of Rituximab Therapy on Microcirculation in Systemic Sclerosis : A Prospective Observational Study

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Introduction: Systemic sclerosis(SSc) characterized by microangiopathy, immune dysregulation and fibrosis. Nailfold capillaroscopy(NFC) non-invasively assess microvascular changes. Rituximab may improve microcirculation by reducing endothelial damage .This study aimed to evaluate effects of Rituximab on microvascular changes in SSc.

Methods: A prospective observational study was conducted among consecutive SSc patients being treated with Rituximab. Patients were excluded if they had underlying comorbidities that could affect the systemic vasculature. NFC was done at baseline, 6 months and 12 months. Data collected include age, sex, disease duration, NFC characteristics, microangiopathy evolution score(MES), antibody status and organ involvement .Statistical differences in video-capillaroscopy abnormalities between 6 and 12 months were evaluated using Friedman's test

Results: Twenty seven patients (all were females) were included in this cohort. Among 27 patients, 19 were Diffuse cutaneous Systemic Sclerosis (DcSSc) and 8 were Limited Cutaneous Systemic Sclerosis (LcSSc). Mean disease duration was 3.4 years. Anti-topoisomerase antibody was the most prevalent antibody (72%) followed by anti-centromere (28%).In NFC, giant capillaries and hemorrhages remained stable from baseline to 12 months whereas there was improvement in capillary density and MES by 24% at 12 months.

Conclusion: Rituximab shows potential benefits beyond immunosuppression in SSc. It may aid in restoring microvascular integrity and enhancing capillary regeneration. NFC serves as a useful method to track these changes. Larger studies are warranted to validate its long-term vascular effects.

POR47: Fertility and pregnancy outcomes in Indian women with systemic lupus erythematosus: A single centre cross sectional study from North India

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Introduction: Systemic lupus erythematosus (SLE) primarily affects women of reproductive age and is associated with increased maternal and fetal morbidity. However, Indian data on fertility, contraception, and pregnancy outcomes in SLE remain limited. This study assessed reproductive health and predictors of pregnancy outcomes.

Methods: Married women aged ≥18 years with SLE fulfilling ACR/EULAR or SLICC criteria were included. Demographic characteristics, disease profile, fertility history, contraception use, and pregnancy outcomes were obtained through structured interviews and medical records. Outcomes before and after SLE diagnosis were compared, and predictors of adverse pregnancy outcomes were analyzed using logistic regression.

Results: Among 190 women with SLE (mean age 35.7±8.6 years), anti-dsDNA was elevated in 90% and low complement levels in 60.5%; 7.4% reported primary infertility. Contraceptive use was low (22.1%). Of 423 pregnancies, 309 occurred before and 114 after diagnosis. Post-diagnosis, cesarean deliveries increased (16% vs 22.9%), live births decreased (76.4% vs 57.9%), and spontaneous abortions increased (17.2% vs 24.6%). Maternal complications increased: gestational hypertension (3.5% vs 0%), preeclampsia/eclampsia (4.4% vs 0.3%), and postpartum hemorrhage (2.6% vs 0%). Neonatal complications increased: SGA/LBW (17.5% vs 9.1%), infections (12.3% vs 8.1%), and congenital heart block (5.3% vs 0%). Pregnancy flares predicted adverse outcomes (OR 2.82, p=0.021).

Conclusion: Women with SLE have poorer pregnancy outcomes post-diagnosis, emphasizing the need for preconception counselling and close monitoring.

POR48: Ten Years with Lupus: Evaluating Quality of Life, Fatigue, Mental Health, psychosocial and clinical determinants in long duration SLE Patients, a cross sectional observational study from a tertiary care centre

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Introduction: Systemic Lupus Erythematosus is a chronic autoimmune condition with known long-term impacts on physical, emotional, and social well-being.

Methods: A cross-sectional observational study was conducted on 79 consecutive SLE patients with disease duration over 10 years, attending lupus OPD. Baseline variables collected included age, sex, marital status, education level, socioeconomic status, residence type (urban/rural), and clinical SLEDAI score was collected. Quality of life was assessed using SF-12 (PCS & MCS), Lupus Impact Tracker (LIT), Fatigue Severity Score (FSS), GAD-7, and PHQ-9.

Results: The median scores observed were: SF-12 PCS 45.7(36-51), SF-12 MCS 53.2(43-58), LIT 25.0(12-47), FSS 29.0 (18-42), GAD-7 6 (3-11), and PHQ-9 6 (3-10). Strong positive correlations were found between LIT and fatigue ($\rho = 0.722$), anxiety ($\rho = 0.638$), and depression ($\rho = 0.748$) scores (all $p<0.0001$). SF-12 PCS was negatively correlated with PHQ-9 ($\rho = -0.609$), FSS ($\rho = -0.598$), GAD-7 ($\rho = -0.326$) and LIT ($\rho = -0.584$) and SF-12 MCS was negatively correlated with PHQ-9 ($\rho = -0.600$), FSS ($\rho = -0.514$), GAD-7 ($\rho = -0.528$) and LIT ($\rho = -0.674$) ($p<0.001$).Socioeconomic status showed positive correlation with FFS score. ($\rho = 0.238$, $p = 0.035$).

Conclusion: Patients with long-standing SLE experience considerable impairment in quality of life, particularly in relation to fatigue, depression, and anxiety. Routine QOL assessments and psychosocial support should be integral to long-term SLE management.

POR49: Cardiovascular Risk Stratification in Psoriatic Arthritis: Influence of Metabolic Syndrome and Application of QRISK3 and Heart Age Metrics

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Introduction: Psoriatic arthritis is a chronic inflammatory disease linked to metabolic syndrome and increased cardiovascular risk. This study evaluated MetS prevalence in PsA patients and examined its association with disease activity, treatment patterns, and cardiovascular risk using QRISK3 and relative risk scores.

Methods: This cross-sectional study (July 2024–July 2025) enrolled 103 PsA patients meeting CASPAR criteria. Nine patients (8.7%) with established CAD were excluded, leaving 94 PsA patients analyzed alongside 94 age- and sex-matched controls. MetS was defined by modified NCEP ATP III criteria. Data included demographics, anthropometry, disease duration, treatment, DAPSA, PASI, labs, and cardiovascular risk using QRISK3 and RR scores. Statistical analysis used nonparametric and correlation tests.

Results: At baseline, 8.7% (9/103) of PsA patients had established CAD and were excluded as secondary prevention cases. MetS prevalence was significantly higher in PsA patients (45.7%) than controls (30.9%, $p = 0.034$). PsA patients with MetS demonstrated markedly higher RR scores (median 2.7 vs 1.3, $p < 0.001$), whereas controls with MetS had lower RR values (median 1.55, $p = 0.002$). QRISK3 was significantly elevated in PsA patients aged 41–60 years (14.8%

vs 9.2%, p = 0.035). Both QRISK3 and RR increased with age. PASI correlated significantly with RR, while DAPSA showed weaker association. MetS was unrelated to disease duration, onset age, or treatment.

Conclusion : Nearly half of PsA patients had metabolic syndrome with significantly increased cardiovascular risk, especially aged 41–60 years. Established CAD was present in 8.7%. Skin severity correlated with higher RR, supporting routine use of QRISK3 and RR for early cardiometabolic screening and prevention.

POR50: Clinical Spectrum of Anti-PL-7 Antibody–Associated Disease: A Retrospective Study

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Introduction: Anti-PL-7 is a myositis-specific autoantibody directed against threonyl–tRNA synthetase and is associated with anti-synthetase syndrome. Clinical manifestations are heterogeneous , may occur with minimal or absent muscle involvement. To describe the clinical spectrum and treatment patterns of patients with anti-PL-7

Methods: We performed a retrospective review of patients who tested positive for anti-PL-7 antibodies between January and October 2025. Demographic characteristics, clinical manifestations, radiological findings, pulmonary function parameters, and treatment details were extracted from electronic medical records. Anti-PL-7 antibodies were detected using the Euroline immunoblot assay, and an intensity ≥1 was considered positive.

Results: Fourteen patients were identified: 12 (85.7%) females and 2 (14.3%) males. The median age at presentation was 48.5 years (IQR 13.5). The most common clinical manifestations were polyarthralgia (57.1%) and interstitial lung disease (ILD) (42.8%). Raynaud's phenomenon was present in one patient (7.1%). Other features included cough (21.4%) and fever (14.3%). Among ILD patients, high-resolution CT patterns included NSIP with organizing pneumonia overlap (33.3%), mixed NSIP (33.3%), cellular NSIP (16.7%), and organizing pneumonia (16.7%). Only one patient (7.1%) had clinical myopathy with mild weakness (MMT-8 score 75/80). Mean forced vital capacity at diagnosis was 1.39 L (58% predicted). For ILD management, one patient received azathioprine and five received mycophenolate mofetil

Conclusion: Anti-PL-7–positive patients predominantly present with ILD, often without overt myopathy. Early recognition of this phenotype facilitates prompt diagnosis and appropriate immunosuppressive therapy

POR51: Manual Assessment of Explainable Artificial Intelligence Using Large Language Model–Based Agents for Screening Rheumatoid Arthritis

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Introduction: Rheumatoid arthritis affects ~1% globally, with Indian patients often presenting late. Mobile LLM-based AI tools may enable early screening, but clinician trust requires

transparent, clinically sound reasoning beyond diagnostic accuracy.

Methods: A proprietary dataset, PreRAID (Pre-Screening Rheumatoid Arthritis Information Database), was developed from physician-confirmed RA cases, comprising 280 cases for knowledge base construction and 70 for testing. Six LLMs (four closed-source, two open-source) were evaluated using three agentic configurations: single-agent without knowledge base access,single-agent with retrieval-augmented generation, and a dual-agent setup. Diagnostic performance was assessed, and reasoning quality independently evaluated by three rheumatologists using predefined criteria

Results: DeepSeek R1 achieved the highest diagnostic accuracy (82%) in the single-agent framework with knowledge base access, followed by OpenAI o1 and o3-mini (80% each) (Table 1). Dual-agent frameworks demonstrated reduced accuracy. Reasoning quality varied widely and did not consistently align with diagnostic accuracy; models with superior reasoning often showed poorer diagnostic performance (Table 1).

Conclusion: A significant misalignment exists between diagnostic accuracy and reasoning validity in LLM-based RA screening. While current models may be suitable for preliminary screening, robust and clinically aligned explainable AI frameworks are essential before broader clinical deployment.

POR52: Clinical, Immunological, and Radiological Characteristics of Anti-Synthetase Syndrome: A Cross-Sectional Study

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Introduction: Inflammatory myopathy in this cohort was predominantly associated with antisynthetase syndrome, characterized by high ILD prevalence, myositis, antisynthetase antibodies, restrictive lung disease, and supportive laboratory, radiological, and histopathological findings.

Methods: This is a hospital-based, observational cross-sectional study. Consecutive patients were enrolled over an 18-month period. Demographic details, clinical manifestations across musculoskeletal, pulmonary, dermatological, and systemic domains were recorded. Immunological profiling included myositis-specific antibodies. ILD was evaluated using (HRCT) and (FVC, DLCO). Histopathological assessment of muscle and skin was performed when indicated. Statistical analysis was conducted using SPSS v19.

Results: This descriptive observational study of 24 patients with inflammatory myopathy showed a mean age of 46 years with mild male predominance. Interstitial lung disease was the predominant manifestation (≈92%), commonly presenting as organizing pneumonia or NSIP. Myositis was universal, with frequent fever, arthritis, and muscle weakness. Muscle enzymes were commonly elevated. Myositis-specific antibodies, especially anti-Jo-1 and Ro-52, were frequent, supporting antisynthetase syndrome. Pulmonary function tests showed a restrictive pattern. ANCA was negative in all, and limited biopsies confirmed inflammatory myopathy.

Conclusion: Inflammatory myopathy in this cohort was predominantly associated with

antisynthetase syndrome, characterized by high ILD prevalence, myositis, antisynthetase antibodies, restrictive lung disease, and supportive laboratory, radiological, and histopathological findings.

POR53: Clinico-radiological profile and inflammatory markers in psoriatic arthritis

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Introduction: Psoriatic arthritis (PsA) arises from a complex interplay of genetic, environmental, and immunological factors, with varied clinical presentations and an uncertain prevalence. Despite efforts to assess disease activity using biomarkers like CAR, NLR, PLR, MPV, SII, SIRI, no concensus exists on optimal indexes.

Methods: This prospective observational study included all patients of PsA seen over 1-year. Demographic, clinico-radiological profile and correlation of new biomarkers with disease parameters like Psoriasis Area and Severity Index (PASI), Nail Psoriasis Severity Index (NAPSI), and number of swollen and tender joints were evaluated.

Results: Among 31 patients with PsA (70% males with mean age of 44 ± 15 years), mean age of onset is 33 years. The most common pattern was category 1 (32%), followed by category 6 (29%). Skin and nail changes were present in 84% (median PASI 1.5) and 55% (median mNAPSI 10) of the cases respectively. Enthesitis, dactylitis and ocular involvement were seen in 35%, 35% and 10% of cases, respectively. Among patients with peripheral arthritis, 35% have erosions (median Steinbrocker score 26) and in spondyloarthritis, 42% have sacroilitis and 13% syndesmophytes (median BASRI 4.5). Biomarkers like NLR, PLR, MLR, SII, SIRI showed a stronger correlation with disease activity scores (cDAPSA, MDA-5, HAQ-DI) compared to CRP level ($p<0.001$).

Conclusion: Identification of new inflammatory biomarkers which corelates better with disease severity in psoriasis, would improve early diagnosis and management of PsA patients.

POR54: Study Of Association Between Nail Fold Capillaroscopy Patterns And Bone Mineral Density In Systemic Sclerosis Patients - A Cross Sectional Study

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Introduction: SSc is characterized by early microvascular damage and fibrosis. Nailfold capillaroscopy reflects microangiopathy severity, while reduced BMD and increased fracture risk are increasingly recognized in SSc. Data linking NFC severity with bone health and FRAX-estimated fracture risk remain limited.

Methods: This study was conducted among 68 patients fulfilling the 2013 ACR/EULAR classification criteria for SSc were included. NFC patterns were classified as Early, Active, Late. BMD assessed by DEXA at the lumbar spine and femoral neck. 10 year fracture risk for major osteoporotic fracture (MOF) and hip fracture (HF) was estimated using FRAX. Statistical analysis was performed using SPSS v25.

Results: The mean age was 41.8 ± 10.9 years, with female predominance (94.1%). LC and DC accounted for 57.4% and 42.6% of cases. NFC patterns were mainly Active (54.4%) and Late (33.8%). DC SSc showed significantly lower lumbar spine and femoral neck BMD than LC ($p < 0.05$). BMD declined with increasing NFC severity (lumbar spine: $H = 12.84$, $p = 0.004$; femoral neck: $H = 10.62$, $p = 0.014$). FRAX-MOF and hip fracture risk increased stepwise from Early to Late NFC patterns ($p = 0.002$ and $p = 0.009$, respectively).

Conclusion: Advanced NFC patterns in systemic sclerosis are associated with lower BMD and higher FRAX-estimated fracture risk. NFC help identify patients at increased risk of skeletal involvement.

POR58: The Great Mimicker Strikes Twice: TEN-Like Skin and Lupus Enteritis in Systemic Lupus Erythematosus

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Introduction: Systemic lupus erythematosus (SLE) rarely presents with simultaneous severe mucocutaneous and gastrointestinal involvement. TEN-like acute cutaneous lupus and lupus enteritis represent uncommon, high morbidity disease phenotypes requiring early recognition and aggressive immunosuppression to prevent complications, organ damage, and mortality.

Case Report: A 30 year old female presented with ten days of abdominal pain, recurrent vomiting, and melena, followed by progressive mucocutaneous lesions over face, trunk, and limbs, oral ulcers, and fever. Examination revealed diffuse erythema over malar region involving nasolabial folds, erosive oral lesions with crusting, ocular discharge, non palpable purpuric rash over extremities, and genital erosions. Investigations showed pancytopenia with hemoglobin 6.4 g/dL, white cell count 2.76×10^9/L, platelets 20×10^9/L, creatinine 1.9 mg/dL, ferritin 942 ng/mL, low complement levels, ANA positive mixed pattern, strongly positive anti dsDNA, anti SSA, anti histone, and anti nucleosome antibodies. Urine protein creatinine ratio was 1.48. Contrast CT abdomen demonstrated circumferential bowel wall thickening involving ascending colon, transverse colon, jejunal and ileal loops, moderate ascites, and dilated small bowel loops suggesting lupus enteritis with subacute intestinal obstruction. She received pulse corticosteroids followed by cyclophosphamide as per NIH protocol with marked clinical and laboratory improvement.

Conclusion: This case highlights rare severe SLE manifestations including TEN like cutaneous lupus and lupus enteritis causing intestinal obstruction. Early differentiation from drug induced TEN and prompt aggressive immunosuppression are essential to improve outcomes and reduce morbidity in severe multisystem lupus presentations.

POR59: Septic Arthritis–Like Flares After Switching Biologic Therapy in Psoriatic Arthritis: A Diagnostic Pitfall

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Case presentation: A 66-year-old man with an initial history of sero-negative polyarthritis managed with Suphasalazine, later developed mild psoriatic skin rash and nail changes, axial

involvement and sacroiliitis warranting IV Infliximab treatment. Cutaneous and Musculoskeletal manifestations responded remarkably to Infliximab which was withdrawn due to unavailability, switching him to Methotrexate and Tofacitinib.

Consequently, he developed right sided painful erythematous swelling of elbow with impaired joint motions treated as septic arthritis with arthrotomy and antibiotics. Subsequently he developed similar symptoms of the right knee necessitating arthrotomy with mild symptomatic improvement. Joint fluid showed leukocytosis (>50,000/mm^3), negative cultures and crystals with normal serum uric acid on repeated occasions. Investigations excluded tuberculosis. A fortnight later he presented with painful right wrist swelling and elevated inflammatory markers after which he was started on a trial of Intramuscular followed by intravenous steroids with remarkable recovery. His Sulphasalazine was optimized but steroid wean off was only possible after the commencement of adalimumab following which the patient remained symptom free.

Discussion: In a patient on treatment for Psoriatic arthritis (PsA) diagnosing disease flare in the context of abrupt onset mono or oligoarticular disease may be challenging. A flare can closely mimic septic arthritis with high joint fluid cell counts and it's reasonable to treat patients for sepsis in the background of immunosuppression. Poor antibiotic response and steroids responsiveness may be helpful in diagnosing such cases.

Conclusion: Although JaKi are known to have comparable efficacy to TNFi in PsA, certain phenotypes may respond better to TNFi.

Beyond Sicca: Heterogeneous Myositis Phenotypes in Sjögren's disease- A Case Series. When the Going Gets Dry

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Case Presentation: Case 1- 30-year-old lady presented with recurrent episodes of progressive limb weakness. Initially treated as Guillain barre syndrome, subsequent episode led to ascending paralysis with respiratory failure. Inflammatory markers were elevated and neurophysiology demonstrated ganglionopathy and sensory-motor axonal polyneuropathy with myopathic changes. MRI revealed long segment myelitis and muscle edema consistent with inflammatory myopathy. Creatinine phosphokinase (CPK) was elevated. Muscle biopsy demonstrated endomysial lymphocytic infiltration with perivascular inflammation. Autoantibodies, initially negative, later became positive for ANA and anti-La, with subsequent sicca symptoms. She was diagnosed with Sjogren disease (SjD)-associated inflammatory myopathy with positive Mi-2a antibodies and showed marked improvement with corticosteroids and rituximab.

Case 2- A 62-year-old lady with diabetes presented with progressive unilateral thigh pain and swelling over 8 months. Repeatedly treated as pyomyositis with lack of response, elevated inflammatory markers, CPK and electrophysiological evidence of focal myositis prompted muscle biopsy which revealed necrotizing myositis. ANA and Anti-La antibodies and dramatic response immunosuppression confirmed a diagnosis of SjD associated focal myositis.

Case 3- 41-year-old lady presented with progressive proximal limb weakness associated

with sicca symptoms, elevated inflammatory markers and CPK. Muscle biopsy and EMG confirmed inflammatory myositis with positive ANA, anti-Ro (SSA), Anti-La (SSB) and Anti-Ro52 antibodies with other myositis specific antibodies being negative. She had a marked response to immunosuppression.

Discussion and Conclusion: Inflammatory myositis is a rare manifestation of SjD and can have a heterogenous presentation. While anti La antibodies are unusual to present in isolation, they are more specific to SjD compared to antiRo and may represent a myositis phenotype of SjD.

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1. **Patient-Centered Approach –** We prioritize real patient needs, ensuring that every initiative is meaningful and impactful.
2. **Strong Medical Collaboration –** By involving doctors, researchers, and healthcare professionals, we keep our community informed with the latest medical advancements.
3. **Inclusive & Interactive Engagement –** Through lectures, Q&A sessions, workshops, and patient stories, we create a dynamic platform for learning and sharing.
4. **Mental Health Awareness –** Recognizing that arthritis affects more than just the body, we advocate for mental and emotional well-being alongside physical health.
5. **Unwavering Support (NOT SYMPATHY) –** Our focus has always been on empowering patients, fostering self-reliance rather than seeking pity.

Next is reaching out to the government and the IRA, which is a crucial step in advocating for arthritis patients, especially when it comes to influencing government policy related to healthcare insurance.

By working alongside the IRA and pushing for policy change, we can create a more inclusive healthcare system for arthritis patients. This partnership can help to ensure that arthritis care becomes more accessible, affordable, and prioritized within India's health policy and insurance systems.

As Chairperson of the Patient Support Group for Arthritis, I extend my deepest gratitude to everyone who makes our journey possible.....

Deepa Mehta
Chairperson MAI

Established in 2014, Antardhwani has emerged as India's premier patient advocacy group for **Spondyloarthritis and Rheumatoid arthritis**. With a robust network of 8000+ patients across India, we function as a strategic ally to the rheumatology community in India, bridging the gap between consultation and long-term disease management through structured education and psychosocial intervention

Core clinical support pillars

1. **Treatment adherence and Counselling** – Our dedicated helpline provides evidence-based guidance on biological therapy protocols, flare management and reproductive counselling in autoimmune conditions. We facilitate financial accessibility through 30% diagnostic subsidies for our members.
2. **Medical Education** – We organise rheumathons, webinars and specialised podcasts to allow rheumatologists to address clinical misconceptions in a controlled, peer-supported group to improve treatment compliance
3. **Psychosocial counselling** – Recognizing that rheumatic diseases are associated with depression and occupational dysfunction, our sessions target the biopsychosocial impact of these diseases. We help mitigate stigma and improve quality of life.
4. **Patient empowerment** – We assist in standardizing patient health records and guide patients for various government welfare schemes and insurance.

A Collaborative Referral Partnership

Antardhwani invites rheumatologists to leverage our decade-long expertise and large patient community through collaboration. By referring patients to Antardhwani, you ensure that patients receive supplementary care, peer support and information, physical rehabilitation guidance Together we can transform the natural course of rheumatic diseases, integrating clinical excellence with a decade-long term patient empowerment program.

SOCIAL MEDIA HANDLES

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SCLERODERMA INDIA

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SOCIAL MEDIA HANDLES

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Scleroderma india was **founded in 2017** and was registered in 2020. Late Ms Neetu Wadhwa founded it. We are a registered NGO. Today it's run by trustees Rashmi Bhasin, Pinky Thakral and Vikrant Wadhwa.
We spread awareness on social media with educational posts and webinars by doctors. We educate our fellow patients by helping them understand that it is possible to live a relatively normal life with lifestyle modification and a positive mindset

Our Motto 'YOU ARE NOT ALONE'

Scleroderma India was founded for the need to spread awareness and lend support to fellow patients and caregivers. It is a body run by fellow patients where we educate patients with

1. Knowing about the disease
2. Pointing them in the right direction about treatment and medical representative
3. Lending mental support
4. Giving them a safe space to talk to fellow patients ask questions
5. Helping patients find reasonable medicines
6. Helping with periodic financial help if required

MYOSITIS INDIA

MYOSITIS INDIA
PATIENT CARE FOUNDATION

SOCIAL MEDIA HANDLES

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Myositis India is a national patient support and advocacy group dedicated to individuals living with Myositis in India. The initiative aims to bridge gaps between patients, caregivers, clinicians, and researchers by providing reliable information, structured support, and opportunities for academic collaboration

A. Advantages for Patients and Caregivers:

1. Access to accurate, evidence-based disease education curated with clinician input
2. Multilingual educational resources
3. Patient and caregiver webinars addressing diagnosis, treatment options, rehabilitation, and long-term disease management
4. Emotional and peer support through moderated community engagement
5. Improved awareness of the importance of follow-up, adherence, and outcome assessments

B. Academic and Clinical Value for Rheumatologists:

1. Access to a well-informed patient community, facilitating better clinical communication and adherence
2. Opportunities to participate in physician-led webinars, panel discussions, and academic forums focused on myositis
3. Support for awareness around observational studies, registries, and clinical trials, aligned with Indian regulatory frameworks
4. A collaborative platform to share evolving perspectives on diagnostics, outcome measures, and treatment strategies
5. Strengthening India's myositis research ecosystem through ethical patient engagement

Digital Access and Patient Education Tools:

All official Myositis India platforms, resources, and updates can be accessed via the
Linktree: https://linktr.ee/myositisindiaofficial

SJOGREN'S INDIA

For Living well with Sjogren's syndrome

Sjögren's India
For Living Well with Sjögren's Disease

Patient Testimony

"Before attending an SI meeting, I thought this was onlly my struggle... but now I can relate, share and get guidance for dealing with this misunderstood disease."

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Sjogren's India (SI) is a patient-initiated and managed support group for individuals living with Sjogren's Disease (SjD). SI was **co-founded by two patients in 2006**. Today, Sjogren's India is the trusted ally for 380 registered SjD patients and their families from across India and abroad.

What does Sjogren India Do?

1. **Education and Counselling -** SI's experienced patient volunteers provide one-to-one counselling via phone, WhatsApp and virtual or in-person group sessions. Over the years, SI has organized over 25 events with over 80 national and international experts interacting with patients.

2. **Public Awareness –** We run awareness campaigns on radio, television and social media, as well as live streaming webinars and the flagship event 'Sjogren's Dialogues' on World Sjogren's Day. In collaboration with PatientsEngage, SI has published Patient stories, educational videos, a research poster and several webinars for public awareness.

3. **Networking -** SI is a member of the International Sjogren's Network (ISN) and EULAR's IPARE Working Group. SI participated as a member of a Community Advisory Board for making Clinical Trials more patient friendly. SI was also a member of the OMERACT Sjogren's Working Group. SI was involved in the CoVAD study as a patient partner and is a co-author for a manuscript and an abstract submitted for EULAR 2026. SI has facilitated the formation of the Patients Alliance for Rheumatic Diseases (PARD) and is working towards developing meaningful collaborative projects with other Patient Support Groups in India.

Kirtida Oza
Co-founder and Director

LUPUS TRUST INDIA

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Lupus Trust India is a national, **patient led organization founded in 2018, headquartered in Kochi, Kerala and operational across 12 states**. We currently support over 1500 patients living with SLE and related autoimmune conditions, many of whom are young and juvenile onset patients.

Core pillar of our work is structured patient education that reinforces patient-based care. We actively **educate patients regarding the role of rheumatologist** as the primary physician and decision maker in lupus care, and how multidisciplinary involvement is best coordinated via a rheumatologist.

Our role is complementary to clinical care. From a clinical perspective, we consistently observe that **patients connected to credible support groups demonstrate better treatment compliance and improved psychosocial outcomes**. We also work across language and regional contexts, ensuring patient education is available in multiple Indian languages to improve comprehension and compliance.

On the policy front, Lupus Trust India **played a role in bringing lupus into parliamentary discussions** for the first time, with the support of Sri Hibi Eden (MP), particularly around access, affordability and inclusion under national health and social welfare frameworks

Vachasamrita Saiju
Chairperson and co-founder
Lupus Trust India

VASCULITIS SOCIETY

SOCIAL MEDIA HANDLES

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Our services are specifically designed to address the unique needs of those with this condition.

Exercise for Vasculitis program: We offer personalized fitness plans that enhance circulation, reduce inflammation, and improve overall mobility. Our knowledgeable trainers ensure that every exercise regimen is safe, effective, and adaptable to your specific health requirements, empowering you to achieve your fitness goals without compromising your well-being.

Nutrition for Vasculitis and Meditation for Vasculitis services: Our nutrition experts create customized meal plans that focus on anti-inflammatory foods, essential nutrients, and balanced diets to support your body's healing process. Meanwhile, our meditation sessions provide tools for stress reduction and mental clarity, essential for managing the emotional and physical challenges of vasculitis.

Together, these holistic approaches form a robust support system, helping you lead a healthier, more balanced life. Choose us for a compassionate, integrative approach to managing vasculitis and enhancing your quality of life.

Unscripted Journey - Patient stories to inspire you

WHERE HEALING HANDS MEET THE CALL OF WILD

Dr Prakash Joshi, MD
Professor, Department of General Medicine
SAIMS, Indore

In the demanding world of medicine, where precision, discipline, and responsibility shape everyday decisions as a reassuring presence beyond hospital corridors and consultation rooms lies another dimension of my life—one that reflects the same depth of observation and commitment, expressed through the lens of wildlife photography.

While I started pursuing wildlife photography seriously in 2018, my relationship with nature is far older. It traces back to childhood, to a time when birds on electric wires, fleeting glimpses of animals, and wildlife documentaries sparked a quiet fascination. These documentaries were more than entertainment; they were early lessons in wonder. Even then, a simple thought lingered—what if this world could be experienced directly, not mediated by a screen, but felt through silence, distance, and presence?

That question found its answer years later inside a tiger reserve, during a moment that would redefine my understanding of the natural world. It was early morning, the forest immersed in soft, filtered light. The tall trees stood motionless as if holding their breath. And then a tiger emerged—unhurried, self-assured, moving through the forest with complete ownership of space. The animal came remarkably close to the gypsy, close enough for its breath to be sensed. In that instant, observation turned into connection. The forest ceased to be a destination and became a living, breathing reality.

That encounter marked a turning point. From that day forward, wildlife was no longer an interest—it became an integral part of my inner life. The camera in my hands evolved into a tool of awareness rather than pursuit.

Over the years, my photographic journey has taken me across many of India's most significant wildlife landscapes, including Kanha, Bandhavgarh, Panna, Pench, Tadoba, Satpura, Corbett, and Bharatpur Bird Sanctuary, along with several other tiger reserves and sanctuaries. My work has also extended beyond national boundaries into Africa, where vast ecosystems and primal rhythms offer profound lessons in balance and coexistence.

Tigers remain central to my work and my heart but my lens embraces a broader narrative—birds in flight, animals at rest, moments of alertness, and the quiet interactions that often go unnoticed. Rather

than seeking spectacle, I prioritized authenticity. My images allow subjects to exist naturally within their environment, revealing character through stillness as much as through action.

Patience lies at the core of photographic philosophy. Long hours of waiting, careful reading of light, and a deep respect for animal boundaries define his process. This approach mirrors the principles that guide and medical practice. Medicine has trained me to observe closely, to exercise restraint, and to act with empathy and precision. These qualities translate seamlessly into the field, where timing and understanding often matter more than equipment or speed.

For me wildlife photography is not a diversion from medicine, but a counterbalance to it. Clinical life demands constant decision-making and accountability; the forest demands surrender and acceptance. In the wilderness, control gives way to humility. Photography becomes a meditative practice—one that restores perspective, sharpens awareness, and nurtures mental well-being. It is here that professional discipline and personal reflection converge.

My work has been widely acknowledged. And showcased in exhibitions, recognized through awards in competitions, and published in newspapers on multiple occasions ,through social media, I continue to share my work with a broader audience, extending the experience of the wild to those who may never encounter it first hand. Yet recognition remains secondary to purpose.

Conservation awareness forms a foundational pillar of his photographic journey. Each image, I believe, carries an ethical responsibility. Beyond visual appeal, my photographs aim to foster respect for wildlife and highlight the delicate balance that sustains ecosystems. By creating emotional connection, I hope to encourage thoughtful coexistence rather than passive admiration.

My journey serves as a reminder that professional excellence and personal passion are not opposing forces. On the contrary, they can strengthen one another. My journey illustrates how attentiveness, empathy, and patience—qualities essential to good medicine—find renewed meaning when practiced in the natural world.

In an era defined by speed and overstimulation, my work offers a quieter, more deliberate rhythm. It invites reflection. It encourages presence. And it subtly reminds us that reconnecting with nature is not a luxury, but a necessity.

Takeaway: In caring for others, professionals often forget to nurture their own sense of wonder. Our journey suggests a simple yet powerful truth—when we allow ourselves the space to reconnect with what inspires us, we return to our work not only more balanced, but more human.

THE CASE THAT REFUSED TO BEHAVE

Dr Aradhana Singh
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SMS Hospital, Jaipur, Rajasthan

The patient arrived on an ordinary clinic day, the kind that lulls you into efficiency. Symmetric joint pain. Morning stiffness. Raised inflammatory markers. Nothing flamboyant, nothing alarming. The signs lined up neatly, almost obediently, and my mind reached for a diagnosis before my chair had fully settled.

I named it quickly as RA, confidently. It felt good to be decisive.

At that stage of my training, certainty felt like competence. Rheumatology, I believed, was about recognizing patterns—and I had recognized this one. I explained the disease to the patient, outlined the plan of treatment, and watched relief wash over the patient's face. A diagnosis, after all, can feel like rescue.

But diseases have a way of resenting premature certainty.

The weeks that followed were quietly rebellious. The response to treatment was underwhelming. New symptoms crept in—soft at first. Dryness that lingered too long. Fatigue that felt disproportionate. Serology returned with mixed signals, inconvenient antibodies that refused to strengthen my original narrative.

Each time, I adjusted around the diagnosis instead of questioning it.

There was a moment—clear in hindsight—when I should have stopped and reconsidered. Instead, I reassured. I waited. I trusted my first impression more than the evolving patient in front of me.

Then came the turning point. The patient landed up in the OPD with new and aggressive symptoms with multiple organ involvement. The kind that changes the tone of the room. The kind that makes silence heavy after the reports are read.

That was when the discomfort arrived—not clinical, but personal.
I went back to the beginning. Not to the lab values or criteria, but to the story. And suddenly, the

fragments rearranged themselves into a different picture altogether. One I should have seen earlier. One that had been whispering while I was too busy being sure.

The final diagnosis emerged without drama, but the realization hit hard: my earliest mistake in rheumatology was not choosing the wrong diagnosis—it was choosing it too soon.
Rheumatology taught me that day what no textbook had emphasized enough: diagnoses here are provisional, not proclamations. Diseases evolve. Antibodies mislead. Patients do not read classification criteria before falling ill.

That case changed the way I practice. I now pause when a diagnosis feels too obvious. I listen for discordant notes. I remind myself that in rheumatology, uncertainty is not a gap in knowledge—it is part of the discipline.

Looking back, that patient gave me an education no lecture could. The error humbled me, slowed me down, and quietly reshaped my clinical instincts.

It was my first diagnostic mistake in rheumatology. And, in many ways, my first real lesson in becoming a rheumatologist.

BEYOND PRESCRIPTIONS: STORIES FROM THE WARD AND OPD

Dr Vaibhav Yadav
Assistant Professor
Department of General Medicine
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Rheumatology often involves managing chronic diseases, long-term medications, and patients who live with persistent pain. Yet, amid serious consultations and busy rounds, small moments of unintended humor remind us that medicine is ultimately about people, not just pathology.

During one ward round, while taking routine history, I moved to obstetric details and asked the patient in Hindi,

“Koi baccha kharab hua hai kya?”
Before the patient could respond, her husband—already trying to control their two energetic children rolling and playing across the bed—answered instantly,

“Doctor saab, ye do hue hain… aur dono hi kharab hue hain!”
Almost on cue, both children continued their playful acrobatics, perfectly validating his remark. The team paused briefly before laughter spread across the ward. The patient herself, smiling, clarified that to him, any child who refuses to sit quietly automatically qualifies as kharab.
Another memorable encounter occurred in OPD while assessing pain severity in an arthritis patient. As part of routine evaluation, I asked,
“On a scale of 1 to 10, how severe is your pain and when it is worst?”
After thinking for a moment, the patient replied,

“ _*Subah 9 hota hai, shaam ko 6.”*_
Before I could proceed further, his wife calmly added,

_*“ _ *Aur jab main kaam bolti hoon tab 11 ho jata hai*_
The consultation room burst into laughter, and even the patient joined in, admitting with a smile,

*“Doctor saab, asli problem ghar par hoti hai.”*
Moments like these remind us that while we treat joints and inflammation, humor often brings relief faster than medicines—for patients, families, and doctors alike.

Take-Home Messages

* Obstetric history needs careful wording… especially in front of honest fathers.
* Spouse history is often more accurate than patient history.
* Pain score rises instantly when housework is prescribed.

THE MAGIC OF CMC VELLORE

Dr. Abhilasha Manwatkar
Assistant Professor, Rheumatology
AIIMS Nagpur

I have worked in many parts of India, but what I learned at CMC Vellore is difficult to explain. Some places teach you skills; a few places change how you think. If I had to summarise the importance of CMC in one line, I would tell my family this: *if something ever happens to me, please take me to CMC Vellore.*

After a few years at Mumbai hospital, my interest gradually shifted toward rheumatology. Medicine, however, remained my first love. Yet, to understand disease in depth…to see beyond symptoms and numbers…I realised I had to step away from the familiar. That decision brought me to CMC. At that time, I knew very little about its history or its aura. To me, it was simply a private charitable hospital.

My first impression was confusion. The wards had no air-conditioning. The buildings looked older than India's independence. Drinking water had to be bought separately. I remember thinking, *why do people speak about this place with such reverence?*

I was shy then. On the first day of introduction, I held the microphone with trembling hands. My voice shook. I barely spoke.

Then I learned the story of CMC...and something quietly shifted inside me.

Dr Ida Sophia Scudder was born in 1870 in South India. She never planned to follow her missionary parents. But one night in Tindivanam, three young women died in childbirth because cultural norms prevented them from being treated by a male doctor. That single night altered her life's direction. She chose medicine not for recognition, but to ensure that such deaths would never happen again. From a one-bed clinic in Vellore grew hospitals, nursing schools, and India's first medical school for women. What began as a response to suffering became Christian Medical College…an institution built on service, discipline, and compassion.

How could one person's determination create something so enduring? That question stayed with me.

Working at CMC slowly changed how I practised medicine. The laboratory taught me integrity. If a report was negative, it was truly negative…no shortcuts, no arguments. Every step, from sample dilution to ELISA and immunofluorescence, was handled by experienced hands. Interestingly, we rarely relied on routine ANA blot testing. At first, I did not understand why. Later, I realised the intent: CMC trains you to think about disease, not hide behind tests.

The teaching schedule was relentless. I often joked that the number of classes I attended at CMC

equalled all the classes I had attended in my entire life. There were scheduled academic sessions, followed by MDT meetings every afternoon…every single day except Sundays and vacations. There were no special holidays for Diwali, Holi, or Pongal. I worked at OPDs on festivals. One day, my OPD continued until 3 a.m. There was no fixed lunch break. You ate when you could, and then returned to patients. Because everyone worked this way, it somehow felt normal.

The nursing staff were the silent pillars of the hospital. Disciplined, knowledgeable, and deeply committed, they often knew more than anyone else. Steroid pulses would not begin unless potassium levels were corrected. Their attention to hygiene, bedsores, ulcers, and basic care, done in simple, traditional ways…was extraordinary. Watching them taught me what responsibility truly means.

In rheumatology, patients are chronic, and responsibility does not end at discharge. Patients wrote emails directly to us. They returned for follow-ups and were allotted back to the same doctor. Many of my patients spoke Bengali; they were simple, trusting, and treated CMC like a temple. At the time, I did not fully understand why.

One patient with severe SLE changed that forever. She became critically ill, was intubated, and required CPR. I was convinced she would not survive. I sent her to the ICU, carrying quiet resignation. Two weeks later, she walked back into my ward. From that day onward, something changed in me. I carried a different kind of confidence, quiet, grounded, and unshakeable.

I also learned from my teachers. When I joined, Dr Danda Sir had already retired. People said we were fortunate…earlier, work continued until 5 a.m., followed by ward rounds at 8 a.m. Just hearing this terrified me. When he later came as an examiner, I was extremely nervous. He gave honest feedback. I felt bad, but I improved within two months. Later, when he spoke to me about his journey and dedication to CMC, my respect for him grew immensely. Once, he stopped at CMC just to sign a letter of recommendation while travelling to Bangalore. I often wonder…does CMC create such people, or does it simply attract them?

The mentor–mentee system was something I had never experienced before. Even as an assistant professor, I was guided by seniors…not just academically, but in life decisions. It reminded me that growing older does not mean knowing everything.

CMC changed me personally, too. From a girl whose voice shook while holding a microphone, I became someone who could speak in front of twenty people every week. I learned to communicate, to teach, to listen. I learned Hindi, Bengali, Tamil, and Telugu words, and with them, their cultures and music. Medicine here required language, empathy, and adaptation.

Even research, which I once avoided, slowly became meaningful. I had believed research demanded too much effort for too little return. When my first paper was accepted, I understood that research is not separate from patient care. It is how one patient's story quietly teaches the world to treat the next patient better.

Even now, I often ask myself - does CMC make people like this, or does it choose them?

I still don't know the answer.

But I know this: **CMC Vellore did not just train me as a doctor. It quietly, steadily, and permanently changed who I am.**

CASES THAT CHANGED ME AS A DOCTOR

Dr. Illiasul Ibad
Assistant Professor, Department of Clinical Immunology and Rheumatology
Christian Medical College, Vellore

Medicine trains us to listen: a murmur through a stethoscope, a rhythm traced on paper, a number that crosses a threshold. But it rarely teaches us how to hear people. Some illnesses do not announce themselves. They settle quietly into the body and begin to alter life in small, almost deniable ways. I learned this from two women who walked into my life at different times, carrying different illnesses, but sharing the same burden: not being believed.

The First Woman: The Drummer's Fading Rhythm
Here is the first woman. She was 41, a drummer (Chenda percussionist) by passion: the kind of woman who could carry 10–15 kg drums on her shoulders, perform for 1–2 hours straight in concerts, and still go home to finish her chores. Strong, lively, unstoppable. Until one month ago, her rhythm began to fade.
It started subtly: dull, non-specific abdominal pain and an overwhelming fatigue she couldn't explain. No vomiting. No weight loss. No bowel change. Just constant tiredness. Her first visit to a clinic ended with a simple verdict: “fibromyalgia.”

She was started on pregabalin and told to rest. But her fatigue deepened. She felt slower, quieter, disconnected from her own energy. Then, as if to complicate the picture, she caught a mild viral upper respiratory infection. It was dengue season, and every patient with fever got a CBC. Her platelet count was 65,000: low enough for concern. She was labeled “probable dengue” and admitted for observation. The usual admission bundle followed: routine vitals, hydration, and an ECG. Everyone glanced at it and moved on. “Normal,” they said.

But I was a final-year trainee back then, fascinated by ECGs. I had a rule: never sign off an ECG without reading it step by step. So I sat down with her tracing

- Rate: normal.
- Rhythm: normal.
- PR interval: fine.
- QRS: fine.

Then, something odd. The QT interval looked shorter than expected, and there were U waves trailing behind the T waves in the chest leads. It didn't feel right. I went back to her bedside: she didn't look sick enough for dengue. No rash, no mucosal bleed, no postural drop. Just fatigue, muscle ache, and apathy. That ECG became our first clue.

We decided to chase the short QT. Serum calcium came back: 18.2 mg/dL.

PTH: 96 pg/mL (elevated).
24-hour urinary calcium: 987 mg (high).
It wasn't dengue. It wasn't fibromyalgia. It was severe hypercalcemia due to primary hyperparathyroidism.

Further evaluation with a sestamibi parathyroid scan revealed an inferior parathyroid adenoma: small, silent, but potent enough to push calcium sky-high and drain her energy. We treated her with IV fluids and furosemide, carefully lowering the calcium. Within days, her fatigue began to lift. The sparkle in her eyes returned.

A week later, she smiled and said, "Doctor, I feel light again… my body is mine now."

She later underwent surgery for the adenoma and fully recovered.

Two lessons from this case will stay with me forever:

1. Never ignore what the patient says. Her words: "I feel tired all the time," were brushed off twice, once as fibromyalgia and again as dengue. The body had been whispering for months.

2. Always read the ECG systematically. Even a "normal" tracing can hide a crucial clue. A short QT may not shout for attention, but it can change everything.

Medicine teaches us science every day. But sometimes, patients teach us humility. The drummer's rhythm came back once her calcium came down. And I learned that the best rhythm in medicine is listening: first to the patient, then to the body, and finally to your instincts.

The Second Woman: The Teacher's Unpredictable Story

The second woman arrived years later. I had just finished my MD in Medicine and was working as a young physician. Rheumatology fascinated me even back then.

One evening in the OPD, a 62-year-old retired schoolteacher walked in: calm, polite, but visibly tired. She spoke softly. "Doctor… I'm not the same since retirement. My joints ache, my mouth feels dry, I sweat and tremble at times… and people think I'm depressed."

She had seen multiple doctors. Labeled as stress, menopausal, functional. No one really believed her.

Her symptoms had actually started months earlier: episodes of palpitations, sweating, tremors, always relieved after food. She thought it was anxiety. So did everyone else.

Then came the fatigue, joint pains, and dryness of mouth and eyes. Outside, she was diagnosed as rheumatoid arthritis due to a mildly positive RF. Started on low-dose steroids and hydroxychloroquine. But something didn't fit: no deformities, no synovitis.

On examination, her mouth was parched, dental caries were evident, Schirmer's test showed only 3 mm wetting. It fit perfectly: Sjögren's syndrome. Anti-SSA came back positive. Finally, we had a name for her fatigue and dryness.

Or so I thought.
Weeks later, she returned: still exhausted. The tremors and sweating persisted. One day, she was near the hospital when another episode struck. She collapsed and was rushed to casualty. Her blood glucose was 40 mg/dL.
That changed everything.
We started evaluating for hypoglycemia:
Insulin > 600 µIU/mL
C-peptide 5.8 ng/mL
CT abdomen: normal
HbA1c: 5.4 %

We suspected Hirata's disease: Insulin Autoimmune Syndrome: and sent insulin antibody levels. It took time, but came back strongly positive (49 U/mL).

What is Hirata's Disease? Also called Insulin Autoimmune Syndrome (IAS): a rare autoimmune disorder where the body forms antibodies against its own insulin. These antibodies bind insulin after meals (causing high sugar), then release it unpredictably later, triggering sudden hypoglycemia.

She was referred to a higher center, where she was treated with steroids and rituximab. Her sugars stabilized, symptoms resolved.

Three months later, she returned: smiling. “Doctor, I'm fine now… thank you for believing me.”

In retrospect, everything made sense: the fatigue wasn't depression. The tremors weren't anxiety. It was her immune system writing its own unpredictable story.

Lessons:
1. Never ignore unexplained symptoms.
2. Don't label what you can't explain.

Medicine teaches science. Patients teach humility. She taught both.

“Doctor, I'm fine now… thank you for believing me.”

That sentence stays with me. Because neither woman demanded brilliance. They asked for attention. Their illnesses did not shout. They changed posture, pace, expression: leaving clues in plain sight.

Medicine is fluent in emergencies. But wisdom often lies in noticing what unfolds slowly. Listen before you label. Read before you dismiss. Respect what does not fit neatly. Science saves lives. But belief restores people. And sometimes, healing begins the moment someone finally pays attention.

WHEN MEDICINE ALONE IS NOT ENOUGH: A RHEUMATOLOGIST'S REFLECTION

Dr. Israrul Haque MD,
DM (Resident), MRCP (UK) SCE Rheumatology
IPGMER, Kolkata, India

In the ward, decisions are often reduced to numbers—creatinine values, estimated glomerular filtration rates, stages of chronic kidney disease. For a young woman with systemic lupus erythematosus and lupus nephritis, now in stage 4 chronic kidney disease, our decision during rounds was clear and clinically justified: escalation of immunosuppression would not meaningfully alter the trajectory of her renal disease. It was a rational decision, grounded in evidence.

Yet this was not the reason her story stayed with me.
What compelled me to write was not the severity of her lupus, but the long silence between her clinic visits—the three years of irregular therapy that had quietly, steadily eroded her kidneys. When I asked her where she lived, she replied, "Nawada district, Bihar." Almost reflexively, I asked why she had stopped treatment, even after being under the care of one of the most dedicated rheumatology centres in the country, the Institute of Postgraduate Medical Education and Research. I asked her, perhaps too bluntly, whether she wanted to live.

She responded not with words, but with tears.
She cried inconsolably, even as her mother tried to comfort her. When she finally gathered herself, I asked about her family. She was married in 2019, likely at the age of 18 or even earlier. By the age of 25, she had three children. Repeated pregnancies, relentless childcare responsibilities, and the daily struggle to survive had gradually pushed her own illness to the margins. Follow-ups became impossible; medicines became secondary.

When I asked about her husband, she cried again. After a long pause, she said he had left her a year ago. Even before that, he had not provided money for medicines or helped her travel nearly 500 kilometers to Kolkata for treatment. When I asked why she had not continued at least prednisolone—the cheapest and most basic of her prescribed drugs—she said a local practitioner had warned her that the medicine was harmful.

I then turned to her mother and asked why she had been married so young, and to an unemployed man. Her mother was initially silent. Later, she said she had four daughters, no fixed household income, and that marrying them off was the only solution she could see.

It was difficult to escape the feeling that failure existed at every level.
This was not merely a failure of adherence or patient education. It was the failure of social structures, economic security, gender equity, and healthcare delivery beyond tertiary hospitals. As rheumatologists, we increasingly practice sophisticated, evidence-based medicine. Almost every state in the country now has trained rheumatologists. And yet, stories like this remind us how extraordinarily difficult it remains to actually treat a patient.

Disease outcomes are not determined by genetics, epigenetics and treating Rheumatologist alone. They are shaped just as powerfully by geography, poverty, misinformation, and absence of support. Social history and economic history are not adjuncts to clinical history; they are central to it.

We encounter this repeatedly—patients stopping methotrexate because a pharmacist labels it "a cancer drug," or abandoning steroids because a quack declares them dangerous. When primary care providers and unqualified practitioners fail to recognise their limitations, and when referral pathways break down, the burden ultimately falls on the patient.

This case forces us to confront an uncomfortable truth: no matter how advanced our therapies become, malfunction at any level of the healthcare ecosystem leads to dysfunction of the whole. Treating rheumatic disease in India requires more than trained specialists—it requires a system where every level understands its role, its strengths, and its boundaries.

Until then, medicine alone will continue to fall short—for reasons that no laboratory test can measure.

IS IT THE END, OR THE BEGINNING OF ANOTHER END?

Dr. Deepak Ramchandra Malgutte[1],
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Lokmanya Tilak Municipal Medical College & General Hospital, Mumbai

Dr. Nilesh Nolkha[2]
[2] Professor, Department of Clinical Immunology and Rheumatology,
Topiwala National Medical College & B.Y.L. Nair Charitable Hospital,
Mumbai, Maharashtra, India;
Founder, Nirjara Multispeciality Clinic, Mumbai

Is It the End, or the Beginning of Another End?

Completing a DM or DNB in Rheumatology is an academic triumph. We are trained to recognise rare autoimmune diseases, interpret complex immunological markers, understand genetic and molecular pathways, and manage diagnostic uncertainty with confidence. Identifying subtle patterns and making difficult clinical calls becomes part of who we are.

Yet, once training ends, we realise something important.
While diagnosing a rare connective tissue disease may feel familiar, understanding how to function outside the education system feels far more difficult. Decisions now revolve around life, not lectures. Should one join a corporate hospital or build an independent clinic? Is early private practice a risk or an opportunity? How does one run a clinic, manage staff, handle pharmacy-related issues, understand finances, or plan long-term stability?

These are not clinical questions. But they define our professional future.
Ironically, these dilemmas are not new. They are discussed repeatedly—but never formally. They surface during coffee breaks at conferences, over lunch or dinner after academic sessions, during phone calls between colleagues, or in quiet peer conversations. Everyone talks about them. Everyone struggles with them. Yet they are rarely addressed on a structured platform, never taught systematically, and almost never discussed openly in front of an audience.

This gap creates quiet stress. Not because young rheumatologists lack capability, but because expectations suddenly change. Confidence inside the consultation room may coexist with uncertainty outside it. Growth feels slow. Comparisons feel unfair. And progress feels invisible.

As **William Osler**, often called the father of modern medicine, once said:
"Medicine is a science of uncertainty and an art of probability."
That uncertainty does not end with disease—it extends into professional life.

Recognising this reality, **Dr. Nilesh Noolka** led the **Clinician to Clinipreneur program**, first held in Mumbai on 14 December 2025. The intent was to give these long-ignored concerns a formal voice—to bring real-world challenges onto the stage. The program focused on areas often absent from training: practice models, corporate versus independent careers, financial awareness, pharmacy-related decisions, technology use, and the importance of physical and mental well-being.

More than advice, the program offered perspective.
It reminded young rheumatologists that success is not instant, clarity is not immediate, and uncertainty is not incompetence. Growth after training is gradual—and that is normal.

The transition from clinician to independent professional is not about knowing everything on day one. It is about navigating uncertainty with time, responsibility, and the willingness to learn beyond textbooks.

Articular Arts

Extra-academic talent of our Rheumatology colleagues

MUSIC: MY SANCTUARY BEYOND MEDICINE

Dr Renu Saigal
MD, FICP, MAMS, FRCP (Glasgow), FACP
Former: Sr Prof. & Head, Department of Medicine,
In-charge Rheumatology Services,
SMS Medical College and Hospital, Jaipur

Amidst the demands and responsibilities of my professional life, I have always believed in nurturing a space that belongs purely to the heart.

There was a time when my days were defined entirely by clinics, case discussions, and the constant pursuit of better patient care. Medicine has always been my calling—demanding precision, responsibility, and emotional resilience. Yet within that structured world, I sensed the need for a quieter, more reflective space—one that would nurture the inner self.

That space emerged during the challenging days of the COVID pandemic. The mental stress was overwhelming for everyone, especially those in the medical profession. It was during this time, by God's grace, that I came into contact with a music teacher who introduced me to the disciplined beauty of classical music. What began as a search for solace gradually evolved into a meaningful journey.

After long and intense clinical days, music became my sanctuary. It restores equilibrium, nurtures empathy, and reconnects me to silence and reflection. In many ways, music and medicine are not separate journeys but complementary paths—one heals the body, the other nourishes the spirit. Together, they create harmony not only in life, but within the self.

Encouraged by my guru, after completing my preliminary training, I had the privilege of performing on stage. That moment was deeply fulfilling. Today, this pursuit of music brings me immense mental peace and helps me navigate professional stress with greater calmness and balance.

For me, music is not merely a hobby—it is a gentle reminder that even amidst responsibility and rigor, the soul must find its own rhythm.

BIRDS OF INDIA
PHOTOGRAPHS & CAPTIONS

Col Arun Hegde
Prof Medicine & Consultant Rheumatologist,
Commanding Officer
Military Hospital, Belagavi

Asian Paradise Flycatcher (Female)
Terpsiphone paradisi
Belagavi, India
A female Asian Paradise Flycatcher perched alertly in forest undergrowth. The grey head, rufous wings, and elongated tail are characteristic of the species, which is widely distributed across the Indian subcontinent.
© Arun Hegde

Verditer Flycatcher
Eumyias thalassinus
Belagavi, India
A Verditer Flycatcher pauses at a water source, displaying its vivid turquoise-blue plumage. This insectivorous species is commonly found near streams and open woodland.
© Arun Hegde

Rufous Treepie
Dendrocitta vagabunda
Belagavi, India
A Rufous Treepie showing its long tail, rufous body, grey head, and contrasting white wing patch. An intelligent and adaptable bird, often seen close to human habitation.
© Arun Hegde

White- cheeked Barbet

Psilopogon viridis

Belagavi, India.

A green barbet found only in southern India. Note the dark brown head and characteristic white patterns on its face: a white patch around the ear-coverts, a white line above the eye, and a white throat. Also note the brown neck and breast with white streaks. Immature is duller and has less well-defined patterns on the face than adults. Heard more often that seen; constantly calls “kutrook-kutrook-kutrook” in mid-day hours from all kinds of wooded patches from small parks and homesteads to wet evergreen forests.

© Arun Hegde

Brown winged Kingfisher

Pelargopsis amarouptera

Bhitarkarnika National park, Odisha, India

Chunky large orangish kingfisher with an oversized head and a scarlet dagger of a bill. No other large kingfisher in its range has dark brown wings. Bright blue-green rump patch most obvious in flight. Restricted to coastal habitats such as mangroves and estuaries. Gives loud, fluid-sounding whistles as well as harsh grating rattles.

© Arun Hegde

Chestnut-capped Babbler

Timalia pileata

Bhitarkarnika, India

A Chestnut-capped Babbler observed in dense foliage. The chestnut crown and white throat contrast with the brown body, typical of this social undergrowth species.

© Arun Hegde

RHEUMATOLOGY IN THE AGE OF THE INTERNET

Dr Rahul Ladda
Consultant Rheumatologist
Rheumaderm Rheumatology and Skin Clinic
Kalaburgi, Karnataka

Once patients came with pain and trust,
Reports in hand, complaints a must.
They spoke, we heard, the story grew,
No search bar telling what was true.

Now patients enter, phone held tight,
Google glowing, data bright.
Before I ask, they gently grin,
“Doctor, I checked the net - log in.”

WhatsApp groups have solved the case,
With folded hands and urgent pace.
One forward swears, with holy might,
“All joint pain means uric acid spike.”

Instagram reels in sixty seconds show
From limping walk to marathon glow.
“No tablets, no doctors, no fear
Just this oil, link in bio, dear.”

Google adds ten diseases more,
Rare ones I've not seen before.
Symptoms ticked like shopping list,
Context quietly gets dismissed.

When I press the joint "Aiyo!" then,
The internet falls silent again.

No reel can feel that tender sigh,
No post knows why the tears run dry.

WhatsApp knows cures from auntie's friend,
Instagram filters hide the end.
Google predicts the worst by night,
By morning, hope is out of sight.

Yet still the net has played its part
Patients come early, ask questions smart.
They read, they learn, they care to know,
Which path to take, which way to go.

So I smile and say, not fighting tone,
“Use the net , but not alone.”
Let Google guide, WhatsApp amuse,
Instagram inspire - but don't confuse.

For when the joint must tell its tale,
And human judgment must prevail,
The screen steps back, the doctor leans
Between the lines, between the screens.

In this age of swipe and rhyme,
Rheumatology keeps its time.
Let the internet trend and shine
Healing, my friend, is still offline.

DOCTORS WITH A MELODY: THE MAKING OF INDORE'S MUSICAL ENSEMBLE, THE HEALING STORM

Dr Akshat Pandey
Consultant Rheumatologist
Apollo Hospitals, Indore

Music has always been a universal language that transcends boundaries—and doctors, too, often share a deep connection with it. Driven by this belief, I envisioned forming a unique musical band in Indore, composed exclusively of doctors. To the best of my knowledge, there has never been a group in India that brings together specialists and super-specialists who not only excel in medicine but are also accomplished multi-instrumentalists.

While several doctor groups perform through orchestras or karaoke-based acts, my aspiration was to unite exceptionally talented professionals on a common platform to create live, soulful music—crafted and performed with the finesse of seasoned artists.

This dream took years to materialize. The journey began with my longtime friend, MBBS senior and vocalist, Dr. Selexi Verma, a Radiologist blessed with an extraordinary voice. Having shared over 15 years of musical camaraderie, we finally found the time and energy beyond our clinical duties to transform our long-standing idea into reality.

Joining me in the band as a Rheumatologist by profession, a saxophonist by passion and an accompanying guitarist, we were quickly joined by Dr. Ashvini Dash, a seasoned plastic surgeon with more than twenty years of experience in Indore and a talented guitarist whose sense of humour,charm and modesty motivate everyone in his presence. Another key addition was Dr. Sushant Aildasani, a Neurologist from Apollo Hospitals, Indore. His quick adaptability and enthusiasm strengthened our lineup further.

Our rhythm section came alive with the youngest member, Rishabh Jain, a dynamic drummer whose technical acumen keeps our performances seamless. On percussion and vocals, we have Dr. Amit Verma, Professor and Head of the Department of Pathology, SAMC & PGI Indore, who adds great musical depth to our ensemble.

We also have Dr. Piyush Manker, a dedicated dentist by profession and a passionate bass

guitarist at heart! Mastering the bass guitar demands first conquering the fundamentals of standard guitar playing. While recruiting a bassist for our band was easy, discovering a doctor with such musical prowess proved a delightful challenge.

Complementing the team are two immensely talented non-medical artists—Mr. Sagar Dhaigude (Octopad) and Mr. Rupak (Keyboard)—both integral to our collective harmony.

Our first live concert on July 6, 2025, in Indore was met with overwhelming appreciation, drawing an audience of nearly 800 music lovers. Since then, the band has performed at several prestigious platforms, including IRACON 2025 at Yashobhoomi, New Delhi, NSICON, and state-level conferences on Diabetes and Cardiology at Bhopal, as well as at the RDGMC Alumni Meet, Ujjain.

Despite our demanding professional commitments—ranging from outpatient consultations and surgeries to academic responsibilities and teaching—music remains the common thread that binds us all. We make it a point to gather regularly, once or twice a week, for late-evening jam sessions in our dedicated music room in Indore. These sessions, held after clinic hours and often extending into the night, serve as a refreshing space where our shared passion for music rejuvenates our minds beyond the realms of medicine.

What began as a shared dream among a few musically inclined doctors has now grown into a symbol of passion, teamwork, and artistic expression—reminding us that medicine heals the body, but music heals the soul.

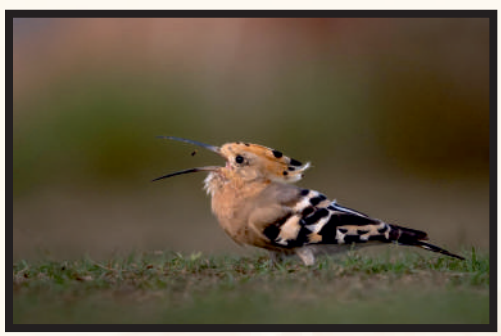
WILDLIFE PHOTOGRAPHY

Dr Sunil Kumar Mavidi, DM
Consultant Rheumatologist
Sunil Rheumatology and Immunology Centre
Visakhapatnam

PEEK- A- BOO: A pair of **spotted owlets** play a game of peek-a-boo from their cozy wooden hideaway. With golden sun-eyes and feathery polka-dot coats, they keep a watchful watch over the forest. Photographed at Keoladeo National Park, Bharatpur, India.

PEREGRINE FALCON

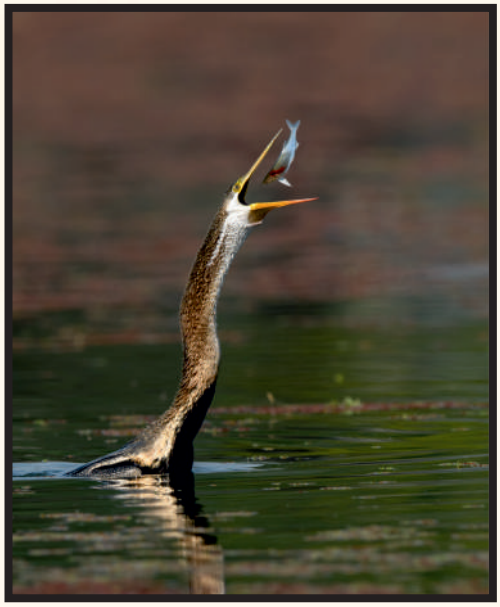
The Peregrine Falcon (Falco peregrinus): Famous for being the fastest animal/bird on the planet, reaching speeds of over 200 miles per hour during its signature hunting dive. Photographed at Sundarbans National Park, India.



COMMON HOOPOE: This zebra-winged bird wears a crown of feathers that opens like a royal fan. It uses its long, curved beak to dig for snacks hidden deep in the grass.

EGRETS

Two Little Egrets gliding gracefully against a golden, sun-drenched sky. These elegant white birds are a common sight in wetlands, known for their slow, rhythmic wingbeats and S-shaped necks.



INDIAN DARTER: This long-necked snakebird uses its sharp beak like a spear to hunt for fish deep underwater. After catching a meal, it flick-tosses the fish into the air to unstick it from its needle-like bill. It then catches the snack in mid-air and swallows it head-first so the scales don't get stuck in its throat. Photographed at Keoladeo National Park, Bharatpur, India.

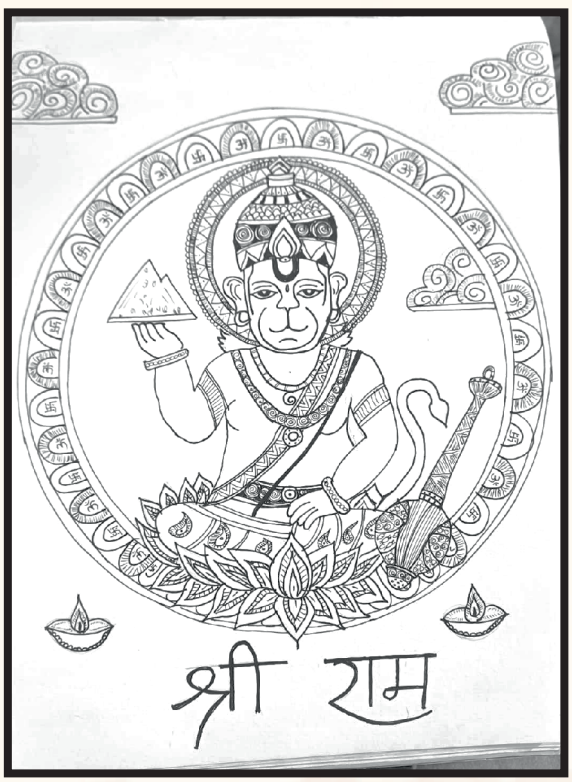
GREEN VINE SNAKE: A long, lime-green ribbon with a pointy nose drift through the leaves like a living branch. With its curious golden slit-eyes, this slender vine-mimic plays a never-ending game of hide-and-seek in the trees. Photographed at Jim Corbett National Park, India.

BENGAL TIGER: A living shadow of gold and ink strides boldly across the dusty path of its jungle kingdom. With a steady gaze and velvet paws, the striped beauty claims the road as tourists watch in silent wonder. Photographed at Jim Corbett National Park, India.

INCIDENTAL ART

Dr Keerthi Talari
Senior Consultant Rheumatologist
Yashoda Hospitals, Secunderabad

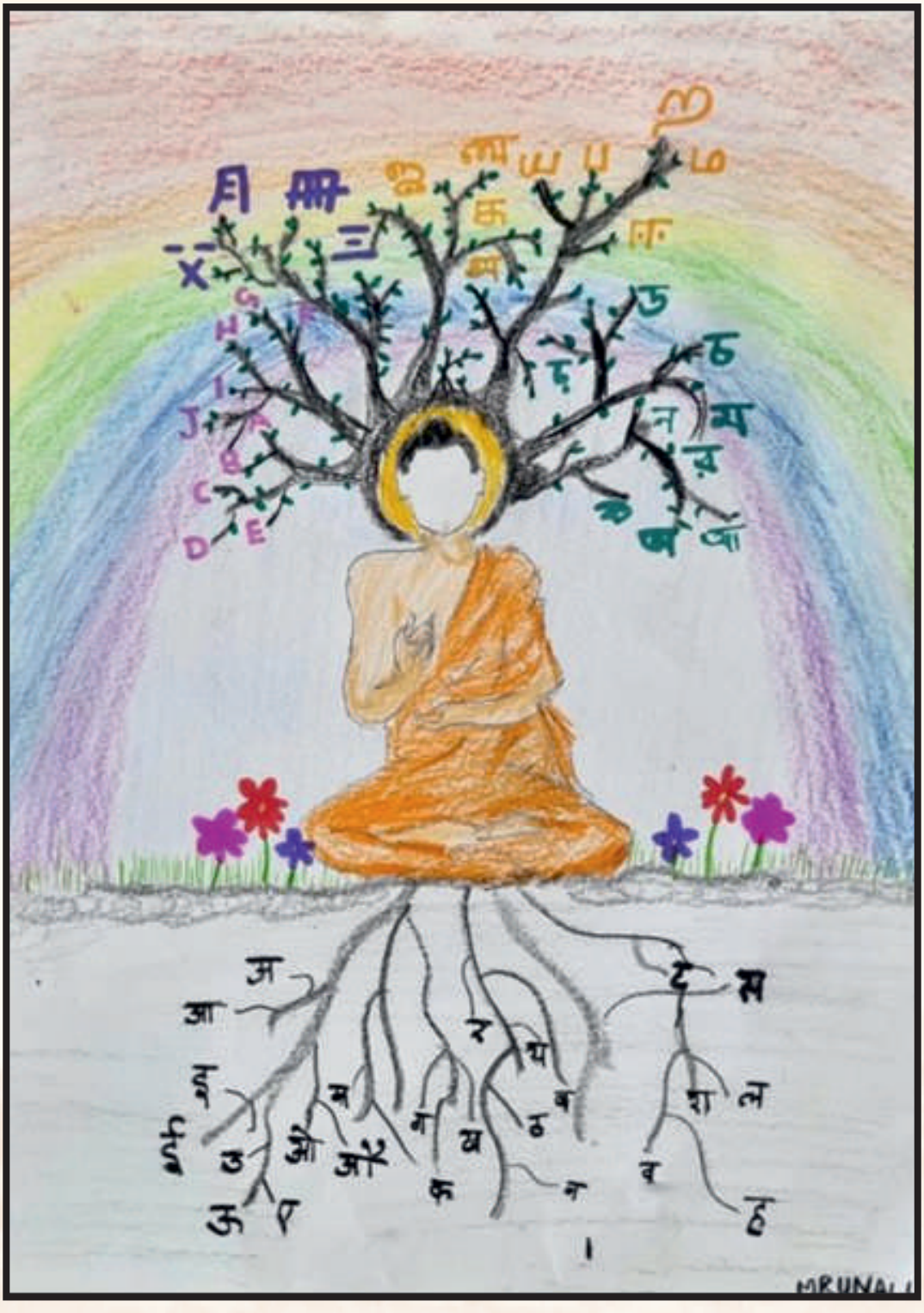
Inspired while accompanying my daughter Chaitra during her art classes, I discovered mandala art and created mandala representations of Lord Hanuman and Lord Shiva.



THE MINI-RHEUM REPORTER

1

Language is the roadmap of Culture
Learn a new language and get a soul



Mrunali, 8yr
D/o Dr Meha Sharma

②

Indian art is a living heritage where storytelling, symbolism, and everyday life intertwine, reflecting centuries of cultural wisdom through region-specific visual languages.

By Chaitra Bommakanti, 8 years
D/o Dr Keerthi Talari
Hyderabad

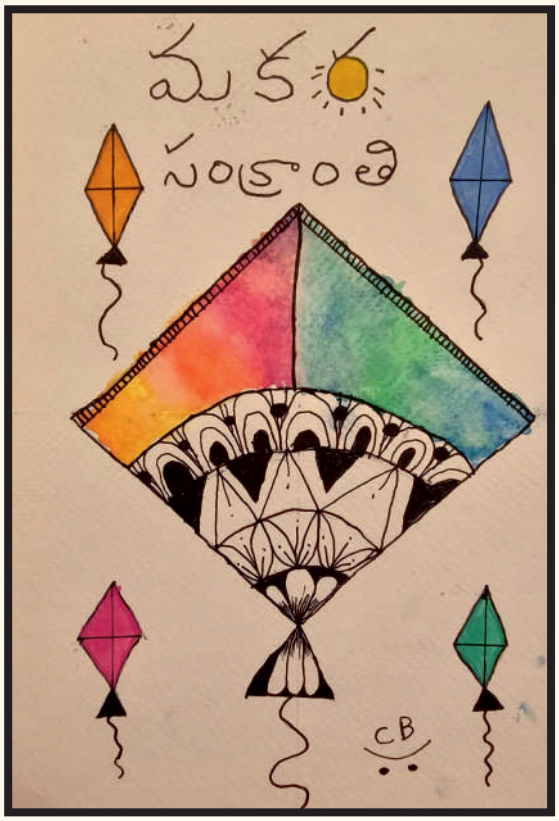
Warli art symbolically portrays the triumph of dharma through the fall of Ravana

Saura tribal art depicting the Mahabharata episode of "Ashwatthama Hatha Kunjaram," symbolizing moral ambiguity in war

Kalamkari artwork featuring lotuses, reflecting purity, resilience, and renewal in Indian tradition

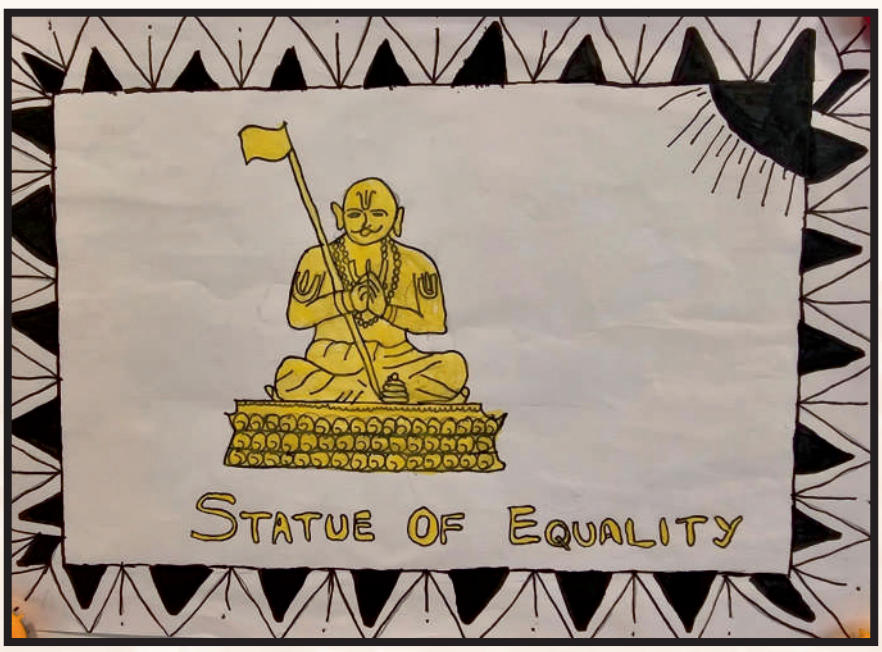
A mandala-inspired tortoise representing stability, longevity, and balance, drawn from Indian symbolism

In Madhubani art, the owl represents wisdom and watchfulness, reminding us to learn, observe, and think before we act



Indian art captures festivals in bright colors – Makar Sankranti

Indian art transforms monuments into stories on paper
Draupadi Rath (Mahabalipuram)

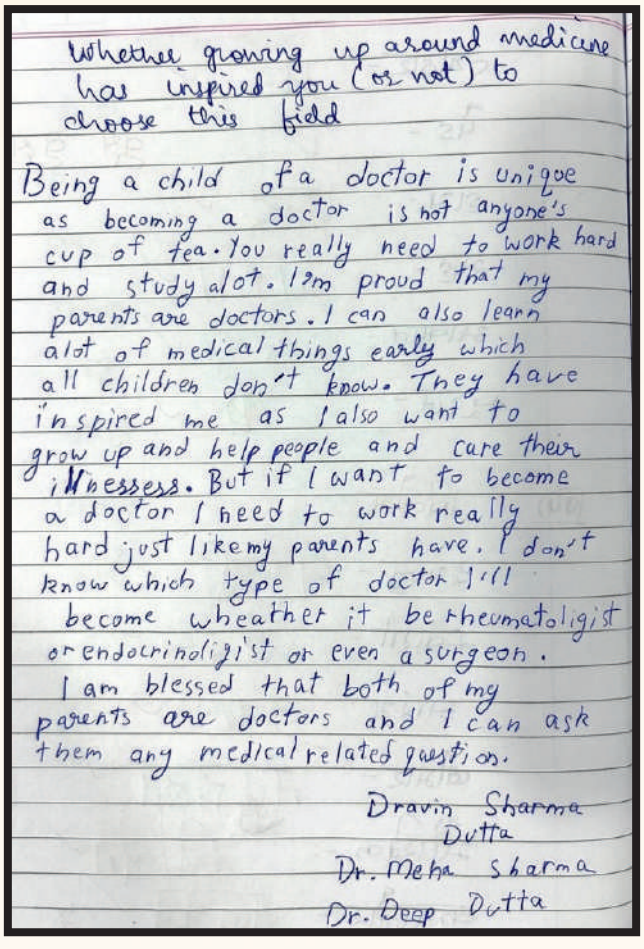


Statue of Equality (Hyderabad)

③ WITTY HUMOUR: THROUGH THE EYES OF INNOCENCE

Aniketh Vemuri, 9 years
S/o Dr Madhuri H R
Hyderabad

④ WHETHER GROWING UP AROUND MEDICINE HAS INSPIRED YOU TO CHOOSE THIS FIELD



Whether growing up around medicine has inspired you (or not) to choose this field

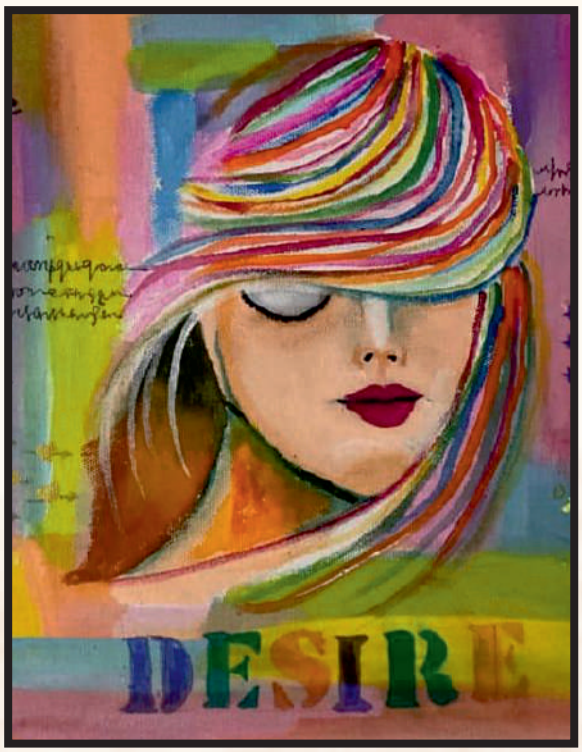
Being a child of a doctor is unique as becoming a doctor is not anyone's cup of tea. You really need to work hard and study alot. I'm proud that my parents are doctors. I can also learn alot of medical things early which all children don't know. They have inspired me as I also want to grow up and help people and cure their illnessess. But if I want to become a doctor I need to work really hard just like my parents have. I don't know which type of doctor I'll become wheather it be rheumatologist or endocrinologist or even a surgeon. I am blessed that both of my parents are doctors and I can ask them any medical related questions.

Dravin Sharma Dutta
Dr. Meha Sharma
Dr. Deep Dutta

Being a child of doctor is unique as becoming a doctor is not everyone's cup of tea. You really need to work hard and study a lot. I am proud that may parents are doctors. I can learn a lot of medical things early which most children don't know. They have inspired me as I also want to grow up and help people and cure their illness. But if I want to become a doctor, I need to work really hard like my parents have. I don't know which kind of doctor I will become, a rheumatologist, an endocrinologist or a surgeon. I am blessed that my both parents are doctors, and I can ask them medical related questions.

Dravin Sharma, 10 yrs
S/o Dr Meha Sharma
Delhi

5



Artist: Ridhi Singh 11 year
d/o Dr. Neena chitins
Mumbai

POWER OUR PAIN

6

Sai Cherith, 11 years
S/o Dr Vijaya Prasanna
Hyderabad

⑦ THE ONLY THING THAT OVERCOMES HARD LUCK IS HARD WORK

I will start with his wonderful quote, which I firmly believe in

" *The only thing that overcomes hard luck is hard work*"

Most of us feel that growing up in a family of intellectuals is a big blessing, indeed it is, but it comes with its own baggage.

I live in a family where my parents had been topper through out their school and entrance exams, my mama ji cracked the civil service in his first attempt, my nana ji (maternal grandfather started his own school at age of 60 years and running it successfully at age of 83 years), my aunt (maasi) is the most loved teacher in Ireland. So (I am not in a situation to brag), after seeing my background, you must have realised that I was born to work harder to prove myself.

I am just 12 years old, but I had been made aware quite early that I would have no time to slack. I could not afford late mornings or a messy home. I learnt my lesson quite early, the day I stepped into school, be it nursery, that I could not afford to skip studies. My morning would start with my father's hospital calls for 6 am insulin doses (I almost ended up memorising doses), my mom getting ready with phone stuck to one ear (managing admitted sick patients) and rushing out for a hospital with a hug and sweet smile while bidding goodbye (she would leave earlier than us). So, we (my younger sister and I) grew up, still growing, in a disciplined environment where our grandparents would narrate stories of wisdom and achievements of our parents.

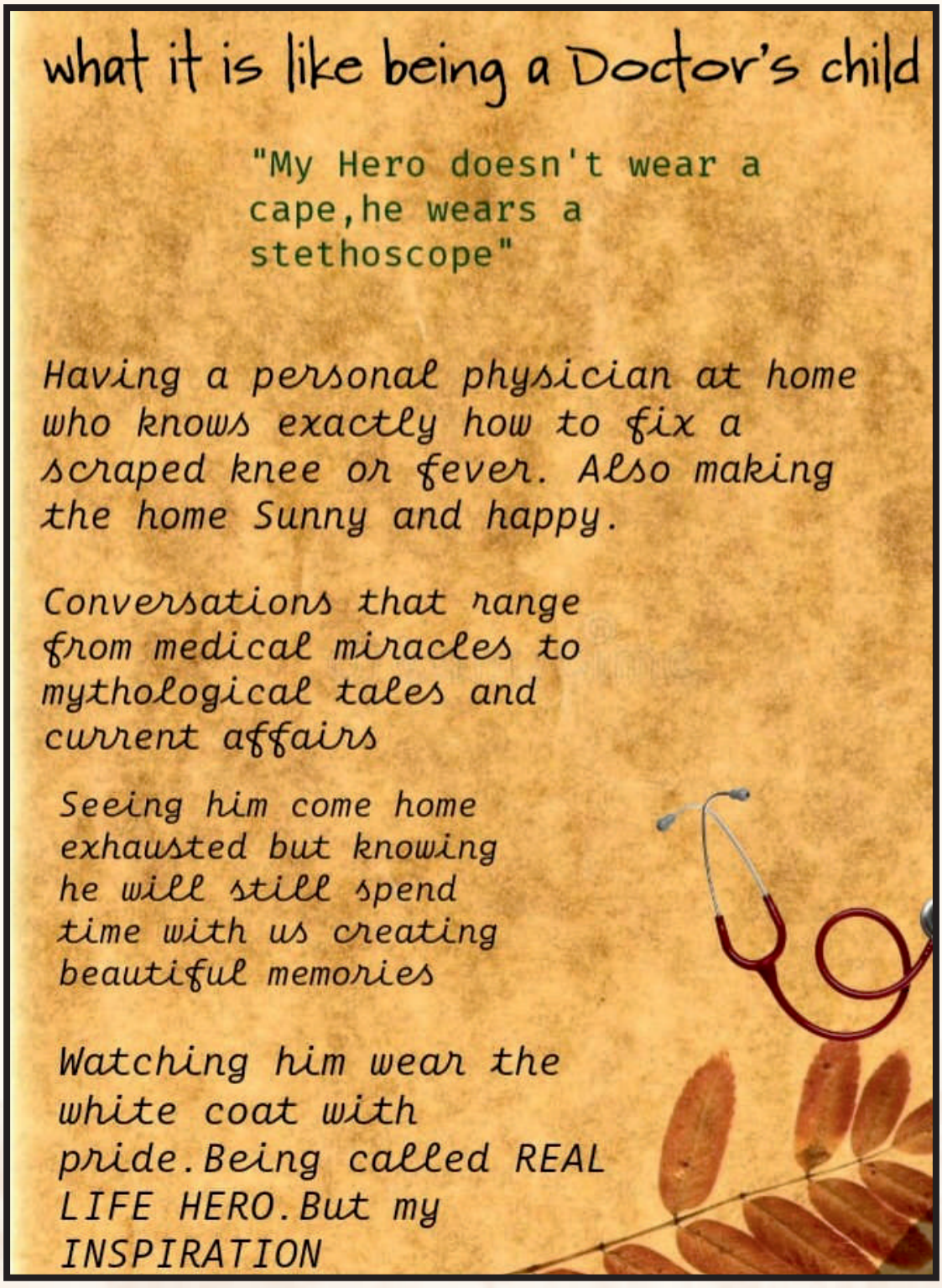
Though it's tough to compete with my own parents, I have evolved to be strong enough to rise and dream of. I would walk ahead carrying the legacy of my family with love and faith that my parents have bestowed on me. I believe my parents' hard work gave me a purpose in life. So, indeed, it's a blessing to be born in the family of intellectuals.

With lots of love

Nandini , 12 years
(A proud daughter of Dr Aarti Sharma and Dr Deepak Khandelwal)
Delhi

BEING A DOCTOR'S CHILD

8



Advika Khare, Class 8th
D/o Dr Apurva Khare
Professor, Medicine
L N Medical College, Bhopal

⑨ BEHIND THE WHITE COAT: THE LIFE FEW TRULY UNDERSTAND

Not every branch of medicine is widely understood, yet these less-understood branches play a very crucial role in protecting a person's quality of life. Rheumatology is one such field.

Instead of being widely recognized, rheumatology is a field that quietly works in the background, helping people preserve something many of us rarely think about, the ability to move freely and live without constant discomfort. To me, rheumatology is about understanding conditions where the body begins to fight itself, requiring not
just medical expertise but patience and long-term care. What makes this branch of medicine especially remarkable is its focus on improving everyday life, allowing individuals to continue doing ordinary things that suddenly become precious when they are at risk. Something that fascinates me most about how complex these illnesses is that *no two patients experience them in the same way*. Hearing about rheumatology from my father has shown me how complex the human body truly is and how important it is to approach illness with both knowledge and empathy. It has also made me realize that some of the most impactful areas of medicine are not always the most visible, yet they transform lives in lasting ways.

What makes many rheumatic diseases especially challenging is that they are often invisible and lifelong. A person may look completely healthy while silently dealing with discomfort and exhaustion. Conditions such as lupus are chronic and usually require ongoing treatment rather than a permanent cure. This invisibility can sometimes make the struggle harder, because others may not fully understand what the patient is experiencing. Unlike most injuries, there is no cast or bandage to signal that someone needs support. Yet these diseases can influence nearly every aspect of a patient's life, from work and school to relationships and mental well-being. Knowing this has changed the way I see strength. Strength is not always loud or dramatic; sometimes it is simply waking up each day and continuing despite pain.

Another realization I have had is how little these diseases are talked about. Whenever I tell my friends that my father is a rheumatologist, I am almost always met with the same confused question: “What is that?” Most people know about fields like cardiology or pediatrics, but rheumatology rarely comes up in everyday conversations. One day in class, while we were reading an economics chapter, an example mentioned a person suffering from arthritis. While the term was familiar to me, I noticed that almost all of my classmates were hearing it for the very first time. This is just one example of how little people know about rheumatic diseases. This lack of awareness can delay diagnosis for patients. A common misconception is that conditions like arthritis affect only older adults, which often prevents people from recognizing how widespread they truly are. Greater awareness is essential, as these illnesses can also impact working individuals and even children, making early understanding and support more important than ever. Increasing awareness is important so that people recognize symptoms earlier and seek proper medical care. Education through schools, social media, and public discussions could make a meaningful difference. Even small conversations can spark curiosity and understanding. I believe that the more we

talk about rheumatic diseases, the less isolated patients will feel.

Even though many rheumatic and autoimmune diseases are lifelong, they do not define a person's future or prevent them from achieving their goals. With the right care, resilience, and determination, patients can continue to live meaningful and successful lives. Many well-known public figures are living examples of this strength. Singer and actress Selena Gomez has openly shared her journey with an autoimmune condition while continuing to grow in her career and use her voice to spread awareness. Similarly, supermodel Gigi Hadid has spoken about managing a chronic autoimmune illness while thriving in the fast-paced and demanding fashion industry. Their stories remind us that although these conditions may remain a constant part of life, they do not diminish a person's potential. Instead, they highlight the power of perseverance and the reality that individuals can adapt, succeed, and shine despite the challenges they face.

Growing up as a doctor's daughter has shown me a life that runs on dedication rather than a predictable schedule. Phone calls can come at the most unexpected times, and workdays can stretch far beyond what most people would consider normal, sometimes lasting ten hours or more. What amazes me is that the learning never stops; there are evenings when my parents are still reading, researching, or discussing cases long after the day is technically over. Sometimes it even feels like they study more than I do. This environment has taught me discipline without anyone needing to say it aloud. It has also helped me understand that medicine is not just a profession; it is a commitment that extends beyond hospital walls and into everyday life. Watching this has made me more responsible and aware, shaping how I view the world and success.

All of these experiences naturally led me to the greatest influence in my life: my parents. Every day, they devote their energy to improving someone else's quality of life. Much of a doctor's effort goes unnoticed by the wider world, yet its impact is deeply felt by the people they help. Their compassion and persistence inspire me more than words can express. Seeing how much they give has encouraged me to consider following a similar path when I grow older, not just because it is admirable, but because it is meaningful. They showed me, and keep showing me, just how amazing the science of the human body truly is. Seeing my parents work has shown me that no career can be as interesting and rewarding as being a doctor. If rheumatology teaches us anything, it is that healing is not always about curing; sometimes it is about supporting, guiding, and standing beside someone for the long journey. From understanding the science to witnessing the human side of care, everything connects back to one value I learnt from my father: a life dedicated to helping others is a life truly well lived.

Ojas Malviya , 14 yrs
D/o Dr. Sourabh Malviya, Indore

⑩ THE OVERLOOKED SIDE OF A DOCTOR'S LIFE

In today's life, doctors are looked upon, described and imagined as a life of achievements and honour. But behind all that is a personal life full of strain & burnouts

- ☆ Stressful life - The life of the patient depends on the doctor, putting a lot of stress. The doctor has to stay in the hospital even on his or her birthday putting strain on his personal relationships.
- ☆ Occupational hazard- There-is small room for error in this profession, causing one mistake to take the life of the patent. Moreover, the doctor can also get infected by the patients disease or by Sleep deprivation. Because the infection does-not take a break, the doctor and surgeon have to be available at all times, causing seep deprivation, which can cause poor judgement and awareness .
- ☆ Income gap: due to expensive coaching and medical college doctor has to take huge loan. While the overall salary is high he/she has to pay the amount by struggling hard.
- ☆ High burnout rates: The amount of stress can cause doctor to resign his post or leave this field.

While most of us focus on the positive side of doctors, but we can't ignore this side of the occupation also.

Vatsal Bhadu 14y, Class 8
S/o Dr Danveer Bhadu
AIIMS, Delhi

⑪ THE RHEUMATOLOGIST'S CHILD

Praurochita Pradhan
15 yr, Class 10, ODM Public School, Bhubaneswar
D/o Dr Prasanta Padhan
Head, Department of Rheumatology
Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar

I belong to a doctor's family! My father and mother both are doctors, and both of them are equally inspiring. Papa, he's a rheumatologist, dealing with joint pain for the past 20-30 years. I've seen him examining the patients in the best way possible by asking history and much more. Whenever I ask him about his favourite place of work and where he learnt the most about rheumatology, he gives the highest credits to CMC Vellore. Yes, even other institutions were a big part of him, but CMC was something special to him. It was even more special to him because of Late Danda Sir. Undoubtedly, I can always understand from Papa the love and respect he has for Sir.

Each and every day when Papa returns from the hospital on his opd days, I ask him, how many patients did you see today? And did any children come to your opd today? He would always reply the correct number and definitely mention the cities from which most patients came, and most likely say yes, there were two or three children too. I've always seen my father being genuine; he has always taught my sister and me not to run behind money but run behind making patients alright. He says, nothing can bring more happiness than giving a patient a smile and a patient's attendant and family a smile. He doesn't go for practice, stays with us in the evening and discusses what it is like to be a doctor. Passion is the most important thing.

I've always heard Papa discussing with his residents about various diseases like Arthritis, SLE, ILD, and Lupus on a daily basis. He has always ensured not to exploit the patients by asking them to do various tests or take expensive treatment. This part is something that inspires me the most from him. As far as I've understood, steroids are an important treatment for rheumatological diseases and an injection of Rituximab, I don't know more. Common people really have a mindset not to take steroids, I've seen Papa explaining it in the best way possible about its necessity for the disease to be cured.

To be a doctor's girl, in simple terms, is special. A very different life from others, honestly. There is so much to learn from them. Most importantly, discipline. I've seen Papa's young patients crying out with pain and suffering, it keeps me inspired to take a proper diet and live a healthy lifestyle to avoid such diseases. Papa has always told me, don't go for NEET, don't be a doctor (this is an honest thing). I had also never thought of becoming a doctor because of the stressful life and all the negative aspects. However, when I had to choose my stream, I sat one day and thought what would be the best for me, something in my brain and heart told me, you should be doctors like your parents. Like your parents had

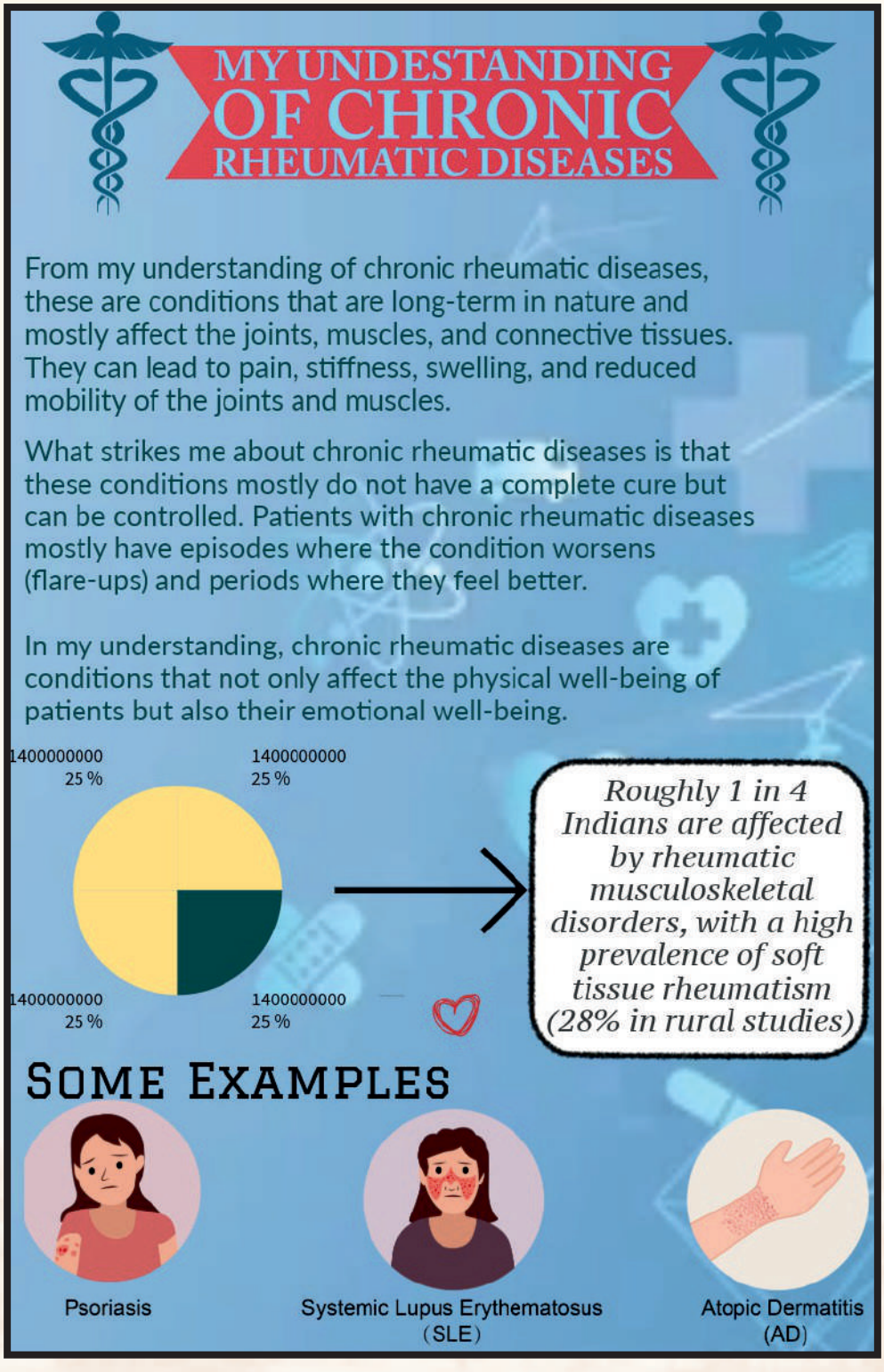
a deep impact on me. It is definitely boring to listen to ILD, CBC, and CRP day and night, but that's just how it is. Mumma is a pulmonologist, Papa is a rheumatologist, and I hear them discussing diseases every time (most commonly ILD, haha). Most importantly, they're never jealous of each other, but they learn from each other.

I wish to be a doctor like my parents and serve society in the future!

Thank you for giving me this wonderful opportunity to write this and speak my heart out.

⑫

MY UNDERSTANDING OF RHEUMATIC DISEASES



Arivan Khare, Class 11th
S/o Dr Apurva Khare
Professor, Medicine
L N Medical College, Bhopal

⑬ STEPPING INTO MY FATHER'S SHOES

Growing up with a parent who is a doctor is both inspiring and difficult. On one hand, I am proud of my father and the work he does. Knowing that he helps people when they are sick or scared makes me admire him. His work has taught me the value of hard work, responsibility and caring for others. I have learnt to stay calm in the most stressful situations, also because that is how he usually handles emergencies.

At the same time, having a doctor as a parent sometimes makes me feel lonely. His long hours and unpredictable schedule often mean missed family time, school events, or simple moments at home. Even when he is present, he is sometimes exhausted or distracted, and I sometimes feel hesitant to share my problems because he already deals with so much at work. There are times when he just ignores listening to me, also. There are moments when I wish my father could just be there for me without thinking like a doctor. Thankfully, my mother isn't a doctor, and that's what brings solace to my family. Atleast, we don't discuss medical terms and complex cases on the dining table or during family time.

Despite these challenges, growing up this way has made me more independent and resilient. I learned early to manage on my own and to finish my work in time before he is home. I also learned to schedule my tasks more meticulously so that I can spend time with him when he is free. I understand that his absence is not a lack of love but a result of responsibility and dedication. Still, understanding does not always erase the feeling of missing him.

In the end, my feelings are mixed. I carry both pride and frustration, admiration and longing. Being the child of a doctor has shaped who I am, teaching me empathy, strength, and patience. While it is not always easy, this life has helped me grow even through its challenges. I know it isn't easy to live as a responsible doctor, catering to everyone's health problems, yet I am inspired to follow my father's footsteps and hope to become a good clinician.

Prapti Kedia 17 yrs
D/o Dr. Arun Kedia
Raipur

⑭ ILLNESS, IDENTITY AND ART: THE FLANNERY O CONNOR STORY

Disease has often given birth to unique lenses of expression. Literary history is rich with writers whose work has been forged in the crucible of illness, from themes of the “white plague” and morbidity in Keats's odes to the light-starved cosmology in Milton's greatest work **Paradise Lost**, composed after his blindness.

For women, this intersection of creativity and illness has been further complicated by historical medical neglect. Symptoms of chronic illness were often dismissed as “female hysteria,” a pattern tragically familiar to many with Lupus. This autoimmune disease, which disproportionately affects women, presents with elusive, fluctuating pain and fatigue, long mistrusted by medical practitioners. No writer embodies this convergence more powerfully than **Flannery O'Connor**. Diagnosed with lupus at twenty-five, O'Connor retreated to her Georgia farm, her life shrinking to a porch and a typewriter. Critically, her illness directly shaped her chosen literary form. Lupus robbed her of the stamina to write for hours on end, making the dense, impactful short story her natural medium over the sustained effort of a novel. Her fiction, often categorized as Southern Gothic, explores the intersection of flawed humanity and divine grace through a startling lens. The grotesque physicality of her characters, like their blindness, amputations, and disfigurements, reads as a direct transcription of her own bodily reality. She knew what it was to live in a body that was a source of limitation and betrayal, and this lived experience gave her the unsentimental authority to render her characters' flaws.

This authority extends to the most defining feature of her work, that is, a theological vision where grace is often preceded by violent, bodily disruption. As a devout Catholic, O'Connor populated her stories with depraved characters and shocking acts of violence, an incongruity she explained by stating that violence “is strangely capable of returning my characters to reality and preparing them to accept their moment of grace.” Her narratives orchestrate a divine stripping of human comfort and hubris, using the degradation of the corporeal to shatter her characters' pride. Nowhere is this process more starkly illustrated than in her iconic story, "**A Good Man Is Hard to Find**." The grandmother, a figure of superficial piety and manipulative nostalgia, is relentlessly comfortable in her selfish worldview. Her journey culminates in a sudden, violent encounter with the serial killer, The Misfit in a roadside ditch. Faced with imminent death, her social pretenses are obliterated. In her final, desperate moment, she reaches out to touch The Misfit and calls him one of her own children. This is a fleeting, ambiguous gesture of grace born from utter devastation. The violence of the scene is the instrument that returns her to the fundamental reality of her mortality and her need for connection, perfectly embodying O'Connor's artistic principle.

In a culture that prized health and feminine delicacy, O'Connor herself, a young woman on crutches, her face altered by steroids, lived as an outsider. This perspective allowed her to create characters who are physical and spiritual freaks, whose very bodies force a violent conflict that could lead to redemption. It is unfortunate that the “grotesque freak” remains a lived reality for many who suffer from lupus today. It is our modern duty to look beyond the “grotesque” surface, be it of a character or a person, and listen

seriously to the narratives of chronic illness. O'Connor's gothic vision, where grace arrives through the breaking of a flawed body, was a product of her disease; **our understanding must be a product of our empathy.**

Ashita Kedia
3rd year BA Honours (Eng Literature)
Miranda House, New Delhi
D/o Dr Arun Kedia, Raipur

⑮ THE INHERITANCE OF EMPATHY

Dear Mom,

Our home was never only a home. It held the quiet presence of hospital corridors, patient stories, and responsibilities that did not end with office hours. Phone calls interrupted meals, and conversations often paused midway. Even as a child, I understood that medicine stayed with you. It followed you home, and in doing so, shaped all of us.

I did not understand illnesses or treatments then. What I understood was care. I noticed how your voice softened after long days, how patience seemed endless, and how listening was never hurried. Patients entered our lives without names or faces, but their struggles were real enough to leave a mark. Through you, I learned that pain does not always demand urgency. Often, it asks for steadiness.

I did not grow up learning anatomy. I grew up learning how chronic illness lives alongside people, not above them. From the outside, rheumatology looked less like dramatic cures and more like commitment. Healing was not always about fixing. It was about reassurance, continuity, and trust built slowly over time. Watching you, I learned that empathy is not separate from clinical expertise. It is what allows it to heal.

Today, I am a designer. My work is about understanding people deeply and designing solutions that respond to real human needs. Much of what I do echoes what I absorbed growing up. Listening before acting. Asking better questions. Respecting complexity rather than rushing to conclusions. Whether in medicine or design, meaningful solutions begin with empathy and are sustained by patience.

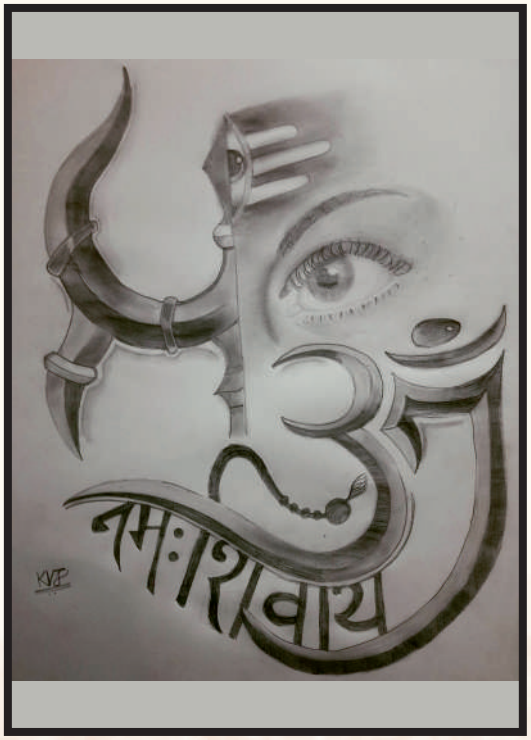
Growing up in your world also meant growing up around learning that never stopped. Academic discussions, conferences, and initiatives like WHIRA were not just professional milestones. They were lessons in leadership, collaboration, and responsibility. I saw what it meant to create impact not only through individual practice, but by building communities that support and uplift others.

This letter is not about professions or paths. It is about gratitude. I may not have inherited your practice, but I inherited your way of seeing people, with attentiveness, respect, and compassion. That inheritance shapes how I think, how I design, and how I show up in the world.

Thank you for bringing your work home, even when it was heavy. Thank you for teaching me, without ever meaning to, that empathy is a strength. It remains the most valuable thing I carry forward.

With love,
Your beloved daughter,
Karmishtha
D/o Dr. Kavita Krishna
Professor & Head Department of Clinical Immunology & Rheumatology at Bharati Vidyapeeth University Medical College & Hospital, Pune

16



Artist: Kaustubh Vardhan Joshi
4th year MBBS student, SKNMC Pune
s/o Dr Vaijayanti Lagu Joshi
Senior Consultant rheumatologist
Deenanath Mangeshkar Hospital.
Pune, Maharashtra, India

WRF SUMMIT 2023
HIGHLIGHTS (JAIPUR)

WRF SUMMIT 2023
HIGHLIGHTS (JAIPUR)

WRF SUMMIT 2023
HIGHLIGHTS (JAIPUR)

WRF SUMMIT 2024
HIGHLIGHTS (CHENNAI)

FACULTY HIGHLIGHTS

CROSSWORD

Dr Ishan Verma, MD
Associate professor, General Medicine,
AIIMS Nagpur

1	2				■	■	3					■	4
5				■	■	■		■	■	■	■	■	
	■	■	6	■	■	■	7	8		9	10		11	
	■	■		12	■	■	13					■		
14		15			■	■			■		16			
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24				■		■	■	■	■	25	■		■	■
	■	26				■	■	■	■	27			28	■
	■		■	■	■	■	■	■	■		■	29		
■	30												■	■

Left to right

1. Inflammasome protein encoded by the MEFV gene
3. Sounds like a febrile arthritis, but is a Bhilai factory product
5. Adult-onset autoinflammatory disease with fever, arthritis, and salmon-coloured rash
7. PIP joint nodule in Osteoarthritis
13. Target of belimumab in Lupus
14. Autoinflammatory disorder due to IL-36RA deficiency
16. Chemiluminescence-based immunoassay used in autoantibody testing
17. Lateral thigh rash characteristic of dermatomyositis
18. Mitochondrial flavoprotein that serves as a key mediator of caspase-independent apoptosis
20. Osteocyte-derived inhibitor of Wnt signalling; target of Romosozumab
21. Oral C5a receptor (C5aR1) inhibitor used in ANCA-associated vasculitis
23. Key transcription factor regulating inflammatory and immune responses
24. Can show pitting, onycholysis or “oil-drop” change in psoriasis
26. Provocative test for shoulder impingement syndrome
27. Tick-borne spirochaetal infection causing migratory arthritis
29. Element essential for haemoglobin synthesis
30. Anti-CD20 therapy for lupus nephritis supported by the REGENCY trial

Top to bottom

1. Not a rheumatology term—crispy Indian snack
2. Paraneoplastic antibody causing cerebellar ataxia, classically with breast/ovarian cancer
3. Scientist linked to folic acid and methotrexate
4. Lupus autoantibody correlating with disease activity and nephritis
6. Fc receptor-like family of immunoregulatory receptors on B cells
8. Prefix denoting involvement of less than 5 joints
9. Current term for chronic idiopathic urticaria
10. High-sensitivity marker of low-grade systemic inflammation
11. You are solving this crossword here
12. Composite disease activity score used in rheumatoid arthritis
15. JAK1/3 inhibitor
17. Autoinflammatory syndrome due to mevalonate kinase deficiency
18. Recombinant IL-1 receptor antagonist
19. Diabetic cheiroarthropathy sign
22. Reduced haemoglobin concentration in blood
25 Autoinflammatory disease due to NOD2 mutation with arthritis, uveitis, and rash
28 C5–C6 brachial plexus palsy (waiter's tip)

1 P	2 Y	R	I	N	■	■	3 S	T	E	E	L	■	4 D
5 A	O	S	D	■	■	■	U	■	■	■	■	■	S
P	■	■	6 F	■	■	■	7 B	8 O	U	9 C	10 H		11 R	D
A	■	■	C	12	■	■	13 B	L	Y	S	S	■	A	N
14 D	I	15 T	R	A	■	■	A	I	■	U	16 C	L	I	A
■	17 H	O	L	S	T	E	R	G	■	■	R	■	P	■
18 A	I	F	■	■	■	■	A	O	■	■	P	■	U	■
N	D	A	■	■	19	■	O	■	■	■	■	■	R	■
A	20 S	C	L	E	R	O	S	T	I	N	■	■	■	■
K	■	I	■	■	21 A	V	A	C	O	P	22 A	N	■	■
I	■	T	■	■	Y	■	■	■	■	■	23 N	F	K	B
24 N	A	I		■	E	■	■	■	25 B	■	E	■	■	■
R	■	26 N	E	E	R	■	■	■	27 L	Y	M	28 E	■	■
A	■	I	■	■	■	■	■	■	A	■	29 I	R	O	N
■	30 O	B	I	N	U	T	U	Z	U	M	A	B	■	■

Top to bottom-

1. PAPAD
2. YO
3. Subbarao
4. DSDNA
6. FCRL
8. Oligo
9. CSU
10. HsCRP
11. Raipur
12. DAS
15. Tofacitinib
17. HIDS
18. Anakinra
19. Prayer sign
22. Anemia
25. BLAU
28. ERB

Left to right-

1. Pyrin
3. Steel
5. AOSD
7. Bouchard
13. Blys
14. DITRA
16. CLIA
17. Holster
18. AIF
20. Sclerostin
21. Avacopan
23. Nfkb
24. Nail
26. Neer
27. Lyme
29. Iron
30. Obinutuzumab

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RA - Rheumatoid arthritis; SLE - Systemic lupus erythematosus
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