
Irish Society
for Rheumatology
Spring Meeting
4-5 May 2023

Welcome Message from the ISR President Prof Geraldine McCarthy

Dear Guests, Colleagues and Friends,

Welcome to Kilkenny for the 2023 Spring meeting. Lockdown seems like a dim distant memory now. Many thanks to our colleagues at St James Hospital who have put together a brilliant programme and this meeting promises to be an excellent one.

On Thursday afternoon, there will be a CAG session chaired by Professor David Kane and this will set the scene for the meeting. Hopefully there will be excellent attendance. The first invited talk will be by Professor Mike Putman from the Medical College of Wisconsin who will speak about evidence-based medicine in Rheumatology. Then Dr Michele Doran and Professor Barry O'Shea will bring us up to date regarding our national registries in rheumatoid arthritis and ankylosing spondylitis. Last on the official agenda will be a presentation from Professor Gaye Cunnane with an intriguing title. I suspect there will be some health and history involved but it will certainly be excellent.

On Friday morning, Dr Brenda Griffin will speak about renal diseases of importance to us as Rheumatologists followed by Professor John Pauling from the University of Bristol who will discuss challenges in vascular ischemia of scleroderma with, hopefully, some approaches to dealing with them. We will then have presentations of clinical cases, which never fail to keep us thinking. Finally, Dr Natasha Jordan will inform us regarding what we need to know about the management of pregnancy in lupus.

I believe Rheumatology in Ireland continues to be in a very positive place. The applications for training in Rheumatology are increasing and I believe we can continue to recruit excellent candidates by making efforts to expose medical students and junior staff to our wonderful specialty.

I would like to offer special thanks to Michael Dineen and Marie Caston who have yet again worked extremely hard to make this meeting a success.

I am especially grateful to our colleagues in the Pharmaceutical Industry for the continued support of ISR and its members.

Finally, my sincere thanks to the members of ISR board for all their continued support and enthusiasm.

Please enjoy this opportunity to get together, share ideas and maybe have a laugh as well!

Prof Geraldine McCarthy
President ISR

ISR Spring Meeting
4-5 May 2023
Programme

Thursday, 4 May 2023

13.00 - 14.00	**Light Lunch**
14.00 - 14.50	**CAG: Chaired by President ISR**
14.50 - 15.00	**Opening Address: Prof Geraldine McCarthy,** President ISR
15.00 - 15.45	***Misconceptions and Opportunities: Evidence-based Medicine in Rheumatology*** **Prof Mike Putman,** Director of the Vasculitis Program, Medical College of Wisconsin USA
15.45 - 16.15	**Coffee and biscuits**
16.15 - 16.45	***Rheumatology registries in RA and AS*** **Dr Michele Doran and Prof Barry O'Shea**, Consultant Rheumatologists, St James's Hospital, Dublin
16.45 - 17.45	***In the direction of flow - the intermingling of history and humanity*** **Prof Gaye Cunnane**, Consultant Rheumatologist, Director of Health and Wellbeing, RCPI
19.30	**Dinner**

Friday, 5 May 2023

09.00 - 09.45	***What rheumatologists need to know about the kidneys*** **Dr Brenda Griffin,** Consultant Nephrologist, St James's Hospital, Dublin
09.45 - 10.30	***Challenges in peripheral vascular ischaemia in scleroderma-spectrum disorders*** **Dr John Pauling,** Consultant Rheumatologist, University of Bristol, UK
10.30 - 11.00	**Coffee and biscuits**
11.00 - 12.00	**Clinical case presentations**
12.00 - 12.45	***Management of Pregnancy in SLE*** **Dr Natasha Jordan,** Consultant in Adolescent Rheumatology, SJH and Children's Hospital Ireland
12.45	**Prize-giving and close of meeting followed by lunch.**

Biographical Sketches

Speakers

Dr Natasha Jordan
Consultant Adult and Adolescent Rheumatologist
St James's Hospital and Children's Health Ireland

Dr Natasha Jordan is a Consultant Adult and Adolescent Rheumatologist, currently based at St James's Hospital and Children's Health Ireland, Crumlin. Her major clinical and research interest is SLE.
Dr Jordan studied Medicine at University College Dublin followed by Higher Specialist Training in General Internal Medicine and Rheumatology. She subsequently worked for 5 years at the Louise Coote Lupus Unit at St Thomas' Hospital in London, where she was the recipient of both an Arthritis Research UK Fellowship and a Graham Hughes Clinical Research Fellowship. She obtained her PhD from King's College London investigating the genetics of lupus nephritis. She then took up a substantive post as a Consultant Rheumatologist for 7 years at Addenbrooke's Hospital in Cambridge. While in Cambridge, she was Deputy Director of the Rheumatology Clinical Research Unit and ran phase II and III trials in SLE. In 2018, she led the bid for Addenbrooke's to become a Lupus UK Centre of Excellence, recognising the unit's commitment to providing high quality care for patients with SLE.
Dr Jordan's interest in pregnancy management in SLE began during her time at St Thomas' and continued while in Cambridge where she worked collaboratively with the departments of Nephology and Obstetrics in providing combined care clinics for pregnant patients with complex autoimmune rheumatologic conditions.

Professor Mike Putman
Assistant Professor of Medicine
Medical College of Wisconsin

Mike Putman is an Assistant Professor of Medicine at the Medical College of Wisconsin, where he is the Medical Director of the Vasculitis Program and maintains an active practice in general rheumatology. He is involved in education and currently serves as the Associate Program Director for Rheumatology and Associate Program Director for Internal Medicine. His research interests include clinical trials in vasculitis and myositis, "big data" epidemiology, and meta-research. He is also an Associate Editor of the journal Rheumatology and hosts the Evidence Based Rheumatology podcast.

Professor Gaye Cunnane
Consultant Rheumatologist
St James's Hospital, Dublin

Gaye Cunnane, PhD, MB, FRCPI, is a Professor of Rheumatology at Trinity College Dublin and a Consultant Rheumatologist at St James's Hospital. She is also the Director of Health and Wellbeing at the Royal College of Physicians of Ireland. Previous roles have included National Specialty Director for Rheumatology, Regional Programme Director for Basic Specialist Training, Director of Post-graduate Education at St James's Hospital and she is a past-President of the Irish Society for Rheumatology.

Dr Brenda Griffin
Consultant Nephrologist at St James's Hospital and Tallaght University Hospital and is a clinical senior lecturer at Trinity College Dublin.

Training: Dr Griffin trained in multiple Irish and UK centres (GN fellowship in Imperial College London) and gained specialist registration in 2012. Her experience spans the full spectrum of clinical nephrology. Dr Griffin worked as locum consultant nephrologist in Cork University Hospital and the Queen Elizabeth Hospital, Birmingham before taking up her current post in 2016.
Training in the cosmopolitan and multicultural cities of London and Birmingham gave Dr Griffin first-hand experience of the care needs of a diverse patient population. Both the renal department and St James's Hospital aim to promote health and minimise disadvantage in at risk groups.
Dr Griffin first worked in St James's Hospital when she was completing her medical senior house officer (SHO) training in the Federation Hospitals. She was delighted to return as consultant nephrologist.
Education: Dr Griffin graduated from the Royal College of Surgeons in Ireland in 1997 and was awarded an MSc from Trinity College Dublin in 2006 and a PhD from University College Dublin in 2013 for independent experimental research in the area of molecular medicine.
Diabetic kidney disease was the subject of Dr Griffin's PhD thesis.
Special Interests: Chronic kidney disease
Vascular access - Clinical Lead in Vascular Access in the Dublin Midlands Group
Nephrology in haematology patients especially sickle cell disease

Prof John Pauling
Consultant Rheumatologist
North Bristol NHS Trust & University of Bristol

John is consultant rheumatologist at North Bristol NHS Trust and Honorary Senior Lecturer at the University of Bristol Medical School. His major research interest relates to peripheral vascular outcomes in Raynaud's phenomenon and systemic sclerosis. John leads the international Scleroderma Clinical Trials Consortium Vascular Working Group to devise better methods for assessing peripheral vascular complications in SSc. He is co-chair of the OMERACT Systemic Sclerosis Digital Vascular Special Interest Group. He is a member of the EULAR Microcirculation Study Group. He is an active member of the UK Scleroderma Study group and sits on the executive committee of the Scleroderma Clinical Trials Consortium.

ISR Board members

Professor Geraldine McCarthy
President
Consultant Rheumatologist
Mater Misericordiae University Hospital Dublin and Full Clinical Professor of Medicine University College Dublin

Geraldine McCarthy graduated in Medicine from NUI. She received her Fellowship in Rheumatology at the Medical College of Wisconsin. Her research has focused on the biological effects of calcium-containing crystals in degenerative joint disease as well as in atherosclerosis and breast cancer. Promoted to Associate Professor of Medicine at the Medical College of Wisconsin in 1996 where she remained until her return to Dublin.
Prof McCarthy was appointed Consultant in Rheumatology at the MMUH and Cappagh National Orthopedic Hospital Dublin in 1999 where she continues to run a busy clinical practice. She teaches as part of the University College Dublin Faculty of Medicine where she was the first clinician to be appointed Full Clinical Professor of Medicine through the Clinical Pathways in 2009.
Geraldine has current international collaborations in the UK, USA, Europe, Australia, New Zealand and Canada, particularly in relation to calcium crystal deposition diseases as well as gout. She continues her involvement in bench research related to the pathogenesis of basic calcium phosphate crystal–induced joint disease and participates in and contributes to numerous international collaborations related to gout. Other research interests include platelet activation in inflammatory arthritis and its role in enhanced cardiovascular risk. She also participates in collaborative studies of the pathogenesis of giant cell arteritis and HIV-associated bone pathology.
Author of over 130 publications, including original manuscripts, editorials, reviews and book chapters and has spoken at many national and international meetings. She has been winner of several research and teaching awards and has mentored many medicine and science graduates in clinical practice and in research.

Dr Claire Sheehy
Honorary Secretary
Consultant Rheumatologist
University Hospital Waterford

Dr Claire Sheehy is a Consultant Rheumatologist in University Hospital Waterford. A graduate of Trinity College Dublin, she completed the higher specialist training in rheumatology and general medicine, and was awarded an MD for work exploring the role of anti TNF therapy in early rheumatoid arthritis. She undertook a fellowship in connective tissue disease and vasculitis between Norfolk and Norwich University Hospital, and Addenbrookes Hospital. She took up her post in 2012; her current clinical interests include early inflammatory arthritis and connective tissue disease.

Dr Shawn Chavrimootoo
Honorary Treasurer
Consultant Rheumatologist,
Our Lady's Hospital,
Navan, Co Meath.

Shawn Chavrimootoo is a Consultant Rheumatologist at Our Lady's Hospital, Navan, Co. Meath. He graduated in Medicine from RCSI, Dublin in 2002 and developed an interest in Rheumatology during his Senior House Officer years in Connolly Hospital, Blanchardstown. Following this, he completed higher specialist training in Cork University Hospital, Kerry General Hospital, Connolly Hospital and St Vincent's University Hospital in Dublin. He was appointed to his Consultant Rheumatologist post in 2013 when he joined Dr Ramakrishnan at Our Lady's Hospital, Navan, from where they currently provide a regional Rheumatology service for the North East of Ireland. His clinical interests include osteoporosis as well as gout, inflammatory arthritis, spondyloarthritis, connective tissue disease and vasculitis.

Dr Nicola Ambrose
Consultant Rheumatologist,
Blackrock Clinic, Co Dublin

Dr Nicola Ambrose is a graduate of Trinity College Dublin. She completed her specialist training in rheumatology and general internal medicine in Ireland, before obtaining an Arthritis Research UK (ARUK) fellowship to undertake a PhD at Imperial College London, studying inflammation in Behçet's Syndrome. She then obtained a Richard Steeven Fellowship from the HSE to undertake a Clinical Fellowship at the ARUK Adolescent Rheumatology Centre of Excellence at University College London Hospital (UCLH). She stayed at UCLH as an Adolescent and Adult consultant rheumatologist, and was the Clinical Lead for Adolescent Rheumatology. Special interests: Adolescent and Young Adult Rheumatology including JIA; Behçet's Syndrome; SLE and dermatomyositis; Gout Osteoporosis and fracture secondary prevention; Inflammatory arthritis. She has published 23 peer review papers as well as 6 book chapters.

Dr Elizabeth Ball
Consultant Rheumatologist
Musgrave Park Hospital/
Belfast City Hospital

Dr Liz Ball is a graduate of Queen's University Belfast and was appointed as a Consultant Rheumatologist at Musgrave Park Hospital/ Belfast City Hospital in 2014. She is also an Honorary Lecturer at Queen's University Belfast. She has a special interest in autoimmune disease and lupus and was awarded an MD entitled 'A study of hand arthritis in Systemic Lupus Erythematosus from a Clinical, Imaging and Cytokine Perspective' from Queen's University in 2013. She is involved in postgraduate medical education and holds a Training Programme Director role within the Northern Ireland Deanery and is currently completing a Masters in Clinical Education. She is a musculoskeletal ultrasound tutor and regularly teaches regionally and nationally.

Photo Gallery

Dr Ashley Elliott - Winner Young Investigator Award

Dr Gerry Coghlan - Speaker

Prof Doug Veale, Prof Ursula Fearson, Dr Ronan Kavanagh, Prof Sandy Fraser and Prof Suzanne Donnelly

Photo Gallery

Dr Sarah Quidwai and Dr Salamet Ali

Dr Mark Leith and Dr Ashley Elliott

Photo Gallery

Prof Ian Bruce - Speaker

Prof Gaye Cunnane - Speaker

Dr Jeff Lee, Prof Ursula Fearon and Prof Doug Veale

Dr Andrew Cairns
Consultant Rheumatologist,
Musgrave Park Hospital, Belfast

Dr Andrew Cairns graduated in Medicine from Queen's University Belfast in 1995. He completed specialist training in Belfast and also at the Rheumatic Diseases Unit in Edinburgh. He was awarded an MD by thesis entitled "Leucocyte surface receptor expression of relevance to apoptotic cell clearance in systemic lupus erythematosus" from Queen's University Belfast in 2001, and an MSc in Sport and Exercise Medicine from the University of Ulster in 2008. He is a Consultant Rheumatologist at Musgrave Park Hospital Belfast where he was appointed in 2004, and also provides rheumatology clinics at Belfast City Hospital. He is a Fellow of the Royal Colleges of Physicians of Ireland, London and Edinburgh. He has published in a wide range of rheumatic diseases and is an enthusiastic proponent of musculoskeletal ultrasound.

Dr Michele Doran
Consultant Rheumatologist and General Physician, St James's Hospital Dublin

Dr. Michele Doran has been working as a Consultant Rheumatologist and General Physician at St. James's Hospital, Dublin since 2003. She graduated in Medicine from UCD in 1993, and completed her clinical training in General medicine and Rheumatology in Dublin and Bath, UK. She undertook a 2 year Research Fellowship at Mayo Clinic, Rochester, USA, where she completed an MD degree with research relating to the Epidemiology of Rheumatoid Arthritis. During this time she completed a Master's Degree in Biomedical Sciences, Clinical Research, in the Mayo Clinic Graduate School. She was involved with the establishment of and is on the steering committee for the Rheumatoid Arthritis Biologics Registry of Ireland (RABRI).

Professor Ursula Fearon
Head of Molecular Rheumatology,
School of Medicine,
Trinity Biomedical Sciences Institute,
Trinity College Dublin.

Professor Ursula Fearon is head of Molecular Rheumatology, School of Medicine, Trinity Biomedical Sciences Institute, Trinity College Dublin. Professor Fearon's research is a bench-to-beside translational approach, focusing on understanding the underlying mechanisms that drive disease pathogenesis; her team specifically examine components of joint inflammation at a cellular and molecular level to dissect out the signalling and gene pathways that are involved in the pathogenesis of inflammatory arthritis and rheumatic diseases. She has established strong collaborative research networks across Europe, USA and Singapore. Professor Fearon, has been awarded significant research funding from Arthritis-Ireland, Health Research Board, Science Foundation Ireland, IRCSET, European-ASPIRE, JU Innovative Medicines Initiative (IMI) and Maeve Binchy Funding for Arthritis Research, in addition to industry collaborative partnerships. She has published extensively in high impact peer-reviewed journals, and her research has been awarded several National/International awards.

Professor David Kane
National Lead for Rheumatology
HSE Clinical Programme
Consultant Rheumatologist,
Tallaght University Hospital, Dublin

Prof David Kane attended medical school at Trinity College, Dublin, Ireland and was conferred MB BCh BAO BA in 1991, PhD in 2002 and FRCPI in 2006. He has trained in rheumatology with Prof. Barry Bresnihan and Prof. Oliver FitzGerald at St. Vincent's University Hospital, Dublin, Ireland and with Prof Roger Sturrock, Prof Iain McInnes and Dr Peter Balint at Glasgow Royal Infirmary, Glasgow, United Kingdom. He was appointed as Senior Lecturer in Rheumatology at the University of Newcastle (2003-2005) and is currently working as Consultant Rheumatologist at the Adelaide and Meath Hospital and Clinical Professor in Rheumatology at Trinity College Dublin. His special interests are musculoskeletal ultrasound, spondyloarthopathy and synovial inflammation. He is a member of the European Working Party on Musculoskeletal Ultrasound and the OMERACT special interest group on musculoskeletal ultrasound, previous organiser of the BSR Musculoskeletal Ultrasound course and is Faculty member of the EULAR Musculoskeletal ultrasound course. He has served as a Board member of the Irish Osteoporosis Society, as President and Treasurer of the Irish Society for Rheumatology and is currently a Board member of Arthritis Ireland.

Dr Emma Jane MacDermott
Consultant Paediatric Rheumatologist,
CHI Crumlin

Emma Jane MacDermott, is a Consultant Paediatric Rheumatologist in CHI Crumlin where she joined the team in 2012 and has helped oversee the ongoing growth and development of the paediatric rheumatology department into a dynamic national service, now including a growing research and education component. With a special interest in education she enjoys working with patients, parents and medical providers to raise the profile and understanding of rheumatologic disease. She works with the national advocacy groups continuing to raise the profile for Irish paediatric rheumatology patients. Areas of interest include Juvenile arthritis, Paediatric Lupus and autoinflammatory disease.
A graduate of University of Dublin, Trinity College Medical School she pursued her post graduate training in paediatrics, becoming a member of the Royal College of Physicians in 2001. She subsequently moved to New York, where she completed a fellowship in Paediatric Rheumatology, from Weill Cornell Medical School, working at Hospital for Special Surgery and the Cornell Campus of New York Presbyterian Hospital as Assistant Attending in Paediatric Rheumatology at Hospital for Special Surgery and Assistant Professor of Paediatrics at Weill Cornell Medical School until her return to Ireland in 2012.
Emma is a member of the Royal College of Physicians of Ireland, the American College of Rheumatology, the Irish Rheumatology Society, the British Society of Adolescent and Pediatric rheumatology.

Photo Gallery

Dr Shawn Chavrimootoo and Dr Liz Ball

Dr Mary Canavan

Dr Maurice Barry and Prof Eamonn Molloy

Dr Wan Lin Ng
SpR Rep on ISR Board

Dr Wan Lin Ng is a medical graduate from the Royal College of Surgeons in Ireland (RCSI). She has completed her basic specialist training in Ireland and is currently in the higher specialist training programme in Rheumatology. She is a recipient of the StAR MD scholarship from RCSI and the ISR Rheumatology Patient Initiative Fund. With a keen passion in teaching and education, Dr Ng was previously an affiliate tutor with University of Limerick and RCSI. Her clinical interests include connective tissue disease related interstitial lung disease and musculoskeletal ultrasound.

Professor Barry O'Shea
Consultant Rheumatologist,
St James's Hospital, Dublin

Barry O'Shea is a Consultant Rheumatologist in St James's Hospital, and a Clinical Associate Professor in the School of Medicine in Trinity College Dublin. He took up his position in St James's in 2010. During his specialist training in Rheumatology he worked in St Vincent's Hospital, Waterford Regional Hospital, St James's Hospital and the Mater Hospital. He was the inaugural recipient of the Irish Society for Rheumatology / Wyeth Travelling Fellowship award. This facilitated the completion of his training in the University of Toronto and Toronto Western Hospital, Canada. He went on to undertake a Research Fellowship in Toronto with Dr Robert Inman with a focus on patients with Ankylosing Spondylitis and Psoriatic Arthritis. He has presented at the American College of Rheumatology Annual Meeting on this work. He is an active member of ASAS (Assessment of SpondyloArthritis international Society), an international group of experts in the field of Ankylosing Spondylitis. He is a co-founder and principal investigator of ASRI – the Ankylosing Spondylitis Registry of Ireland, a national database of patients with AS from across Ireland. He is the Clinical Lead for Rheumatology in St James's Hospital. In 2022 he was appointed the National Speciality Director (NSD) for Rheumatology training in the Royal College of Physicians of Ireland.

Dr John Ryan
Consultant Rheumatologist,
Cork University Hospital, Cork

Dr John Ryan is a graduate of the Royal College of Surgeons in Ireland, he completed his higher medical training in rheumatology and general internal medicine in Ireland. He undertook a fellowship at the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) in Bethesda, Maryland. During this time he undertook translational research into disordered innate immunity manifesting as recurrent fever syndromes. He joined Dr Sinead Harney in the Rheumatology service at Cork University Hospital in 2010. The Rheumatology department has since expanded to include Dr Grainne Murphy. In July 2017 he took up the post of National Specialty Director for Rheumatology.

Dr Bryan Whelan
Consultant Rheumatologist
Our Lady's Hospital, Manorhmailton,
Co Leitrim

Dr Bryan Whelan is a Consultant Rheumatologist in Our Lady's Hospital in Manorhmailton, Co Leitrim and an Honourary Senior Lecturer in Medicine in NUIG. He qualified from UCD in 2000 and completed BST in the Mater Hospital in Dublin. He completed SpR training in Rheumatology in CUH, the Mater Hospital and University College London. He has an MD and Masters Sports and Exercise Medicine from UCC and an MSc in Epidemiology from the London School of Hygiene and Tropical Medicine. He is currently a board member of Arthritis Ireland, the SUH Research and Education Foundation, a member of the Academic Committee of the FSEM and a member of the Advisory Committee for Human Medicines Clinical Trials Subcommittee of the HPRA. His current research interests include muscle disease, exercise in rheumatology and osteoarthritis.

Dr Maria Wray
Consultant Rheumatologist
Antrim Hospital, Northern Ireland.

Dr Maria Wray is a consultant rheumatologist in Antrim Hospital, Northern Ireland. She graduated from Queens University Belfast in 2000 and began rheumatology training in Northern Ireland. She then undertook long term specialty doctor roles in rheumatology firstly in Musgrave Park Hospital and then the South East Health Trust where she developed particular expertise in musculoskeletal and vascular ultrasound. She was awarded specialist registration in rheumatology and joined the team in Antrim hospital in the Northern Trust as a consultant in 2018. Her specialist interests include PMR/GCA and "fast track" diagnostic imaging.

Photo Gallery

Ms Caitriona Stafford, Ms Irene Joseph and Ms Linzi Martin

Audience View

Photo Gallery

Mr Neil Herbert, Lilly; Dr Clare Matthews, Dr Elizabeth Ball, Dr Vivienne McGoldrick, Dr Nicola Maiden.

Mr David McCan, AbbVie, Dr Sharon Cowley and Mr David Lynch, AbbVie

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Dr J. Lee 2006 - 2008
Craigavon

Dr P. O'Connell 2004 - 2006
Dublin

Prof O. FitzGerald 2002 - 2004
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Mater Misericordiae University Hospital Dublin
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Dr Bryan Whelan
Consultant Rheumatologist
Our Lady's Hospital, Manorhmailton, Co Leitrim

Dr Maria Wray
Consultant Rheumatologist
Antrim Hospital, Northern Ireland

Photo Gallery

Dr Maura McCarron, Dr Maria Wray, Dr Rory McKane, Dr Aubrey Bell

Mrs Sandra Taggart, Dr Gary Wright, Mrs Pippa Cullum

Message from Michael Dineen

Dear Friends,

As we arrive in Kilkenny for our Spring Meeting, to a venue that has been good for ISR in the past, I sense a very positive feeling regarding our Society. We will continue the 'later dates' for our Spring Meeting and we have again organised this meeting over two days. It is easier to travel in the summer weather than in March or April.

Our thanks to the academic team from St James's Hospital for this programme and we look forward to the contribution of our colleagues in St Vincent's for our Autumn Meeting at Fitzpatrick's Castle Hotel, Killiney on 21 and 22 September.

With five weeks still to go to our Spring meeting and have almost 100 rooms booked and approx. 24 pharma stands. This gives an idea of the demand for places. Although the overall cost of organising meetings is on the rise we are still endeavouring to maintain registration fees for delegates at previous levels - a policy that the Board is anxious to continue.

Our relationship with our partners in Industry is excellent. While we have lost one or two of our friendly companies, there are always new ones emerging. This obviously bodes well for the future of the ISR conferences and any subsidiary sponsorship by the industry. The Board of ISR has recently taken over the responsibility for the 'biologic registries' which will be important for the long term success and funding of the projects.

We continue to offer our trainees opportunities to improve their skill sets. The Young Investigator Award is highly regarded and much sought after. So are the medals for the best clinical and scientific orals.

Last year we reintroduced the Bernard Connor Medal in order to create interest among BST trainees in medicine. The intention is to promote interest in the field of Rheumatology and to support engagement with the Irish Society for Rheumatology. The interest to date has been less than what was anticipated.

This years Bernard Connor Medal winner will receive €500 and a silver inscribed medal. Let us all put our shoulders to the wheel and encourage aspiring clinicians and researchers to participate and claim this worthy trophy to add to their CVs.

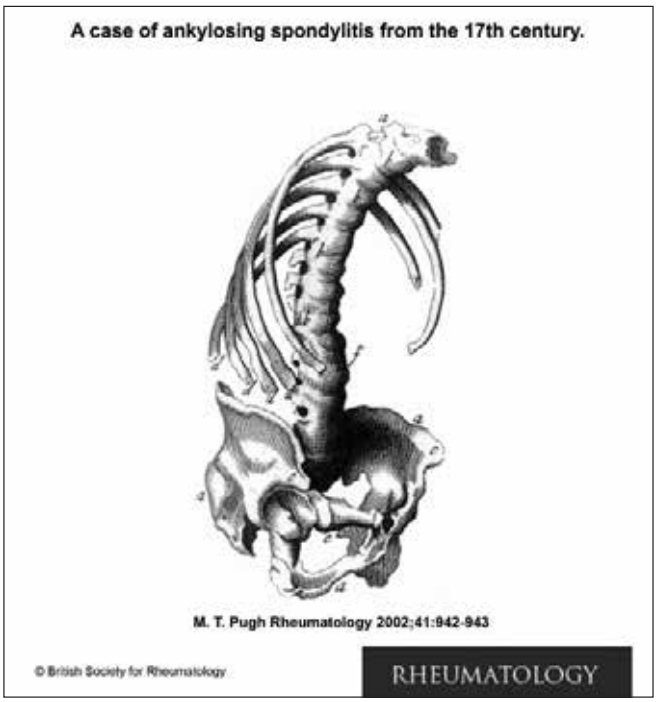
We have just completed a major trawl of ISR Membership. While we have attracted a large number of new members sadly quite a few have not recently renewed their subscriptions. It is our collective responsibility to raise awareness of rheumatic disease associated research and we must support the profession in delivering a high-quality care for patients in addition to lobbying centrally to influence government policy.

This can best be achieved by having one strong voice promoting ISR. To this end I would ask you to ensure that you are a fully registered member of the society.

Finally, I must pay tribute to the lady who not only brought us through the pandemic but who has guided us for the past four years, a feat never previously achieved. To Geraldine McCarthy and the Board who supported her, well done – **You have certainly done some service.**

Michael Dineen
Chief Executive ISR

ISR Bernard Connor Medal



The Irish Society for Rheumatology (ISR) has established the Bernard Connor Medal to promote interest in the field of rheumatology among BST trainees in medicine, *and to support engagement with the Irish Society for Rheumatology.*

The award is open to BST trainees in medicine who *fulfil the eligibility criteria below*. In addition to receiving the Connor Medal, the winner will be invited to attend the annual scientific meeting of the ISR to present their work to the membership, as a guest of the society. *Additionally, and at the discretion of the judging panel, up to two runners-up may be awarded full registration to attend the ISR annual scientific meeting.*

Bernard Connor

The Connor Medal is named in honour of Bernard Connor, an Irish physician who observed and described the characteristic skeletal and clinical features of ankylosing spondylitis in 1693, while a medical student in Paris. This award will be made annually on the basis of competitive submission.

Theme

The theme chosen by the ISR is a **Case Report with relevance to rheumatology.**
The submitted work should highlight your observations/insight in relation to rheumatology as a BST trainee.

Submission Only one submission per trainee will be accepted.

Case Report

The case report should be submitted in full form and present the details of an interesting case followed by a discussion on your observations of the key points of interest, and relevant literature review. These should be submitted in full [**max 800 words**, concluding with summary key message and references (up to 5 in total)].

Bernard Connor Medal contd...

Eligibility

- Applicants must be registered RCPI BST trainees by 1 July 2023.
- Applicants must submit completed entries to the ISR by the 11 August 2023.
- It is hoped that the re-launch of this competition in its new format will not only honor a great clinician but also draw the specialty of Rheumatology to the attention of BST trainees. The ultimate aim is to attract more trainees to HST in Rheumatology.

How to Apply

Application forms for the Connor Medal will be available for download from the ISR website www.isr.ie. These must be completed in full and returned together with your submission to info@isr.ie

Closing Date: 11 August 2023.

Judging Criteria

The Medal will be awarded according to the criteria below which will be applied to all submissions

- Relevance of the submitted work to rheumatology
- Originality and Merit of the work

The Board of the Irish Society for Rheumatology shall convene the judging panel. The decision of the panel shall be final and no correspondence on submissions or final outcome can be entered into.

**Dr Samantha Banford,
Winner of the 2022 Bernard Connor Medal**

Photo Gallery

Dr Alwin Sebastian, Dr Sumreen Sarafaz, Dr Taimoor Mounis, Dr Saleha Huma, Dr Aniah Farouk

Audience View - Dr John Paul Doran, Dr Eithne Murphy, Dr Maurice Barry and Dr Michele Doran

Photo Gallery

Dr Ashley Elliott, Dr Maria Wray, Dr Annmarie McShane and Dr Aidan O'Neill

Dr Duncan Rogers - Speaker

Mrs Taggart and Dr Allister Taggart

CLINICAL PRESENTATIONS

Abstract No.	Clinical Cases Presentation	Time
23S115	No. 1	11.00
23S120	No. 2	11.15
23S135	No. 3	11.30
23S137	No. 4	11.45

CLINICAL CASES

23S101 **CLINICAL CASE**

NXP2 dermatomyositis with bulbar weakness requiring mechanical ventilation and treatment with high dose corticosteroid, IVIG, cyclophosphamide and rituximab.

Author(s)
Dr. Brona Dinneen Dr. John Stack Prof. Geraldine McCarthy

Department(s)/Institutions
Rheumatology Department, Mater Misericordiae University Hospital Dublin

Introduction
A 28-year-old female presented with a 6-week history of rapid onset proximal muscle weakness, dysphagia and inflammatory rash (heliotropic rash, Gottren's papules and livedo reticularis). 3 weeks prior to developing symptoms she had tested positive for SARS-CoV-2.

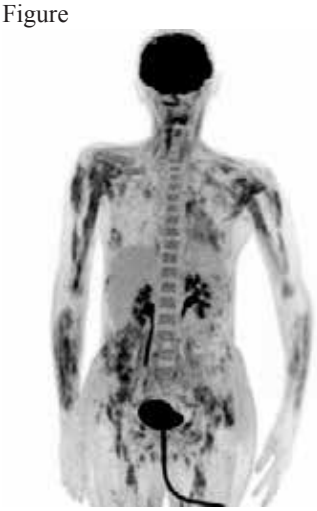
Aims/Background
On examination her MMT was 72/150. She had marked anasarca. Laboratory assessment showed CK of 9270 IU/L. Extended myositis panel revealed a positive NXP2 antibody. Full body MRI showed extensive muscle oedema in the adductor and flexor muscle groups of the shoulder and hip girdle and erector spiny muscles. Muscle biopsy and skin biopsies were both consistent with dermatomyositis. CT- PET showed evidence of intense muscle uptake particularly in the proximal muscles of the forearms without any findings suggestive of an underlying malignancy. Initial treatment consisted of high dose intravenous methylprednisolone followed by oral taper and a course of IVIG (2g/kg). She was initiated on mycophenolate 500mg twice daily.

Method
Her condition worsened and 2 weeks following her initial presentation she was requiring hoist to transfer, with worsening dysphagia. Gut malabsorption was suspected as she now had ascites, worsening anasarca and bilateral pleural effusions in the setting of low albumin (26g/L) and hyponatraemia (125 mmol/L). Due to deteriorating peak flow measurements at ward level, the patient was moved to the intensive treatment unit (ITU) where she was intubated for airway protection and commenced on total parenteral nutrition. Combination of IV hydrocortisone, cyclophosphamide, and rituximab was prescribed.

Results
Symptoms slowly improved with recovery of muscle function (MMT8=147/150), resolution of skin rash and dysphagia. CK peaked at 21525 IU/L before slowly returning to normal range. She remains well 6 months post discharge and has been switched to mycophenolate 500mg twice daily. Prednisone has been discontinued

Conclusions
In this report we present a case of COVID-19-related DM with multiple factors associated with poor prognosis. Previous case reports have described successful use of IVIG for steroid refractory DM with bulbar involvement and anasarca. To our knowledge this is the first case report of a DM patient with anasarca and bulbar involvement to be successfully treated with a combination of IVIG, high dose corticosteroid, cyclophosphamide, and rituximab.

Figure



23S103 **CLINICAL CASE**

Alemtuzumab – here today, not gone tomorrow.

Author(s)
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Department(s)/Institutions
Rheumatology Department, Mater Misericordiae University Hospital

Introduction
Autoimmune adverse effects from alemtuzumab may present years after receiving the drug.
Aims/Background
We present a patient who developed new polyarthropathy resistant to oral steroids after receiving alemtuzumab years ago.

Method
A 37-year-old female presented with polyarthritis affecting her wrists, knees and ankles on a background of relapsing-remitting multiple sclerosis (MS) which was well-controlled on ocrelizumab (anti-CD20). She had received 2 doses of alemtuzumab (anti-CD52) therapy 4 years prior to presentation. Initial impression was inflammatory arthritis. She was treated with oral prednisone, with nil improvement. She then developed fevers, pleuritic chest pain and ongoing arthritis severely limiting her mobility. Repeat bloods revealed white cell count (WCC) of 16.9x 10^9 (raised neutrophils), CRP 331 mg/L, ESR 105 mm/hr, Ferritin 609 ug/L. Autoimmune serology was negative. Synovial fluid aspirate of right knee showed high WCC. Fluid culture was sterile and was sent for 16s PCR. CT TAP was negative for any acute inflammatory process. As she did not respond to high dose oral steroids, we prescribed pulsed IV methylprednisolone with marked clinical improvement. Her condition immediately worsened when switched to oral steroids. As her symptoms resembled adult-onset Still's disease (fever, serositis, marked acute phase response, neutrophil leucocytosis, seronegative) and was requiring high doses of steroids to control symptoms we added tocilizumab (anti-IL6). This resulted in clinical improvement which ultimately allowed for steroid tapering and discharge home. Joint fluid PCR was subsequently positive for Ureaplasma Urealyticum.

Results
Her polyarthritis resolved with methylprednisolone and tocilizumab. She was also started on doxycycline to treat Ureaplasma Urealyticum infection. Our working diagnosis is that this patient developed a Stills-like reactive arthritis secondary to Ureaplasma Urealyticum

Photo Gallery

Prof Geraldine McCarthy, President ISR

Prof Michael Doherty - Speaker

Dr Gary Wright, Dr Andrew Cairns, Dr Alister Taggart,
Prof. Emilio Filippucci, Dr Eithne Murphy & Dr Carmel Silke

infection in the context of active B cell depleting therapy. The hyper-inflammatory immune response we suspect was a consequence of prior alemtuzumab therapy (Still's disease is a recognised adverse event associated with alemtuzumab). Alemtuzumab exerts its mechanism via rapid and sustained T cell depletion. Immune reconstitution favours the proliferation of chronically active memory T cells predisposing to autoimmune adverse events which tend to peak 3 years after receiving therapy.

Conclusions
Alemtuzumab can predispose/contribute to autoimmune adverse events which may present years after receiving therapy.

23S104 **CLINICAL CASE**

Two cases of Polyarteritis Nodosa presenting as Myositis

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Introduction
This report contains two cases that presented with signs consistent with idiopathic inflammatory myositis. Both cases had a unifying diagnosis of Polyarteritis Nodosa(PAN).PAN is a systemic necrotizing inflammation of blood vessels that typically affects small and medium sized arteries. This highlights the need for a broader differential for the aetiology of lower limb pain and the possibility of PAN to present with predominant muscle involvement.

Aims/Background
Included are two cases, A and B Case A is a 67 yr old male referred from his GP with bilateral lower limb pain for 6 weeks. He initially had been in a private hospital with a provisional diagnosis of polymyositis made. He was started on tapering steroids which had stopped 2 weeks prior to review. On exam he had bilateral thigh tenderness. Case B is a 66 yr old male with 8 week history of progressive bilateral lower limb pain on a background of T2DM who had received one week of steroids prior to review. On exam he had bilateral thigh tenderness with normal power.

Method
They both had raised inflammatory markers on admission with Case A CRP:62 ESR:103 and Case B CRP:266 ESR:66.An extended Myositis panel was done with all markers negative in both cases. Case A had a PET CT showing low-grade metabolic activity within the lower limb arteries and a muscle biopsy with no evidence of inflammatory myopathy.PET CT for Case B showed patchy increased tracer uptake in the musculature of the thighs and a muscle biopsy showed Vasculitis.

Results
Case A was discharged on Methotrexate 25mg and prednisolone 40mg and Case B discharged on prednisolone 60mg and methotrexate 25 mg. They were followed up in clinic after 2 months and both had evidence of clinical remission from PAN.

Conclusions
Both patients have PAN with very similar albeit under-recognised clinical presentations. They ultimately had two different confirmatory tests (PET and muscle biopsy),with the corresponding test being negative in the other patient. They were treated for PAN as per the ACR guidelines for remission induction therapy with Glucocorticoids with non- GTC immunosuppressive. PAN should be suspected in cases of diffuse myopathy, especially in the context of a systemic disease.

23S105 **CLINICAL CASE**

"I Can't Feel My Legs"

Author(s)
E. McCabe, B. Louis, J. Kinsella, L. O'Neill, L. Rooney

Department(s)/Institutions
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Introduction
A 54 year old lady presented to the Rheumatology clinic with bilateral lower limb sensory symptoms for one year. She had been attending since 2016 with an undifferentiated inflammatory arthritis and severe bilateral anterior uveitis on infliximab.

Aims/Background
She described bilateral lower limb numbness which had ascended to involve the trunk, "burning" pain in her right leg and unsteadiness. Power was 5/5 in both upper and lower limbs with reduced sensation in her left lower limb and increased on the right. Reflexes were normal. She had an ataxic gait.

Method
She was admitted to hospital for further work-up. Initial routine blood work was normal. ESR was 55 with a normal CRP 4.6. Viral screen was negative. ANA was 1:400, negative ENA, dsDNA, ANCA. Her MRI brain and whole spine showed extensive enhancing periventricular juxtacortical and infratentorial white matter lesions and enhancing T2 high signal lesions throughout the cervical and thoracic cord in keeping with active demyelinating lesions.

She was seen by Neurology who recommended IV methylprednisolone 1g od for three days and then prednisolone 40mg. There was an improvement in her sensory symptoms and mobility. CSF WCC was elevated at 16 with > 99% lymphocytes, no organism growth and negative CSF viral screen. CSF protein and glucose were normal. She had oligoclonal bands present in CSF and not serum. Aquaporin 4 antibodies were negative, however her anti-MOG antibodies were positive. A diagnosis of MOGAD (myelin oligodendrocyte glycoprotein antibody-associated disease) was made. She was started on Rituximab therapy in hospital.

Results
MOGAD is a demyelinating disease with a prevalence of 2.5 per 100,000. Distinguishing MOGAD from MS is important as some therapies for MS appear to be ineffective for MOGAD. Features that favour MOGAD are MOG-IgG antibody positivity, absence of CSF oligoclonal bands and resolution of T2 lesions over time. Acute flares are managed with steroids. Refractory cases may respond to IVIG or plasma exchange. RTX, MMF, AZA and IVIG have been used in relapsing disease.

Conclusions
Our patient's symptoms continued to improve following RTX. Repeat MRI imaging showed resolution of contrast enhancement of white matter and cord lesions.

Figure

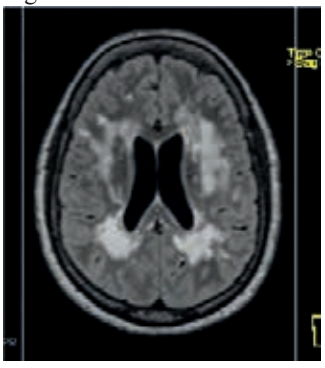
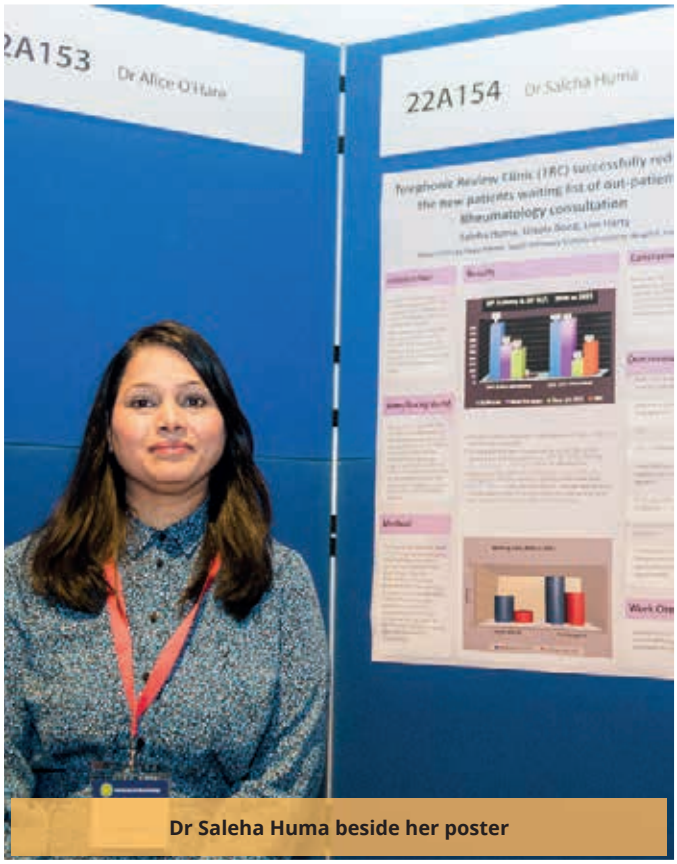


Photo Gallery

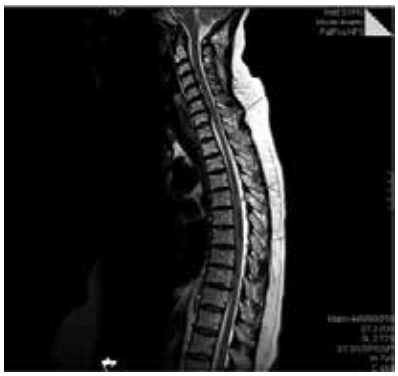


Dr Saleha Huma beside her poster

Prof. Emilio Filippucci - Speaker

Dr Mary Canavan Dr Viviana Marzaiola, Dr Leah Rooney

Figure



23S106 **CLINICAL CASE**

Atypical Polymyalgia Rheumatica in Young Adult

Author(s)
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Introduction
Polymyalgia Rheumatica (PMR) is a common inflammatory condition after 50 years. It typically presents with shoulder, neck and pelvic pain and stiffness associated with raised inflammatory markers.

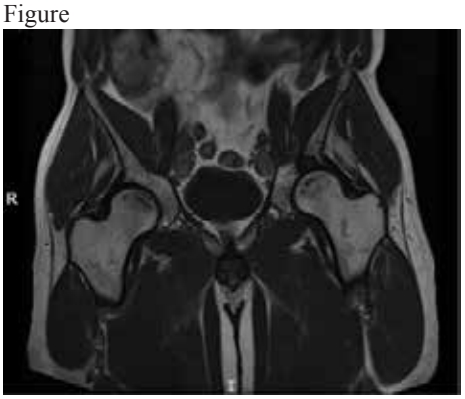
Aims/Background
We report a case of PMR in a young male with atypical clinical presentation

Method
40 year/male presented in Rheumatology OPC with three months history of back and pelvic pain with EMS for upto 40 minutes and marked fatigueability. CRP 19 and ESR 52. Initial assessment was inflammatory Axial Spondyloarthropathy
MRI spine and SIJ as discussed in radiology MDT didn't show inflammation in spine or SIJ. It showed bilateral Osteonecrosis of Femoral heads and ill-defined oedema in left acetabulum. A repeat MRI and CT pelvis showed sclerotic lesion in left Acetabulum. It was discussed in bone/soft tissue tumour MDT with conclusion of non-malignant lesion.
The pain progressed to involve both shoulders despite regular NSAIDs. The stiffness became generalised for up to 2 hours. Fatigue worsened. CRP was 19, 25 and 20 whereas ESR was 52 48 and 40 with an interval of a month each.
Multiple investigations including WCC,Hb,U&Es,LFTs,bone profile,RF,CCP, ANA,ENA,ANCA,SPEP, Myeloma screen,urine analysis,CXR and ECG were normal.
He went on to have PETCT suggestive of arthritis in both shoulders, Sternoclavicular joints, 1st Costochondral joint, left hip and posterior spinal ligament. The findings were discussed in Radiology MDT again with a conclusion of PMR

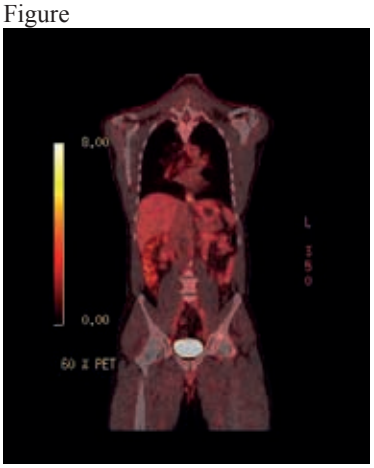
Results
Prednisolone 20mg was started. Symptoms improved within 3 days. CRP improved to 4 and ESR 18. They remained normal after two and three months. He is currently on tapering dose of Prednisolone without recurrence of symptoms and rise in inflammatory markers

Conclusions
Although PMR is commonly seen in over 50 years but it can present in young adults like in this patient. Pattern of joint involvement on PET CT and dramatic response to steroids confirmed the diagnosis.
Because of young age and atypical presentation rheumatologist required multiple investigations including PET CT to diagnose PMR that otherwise is diagnosed clinically with raised inflammatory markers.
Osteonecrosis of Femoral heads in the absence of any other positive investigation was thought most likely secondary to Inflammation that again is not very common presentation of PMR.

Figure



Figure



23S107 **CLINICAL CASE**

A case of minocycline induced anti-neutrophil cytoplasmic antibody (ANCA) vasculitis

Author(s)
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Introduction
A 16 year old boy presented with a three day history of progressive bilateral lower limb weakness. He had preceding upper respiratory tract symptoms treated with prednisolone and amoxicillin/clavulanic acid. He had a background of acne vulgaris for which he had been taking minocycline. He had no rashes or prior drug use.

Aims/Background
ANCA vasculitis is a heterogeneous group of multi-system autoimmune diseases which affect predominantly small vessels. Vasculitis associated with minocycline is rare but well described. We present a potential case of minocycline associated ANCA vasculitis

Method
Lower limb power was globally reduced 3/5 and tone reduced. Gait was broad-based and ataxic. Lower limbs reflexes were reduced with downgoing plantars. Upper limbs and cranial nerves were intact. He subsequently developed a new oxygen requirement, upper limb weakness with power 2/5 and dysphagia.

Results
Bloods showed a microcytic anaemia and deranged liver function tests in an intrahepatic pattern. Initial treatment with IVIG for potential Guillain Barre Syndrome was performed with ICU admission with minimal improvement. Subsequently, cytoplasmic ANCA and PR3 were positive. Given the lack of response to IVIG and positive antibodies, he was treated IV methylprednisolone and rituximab for a potential ANCA vasculitis. Tapering oral steroids commenced with vitamin D and PCP prophylaxis. Liver biopsy showed chronic inflammation which was felt likely drug induced secondary to minocycline. Nerve conduction studies showed a mixed motor and sensory polyneuropathy. The patient made a gradual but full improvement over 6 weeks.

Conclusions
It is important to consider minocycline as a rare trigger for ANCA vasculitis.

23S110 **CLINICAL CASE**

Giant Cell Arteritis of the Prostate

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Introduction
A 67-year-old gentleman was referred to our institution after radical prostatectomy for Gleason score 9 prostate adenocarcinoma. Histology from his prostate showed necrotising vasculitis with focal giant cells in the medium and large vessels adjacent to and within the prostate parenchyma.

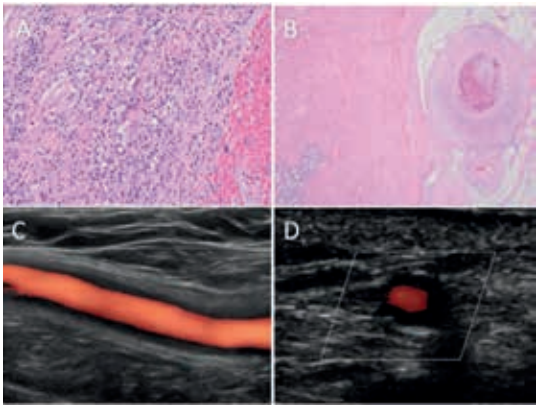
Aims/Background
He described abrupt onset of bilateral shoulder and hip girdle pain and stiffness 14 months prior to his prostate biopsy in keeping with polymyalgia rheumatica (PMR). He attended his general practitioner and received a non-steroidal anti-inflammatory drug which did not control his symptoms and subsequently received bilateral shoulder joint injections on two separate occasions. At the time of the initial review by Rheumatology, he still described early morning stiffness. He also recalled intermittent, headaches, affecting the bilateral temporal region and occipital region. He had no scalp tenderness, jaw claudication, or visual disturbance. He reported some night sweats at the outset of his symptoms, but these subsided in the initial few weeks and did not recur. He also described ongoing fatigue.

Method
On examination, the patient had bilateral palpable non-tender temporal pulses. He had mildly reduced range of motion to the right shoulder in flexion and abduction. Axillary artery bruits were present bilaterally.

Results
Erythrocyte sedimentation rate was 15mm/hr. Temporal and axillary artery ultrasound showed evidence of vasculitis with halo sign in 5/6 branches of the temporal arteries assessed with associated intima-media thickening and increased axillary artery thickness bilaterally with a positive slope sign and halo sign. The patient was treated with 40mg of prednisolone. Given his prostate cancer was considered cured after surgical resection, no further treatment was required. He was commenced on tocilizumab 162mg weekly. His symptoms of GCA/ PMR resolved and his prostate cancer remains in remission.

Conclusions
Vasculitis of the prostate is uncommon. There have been some reported cases in conjunction with anca-associated vasculitis manifesting in the prostate vessels. There have been two previously reported cases of isolated prostate vasculitis with identification of giant cells on histology. Neither of these cases had features of systemic vasculitis.

Figure



23S112 **CLINICAL CASE**

A Case of Scleroderma Overlap Syndrome

Author(s)
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Department(s)/Institutions
Department of Rheumatology, Beaumont Hospital, Dublin

Introduction
Scleroderma overlap syndrome is a rare disease that presents with two or more connective tissue disorders, most commonly dermatomyositis in conjunction with scleroderma. It is associated with a wide variety of symptoms and can be difficult to diagnose. It is associated with specific antibodies.

Aims/Background
We report on the successful management of a complex patient presenting to Beaumont Hospital with dermatomyositis and scleroderma overlap syndrome causing slurred speech, weight loss and the concern regarding malignancy.

Method
A 78-year-old lady, with a history of ovarian malignancy successfully treated 5 years previously, presented with slurred speech and deterioration in her swallow. She reported profound fatigue, neck pain and difficulty mobilising. Eighteen months previously, she had a rash on her hands and on admission, sclerodactyly without features of Raynauds . She had previously attended another hospital where her statin was held for a presumptive diagnosis of statin induced myopathy, with no improvement.

Investigations
CK >3500.
An EMG showed evidence of myopathy/myositis.
Myositis panel results showed Mi-2b weakly positive, Ku positive, PM-Scl-75 positive, consistent with myositis and systemic sclerosis overlap . Tif1-gamma was negative.
A PET-CT ruled out recurrence of malignancy, and demonstrated diffuse uptake in her muscles, in keeping with dermatomyositis.

Photo Gallery

Dr Salamet Ali and Dr Kamil Khan

Dr Jo Kitchen and Dr Eithne Murphy

Dr Mark Leith, Dr Julie-Ann Henderson, Dr Jonathan McKnight, Dr David McCormick, Dr Ashley Elliott, Dr Kerry Aston

Photo Gallery

Dr Miriam O'Sullivan, Dr Len Harty and Dr Orla Ni Mhuircheartaigh

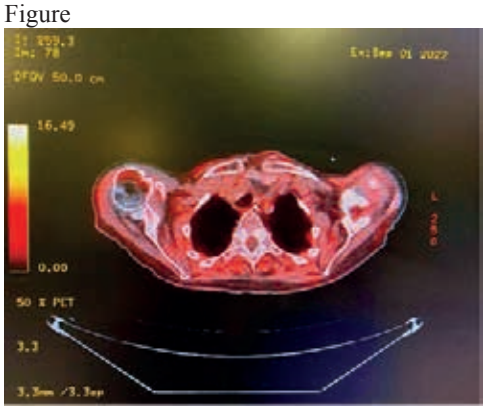
ISR Novartis Sp R Training Day Oct 2022 at Fitzpatrick Castle Hotel, Killiney

Management
IV Immunoglobulin was started for five days with pulsed methylprednisolone.
She was subsequently commenced on mycophenolate mofetil. Due to deteriorating swallow, had a NG tube in during her admission which was changed to a PEG due to inadequate calorie intake. Following a further course of IVIg, a tapering dose of prednisolone and mycophenolate mofetil were continued on discharge home.

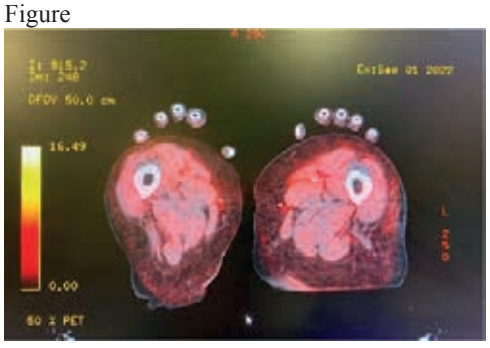
Results
Scleroderma overlap syndrome was successfully managed with IVIg, methylprednisolone followed by mycophenolate mofetil and a slow tapering dose of prednisolone. Her CK is now normal, PEG has been removed and the patient is significantly improved.

Conclusions
This case highlights that scleroderma overlap syndrome is a rare condition, which can be difficult to diagnose. Early recognition of this through a detailed history and examination, involving other specialties and focused investigations, including a myositis panel on bloods, will all assist in confirming a diagnosis and guiding a focused treatment plan.

Figure



Figure



23S113 **CLINICAL CASE**

"The Eyes Have It!!!...A Sight to Behold!" – The Ocular Signs & Complications of Vascular Ehlers-Danlos syndrome

Author(s)
Robert Harrington Laura Durcan

Department(s)/Institutions
Beaumont Rheumatology Department

Introduction
Ms. A is a 34-year-old woman who was originally investigated for a collagen disorder in her 20s after severe bleeding and poor wound healing post varicose vein stripping. She looked distinctly different to the rest of her family with large eyes, narrow nose and thin lips and a propensity to easy bruising. She was diagnosed with vascular Ehlers Danlos (vEDS) after specialist review and genetic testing ex domo.

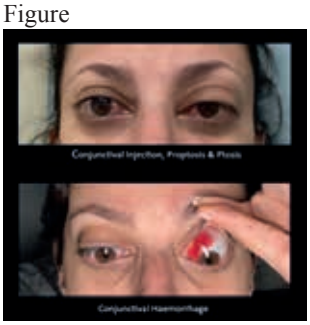
Aims/Background
In recent months, Ms. A began to develop a whooshing sound and diplopia with exertional Valsalva while weight training. She presented with progressive swelling, pain and protuberance of the left eye. Examination revealed chemosis, marked conjunctival injection and pulsating proptosis of the left globe with evident mild bilateral 6th nerve palsies. Visual acuity was preserved at 6/5 with normal optic nerve and disc.

Method
Neuroimaging with CT intracranial angiogram and MRA demonstrated bilateral carotico-cavernous fistulae with engorgement of both superior ophthalmic veins, left greater than right. Ms. A proceeded to endovascular embolization of the left carotid cavernous fistula but developed a complete left 6th nerve palsy and a complete left 3rd nerve palsy in the post-operative period. Treatment with high dose dexamethasone was commenced with partial recovery to date though full resolution of symptoms remains uncertain.

Results
vEDS displays an autosomal dominant inheritance pattern though 50% of cases are de novo mutations affecting the gene for type 3 collagen. vEDS patients do not display increased skin laxity and hypermobility evident only in the fingers. Approximately 30% have the classical EDS facies of bulging eyes, pinched nose, thin lips and hollowed cheeks. In young adults, vEDS may present as otherwise unexplained spontaneous bowel perforations and pneumothoraces. Pregnancy confers a 5% mortality risk due to uterine rupture. Ultimately 80% of cases present by age 40 with a vascular event.

Conclusions
Carotid cavernous fistula is an abnormal arteriovenous communication between the carotid artery or its branches and the cavernous sinus. The dramatic clinical findings in this case of vEDS are explained by the pathologic high-pressure flow of arterial blood into the cavernous sinus impeding venous return and engorging and arterialising the superior ophthalmic vein.

Figure



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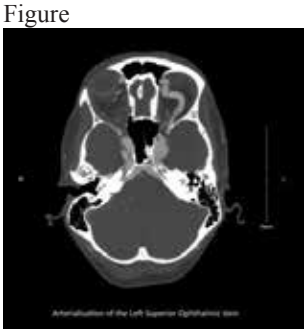

23S114 CLINICAL CASE

Hypogammaglobulinemia and prolonged Covid pneumonia in a patient on maintenance Rituximab

Author(s)
Samreen Tariq Muhammad Jehangir Malaz Ahmed Catriona Stafford Martina Carolan Shawn Chavrimootoo Omer Hussein

Department(s)/Institutions
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Introduction
case report
Aims/Background
Rituximab,a B-cell depleting agent indicated for the treatment of many rheumatological conditions,is associated with dampened humoral response and secondary hypogammaglobulinemia. Recent studies have suggested that immunocompromised patients who develop COVID-19 can demonstrate prolonged viral shedding.We report a case of an immunocompromised patient on maintenance rituximab with new onset recurrent pneumonia after Covid-19 infection and prolonged viral shedding for up to 262 days from initial infection.

Method
A 56 y/o gentleman,with a history of Rheumatoid Arthritis(RA),initially presented to the hospital in January 2022 with one week of cough and shortness of breath. An oropharyngeal swab was positive for SARS-Cov-2, but he did not require hospitalisation. His RA was treated with maintenance Rituximab every 6 months, since 2016 and he received his last infusion one month after the COVID infection.His serum immunoglobulins (IgG's) were normal at that stage and four months afterwards
From March 2022 to August 2022, he was hospitalised four times with waxing and waning appearing pneumonia requiring treatment with antibiotics and steroids.But his respiratory symptoms returned within a few days upon discontinuing steroids and antibiotics. Computed tomography (CT) imaging demonstrated recurrent ground glass opacities with bilateral interstitial pneumonia,compatible with COVID-19.He continued to have COVID detected in his bronchoalveolar lavage. Due to relapsing hypoxemia and radiographic progression,a diagnosis of organizing pneumonia as sequelae of COVID-19 was considered.High dose oral steroid therapy was started but,within 5 days of discharge, he represented to the hospital with respiratory failure and required intubation and ventilation.Subsequent medical therapy included steroids,two courses Remdesivir and Tocilizumab.He also received Intravenous immunoglobulin as his IgGs were now found to be low.

Results
He was extubated after 15 days and demonstrated a gradual clinical recovery with progressive reduction in oxygen therapy and eventual negative swab by RT-PCR.He in currently on weekly Tocilizumab for his RA and receives monthly IVIG replacement as recommended by Immunology.

Conclusions
This is a unique case as the patient had been on rituximab for 6 years without any major infections and then developed hypogammaglobulinemia following COVID-19.In high risk patients with symptomatic hypogammaglobulinemia,intravenous immunoglobulin should be considered prior to and during rituximab use.

23S115 CLINICAL CASE

"Go With the Flow or No?!" – When Digital Ulceration is Not Strictly Vascular: An Unusual Case of Acrodermatitis Continua of Hallopeau?

Author(s)
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Department(s)/Institutions
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Introduction
Mrs. R is a 77-year-old woman with an initial working diagnosis of severe primary Raynaud's based on seasonal digital ulceration, marked cold sensitivity, and prolonged capillary refill time. Initial investigations returned a negative ENA, ANCA, RF, cryoglobulins, anti-phospholipid antibodies, viral screen, anti-synthetase antibody panel and normal complements. An endothelin receptor antagonist was trialled with Bosentan along with low dose aspirin but progressively worsening ulceration particularly at left hand ring finger necessitated the introduction of seasonal prostacyclin infusions.

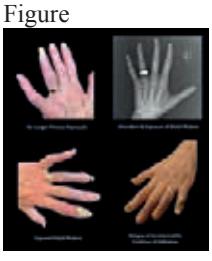
Aims/Background
Further diagnostics returned a borderline elevated estimated pulmonary artery pressure on transthoracic but subsequent cardiac MRI was normal. Cryoglobulins were not detected on multiple repeat samples. Despite worsening ulceration of finger pulps, toes were unaffected and no features of CREST, diffuse systemic sclerosis or connective tissue disease developed.

Method
2 years into treatment course, soft tissue ulceration and gangrene of the left 4th digit finger pulp exposed the distal phalanx to open air. Unlike acro-osteolysis, there was no plain evidence of resorption of the neighbouring distal phalanges. Repeat extended myositis panel returned a positive signal recognition particle (SRP) antibody which was deemed clinically insignificant given the lack of myopathy and the absence of similar cases in the literature.

Results
Swab of the denuded distal phalanx grew MRSA resistant to flucloxacillin and ciprofloxacin. Treatment was commenced with clindamycin. CT angiogram of the left upper limb showed grossly patent arterial vasculature with the exception of the ulnar artery which showed probable low flow but no focal occlusion. Punch biopsy of the left hand 5th digit showed non-specific chronic dermal inflammation and mild perivascular inflammation but no true vasculitis, granulomatous or pustular inflammation or features of lupus or scleroderma.

Conclusions
Eventually the exposed distal phalanx auto-amputated. Onycholysis and retropulsion of neighbouring finger nails supported the new working diagnosis of Acrodermatitis Continua of Hallopeau which is a rare inflammatory pustular psoriasis-like dermatosis of fingers and toes. After MDT discussion with dermatology, methotrexate was commenced with slow but gradual improvement with healing of finger pulp ulceration and onycholysis. Relapse of ulceration this winter however has necessitated the introduction of TNF inhibitor. To date all other fingers have been spared.

Figure



23S116 **CLINICAL CASE**

Roaccutane unmasking Dermatomyositis

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Introduction
Roaccutane (Isotretinoin) is a drug used commonly to treat acne. Although muscle pain and joint pain are reported as very common side effects of Roaccutane (1), myositis associated with this drug is a rare clinical condition (2).

Aims/Background
We present a case of a 40-year-old physically fit and healthy gentleman who was diagnosed with adult-onset acne by Dermatology outpatients and was started on Roaccutane. A few weeks into his treatment he presented with c/o myalgias and proximal muscle weakness. His clinical exam at the time showed muscle power of 4/5. He also had skin involvement, including a periorbital rash, shawl sign and gotterns papules. His initial CK was approximately 9000. He was started on the steroid to which he had a dramatic response. Further investigations confirmed the diagnosis of Dermatomyositis with inflammation in b/l MRI of quadriceps muscles. Muscle biopsy showed MHC class 1 upregulation and was suggestive of an immune-mediated process. His myositis-specific antibody was positive for Anti Mi2 and anti PM 75.
His follow-up outpatient appointment noticed a significant improvement both in his muscle power (5/5) and CK levels. Methotrexate was added to his treatment. However, CK rose again off the steroid and patient was not fully back to his baseline, so IVIG was added to the treatment with Methotrexate

Conclusions
Drug-induced myositis should be taken into consideration in the differential diagnosis of myopathic pain. Early recognition and prompt treatment with immunosuppression are key to achieving remission and avoiding complications.

23S117 **CLINICAL CASE**

A Rare Case of Mixed Cryoglobulinemia with Hepatitis B Infection and Waldernstorms Macroglobulinemia

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Introduction
Cryoglobulinemia is a rare inflammatory syndrome,that causes small-to medium vessel vasculitis.Type I Cryoglobulinemia is associated with lymphoproliferative diseases,while type II, and type III, called mixed cryoglobulinemia (MC), are associated with chronic infections mainly hepatitis C virus (HCV),rarely Hepatitis B virus (HBV) and other systemic autoimmune disorders.We report a unique case of MC in a patient with chronic HBV infection and Waldernstorm's Macroglobulinemia (WM).

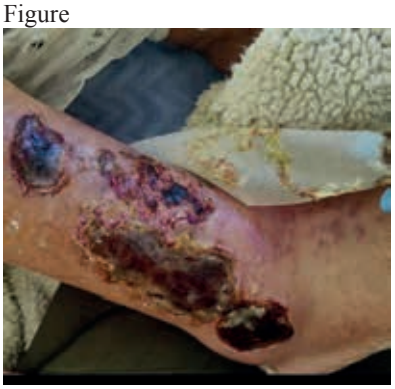
Aims/Background
Case report

Method
A 67 year-old woman with previous medical history of hypertension,atrial fibrillation and peripheral vascular disease, was admitted due to intermittently appearing purpuric rash and ulcerating lesions on both legs and progressive dyspnea over the previous three months.The rheumatology team was asked to assess, whether this lady had an underlying vasculitis.Examination revealed purpuric papules of the lower extremities and deep ulcers on the right leg. There was bilateral pedal oedema to the mid shins.Laboratory tests revealed normocytic anemia, positive rheumatoid factor,positive cryoglobulins,high IgM,hypocomplementemia,elevated BNP and elevated creatinine. Viral screening was consistent with a new diagnosis of chronic Hep B infection.Cutaneous biopsy of the ulcers revealed leucocytoclastic vasculitis.

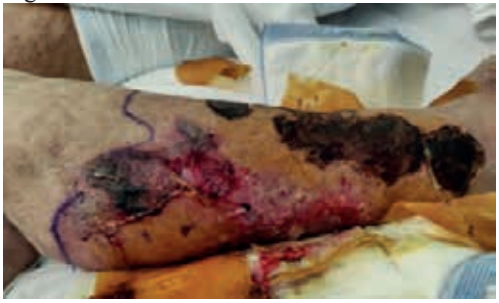
Results
A diagnosis of MC secondary to chronic HBV infection was made. Initial immunosuppressive therapy with methylprednisolone and subsequent oral prednisolone was commenced.HBV was treated with Entecavir. Despite the treatment, the ulcers did not improve so it was decided to treat with Rituximab. Unfortunately three weeks into admission, the patient had a huge clinical deterioration and an acute drop in Hb,rising inflammatory markers. Blood cultures revealed E.coli septicemia which was treated with IV meropenem and Linezolid. The Haematology team was involved to assess for hyperviscosity syndrome or lymphoproliferative disorder. Bone marrow biopsy revealed Waldernstorm's Macroglobulinemia (WM). The diagnosis of MC secondary to WM in a patient with chronic HBV was made. Decision to treat with Rituximab 375mg/m2 weekly dose was made, after sepsis resolution. The patient responded very well to the treatment and was discharged home after 5 weeks.

Conclusions
It was unusual that our patient had potentially two causes for her cryoglobulinemia, both HBVand WM. The use of rituximab in our patient to treat the WM and concomitant anti-viral HBV therapy proved to be safe and effective therapeutic option.

Figure



Figure



23S118 CLINICAL CASE

The Depth of Darkness: Scleral melt due to Rheumatoid Arthritis

Author(s)
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Introduction
We present a rare case of scleral melt (SM) secondary to longstanding Rheumatoid Arthritis(RA). RA is a systemic disease that can affect the eyes. The ophthalmic manifestations of RA include keratoconjunctivitis sicca, episcleritis, scleritis, peripheral ulcerative keratitis, and retinal vasculitis.

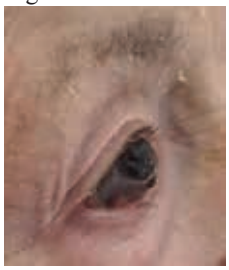
Aims/Background
Case report

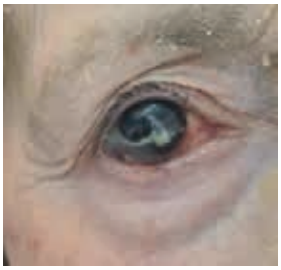
Method
A 72-year-old lady with rheumatoid factor and anti-CCP positive RA diagnosed at the age of 40 presented with three weeks history of right eye pain, discharge, and decreased visual acuity. She was diagnosed with scleral melt, and enucleation was performed due to the severity of the disease and disruption of the integrity of the globe. At the time of diagnosis of RA in 1990, she was treated with steroids, Gold, Penicillamine and Methotrexate. She had poor medication compliance and self-stopped treatment. Over the years, she developed advanced RA deformities and extra-articular manifestations, including visual abnormality. On presentation, she also had infected grade 3 sacral ulcers, and her MRI scan confirmed Osteomyelitis. She was treated with a prolonged course of broad-spectrum antibiotics. She was commenced on high-dose oral steroids (Prednisolone 1mg/kg) and a TNF inhibitor, Adalimumab. She responded well to the treatment, and her symptoms and mobility improved. However, the infection was slow to settle due to the depth of the ulcers and steroid therapy. Despite aggressive immunosuppression, she, unfortunately, developed left-eye scleral melt. At this stage, Adalimumab was switched to Infliximab infusion and Methotrexate 10mg once weekly. Again she responded well to Infliximab, and her visual acuity improved. However, she did not attend her subsequent appointments for Infliximab after two doses.

Results
A multiple-disciplinary approach was taken from the start. Still, despite several interactions and explanations of the importance of the treatment to avoid major organ damage, our patient remained non-compliant.

Conclusions
Scleral melt is a rare complication of RA. We highlight the severity of extra-articular manifestations of RA affecting the quality of life. Aggressive management of RA (As per EULAR recommendations) promptly helps reduce the risks of severe complications associated with RA. Compliance with the treatment and education of the patient is pivotal.

Figure





23S119 CLINICAL CASE

A case of a difficult to treat Juvenile Idiopathic Arthritis

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Introduction
We present the case of a 23-year-old female with a diagnosis of polyarticular Juvenile Idiopathic Arthritis (JIA). This case aims to highlight the complex nature of JIA progression and conversely, the modifications to management for symptom control.

Aims/Background
Following diagnosis at 12 years of age, she was commenced on cDMARD therapy, Methotrexate. At her follow up appointment she had persistent polyarthralgia, early morning stiffness and joint swelling not responsive to NSAID therapy, and was switched to Enbrel in 2013. Due to recurrent episodes of bilateral uveitis, she was switched to Humira, with minimal symptoms for 2 years. She transitioned to adult services with predominantly right knee arthritis.

Method
At 17 years of age her condition progressed from peripheral to axial symptoms. MRI imaging and examination of her pelvis was consistent with bilateral sacroiliitis, requiring a switch to another anti-tnf alpha agent, Simponi. Her Simponi was stopped 4 months later due to recurrence of uveitis and she was commenced on Orencia with subcutaneous Methotrexate. Her disease was quiescent for 3 years.
She was switched to Tocilizumab in 2019, due to debilitating fatigue. She was unfortunately hospitalised after her 5th dose secondary to neutropenic sepsis. She was recommenced on Orencia and MTX but her disease progressed with bilateral TMJ pain, fatigue and polyarthritis. We switched to Infliximab infusion, and remission was achieved.
She had a planned pregnancy at 21 and continued her infliximab until 26 weeks gestational age. She then had a treatment gap until 6 weeks postpartum when her disease flared. Her disease was incredibly difficult to treat and she required multiple courses of glucocorticoids. She was retried with infliximab but developed an infusion reaction. She subsequently failed Upadacitinib, Secukinumab and Salazopyrin.

Results
After 4 months she started Anakinra and subcutaneous methotrexate and her disease remains stable.

Conclusions
This case highlights the difficult nature of managing polyarticular JIA particularly in post-partum period. It also opens a debate if whether continuation of anti-tnf alpha therapy throughout her entire pregnancy would have minimised the risk of her disease flaring.
Home

Photos Gallery

Ms Mary O'Donnell, Ms Theresa Higgins - Nordic; Ms Maureen Kennelly, Celltrion; Ms Mary Mayes - Nordic; and Ms Claire Lenihan - Celltrion

Dr Gary Wright, Mrs Claire Wright, Mrs Sally Doherty, Prof Michael Doherty and Dr Ronan Kavanagh

Photo Gallery

Dr Claire Sheehy, Dr Bernardette Lynch and Dr Eithne Murphy

Prof Ursula Fearon, Dr Mary Canavan and Prof Gerry Wilson

23S120 CLINICAL CASE

Bilateral Longus Coli Muscle Inflammation as an Unusual Cause of Neck pain in the Ankylosing Spondylitis Patient – A New Association?

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Introduction
The longus coli muscle is one of four prevertebral muscles. It spans the anterior surface of the entire cervical spine and the first 3 thoracic vertebrae, acting to flex the head bilaterally and unilaterally tilt the head at the cranio-cervical junction. According to literature, longus coli muscle inflammation is a rare cause of neck pain. There seem to be several suggested aetiologies including calcium hydroxyapatite deposition within the muscle belly, retropharyngeal abscess, trauma, neck mass and discitis and all are usually unilateral. We present to our knowledge, the first case of bilateral inflammation of longus coli muscle in an ankylosing spondylitis patient.

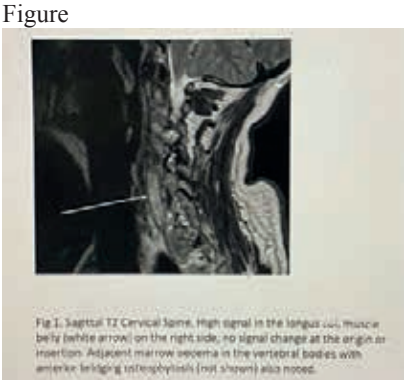
Aims/Background
The patient is 54-year-old male with a 30-year history of ankylosing spondylitis. He is currently treated with Adalimumab (Humira) to which he had a good response, however for over 6 months he developed worsening cervical spine pain associated with dysphonia, odynophagia and reduced range of cervical spine movements.

Method
Not all symptoms could be attributed to the patient's underlying inflammatory disease. Further imaging including CT and MRI scanning were carried out as well as assessment of swallow.

Results
Bloods showed a leucocytosis (18.9 x 10^9/L) and elevated ESR (104 mm/hr). Barium swallow was normal. Cervicothoracic MRI then revealed bilateral, symmetrical diffuse swelling of the longus coli muscle (myositis) extending into the upper chest with associated oedema of adjacent vertebral bodies. These changes were not present on MRI one year prior. There was no evidence of discitis or tendonitis and interestingly, CT scanning did not show any calcification within the muscle.

Conclusions
The purpose of this case is to highlight the potential association between ankylosing spondylitis and longus coli muscle inflammation. To our knowledge there are no previous reports of longs coli inflammation in ankylosing spondylitis. We are investigating whether this changes are mechanical or inflammatory in nature. This case reiterates the importance of recognising and identifying alternative causes of neck pain in ankylosing spondylitis patients.

Figure



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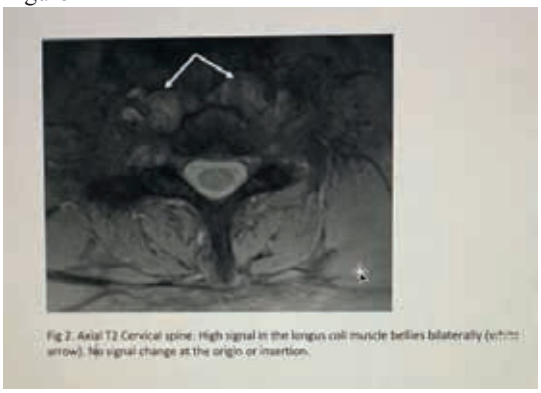

23S121 CLINICAL CASE

A Rare presentation of Psoriatic Arthritis in a patient of Osteopetrosis

Author(s)
Maryum Binte Yousaf, Len Harty, Saleha Huma, Shehla Farrukh

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Introduction
Osteopetrosis is a rare disease affecting bone density with an incidence rate of approximately 1 in 20,000 in autosomal dominant and 1 in 250,000 in autosomal recessive type. Co-existence of psoriatic arthritis with osteopetrosis is an even rarer phenomenon that has not been reported before. We present a case of psoriatic arthritis in a patient of autosomal dominant type 2 osteopetrosis also known as Albers-Schönberg disease.

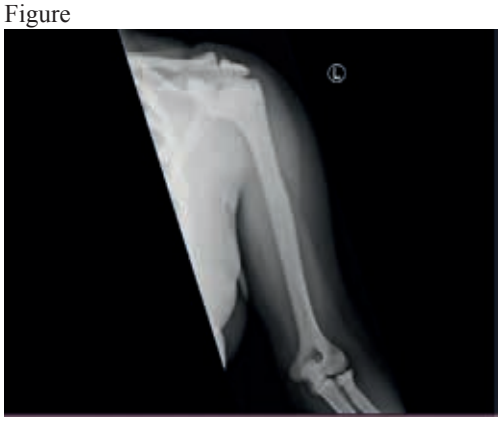
Aims/Background
A 38-year-old lady was referred to us with pain, swelling, stiffness and weakness of her left wrist with pain radiating to forearm. She had a background history of autosomal dominant type 2 osteopetrosis (CNCL7 gene mutation found on genetic testing) with marrow insufficiency associated with recurrent infections, lumbar spine and long bone pathological fractures, winging of right scapula and pulmonary emboli. She had a long-standing history of joint aches and pains which were always attributed to her osteopetrosis. She developed oligoarthritis of left wrist and elbow.

Method
She was subsequently seen in Rheumatology where a detailed history revealed dactylitis and cutaneous psoriasis affecting natal cleft. X-rays of knees and ankles demonstrated preserved joint space. She was diagnosed with psoriatic arthritis. She was started on tapering dose of steroids along with loading dose of secukinumab. Methotrexate was avoided because of established bone marrow insufficiency secondary to osteopetrosis. WBC was 3.6 with neutrophils of 1.78, Hb of 9.9 and platelets of 173.

Results
She developed oral thrush, recurrent infections and nasty pulmonary emboli in the year to follow which led to discontinuation of secukinumab multiple times. Once she was established on the drug, she showed some response with improvement in her joint symptoms.

Conclusions
This is the first case of psoriatic arthritis described in a patient of osteopetrosis. Osteopetrosis complicates the algorithm for treatment of psoriatic arthritis in this case.

Figure



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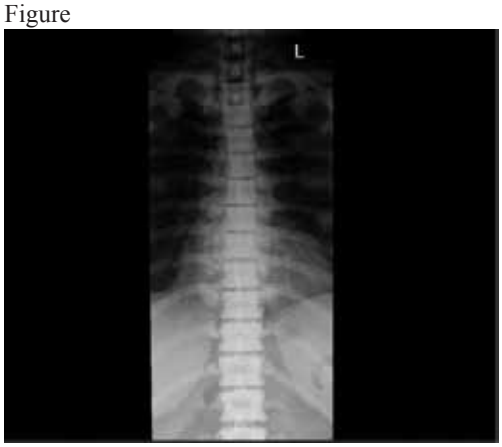

23S122 **CLINICAL CASE**

A long way from the Silk Road

Author(s)
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Introduction
Neurobehcet's is rare manifestation of the Behcet's syndrome occurring in less than 10% of all cases and presenting predominantly in males. Here we describe a case of Neurobehcet's that mimicked infectious aetiologies resulting in a diagnostic dilemma

Aims/Background
A 34 year old Nicaraguan male presented to Tallaght University Hospital and Naas General Hospital on multiple occasions over a period of 8 months. His initial presenting symptoms included fevers, headaches and abdominal pain on two occasions. His symptoms and inflammatory markers improved over a short period and he was discharged home.

Method
His next presentation 2 months later was characterised by recurrent fevers (as high as 40.2 degrees Celsius), abdominal pain, headaches and bilateral limb weakness. A Neurology opinion was sought who noted pyramidal signs with spastic paraparesis on clinical examination. Lumbar puncture demonstrated significantly raised white cell with predominant (99%) polymorphic cells and markedly elevated protein 268mg/dL. Magnetic Resonance imaging of the brain demonstrated a tiny focus of right occipital horn intraventricular diffusion restriction, suspicious for ventriculitis. Given the above findings there was a high clinical suspicion of tuberculous meningitis and patient was commenced on rifampicin, isoniazid and pyrazinamide with 40mg Prednisolone dose tapering by 5mg/week with initial excellent clinical response.

Results
Fevers and lower limb weakness reoccurred when tapered prednisolone dose was reduced to 15mg and he was admitted to hospital again for further evaluation. By this point both CSF and peripheral cultures were negative for Tuberculosis. At this point further history obtained from the patient revealed a long history of mouth ulcers involving tongue, buccal mucosa and gums with attacks occurring three to four times every year since childhood. A Multidisciplinary discussion between Rheumatology, Neurology and Infectious Disease proposed a diagnosis of Neurobehcet's based on long history of mouth ulcers, aseptic meningitis, pyramidal signs and excellent response to high dose steroids. He was commenced on prednisolone 1mg/kg and subcutaneous infliximab and his disease is now in remission

Conclusions
This case highlights the importance of accurate clinical history and examination findings with extensive multidisciplinary input to obtain the correct diagnosis

23S123 **CLINICAL CASE**

Stroke and valvular heart disease in Lupus and Seconday Anti Phospholipid Antibody Syndrome

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Introduction
Libman-sacks endocarditis (LSE), which can be associated with Lupus or APS, is very rare with a prevalence of 0.9-1.6%. We report a case of a patient with multifocal strokes secondary to LSE, treated with anticoagulation and immunosuppression, without further recurrence.

Aims/Background
Case Report

Method
A 36 year old lady with an 8 year history of SLE, was admitted to the hospital with acute confusion, hallucinations, and progressively worsening headaches over the previous 4 weeks. Past complications of SLE included biopsy proven grade IV lupus nephritis and previous cardiac tamponade. She was also positive for anti-cardiolipin antibodies twice in the past but had no history of thrombosis. Her current Lupus treatment included six monthly Rituximab infusions, hydroxychloroquine and 10mg prednisolone.
Neurological exam on the day revealed expressive and receptive dysphasia and poor coordination. There was no neck stiffness. She also had a very loud systolic murmur and significant pedal oedema up to the knees. Laboratory investigations revealed anaemia, elevated CRP and ESR, low complements and elevated dsDNA. The patient's creatinine was elevated at 151 umol/l, in keeping with lupus nephritis.
Brain magnetic resonance imaging revealed numerous small cortical and subcortical acute infarcts in multiple vascular territories. Transthoracic echocardiogram (TTE), requested to assess for cardio-embolic phenomenon, showed dilated left ventricle and reduced ejection fraction of 45%. A subsequent trans-oesophageal echocardiogram (TOE) showed a myxomatous leaflet of mitral valve

with severe eccentric mitral regurgitation. Infectious evaluation including multiple blood cultures were negative. The patient was ultimately diagnosed with LSE of the mitral valve, severe lupus flare and APS.

Results
The patient completed pulse therapy with methylprednisolone and commenced intravenous cyclophosphamide. She was started on warfarin for stroke prevention. She made a good clinical recovery without any further strokes.

Conclusions
Patients with lupus presenting with stroke symptoms should be worked up thoroughly for possible valvular heart disease. TTE alone can miss the diagnosis and acquisition of TOE should be requested. Timely detection of cardiac manifestation and treatment with immunosuppressants and anti-coagulation can reduce the mortality of these patients.

23S124 **CLINICAL CASE**

An Insidious case of Angiosarcoma in complicated pericarditis

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Department(s)/Institutions
Cork University Hospital Rheumatology Department Dermatology Department Cardiology Department Oncology Department

Introduction
A 49 year old man was referred to Rheumatology for consideration of an autoimmune aetiology for recurrent pericarditis, complicated by cardiac tamponade and serositis.

Aims/Background
He presented initially to cardiology with symptoms and echocardiographic evidence of pericarditis. He was treated with NSAIDs and colchicine initially; 2 months later however, he represented with chest pain and cardiogenic syncope. He necessitated emergent pericardiocentesis for cardiac tamponade in the emergency department. CT imaging showed serosal inflammation only. His pericardial and pleural effusion was exudative with benign cytology. His autoimmune screen was negative and a broad infectious workup unremarkable. Given the severity of his presentation, he commenced prednisolone. Anakinra was added for presumptive autoimmune relapsing pericarditis leading to complete resolution of his symptoms.

Method
6 months later, he was re-admitted with a 2 week history of haemoptysis, non-pleuritic chest tightness, shoulder girdle stiffness, malaise, myalgia and night sweats. He had no peripheral stigmata of infective endocarditis, connective tissue disease and no synovitis. CT of Thorax, Abdomen and Pelvis reported multifocal pulmonary and mediastinal nodular calcified lesions. A sub-centimetre lesion within the liver was suspicious for metastases. He also had lytic lesions in his right posterior second rib, T8 vertebral body and right hemipelvis. Echocardiogram showed a large highly mobile linear structure in the right atrium. Cardiac MRI confirmed enhancing mass-like thickening of the right atrium and intra-atrial septum (3.2cm) with peripherally enhancing mediastinal and pericardial nodules and a pericardial effusion. Bronchoscopy was performed, Bronchoalveolar lavage showed no malignant cytology and culture grew proteus mirabilis.

Results
Even with benign cytology, the radiology findings were most in keeping with malignancy. However, despite the presence of multiple nodules, obtaining a targeted biopsy was challenging due to their location. On thorough physical examination, there were 3 small dark-blue discoloured nodules present on his lower back. Biopsy of one of these lesions was consistent with an angiosarcoma. He was commenced on chemotherapy (Doxorubicin/Ifosfamide) with improvement clinically and on follow up imaging and with resolution of serositis.

Conclusions
This case highlights the importance of extensive physical examination and close monitoring in patients with complex or atypical presentations of pericarditis.

23S125 **CLINICAL CASE**

Gout Flare Leading to Pregnancy Diagnosis

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Introduction
This case highlights unusual case of gout in pregnancy. Gout is the most common inflammatory arthritis. However, it is an exceptionally rare phenomenon in reproductive and pregnant woman. This is because oestrogen is thought to have uricosuric effect.

Aims/Background
39 year old lady was referred to inpatient Rheumatology services for presumed reactive arthritis. She had a 2 week history of left ankle pain and swelling, and right knee pain gradually worsening. She was unable to weight bear due to this. 6/52 prior to this, she had flu-like illness and vomiting. 3 months previously, she was commenced on triple therapy treatment for Helicobacter pylori.
Her background history was polycystic ovarian syndrome (PCOS) diagnosed at the age 16. She has a high BMI. She had been on Metformin for her PCOS management. She was also recently diagnosed with Type 2 Diabetes Mellitus (HbA1c 57).
As part of her investigation work up, pregnancy test was performed and it came back positive (at 5/40 gestation). This was unexpected as she has been struggling to conceive due to her PCOS.

Method
On examination, she had raised BMI>35. She had tender and swollen erythematous right knee effusion. Clinically, she was not septic and no peripheral stigmata of inflammatory arthritis. Serum urate was 678. Right knee X-ray showed effusion with no erosion or degenerative arthropathy. Her joint aspirate showed crystals consistent with gout.

Results
She was diagnosis with crystal proven gout that was triggered in early pregnancy and recent diagnosis of T2DM on the background of PCOS. She does have increased risk of gout due to her PCOS, but interestingly her first manifestation was during pregnancy.

Conclusions
The case highlights the importance of pregnancy testing in reproductive age woman. Although it is rare in pregnancy, gout should be part of the main differential diagnosis for patients presenting with monoarthritis. There is still not enough evidence regarding safety of urate-lowering therapy in pregnancy.

Photo Gallery

Ms Sarah Dawson, Ms Rachel Kenny, Ms Aileen Woods, Dr Sarah Quidwai, Ms Marian Hayden

Dr Wan Lin Ng, Dr Brona Dinneen, Dr Aoife Cox, Dr Cillian Hurst and Dr Imran Ali

Photo Gallery

Dr Aubrey Bell

Prof. Barry O'Shea and Dr Clare Matthews

Ms Linzi Martin, Ms Darina Murhy, Ms Clara Bannon, Ms Martina Corbett and Ms Mary Gillespie

23S126 CLINICAL CASE

The Use of PET-CT to Monitor Disease Activity in Juvenile Dermatomyositis.

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Introduction
This case demonstrates that PET-CT can be a useful means of assessing disease activity in patients with juvenile dermatomyositis with extensive calcification which can interfere with other imaging modalities.

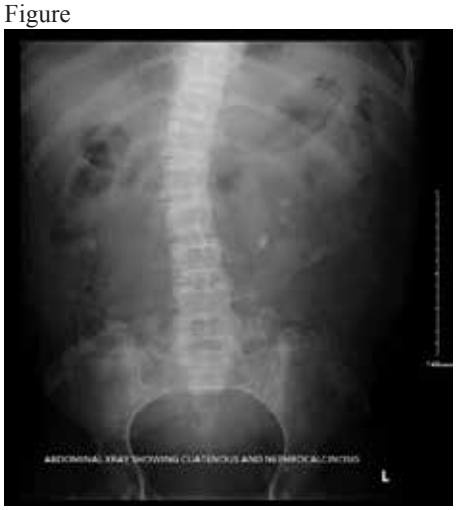
Aims/Background
We present the case of 26 yr old female diagnosed with juvenile dermatomyositis age 6 when she presented with myositis, rash and large joint arthritis. Initial treatments included pulse steroids, azathioprine, cyclosporine, methotrexate and IVIG followed by a number of courses of cyclophosphamide and infliximab.

Method
She became wheelchair bound despite rehabilitation with extensive ulcerating calcinosis and contractures. She then developed abnormal liver function tests attributed to hepatic steatosis and methotrexate toxicity. Further metabolic derangements included dyslipidemia, hypertension, insulin resistance, obstructive sleep apnoea and PCOS. She has an abnormal body composition as a result of her cutaneous calcification.
In addition to the widespread cutaneous calcification she developed a staghorn calculus, requiring uretic stenting and osteoporosis. She continued to have difficult to control muscle disease with ongoing activity on PET-CT and was commenced on mycophenolate mofetil.

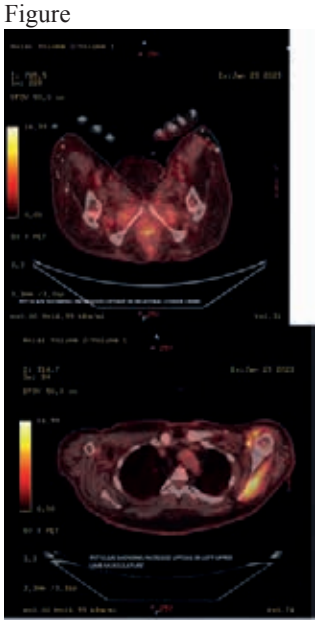
Results
Her antibodies include MDA5, NXP2, SAE1 and PMscl100. She does not have any Raynauds nor scleroderma-like skin changes. Recently her mycophenolate therapy has been interrupted by derangements in her liver function and as a result she has had a flare in her muscle symptoms. CK had been stable in the low normal range over last 5 year but recently increased to 204. We suspected disease activity.

Conclusions
This has been confirmed with a second PET-CT which shows new increase FDG uptake in left upper extremity and bilateral lower limb musculature along with mild cutaneous activity in left posterior thigh signifying disease activity. She is being worked up for rituximab therapy in addition to her mycophenolate mofetil.

Figure



Figure



23S127 CLINICAL CASE

Successful Medical and Surgical Management of 'AAA' Critically Unwell Rheumatology Inpatient

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Introduction
Idiopathic retroperitoneal fibrosis is a rare condition characterized by the presence of inflammatory and fibrous tissue in the retroperitoneum. The tissue is generally localised around the infrarenal portion of the abdominal aorta and iliac arteries often encasing the ureters or other abdominal organs.Rapid diagnosis and early specialist treatment are key to good patient outcomes. Here a very ill gentleman who benefitted from a multidisciplinary approach is discussed.

Aims/Background
A 62 yo man presented with a 9-day history of back pain and anuria. He had a raised serum creatinine of 1848umol/L, K+ 7.4mmol/L, pH 7.23, Bicarbonate 15 and CRP of 74. A CT angiogram confirmed an abdominal aortic aneurysm (AAA) (noted on ultrasound) and multifocal irregularity of the aorta and periaortic inflammatory mass causing bilateral hydronephrosis. (FIGURE 1)
He was transferred urgently under the Vascular team to ICU. He had emergency dialysis and bilateral nephrostomies were inserted with a rapid recovery in renal function and per-urethral urine flow, but on day 5 developed acute onset left-sided weakness and dysarthria. Acute evolving occipital and right medulla infarcts appeared on CT cerebral angiogram. Features suspicious for aortitis and vasculitis of the right MCA branches were noted. He was commenced on IV methylprednisolone, with an immediate resolution of symptoms and switched to oral steroids on day 3. The rheumatology team noted normal Rheumatoid Factor, anti-CCP, IGG4, ANA and ANCA. The vascular team performed an EVAR and a CT guided biopsy of the inflammatory mass revealed findings possibly consistent with a large/medium vessel vasculitis.

Method
Inpatient Rituximab 2 weeks apart was followed by tapering dose of steroids on discharge, and non-functioning nephrostomies remained in situ. His kidney function reached a new baseline-CKD stage IIIb.

Results
In clinic, he had intermittent claudication and switched to methotrexate with maintenance Rituximab 1g dose after 6 months. Follow up CT angiogram revealed reduced aortic thickening while inflammatory markers were normalised.

Conclusions
In summary, a critically unwell man with vascular, renal, urological and neurological manifestations of retroperitoneal fibrosis is presented, who had mild residual intermittent claudication and CKD. Expert management from multiple specialists resulted in an excellent outcome.

23S128 **CLINICAL CASE**

It's all in your head

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Department(s)/Institutions
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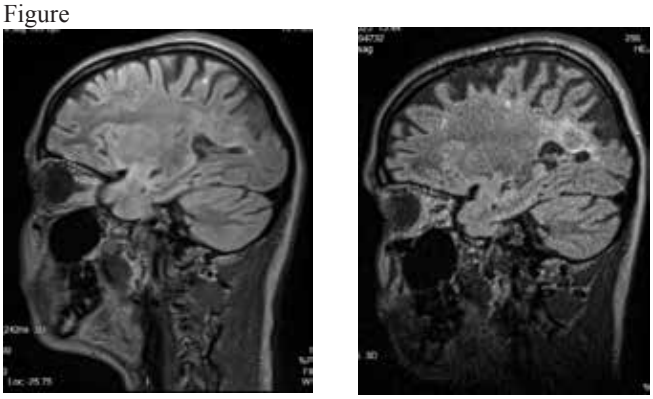
Introduction
A 41 y/o lady with a 4yr history of SLE presented to ED with acute onset right sided facial anaesthesia, parasthesia and a right sided facial droop on 09/01/23.

Aims/Background
Her background SLE diagnosis 2019 which initially presented with headache, a right seventh nerve palsy and glosso-pharyngeal neuralgia which resolved spontaneously over six days. MRI brain in 2019 showed extensive white matter signal abnormality in the supratentorial brain bilaterally of uncertain significance. CSF unremarkable and negative for oligoclonal bands. She subsequently developed systemic symptoms w/ arthralgia, dry eyes, dry mouth and a photosensitive facial rash. Her bloods were positive for anticardiolipin antibody IGG and IGM with a low C4 level; positive antinuclear antibody levels and anti DS-DNA antibodies (ANCA negative). Following extensive neurology involvement, a unifying diagnosis of systemic lupus erythematosus was made and an agreed plan for hydroxychloroquine was commenced with no active symptoms until 2023.

Method
Following her 2013 presentation, urgent CT brain and CT angiogram were obtained on this presentation and were both reported as unremarkable. MRI brain, (i) a small established interval cerebellar infarct and (ii) similar extensive white matter signal abnormality, but also new volume loss in the frontal and parietal lobes out of keeping with age. Subtle cognitive defecits were noted, formal neuropsychological testing pending. Of note, she was noted to have a previously strongly positive B2-Glycoprotein 1 antibody of 165 (normal <7) and a positive lupus anticoagulant in 2019. Repeat screen pending.

Results
She was commenced on steroids and cyclophosphamide therapy for a working diagnosis of a CNS vasculitis secondary to lupus, however an alternatIve hypothesis of microangiopathic thrombosis due to an antiphospholipid syndrome is also proposed.

Figure



23S129 **CLINICAL CASE**

Limited cutaneous systemic sclerosis, SLE, Large vessel vasculitis Overlap syndrome in a young Angolian patient

Author(s)
Saleha Huma, Shehla Farrukh, Maryum Binte Yousaf, John Bourke, Michael O'Shaughnessy, Len Harty

Department(s)/Institutions
Department of Rheumatology, SIVUH Department of Plastic surgery, SIVUH Department of Dermatology, SIVUH

Introduction
SSc-SLE Overlap syndrome is rare. Little is known about the epidemiology, clinical characteristics, and survival of SSc-SLE overlap syndrome.

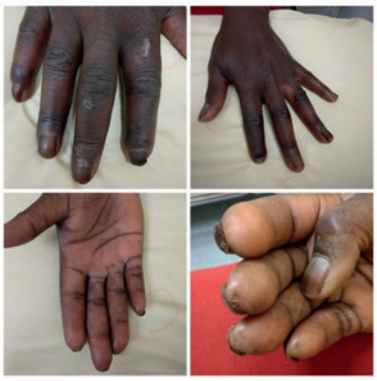
Aims/Background
A 42-year-old male presented with cutaneous lupus, scarring alopecia, positive ANA in 2015, later developed Scleroderma with positive anti-centromere antibodies in 2018. He represented with digital ischemic ulcers in 2021, despite taking hydroxychloroquine (HCQ) and nifedipine.

Method
Now had multiple digital ulcers, thickening of skin mRSS 6, diffuse alopecia and arthritis of PIPs, knee and hip joints. Laboratory results showed WBC 3.1, lymphocyte 0.80, ESR 33, CRP 5, normal Complement C3 and C4, positive ANA, anti-centromere, anti-Sm, anti U1 RNP, anti-Ribosomal P Protein, anti-PM-Scl 100 antibodies. He received Iloprost, corticosteroids, nifedipine, sildenafil and HCQ 200 mg bd with addition of bosentan 125 mg tds and Cellcept 1 mg bd upon further relapse when he had gangrene at tips of 2nd, 3rd, and 4th digits of Right hand and 2nd digit of Left hand. CT angiogram arch of aorta (to see any source of emboli) showed short occlusion and tight stenosis of left subclavian artery suggesting large vessel vasculitis LVV. Echo showed EF of 50% with no PAH.

Results
Remission was induced with 5 cycles of cyclophosphamide followed by 6 monthly Rituximab, bosentan and nifedipine. Plastic surgery led hand therapy achieved full ROM in the hands. RP was controlled with no further ulcers. No other complications were seen.

Conclusions
A collaborative multidisciplinary approach involving dermatology, plastic surgery, occupational therapy, emergency medicine, radiology and rheumatology resulted in optimal outcome for this critically ill patient with rare presentation of SSc/SLE/LVV Overlap syndrome, now maintaining on Rituximab immunosuppressant with bosentan and nifedipine vasodilators.

Figure



Figure

AUTOIMMUNE PROFILE				
#	2015	2018	2019	2021
ANA	+ (1:640)	+	+	+ 1S (positive >1)
dsDNA	-	-	-	-
Anti Sm Ab	-	-		+ 14 (range < 7)
Anticentromere Ab	-	+		+ 240 (range < 7)
Anti Scl 70 Ab	-	-	-	-
Ribosomal P Protein Ab		+		
U1 RNP Ab	-	-		+ 44 (range < 5)
Anti Mitochondrial Ab	-	-	-	4 (Equivocal 4-6)
Anti Smooth Muscle Ab	-	-	-	-
MYOSITIS SCREEN (28/10/2021)				
Anti PM SCL 100 Ab				Weakly positive

23S130 **CLINICAL CASE**

No way it's NOMID!

Author(s)
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Department(s)/Institutions
Rheumatology Department - Tallaght University Hospital

Introduction
18 y/o lady with NOMID/CINCA syndrome (diagnosed aged 6), chronic glucocorticoid use since aged 2 when diagnosed with rheumatoid oligoarthritis. Refugee presents to Rheumatology clinic for management of above and her primary subjective issue was profound growth retardation as well as foot and knee pain.

Aims/Background
Little known background due to language barrier and difficulty accessing records. On exam very short stature 105cm, weight 18.3kg, bilateral cheek erythema, cushingoid facies and valgus bone deformity.
XR of bilateral wrists, hands, femurs, tibia / fibulas showed marked skeletal immaturity for age, growth plates unfused with stippled, deformed appearances of multiple growth plates/epiphyses and decreased bone density. Hand and wrist bone age 4.65 years as per Greulich-Pyle scale. DEXA scan pending. These radiological features characteristic for NOMID.

Method
Bloods: Amyloid A 109; cortisol 27; Growth hormone 0.44
Commenced on anakinra in April 2022 and steroids slowly reduced, joint pain much improved.

Results
Reviewed by endocrinology who noted she is primary amenorrhoeic, limited sexual development and diagnosed hypothalamic pituitary adrenal axis suppression due to long history of glucocorticoid use. Referred to paediatric endocrinology department for further investigation and management of growth hormone deficiency. She has hypogonadotropic-hypogonadism and oestrogen replacement therapy is planned which may expedite the fusion of her growth plates.

Conclusions
This case demonstrates challenges in managing chronic diseases with patients newly presenting to clinics with limited medical records.

Figure

23S131 **CLINICAL CASE**

Eosinophilic fasciitis likely secondary to an immune checkpoint inhibitor

Author(s)
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Introduction
Immune checkpoint inhibitors (ICIs) have revolutionised the treatment of several cancers. ICIs enhance the patient's immune system through cytotoxic T-lymphocytes associated protein-4 and programmed cell death-1 and its ligand. However, the over-activation of the immune system causes a variety of immune-related adverse events (irAE), including rheumatological. We report a case of eosinophilic fasciitis (EF) likely secondary to an ICI, Pembrolizumab.

Aims/Background
An 82-year male presented with joint pain in his thumbs and wrists associated with skin thickening and induration of forearms and lower limbs. He also complained of myalgia, fatigue and reduced manual dexterity. Background history revealed treatment for Hodgkin's Lymphoma (HL) in 2016. Following the relapse of HL in 2019, he was commenced on Pembrolizumab and remained in remission.

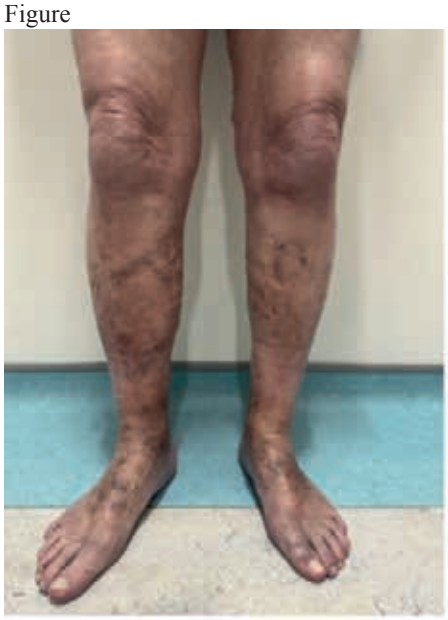
Method
Eosinophilic fasciitis (EF) is a rare scleroderma-like disorder. The absence of Raynaud's phenomenon, organ involvement and lack of autoantibodies differentiates it from systemic sclerosis. Possible causes of EF include medications, haematological malignancies or idiopathic. EF usually spares the trunk and face and may be associated with peripheral eosinophilia. There are no censuses guidelines for treatment. However, patients with EF tend to respond well to steroids and oral immunosuppressants.

Results
Blood results revealed a normal complete blood count with no lymphopenia or eosinophilia. The connective tissue disease screen

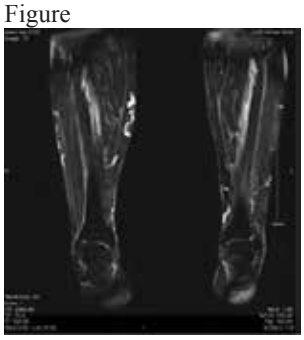
was negative, including anti-centromere antibodies and anti-SCL 70. MRI whole body showed fasciitis, predominantly in the thighs. Skin biopsy showed moderate mononuclear lymphocytes and plasma cells. Pembrolizumab was discontinued, and he was commenced on Prednisolone and Methotrexate. The skin thickening and induration improved.

Conclusions
ICIs are commonly used medications for malignancies and should be considered as a cause for EF in patients presenting with scleroderma-type skin features.

Figure



Figure



23S132 **CLINICAL CASE**

"Something Fishy About a Psoriatic Arthritis Diagnosis"

Author(s)
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Tallaght University Hospital

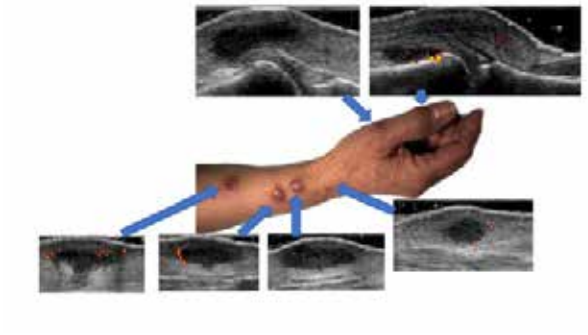
Introduction
Diagnosing the underlying cause of arthritis can be challenging at times. Although a scaly rash and nail lesions associated with swollen finger joints can indicate psoriatic arthritis, rare mimics exist. In this case study, we describe a patient who initially appeared to have psoriatic arthritis but was later diagnosed with Mycobacterium marinum infection by carefully considering the patient's history, examinations, and ultrasound findings.

Case Presentation:
A 59-year-old woman with no prior medical conditions presented with pain and swelling in her right first interphalangeal (IP) and metacarpophalangeal (MCP) joints, red scaly lesions on the dorsum of her right forearm, and onycholysis of her right first nail. Her inflammatory markers were normal, and she had no fever or other constitutional symptoms. Ultrasound revealed swelling within the right first IP joint and hypoechoic soft tissue thickening superficial to the extensor tendon. The patient reported cleaning her fish tank before the onset of her symptoms and experiencing pain in her thumbnail with discharge, which resolved within 1 week with clindamycin. Biopsy results indicated a nontuberculous mycobacterial infection, which we are confident will be shortly confirmed as M. marinum (final culture results pending).

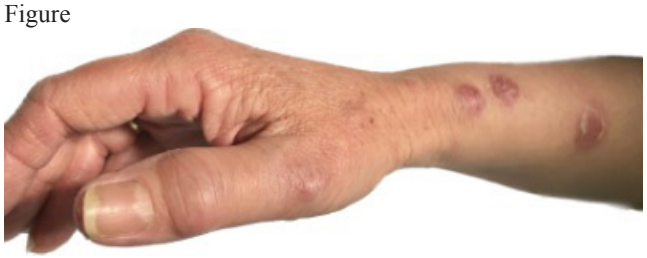
Discussion:
M. marinum is an uncommon skin infection acquired from aquatic sources, including fish tanks. Rare cases have involved hand joints, mimicking inflammatory arthritis and leading to inappropriate treatment with immunosuppressants. The differential diagnosis includes other infections causing nodular lymphangitis, such as sporotrichosis. A tissue biopsy can be used to confirm the diagnosis.

Conclusions
M. marinum infection can mimic psoriatic arthritis, but thorough history taking, examination, and ultrasound can provide clues to the correct diagnosis. Accurately identifying the underlying cause of arthritis is crucial for proper management.

Figure



Figure



23S133 **CLINICAL CASE**

All Aboard!

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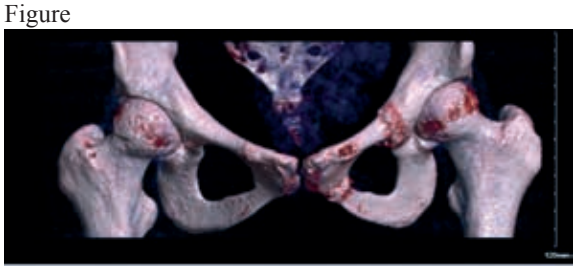
Introduction
Stress fractures are overuse injuries to normal bone caused by increased load and/or increased repetitions. They have long been described in runners and athletes and generally occur more commonly in the lower extremities.

Aims/Background
A 26 year old waitress working on board a ship in the Caribbean presented to the emergency department with worsening left sided hip pain. The pain was gradual in onset and there was no history of trauma. There was a history of weight loss two years ago resulting in amenorrhoea for 6 months but this weight loss was quickly regained. There was a strong family history of breast and bowel cancer. A plain film of her pelvis revealed an undisplaced healing inferior pubic ramus fracture.

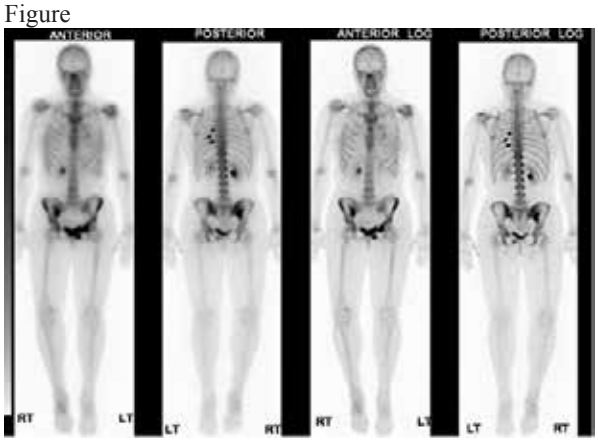
Results
Initial bloodwork showed an iron deficiency anaemia. Following the xray pelvis, a CT pelvis revealed several "sclerotic looking lesions" suspicious for metastatic disease. Tumour markers including alpha fetoprotein, beta hCG, CA 125 and CA 19-9 were negative. Serum protein electrophoresis and anti transglutaminase antibody was also negative. Cortisol, prolactin and PTH was normal. CT TAP did not show any malignancy. MRI pelvis revealed ultiple bilateral pelvic fractures with associated bone oedema. A nuclear medicine bone scan showed multiple areas of focally intense activity at the posterior segment of left sided ribs 7 to 9 and in the left superior pubic ramus, left inferior pubic ramus and left iliac bone. A DEXA scan showed osteopaenia.
Bone turnover bloods showed a P1NP of 70ng/mL, normal CTX-crosslaps, osteocalcin and bone alkaline phosphatase.

Conclusions
This patient is an unusual case of multiple stress fractures most likely as a result of carrying very heavy trays while waitressing. There was no evidence of an underlying metabolic bone disease although she has been recommended for endoscopy in the context of iron deficiency anaemia despite no gastrointestinal symptoms and a negative anti tTG. A vitamin D level was low at 32nmol/L and she has been started on supplementation. She is making a good recovery and is now mobilising with two crutches. Advice on maintaining a stable body weight and nutrition were given.

Figure



Figure



23S134 **CLINICAL CASE**

Unusual Calcifications

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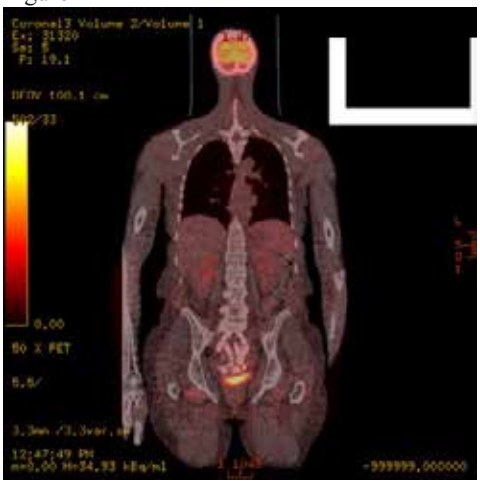
Introduction
Low vitamin D during bisphosphonate therapy has been shown to result in suboptimal changes in bone mineral density in several studies. Bisphosphonates such as IV Zolendronic acid have been shown to have an anti myeloma effect and are included as treatment in international multiple myeloma guidelines. We present an unusual manifestation of vitamin D deficiency in the setting of monthly IV bisphosphonate.

Aims/Background
A 55 year old female with a background of multiple myeloma was referred to the rheumatology department with widespread pain. The pain affected all joints and muscles in no particular pattern, was constant and not associated with any joint swelling. Symptoms improved while on steroids and was further controlled with opiates. Examination revealed non tender palpable nodules along her right forearm. There was no joint tenderness and normal range of motion in all joints. Xray did not show any calcinosis.
Prescribed medications included maintenance Lenalidomide and monthly IV Zolendronic acid. A CT skeleton at the time of presentation did not show any evidence of bony lesions.

Results
Initial bloodwork at the time of rheumatology referral showed a mild pancytopaenia with normal renal, liver and bone profiles. Inflammatory markers were normal and rheumatoid factor was negative. A PET CT was organised to assess for relapse of multiple myeloma. This revealed confluent symmetrical calcification of the abdominal wall sheath and in the soft tissues of the proximal limbs, notably in the shoulders and adductor compartments of the hip joints. There was also mild generalised oedema of the skin.
Vitamin D was found to be very low at 22nmol/L.

Conclusions
A loading dose of vitamin D was prescribed and a 70% improvement in symptoms was described at 8 weeks. There was complete resolution of palpable nodules in the forearm. Our patient is now off opioid analgesia and reports minimal joint pain. We conclude that the severe arthralgias and abnormal soft tissue calcification was as a result of low vitamin D in the setting of monthly IV bisphosphonate. This has not been previously described in the literature. Vitamin D should always be evaluated before bisphosphonate therapy use.

Figure



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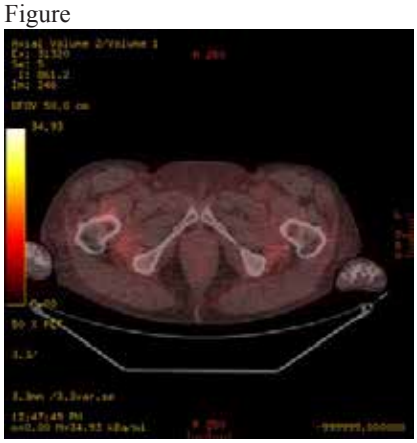

23S135 CLINICAL CASE

A New Rash

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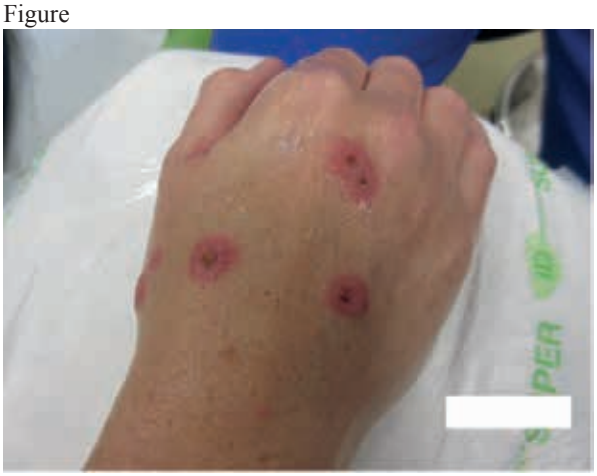
Introduction
This is a case report describing the atypical presentation of Crohn's disease, with primarily cutaneous symptoms, in a 35 year old patient.

Aims/Background
The patient has a background of small and large bowel Crohn's disease, managed on Azathioprine and Prednisolone and a recent new diagnosis of IBD associated inflammatory arthritis.
There is a significant history of renal transplant for end stage renal disease secondary to Sulfasalazine related chronic tubular interstitial nephritis and subsequent immunosuppression with Tacrolimus.

Method
In November 2020, a 35 year old female presented to routine nephrology clinic review and was noted to have a new rash. This came on after completing a short course of steroids for a Crohn's flare. The rash started on her hands and then spread to the right ear, abdomen and lower legs. The rash began as small pustules which enlarged into nodules with a central crust. It was painless and non pruritic.
Initial impression from Dermatology was of a neutrophilic dermatosis and the patient was started on Dermovate ointment topically. Skin biopsy however, showed small non caseating granulomas in the dermis and subcutaneous tissue. This was discussed at the Dermapathology MDT and thought to be consistent with cutaneous manifestation of IBD.
She was started on Adalimumab for the inflammatory arthritis and Crohn's disease and at 8 weeks had complete resolution of the rash with post inflammatory pigmentation only at the sites of previous lesions. Her Azathioprine had been reduced.

Conclusions
Cutaneous manifestations of extraintestinal IBD flares are not uncommon. They range from mild to severe but also vary significantly in character. Erythema nodosum and pyoderma gangrenosum are well described. Non caseating granulomatous deposits in the skin tissue is the most rare dermatological manifestation of IBD and has been referred to as metastatic Crohn's disease in the literature. This is diagnosed using skin biopsy, where the histopathological findings can be consistent with that of the underlying gastrointestinal disease, as demonstrated In this case.
This is treated with the same anti-inflammatory and immunosuppressive therapies as in intrabdominal IBD without extraintestinal manifestation.

Figure



23S136 CLINICAL CASE

A rare case of Eosinophilic Granulomatosis with Polyangiitis (EGPA) leading to Myocarditis and Severe Mononeuritis Multiplex.

Author(s)
Mohamed Idris , Ronan Mullan

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Department of Rheumatology Tallaght University Hospital

Introduction
Eosinophilic Granulomatosis with Polyangiitis (EGPA) is a rare form of anti-neutrophil cytoplasm antibody (ANCA)-associated vasculitis. We report a clinical case of EGPA with Cardiac and severe nervous system involvement.

Aims/Background
We present a 52 years old female whom within one month presented twice to the Emergency Department with lower back pain, Hand and feet arthralgia and was discharged on both occasions with analgesia and Physiotherapy referral. She presented on this admission with difficulty in holding objects with both hands and give away weakness in her leg. She has background history of Sinusitis and a recent diagnosis of Asthma.

Method
Paraesthesia and weakness in the distal muscle groups of both hands and feet with power Grade 3/5 on Wrist extension and flexion Bilaterally, Finger Flexion G4/5 on Right hand and G3/5 on the left. There was pronounced reduced left Ankle Dorsiflexion, Ankle plantarflexion G1/5 and G2/5 respectively.

Results
Laboratory studies revealed elevated troponin level 1006ng/L, BNP 1435pg/ml, leukocyte count 21×109/l with predominant eosinophilia 13.3×109/l, Erythrocyte sedimentation rate 36mm/hr, C-reactive protein 90mg/L. Anti Myelo-peroxidase (MPO)-ANCA was strongly positive 134U/ml.
ECG showing Minor ST depression in inferio-lateral leads .Echocardiography showed left ventricular systolic function 49% and Pericardial Effusion.
Baseline Cardiac MRI showed low normal ventricular function.
Electromyography and Nerve Conduction Study indicated acute axonal injury consistent with Mono-Neuritis Multiplex. Sural nerve biopsy was suggestive of Neuropathic changes.
Computed tomography of the lung and sinus revealed Ground glass changes, small Nodule and moderate paranasal sinusitis.

Conclusions
On the basis of clinical features, including history of Sinusitis and Bronchial Asthma, Eosinophilia, Mono neuritis multiplex, MPO-ANCA positive and Myopericarditis a diagnosis of EGPA was made. Pulse therapy with Methylprednisolone and Cyclophosphamide as induction due to the life-threatening cardiac involvement and rapid neurological deterioration.
Multidisciplinary meeting concluded to initiate Mepolizumab as Maintenance therapy alongside Glucocorticosteroids tapering dose.
The patient had a good symptomatic and biochemical recovery and currently engaging with neurology rehabilitation.
Cardiac involvement is the major cause of morbidity and mortality in these patients .Early recognition and treatment initiation are essential to improve patients outcome and to prevent the establishment of fixed peripheral neuropathy.

23S137 **CLINICAL CASE**

An Uncommon Cause of Foot Drop

Author(s)
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Introduction
Foot drop refers to an inability to lift the forefoot due to weakness of the dorsiflexors. For patients presenting acutely a broad differential exists however compressive peroneal neuropathies are the most common cause

Aims/Background
In this study, we present an uncommon case of foot drop caused by an intraneural ganglion cyst of the common peroneal nerve in which the initial diagnosis was made by bedside ultrasonography

Method
Case report

Results
Case report

Conclusions
Compressive common peroneal neuropathy by an intraneural ganglion cyst is rare and the condition is therefore not commonly diagnosed or misdiagnosed. A ganglion cyst should be considered as a differential diagnosis of foot drop. Point of care, ultrasound is a valuable tool for not only identifying the lesion, but also to aid in treatment through decompression by aspiration.


Figure 2

Figure

AN UNCOMMON CAUSE OF FOOT DROP

Abuelgasim, Omer O'Neill, Lorraine Browne, Peter

Department of Rheumatology, University Hospital Kerry

INTRODUCTION

Foot drop refers to an inability to lift the forefoot due to weakness of the dorsiflexors. For patients presenting acutely a broad differential exists however compressive peroneal neuropathies are the most common cause.

Compression of the common peroneal nerve at the level of the fibular head is the most common site of entrapment.

In this study, we present an uncommon case of foot drop caused by an intraneural ganglion cyst of the common peroneal nerve in which the initial diagnosis was made by bedside ultrasonography.

CASE PRESENTATION

A 60 - year - old female known to our service with arthralgias and a positive rheumatoid factor, presented with acute onset left foot drop. There was no history of recent trauma or surgery. She was otherwise well with no constitutional or systemic symptoms. She reported mild back pain with no associated radicular symptoms. Physical examination of the left foot and ankle revealed weakness of dorsiflexion, eversion, and toe extension. Inversion, plantar flexion, and toe flexion were intact. Sensation of the left lateral leg and dorsal aspect of the foot was decreased.

Bedside ultrasound of the left knee demonstrated an anechoic cystic lesion arising from the proximal tibiofibular joint causing a mass effect on the adjacent common peroneal nerve, resulting in nerve enlargement and loss of the normal internal striated echotexture. Subsequently, MRI of the left knee demonstrated a 10x6 mm cyst extending along the common peroneal nerve from behind the lateral femoral condyle, extending inferiorly along the lateral margin of the head of the fibula and extending anteriorly into the anterior compartment. It measured approximately 6.5 cm in long axis diameter and felt consistent with extensive peroneal nerve ganglion (Figure 1).

DISCUSSION

Ganglion cysts causing compression of the common peroneal nerve are uncommon causes of a foot drop. A systematic review reported in 2022 identified only 570 published cases of an intraneural ganglion cyst of the common peroneal nerve (1).

They can be classified as either intraneural or extraneural lesions. Intraneural ganglion cysts are rare, fluid-filled formations within the epineural sheath of peripheral nerves that can cause nerve compression.

On ultrasound, these lesions appear as anechoic, multiloculated, cystic fluid collections that track along the path of the common peroneal nerve which can cause mass effect resulting in nerve enlargement. Ultrasonographic measurements and cut off values of the common peroneal nerve cross sectional area at the fibular head

They can be classified as either intraneural or extraneural lesions. Intraneural ganglion cysts are rare, fluid-filled formations within the epineural sheath of peripheral nerves that can cause nerve compression.

On ultrasound, these lesions appear as anechoic, multiloculated, cystic fluid collections that track along the path of the common peroneal nerve which can cause mass effect resulting in nerve enlargement. Ultrasonographic measurements and cu off values of the common peroneal nerve cross sectional area at the fibular head have been reported but validation data is limited.

Both ultrasound and MRI provide important information about both the anatomy of the underlying nerves and muscles, as well as the pathoanatomy. Ultrasound is widely available and less expensive, although it is limited in its ability to penetrate bony structures. Ultrasound allows for high spatial resolution and continuous visualization of the nerve, and with the development of ultra-high frequency transducers, the spatial resolution for visualizing the more superficial peripheral nerves can be as high as 30mm. MRI is a more expensive study and not readily available but carries less user dependence when imaging peripheral nerve lesions when compared to ultrasound.

The treatment is often surgical although some case studies support ultrasound-guided needle aspiration and steroid injection of the cyst with good outcomes.

CONCLUSION

Compressive common peroneal neuropathy by an intraneural ganglion cyst is rare and the condition is therefore not commonly diagnosed or misdiagnosed. A ganglion cyst should be considered as a differential diagnosis of foot drop. Point of care, ultrasound is a valuable tool for not only identifying the lesion, but also to aid in treatment through decompression by aspiration.

Photo Gallery

Dr Emma MacDermottt, Prof Geraldine McCarthy and Dr Michele Doran

Dr Leah Rooney, Ms Alexia Kelly, Ms Lorna Freeman, Ms Hannah Woods

Photo Gallery

Dr Viviani Marzaioli

Mr Bryan Walsh, Amgen; Dr Barry Sheane

Audience View

23S138 CLINICAL CASE

An Unusual Case of Severe Systemic Vasculitis

Author(s)
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Department(s)/Institutions
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Introduction
We are presenting case of systemic vasculitis that occur after Covid-19 vaccination

Aims/Background
Vasculitis involves inflammation of the blood vessels. Vasculitis can be caused by infection such as Hepatitis B and C, immune system disorders or reaction to certain drugs.

Method
A 55 year old male presented to ED with fever, severe night sweats and significant weight loss associated with myalgia and headache which was atypical for GCA. These symptoms had developed shortly after he received Covid-19 vaccination. He had no other symptoms suggestive of arthritis or connective tissue disease. He denied chest pain, shortness of breath, skin rash or symptoms of inflammatory bowel disease.

Results
His bloods showed ESR 59, CRP 50, mild leukopenia and neutrophilia. He had a negative auto immune and viral screen. SPEP was normal. Bone marrow aspirate showed only a mild reactive process with no clear sign of an infective process. ANCA was weakly positive with atypical but normal PR3 and MPO. CT thorax, abdomen and pelvis showed no evidence of malignancy. The patient was treated with PO antibiotics in hospital without obvious immediate benefit. His symptoms subsequently resolved spontaneously. He was asymptomatic when he attended for an outpatient PET scan 3 months later and remained asymptomatic when he was reviewed at OPD with the results. Despite his lack of symptoms the PET Scan showed extensive large vessel vasculitis involving the entire aorta, proximal great vessels and bilateral common iliac arteries. There was also evidence of synovitis affecting bilateral glenohumeral joints consistent with inflammatory arthritis. Treatment was commenced with a tapering dose of prednisolone, starting at 40mg daily. He was noted to have a recurrence of arthralgia when he was reviewed in the OPD a few months later. He was commenced on treatment with the IL6 inhibitor Tocilizumab. Since starting on Tocilizumab his symptoms have improved significantly and steroid therapy has been discontinued.

Conclusions
There are 2 cases of systemic vascilitis post Covid-19 vaccination described in the literature. Due to the temporal relationship with vaccination and onset of symptoms it is possible that the onset of vasculitis in this case was consequent on recent Covid 19 vaccination.

Figure

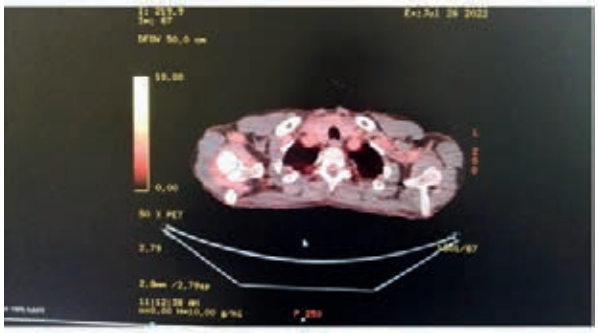

Photo Gallery

Ms Suzanne Alston, Ms Nicole O'Keeffe, Ms Grainne Cussen and Ms Karen Quinn

Dr Eithne Murphy, Dr Frances Stafford, Ms Jill Mullins (BMS)

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