ARMD

ARCHIVES IN RHEUMATIC AND MUSCULOSKELETAL DISEASES

REVISTA DIGITAL DO INSTITUTO PORTUGUÊS DE REUMATOLOGIA

NOVEMBRO 2025 • N.O 1 • EDIÇÃO ESPECIAL XXXIII JORNADAS INTERNACIONAIS IPR

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Archives in Rheumatic and Musculoskeletal diseases

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PROGRAMA

XXXIII JORNADAS INTERNACIONAIS DO INSTITUTO PORTUGUÊS DE REUMATOLOGIA

19, 20 e 21 DE NOVEMBRO

PROGRAMA

2025

Faculdade de Medicina Dentária

LISBOA | PORTUGAL

XXXIII JORNADAS INTERNACIONAIS DO INSTITUTO PORTUGUÊS DE REUMATOLOGIA

19 - 21 NOVEMBRO 2025

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XXXIII JORNADAS INTERNACIONAIS DO INSTITUTO PORTUGUÊS DE REUMATOLOGIA

19 - 21 NOVEMBRO 2025

COMISSÃO CIENTÍFICA

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Dra. Beatriz Mendonça	Dra. Joana Borges	Dr. Paulo Clemente Coelho
Dra. Cândida Silva	Dra. Leonor Reynolds	Dra. Sofia Cláudio
Dra. Cláudia Miguel	Dra. Manuela Micaelo	Dra. Susana Fernandes

GRANDES TEMAS

1. TERAPÊUTICAS DAS DOENÇAS IMUNOMEDIADAS: O PRESENTE E O FUTURO
2. VACINAÇÃO: O DOENTE IMUNOCOMPROMETIDO NO MUNDO GLOBALIZADO
3. OSTEOPOROSE: ESTRATÉGIAS TERAPÊUTICAS
4. DOR CRÓNICA: MÚLTIPLAS CAUSAS, MÚLTIPLAS SOLUÇÕES

19 NOVEMBRO
QUARTA-FEIRA

RHEUMA 2025 STAR COURSE

14H00 - 15H50

4º RHEUMA STAR COURSE (CURSO DE INTERNOS) – 1.ª PARTE

COORDENAÇÃO
Dr. Luis Miranda, IPR
Dra. Cláudia Miguel, IPR

1. **Amiloidose (etiologia e diagnóstico)**
   Dra. Maria Pontes Ferreira, Interna de Formação Específica em Reumatologia, 5º ano

2. **Tratamento do Fenómeno de Raynaud e úlceras digitais na Esclerose Sistémica**
   Dra. Cláudia Oliveira, Interna de Formação Específica em Reumatologia, 5º ano, Unidade Local de Saúde da Região de Aveiro

3. **Síndromes Scleroderma-like**
   Dra. Sofia Ferreira Azevedo, Interna de Formação Específica em Reumatologia, 5º ano, Unidade Local de Saúde da Região de Aveiro

4. **Osteoporosis in Oncology: Current Perspectives**
   Dra. Inês Genrinho, Interna de Formação Específica em Reumatologia, 5º ano, ULS de Viseu Dão Lafões

5. **EULAR recommendations for use of antirheumatic drugs in reproduction, pregnancy, and lactation: 2024 update**
   Dra. Inês Correia Santos, Interna de Formação Específica em Reumatologia, 5º ano, ULS de Viseu Dão Lafões

6. **Diagnóstico e tratamento da Síndrome VEXAS**
   Dr. Fernando Albuquerque, Interno de Formação Específica em Reumatologia, 5º ano, ULS de Coimbra

7. **Guidelines da BSR 2025 sobre tratamento da espondilartrite axial**
   Dr. Roberto Pereira da Costa, Interno de Formação Específica em Reumatologia, 5º ano, ULS de Santa Maria

COFFEE BREAK

16H10 - 18H00

4º RHEUMA STAR COURSE (CURSO DE INTERNOS) – 2.ª PARTE

8. **Abordagem da aftose oral**
   Dra. Carolina Ochôa Matos, Interna de Formação Específica em Reumatologia, 5º ano, ULS de Santa Maria

9. **Síndrome de ativação macrofágica em doenças reumáticas - diagnóstico e abordagem terapêutica**
Dra. Ana Rita Lopes, Interna de Formação Específica em Reumatologia, 5º ano, ULS de Santa Maria

10. **CAR T-cells em Reumatologia**
Dra. Mariana Emília Santos, *Interna de Formação Específica em Reumatologia, 5º ano, ULS de Lisboa Ocidental*

11. **Crioglobulinemia e/ou vasculites mediadas por imuno-complexos**
Dra. Ana Catarina Abreu, *Interna de Formação Específica em Reumatologia, 5º ano, ULS de Almada-Seixal*

12. **Papel dos inibidores da SGLT2 na Reumatologia**
Dra. Margarida Lucas Rocha, *Interna de Formação Específica em Reumatologia, 5º ano, ULS do Algarve*

13. **Síndromes de hipermobilidade/doenças hereditárias do tecido conjuntivo**
Dr. João Daniel Carvalho, *Interno de Formação Específica em Reumatologia, 5º ano, Hospital Dr. Nélio Mendonça - SESARAM, EPERAM*

14. **Manifestações oculares nas doenças reumáticas sistémicas**
Dra. Maria João Cadório, *Interna de Formação Específica em Reumatologia, 4º ano, ULS Coimbra*

20 NOVEMBRO
QUINTA-FEIRA

8H30 — **ABERTURA DO SECRETARIADO**

9H00 - 10H20 — **FLASH REUMATOLÓGICO**

Presidente - Dr. José António Melo Gomes, *Reumatologista, IPR*
Moderador 1 - Dra. Helena Madeira, *Reumatologista, IPR*
Moderador 2 - Dr. Vítor Teixeira, *Reumatologista, ULS do Algarve, Lusíadas Saude Algarve*

1. **Capilaroscopia - O seu valor atual no seguimento das doenças imunomediadas**
Dr. *Paulo Coelho, Reumatologista, IPR*

2. **Abordagem atual dos nódulos reumatóides**
Dra. Ana Leão, Interna de Formação Específica em Reumatologia, 2º ano, IPR

3. **Polineuropatia e doença reumática**
Dra. Leonor Reynolds, Interna de Formação Específica em Reumatologia, 2º ano, IPR

4. **Síndromes auto-inflamatórios – quando suspeitar?**
Dra. Bárbara Lobão, Especialista de Medicina Interna, ULS da Arrábida

5. Sindrome Túnel Cárpico: a ponta do iceberg
Dra. Ana Rita Vieira, Interna de Formação Específica em Reumatologia, 3º ano, IPR

6. Abordagem inicial de alteração das provas hepáticas no doente reumatológico
Dr. Salvador Bodião, Interno de Formação Específica em Gastrenterologia, 3º ano, da ULS de São José

7. Dieta, fome e excessos - efeitos no sistema imunitário
Dr. José Vaz Patto, Reumatologista, IPR

8. Quando a densidade mineral óssea está muito acima do esperado
Dra. Beatriz Santos, Interna de Formação Específica em Reumatologia, 1º ano, IPR //
Dra. Eugénia Simões, Reumatologista, IPR

10H20 – 11H00 **CERIMÓNIA DE ABERTURA**

11H00 – 11H45 *COFFEE BREAK*

DISCUSSÃO DE POSTERS

3 Grupos – Moderadores:

- *Dr. Miguel Sousa, Reumatologista, IPR*
- *Dra. Nathalie Madeira, Reumatologista, Serviço de Reumatologia da Unidade Local de Saúde da Guarda, Faculdade de Ciências da Saúde/Centro Académico das Beiras*
- *Dra. Maria Inês Seixas, Reumatologista, Unidade de Reumatologia, ULS de Viseu Dão-Lafões*

11H45 – 12H15 **CONFERÊNCIA PLENÁRIA – Lipossomas com MTX no tratamento da AR**

Prof. Doutor Artur Cavaco Paulo , Engineering, PhD, CEO, CSO Solfarcos Soluções Farmacêuticas e Cosméticas, Lda, Professor na Universidade do Minho no Dep. Eng. Biológica

Presidentes

- Prof. Doutor Fernando Pimentel Santos, Reumatologista, Nova Medical School, Universidade NOVA Lisboa, Diretor Serviço de Ensino Pré e Pós-Graduado da ULS de Lisboa Ocidental, Presidente da Sociedade Portuguesa de Reumatologia

- Prof.ª Doutora Helena Santos, *Reumatologista, IPR*

12H30 – 13H00 **SIMPÓSIO SATÉLITE AMGEN - Agir hoje para evitar fraturas amanhã**

- Dr. Filipe Araújo (Reumatologia)
- Dra. Madalena Rodrigues (MGF)

13H00 – 14H00 *ALMOÇO*

14H00 – 15H30

MESA REDONDA 1 - Novidades Diagnósticas e Terapêuticas em 15 slides: Terapêutica das doenças imunomediadas – o presente e o futuro

Presidente - Prof. Doutor José Miguel Bernardes, Reumatologista, Assistente Hospitalar graduado de Reumatologia da ULS de São João do Porto, Professor Auxiliar convidado de Reumatologia da Faculdade de Medicina da Universidade do Porto (FMUP)

Moderador 1 - Dra. Cândida Silva, Reumatologista, IPR

Moderador 2 - Dra. Elsa Sousa, Serviço de Reumatologia, Hospital de Santa Maria, Unidade Local de Saúde de Santa Maria, Centro Académico de Medicina de Lisboa, Clínica Universitária de Reumatologia, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa

1. ***Já sabemos responder a tudo sobre a utilização de inibidores da JAK?***
   Dra. Susana Fernandes, Reumatologista, IPR

2. ***O futuro da inibição da IL-17 e da IL-23***
   Prof. Doutor Filipe Barcelos, Reumatologista, IPR

3. ***Novas oportunidades terapêuticas no LES***
   Dra. Maria Rato, Reumatologista, ULS de São João, Porto

4. ***Novas perspetivas da utilização das CAR-T cells e T-cell engagers***
   Dra. Daniela Alves, Serviço de Hematologia e Transplantação de Medula, UL

15H30 – 16H00 *COFFEE BREAK*

16H00 – 17H15

MESA REDONDA 2 – Gestão do doente reumático sob terapêuticas imunomoduladoras

Presidente - Dra. Maria João Salvador, Reumatologista, Assistente Hospitalar Graduada da ULS de Coimbra - HUC Coimbra

Moderador 1 - Dra. Susana Fernandes, Reumatologista, IPR

Moderador 2 - Dra. Ana Catarina Duarte, Reumatologista, Serviço de Reumatologia da Unidade Local de Saúde de Almada-Seixal

1. **Vacinação no mundo globalizado**
   Dra. Daniela Lages, Médica especialista em Doenças Infecciosas, Serviço de Infecciologia, Hospital Garcia de Orta

2. **Rastreios neoplásicos**
   Dr. Tiago Barroso, Assistente Hospitalar de Oncologia Médica, ULS Santa Maria

3. **Rastreio da Tuberculose Latente**
   Dra. Carina Silvestre, ULSO - Torres Vedras
   Comissão Trabalho de Tuberculose - Sociedade Portuguesa de Pneumologia

17H15 – 18H30

CONCURSO DE IMAGENS

Presidente - Dr. Pedro Abreu, Reumatologista, ULS de Castelo Branco, EPE

Moderador 1 - Dr. Paulo Coelho, Reumatologista, IPR

Moderador 2 - Dra. Diana Gonçalves, Reumatologista, ULS Póvoa de Varzim/Vila do Conde

21 NOVEMBRO
SEXTA-FEIRA

09H00 – 10H30

COMUNICAÇÕES LIVRES

Presidente - Dra. Patrícia Pinto, Reumatologista, Assistente Hospitalar Graduada de Reumatologia, Reumatologista Pediátrica, Docente convidada da cadeira de Reumatologia da Faculdade de Medicina Universidade do Porto

Moderador 1 - Dra. Manuela Parente, Reumatologista, IPR

Moderador 2 - Dra. Joana Rodrigues, Reumatologista, Médica Assistente no Serviço de Reumatologia da Unidade Local de Saúde da Cova da Beira, Portugal

1. **Pregnancy outcomes in anti-SSA/SSB positive patients with rheumatic diseases: a retrospective study from a tertiary center**
   Dra. Catarina Rua, Interna de Formação especifica em Reumatologia, 4.º ano, ULS de Gaia e Espinho

2. **Interstitial Lung Disease in Mixed Connective Tissue Disease: A Distinct Clinical Pattern Compared to Systemic Sclerosis and Myositis?**
   Dra. Filipa Marques Costa, Interna de Formação Específica em Reumatologia, 4º ano, ULS de Santa Maria

3. **Fracture incidence and risk factors in peritoneal dialysis patients**
   Dra. Mariana Diz Lopes, Interna de Formação Específica em Reumatologia, 4º ano, ULS de São João

4. **Radiofrequency Echographic Multi Spectrometry (REMS) technology in the evaluation of glucocorticoid-induced osteoporosis in systemic lupus erythematous patients**
   Dra. Mariana Diz Lopes, Interna de Formação Específica em Reumatologia, 4º ano, ULS de São João

5. **Key features of paediatric Sjögren's disease a portuguese cohort overview**
   Dra. Bianca Paulo Correia, Interna de Formação Específica de Reumatologia, 4º ano, ULS de Santa Maria, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa (CAML)

6. **Can salivary gland ultrasound replace minor salivary gland biopsy in primary Sjögren's syndrome? evidence from a real-world cohort**
   Dra. Sara Alves Costa, 3º ano, Interna de Formação Específica em Reumatologia, ULS de Coimbra

7. **Immune-mediated necrotizing myopathy in the portuguese population – a multicentric nationwide study**
   Dra. Anita Cunha, Maria Pontes Ferreira, Serviço de Reumatologia, ULS do Alto Minho, Ponte de Lima

10H30 – 11H15 COFFEE BREAK

DISCUSSÃO DE POSTERS

3 Grupos – Moderadores:

Dra. Dina Medeiros, *Reumatologista, IPR*

Dra. Carina Lopes, *Reumatologista, Assistente Hospitalar de Reumatologia no Hospital de Egas Moniz, ULS de Lisboa Ocidental, Docente da Unidade Curricular do Aparelho Motor – Reumatologia da NOVA Medical School, Universidade Nova de Lisboa*

Dra. Ana Teresa Melo, *Reumatologista, Unidade de Reumatologia, Hospital Santo António dos Capuchos, ULS de S. José*

11H15 – 12H45

MESA REDONDA 3 – Osteoporose: conceitos e estratégias

Presidente – Dr. José Carlos Romeu, Reumatologista, Hospital Nossa Senhora da Arrábida, Setúbal

Moderador 1 – Dra. Eugénia Simões, Reumatologista, IPR

Moderador 2 – Dra. Ana Raposo, Reumatologista, Assistente graduada Reumatologia, Diretor

1. Prevenção primária de fraturas
Prof.ª Doutora Anabela Barcelos, Reumatologista, Diretora do Serviço de Reumatologia da ULS da Região de Aveiro, Professora Auxiliar Convidada do Departamento de Ciências Médicas da Universidade de Aveiro, Investigadora Clínica na Unidade EpiDoC, NOVA Medical School, Universidade NOVA de Lisboa

2. Risco iminente de fratura
Dra. Ana Roxo Ribeiro, Reumatologista, ULS de Braga

3. Posicionamento e utilização das novas terapêuticas
Dra. Sofia Pimenta, Reumatologista, Assistente Hospitalar Graduada do Serviço de Reumatologia da ULS São João, Porto, Assistente convidada da Faculdade de Medicina da Universidade do Porto

12H45 – 14H00 ALMOÇO

14H00 – 15H30

MESA REDONDA 4 – Curso Monotemático Dr. João Figueirinhas
Dor crónica não inflamatória

Presidente – Dr. Luís Maurício Santos, Reumatologista, Hospital do Divino Espírito Santo – de Ponta Delgada

Moderador 1 – Dr. Augusto Faustino, Reumatologista, IPR

Moderador 2 – Dra. Marília Rodrigues, Reumatologista, Serviço de Reumatologia, ULS da Região de Leiria

1. **Novas abordagens farmacológicas da dor**
   Dr. Tiago Beirão, Interno de Formação Especifica em Reumatologia, 5º ano, Unidade Local de Saúde Gaia e Espinho

2. **Radiofrequência, crioneurólise e bloqueio anestésico dos nervos periféricos**
   Dr. Jorge Barbosa, Médico Fisiatra, CIPS (Certified Interventional Pain Sonologist) pelo World Institute of Pain, Assistente Graduado e Coordenador da Unidade Funcional de Reabilitação Músculo-Esquelética e do Estágio de Intervenção Ecoguiada do Serviço de Medicina Física e de Reabilitação (MFR) da ULSLO, Membro da Unidade da Dor da ULSLO, Assistente Convidado de MFR da NOVA Medical School, Editor-Associado da Revista da Sociedade Portuguesa

3. **Procedimentos percutâneos no tratamento da dor lombar**
   Dr. Diogo Roque, Hospital de Santa Maria (ULS Santa Maria, Serviço de Neurocirurgia), Hospital da Luz

15H30 – 16H00 *COFFEE BREAK*

16H00 – 17H30 **MESA REDONDA 5 – Multidisciplinaridade em Reumatologia: para além da articulação**

Presidente - Prof.ª Doutora Ana Rodrigues, Reumatologista, Nova Medical School, Hospital dos Lusíadas de Lisboa

Moderador 1 - Dra. Joana Borges, Reumatologista, IPR

Moderador 2 - Dra. Joana Sousa Neves, Reumatologista, Serviço de Reumatologia, ULS de Braga

1. **Pico monoclonal na eletroforese das proteínas: quando referenciar?**
   Dra. Cátia Gaspar, *Instituto CUF Oncologia-CUF Descobertas*

2. **Alopécia e queda de cabelo: avaliação clínica e tratamento**
   Dr. Rui Oliveira Soares, Trichology Coordinator and Clinical Director CDL. Lisbon, Former General Secretary - Grupo de Tricologia da Sociedade Portuguesa de Dermatologia e Venereologia, Teacher in International Master in Trichology and Hair Transplant, European Hair Research Society Member, International Trichoscopy Society Member, Ukrainian Hair Research Society Honorary Member, Liga Portuguesa Contra o Cancro NRS - Secondary Prevention Coordinator

3. **Risco trombótico nas doenças reumáticas**
   Prof.ª Doutora Maria José Santos, Reumatologista, Serviço de Reumatologia, Hospital Garcia de Orta, Unidade local de saúde de Almada-Seixal, Almada

17H30 – 18H00 **ATRIBUIÇÃO DE PRÉMIOS. ENCERRAMENTO DAS JORNADAS**

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XXXIII JORNADAS INTERNACIONAIS DO INSTITUTO PORTUGUÊS DE REUMATOLOGIA

18, 19 e 20 NOVEMBRO 2026

SAVE THE DATE

2026

SESSÕES DO PROGRAMA

CAPILAROSCOPIA – O SEU VALOR ATUAL NO SEGUIMENTO DAS DOENÇAS IMUNOMEDIADAS

Paulo Clemente Coelho

Instituto Português de Reumatologia

A Capilaroscopia tem o seu principal papel na avaliação diagnóstica do Fenómeno de Raynaud e das doenças imunomediadas com envolvimento microcirculatório importante. Alguns estudos sugerem também que a Capilaroscopia pode ter um papel para avaliar a progressão da doença e a eficácia do tratamento. Os achados anormais do leito capilar podem evoluir com o tempo, refletindo o agravamento do dano microvascular ou o resultado da intervenção terapêutica. Apesar desta evidência de deteção da evolução ser menos consistente em relação ao papel da evidência diagnóstica no uso da Capilaroscopia, alguns estudos sugerem que o acompanhamento regular ajuda a avaliar a progressão dos padrões capilaroscópicos e pode correlacionar-se com o envolvimento de órgãos internos e a resposta ao tratamento.

ABORDAGEM AOS NÓDULOS REUMATÓIDES

Ana Bispo Leão

Instituto Português de Reumatologia

Os Nódulos Reumatóides (NR) constituem a manifestação extra-articular mais frequente na Artrite Reumatóide (AR), observando-se em cerca de 20 a 30% dos doentes. São fatores de risco para o seu aparecimento o tabagismo (principalmente no sexo masculino), maior duração e atividade da doença, AR seropositiva (FR+ e/ou anti-CCP+) e HLA-DRB104:01. Os NR estão ainda associados a maior probabilidade de ocorrência de outras manifestações extra-articulares, gravidade da AR, complicações cardiovasculares e mortalidade.

A apresentação clínica mais frequente constitui a forma subcutânea, com localização preferencial em áreas de pressão ou extensão, como o olecrâneo, dorso dos dedos, tuberosidade isquiática, região sagrada, occipital e tendão de Aquiles. Caracterizam-se como lesões firmes, indolores, de dimensões variáveis entre 0,2 e 5 cm, podendo ser móveis ou aderentes aos planos profundos. São frequentemente assintomáticos, no entanto podem cursar com dor, ulceração, infeção, limitação funcional ou compressão nervosa. O diagnóstico é clínico, sendo a biópsia reservada para casos com apresentação atípica, crescimento rápido ou perante complicações associadas. Quando presentes e histologicamente confirmados, são patognomónicos da Artrite Reumatóide.

Os NR podem também afetar outros órgãos, destacando-se o envolvimento pulmonar, onde se apresentam como solitários/múltiplos, subpleurais, de tamanho variável, até 8 cm. Podem surgir complicações associadas aos mesmos nomeadamente: cavitação, pneumotórax, infeção ou derrame pleural. Outras localizações menos frequentes incluem o coração, o sistema nervoso central (meninges), fígado, rim, olho ou mucosa oral.

O diagnóstico diferencial inclui tofos gotosos, calcinose cutânea, lipoma, fibromas, sarcoidose, tuberculose pulmonar ou lesões neoplásicas.

Destaca-se ainda a Nodulose Acelerada, fenómeno caracterizado pelo aparecimento súbito ou crescimento rápido de NR em doentes sob terapêutica DMARD convencional ou biológica, onde se destaca o Metotrexato, Leflunomida, anti-TNFs e anti-IL6.

A Nodulose Reumatóide corresponde à presença de NR em doentes sem sinovite crónica.

O tratamento dos NR deve ser individualizado, tendo em conta a sintomatologia e relação com DMARDs. Os nódulos assintomáticos não exigem intervenção específica, estando indicada apenas a vigilância clínica.

Nas formas complicadas, pode recorrer-se à infiltração local com glucocorticóides ou excisão cirúrgica. Nos

casos de Nodulose Acelerada, recomenda-se a suspensão do cs/bDMARD associado. O controlo rigoroso da atividade inflamatória da AR é essencial

para prevenir o aparecimento e progressão das manifestações nodulares.

Os NR representam, assim, um marcador clínico e histopatológico de doença mais severa, traduzindo a natureza sistémica da AR e merecendo a sua integração na estratégia terapêutica do doente.

POLINEUROPATIA E DOENÇA REUMÁTICA

Leonor Reynolds de Sousa

Instituto Português de Reumatologia

Introduction: Peripheral polyneuropathies are common disorders with a wide spectrum of etiologies, ranging from metabolic and infectious to immune-mediated causes. In rheumatic diseases, neuropathic involvement is frequent and may arise through diverse mechanisms, including immune-mediated injury, vasculitic processes, mechanical compression, and drug toxicity. Clinical presentation, underlying pathophysiology, and diagnostic approach are often complex and require a multidisciplinary evaluation.

Diagnosis and Classification: A comprehensive clinical assessment remains the cornerstone of diagnosis. Detailed anamnesis should focus on comorbidities, family history, and exposure to potentially neurotoxic medications. Neurological examination should include assessment of sensory and motor function, autonomic symptoms, and search for systemic manifestations as skin lesions, lymphadenopathy, or organomegaly. Nerve conduction studies (NCS) and electromyography (EMG) are essential to confirm neuropathy, determine severity, and differentiate axonal from demyelinating patterns. Laboratory workup - performed stepwise - should screen for metabolic, infectious, and autoimmune causes. Nerve or muscle biopsy may be required in rapidly progressive or atypical cases to confirm vasculitis or infiltrative disease.

Neuropathy Patterns in Rheumatic Diseases: Rheumatic disorders may present with several neuropathic phenotypes. Sjögren's syndrome and systemic lupus erythematosus (SLE) are among the most common causes, followed by rheumatoid arthritis (RA) and systemic vasculitides. Neuropathy may manifest as mononeuritis multiplex, distal symmetric polyneuropathy, small fiber neuropathy, or cranial neuropathies. Vasculitic neuropathy typically results from inflammation of medium or small vessels, producing acute and painful asymmetric deficits that may evolve into diffuse polyneuropathy. Non-vasculitic inflammatory neuropathies, such as chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), may also occur, occasionally triggered by immunomodulatory agents like anti-TNF therapy. In addition, compressive neuropathies (e.g., carpal tunnel, ulnar entrapment) are frequent in RA due to joint deformities and tenosynovitis.

Differential Diagnosis: Conditions that mimic neuropathic pain, such as arthropathy-related deformities, glucocorticoid-induced myopathy, or fibromyalgia with neuropathic features, must be carefully excluded. Infectious diseases with rheumatic manifestations, including Lyme disease and leprosy, can also produce peripheral neuropathy. Coincidental neuropathies, such as those related to diabetes, alcoholism, or nutritional deficiencies, may coexist with rheumatic disease.

Treatment and Prognosis: Management depends on the underlying mechanism. Immunosuppressive therapy, including corticosteroids, cyclophosphamide, or rituximab, remains the mainstay in vasculitic and immune-mediated neuropathies. Discontinuation or substitution of offending drugs may be indicated in iatrogenic cases (e.g., leflunomide, anti-TNF agents). Compressive neuropathies benefit from local interventions. Symptomatic control of neuropathic pain with anticonvulsants, antidepressants, or topical agents improves quality of life. Early recognition and targeted therapy are crucial to prevent irreversible axonal loss and functional disability.

Conclusion: Peripheral neuropathy represents a significant but often underrecognized manifestation of rheumatic diseases. A structured diagnostic approach integrating clinical, electrophysiological, laboratory, and pathological findings is essential to identify the underlying cause. Timely and individualized management can substantially improve outcomes, highlighting the importance of close collaboration between rheumatologists and neurologists in the care of these complex patients.

SÍNDROMES AUTO-INFLAMATÓRIOS – QUANDO SUSPEITAR?

Bárbara Lobão

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As síndromes autoinflamatórias constituem um grupo heterogéneo de doenças caraterizadas por episódios inflamatórios sistémicos recorrentes ou persistentes, sem evidência de causa infeciosa, neoplásica ou autoimune. Resultam de desregulação do sistema imunitário inato e incluem entidades monogénicas, poligénicas e síndromes adquiridas com mutações somáticas. Desde a identificação do gene da febre mediterrânica familiar (FMF) em 1997, mais de 50 genes foram associados a estas patologias, reconhecendo-se cada vez mais apresentações atípicas ou de início tardio na idade adulta.

Esta apresentação pretende uma abordagem prática destas doenças com revisão dos padrões clínicos e laboratoriais que devem levantar suspeita, incluindo inflamação recorrente ou persistente, fenótipo não explicável por infeção ou autoimunidade, refratariedade terapêutica, envolvimento seroso, cutâneo, osteoarticular ou hematológico, e história familiar sugestiva. A classificação por mecanismos fisiopatológicos — inflamassomopatias, interferonopatias, NF-κB-patias e outros mecanismos — é explorada para facilitar o raciocínio clínico. Exemplos incluem FMF, TRAPS, CAPS, défice de mevalonato-quinase, doença de Still, osteomielite multifocal recorrente crónica e a síndrome VEXAS.

Apresenta-se um algoritmo prático de abordagem diagnóstica, integrando exclusão etiológica, caracterização do padrão das crises, testes laboratoriais, imagem e a interpretação de testes genéticos, incluindo a necessidade de considerar mutações somáticas com baixa frequência alélica e, quando indicado, repetição de testes ou estudo em medula óssea.

Salienta-se a importância da identificação precoce destas entidades de forma a prevenir complicações como amiloidose AA e permitir intervenções dirigidas altamente eficazes.

SÍNDROME DO TÚNEL CÁRPICO – A PONTA DO ICEBERG

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Síndrome do Túnel Cárpico (STC) designa a neuropatia do nervo mediano a nível do punho, causada pela compressão crónica a este nível. Trata-se da neuropatia de compressão mais comum. Clinicamente, caracteriza-se pela presença de parestesias ou disestesias, tipicamente de predomínio noturno, no território do nervo mediano (1-3º dedos e face medial do 4ºdedo). Na ausência de tratamento, os sintomas podem progredir para hipostesia e fraqueza muscular.

Aquando da investigação de STC, importa ter em consideração diversos fatores de risco, sendo os mais conhecidos sexo feminino, idade, obesidade, fatores relacionados com o trabalho e gravidez. Contudo, a síndrome pode também estar associada a outras patologias, nomeadamente, reumáticas (artrite reumatoide, outras doenças articulares inflamatórias, tenossinovite e tofos gotosos), ortopédicas (complicação de fratura distais do rádio), neurológicas (polineuropatias, sendo de destacar em Portugal a paramiloidose familiar), endocrinológicas (diabetes, hipotiroidismo e acromegalia). De referir ainda a importância da história familiar, sobretudo nos casos de STC bilateral. Como etiologias mais raras, são ainda de considerar variações anatómicas vasculares, infeções, ou lesões ocupantes de espaço.

Assim, é fundamental realizar uma história clínica detalhada e um exame objetivo completo, para uma melhor abordagem diagnóstica, de modo a esclarecer se se trata de uma STC isolada, ou no contexto de outra doença que beneficiará, em primeira instância, do tratamento da doença de base e reavaliação posterior da STC. No que concerne ao exame objetivo, destacam-se as manobras de Tinel e Phalen. O diagnóstico e a gravidade do quadro são confirmados por eletromiograma (EMG) e, eventualmente, avaliação complementar com ecografia para avaliar a presença de artrite / tenossinovite, ou, mais raramente, ressonância magnética para identificação de alterações vasculares.

Nos casos leves a moderados, poder-se-á optar por tratamento conservador: ferramentas ergonómicas, talas ou corticoesteróides locais. Nos casos severos, identificados no EMG e/ou na ausência de melhoria com medidas conservadoras, poderá ser ponderada cirurgia aberta ou minimamente invasiva. A taxa de sucesso da cirurgia ronda os 75-90%, com complicações inferiores a 1%.

No futuro, seria importante a criação de critérios de diagnóstico e abordagem terapêutica *standard*, de modo a promover uma abordagem terapêutica semelhante entre centros.

ABORDAGEM INICIAL DA ALTERAÇÃO DAS PROVAS HEPÁTICAS NO DOENTE REUMATOLÓGICO

Salvador Bodião

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As alterações das provas hepáticas são frequentes em doentes com doenças reumatológicas e podem resultar do envolvimento sistémico da própria doença, da hepatotoxicidade dos fármacos ou da coexistência de uma hepatopatia primária. Reconhecer estes cenários é essencial para evitar suspensões terapêuticas desnecessárias e identificar precocemente situações potencialmente graves. Até 40% dos doentes apresentam elevações ligeiras e transitórias das enzimas hepáticas, muitas vezes sem doença estrutural.

Entre as causas medicamentosas, o metotrexato e a leflunomida são os agentes mais frequentemente implicados, enquanto biológicos e inibidores de JAK acarretam risco significativo de reativação do vírus da hepatite B, mesmo em infeção resolvida. As hepatopatias autoimunes — hepatite autoimune, colangite biliar primária e colangite esclerosante primária — constituem causas primárias relevantes, sobretudo na síndrome de Sjögren e na esclerodermia limitada. A abordagem deve seguir um algoritmo simples baseado em três eixos: envolvimento sistémico, fármaco e doença hepática primária, com rastreio viral prévio e monitorização regular para garantir a segurança da terapêutica.

DIETA, FOME E EXCESSOS-EFEITOS NO SISTEMA IMUNITÁRIO

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Inicia-se a apresentação com uma abordagem crítica acerca dos trabalhos científicos e dietas propostas, suas metodologias e diferentes vieses, com o olhar numa dieta futura verdadeiramente personalizada.

Serão abordados diferentes aspetos da nutrição determinados pela genética/epigenética nomeadamente a alimentação da grávida, peso à nascença e a amamentação. Informação da "epigenética exógena" através dos miRNAs exógenos (xenomiRNAs) dos alimentos ingeridos.

A estabilização da microbiota intestinal na infância, as suas interações e a dificuldade de a modificar.

Uma chamada de atenção à frequência das dietas que os doentes experimentam antes de obterem dum médico ou nutricionista qualquer orientação.

Entre as diferentes dietas aprofunda-se a dieta mediterrânica, sua definição e resultados em pessoas saudáveis e prevenção e tratamento na AR.

Desenvolvem-se diferentes aspetos do jejum intermitente, aplicado a algumas doenças,

nomeadamente reumáticas e seus mecanismos imunológicos.

Breve referência à importância da crono-nutrição.

Não esquecer o papel dos micronutrientes (ex. selénio e zinco) e Vitaminas (ex A, C e D)

Relevância da pandemia da Obesidade nos países desenvolvidos e suas relações com o sistema imunitário.

QUANDO A DENSIDADE MINERAL ÓSSEA É MUITO ACIMA DO ESPERADO

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A determinação da densidade mineral óssea pela Absorciometria de Raios X de Dupla Energia (DXA) constitui um pilar diagnóstico na osteoporose e osteopenia. Todavia, o achado de alta densidade mineral óssea, frequentemente denominado na literatura por *High Bone density (HBD)* ou *High Bone Mass* (HBM), representa um desafio diagnóstico. Por um lado, não existe um cut off de Z-score e/ou T-score estabelecido. Por outro lado, incorre-se o risco de rotular como "saudável" um indivíduo com risco aumentado de fratura, quer pela condição clínica subjacente, quer por mascarar uma osteoporose/osteopenia. Esta revisão consolida a compreensão atual deste tema, alertando para o espectro etiológico e implicações clínicas inerentes. De forma geral, a etiologia pode ser estratificada em 3 categorias: 1) artefactos mimetizadores que incluem condições degenerativas e inflamatórias (por exemplo, espondilose, DISH, fraturas vertebrais), bem como artefactos extravertebrais (próteses e calcificações vasculares, lítiase); 2) HDB focal, nomeadamente pela Doença de Paget ou lesões osteoblásticas metastáticas de neoplasias primárias (por exemplo, próstata, mama, estômago, colon, pulmão); 3) HDB generalizada que abrange doenças adquiridas (por exemplo, acromegalia, osteodistrofia renal, mielofibrose, mastocitose, excesso de flúor) e formas genéticas, menos frequentemente (por exemplo, mutações em LRP5, CLCN7, etc).

Assim, o trabalho visa notar este achado da HBD como um possível *red flag* para doenças sistémicas sejam metabólicas, neoplásicas ou genéticas. Salienta-se a necessidade de definição de critérios apropriados, bem como de utilizar outros exames complementares na interpretação da densidade mineral óssea. A investigação de HBD, pode fornecer pistas na criação de terapêuticas futuras da osteoporose.

IMPROVED BIOAVAILABILITY AND SPECIFICITY TO TARGET RHEUMATOID ARTHRITIS WITH FOLATE-BASED LIPOSOMES ENCAPSULATING METHOTREXATE (FBL-MTX)

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Keywords: Targeted-liposomes, methotrexate, rheumatoid arthritis, non-clinical studies, clinical trials

Introduction: With a prevalence ranging from 0.3% to 1.1% in North America and Europe, Rheumatoid Arthritis (RA) is the most common form of chronic inflammatory arthritis. Discoveries concerning RA pathogenesis have led to the development of new

agents with specific molecular targets, which have transformed the prognosis for numerous RA patients [1].

Objectives: Over the past 15 years, our aim has been creating an innovative formulation for RA, starting from basic research and translating it into an investigational medicinal product.

Methods: Co-funded by 2 European projects [2,3] that culminated with the foundation of a start-up which is now investing their own funds in bringing a biotechnological methotrexate (MTX)-based treatment to RA patients, our core team has been leading the full cycle of this drug development. From the ideation of a folate-decorated liposome encapsulating MTX and the development of its production method at lab scale, to the mechanism of action, efficacy and toxicological analysis in cell lines and animal models, and overcoming the challenges in the scale-up and setting of the GMP manufacturing process. The first-in-human (FIH) clinical study was performed in 2020/21 and the proof-of-concept (PoC) phase IIa clinical study with RA patients is taking place during the second semester of 2024.

Results and Conclusions: FBL-MTX are pegylated liposomes with less than 150 nm encapsulating MTX and bearing folate at the surface, anchored in the lipid bi-layer through the SP-DS3 peptide which targets activated macrophages on RA [4].

In the *in vivo* studies performed in mice, FBL-MTX showed to be more effective than MTX in preventing and alleviating the arthritic signs and symptoms with adequate safety [5]. Full toxicological GLP studies in rats and dogs showed that repeated doses given by IV injection every two-weeks were safe and well tolerated up to 2.5 mg (human equivalent dose).

The effect of FBL-MTX in humans has been evaluated in a placebo-controlled FIH study including a total of 32 healthy subjects. The biodistribution profile was determined following a single IV administration of FBL-MTX up to 2.5 mg, showing $t_{1/2}$ and t_{last} similar to what is found for MTX SC injection in standard treatments (15-20 mg). FBL-MTX was considered safe and well tolerated.

The local tolerance of FBL-MTX to a SC injection and its biodistribution was assessed with repeated injections of 2.5 mg in minipigs. The pharmacokinetics profile was typical of a SC injection, with reduction of the C_{max} and $t_{1/2}$ compared to an equivalent IV injection, though with longer t_{last}.

These results are very promising for FBL-MTX being an alternative to MTX SC injection, with once or twice monthly administrations. This scheme is being tested in the PoC phase 2a clinical ending this Winter.

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COMUNICAÇÕES LIVRES

CL 01 - PREGNANCY OUTCOMES IN ANTI-SSA/SSB POSITIVE PATIENTS WITH RHEUMATIC DISEASES: A RETROSPECTIVE STUDY FROM A TERTIARY CENTER

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Purpose: Anti-Ro/SSA and anti-La/SSB antibodies are associated with neonatal lupus, congenital heart block, and other pregnancy complications, making close monitoring essential in patients who are positive for these antibodies.

Objectives: To evaluate maternal, fetal, and pregnancy outcomes in anti-SSA/SSB-positive patients with autoimmune rheumatic diseases (RMDs) and assess the impact of close multidisciplinary care.

Methods: A retrospective analysis was conducted on 94 pregnancies in 85 patients with RMDs between 2021 and 2024. Patients were grouped based on anti-SSA/SSB antibody status (9 anti-SSA/SSB-positive and 85 negative, determined via ELISA). Diagnoses included rheumatoid arthritis, psoriatic arthritis, axial spondyloarthropathies, systemic lupus erythematosus (SLE), primary Sjögren's syndrome (pSS), mixed and undifferentiated connective tissue diseases, Behçet's disease, and juvenile idiopathic arthritis. Outcomes were assessed using Fisher's exact, chi-square, and t-tests for categorical and metric variables.

Results: Anti-SSA/SSB-positive pregnancieCs were associated with higher rates of SLE and/or pSS (66.7% vs. 17.6%; p = 0.002), hypertension (22.2% vs. 2.40%, p=0.046) and increased hydroxychloroquine use (88.9% vs. 24.7%; p < 0.001). Emergency cesarean section rates were significantly higher in this group (57.1% vs. 13.8%; p = 0.014). Fetal birth weight was slightly higher among anti-SSA/SSB-positive pregnancies (3171 ± 181.5 g vs. 3045 ± 578.2 g; p = 0.038), but no significant differences were found in preterm births, composite maternal complications, or fetal/neonatal outcomes. No neonatal lupus or congenital heart block were recorded.

Conclusion: Despite higher rates of SLE and pSS and increased cesarean sections in anti-SSA/SSB-positive pregnancies, no significant differences in major maternal or fetal outcomes were observed. These findings suggest that close multidisciplinary care may mitigate risks. Larger cohort studies are needed to validate these results and guide care strategies.

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TABLE 1 – BASELINE CHARACTERISTICS AND CLINICAL OUTCOMES OF PATIENTS WITH POSITIVE AND NEGATIVE SSA/SSB AUTOANTIBODIES

VARIABLE	Negative anti-SSA/SSB autoantibodies (n=85)	Positive anti-SSA/SSB autoantibodies (n=9)	p-value
BASELINE VARIABLES			
AGE OF CONCEPTION (YEARS, MEAN ± SD)	32.8±5,05	33.7±6.12	0.260
MATERNAL AGE > 35 YEARS	27.1%	44.4%	0.273
PRESENCE OF LÚPUS AND/OR PSS (%)	17.6%	66.7%	**0.002***
ACTIVE DISEASE IN LAST 12 MONTHS (%)	38.1%	55,6%	0.475
OBESITY (%)	26.5%	50.0%	0.564
PREVIOUS PREGNANCY LOSS (%)	23.5%	22.2%	0.647
PRECONCEPTION APPOINTMENT (%)	69.5%	55.6%	0.459
ACTIVE SMOKER (%)	8.30%	22.2%	0.209
DIABETES (%)	0.00%	0.00%	N/A
THYROID DISEASE (%)	8.30%	11.1%	0.572
HYPERTENSION (%)	2.40%	22.2%	**0.046***
CHRONIC KIDNEY DISEASE (%)	3.60%	0.00%	0.724
CORTICOSTEROID USE (%)	35.3%	44.4%	0.718
BDMARD USE (%)	35.3%	11.1%	0.263
CDMARD USE (%)	25.9%	22.2%	0.585
HYDROXYCHLOROQUINE (%)	24.7%	88.9%	**<0.001***
PREGNANCY OUTCOMES			
COMPOSITE ENDPOINT	21.3%	57.1%	0.057

PREGNANCY LOSS < 20 WEEKS (%)	6.10%	22.2%	0.141
PREGNANCY DURATION (WEEKS, MEAN ± SD)	37.5±4.95	38.69±1.01	0.394
EMERGENCY C-SECTION (%)	13.8%	57.1%	**0.014***
FETAL AND NEONATAL OUTCOMES			
COMPOSITE ENDPOINT	26.1%	14.3%	0.672
MEAN WEIGHT AT BIRTH (GRAMS, MEAN ± SD)	3045±578.2	3171±181.5	**0.038***
LOW BIRTH WEIGHT < 2500G (%)	10.9%	0.00%	0.467
FETAL GROWTH RESTRICTION (%)	10.1%	12.5%	0.603
PRE-TERM BIRTH < 37 WEEKS (%)	**18.9%**	**0.00%**	**0.345**
ICU ADMISSION AFTER BIRTH (%)	4.50%	0.00%	0.739
MATERNAL OUTCOMES			
COMPOSITE ENDPOINT	23.3%	37.5%	0.401
RMD FLARE (%)	10.4%	25.0%	0.237
GESTATIONAL DIABETES (%)	6.70%	12.5%	0.467
GESTATIONAL HYPERTENSION (%)	6.80%	0.00%	0.590
PRE-ECLAMPSIA (%)	8.10%	12.5%	0.527
ANTEPARTUM HAEMORRHAGE (%)	4.20%	0.00%	0.723
COMPOSITE ENDPOINT (MATERNAL, PREGNANCY AND/OR FETAL/NEONATAL OUTCOMES)	54.1%	71.4%	0.454

BDMARD - BIOLOGIC DISEASE-MODIFYING ANTIRHEUMATIC DRUG; CDMARD - CONVENTIONAL DISEASE-MODIFYING ANTIRHEUMATIC DRUG; C-SECTION - CESAREAN SECTION; FGR - FETAL GROWTH RESTRICTION; PSS - PRIMARY SJÖGREN'S SYNDROME; RMD - RHEUMATIC DISEASES; SLE - SYSTEMIC LUPUS ERYTHEMATOSUS; SD - STANDARD DEVIATION; UCI - INTENSIVE CARE UNIT

CL 02 - INTERSTITIAL LUNG DISEASE IN MIXED CONNECTIVE TISSUE DISEASE: A DISTINCT CLINICAL PATTERN COMPARED TO SYSTEMIC SCLEROSIS AND MYOSITIS?

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Introduction: Mixed connective tissue disease (MCTD) shares overlapping features with systemic sclerosis (SSc) and idiopathic inflammatory myopathies (IIM) with a proportion of patients presenting with interstitial lung disease (ILD). However, there are limited comparative data regarding ILD severity, progression patterns, and outcomes between each of these entities.

Objectives: We aimed to assess whether the patients with MCTD-associated interstitial lung disease (MCTD-ILD) have a higher proportion of severe/progressive lung disease during a 4-year (±12 months) follow-up period, when compared to patients with SSc-ILD and myositis-ILD.

Methods: We retrospectively included patients diagnosed with MCTD, SSc, IIM and associated ILD on chest computed tomography (CT) scans with a follow-up of 4 years (±12 months). All patients with MCTD-ILD (n=13) were matched with SSc-ILD and IIM-ILD in a 1:1:1 ratio based on age and disease duration. The primary outcome was the proportion of patients with at least one period of ILD progression, defined as an annual ≥10% relative decline in forced vital capacity (FVC). Secondary analyses included annual assessments of FVC, diffusing capacity of carbon monoxide (DLCO) and mortality. Differences between groups were assessed using chi-square, Fisher's exact, Mann-Whitney or ANOVA tests as appropriate. We considered definite associations when $p<0.05$.

Results: Thirty-nine patients were included, with 33 (84.6%) being female. Mean age at ILD diagnosis was 47.6 ± 2.6 years and median time from initial disease diagnosis to ILD diagnosis was 12 months (IQR 30). The most frequent IIM subtype was antisynthetase syndrome (n=7) followed by dermatomyositis (n=6), of which 3 were positive for anti-MDA5 and the remaining for anti-SAE, anti-EJ and anti-TIF1γ respectively. The extent of lung involvement on CT scan was not significantly different across the three groups. Most patients (n=28, 72%) exhibited a non-specific interstitial pneumonia pattern (NSIP)

However, SSc-ILD patients presented significantly more frequently with usual interstitial pneumonia (UIP) than MCTD-ILD patients (n=4, 30.8% vs. n=0, 0%, $p<0.007$). The extent of ILD on CT scan was not significantly different between groups. During the follow-up period, SSc-ILD patients showed a higher proportion of individuals with at least one annual ≥10% relative decline in FVC, approaching statistical significance (n=0 vs. n=5, $p=0.054$) when compared to MCTD-ILD patients. Conversely, IIM-ILD patients were more likely to experience a relative decline in FVC of >10% ($p=0.026$) within the first year of follow-up when compared with MCTD-ILD patients. At 3 years of follow-up, the mean relative decline in DLCO was significantly greater in SSc-ILD patients when compared to MCTD-ILD patients (mean difference of 24.7%, $p<0.001$). No other significant differences were found between groups in FVC or DLCO decline at annual follow-up assessments. No deaths were recorded during follow-up.

Conclusion: MCTD-ILD apparently has a more favorable clinical presentation than SSc-ILD or IIM- ILD. IIM-ILD and SSc-ILD demonstrated a more rapid decline in pulmonary function, particularly in the first years of

follow-up. These findings highlight distinct ILD progression and underscore the importance of disease-specific monitoring strategies. However, larger, prospective studies are needed to confirm these observations.

Table 1 – Demographics and clinical features at baseline

	All (n=39)	MCTD (n=13)	IIM (n=13)	SSc (n=13)	p value
Gender, n (%)					0.541
Female	33 (84.6)	12 (92.3)	10 (76.9)	11 (84.6)	
Male	6 (15.4)	1 (8.3)	3 (23.1)	2 (15.4)	
Age at ILD diagnosis*, mean ± SD	47.6±2.6	45.7±4.2	45.11±4.1	51.6±5.0	0.380
Time to ILD diagnosis (months)*, median (IQR)	12.0(30.0)	7.0(22.0)	2.0(51.0)	25.0(64.0)	0.308
Smoker*, n (%)					0.339
Yes	8(20.5)	1(8.0)	3(23.0)	4(30.8)	
No	22(56.4)	8(61.5)	8(61.5)	6(46.2)	
Extension of lung involvement at baseline, n (%)*					0.091
<10%	11 (28.2)	7 (53.8)	2 (15.4)	2 (15.4)	
10-20%	11 (28.2)	4 (30.8)	3 (23.1)	4 (30.8)	
>20%	16 (41.0)	2 (15.4)	7 (53.8)	7 (53.8)	
Pattern of involvement at baseline, n (%)					<0.007
NSIP	28 (72.0)	9 (69.2)	10 (76.9)	9 (69.2)	
UIP	4 (10.2)	0	0	4 (30.8)	
LIP	1 (2.6)	1 (7.8)	0	0	
OP	3 (7.7)	0	3 (23.1)	0	
BO	2 (5.1)	2 (15.4)	0	0	
IPF	1 (2.6)	1 (7.8)	0	0	
% FVC at baseline, mean ± SD	76.6±3.9	76.8±7.7	75.3±6.3	77.5±6.9	0.958
% DLCOc SB at baseline*, mean ± SD	59.0±2.8	64.1±5.8	57.4±4.3	55.8±4.6	0.748

*2 missing values for DLCO; *9 missing values for smoking habits; *1 missing value for the extension of lung involvement at baseline.
IQR – interquartile range; SD – standard deviation; NSIP – nonspecific interstitial pneumonia; UIP – usual interstitial pneumonia; LIP – lymphoid interstitial pneumonia; OP – organizing pneumonia; BO –bronchiolitis obliterans; IPF – interstitial pulmonary fibrosis.

Table 2 – Proportion of patients with decreased FVC and DLCO in each subgroup at yearly follow-up

	MCTD (n)	IIM (n)	p value	MCTD (n)	SSc (n)	p value
Relative decrease of FVC >10% at 1-year from baseline (±12months)	0	4	**0.026**	0	**2**	**0.505**
Relative decrease of FVC >10% between year 1 and 2 of follow up (±12months)	0	0	*	0	3	0.505
Relative decrease of FVC >10% between year 2 and 3 of follow up (±12months)	0	1	1.000	0	1	1.000
Relative decrease of FVC >10% between year 3 and 4 of follow up (±12months)	0	0	*	0	2	0.467
Absolute decrease of FVC >10% at 1-year from baseline (±12months)	0	2	0.200	0	1	1.000
Absolute decrease of FVC >10% between year 1 and 2 of follow up (±12months)	0	0	*	0	2	0.524
Absolute decrease of FVC >10% between year 2 and 3 of follow up (±12months)	0	0	*	0	1	1.000
Absolute decrease of FVC >10% between year 3 and 4 of follow up (±12months)	0	0	*	0	2	1.000
Absolute decrease of FVC >5% at 1-year from baseline (±12months)	3	1	0.569	3	6	1.000
Absolute decrease of FVC >5% between year 1 and 2 of follow up (±12months)	2	1	1.000	2	3	1.000
Absolute decrease of FVC >5% between year 2 and 3 of follow up (±12months)	0	1	1.000	0	2	1.000
Absolute decrease of FVC >5% between year 3 and 4 of follow up (±12months)	0	2	1.000	0	0	*
Relative decrease of FVC >5% at 1-year from baseline (±12months)	3	1	0.569	3	6	1.000
Relative decrease of FVC >5% between year 1 and 2 of follow up (±12months)	2	1	1.000	2	3	1.000
Relative decrease of FVC >5% between year 2 and 3 of follow up (±12months)	1	1	1.000	1	2	1.000
Relative decrease of FVC >5% between year 3 and 4 of follow up (±12months)	0	2	1.000	0	0	*
Relative decrease of DLCO>15% at 1-year from baseline (±12months)	1	0	0.467	1	3	1.000
Relative decrease of DLCO>15% between year 1 and 2 of follow up (±12months)	0	0	*	0	4	0.231
Relative decrease of DLCO>15% between year 2 and 3 of follow up (±12months)	1	2	1.000	1	1	1.000
Relative decrease of DLCO>15%between year 3 and 4 of follow up (±12months)	0	0	*	0	1	1.000
Absolute decrease of DLCO>15% at 1-year from baseline (±12months)	1	0	1.000	1	2	1.000
Absolute decrease of DLCO>15% between year 1 and 2 of follow up (±12months)	0	0	*	0	0	*
Absolute decrease of DLCO>15% between year 2 and 3 of follow up (±12months)	1	1	1.000	1	0	0.250
Absolute decrease of DLCO>15%between year 3 and 4 of follow up (±12months)	0	0	*	0	0	*

*Nule value, no statistical analysis was performed.

CL 03 - FRACTURE INCIDENCE AND RISK FACTORS IN PERITONEAL DIALYSIS PATIENTS

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Introduction: Predicting fracture risk in chronic kidney disease (CKD) patients is still a challenge in clinical practice. Particularly in peritoneal dialysis (PD), there is scarce evidence about fracture incidence and risk.

Objectives: The aim of this study was to evaluate the incidence of fractures in a cohort of PD patients and its association with risk factors.

Materials and Methods: This was an observational, retrospective, single-center study of a cohort that received PD for a period ≥ 12 months. Patients were excluded if they received hemodialysis and/or were submitted to renal transplantation before the period of PD. Data from yearly blood tests and clinical evident fractures were collected. FRAX was calculated with the *web-based* FRAX tool (portuguese version) including CKD as secondary osteoporosis. Radiographies of the thoracic and/or lumbar spine were evaluated for vertebral fractures.

Results: A total of 184 patients were included with a median PD vintage of 43(26-66.8) months (table 1).

Mean age at the beginning of PD was 52.7 (14.3) years and 51.1% (n=94) were males. Twenty-two (12%) fractures were identified, representing an incidence rate of 33/1000 patient-year. Patients with fractures during follow-up were older (p<0.001), had longer PD vintage (p=0.021), had a lower body mass index (BMI) (p=0.009), and more incidence of previous fractures (p=0.007). Additionally, they had lower (but within the normal range) phosphorus levels (p<0.001) and lower PTH levels (p=0.019), with more patients with parathyroid hormone (PTH) levels ≤150pg/mL (p=0.040). The vascular calcification score (VCS, Adragao) was higher in patients with fractures (p=0.007). During follow-up, patients with fractures had a higher death rate (p=0.022).

FRAX score was significantly higher in the group with fractures, with 4 (26.7%) of the patients with a high fracture risk calculated by FRAX (p=0.040). Area under the curve for major fracture risk was 0.755 (95% CI 0.651-0.858) and 0.734 (95% CI 0.622-0.846) for hip fracture risk.

When adjusting for the existence of previous fractures (the factor with most impact in the relevant outcome), older age, time on PD, lower BMI, lower PTH, and higher FRAX score independently predicted the occurrence of fractures (table 2). VCS also increased the odds of sustaining a fracture, but this did not achieve significance in the multivariate analysis.

Discussion: In this young and equally sex-distributed cohort of PD patients, fracture incidence was high, and its risk increased with age, PD vintage and PTH levels ≤150pg/mL. FRAX had a good performance in predicting fractures in these patients

Table 1- Characteristics of the groups with and without fractures during follow-up. Data are reported as mean (SD) for normally distributed variables, median (IQR) for non-normally distributed variables, or percentage for categorical variables. P-values were calculated using Mann-Whitney U test for continuous variables and Chi-square with Fisher exact test for categorical variables.

	Fractures during follow-up **n=22 (12%)**	**No fractures during follow-up** **N= 162 (88%)**	**p-value**
Age (years) at the beginning of PD, mean (SD)	60.5 (14.5)	51.7 (50.5)	<0.001*
PD vintage (months), median (IQR)	65.5 (43.3-80.8)	40 (25-63)	0.021*
Gender Female, n (%) Male, n (%)	 13 (59.1) 9 (40.9)	 77 (48.7) 81 (51.3)	0.093
Current smoker, n (%)	0	10 (6.3)	0.691
Alcohol consumption, n (%)	1 (4.5)	10 (6.3)	0.271
Diabetes Mellitus, n (%)	7 (31.8)	31 (19.6)	0.118
Hypertension, n (%)	18 (81.8)	138 (87.3)	0.717
Dyslipidemia, n (%)	16 (72.7)	89 (56.3)	0.341
Body mass Index, median (IQR)	22.5 (10-24.7)	24.8 (21.9-27.8)	0.009*
Fracture before PD, n (%)	5 (22.7)	6 (3.8)	0.007*
CCT during PD period, n (%)	4 (18.2)	10 (6.3)	0.097
FRAX® (without BMD), median (IQR) Major osteoporotic Hip Fracture	 7.5 (2.5-9) 2.4 (0.5-3)	 1.9 (1.4-3.8) 0.3 (0.1-1.2)	<0.001* <0.001*
High-Fracture Risk with FRAX® (without BMD), n (%)	4 (26.7)	9 (6.3)	0.040*
Biochemistry, median (IQR)			
Albumin	37.0 (32.7-39.8)	38 (35.6-40.3)	0.523

Calcium, mg/dL	4.4 (4.3-4.7)	4.5 (4.3-4.7)	0.386
Phosphorus, mg/dL	4.1 (3.8-4.8)	4.9 (4.4-5.4)	<0.001*
Magnesium	1.7 (1.6-1.8)	1.7 (1.6-1.8)	0.129
Alkaline Phosphatase, U/L	114 (84.1-158.5)	96.8 (75.5-122.5)	0.0.91
PTH, pg/mL	330.8 (236.8-389.5)	429.8 (325.7-553.2)	0.019*
Hemoglobin, g/dL	11.7 (10.9-12.3)	10.9 (10-12)	0.027*
Platelets	213 (179.3-235)	221.3 (186.3-260.1)	0.313
C-reactive protein, mg/L	7.3 (2.5-14.6)	3.8 (1.5-9)	0.413
HbA1c,	5.8 (5.7-7.5)	5.6 (5.3-6.5)	0.182
PTH category, n (%) **≤ 150 pg/mL** **150-300 pg/mL** **>300 pg/mL**	 3 (13.6) 6 (27.3) 13 (59.1)	 5 (3.1) 29 (18) 127 (78.9)	0.038*
Vascular calcification score (Adragao Score), median (IQR)	2 (0-4)	0 (0-2)	0.007*
Death, n (%)	8 (36.4)	34 (21.5)	0.022*

Footnote: *statistically significant, p<0.05;

PD: peritoneal dialysis; SD: standard deviation; IQR: interquartile range; CCT: corticosteroid; PTH: parathyroid hormone; VCS: vascular calcification score; FRAX: Fracture risk assessment; BMD: bone mineral density

	Univariate logistic regression				Multivariate logistic regression			
Parameters	OR	OR lower 95% CI	OR upper 95% CI	p-value	OR	OR lower 95% CI	OR upper 95% CI	p-value
Age	1.047	1.012	1.084	0.009*	1.038	1.002	1.076	0.038*
PD vintage (months)	1.013	1.001	1.026	0.034*	1.016	1.004	1.029	0.011*
BMI	0.928	0.876	0.980	0.008*	0.925	0.874	0.979	0.007*
Previous fracture	7.598	2.095	27.551	0.002*	--	--	--	--
PTH	0.996	0.993	0.999	0.010*	0.997	0.994	1.000	0.036*
PTH (≤150 vs >300pg/mL)	5.862	1.255	27.368	0.024*	5.075	1.017	25.327	0.048*
Phosphorus	0.451	0.254	0.804	0.007*	0.501	0.279	0.909	0.023*
VCS	1.186	1.006	1.397	0.042*	1.179	0.996	1.397	0.056
VCS (≥3)	2.512	0.993	6.358	0.052*	2.148	0.814	5.671	0.123
FRAX without BMD for major fracture	1.001	1.000	1.002	0.019*	0.019	1.001	1.003	0.012*
FRAX without BMD for hip fracture	1.001	1.000	1.002	0.006*	1.002	1.001	1.003	0.004*

Table 2 – Regression models for outcome variable (Fractures, n=22)

Footnote: *statistically significant, p<0.05; **adjusted for previous fracture

PD: peritoneal dialysis; PTH: parathyroid hormone; VCS: vascular calcification score; FRAX: Fracture risk assessment; BMD: bone mineral density; OR: odds ratio; CI: confidence interval

CL 04 - RADIOFREQUENCY ECHOGRAPHIC MULTI SPECTROMETRY (REMS) TECHNOLOGY IN THE EVALUATION OF GLUCOCORTICOID-INDUCED OSTEOPOROSIS IN SYSTEMIC LUPUS ERYTHEMATOUS PATIENTS

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Introduction: Patients with Systemic Lupus Erythematosus (SLE) have an increased risk of osteoporosis, particularly those undergoing glucocorticoid (GC) therapy. Dual-energy X-ray absorptiometry (DXA) remains the gold standard for bone mineral density (BMD) assessment, while Radiofrequency Echographic Multi Spectrometry (REMS) is emerging as a radiation-free alternative.

Objectives: To assess the diagnostic agreement between REMS and DXA for BMD in patients with SLE.

Materials and Methods: This cross-sectional observational study included 106 SLE patients referred for DXA evaluation that underwent BMD assessment at the lumbar spine (LS), femoral neck (FN), and total hip (TH) using both DXA and REMS. We compared BMD and T-scores between methods and analyzed diagnostic agreement using linear regression, Cohen's kappa and Bland-Altman analysis. Receiver operator characteristic (ROC) curves evaluated the ability of REMS and DXA to discriminate fracture status.

Results: A total of 106 patients with SLE were included, mostly women (88.7%) and with a mean age of 53.6±13.9 years old. Forty-four patients were receiving GCs, the mean life-time cumulative dose was 8.5 g pred eq (IQR 2.4-18.2) and the mean cumulative duration of use was 71 (IQR 17.3-180) months. Fifteen (14.2%) patients had previous vertebral fractures and 36 (34.0%) had sustained previous non-vertebral fractures.

BMD measurements at all sites were significantly lower with REMS when compared with DXA: FN (0.711±0.127 vs 0.858±0.143, p<0.001), LS (0.891±0.184 vs 1.086±0.172, p<0.001) and TH (0.836±0.123 vs 0.911±0.1421, p<0.001). In SLE patients currently on GC, we found similar differences in REMS and DXA BMD levels.

Table 1 shows the agreement and association metrics between REMS and DXA. In the entire cohort, the correlations between the T-scores from DXA and REMS were significant but moderate. In the group of GC-treated patients, we similarly found statistically significant but weak to modest correlations between T-scores derived from DXA and REMS. The Bland–Altman plots obtained to evaluate the differences between BMD values measured with DXA and REMS showed no bias (Figure 1).

The Cohen's κ for osteoporosis diagnosis between REMS T-score and DXA T-score was 0.229 (95% CI -0.028, 0.486) at the LS, 0.301 (95% CI 0.044, 0.558) at FN and 0.311 (0.083, 0.539) at any site, representing a slight to fair agreement between the two methods. In the GC treated group, the Cohen's κ correlation coefficients for the diagnosis of osteoporosis reflected a moderate agreement.

ROC curves in all patients demonstrated that REMS exhibited similar discriminative ability compared to DXA across all skeletal sites evaluated. When a clinical predictor model based on age, sex, and BMI was included, its standalone AUC was 0.735 (95% CI 0.582-0.889) at the FN and 0.729 (95% CI 0.574-0.884) at the LS, comparable to REMS and DXA (Figure 2). Given the comparable AUC values observed between REMS, DXA, and the clinical predictor model (age, sex, and BMI) for identifying fractured patients, we modeled predicted BMD using age, sex, and BMI as predictors, and compared with the observed BMD values from REMS and DXA at each skeletal site. The r^2 values for REMS

were higher at all sites: LS (r^2=0.569), FN (r^2=0.696), and TH (r^2=0.744), compared to the corresponding r^2 values for DXA (0.208, 0.489, and 0.429, respectively).

Discussion: In conclusion, we found only a moderate agreement between REMS and DXA in osteoporosis classification, and REMS consistently underestimated BMD values. REMS measurements showed stronger associations with age, sex, and BMI than DXA. While this could indicate better alignment of REMS with clinical risk factors, it also suggests that REMS estimates may be more dependent on anthropometric characteristics and may warrant further calibration or adjustment in specific populations. Further longitudinal studies are warranted to validate its predictive value and clinical utility.

Table 1 - Association and agreement between radiofrequency echographic multispectrometry (REMS) and dual x-ray absorptiometry (DXA) in the overall SLE population and glucocorticoid (GC) treated group; * represents p<0.05

	GC treated (n=44)			All patients (n=106)		
Anatomical site	Lumbar Spine	Femoral Neck	Total Hip	Lumbar Spine	Femoral Neck	Total Hip
Valid cases (n)	34	35	34	79	82	78
Sensitivity° (%)	33.3	60.0	0.0	33.3	45.5	40.0
Specificity° (%)	91.4	94.9	94.6	90.4	89.5	96.6
K	0.239	0.549*	-0.036	0.229	0.301*	0.366
R	0.40*	0.41*	0.50*	0.54*	0.50*	0.61*
r^2	0.16*	0.17*	0.25*	0.29*	0.25*	0.37*
Regression line slope	0.40	0.39	0.44	0.42	0.34	0.65
SEE (g/cm^2)	0.190	0.127	0.132	0.157	0.118	0.109
Average differences (mean±1.96 SD, g/cm^2)	0.171±0.211	-0.128±0.165	-0.066±0.152	-0.197±0.192	-0.146±0.136	-0.078±0.165
	All sites			All sites		
Diagnostic Classification, n (%)	Normal	Osteopenia	Osteoporosis	Normal	Osteopenia	Osteoporosis
REMS	18 (40.9)	20 (45.5)	6 (13.6)	39 (36.8)	48 (45.3)	19 (17.9)
DXA	14 (31.8)	16 (36.3)	12 (27.3)	36 (34.0)	50 (47.2)	20 (18.9)

°for classification of osteoporosis REMS vs DXA T-scores

Fig.1 Bland-Altman plots comparing the radiofrequency echographic multispectrometry (REMS) and dual x-ray absorptiometry (DXA) bone mineral density (BMD) measurements in all patients (a) and in the GC-treated group (b)

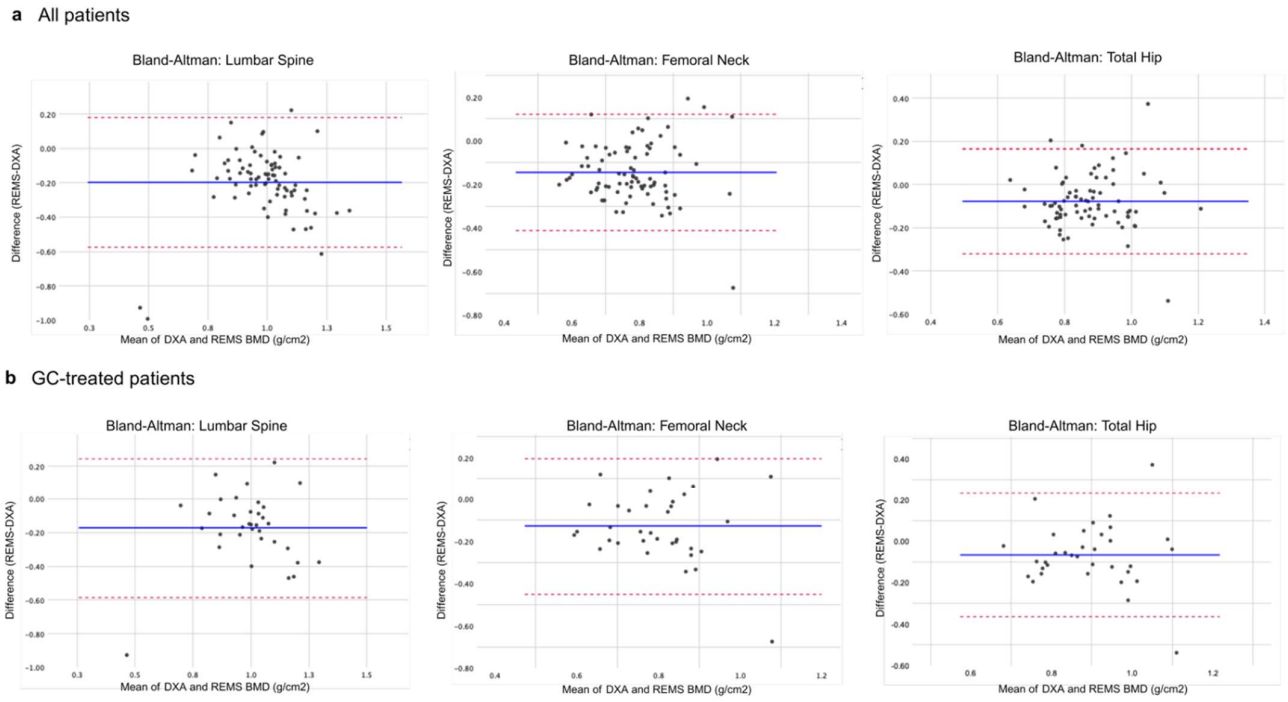
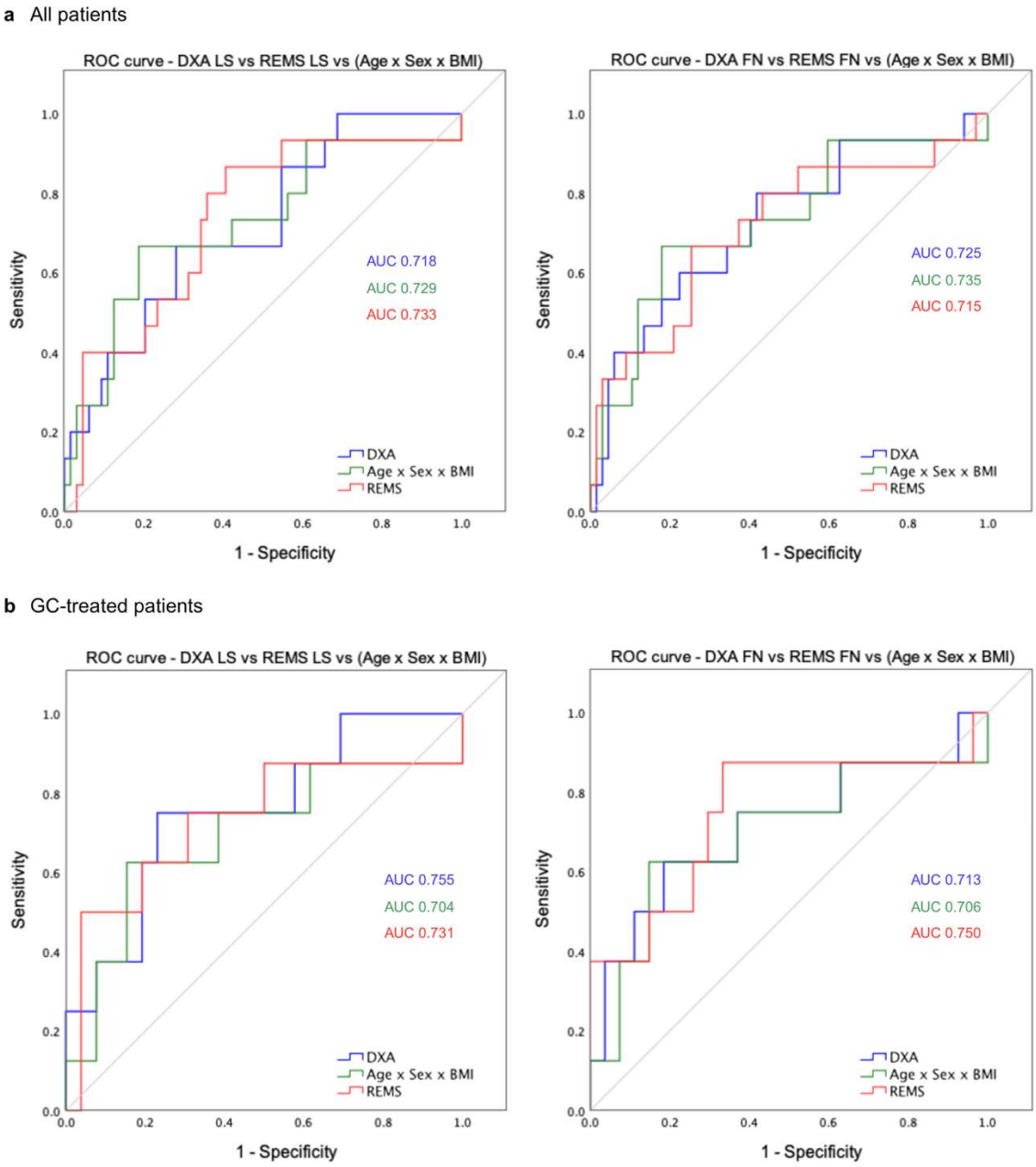


Fig.2 Receiver operator characteristic (ROC) curves comparing the diagnostic performance of dual x-ray absorptiometry (DXA) and radiofrequency echographic multispectrometry (REMS) and the clinical predictor model (age x sex x body mass index) in identifying morphometric vertebral fractures in all patients (a) and in glucocorticoid (GC) treated patients (b)



CL 05 – KEY FEATURES OF PAEDIATRIC SJÖGREN'S DISEASE: A PORTUGUESE COHORT OVERVIEW

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Introduction: Paediatric Sjögren's disease (pSjD) is a rare autoimmune condition presenting with diverse systemic manifestations, which often lead to diagnostic delays and underrecognition. Its identification and classification are challenging due to the absence of paediatric-specific criteria, often requiring reliance on clinical expertise and adult guidelines.

Objectives: To describe the clinical and laboratory features of paediatric SjD across the disease course, highlighting the need for better recognition and earlier diagnosis in clinical practice.

Methods: We conducted a retrospective study using data from patients followed in the Paediatric Rheumatology Units of two Portuguese university hospital centres, between January 2014 and March 2025. All patients were younger than 18 years at disease onset. Data were collected from electronic medical records, including clinical history, diagnostic investigations, and disease activity scores at the last recorded visit.

Results: A total of 16 patients with pSjD were included, of whom 81.3% were female, with a mean disease onset age of 12.9 ± 3.8 years (range 3–17) and a mean disease duration of 6.2 ± 4.5 years at the last visit (mean follow-up: 4.9 ± 4.8 years). Fifteen patients (93.8%) had primary SjD, and one had SjD associated with systemic lupus erythematosus (SLE).

Recurrent parotid swelling was observed in 43.8% of patients at presentation, and 18.8% had persistent glandular enlargement. During follow-up, 81.3% reported dry mouth and 75.0% dry eyes. Other common manifestations included lymphadenopathy (68.8%), arthritis/arthralgia 2 (43.8%), constitutional symptoms (37.5%), hematologic involvement (37.5%), and Raynaud's phenomenon (18.8%). Two patients (12.5%) had central nervous system manifestations (seizures and psychiatric symptoms).

Hypergammaglobulinemia was present in 75%, with a median IgG of 2256 mg/dl (IQR 1272). All patients were ANA-positive, 93.8% had SSA/Ro and 56% had SSB/La antibodies, and 57.1% were rheumatoid factor (RF)-positive. Hypocomplementemia (low C3) was present in 31.3% of patients.

In our cohort, 62.5% (10/16) had a positive salivary gland ultrasound (OMERACT ≥2), 42.9% (6/14) a positive salivary gland biopsy (focus score ≥1), 54.5% (6/11) had a positive Schirmer's test, and 28.6% (2/7) abnormal unstimulated sialometry.

The median ESSDAI score at the last visit was 1.0 (IQR 1.0). Most patients (75%) were being treated with hydroxychloroquine, 25% with oral corticosteroids, 18.8% with rituximab, 6.3% with methotrexate, and 6.3% with etanercept, at the time of the last visit. Only 56.3% of patients met the 2016 EULAR/ACR criteria for adult SjD.

Conclusions: In this Portuguese cohort of paediatric Sjögren's disease, *sicca* symptoms, parotid swelling, lymphadenopathy, and joint involvement were common, alongside high rates of

hypergammaglobulinemia and positivity for ANA and SSA/Ro antibodies. Glandular and biological involvement appear to be prominent in children with SjD, with most patients achieving remission or low disease activity with systemic therapies. Our findings contribute to a better understanding of paediatric Sjögren's disease and may support future development of paediatric-specific classification criteria.

CL 06 - CAN SALIVARY GLAND ULTRASOUND REPLACE MINOR SALIVARY GLAND BIOPSY IN PRIMARY SJÖGREN'S SYNDROME? EVIDENCE FROM A REAL-WORLD COHORT

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Introduction: Minor salivary gland biopsy (MSGB) remains the reference for primary Sjögren's syndrome (pSS), however it is invasive. Salivary-gland ultrasound (SGUS) may provide a simpler alternative.

Objectives: The primary objective was to evaluate whether SGUS (OMERACT score) associates with MSGB positivity (focal lymphocytic sialadenitis with focus score (FS) ≥1/4 mm²) and to quantify SGUS accuracy. Secondary objectives included assessment of inter-reader reliability and sensitivity analyses.

Methods: Adults followed at ULS Coimbra who fulfilled the 2016 ACR/EULAR classification criteria for pSS were included. Descriptives (e.g. age, sex, disease duration, biopsy year and immunologic status were summarized as median (IQR) or n (%). SGUS of bilateral parotid and submandibular glands was scored 0–3 per gland (OMERACT) and considered positive if ≥1 gland was graded ≥2. MSGB was abstracted as positive (FS≥1), negative/unspecific, inconclusive/insufficient, or unknown. **Primary analysis included only patients with valid biopsies (positive/negative) and** included 2×2 accuracy metrics with Cohen's κ (with 95% CIs), ROC for continuous SGUS (sum of 4 gland scores, 0–12; and maximum gland score, 0–3), and binary logistic regression for biopsy positivity adjusted for observer (reader 1 vs 2). **Sensitivity analyses reclassified inconclusive as negative and** reanalyzed results by reader. Inter-reader reliability was assessed in a 6-patient subset using weighted Cohen's κ.

Results: Thirty-six patients with SGUS (88.9% female; median current age 64.5 [IQR 16.75]; median age at diagnosis 54.0 [IQR 17.25]; anti-SSA 80.6%, anti-SSB 41.7%) were included, of whom **24** had valid paired biopsies (biopsy-positive 62.5%; performed between 2000 and 2025). Inter-reader agreement for OMERACT grades was moderate to perfect (weighted κ 0.54–1.00). SGUS was positive in 58.3%. Against biopsy, SGUS showed **sensitivity 73.3%**, **specificity 66.7%**, **PPV 78.6%**, **NPV 60.0%**, **accuracy 70.8%**, and **κ=0.391** (fair; p=0.054). Continuous metrics showed fair discrimination: total SGUS score AUC 0.726 (95% CI 0.506–0.945; Youden-optimal cut-off ≥5 giving Se 73.3%/Sp 77.8%) and maximum SGUS AUC 0.722 (95% CI 0.511–0.933; optimal ≥2, matching the prespecified rule). In a reader-adjusted logistic model, SGUS positivity remained associated with biopsy positivity (aOR 6.91, 95% CI 0.95–50.31; p = 0.056) with no significant observer effect. Results were directionally robust when inconclusive biopsies were reclassified as negative (n = 26; Se 73.3%, Sp 54.5%, κ 0.28; aOR 3.13, 95% CI 0.59–16.64; p = 0.18) and when examined per reader (Observer 1: Se 80%, Sp 50%; Observer 2: Se 60%, Sp 100%; small denominators).

Discussion: In this pSS cohort, SGUS aligned with biopsy in a clinically meaningful, but not perfect, way. Using the prespecified rule (any gland OMERACT ≥2), we observed a balance of true positives and true negatives with a modest number of false negatives and false positives, yielding moderate discrimination and fair agreement versus MSGB, consistent with previous studies. Importantly, the method was reproducible

(inter-reader κ 0.54–1.00) and the association with biopsy positivity remained strong after adjusting for reader, indicating no clear reader effect. ROC analyses supported our a priori rule (the highest gland grade ≥2 was also the optimal cut-off) and suggested that a higher total SGUS threshold (≥5/12) can improve specificity when a more conservative stance is desired.

Conclusion: Our data support SGUS as a valuable, less-invasive tool that may reduce biopsy need in selected patients. Larger, prospective studies combining SGUS with serology and glandular function are warranted.

CL 07 - IMMUNE-MEDIATED NECROTIZING MYOPATHY IN THE PORTUGUESE POPULATION – A MULTICENTRIC NATIONWIDE STUDY

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Background: Immune-mediated necrotizing myopathy (IMNM) is a rare heterogeneous idiopathic inflammatory myopathy (IIM) characterized by proximal muscle weakness, markedly elevated creatine kinase (CK), and frequent need for immunosuppressive therapy (1,2). Three serological subgroups are recognized: anti–signal recognition particle (SRP), anti–3-hydroxy-3-methylglutaryl–coenzyme A reductase (HMGCR), and seronegative IMNM (2). Data on IMNM epidemiology and clinical features in the Portuguese population remain scarce.

Objectives: To characterize the IMNM cohort of the Rheumatic Diseases Portuguese Registry (Reuma.pt/Myositis), including clinical, serological, and histopathological features, comorbidities, and treatment patterns.

Methods: Multicentric, cross-sectional observational study including patients with a clinical IMNM diagnosis, as determined by the attending physician, registered in Reuma.pt/Myositis (3). Data collected comprised demographic and clinical characteristics, including muscle strength, extra-muscular manifestations, muscle enzymes, autoantibodies, biopsy, electromyography (EMG) and magnetic resonance imaging (MRI) findings, comorbidities, prior statin use and treatments. Data were analysed using SPSS v.28.

Results: A total of 30 patients were included from the 603 registered in Reuma.pt/Myositis (**Table 1**). The median age at onset was 59 years (IQR 21), 70% were females, and 88% Caucasian. The disease presented with subacute or insidious symmetric proximal weakness in 97% of cases. Median CK at diagnosis was 8357 U/L. Extra-muscular involvement included dysphagia (37%), Raynaud phenomenon (35%), and cardiac (17.9%) or pulmonary manifestations (21.4%). Autoantibody subgroups included 11 anti-HMGCR, 10 anti-SRP, and 5 seronegative patients. Previous statin use was reported in 7 of 11 anti-HMGCR positive patients. Muscle biopsy revealed predominant

myofiber necrosis with sparse inflammatory infiltrate in 90% of the patients. EMG and MRI supported myopathic changes in most cases. Regarding treatment, glucocorticoids were used in 80%, IVIG in 50%, rituximab in 43%, and at least one conventional immunosuppressant in 66.6% of patients. Malignancy was uncommon (6.7%). Significant differences across IMNM subgroups were observed for age at diagnosis and disease onset, with anti-HMGCR–positive patients being older than anti-SRP patients (p=0.011 and p=0.05,respectively). Female sex was more frequent in the SRP subgroup (p=0.012). Treatment patterns also differed: methotrexate was more commonly prescribed in HMGCR compared with seronegative patients (p=0.036).

Discussion: IMNM is estimated to represent approximately 3.6%–38% of all IIMs. Accordingly, the 5% proportion observed in the Reuma.pt/Myositis registry is consistent with prior reports (1,4–6). Clinically, our cohort mirrors typical IMNM features (1,4,7,8). Subacute/insidious onset, symmetric proximal weakness, and markedly elevated CK are consistent with previous descriptions (1,7). Muscle biopsy frequently showed myofiber necrosis with sparse inflammatory infiltrate, reflecting canonical necrotizing myopathy (4). Muscle MRI revealed oedema and early muscle damage, as seen in other series (8). Differences across subgroups mirror previous reports (2,7,9): anti-HMGCR–positive patients were older, whereas anti-SRP–positive patients showed a female predominance. Most anti-SRP patients were treated with glucocorticoids, IVIG, and rituximab, presumably reflecting a more aggressive disease course, as described in the literature (1,2,6,7). These results highlight the heterogeneous but distinct clinical profile of IMNM compared with other IIM subtypes (1,2,5,7).

Conclusion: IMNM represents a rare but clinically significant subset of IIM in Portugal. Serological subgroups exhibit distinct demographic, clinical, and therapeutic patterns. This nationwide characterization provides a foundation for prospective studies and inter-centre collaboration in this rare disorder.

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Table 3: Demographic, clinical, laboratory, comorbidity, and treatment characteristics of patients with immune-mediated necrotizing myopathy (IMNM), overall and by serological subgroup (seronegative, anti-SRP, and anti-HMGCR).

	All (n=30)		Seronegative (n=5/26)		Anti-SRP (n=10/30)		Anti-HMGCR (n=11/26)		p-value
Age at diagnosis* - median (IQR)	60.0	(15.0)	55.0	(21.0)	49.0	(20.0)	67.0	(15.0)	**0.02**
Age at disease onset* - median (IQR)	59.0	(21.0)	55.0	(20.0)	49.0	(16.0)	66.0	(16.0)	**0.01**
Female sex - n/N (%)	21/30	(70.0)	3/5	(60.0)	10/10	(100.0)	5/11	(45.5)	**0.016**
Caucasian - n/N (%)	22/25	(88.0)	4/4	(100.0)	7/9	(77.8)	10/10	(100.0)	NS
Portuguese - n/N (%)	26/28	(92.9)	5/5	(100.0)	7/9	(77.8)	11/11	(100.0)	NS
Disease characteristics - n/N (%)									
Insidious disease onset	29/30	(96.7)	5/5	(100.0)	9/10	(90.0)	11/11	(100.0)	NS
Symmetric proximal limb weakness	28/29	(96.6)	4/5	(80.0)	10/10	(100.0)	11/11	(100.0)	NS
Neck flexors weaker than extensors	16/25	(76.0)	3/4	(75.0)	9/10	(90.0)	5/8	(62.5)	NS
Ventilatory and pharyngeal weakness	1/30	(3.3)	0/5	-	1/10	(10.0)	0/11	-	NA
Esophageal involvement	11/30	(36.7)	0/5	-	4/10	(40.0)	6/11	(54.5)	NA
Gastrointestinal involvement	1/28	(3.6)	0/5	-	0/9	-	1/11	(9.1)	NA
Pulmonary involvement	6/28	(21.4)	1/5	(20.0)	3/9	(33.3)	0/11	-	NA
Cardiac involvement	5/28	(17.9)	1/5	(20.0)	3/9	(33.3)	0/11	-	NA
Arthralgia and/or arthritis	7/28	(25.0)	1/5	(20.0)	2/9	(22.2)	1/11	(9.1)	NS
Raynaud phenomenon	10/29	(34.5)	1/5	(20.0)	5/9	(55.6)	1/11	(9.1)	NS
Periungual changes	4/27	(14.8)	0/5	-	3/9	(33.3)	0/10	-	NA
Calcinosis	1/28	(3.6)	1/5	(20.0)	0/9	-	0/11	-	NA
MMT8* - median (IQR)	114.0	(39.0)	137.0	-	114	(41.0)	104.5	(46.0)	NA
IMNM suggestive biopsy - n/N (%)	18/20	(90.0)	4/4	(100.0)	3/5	(60.0)	9/9	(100.0)	NS
Myopathic changes on EMG - n/N (%)	18/22	(81.8)	4/4	(100.0)	5/6	(83.3)	9/10	(90.0)	NS
Myositis changes in MRI - n/N (%)	11/13	(84.6)	3/3	(100.0)	3/3	(100.0)	4/5	(80.0)	NS
CK elevation - n/N (%)	29/29	(100.0)	5/5	(100.0)	10/10	(100.0)	11/11	(100.0)	NA
LDH elevation - n/N (%)	22/22	(100.0)	5/5	(100.0)	6/7	(85.7)	9/9	(100.0)	NS
Liver enzymes elevation - n/N (%)	24/25	(96.0)	5/5	(100.0)	6/7	(85.7)	10/10	(100.0)	NS
Comorbidities - n/N (%)									
Diabetes mellitus	9/24	(37.5)	0/5	-	0/7	-	8/10	(80.0)	NA
Dyslipidaemia	14/24	(58.3)	3/5	(60.0)	2/7	(28.6)	8/10	(80.0)	NS
Hypertension	14/24	(58.3)	3/5	(60.0)	3/7	(42.9)	8/10	(80.0)	NS
Obesity	1/24	(4.2)	0/5	-	0/7	-	1/10	(10.0)	NA
Neoplasm	2/30	(6.7)	0/5	-	1/10	(10.0)	1/11	(9.1)	NA
Osteoporosis	2/24	(8.3)	0/5	-	2/7	(28.6)	0/10	-	NA
Smoking (active or ex-smoker)	3/23	(13.0)	1/4	(25.0)	0/6	-	2/9	(22.2)	NA
Previous statin use - n/N (%)	10/30	(33.3)	1/5	(20.0)	2/10	(20.0)	7/11	(63.6)	NS
Treatment, type - n/N (%)									
Glucocorticoids	24/30	(80.0)	4/5	(80.0)	8/10	(80.0)	9/11	(81.8)	NS
Intravenous Immunoglobulin	15/30	(50.0)	0/5	-	8/10	(80.0)	7/11	(63.6)	NA
Azathioprine	4/30	(13.3)	1/5	(20.0)	3/10	(30.0)	0/11	-	NA

Methotrexate	16/30 (53.3)	1/5 (20.0)	4/10 (40.0)	9/11 (81.8)	**0.018**
Mycophenolate	6/30 (20.0)	1/5 (20.0)	3/10 (30.0)	1/11 (9.1)	NS
Cyclophosphamide	3/30 (10.0)	0/5 -	2/10 (20.0)	0/11 -	NA
Rituximab	13/30 (43.3)	1/5 (20.0)	9/10 (90.0)	2/11 (18.2)	NS

Footnote: CK – Creatine Kinase; EMG – Electromyography; HMGCR – 3-Hydroxy-3-Methylglutaryl-Coenzyme A Reductase; IMNM – Immune-Mediated Necrotizing Myopathy; IQR – Interquartile Range; LDH – Lactate Dehydrogenase; MMT8 – Manual Muscle Testing of 8 muscle groups; MRI – Magnetic Resonance Imaging; NA – Not applicable; n/N (%) – Number of patients with the feature / total number of patients assessed; NS – Not significant; SD – Standard Deviation; SRP – Signal Recognition Particle.

*1 missing value for age at diagnosis and 1 for age at disease onset, 13 missing values for MMT8.

POSTERS

PO 01 - PRÁTICAS DE RASTREIO DA OSTEOPOROSE EM DUAS UNIDADES DE SAÚDE FAMILIAR DO NORTE DE PORTUGAL – UM ESTUDO TRANSVERSAL

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Introdução: A osteoporose é uma doença caracterizada pela perda da massa óssea e deterioração microestrutural do osso, conduzindo a maior fragilidade e risco de fratura. Em Portugal, afeta 10,2% dos adultos e constitui um importante problema de saúde pública, responsável por aproximadamente 40.000 fraturas anuais e custos diretos estimados em 216 milhões de euros/ano. A prevenção e o diagnóstico precoce são, assim, fundamentais. Nos Cuidados de Saúde Primários (CSP), o FRAX-Port, versão validada do Fracture Risk Assessment Tool para Portugal, permite estimar o risco de fratura major e da anca a 10 anos, orientando decisões terapêuticas baseadas em risco. Apesar da sua relevância, a aplicação do FRAX-Port na prática clínica parece ser, ainda, limitada.

Objetivos: Avaliar a taxa de aplicação do FRAX-Port em utentes com 50 ou mais anos em duas Unidades de Saúde Familiar (USF) do norte de Portugal e analisar a adequação da abordagem clínica subsequente à sua utilização, de forma a caracterizar as práticas de rastreio e gestão do risco fraturário nos CSP.

Material e Métodos: Realizou-se um estudo transversal entre maio e agosto de 2024, envolvendo utentes com idade igual ou superior a 50 anos observados em consulta presencial nas duas USF incluídas. As listagens de utentes elegíveis foram obtidas através da plataforma MIM@UF®. Foram excluídos os doentes com diagnóstico ativo de osteoporose. A amostragem foi aleatória, com base num tamanho amostral significativo previamente calculado. Foram consultados os processos clínicos eletrónicos individuais dos utentes incluídos e foram recolhidas variáveis sociodemográficas (idade, género, escolaridade e USF de origem) e clínicas: presença de critérios diretos para tratamento (fraturas de fragilidade prévias), aplicação do FRAX-Port nos últimos cinco anos, categoria de risco obtida e adequação da abordagem subsequente (vigilância, densitometria ou tratamento). Foi efetuada uma análise estatística descritiva.

Resultados: Dos 680 utentes incluídos na amostra, 1.3% (N=9) apresentavam fraturas de fragilidade prévias, configurando critério direto para tratamento farmacológico; contudo, apenas um (11.1%) destes iniciou terapêutica, três (33,3%) realizaram densitometria sem tratamento posterior e cinco (55,6%) mantiveram-se em vigilância. Dos restantes 671 utentes assintomáticos e elegíveis para rastreio, em 97.6% dos casos (N=655) não foi aplicado o FRAX-Port, apurando-se uma taxa de aplicação de 0,3% na USF A e 4,5% na USF B. Entre os dezasseis utentes (2.4%) cujo risco de fratura foi calculado, a maioria apresentou baixo e moderado risco fraturário - 50% (N=8) e 43.8% (N=7), respetivamente - e um caso (6.3%) apresentou alto risco. A abordagem clínica subsequente foi considerada adequada em 87,5% dos casos em que o FRAX-Port foi aplicado, sendo corretamente pedida densitometria nos doentes de risco intermédio e mantida vigilância nos de baixo risco.

Discussão/Conclusões: A taxa de rastreio do risco de fratura com o FRAX-Port nas duas USF estudadas foi baixa (2,4%), traduzindo uma reduzida integração desta ferramenta na prática clínica dos CSP. Em contraste, quando o FRAX-Port foi aplicado, a interpretação e a decisão subsequente mostraram-se, na maioria, adequadas, sugerindo que o problema reside sobretudo na não aplicação da ferramenta. Estes resultados estão em consonância com um estudo prévio desenvolvido numa outra USF, que revelou uma taxa de rastreio de 0,8%. Apesar de a literatura demonstrar que a utilização do FRAX-Port é custo-efetiva, permitindo orientar o tratamento para os doentes com risco fraturário clinicamente significativo

e reduzir o impacto económico, conclui-se que a adesão é ainda muito inferior ao esperado e sugere uma lacuna importante na gestão do risco de fratura. Desta forma, foi criado um plano de melhoria contínua da qualidade nas USF em estudo, centrado na formação dos profissionais, de forma a promover uma prática preventiva mais eficaz e consistente.

PO 02 - EFFICACY OF METHOTREXATE IN NECROTIZING MYOPATHIES: A CASE SERIES

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Introduction: Immune-mediated necrotizing myopathies (IMNM) are a subgroup of idiopathic inflammatory myopathies, characterized by severe clinical presentation with rapidly progressive muscular weakness and high serum creatine kinase (CK) levels.

IMNM is subclassified based on antibody status: anti-SRP+, anti-HMGCR+, and seronegative forms. IMNM require early, aggressive therapy to prevent long-term disability. Currently, immunosuppressant therapy is needed, methotrexate (MTX) and glucocorticoids (GC) are first line therapies.

The authors present five cases of IMNM treated with GC and MTX.

Results: The series includes 3 females and 2 male, ages between 27 and 68 years, 3 cases anti-HMGCR+ and 2 anti-SRP+. The mean disease duration is 5 years (11 months to 15 years), mean follow-up of 2 years. Two patients had previous exposure to statins (HMGCR+). One patient had lung involvement (SRP+).

All patients were treated with MTX, mean dose of 17,5-25mg, subcutaneous (sc). Patients were also treated with oral prednisolone, mean initial dose of 30mg (10-60mg), 13,5mg at 3 months, 8mg at 6 months, 5,1mg at 12 months (4 patients), 3,5mg at 24 months (3 patients).

All patients decreased CK levels and had improved muscular strength, as presented in table 1.

	Responses at Follow-up			
	3 months	6 months	12 months	24 months
CK response	58,8%	65,1%	78,0% (4 patients)	71,8% (3 patients)
MMT8 response	4,4%	8,4%	13,0% (4 patients)	14,7% (3 patients)

Table 4: Responses at Follow-up of CK levels and MMT8 score

Mean MMT8 score and MMT8 response are represented in table 3, based on antibody profile.

	Mean MMT8 score and MMT8 response			
	3 months	6 months	12 months	24 months
SRP+	121/150 (1,7%)	128/150 (8,0%)	133/150 (12,2%)	139/150 (16,9%)
HMGCR+	139/150 (7,2%)	142/150 (8,8%)	150/150 (13,8%) (2 patients)	150/150 (10.3%) (1 patient)

Table 3: Mean MMT8 score and MMT8 response during follow-up, based on antibody profile.

To date, SRP+ patients have a 133/150 MMT8 score and HMGCR+ 147/150.

Conclusion: All IMNM patients had clinical and analytical improvement with MTX and GC. In our study MTX sc was an effective therapeutic option.

HMGCR+ patients had a better clinical evolution and currently all patients have normal or near normal clinical muscular strength. SRP+ patients had a good initial response to treatment and improvement of muscular function within the follow-up, however without complete recovery of muscular strength.

Our results are consistent with the literature regarding the clinical phenotype of IMNM according to the antibody profile. The anti-HMGCR antibodies in IMNM are associated with a better response to treatment and improved functional muscular prognosis compared to SRP+ patients.

Despite these prognostic and functional differences, all patients improved with immunosuppressive therapy, as demonstrated in our series.

PO CC 03 - DISPLASIA CLEIDOCRANIANA E KNUCKLE PADS - MANIFESTAÇÃO INDEPENDENTE OU VARIANTE FENOTÍPICA?

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Introdução: A displasia cleidocraniana (DCC) é uma doença genética rara do desenvolvimento ósseo, de transmissão autossómica dominante, causada principalmente por mutações no gene *RUNX2*. Caracteriza-se por alterações esqueléticas, como hipoplasia ou ausência das clavículas, alterações craniofaciais e dentárias, além de baixa estatura. Embora o fenótipo esquelético seja predominante, podem ocorrer outras manifestações clínicas, incluindo alterações auditivas e respiratórias. Estes doentes apresentam ainda um risco aumentado de osteoporose (OP) na idade adulta. *Knuckle pads* (KP), também conhecidos como nódulos de Garrod, representam lesões subcutâneas benignas, que se localizam geralmente nas articulações interfalângicas proximais (IFP) ou metacarpofalângicas (MCF) das mãos ou pés. Podem ser idiopáticas, associadas a microtrauma repetitivo ou ainda associadas a certos síndromes genéticos do espectro fibromatoso.

DESCRIÇÃO DE CASO:

Mulher de 30 anos, fisioterapeuta, com diagnóstico de DCC aos 2 anos, sendo a variante descrita c.577C>T p.(Arg193X) em heterozigotia no exão 3 do gene RUNX2. Tratava-se de uma doente previamente seguida em consulta de Imunoalergologia por diagnóstico de asma controlada com terapêutica inalatória, bem como

Otorrinolaringologia por vertigem postural paroxística benigna. A doente foi referenciada à consulta de Reumatologia por queixas, com meses de evolução, de artralgias de ritmo mecânico e noção de presença de nódulos firmes no 2º e 3º dedos da mão esquerda e direita, respetivamente, dolorosos à mobilização da articulação, sem noção de tumefação articular. À observação, apresentava hipoplasia clavicular em relação com a sua doença de base, bem como nódulo móvel em topografia de IFP do 2º dedo da mão esquerda e 3º dedo da mão direita, não flutuante e indolor à palpação, sem aparente relação com a articulação. Analiticamente, não apresentava alterações de relevo. Para melhor descrição da tumefação observada, realizou ressonância magnética (RM), que revelou formação alongada, com baixo sinal das sequências ponderadas em T1, com moderada captação do produto de contraste com hipersinal nas sequências sensíveis ao líquido, em mais provável relação com manifestações de fibromatose no contexto de KP (figuras 1-3). A doente recusou intervenções diagnósticas e terapêuticas posteriores, nomeadamente biópsia para caraterização histológica e infiltração local. Realizou densitometria óssea, com resultado compatível com osteopenia (Densidade Mineral Óssea da coluna lombar de 0.894 g/cm2; Z-score -1.9). Atualmente, encontra-se assintomática com a aplicação de medidas gerais de controlo sintomático e de prevenção, nomeadamente evicção de trauma repetido. Foram ainda introduzidas medidas preventivas de OP dado o risco acrescido nestes doentes, nomeadamente suplementação de vitamina D para valores >30ng/mL, incremento de lácteos na dieta e atividade física regular em carga.

Conclusão:

O diagnóstico de DCC é geralmente feito em idade pediátrica, apresentando um espectro fenotípico variável. Este caso ilustra uma doente com manifestações esqueléticas clássicas, apesar da ausência de outras alterações características fenotípicas comumente reportadas. O caso realça ainda a necessidade de vigilância de co-morbilidades frequentemente associadas. A presença de KP poderá representar uma manifestação independente da doença, de etiologia idiopática, ou ter eventual relação com microtrauma em contexto profissional. No entanto, levanta-se a hipótese de uma variante fenotípica da DCC, previamente não descrita na literatura 1-6.

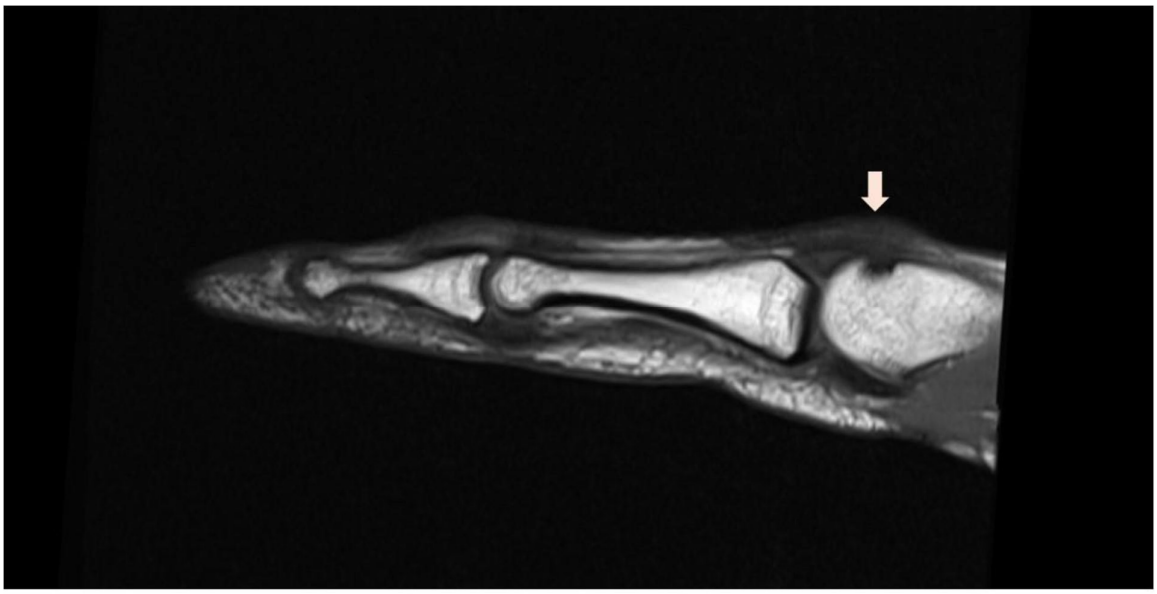

Figura 1. Corte sagital de formação alongada em topografia da vertente dorsal da IFP do 2º dedo da mão esquerda com baixo sinal em sequências ponderadas em T1.

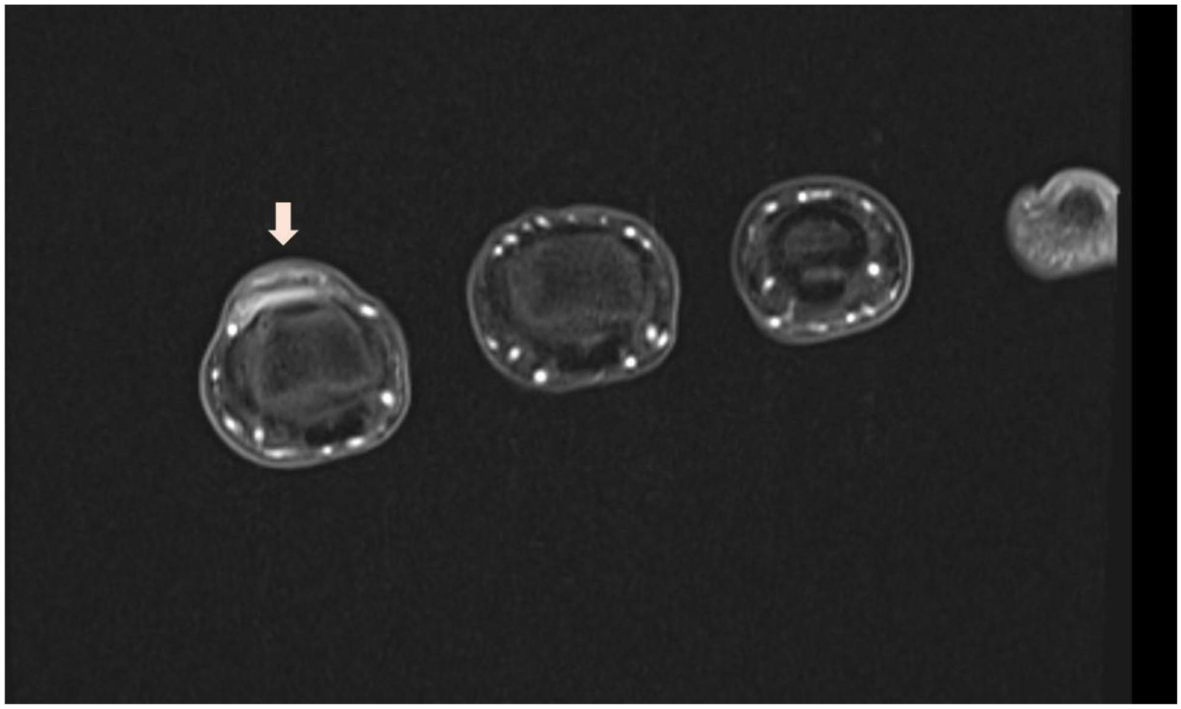

Figura 2. Corte axial de formação em topografia na IFP do 2º dedo da mão esquerda com captação de produto de contraste com hipersinal em sequências sensíveis ao líquido.

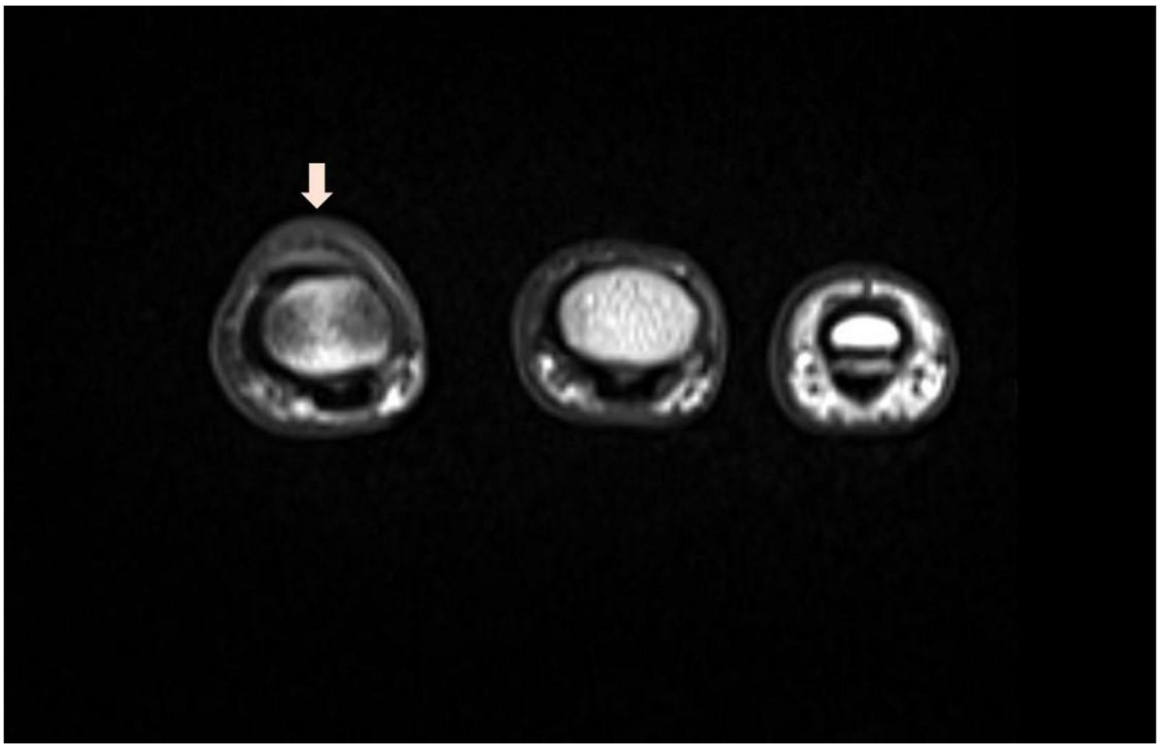

Figura 3. Corte axial de formação em topografia da IFP do 3º dedo da mão direita com captação de produto de contraste com baixo sinal em sequências ponderadas em T1.

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PO 04- GRÃO A GRÃO: CONDROMATOSE SINOVIAL BILATERAL NO IDOSO

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[1]ULS São José

A condromatose sinovial (CS) é uma doença rara que consiste na metaplasia benigna da membrana sinovial, caracterizada pela formação de corpos cartilaginosos livres dentro da articulação. Embora a etiologia exata seja desconhecida, a CS é classicamente descrita como uma condição monoarticular, mais comum no sexo masculino entre a terceira e quinta década de vida, que pode estar associada a fatores de risco, tais como trauma articular e inflamação crónica. O quadro clínico típico envolve dor, crepitação, edema e limitação do movimento articular. A diagnóstico é realizado através da avaliação clínica e de exames de imagem, como a ressonância magnética (RM). No entanto, este caso clínico destaca-se pela sua apresentação atípica, envolvendo um paciente idoso de 86 anos com manifestações bilaterais da doença, o que desafia o perfil epidemiológico e clínico habitual da CS.

O objetivo deste trabalho é apresentar um caso clínico de condromatose sinovial com características atípicas, nomeadamente a idade avançada do paciente e a bilateralidade da condição. Pretende-se, igualmente, sublinhar a importância do diagnóstico diferencial e da investigação detalhada em apresentações incomuns da doença, visando um tratamento adequado e a melhoria da qualidade de vida do paciente.

Um paciente de 86 anos, com história de gonartrose bilateral e queixas de gonalgia mais intensa à direita foi submetido, em 2014, a artroscopia à direita para meniscectomia e sinovectomia, por alterações degenerativas, com bom controlo sintomático posterior. Recentemente, apresentou quadro com queixas bilaterais de gonalgia, tumefação e limitação do movimento que foram geridas com infiltrações com corticoide, com melhoria apenas parcial, bem como surgimento de quistos de Baker no joelho esquerdo, que recorriam após drenagem.

A persistência dos sintomas motivou o encaminhamento para consulta de Medicina Física e Reabilitação. A avaliação clínica detalhada, complementada por ecografia revelou nódulos cartilaginosos hiperecogénicos intra-articulares bilateralmente, o que levantou a suspeita de condromatose sinovial bilateral. Este diagnóstico foi confirmado, posteriormente, por uma ressonância magnética (RM) em que foram descritos focos hipointensos sugestivos de condromatose sinovial.

Dado falência do tratamento prévio, foi realizado um bloqueio ecoguiado dos nervos geniculados do joelho, para controlo da dor, com resultados favoráveis. O paciente mantém seguimento para decidir entre a manutenção do controlo sintomático ou a intervenção cirúrgica, dependendo da evolução clínica.

Este caso sublinha a apresentação atípica da CS num paciente idoso com envolvimento bilateral, desafiando a compreensão convencional da doença. A inespecificidade dos sintomas e a variabilidade clínica realçam a necessidade de um elevado índice de suspeição e de uma investigação imagiológica aprofundada para um diagnóstico preciso. A abordagem multidisciplinar e a gestão individualizada são cruciais para otimizar os resultados em casos atípicos de condromatose sinovial, considerando a idade, a extensão da doença e a resposta à terapêutica

PO 05: IMPACT OF THE 2023 ACR/EULAR CLASSIFICATION CRITERIA ON PREGNANT WOMEN WITH PRIMARY ANTIPHOSPHOLIPID SYNDROME FOLLOWED IN A RHEUMATOLOGY–OBSTETRICS JOINT CLINIC

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Background: Antiphospholipid syndrome (APS) remains a major cause of pregnancy morbidity. The 2023 ACR/EULAR criteria introduced stricter definitions of obstetric morbidity and differential weighting of antibody isotypes and titers to improve specificity. However, concerns persist regarding reduced sensitivity for obstetric-only phenotypes and their clinical implications.

Objectives: We assessed the performance of the new classification criteria in a cohort of pregnant women fulfilling the 2006 Sydney criteria.

Methods: Single-center retrospective study of pregnant women with APS (Sydney criteria) followed at a tertiary multidisciplinary clinic (2009-2024). Patients with secondary APS or those lost to follow-up were excluded. Patients were reclassified using 2023 ACR/EULAR criteria, and clinical and pregnancy outcomes compared between those who met or did not meet the new criteria.

Results: Thirty pregnancies meeting the Sydney criteria were included. All women received standard antithrombotic therapy. There were 24 live births (80.0%) and 6 losses (20.0%). Two small-for-gestational-age newborns, 1 fetal growth restriction, and 1 preterm birth occurred. Twenty-one pregnancies (70%) fulfilled the 2023 ACR/EULAR criteria. Thrombotic APS cases according to Sydney classification decreased from 22 to 19, due to high-risk thrombotic profile (n=1) or isolated IgM (n=2). Likewise, obstetric APS declined from 8 to 2, due to lower weighting assigned to early pregnancy losses (n=4) and isolated IgM positivity (n=2).Patients fulfilling the new ACR/EULAR criteria (n=21) were younger at diagnosis (23.7 ± 6.7 vs. 32.3 ± 4.6 years, p=0.002) compared to those who did not meet the criteria (n=9). In addition, these patients had longer intervals between diagnosis and conception (8.5 vs. 7.0 years, p<0.001) and from the last thrombotic event to conception (8.5 vs. 1.0 years, p<0.001), and were less often obese (BMI >30Kg/m^2) (4.8% vs. 44.4%, p=0.019). Lupus anticoagulant (95.2% vs. 44.4%, p=0.005) and triple aPL positivity (42.9% vs. 0%, p=0.029) were more frequent in patients meeting the new classification criteria, suggesting a higher risk profile. In contrast, patients who did not meet ACR/EULAR criteria had higher prevalence of IgM anticardiolipin antibodies (aCL) (55.6% vs. 14.3%, p=0.032), particularly high titer (>80 units; 44.4% vs. 9.5%, p=0.049) and IgM anti-β2 glycoprotein I antibodies (aB2GPI) (55.6% vs. 14.3%, p=0.032), particularly at low titer (<40 units; 44.4% vs. 4.8%, p=0.019). Low-titer IgG aB2GPI (<40 units) was found exclusively in pregnancies excluded from ACR/EULAR criteria.

Despite a significantly lower birth weight in neonates from mothers meeting the new criteria (2880g vs. 3355 g, p=0.002), the incidence of adverse pregnancy outcomes remained comparable between groups. No further differences were identified – Table 1.

Conclusion: The 2023 ACR/EULAR criteria undervalue the relevance of recurrent early pregnancy loss and isolated IgM positivity, potentially excluding women with clinically significant obstetric APS. In our cohort, pregnancy outcomes were similar regardless of classification status, suggesting that patients failing the 2023 ACR/EULAR criteria may still benefit from standard prophylactic therapy. Although the new criteria

improve specificity for research purposes, reduced sensitivity may inadvertently exclude at-risk patients from both clinical care and scientific studies. Clinical judgment must remain key to the identification and management of obstetric APS.

Table 1. Demographic, clinical, and serological characteristics of pregnant women with APS and comparison between women meeting or not meeting the 2023 ACR/EULAR criteria.

Demographic characteristics	Sydney Criteria (n=30)	2023 ACR/EULAR Criteria		p-value$^{\phi}$
		Classified (n=21)	Not classified (n=9)	
Age at APS diagnosis (years), mean ± SD	26.3 ± 7.3	23.7 ± 6.7	32.3 ± 4.6	0.002
Age at conception (years), mean ± SD	32.3 ± 4.2	31.5 ± 4.3	34.0 ± 3.7	0.144
Time from diagnosis to conception (years), median [IQR]	5.0 [9.0]	8.5 [7.5]	7.0 [7.5]	<0.001
Time from last thrombotic event to conception (years), median [IQR]	6.0 [10.0]	1.0 [4.0]	8.5 [8.0]	<0.001
Classification, n (%)				
Thrombotic APS	22 (73.3)	19 (90.5)	3 (33.3)	0.003
Obstetric-only APS	8 (26.7)	2 (9.5)	6 (66.7)	N/A
Antiphospholipid antibody profile, n (%)				
Persistent LA	24 (80)	20 (95.2)	4 (44.4)	0.005
Anticardiolipin antibodies				
IgM positivity (total)	8 (26.7)	3 (14.3)	5 (55.6)	0.032

IgM <40	0	0	0	N/A
IgM 40-80	2 (6.7)	1 (4.8)	1 (11.1)	0.517
IgM >80	6 (20)	2 (9.5)	4 (44.4)	**0.049**
IgG positivity (total)	14 (46.7)	11 (52.4)	3 (33.3)	0.440
IgG <40	2 (6.7)	2 (9.5)	0	1.000
IgG 40-80	5 (16.7)	3 (14.3)	2 (22.2)	0.622
IgG >80	7 (23.3)	6 (28.6)	1 (11.1)	0.393
Combined IgG and IgM positivity	3 (10.0)	3 (14.3)	0	0.534
aB2GPI				
IgM positivity (total)	8 (26.7)	3 (14.3)	5 (55.6)	**0.032**
IgM <40	5 (16.7)	1 (4.8)	4 (44.4)	**0.019**
IgM 40-80	1 (3.3)	1 (4.8)	0	1.000
IgM >80	3 (10.0)	2 (9.5)	1 (11.1)	1.000
IgG positivity (total)	13 (43.3)	9 (42.9)	4 (44.4)	1.000
IgG <40	4 (13.3)	0	4 (44.4)	**0.005**
IgG 40-80	5 (16.7)	5 (23.8)	0	0.286
IgG >80	6 (20.0)	6 (28.6)	0	0.141
Combined IgG and IgM positivity	6 (20.0)	3 (14.3)	3 (33.3)	0.329
Double positivity (LA and aCL or aB2GPI)	19 (63.3)	12 (57.1)	7 (77.8)	0.419
Triple positivity (LA, aCL, and aB2GPI)	9 (30.0)	9 (42.9)	0	**0.029**
Cardiovascular risk factors and main comorbidities				

High blood pressure, n (%)	2 (6.7)	1 (4.8)	1 (11.1)	0.517
Obesity, n (%)	5 (16.7)	1 (4.8)	4 (44.4)	**0.019**
Diabetes, n (%)	0	0	0	N/A
Dyslipidemia, n (%)	4 (13.3)	3 (14.3)	1 (11.1)	1.000
Smoking, n (%)	2 (6.9)	1 (5.0)	1 (11.1)	0.532
Thyroid disease, n (%)	4 (13.3)	2 (9.5)	2 (22.2)	0.563
Gynecologic adverse condition*, n (%)	2 (6.7)	0	2 (22.2)	0.083
APS treatments during pregnancy, n (%)				
Anticoagulation, n (%)	27 (90.0)	19 (90.5)	8 (88.9)	1.000
Prophylactic LMWH, n (%)	11 (36.7)	5 (23.8)	6 (66.7)	**0.042**
Therapeutic LMWH, n (%)	17 (56.7)	15 (71.4)	2 (22.2)	**0.020**
Warfarin**, n (%)	2 (6.7)	2 (9.5)	0	1.000
Low-dose aspirin, n (%)	30 (100.0)	21 (100.0)	9 (100.0)	N/A
Combined therapy (anticoagulant and antiplatelet), n (%)	27 (90.0)	19 (90.5)	8 (88.9)	1.000
Hydroxychloroquine, n (%)	7 (23.3)	7 (33.3)	0	0.071
Maternal and Perinatal Outcomes during follow up, n (%)				
Early pregnancy loss***, n (%)	4 (13.3)	4 (19.0)	0	0.287
Late pregnancy loss ***, n (%)	1 (3.3)	0	1 (11.1)	0.300
Stillbirth***, n (%)	0	0	0	N/A
Medical termination of pregnancy, n (%)	1 (3.3)	1 (4.8)	0	1.000

Gestational age at delivery (weeks), median [IQR]	39.0 [2.4]	39.3 [2.4]	38.6 [2.7]	0.922
Preterm births, n (%)	1 (4.2)	1 (6.3)	0	1.000
Fetal growth restriction, n (%)	1 (4.2)	1 (6.3)	0	1.000
Small for gestational age, n (%)	2 (10.0)	2 (15.4)	0	0.521
Birth weight at delivery (grams), median [IQR]	3135.0 [506]	2880.0 [498]	3355.0 [600]	**0.002**
Cesarean deliveries, n (%)	17 (73.9)	10 (66.7)	7 (87.5)	0.369
Gestational hypertension, n (%)	1 (3.7)	0	1 (11.1)	0.333
Gestational diabetes mellitus, n (%)	3 (11.1)	2 (11.1)	1 (11.1)	1.000
Preeclampsia, n (%)	0	0	0	N/A
Eclampsia, n (%)	0	0	0	N/A
Maternal thrombotic events during pregnancy/postpartum, n (%)	2 (6.7)	2 (9.5)	0	1.000
Occurrence of adverse pregnancy outcomes, n (%)	8 (26.7)	7 (33.3)	1 (11.1)	0.374

Legend: APS, antiphospholipid syndrome; LA, lupus anticoagulant; aCL, anticardiolipin antibodies; aB2GPI, anti-β2 glycoprotein I antibodies; IgM, Immunoglobulin M; IgG, immunoglobulin G; LMWH, low-molecular-weight heparin;

N/A, not applicable. *Gynecologic adverse condition: includes gynecologic or structural conditions that may impair fertility or complicate pregnancy, such as endometriosis, uterine fibroids, uterine anomalies, or polycystic ovary syndrome. **Warfarin exposure was documented in two pregnancies where conception occurred during ongoing anticoagulation therapy, without prior preconception counselling. One pregnancy ended in spontaneous miscarriage and the other in elective termination during the first trimester. ***Early pregnancy loss: before 12 weeks+6 days of gestation; Late pregnancy loss: between 13 weeks+0 days and 19 weeks+6 days of gestation; Stillbirth: after 20 weeks of pregnancy.

ϕ Data are expressed as mean ± standard deviation (SD), median [interquartile range, IQR], or number (%), as appropriate. p-values refer to comparisons between patients fulfilling and not fulfilling the 2023 ACR/EULAR criteria. Statistical tests used include independent samples t-test, Mann–Whitney U test, and Fisher's exact test, as applicable.

PO CC 06 - MIOPATIA NECROTIZANTE IMUNOMEDIADA ANTI-HMGCR: A PROPÓSITO DE UM CASO CLÍNICO

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Introdução: A miopatia necrotizante imunomediada é uma entidade distinta dentro do espetro das miopatias inflamatórias, caraterizada por necrose e regeneração de fibras musculares com mínima inflamação. Clinicamente manifesta-se por um quadro de instalação aguda de fraqueza muscular proximal simétrica, valores elevados de creatinoquinase (CK) estando frequentemente associada a positividade para os anticorpos anti-SRP (signal recognition particle) ou anti-HMGCR (3-hidroxi-3-metilglutaril-CoA redutase). No entanto, cerca de 20% dos doentes são seronegativos.

As miopatias associadas ao anti-HMGCR estão frequentemente associadas ao uso prévio de estatinas (entre 15-65% dos doentes expostos, dependendo da origem geográfica e da idade). Esta entidade, difere da miopatia tóxica comum induzida por estatinas por não resolver totalmente com a suspensão do fármaco.

O reconhecimento precoce e o tratamento imunossupressor adequado são essenciais para prevenir incapacidade funcional.

Caso clínico: Os autores apresentam o caso clínico de um homem de 68 anos, caucasiano, com antecedentes de hipertensão arterial e dislipidemia. Medicado com lisinopril e sinvastatina desde há 9 meses.

Em janeiro de 2024, após quadro gripal, iniciou dor e fraqueza muscular proximal progressiva nas cinturas escapular e pélvica, com dificuldade em subir escadas e elevar os braços, determinando incapacidade para a corrida, atividade que realizava regularmente e posteriormente limitação em levantar-se de cadeiras. Negava sintomas sistémicos, cutâneos ou articulares. Ao exame objetivo apresentava marcha indiferente, cinturas escapulares com atrofia ligeira dos trapézios; força muscular globalmente G3 ao nível dos ombros, G4 cotovelos punhos e mãos de forma simétrica; cintura pélvica com amiotrofia das coxas, ancas com força muscular dos abdutores, adutores e extensores Grau 2+; flexores G3, extensão dos joelhos, dorsiflexores e flexores plantares G4, contabilizando um MMT8 de 128/150. Sem alterações da sensibilidade.

Analiticamente, verificou-se CK persistentemente elevada (3044 - 2058 U/L), aldolase 25,6 U/L e LDH 492 U/L, sem elevação dos parâmetros inflamatórios. Foi suspensa terapêutica com sinvastatina e iniciada ezetemiba, sem melhoria do quadro clínico.

Da marcha diagnóstica a destacar painel de miosites que revelou anticorpo anti-HMGCR positivo (5,59), com restantes autoanticorpos negativos. Realizou ressonância magnética das coxas que evidenciou "alteração do sinal normal das várias massas musculares de ambas as coxas, com hipersinal nas sequências ponderadas em T2 com supressão de gordura, verificando-se infiltração lipomatosa dos músculos essencialmente a nível da bacia, alterações relacionadas com edema muscular por processo inflamatório".

Assumiu-se o diagnóstico de miopatia necrotizante imunomediada anti-HMGCR.

Iniciou prednisolona 0,5mg/kg/dia e metotrexato subcutâneo 12,5 mg/semana com titulação da dose até os 17,5mg/semana. Concomitantemente iniciou programa de medicina física e de reabilitação dirigido.

Evolutivamente, verificou-se melhoria progressiva da força muscular com MMT8 aos 3 meses de 140/150 e aos 6 meses de 142/150, traduzindo uma melhoria de 11% desde o início do diagnóstico. Relativamente às enzimas musculares assistiu-se a descida da CK para 1536 U/L aos 3 meses e 1374 U/L aos 6 meses, perfazendo uma redução de 33,2% da CK desde o inicio da terapêutica dirigida.

Conclusão: A miopatia necrotizante imunomediada anti-HMGCR é uma entidade rara, mas importante no diagnóstico diferencial das miosites e das miopatias induzidas por estatinas.

O diagnóstico resulta da integração de dados clínicos, laboratoriais, imagiológicos e, quando necessário, histológicos, com os anticorpos anti-HMGCR a desempenharem um papel crucial na confirmação.

Este caso ilustra uma forma de miopatia necrotizante imunomediada associada a exposição prévia a estatina, associação que se correlaciona habitualmente com prognóstico mais favorável e melhor resposta à terapêutica imunomoduladora, sublinhando a relevância da suspeição clínica precoce, dado que o diagnóstico atempado e o tratamento adequado são cruciais para preservar a função muscular.

PO CC 07 - EXPANDING THE SPECTRUM OF AIRE-RELATED AUTOIMMUNITY A CASE OF PRIMARY SJÖGREN'S DISEASE IN EARLY CHILDHOOD

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Introduction: Sjögren's disease (SjD) is a chronic autoimmune disorder characterised by lymphocytic infiltration of exocrine glands, though its molecular pathogenesis remains poorly understood. The autoimmune regulator gene (AIRE) plays a key role in central immune tolerance, and its dysfunction predisposes to autoimmunity, including SjD. In AIRE-deficient mice, early lacrimal gland inflammation and altered signalling pathways mimic SjD, but in humans, this association is rarely reported.

Objectives: To describe a paediatric case of primary SjD likely associated with a heterozygous variant in the AIRE gene.

Methods: Case-report.

Results: A 4-year-old girl, with no relevant past medical history and a family history of systemic lupus erythematosus (maternal grandmother), was referred for bilateral submandibular swelling persisting for over a year, associated with intermittent parotid gland enlargement and recurrent episodes of unexplained high-grade fever (~39 ºC, lasting 1–2 days per month). She also reported xerostomia, with no ocular symptoms. Physical examination revealed elastic, non-tender submandibular swellings measuring ~3.5 cm. Laboratory work-up was unremarkable, except for a positive antinuclear antibody (ANA) (1:320) and elevated serum amyloid A (max 37 mg/L). Extractable nuclear antigens (ENA) panel, anti-dsDNA, and rheumatoid factor were negative. IgG4 and angiotensin-converting enzyme (ACE) levels were normal. Cryoglobulinemia, monoclonal component (free light chain ratio), hepatitis C and HIV were excluded.

Ultrasound revealed enlarged parotid and submandibular glands with heterogeneous echotexture and poorly defined hypoechoic areas, suggestive of chronic inflammation. Ophthalmologic evaluation was normal. Submandibular biopsy showed dense B- and T-cell infiltration forming seven lymphoid aggregates (>50 cells) with germinal centres; with a focus score of 2.1, compatible with SjD. No evidence of IgG4-related disease or lymphoma.

Given the early onset and systemic features, genetic testing was performed and revealed a heterozygous variant of uncertain significance (VUS) in the AIRE gene: c.816G>T (p.Arg272Ser), not previously reported in the literature. Pathogenic AIRE mutations have been described in both autosomal dominant and recessive forms of autoimmune polyendocrine syndrome type 1 (APS-1). Cytokine profiling and familial segregation

studies are ongoing. Treatment with hydroxychloroquine was initiated, with clinical improvement.

Conclusion: This case highlights a rare paediatric presentation of primary SjD likely associated with a previously unreported AIRE variant, pending further clarification through familial segregation studies. It supports the hypothesis that AIRE dysfunction may contribute to early-onset autoimmunity beyond classical APS-1. Findings from AIRE-deficient mouse models reinforce a potential pathophysiological link with salivary gland inflammation. Further studies are needed to elucidate the role of AIRE variants in isolated autoimmune phenotypes such as SjD.

PO 08 - RELAÇÃO ENTRE O USO DE BIFOSFONATOS E O SUCESSO DOS IMPLANTES DENTÁRIOS: REVISÃO NARRATIVA

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Introdução: Os bifosfonatos são amplamente utilizados no tratamento da osteoporose e de outras doenças metabólicas ósseas, atuando na inibição da reabsorção óssea por bloqueio da atividade osteoclástica(1). A sua utilização tem sido associada a complicações maxilofaciais, nomeadamente osteonecrose da mandíbula, e poderá comprometer o processo de osteointegração dos implantes dentários(3,5).

Objetivo: Rever a evidência atual sobre a relação entre o uso de bifosfonatos e o sucesso e sobrevivência dos implantes dentários.

Métodos: Foi realizada uma revisão narrativa da literatura na base PubMed, abrangendo publicações entre 2015 e 2025, utilizando as palavras-chave *bisphosphonates*, *dental implants*, *osteonecrosis* e *osseointegration*. Foram incluídos estudos clínicos, revisões sistemáticas e relatos de caso relevantes.

Resultados: A maioria dos estudos aponta para uma redução na taxa de sucesso dos implantes em doentes sob bifosfonatos, sobretudo com terapêutica prolongada ou por via endovenosa(1,3,4). Persistem, contudo, inconsistências quanto à influência da duração do tratamento, formulação utilizada e período de segurança antes da colocação do implante(2,4).

Conclusão: O uso de bifosfonatos pode afetar negativamente a osteointegração e o prognóstico dos implantes dentários, embora a magnitude desse efeito permaneça incerta. A avaliação individual do risco, a monitorização periódica e a adoção de medidas preventivas são fundamentais. São necessários estudos prospectivos robustos para estabelecer recomendações clínicas seguras.

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PO 09 - (SUB)DIAGNÓSTICO DA OSTEOPOROSE NA UNIDADE DE SAÚDE FAMILIAR TRAVESSA DA SAÚDE: ESTUDO OBSERVACIONAL TRANSVERSAL

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Introdução e Objetivos: A osteoporose é uma doença crónica caracterizada pela diminuição da densidade mineral óssea (DMO) e deterioração da microarquitetura óssea. O diagnóstico baseia-se na osteodensitometria óssea (DEXA), segundo a OMS. O *Scorecard For Osteoporosis in Europe – Scope 2021 Summary Report,* em 2019, estimou que a prevalência em Portugal era de 5,6%, sendo de 22% nas mulheres e 6,7% nos homens com ≥50 anos. O subdiagnóstico e subtratamento mantêm-se, com mais de 23 milhões de pessoas na União Europeia em risco elevado de fratura de fragilidade. De acordo com a auditoria da *International Osteoporosis Foundation* realizada em 2020, os Cuidados de Saúde Primários (CSP) são o principal prestador de cuidados em 16 dos 28 países avaliados, torna-se então essencial caracterizar esta realidade na nossa prática clínica.

Métodos: Neste estudo observacional transversal utilizou-se uma amostra de conveniência de utentes da Unidade de Saúde Familiar (USF) Travessa da Saúde (7 listas). Recolheram-se dados da plataforma MIM@UF (abril 2025) e do programa SClínico. Foi calculada a prevalência do diagnóstico de Osteoporose com base na codificação "L95 - Osteoporose" (ICPC-2). Para análise utilizou-se o teste do qui-quadrado de Pearson e o teste exato de Fisher ($p<0,05$), com o software R 4.4.3.

Resultados: Dos 19.843 utentes inscritos na USF Travessa da Saúde, 661 apresentavam o código "L95 – Osteoporose". A amostra em estudo incluiu 12.588 utentes, dos quais 398 tinham codificação ativa: 94,72% (n=377) mulheres e 5,28% (n=21) homens. Em idades ≥50 anos, a prevalência foi de 12,92% (n=370) nas mulheres e 0,93% (n=21) nos homens. O diagnóstico foi atribuído pelo médico de família em 72% dos casos, por especialista hospitalar em 10%, e 18% sem origem identificada. Apenas 70,6% realizaram DEXA.

Entre os fatores de risco analisados: 82,41% tinham ≥65 anos (18,93% (n=311) mulheres, 1,50% (n=18) homens); 8,79% (n=35) eram fumadores (14,29% (n=5) homens); 3,37% (n=11) tinham IMC <19 kg/m².

Quanto a presença de fraturas, 29,90% (n=119) estavam codificados: 15,26% (n=18) do fémur, 33,61% (n=40) vertebrais e 51,26% (n=61) correspondentes a outras fraturas. A destacar que 70,83% das fraturas ocorreu em idades ≥65 anos. Ainda 100% das fraturas do fémur, 90% das vertebrais e 95,08% das outras ocorreram no sexo feminino.

Destaca-se que 18,09% (n=72) dos utentes codificados com osteoporose tinham diagnóstico densitométrico compatível com osteopenia, para a qual não existe codificação ICPC-2.

A análise bivariada não revelou significância estatística entre o diagnóstico de osteoporose e os fatores de risco analisados, nomeadamente IMC e tabagismo ativo. O mesmo se verificou nos casos de osteopenia.

Discussão e Conclusões: A prevalência de osteoporose identificada reforça o papel central dos CSP, com destaque para o médico de família como principal responsável pelo diagnóstico. No entanto, os valores observados em idades ≥50 anos estão abaixo da média nacional em ambos os sexos, sugerindo um subdiagnóstico, particularmente no sexo masculino, o que poderá refletir a ausência de rastreio sistemático em faixas etárias de risco. Apesar de uma utilização considerável da DEXA, a inexistência de codificação (ICPC-2) para osteopenia limita uma precisão diagnóstica.

A implementação de um protocolo clínico específico poderá optimizar estratégias de rastreio, diagnóstico precoce e tratamento adequado, contribuindo para uma abordagem mais coordenada e eficaz, contribuindo para a prevenção de fraturas e melhoria da qualidade de vida dos utentes.

PO 10 - SHARED EXPERIENCE BETWEEN DERMATOLOGY AND RHEUMATOLOGY IN PSORIASIS AND PSORIATIC ARTHRITIS PATIENTS TREATED WITH GUSELKUMAB

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Introduction: Guselkumab (GUS) has demonstrated sustained efficacy in both psoriasis (PsO) and psoriatic arthritis (PsA) in randomized clinical trials. However, real-world data from Rheumatology practice in Portugal remains limited and mostly based on shared follow-up with Dermatology.

Objectives: To evaluate the real-world effectiveness and safety of GUS in a Portuguese cohort of patients with PsO and PsA managed in Dermatology and Rheumatology practice.

Methodology: We retrospectively identified all patients treated with GUS at both departments of Unidade Local de Saúde Santa Maria, until February 2025. Demographic characteristics, GUS treatment line, prior biologic/ targeted synthetic disease-modifying antirheumatic drugs (b/tsDMARDs), treatment duration (up to August 2025), reasons for switching, and disease activity for skin and joints were analysed at baseline, 6 months, and last visit. Data were captured using Derma.pt and Reuma.pt. Continuous variables were presented as mean ± standard deviation, and categorical variables as percentages.

Results: Between February 2022 and February 2025, a total of 26 patients received GUS, for a period ranging from 4 to 42 months (mean 24.6 ± 10.6). Of these, 24 initiated GUS for active PsO, 11 of them co-managed by Rheumatology for a PsA diagnosis. Two patients were treated exclusively for PsA within a clinical trial setting, where GUS was mandated as the 1st-line bDMARD and continued as per clinical practice.

The mean age at GUS initiation was 56.0 ± 14.6 years, with an equal sex distribution (50% female). GUS was used as 1st-line therapy in 27% of patients and as 2nd-line also in 27%, the large majority (64%) having exclusively PsO. In contrast, it was used as 3rd-line or beyond in 46% of cases, most commonly in patients with PsA (67%), where it was most often the 4th-line therapy.

Treatment discontinuation occurred in only 3/26 patients (11.5%): 2 due to adverse events (AE) and 1 due to primary failure. Notably, AEs included one case of drug-induced cutaneous lupus in a patient with prior positive antinuclear and anti-SSA/SSB antibodies, lready followed due to a diagnostic hypothesis of Sjögren Syndrome; and 1 case of a new diagnosis of Mycosis Fungoides. Other observed AE that did not warrant treatment discontinuation were skin fungal infections, urinary tract infections and a case of Varicella-zoster infection.

PsO patients with concomitant arthritis (PsA) had been frequently previously exposed to b/tsDMARDs, the majority to a TNFi (58.8%), followed by ustekinumab (17.6%) and secukinumab (15.7%).

Concomitant use of conventional synthetic DMARDs (csDMARDs) was observed in 34.6% of patients, primarily methotrexate (n=7, 26.9%). One patient was treated with leflunomide and another with hydroxychloroquine (a patient with drug-induced lupus to secukinumab switched to GUS).

Marked improvement in disease activity was observed, with psoriasis area and severity index (PASI) scores dropping from a baseline mean of 16.6 ± 12.7 to 1.9 ± 3.0 at 6 months, and further to 1.6 ± 3.0 at the latest visit. Eleven patients achieved complete skin remission (PASI 0). Although not the primary indication in most, improvement in joint symptoms (tender/swollen joint counts) was also documented.

Discussion/Conclusion: In this national real-world cohort, GUS demonstrated sustained effectiveness and a favourable safety profile in a diverse PsO/PsA population. Relevant experience with significant and lasting improvements in skin and joint disease is learned, with a notable proportion achieving PsO complete remission. These results support the utility of GUS across the psoriatic disease spectrum and underscore the benefits of coordinated care in managing complex patients.

	Exclusively Pso, n=13	PsO with arthritis (PsA), n=11	Exclusively PsA, n=2	Total, n=26
Current age, years	56.6 ± 13.5	61.2 ± 16.7	51.5 ± 6.4	58.2 ± 14.5
Age at start of GUS, years	54.2 ± 13.4	59.3 ± 16.9	49.5 ± 6.4	56.0 ± 14.6
Female sex, n (%)	6 (46.2)	6 (54.5)	1 (50.0)	13 (50.0)
GUS treatment line (b/tsDMARDs)				
1º, n (%)	4 (30.8)	1 (9.1)	2 (100.0)	7 (26.9)
2º, n (%)	5 (38.5)	2 (18.2)	0	7 (26.9)
3º, n (%)	2 (15.4)	1 (9.1)	0	3 (11.5)
4º, n (%)	1 (7.7)	4 (36.4)	0	5 (19.2)
5º, n (%)	1 (7.7)	1 (9.1)	0	2 (7.7)
6º, n (%)	0	0	0	0
7º, n (%)	0	1 (9.1)	0	1 (3.8)
8º, n (%)	0	0	0	0
9º, n (%)	0	0	0	0
10º, n (%)	0	1 (9.1)	0	1 (3.8)
GUS treatment duration, months	28.6 ± 10.6	20.4 ± 10.4	21.5 ± 2.1	24.6 ± 10.6
Previous b/tsDMARDs (total)	**16**	**35**	**0**	**51**
Tumor Necrosis Factor inhibitor, n (%)	11 (68.8)	19 (54.3)	0	30 (58.8)
Secukinumab, n (%)	2 (12.5)	6 (17.1)	0	8 (15.7)
Ixekizumab, n (%)	1 (6.2)	1 (2.8)	0	2 (3.9)
Ustekinumab, n (%)	2 (12.5)	7 (20.0)	0	9 (17.6)
JAKi, n (%)	0	1 (2.8)	0	1 (2.0)

Tocilizumab, n (%)	0	1 (2.8)	0	1 (2.0)
Concomitant csDMARDs				
Methotrexate, n (%)	2 (15.4)	4 (36.4)	1 (50.0)	7 (26.9)
Leflunomide, n (%)	0	0	1 (50.0)	1 (3.8)
Hydroxychloroquine, n (%)	0	1 (9.1)	0	1 (3.8)
Acitretin, n (%)	1 (7.7)	0	0	1 (3.8)
GUS switch/discontinued, n (%)	2 (15.4)	1 (9.1)	0	3 (11.5)
GUS switch indication				
Adverse event, n (%)	2 (15.4)	0	0	2 (7.7)
Primary failure, n (%)	0	1 (9.1)	0	1 (3.8)
Disease activity at baseline				
PASI, 0-72	18.3 ± 17.6	15.5 ± 5.3	8.0	16.6 ± 12.7
Tender joint count, 0–68	-	0.8 ± 1.4	18.0 ± 7.1	3.9 ± 7.1
Swollen joint count, 0–66	-	1.0 ± 1.6	16.5 ± 6.4	3.6 ± 6.2
Disease activity at 6 months				
PASI, 0-72	2.7 ± 3.8	1.3 ± 2.0	0	1.9 ± 3.0
Tender joint count, 0–68	-	0.9 ± 1.6	0	0.7 ± 1.4
Swollen joint count, 0–66	-	0.5 ± 0.9	0	0.4 ± 0.8
Disease activity at last visit				
PASI, 0-72	1.1 ± 1.4	2.7 ± 4.5	0	1.6 ± 3.0
Tender joint count, 0–68	-	0.6 ± 1.8	0	0.5 ± 1.5
Swollen joint count, 0–66	-	0.2 ± 0.7	0	0.2 ± 0.6

Pso: Psoriasis; PsA: Psoriatic Arthritis; GUS: Guselkumab; b/tsDMARD: Biologic/targeted Synthetic Disease-modifying Antirheumatic Drug; csDMARD: Conventional Synthetic Disease-modifying Antirheumatic Drug; PASI: Psoriasis Area and Severity Index.

The number of missing data per variable was as follows: PASI at baseline 26.9% (n=7); PASI at 6 months 30.8% (n=8); PASI at last visit 15.4% (n=4); Tender joint count at baseline 15.4% (n=2); Tender joint count at 6 months 23.1% (n=3);

PO 11- WHEN SYMPTOMS DON'T MATCH: LOW BACK PAIN, ARTHRITIS, AND RAYNAUD PHENOMENON

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INTRODUCTION: Paraneoplastic rheumatic syndromes are uncommon but clinically significant, as they may mimic primary autoimmune or inflammatory conditions. Inflammatory low back pain or arthritis in patients over 50 years of age, especially when accompanied by constitutional symptoms, should raise suspicion for an underlying malignancy.

CASE DESCRIPTION: Male in his 50s with hypertension and allergic rhinitis was referred from Hematology to Rheumatology for evaluation of anemia in the context of a possible inflammatory rheumatic disease. He reported a one-year history of inflammatory dorsolumbar pain associated and partial response to NSAIDs, but with progressive worsening. Over the previous two months, he developed predominantly nocturnal arthralgias of the hands and feet (without swelling), new-onset biphasic Raynaud phenomenon, anemia, and an unintentional 5-kg weight loss. He denied uveitis, dactylitis, or gastrointestinal symptoms. On examination, there was right knee arthritis and diffuse swelling of the right hand. Sacroiliac joint manoeuvres were negative. Laboratory tests showed normocytic, normochromic anemia (Hb 9.5 g/dL; normal iron studies and ferritin), elevated ESR (111 mm/h) and CRP (12.3 mg/dL), and negative autoantibodies (RF, ACPA, ANA) and HLA-B27. Viral and infectious serologies (HBV, HCV, HIV, CMV, parvovirus B19, syphilis) were negative. Plain radiographs of the hands and sacroiliac joints were unremarkable, and colonoscopy was normal. Given the seronegative, non-erosive polyarthritis, Raynaud phenomenon, inflammatory back pain in a patient over 50, marked systemic inflammation, anemia, constitutional symptoms, and negative viral serologies, a paraneoplastic process was suspected. Urgent CT imaging revealed thickened small bowel loops (~13 cm) (Figure 1) and bulky mesenteric and retroperitoneal lymphadenopathy, without metastatic lumbar lesions. Histology confirmed high-grade B-cell non-Hodgkin lymphoma, stage IVB, with intestinal involvement. Treatment with R-CHOP chemotherapy led to complete resolution of arthritis, axial pain, and Raynaud phenomenon after two cycles, with normalization of inflammatory markers.

DISCUSSION/CONCLUSIONS: This case illustrates paraneoplastic arthritis, Raynaud phenomenon, and inflammatory back pain as the presenting features of intestinal lymphoma. Red flags included age over 50, male sex, seronegative arthritis, constitutional symptoms, markedly elevated inflammatory markers, and complete remission with chemotherapy. Lung adenocarcinoma is the most frequent malignancy associated with paraneoplastic arthritis, followed by hematologic tumours. Importantly, leukaemia and lymphomas may present with paraneoplastic joint manifestations in up to 25% of cases. A multidisciplinary approach was essential to reach the final diagnosis. These findings highlight the importance of distinguishing paraneoplastic arthritis from primary rheumatic diseases, as recognition and treatment of the underlying malignancy are crucial for resolution of joint symptoms.

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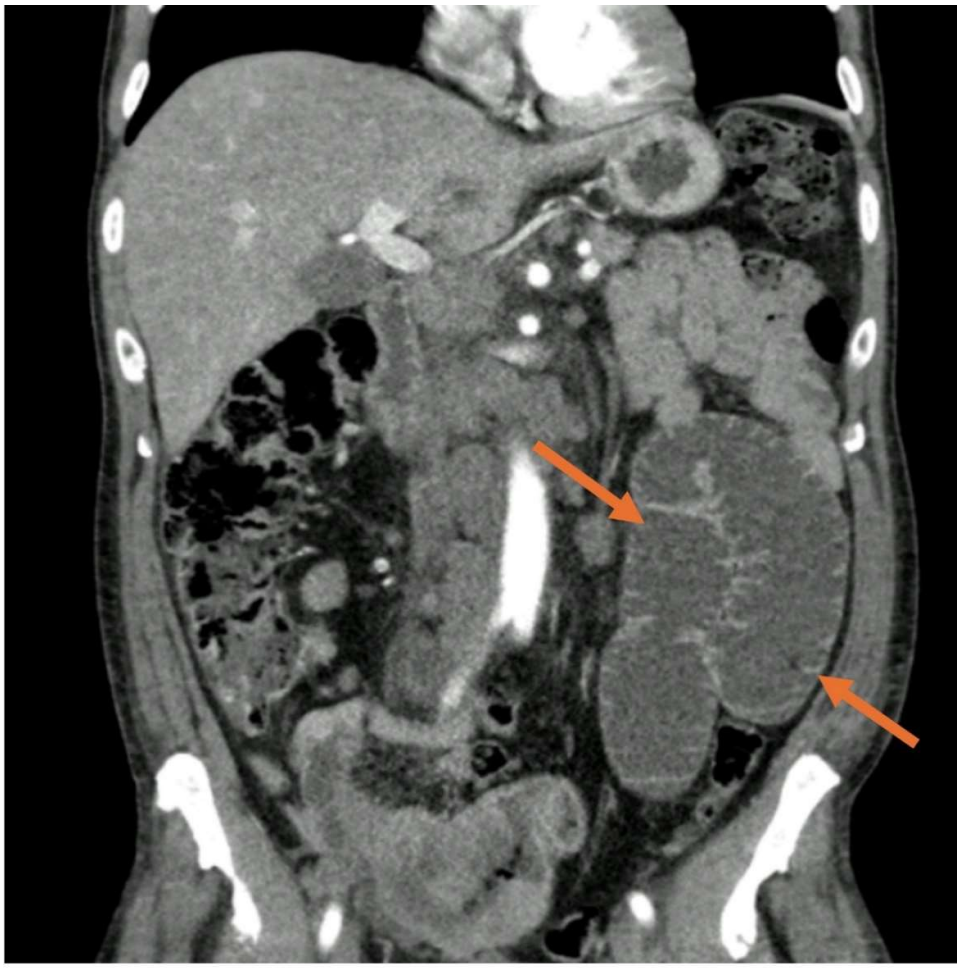

Figure 1. Coronal CT of the abdomen and pelvis showing a cluster of small bowel loops (orange arrows) with nodular wall thickening and mild expansion. The exact extent is difficult to delimit, but the involved segment measures approximately 13 cm.

PO CC 12 -NEUROPATIA VASCULÍTICA SARCOIDE: UMA FORMA RARA DE APRESENTAÇÃO

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Introdução: A sarcoidose é uma doença granulomatosa sistémica caracterizada pela presença de granulomas não caseosos, afetando predominantemente os pulmões e os gânglios intratorácicos. A neuropatia periférica ocorre em cerca de 10% dos doentes, sendo a forma vasculítica rara e pouco descrita.

Descrição de caso: Mulher de 60 anos, com antecedentes de depressão, hipotiroidismo, obesidade grau III e esteatose hepática, com diagnóstico de sarcoidose desde 2012, em contexto de eritema nodoso, poliartralgias inflamatórias, tenossinovite dos tendões aquilianos, peroneais e tibiais posteriores, e envolvimento pulmonar grau III confirmado por biópsia. Tratamentos prévios incluíram corticoterapia, suspensa por iniciativa própria, permanecendo sem medicação dirigida durante o período subsequente. Foi observada em consulta de Reumatologia pela primeira vez em 2016, tendo reiniciado corticoterapia em esquema de redução gradual, e metotrexato 10 mg/semana, titulado até 15 mg/semana por via subcutânea (dose máxima tolerada). Durante o seguimento, houve necessidade de infiltrações periarticulares por tenossinovite ativa. Por persistência de atividade inflamatória, iniciou infliximab 5 mg/kg a cada 8 semanas em julho de 2022, com resposta favorável.

Em março de 2024, a doente apresentou hipostesia e disestesias distais dos membros inferiores, sem défice de força muscular. O estudo laboratorial revelou AST 101 U/L, ALT 80 U/L, CK 733, aldolase 13.2 U/L, sem alterações do hemograma ou da função renal, nem elevação dos parâmetros de fase aguda; a ECA encontrava-se dos valores de referência (107 U/L ao diagnóstico). A eletromiografia revelou mononeuropatias múltiplas axonais dos membros inferiores, sem evidência de miopatia. Foi realizada biópsia do nervo sural, que evidenciou neuropatia axonal ligeira com discreta atividade, sem regeneração, associada a alterações inflamatórias e de vasculopatia, e presença de granulomas não caseosos, achados compatíveis com neuropatia vasculítica sarcoide. Foram excluídas causas infeciosas, incluindo tuberculose.

O tratamento sintomático foi ajustado, com pregabalina 150 mg três vezes por dia, venlafaxina 250 mg uma vez por dia e tapentadol 100 mg em SOS. A corticoterapia foi aumentada para 1 mg/kg/dia, posteriormente suspensa, e o esquema de infliximab encurtado para administração a cada 7 semanas, encontrando-se atualmente em remissão.

Discussão/Conclusão: A neuropatia vasculítica sarcoide é uma manifestação rara da sarcoidose, que se apresenta mais frequentemente como mononeuropatia múltipla axonal, com défice motor e sensitivo assimétricos, de início subagudo ou progressivo. Este caso reforça a importância de considerar esta forma de envolvimento neurológico em doentes com sarcoidose que desenvolvem sintomas neurológicos de novo ou inexplicados.

PO CC 13 - RABDOMIÓLISE INDUZIDA PELA ISONIAZIDA EM DOENTE COM ESCLEROMIOSITE: UM DESAFIO DIAGNÓSTICO

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Introdução: A escleromiosite é uma síndrome de sobreposição rara, frequentemente associada a anticorpos anti-PM/Scl. Nestes doentes, a elevação das enzimas musculares nem sempre reflete atividade da doença, sendo fundamental considerar causas alternativas.

Descrição de caso: Mulher de 49 anos, com diagnóstico de síndrome de sobreposição esclerose sistémica difusa e miosite (escleromiosite) estabelecido em 2013, em contexto de quadro clínico inaugural de fraqueza muscular proximal (MRC 4), elevação das enzimas musculares (AST 48 U/L, CK 259 U/L e aldolase 18.4 U/L), positividade para anticorpos anti-PM/Scl (100 e 75) e envolvimento pulmonar intersticial grave sugestivo de padrão NSIP. Iniciou tratamento com seis pulsos mensais de ciclofosfamida, seguido de terapêutica de manutenção com micofenolato de mofetil (MMF) 2 g/dia, com resposta favorável.

Em novembro de 2017, a dose de MMF foi aumentada para 3 g/dia devido ao agravamento das mialgias e da fraqueza muscular proximal (MRC 4), nova elevação das enzimas musculares (AST 50 U/L, CK 574 U/L e aldolase 13.2 U/L) e eletromiografia compatível com miopatia inflamatória sem sinais de desnervação ativa. Pela persistência do envolvimento muscular e necessidade de corticoterapia sistémica (prednisolona 5mg/dia), foi efetuado switch para rituximab (RTX), com resposta eficaz.

No contexto de avaliação de risco infecioso, iniciou isoniazida 300 mg/dia associada a piridoxina para tratamento de tuberculose latente em julho de 2019. Ao quarto mês, desenvolveu náuseas, astenia, mialgias, elevação das transaminases hepáticas e rabdomiólise, com AST 397 U/L, ALT 196 U/L, LDH 905 U/L, CK 2813 U/L, aldolase >117 U/L e mioglobina 1302 ng/mL, sem alteração da função renal. Foi colocada a hipótese de toxicidade induzida pela isoniazida, pelo foi suspensa. Foram excluídas outras causas de rabdomiólise, incluindo infeciosas. Seguiu-se uma melhoria clínica progressiva, com quase normalização laboratorial ao final de um mês (AST 48 U/L, ALT 32 U/L e CK 327 U/L e aldolase 16.4 U/L e mioglobina 94.9 ng/mL). Em janeiro de 2020, iniciou rifabutina 300 mg/dia, completando o esquema até maio do mesmo ano sem intercorrências.Atualmente, mantém RTX semestral e encontra-se clinicamente estável, com força muscular preservada e normalização das enzimas musculares.

Discussão/Conclusão: Este caso destaca a importância de uma avaliação cuidadosa da elevação das transaminases hepáticas e das enzimas musculares em doentes com escleromiosite, considerando causas alternativas à atividade da doença, como toxicidade farmacológica ou infeções, permitindo ajustes terapêuticos seguros. A monitorização laboratorial periódica é essencial durante a profilaxia com isoniazida, especialmente em doentes com imunodeprimidos.

PO CC 14 - EXUBERANT PALPEBRAL OEDEMA: AN UNDERRECOGNIZED CLINICAL FEATURE OF DERMATOMYOSITIS

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Introduction: Dermatomyositis (DM) is a rare, chronic autoimmune disease characterized by a combination of inflammatory myopathy and heterogeneous cutaneous manifestations. Autoantibodies play a significant role in the pathogenesis and are closely associated with specific clinical phenotypes. Anti-TIF1-γ DM is distinguished by extensive, frequently severe cutaneous symptoms, varying degrees of muscle involvement, and a notable link to malignancy. We present a case of anti-TIF1-γ positive DM with marked palpebral oedema, severe skin rash and mild myopathy.

Case report: A 64-year-old woman has been followed in the Rheumatology clinic since 2012 for DM. Her initial clinical presentation included mild bilateral proximal muscle weakness and erythematous scaly rashes on the hands and fingers, as well as scaly, pruritic, erythema on the anterior upper chest and posterior neck, with a

psoriasis-like appearance. Laboratory tests showed mildly elevated levels of creatine kinase, aldolase, ESR, CRP, and a positive antinuclear antibody (ANA) at a titer of 1/640, with a negative extractable nuclear antigen (ENA) panel. Skin biopsy revealed atrophic and hyperkeratotic epidermis with interstitial and perivascular lymphocytic infiltration, consistent with DM. She received treatment with 60mg prednisolone, tapering, and 15mg subcutaneous methotrexate, resulting in improved muscle response and reduced rash inflammation, with sequelae of poikiloderma. In late 2024, she presented with bilateral palpebral oedema and recurrence of erythematous rash in a sun exposed distribution. The left eyelid oedema progressed in a few months and led to complete visual field obstruction. MRI findings indicated bilateral periorbital inflammation, more pronounced on the left, with features typical of a chronic inflammatory process compatible with dermatomyositis-related orbital involvement. No evidence of intraorbital masses, muscle infiltration, or vascular abnormalities was found. Palpebral and orbicular muscle biopsies showed no evidence of malignancy. Infectious and allergic causes were also ruled out. Repeat immunological screening was conducted revealing positive ANA, ENA, and positivity for anti-TIF1-γ and anti-Ro52. Cancer screening was negative. She was started on prednisolone 40 mg, achieving marked improvement in left eyelid oedema and widening of the palpebral fissure. However, with dose tapering, the oedema worsened. She was then treated with monthly IVIG (2 mg/kg/day for 5 days), resulting in sustained improvement of periorbital oedema and skin lesions at 3 months follow-up. She is scheduled for blepharoplasty aimed at achieving appropriate tissue reduction and improving aesthetic appearance.

Discussion: Although periorbital oedema is a classic cutaneous feature of DM, typically part of the heliotrope rash, it is frequently underrecognized or misinterpreted. Extensive oedema can obstruct the visual field and is often linked to skin disease activity. In our case, we observed significant improvement with prednisolone and IVIG therapy. Due to the wide range of possible causes of periorbital oedema, it is important to rule out infectious, malignant, vascular and allergic conditions through proper evaluation.

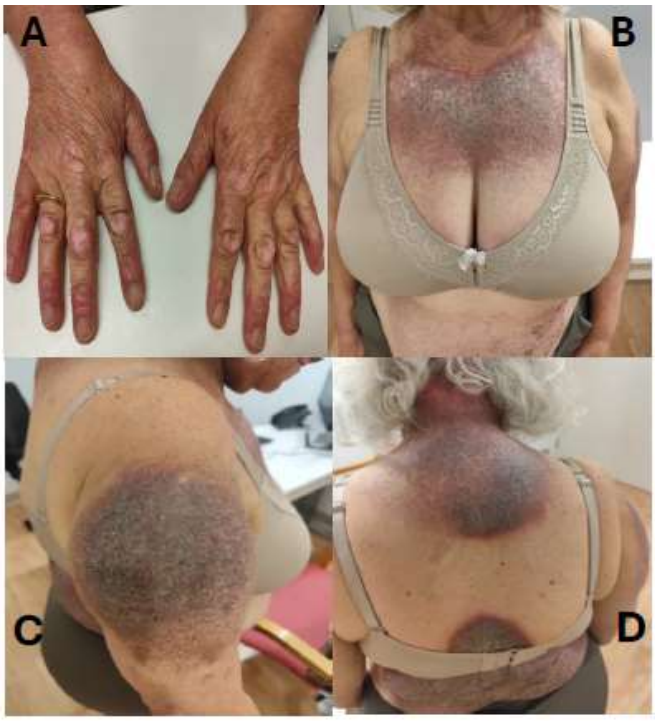

Figure 1. Clinical features of dermatomyositis. (**A**) Gottron's papules; (**B**) V-sign rash with poikiloderma; (**C**, **D**) Shawl sign extending to the lateral arm and scalp rash with poikiloderma, consistent with chronic disease.

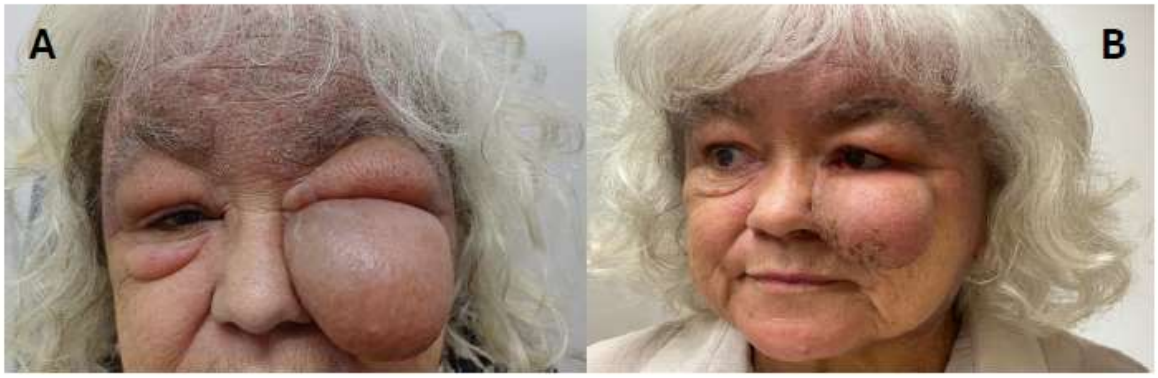

Figure 2. A: Exuberant periorbital oedema, especially of the left inferior palpebra, occluding the left visual field; **B:** 3 months after starting on prednisolone and monthly IGIV with increased eyelid fissure opening.

PO CC 15 - "METOTREXATO E PULMÃO: DE SUSPEITO A TERAPÊUTICO – UM CASO CLÍNICO"

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Introdução: A artrite reumatóide (AR) é uma doença inflamatória autoimune caraterizada pela presença de poliartrite simétrica, podendo levar a destruição e deformidade das articulações sinoviais. É uma patologia sistémica, que pode apresentar diferentes manifestações extra-articulares, tais como o envolvimento pulmonar. Este pode ser consequência da própria doença ou secundário ao seu tratamento. Apresentamos o caso de uma doente com o diagnóstico de AR e a presença de múltiplos nódulos pulmonares, que protelaram o início de terapêutica com metotrexato.

Caso clínico: Doente do sexo feminino, 57 anos de idade, com antecedentes pessoais de osteoporose não fracturária, doença pulmonar obstrutiva crónica, tabagismo ativo e tuberculose latente, tendo realizado terapêutica com isoniazida em 2017. Apresenta seguimento em consulta de Reumatologia por AR erosiva, com fator reumatóide e anti-CCP positivos. O quadro terá tido início em 2012 e o diagnóstico estabelecido em 2017. A doente tem sido também seguida em consulta de Pneumologia, onde para estudo dos nódulos pulmonares, realizou broncofibroscopia, com resultados microbiológicos, micobacteriológicos e citológicos negativos, biópsia transtorácica com agulha e tomografia por emissão de positrões (PET) combinada com tomografia computadorizada (TC). Estes últimos revelaram resultados inespecíficos, descrevendo provável processo inflamatório no contexto da AR, sem se conseguir excluir outras etiologias.

Inicialmente, dado os antecedentes da doente, optou-se por uma tentativa inicial de controlo da doença com leflunomida 20mg id, hidroxicloroquina 400mg id e prednisolona 2.5mg id. Contudo, cerca de 5 anos após o início da terapêutica, objetivamente, a doente mantinha artrite das pequenas e médias articulações de ambas as mãos, descrevendo rigidez matinal prolongada. Analiticamente, apresentava elevação da PCR (1.16mg/dL) e da VS (61mm/H), sendo o valor de DAS28 de 4.69, compatível com doença ativa moderada. A nível pulmonar, na consulta de Pneumologia, a doente manteve controlo dos nódulos pulmonares por TC, sem evidência de melhoria radiológica.

Assim, por manter atividade da doença, após discussão conjunta com a Pneumologia, optou-se pela suspensão da hidroxicloroquina e a introdução de metotrexato oral com titulação progressiva da dose até 20mg por semana. Na TC pulmonar, realizada em junho de 2023, cerca de 1 mês e meio após o início do metotrexato, destacava-se a presença de um nódulo no segmento posterobasal do lobo inferior direito, já descrito anteriormente mas, agora, com evidência de cavitação. Embora a hipótese infecciosa não pudesse ser excluída, por ausência de agravamento clínico ou analítico, e pela terapêutica anterior com isoniazida, optou-se pela manutenção do metotrexato sob vigilância. Na reavaliação imagiológica, 14 meses após a alteração, era visível uma evolução favorável da maioria dos nódulos, destacando-se a perda de cavitação e uma redução dimensional de cerca de 17% do nódulo acima descrito (Fig 1). A nível clínico, o DAS28 era de 2.62.

Discussão e Conclusão: Este caso expõe a complexidade na distinção entre o envolvimento pulmonar na artrite reumatóide de outras patologias pulmonares, nomeadamente em indivíduos com fatores de risco e/ou com o diagnóstico de outras patologias respiratórias, obrigando a uma vigilância apertada

destes doentes. Adicionalmente, o tratamento revela-se igualmente um desafio, tanto pela dificuldade em estabelecer uma solução terapêutica adequada face às comorbilidades dos doentes, como pela potencial toxicidade farmacológica pulmonar.

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PO CC 16 - PULMONARY AMYLOIDOSIS IN SJÖGREN'S DISEASE - A SERIES OF THREE CASES

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Background: Sjögren's disease (SjD) is a systemic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands, leading to sicca symptoms, but also other organ specific manifestations. Pulmonary involvement occurs in 43-75% of cases, with 9-22% of patients being symptomatic. Any lung compartment may be affected, most frequently the interstitium, airways, pleura, or vasculature.

Pulmonary amyloidosis (PA) is a rare manifestation of SjD, reported in 5.5–6.5% of patients, typically presenting as pulmonary nodules. We describe three cases of this uncommon manifestation.

Case #1: A 64-year-old man underwent routine preoperative evaluation, which revealed a pulmonary mass. Lung biopsy demonstrated amorphous material with green birefringence under polarized light following Congo red staining, consistent with amyloidosis. He was referred for evaluation of possible connective tissue disease and reported chronic dry eyes with recurrent keratitis and xerostomia, without parotid swelling. Laboratory tests showed elevated erythrocyte sedimentation rate (ESR, 42 mm/h), antinuclear antibodies (ANA, 1:320, AC-4), anti-Ro52 (135.8), anti-Ro60 (87.3), and mildly increased rheumatoid factor (36.4). Salivary glands ultrasound revealed bilateral parotid abnormalities (OMERACT grades 3 and 2), confirming SjD. Three months later, the pulmonary lesions grew, with polyclonal hypergammaglobulinemia and systemic inflammation prompting treatment with mycophenolate mofetil (MMF). After six months, there was a partial regression of lung nodules and improved systemic activity.

Case #2: A 57-year-old woman with an undifferentiated connective tissue disorder was referred for lung biopsy after multiple pulmonary nodules were identified on imaging. The pathology confirmed amyloidosis (Congo red–positive amorphous material) and the immunohistochemical studies were inconclusive. During evaluation, she reported Raynaud's phenomenon, haemoptysis, fatigue, and a decreased salivary pool was noted despite lack of dry complaints or parotid swelling. Workup revealed ANA (1:640, AC-4), high titres of anti-Ro52 (8560), anti-Ro60 (109030.3), anti-SSB (1066.2), and elevated serum amyloid A (9.67 mg/dL). A diagnosis of SjD with

secondary PA was established. After two years of clinical stability with azathioprine, follow-up CT demonstrated growth of the lung nodules, and MMF was initiated.

Case #3: A 44-year-old woman was referred for evaluation of suspected connective tissue disease following a diagnosis of PA. She reported pleuritic chest pain for several months, with imaging showing a lung nodule and moderate pleural effusion. Biopsy revealed amorphous material with Congo red positivity, confirming PA and the immunohistochemistry was inconclusive. The patient reported dry mouth and eyes, sporadic parotid swelling, fatigue, weight loss, and purpuric rash. Laboratory findings included elevated ESR (98 mm/h), ANA (1:1280), anti-Ro52 (16863.8), anti-Ro60 (>27496.0), anti-SSB (1428.9) and rheumatoid factor (49.6), and polyclonal hypergammaglobulinemia with increased IgG (2653 mg/dL). Salivary glands ultrasound showed parotid and submandibular abnormalities (OMERACT grade 2), consistent with SjD. Over the 5 years of follow-up, the pulmonary nodules remained stable, while cutaneous and systemic activity required intensification of immunosuppression with rituximab.

Discussion: All three patients exhibited localised pulmonary amyloidosis, which appears more common in SjD than systemic forms. In two cases, PA was incidentally detected and prompted the diagnosis of SjD, an atypical but documented clinical sequence. Diagnosis requires histopathological confirmation, and subtyping of amyloid protein through immunohistochemistry or mass spectrometry may inform treatment decisions. However, in these cases, such studies were either not performed or inconclusive.

This case series highlights the need for heightened clinical suspicion of PA as an uncommon and often asymptomatic pulmonary manifestation of SjD. Recognition and accurate characterisation of amyloid deposition are crucial for timely management, as pulmonary amyloidosis may influence systemic disease course and prognosis.

PO CC 17 - EOSINOPHILIC FASCIITIS: DIAGNOSTIC CHALLENGES AND THE ROLE OF ULTRASOUND EVALUATION - A CASE REPORT

Fabiana Gouveia

Introduction: Eosinophilic fasciitis (EF) is a rare and often underdiagnosed connective tissue disorder, resulting in uncertainty about its prevalence (approximately 300 patients reported worldwide). It is characterized by inflammation and fibrosis of the subcutaneous fascia and frequently associated with eosinophilia. Although etiology is unclear, reported triggers include strenuous exercise, trauma, infections, medications, and autoimmune diseases. The differential diagnosis is particularly challenging, since its clinical features may mimic systemic sclerosis (SSc).

Clinical Case: We describe a 21-year-old woman presenting with progressive skin thickening of the limbs, subjective swelling of the wrists and ankles, and progressive functional impairment. Symptoms began during isotretinoin treatment for acne and persisted after discontinuation.

On physical examination, there was diffuse hardening of the integument of the upper and lower limbs (excluding hands and feet), resulting in limited joint mobility and perceived muscle weakness. No arthritis, Raynaud phenomenon (RP), or capillaroscopic abnormalities were observed. Laboratory tests revealed ANA 1:320 (dense fine speckled nuclear pattern), elevated ESR and CRP, peripheral eosinophilia, hypergammaglobulinemia and microcytic anemia. Ultrasound demonstrated fascial and tendon thickening of the forearms and legs and screening for systemic involvement was unremarkable.

A provisional diagnosis of EF was assumed and the patient was started on prednisolone 1 mg/kg/day and methotrexate 15 mg/week. MRI of the forearm confirmed diffuse fascial thickening and a deep skin and fascia biopsy revealed septal panniculitis with eosinophilic infiltrate, consistent with EF.

Treatment led to partial clinical improvement, normalization of inflammatory parameters, and resolution of eosinophilia. Phototherapy and physiotherapy were also recommended.

Discussion/Conclusion: This case illustrates the clinical overlap between EF and SSc (such as skin thickening and joint mobility restriction). However, unlike SSc, EF usually spares the hands and feet, does not cause RP or capillaroscopic changes, and typically lacks visceral involvement.

Ultrasound may be a valuable tool for EF diagnosis and follow-up, as it can detect fascial and tendon thickening in a fast and non-invasive manner, complementing clinical evaluation.

Another interesting aspect of this case is the temporal association with isotretinoin exposure. Although causality cannot be established, isotretinoin has been reported as a possible trigger of other eosinophil-related pathological disorders. This reinforces the need to consider drug exposure as a potential precipitating factor.

The patient showed partial clinical improvement. However, clinical recovery is often slow, and relapses may occur, reinforcing the need for long-term follow-up.

PO 18 - DIFFERENCES BETWEEN PRIMARY AND SECONDARY ANTIPHOSPHOLIPID SYNDROME: INSIGHTS FROM A TERTIARY CENTER COHORT

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Introduction: Antiphospholipid syndrome (APS) is a systemic autoimmune disorder characterized by the occurrence of thrombosis, pregnancy morbidity or microvascular involvement in individuals with persistent antiphospholipid antibodies (aPL).

Objectives: Our primary aim was to compare disease features between primary and secondary APS.

Materials and Methods: We conducted a retrospective observational study including patients with APS followed at the Rheumatology Department of Coimbra University Hospital. Patients were included if they had a clinical diagnosis established by a rheumatologist.

We collected demographic, clinical, laboratory and therapeutic data through medical record review, covering patients followed between 1999 and 2025. Patients were grouped into primary or secondary APS, and intergroup differences were analysed.

Results: A total of 81 patients were included: 82.7% female, and mean age at diagnosis was 39.6 years. Primary APS affected 54.3% of patients, while the remainder had APS associated with systemic lupus erythematosus (37.0%) or other autoimmune diseases (8.6%; n= 7), such as undifferentiated connective tissue disease, rheumatoid arthritis or microscopic polyangiitis.

A total of 66.7% of patients fulfilled the Sapporo criteria, of whom 42.6% also met the 2023 ACR/EULAR criteria. No patient fulfilled the ACR/EULAR criteria alone.

Thrombotic events were the most frequent clinical manifestations: venous in 60 patients, arterial in 24 and microvascular in 3. Obstetric morbidity affected 19.4% of women, mainly early pregnancy loss, followed by premature births due to placental dysfunction, and fetal deaths beyond 10 weeks' gestation.

Venous thrombosis most frequently involved deep veins, pulmonary arteries and cerebral venous sinuses, but also splanchnic, hepatic and inferior vena cava territories. Some patients had recurrent vascular access thrombosis or superficial vein involvement.

Arterial events included stroke, acute coronary syndrome, central retinal artery occlusion and thrombosis of renal, splenic and peripheral arteries.

Microvascular involvement comprised thrombotic microangiopathy with acute kidney injury and recurrent diffuse alveolar haemorrhage. Libman-Sacks endocarditis was observed in two patients.

Among patients with available data, lupus anticoagulant was detected in 88.9%, anti-β2 glycoprotein I antibodies in 49.4%, and anticardiolipin antibodies in 46.9%. Triple positivity was observed in 39.5%. Anti-phosphatidylserine antibodies were tested in 29 patients, with a positivity rate of 27.6%.

The mean age at diagnosis was significantly higher in primary APS (44.2 ± 15.8 vs. 31.7 ± 13.9 years; p = 0.001), with a large effect size (d = 0.82). The median time to diagnosis was 1 year in primary APS and 0 in secondary APS, being significantly longer in primary APS (p = 0.022). In contrast, no statistically significant differences were found between groups regarding sex distribution, clinical features, or aPL profile.

Cardiovascular risk factors and hereditary thrombophilia were also assessed as potential contributors to thrombosis.

Discussion: This study identifies a diagnostic delay in patients with primary as compared with secondary APS that needs to be improved. Current classification criteria are intended to be used as inclusion criteria for clinical trials, but present a low sensitivity for APS diagnosis.

PO CC 19 - ULTRASOUND FOR THE DIAGNOSIS OF GIANT CELL ARTERITIS – IS AXILLARY ULTRASOUND SUFFICIENT TO DETERMINE INVOLVEMENT OF EXTRACRANIAL LARGE VESSELS?

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Introduction: In the most recent European Alliance of Associations for Rheumatology (EULAR) recommendations1, ultrasound of the temporal and axillary arteries is considered the first-line imaging modality for suspected Giant Cells Arteritis (GCA). Systematic inclusion of axillary arteries increases diagnostic sensitivity and provides a baseline for future follow-up, allowing bedside documentation of extracranial large-vessel involvement. However, GCA often affects other extracranial large vessels beyond the axillary arteries, and this involvement may be missed if the ultrasound assessment is limited to the axilla.

Objectives: To discuss the diagnostic value of extended vascular ultrasound in identifying GCA non-axillary extracranial disease that would otherwise remain undetected with standard EULAR-recommended study limited to superficial temporal and axillary arteries, based on a clinical case report.

Materials and Methods: A single case was evaluated through the Hospital de Braga Fast-

Track GCA pathway. Clinical assessments included cranial and extracranial symptoms, ischemic features, and inflammatory markers. Vascular ultrasound was performed initially assessing superficial temporal and axillary arteries according to EULAR recommendations1 but, due to suspected large-vessel involvement, anextended examination including vertebral arteries was performed. The halo sign and increased intima–media thickness were considered indicative of vasculitis. A brief literature review was conducted on extracranial large-vessel involvement in GCA and the diagnostic role of extended vascular ultrasound.

Results

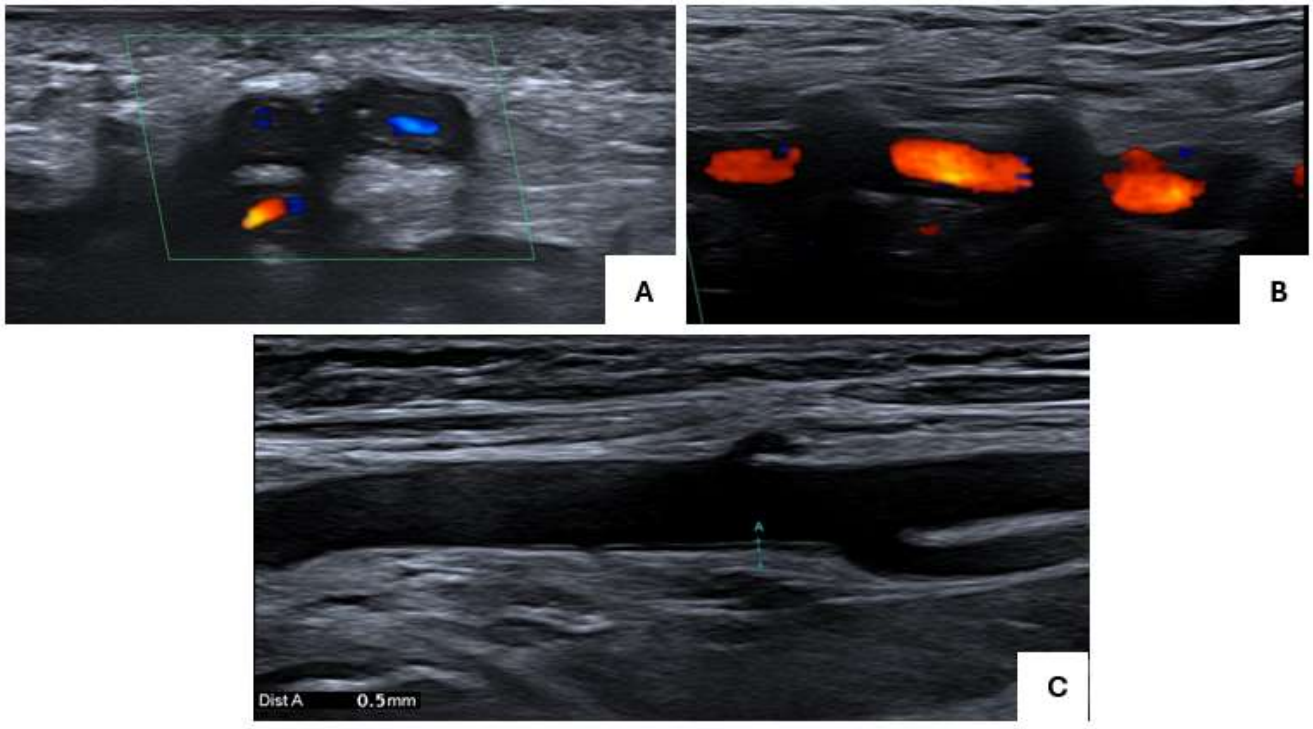
Clinical Case

A 75-year-old woman presented to the Emergency Department following an episode of amaurosis fugax. She also reported persistent, new-onset headaches for the past 3 to 4 months, as well as anorexia, weight loss, and pain with stiffness affecting the shoulder and pelvic girdles with the same duration. Laboratory findings revealed markedly elevated inflammatory markers (Erythrocyte Sedimentation Rate of 117 mm/hr; C-Reactive Protein of 124 mg/L), anemia (Hemoglobine of 10.4 g/dL), and thrombocytosis (771 $\times 10^9$/L).

Given the clinical suspicion of GCA, superficial temporal artery ultrasound was promptly performed via the Fast-Track GCA program, demonstrating a clear non-compressible halo sign in three segments – the common trunk, the frontal branch, and the parietal branch of the right superficial temporal artery.

According to the EULAR1 Guidelines, axillary arteries ultrasound was also performed, and initial axillary artery assessment was unremarkable. However, extended vascular ultrasound revealed involvement of large vessels, including the vertebral artery, where characteristic intima–media thickening and a hypoechoic halo were observed.

Figure 1 – (A) shows a hypoechoic halo in the common trunk of the right superficial temporal artery; (B) demonstrates wall thickening with a hypoechoic halo in the vertebral artery; (C) shows the axillary artery without evidence of wall thickening or halo sign, with normal intima-media thickness of 0.5



The diagnosis of GCA was established based on the combination of cranial and extracranial symptoms, elevated inflammatory markers, and clear ultrasound findings in multiple temporal artery segments and vertebral artery.

Due to ischemic ocular symptoms, treatment was initiated with three daily pulses of 500 mg of methylprednisolone, after which the patient was discharged on oral prednisone at 0.5 mg/kg. During reevaluation in outpatient

clinic, due to a recurrence of headaches and a rise in inflammatory markers, tocilizumab 8 mg/kg monthly was initiated, resulting in clinical stabilization.

Discussion/ Conclusion

Discussion: Despite the superficial temporal and axillary arteries ultrasound, only after an extended ultrasound, was it possible to observe the extracranial involvement of GCA – with vertebral artery findings, as no axillary abnormalities were detected in those arteries.

While axillary artery imaging increases sensitivity and allows baseline documentation, disease may extend to other large vessels, highlighting the need for ultrasound of additional territories such as the vertebral arteries, which may add further diagnostic and comparative value. Similar with this case, in which vertebral artery involvement was observed without axillary artery disease, a study including 133 GCA patients showed that 9% had vertebral arteries as the only large vessels affected, and 3.8% would have been missed with limited ultrasound (limited to superficial temporal and axillary arteries), highlighting the importance of an extended ultrasound examination2.

Despite having diagnosis implications (especially when there is no evidence of cranial disease) and being relevant for follow-up, the detection of extracranial involvement is not unanimously useful for therapeutic purposes and current guidelines for GCA treatment are not fully aligned: the 2021 American College of Rheumatology (ACR) recommendations3 support immunomodulator use (tocilizumab or methotrexate) in addition to steroid therapy in case of extracranial involvement, whereas the 2019 EULAR4 and British Society for Rheumatology (BSR) recommendations5 restrict their use to relapsing disease or to patients at higher risk of glucocorticoid-related toxicity, namely metabolic decompensation of diabetes mellitus or osteoporotic fragility fracture.

Thus, although the therapeutic impact of detecting extracranial involvement is not yet clearly defined, and further studies and clinical trials are needed to clarify its role, this recognition may support earlier consideration of biologic steroid-sparing strategies in selected patients.

Conclusion: While temporal and axillary artery ultrasounds are recommended for GCA diagnosis, exclusive reliance on this approach may fail to detect extracranial large-vessel involvement. This is a paradigmatic case in which exclusive imaging of the axillary artery would have missed extracranial large vessel involvement. Therefore, comprehensive ultrasound assessment beyond the axilla sometimes is essential to diagnose the extracranial involvement and to provide a baseline for follow-up, although its therapeutic implications are not yet consensually established.

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PO 20 - THE ROLE OF ANTIPHOSPHOLIPID ANTIBODIES IN SPONDYLOARTHRITIS- PROSPECTIVE OBSERVATIONAL STUDY

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Introduction. Spondyloarthritis (SpA) includes a group of inflammatory rheumatic diseases were immune dysregulation plays a central role, although pathogenesis is not fully understood. Recent studies have highlighted the presence and potential role of antiphospholipid antibodies (aPL) in SpA, adding complexity to the disease's immunological management.

Aim. To identify the presence of aPL in patients diagnosed with SpA and its relation with different clinical-epidemiological data.

Methods. A prospective observational study between September 2023 and February 2024, was conducted involving adult patients diagnosed with axial SpA (ASAS criteria) followed in rheumatology department in ULS Aveiro. Anticardiolipin and anti-ß2 glycoprotein antibodies and Lupus anticoagulant (AL) were assessed and repeated 12 weeks after, in case of positivity. Lifestyle behaviours, demographic data, comorbidities, thrombotic and obstetric events and therapeutics were collected. Disease activity was measure based on ASDAS PCR, BASDAI and CRP level (taking as a cut-off point 0.5 mg/dL) in the last consultation.

Results. A total of 141 patients were screened, 9 were excluded due to coagulation therapy and active neoplasia. The majority were women (56.1%), with a mean age of 52.2 years (+/-1.22), mean disease duration 10.8 years (+/-3.5), 62.9% were HLAB27 positive, 69.7% were classified as ankylosing spondylitis (AS) and 30.3% as non-radiographic SpA, 26.5% presented peripheral involvement. The most frequent cardiovascular risk factors were dyslipidemia in 42.4%, followed by hypertension (33.3%), obesity (15.9%), diabetes mellitus (9.1%) and hyperuricemia (3%). 40.2% were under biotechnological therapy, being anti-TNF the most common (94.3%). More than one third had

high disease activity with ASDAS>2.1 in 37.1%, BASDAI>4 in 37.4% and raised CPR levels in 36.7%. Positive aPL were found in 16.7% of the sample, of which 40.9% (6.8% of total patients) were confirmed. AL was positive in 59%, followed by IgM anti-ß2 glycoprotein in 40.9% and anticardiolipin IgM in 31.8%. None of the patients with persistently positive aPL presented thromboembolic or obstetric events. Radiographic SpA (p=0.048), obesity (p=0.045) and higher CPR levels (p=0.000) showed a statistically significant tendency to the presence of aPL.

Conclusions. This study identified the presence of aPL in 16.7% of SpA patients, particularly those with radiographic SpA, obesity, and elevated CRP levels. Despite the detection of aPL, there were no associated thromboembolic or obstetric events in patients with persistently positive aPL. These findings suggest that while aPL are present in some SpA patients and correlated with specific clinical-epidemiological factors, their clinical implications in terms of thrombotic risk require further investigation.

PO 21 - REAL-WORLD SAFETY DATA OF FIRST-LINE DRUGS FOR RHEUMATOID ARTHRITIS: INSIGHTS FROM THE PORTUGUESE REUMA.PT DATABASE

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ABSTRACT

Introduction: Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disorder that primarily affects the synovial joints, leading progressive joint destruction.1–4 Early diagnosis and a combination of pharmacologic and non-pharmacologic therapies are key to managing this chronic and debilitating disease effectively.2,5,6

Disease-modifying antirheumatic drugs (DMARDs) are key therapeutic agents for RA.7

Methotrexate (MTX) is the leading conventional DMARD (cDMARD). Biological DMARDs (bDMARDs), including tumor necrosis factor inhibitors (TNFi), are employed when treatment goals are not achieved with first-line therapies or when adverse events (AEs) associated with conventional DMARDs (cDMARDs) necessitate discontinuation.7

The use of cDMARDs and bDMARDs for RA treatment has been associated with various AE.8–13,14 High burden of AE has been associated with higher disease activity and lower likelihood of remission in early RA.15,16

Long-term observational studies and registry data are crucial for understanding the safety profiles of these agents. Although randomized controlled trials (RCTs) have identified differences in the efficacy and safety of these therapies, their findings are inherently constrained by strict inclusion and exclusion criteria. Therefore, there is a critical need to complement RCT evidence with real-world data, which offers a more representative and ecologically valid assessment of treatment safety across broader and more diverse patient populations. No real-world evidence on the safety of first-line DMARDs in patients with RA was identified in the Portuguese population.

Objectives: To assess adverse events (AE) associated with first-line therapies for rheumatoid arthritis (RA) in a real-world setting.

Material and Methods: Retrospective multicenter cohort study of patients fulfilling classification criteria for RA and followed up in 66 rheumatology centers from the Rheumatic Diseases Portuguese Registry (Reuma.pt). All AE reports associated with first-line disease-modifying antirheumatic drugs (DMARDs) up to November 2024 were included. Demographic and clinical data were analyzed, and AE characteristics were investigated. Categorical and continuous variables were compared using chi-square tests and Mann–Whitney U tests, respectively. Statistical significance was defined as $p < .05$.

Results: Among 1 880 AE entries, 377 (20.1%) were attributed to first-line DMARDs, most commonly methotrexate (62.9%) although no information on drug dosage was available. The median age at AE occurrence was 58.6 years (IQR: 19.32), and 82% were female. A

causality assessment was available in 317 reports, with 40.3% deemed "probable," 28.1% "possible," and 10.6% "definitive." Severe AE were reported in 13.2% of cases, with pulmonary involvement being 4 the most common (20.8%). Overall, 46.7% of patients discontinued treatment for any reason. Male sex was significantly associated with severe AE (OR = 2.31; 95% CI: 1.17–4.55; p = .014), and older patients were more likely to experience severe AE (median age 65.7 vs. 57.9 years; p < .001). The most affected body organ systems were gastrointestinal (9.3%), skin (8.2%), and hematological (8.2%). The median time to AE onset from treatment initiation was 1.27 years (IQR: 2.63), and from disease onset was 8.56 years (IQR: 11.76).

Discussion/Conclusions: AE related to first-line RA therapies can lead to significant clinical consequences, including treatment discontinuation. Male sex and advanced age were associated with increased AE severity. The most affected systems appear consistent with known drug safety profiles, particularly that of methotrexate; however, the absence of information regarding drug dosage precludes more detailed conclusions. These findings emphasize the need for individualized monitoring strategies and improved pharmacovigilance to optimize long-term treatment safety and adherence in RA management.

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PO CC 22 - AN UNUSUAL CULPRIT BEHIND JOINT DESTRUCTION: A CASE OF SYRINGOMYELIA-ASSOCIATED CHARCOT ELBOW

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Introduction: Neuropathic arthropathy, or Charcot joint, is a rare but severe form of joint degeneration caused by underlying neurological dysfunction. While it is most observed in the lower extremities—particularly in the context of diabetes mellitus—its manifestation in the upper limbs is uncommon. Syringomyelia is recognized as the predominant cause of Charcot joints in the upper extremity. The condition results from the formation of a syrinx, a fluid-filled cavity within the spinal cord, which disrupts pain and temperature pathways and, over time, leads to motor and sensory deficits. The ensuing joint destruction arises through neurotraumatic mechanisms—repetitive microtrauma and abnormal pressure due to proprioceptive loss—and neurovascular alterations, such as increased osteoclastic activity driven by hyperaemia.

Case report: We present the case of a 64-year-old man with a history of Chiari type I malformation and associated syringomyelia, diagnosed after several years of occipital headache and back pain. He underwent suboccipital craniectomy and C1 laminectomy in 2019. The patient had previously been diagnosed with long-standing ulnar neuropathy and underwent cubital tunnel release in 2016. He was referred due to progressive swelling and reduced mobility of the left elbow over several years, associated with only mild discomfort. Clinical examination revealed marked joint swelling, limitation of both active and passive movement (flexion-extension restricted to 30–90º), and sensory deficits in the upper limbs, predominantly on the left. Radiographs of the elbow showed severe arthrosis with extensive extra-articular ossifications, with clear radiographic progression compared to previous imaging from 2018. Laboratory tests showed no elevation of inflammatory markers, negative rheumatoid factor and anti-CCP antibodies, and normal calcium/phosphate metabolism. Based on the radiographic findings, clinical history, and known syringomyelia, a diagnosis of neuropathic arthropathy of the elbow secondary to syringomyelia was made.

Discussion: Charcot elbow is an uncommon but important differential diagnosis in patients presenting with monoarticular swelling and functional impairment, especially when associated with ulnar nerve

involvement (e.g., paraesthesia, atrophy, clawing). Up to 50% of patients report pain, although many remain painless. Imaging is central to diagnosis: plain radiographs often reveal advanced joint destruction and heterotopic ossifications, while cervical MRI is essential for identifying a syrinx in undiagnosed patients. Management should be individualized, focusing on joint protection and treatment of the underlying neurological condition. In the presence of a syrinx, neurosurgical decompression is indicated to halt progression.

Conclusion: This case highlights the importance of considering neuropathic arthropathy of the elbow in the differential diagnosis of chronic monoarthritis with atypical radiographic features, particularly in patients with known syringomyelia or with suggestive symptoms, such as occipital headache exacerbated by Valsalva manoeuvre, back pain, or radicular symptoms.

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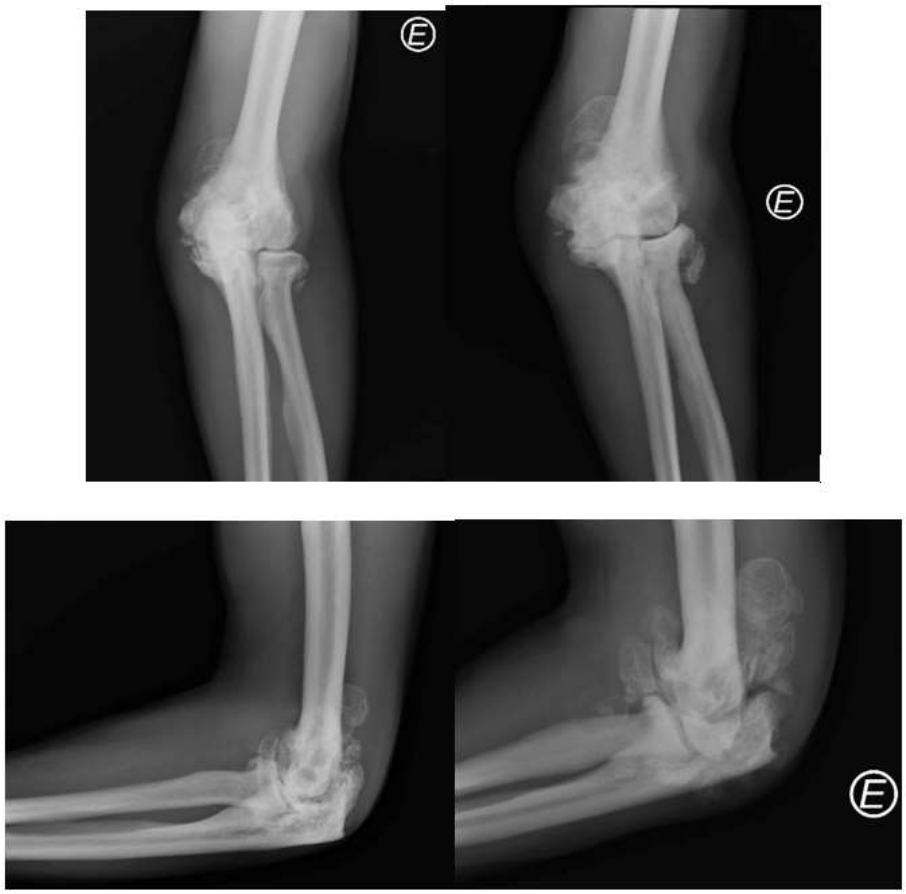
Imagem 1: Radiografia da articulação do cotovelo que demonstra evolução de 2018 (à esquerda) para 2025 (à direita)



PO CC 23 - SILENT ADRENALS, CRACKING BONES: A GLUCOCORTICOID-NAIVE CAH CASE WITH OSTEOPOROSIS

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Introduction: Congenital adrenal hyperplasia (CAH) is an autosomal recessive disorder, most commonly (over 90% of cases) caused by 21-hydroxylase deficiency (21OHD). This enzymatic defect impairs the conversion of 17-hydroxyprogesterone to 11-deoxycortisol, leading to reduced cortisol synthesis and a compensatory increase in adrenocorticotropic hormone (ACTH) secretion. Chronic ACTH stimulation results in adrenal hyperplasia and excessive androgen production. The clinical severity of CAH correlates with the extent of residual 21-hydroxylase activity, ranging from severe to mild presentations.

Low bone density index (BDI) is a well-recognized concern in both children and adults with CAH, often attributed to prolonged exposure to supraphysiological doses of glucocorticoids (GC). However, the role of GC therapy in bone loss remains controversial, and additional contributing factors such as low body mass index and impaired growth during childhood have been proposed.

We report a case of osteoporosis in a glucocorticoid-naive male patient with undiagnosed CAH, highlighting alternative mechanisms of bone fragility in this context.

Case report: A 62-year-old male was referred to rheumatology for persistent osteoporosis despite three years of treatment with alendronate and cholecalciferol. Dual-energy X-ray absorptiometry revealed a lumbar spine T-score of –3.1 and a femoral neck T-score of –2.0. He had sustained a right femoral fracture at age 49 following a fall from a stepladder but denied any additional fractures or height loss. He reported no history of smoking and occasional alcohol consumption.

His medical history was notable for precocious puberty, infertility, reduced libido, and absence of morning erections. There was no family history of hip fractures or hereditary diseases. His adult height was 145 cm. On physical examination, he presented with small testes within the scrotum and otherwise normal external genitalia.

Hormonal workup revealed elevated ACTH (84.7 pg/mL), markedly increased 17-hydroxyprogesterone (90.1 ng/mL), low total testosterone (148 ng/dL), and low morning cortisol (4.9 µg/dL). A Synacthen stimulation test confirmed adrenal insufficiency, with a peak cortisol of 7.7 µg/dL. Testicular ultrasound identified bilateral, irregularly hyperechoic lesions, consistent with testicular adrenal rest tumours (TARTs).

Genetic analysis identified a homozygous pathogenic variant in the **CYP21A2** gene [c.1273G>A p.(Gly425Ser)], confirming the diagnosis of classic congenital adrenal hyperplasia (CAH).

The patient was started on hydrocortisone replacement therapy and antiresorptive treatment with denosumab.

Conclusion: This case highlights an uncommon presentation of CAH, diagnosed in late adulthood in a glucocorticoid-naive patient with severe osteoporosis. It challenges the notion that bone loss in CAH is solely attributable to glucocorticoid therapy and suggests a multifactorial aetiology, potentially involving chronic androgen excess, hypogonadism, and early growth disturbances. Clinicians should maintain a high index of suspicion for CAH in patients with unexplained

osteoporosis and signs of long-standing hormonal imbalance.

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PO CC 24 - ISOLATED RHEUMATIC MANIFESTATIONS IN WHIPPLE DISEASE: AN EARLY DIAGNOSIS?

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Introduction: Whipple's disease is a rare systemic infection caused by Tropheryma whipplei, mostly affecting middle-aged Caucasian men. Associated with poor hygiene and wastewater exposure, it classically evolves in three phases: an early phase (<6 years) with arthralgia and fever; a middle phase (6–8 years) with diarrhoea, abdominal pain and weight loss; and a late phase (>8 years) marked by neurological symptoms. Since seronegative arthritis and/or arthralgia are often early isolated manifestation, misdiagnosis as a rheumatic disease — particularly seronegative rheumatoid arthritis and spondylarthritis — is common, delaying correct diagnosis by an average of 6.7 years.

Case report: We present the case of a 50-year-old Caucasian male with a 1.5-year history of symmetric additive polyarthralgia involving the metacarpophalangeal joints, wrists, elbows, knees, ankles, and feet. The patient presented with episodes of worsening pain and swelling that significantly impaired his gait Laboratory tests revealed elevated inflammatory markers, with a C-Reactive Protein (CRP) of 4mg/dL and an Erythrocyte Sedimentation Rate (ESR) of 53 mm/h. Musculoskeletal ultrasound showed mild-to-moderate synovitis in small joints of the hands and feet.

The patient underwent sequential treatment with methotrexate, deflazacort, colchicine, leflunomide over 1.5 years, without significant clinical improvement. He was then treated with adalimumab for a month, with clear worsening and immediate treatment suspension.

Extensive immunologic, infectious, and oncologic workup was negative. During this work-up, Helicobacter pylori was isolated in gastric biopsies and treated with bismuth, metronidazole and tetracyclines, with transitory improvement. Nearly two years after initial evaluation, synovial fluid from the extensor tendon sheath of the left hand was sent for analysis and tested positive for Tropheryma whipplei by PCR. Upper gastrointestinal endoscopy confirmed the diagnosis through positive duodenal PCR.

Immunosuppressive therapy was discontinued, and the patient received intravenous ceftriaxone (2g/day for 15 days), followed by oral sulfamethoxazole/trimethoprim (800/160 mg twice daily). Clinical improvement was rapid, with significant reduction in joint swelling and restoration of gait function. Laboratory markers also improved, with CRP decreasing from 6.2 to 0.75 mg/dL within five days of antibiotic therapy initiation.

After one month, a slight worsening of ankle arthralgia was noted, accompanied by a mild elevation of CRP (3.29 mg/dL). An immune reconstitution inflammatory syndrome (IRIS) was assumed and managed successfully with a low dose of prednisolone (5 mg/day). At three-month follow-up, the patient remained asymptomatic, and corticosteroid tapering was initiated.

Conclusion: Timely recognition of Whipple's disease is crucial in patients presenting with atypical rheumatologic symptoms, particularly when subtle systemic signs or elevated inflammatory markers are also present. Worsening arthritis or new systemic manifestations following immunosuppressive treatment should raise clinical suspicion. Including Whipple's disease in the differential diagnosis of persistent seronegative arthritis may enable earlier identification and timely initiation of antibiotic therapy, thus reducing the risk of disease progression and complications. Given its ability to mimic other rheumatic conditions, and the potential harm of delayed diagnosis, rheumatologists should be watchful of this rare but treatable disease.

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PO CC 25 - BEYOND THE USUAL SUSPECTS: CRYPTOSPORIDIUM AS A RARE CAUSE OF DIARRHOEA IN SLE

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Introduction: Systemic lupus erythematosus (SLE) is a complex autoimmune disease characterized by multisystem involvement and unpredictable flares. While disease activity is often idiopathic, infectious triggers — particularly in immunosuppressed individuals — may precipitate flares and complicate management. Cryptosporidium spp. is a protozoan parasite known to cause gastrointestinal infections, especially in immunosuppressed hosts. However, its potential to trigger diarrhoea and a lupus flare has not, to our knowledge, been previously reported in the literature.

Case report: We present the case of a 21-year-old female with a recent diagnosis of systemic lupus erythematosus (SLE), with cutaneous, articular, hematologic, and renal involvement (class II lupus nephritis). She was under treatment with hydroxychloroquine 400 mg, mycophenolate mofetil 1000 mg BID, and prednisolone 60 mg.

Shortly after an increase in the dose of mycophenolate, she developed profuse watery diarrhoea, initially presumed to be drug related. Despite dose reduction and oral rehydration, symptoms worsened, prompting

emergency department admission. Laboratory results revealed acute kidney injury (AKIN stage 2), leucocytosis with neutrophilia, hyponatremia, and hyperkalaemia, raising concerns for an infectious trigger and a possible lupus flare, further supported by the presence of a malar rash.

Due to persistent symptoms, low-grade fever, and worsening cutaneous lesions, hospital admission was warranted. Mycophenolate was suspended, empiric antibiotic therapy was initiated, and stool studies were performed, revealing a positive Cryptosporidium spp. antigen.

Following discussion with Infectious Diseases, tentative treatment with nitazoxanide 500 mg BID was started, along with intravenous immunoglobulin (2 g/kg over 3 days). The patient showed marked clinical improvement within 48 hours, including reduced bowel frequency, improved fatigue, and resolution of the cutaneous flare. Renal function also normalized rapidly, suggesting that the acute kidney injury was more likely due to dehydration and infection rather than a lupus nephritis flare — the latter of which could only be confirmed by renal biopsy.

Conclusion: This case represents a rare — and possibly first-reported — instance of an SLE flare precipitated by Cryptosporidium-associated gastroenteritis. It highlights the importance of considering Cryptosporidium spp. as a potential trigger for both diarrhoea and disease activity in patients with lupus. Moreover, it supports the off-label use of nitazoxanide as a therapeutic intervention in selected cases involving immunocompromised patients. Recognizing such associations is crucial for guiding timely diagnosis, targeted infectious screening, and personalized therapeutic interventions in autoimmune patients presenting with gastrointestinal symptoms.

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PO CC 26 - THE WRISTS THAT WOULDN'T HEAL: REFRACTORY STILL'S DISEASE REQUIRING BILATERAL ARTHRODESIS

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Introduction: The management of Still's disease has evolved significantly with the introduction of biologic disease-modifying anti-rheumatic drugs (bDMARDs), particularly IL-1 and IL-6 inhibitors, which have demonstrated efficacy in both systemic and articular phenotypes. Despite therapeutic advances, a subset of patients may develop refractory disease, particularly with persistent articular involvement, leading to irreversible joint damage and functional disability. To our knowledge, this is the first reported case of a patient with Still's disease with articular refractory, destructive disease requiring total wrist arthrodesis.

Case report: We report the case of a 44-year-old man

diagnosed with Still's disease in 2019. Initial symptoms included headache, odynophagia, generalized myalgia, fever, and inflammatory polyarthralgia involving the shoulders, wrists, and knees. He also reported abdominal discomfort and a transient erythematous

rash predominantly on the trunk. Cardiac symptoms prompted further evaluation, revealing myopericarditis. Additionally, he developed a pleural effusion, presenting with dyspnoea and peripheral desaturation, requiring oxygen supplementation at 3 L/min.

Initial therapy with methotrexate, corticosteroids, and colchicine proved insufficient to control disease activity, and multiple bDMARDs were sequentially introduced. Anakinra (Dec 2019–Aug 2020) was discontinued due to secondary failure. Tocilizumab (Aug 2020–Jan 2021) was used without adequate control of oligoarthritis and persistent hyperferritinaemia. Canakinumab (Apr 2021–Mar 2023) achieved systemic control but with ongoing articular activity. After a brief suspension, Canakinumab was reintroduced (Nov 2023–Apr 2024), again with limited articular response. Rituximab (from Apr 2024) and adalimumab (May–Jun 2024) also failed to provide clinical benefit. Infliximab (from Sep 2024) was initiated with good systemic tolerance but persistent inflammatory arthritis of the wrists. Disease control was further complicated by two infectious events: a hepatitis B virus infection and pneumocystosis (both in 2024), which delayed optimization of immunosuppressive therapy.

By early 2025, the patient presented with marked functional impairment and persistent pain in both wrists. MRI revealed advanced osteoarthritis with cartilage loss and multiple erosions, rendering radiosynovectomy unfeasible. Although the initiation of JAK inhibitors was considered, it was ultimately dismissed due to the extent of joint destruction and severe disability, which were judged to outweigh the potential therapeutic benefits. Considering the refractory articular disease and irreversible structural damage, total wrist arthrodesis was proposed. The procedure was successfully performed on the right

wrist in June 2025, with the left wrist surgery planned subsequently. Routine blood tests at the time of surgery were unremarkable, including inflammatory markers.

Discussion: Although Still's disease is often responsive to immunosuppressive therapy, a small subset of patients develops refractory articular manifestations despite multiple lines of targeted biologic treatment. In this case, the patient showed inadequate articular response to IL-1 and IL-6 inhibitors, anti-CD20, and TNF inhibitors, ultimately progressing to severe bilateral wrist damage over a 6-year disease course. Joint destruction in Still's disease is rarely reported, and this case underscores the potential for aggressive, treatment-resistant articular involvement despite optimal pharmacologic management. To the best of our knowledge, this is the first reported case of Still's disease requiring total wrist arthrodesis due to persistent arthritis and advanced joint destruction.

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PO CC 27 - MORE THAN A GOUT FLARE AN ATYPICAL PRESENTATION OF POLYARTERITIS NODOSA

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Introduction: Polyarteritis nodosa (PAN) is a systemic necrotizing vasculitis of medium- and small-sized arteries. Diagnosis is challenging, especially when features are non-specific or overlap with prior

conditions. Recognizing unusual patterns of multisystem involvement becomes critical for early and effective treatment.

Case description: A 66-year-old man with long-standing tophaceous gout and chronic alcoholism was

transferred to Rheumatology on day 12 of hospitalization. The patient presented with migratory joint pain, fatigue, fever (38.9°C), new-onset atrial fibrillation, bilateral pleural effusion, and type I respiratory failure. Cardiac evaluation showed preserved ventricular function without evidence of endocarditis. Laboratory tests revealed marked systemic inflammation (CRP >600 mg/L, ESR 140 mm/h, neutrophilic leukocytosis, thrombocytosis), normocytic anemia, and cholestatic liver enzyme abnormalities. Despite broad-spectrum antibiotics, fever and joint symptoms persisted. Upon rheumatologic evaluation, he exhibited multiple warm, swollen, and tender joints - elbows, wrists, knees, ankles, and midfoot. Arthrocentesis of the right knee yielded thick yellow-brown fluid with negatively birefringent crystals, consistent with monosodium urate. Antibiotics were discontinued, and treatment with oral prednisolone \ ESR remained elevated. Immunologic tests (ANA, ANCA, RF, ACPA) were negative, with no complement consumption. Extensive infectious screening was also negative.

Two months later, he returned with recurrent polyarthritis, new-onset abdominal pain, severe fatigue, 18 kg weight loss, and distal dysesthesias, described as burning and thermal hypersensitivity in the hands and feet, more pronounced on the right side. Neurological examination revealed asymmetric distal sensorimotor deficits, with weakness of the right fifth finger and toe flexors (3/5), and reduced sensation in a stocking distribution. Electromyography showed asymmetric sensorimotor polyneuropathy with axonal/demyelinating features, consistent with mononeuritis multiplex.

As part of the workup for occult malignancy, cervical CT additionally revealed enlargement and heterogeneous enhancement of the left trapezius. MRI confirmed

increased volume and a linear pattern of high T2 signal intensity. Whole-body 18F-FDG PET/CT demonstrated patchy muscular hypermetabolism, mimicking a pseudotumor. No malignancy or relevant pulmonary

infiltrates or nodules were observed.

The pattern of multisystem involvement, suggestive of medium-vessel vasculitis, supported a clinical diagnosis of PAN after exclusion of mimickers. Immunosuppressive induction therapy was initiated with high-dose oral corticosteroids and intravenous cyclophosphamide (CYCLOPS protocol). At the time of this report, after five cycles, the patient improved clinically and analytically, with stable neurological deficits.

Conclusion: This case illustrates the diagnostic challenge of atypical PAN and the growing importance of FDG-PET. We report a complex presentation of this disease, initially confounded by a coexisting crystal arthritis flare. The emergence of abdominal pain, systemic symptoms and mononeuritis multiplex redirected the workup. FDG-PET/CT proved to be crucial, by excluding mimics and revealing vasculitic muscle involvement in the absence of visceral or cardiac findings.

PO CC 28 - TWO VIRUSES AND A VASCULITIS

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Introduction: Polyarteritis nodosa (PAN) is a rare systemic necrotizing vasculitis affecting medium-sized arteries, typically presenting with multisystem involvement including skin, musculoskeletal,

neurological, renal, and gastrointestinal manifestations [1,2] Diagnosis is confirmed by histopathology or angiography. PAN is occasionally associated with viral infections, particularly hepatitis B virus (HBV), and immunocompromised states may alter disease presentation and course. We report a case of PAN in a

patient with coexisting HIV infection on antiretroviral therapy and chronic hepatitis B virus infection, highlighting diagnostic and therapeutic considerations [1,2]

Clinical case: A 46-year-old female, born in Guinea-Bissau, had been followed up since 2022 for mechanical joint pain, dactylitis, and tenosynovitis suggestive of spondyloarthritis. In 2024, she was diagnosed with HIV infection (under antiretroviral therapy) and showed evidence of past hepatitis B infection (HBsAg negative, anti-HBc and anti-HBs positive, undetectable viral load).

Later that year, she developed painful nodular lesions on the lower limbs, clinically compatible with panniculitis, accompanied by bilateral knee arthritis. Laboratory tests revealed an ESR of 107 mm/h and CRP of 0.8 mg/dL, with negative serologies for anti-citrullinated peptide antibodies, rheumatoid factor, anti-nuclear antibodies and antineutrophil cytoplasmic antibodies. HLA-B27 was also negative. Syphilis and hepatitis C serologies were negative. IGRA was initially indeterminate (2024) but converted to positive in January 2025, prompting treatment for latent tuberculosis. Arthrocentesis was non-inflammatory.

A skin biopsy of the nodular lesions (November 2024) demonstrated a medium-sized artery with fibrin deposition and intravascular thrombus, along with a mixed lymphocytic and neutrophilic infiltrate in the vessel wall, suggestive of polyarteritis nodosa (PAN).

In February 2025, she developed a painful red left eye, and ophthalmologic evaluation confirmed scleritis (March 2025). She was started on systemic and topical corticosteroids with partial improvement. After confirmation of undetectable HIV and HBV viral loads, methotrexate was introduced at 10 mg/week and later titrated, leading to resolution of articular and ocular symptoms and allowing gradual glucocorticoid tapering.

Based on clinical, laboratory, and histological findings, a diagnosis of polyarteritis nodosa was established in the setting of HIV infection and previous HBV exposure. To exclude peripheral neurological involvement, an electromyography was performed, showing no abnormalities.

Discussion: This case underscores the diagnostic and therapeutic challenges of managing vasculitis in patients with chronic viral infections. Polyarteritis nodosa may occur in association with both hepatitis B virus and HIV, and distinguishing idiopathic from infection-related forms is essential, as management must be tailored to underlying comorbidities1. Close coordination between Rheumatology, Infectious Diseases, and Ophthalmology allowed effective control of vasculitic activity while maintaining viral suppression.

Conclusion: This case illustrates the importance of maintaining a high index of suspicion for systemic vasculitis in patients with chronic viral infections presenting with multisystem involvement. Early histological confirmation and multidisciplinary management were crucial to achieve disease control while ensuring the safety of immunosuppressive therapy.

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PO CC 29 - CHANGES IN PSORIATIC ARTHRITIS PHENOTYPE AND ITS THERAPHEUTIC CONSEQUENCES – A CASE SERIES

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Introduction: Psoriatic arthritis (PsA) is heterogeneous, with different clinical phenotypes that can evolve or overlap over time. Other musculoskeletal (MSK) and

extraarticular (EAr) manifestations enhance this diversity. Therapeutic choices must consider each manifestation as well patient co-morbidities. When biologic disease-modifying anti-rheumatic drugs (bDMARD) are required, its choice must rely on both MSK and EAr manifestations. Clinically relevant skin psoriasis should prompt treatment with anti-IL-17A, anti-IL-17A/F, anti-IL-12/23 or anti-IL-23; axial involvement with IL-17A, IL-17A/F, anti-TNF or JAKi and uveitis with anti-TNF. Nail psoriasis has shown to respond best to IL-17A and JAKi.

Clinical cases: We present three clinical cases of patients with PsA changing phenotype along disease course. Case 1: 40-year-old female with positive HLA-B27 and rheumatoid-like PsA diagnosed at age 28, initially treated with methotrexate (MTX) and etanercept (ETN) with good response. At age 32 (in 2017), switch of current treatment to sulfasalazine was made due to pregnancy. In post-partum, she presented with phenotype change to axial disease with mild scalp psoriasis and anterior uveitis, requiring start of adalimumab (ADA) with clinical remission since then but no response in psoriasis. Case 2: 39-year-old male with axial PsA diagnosis at age 32 with mild scalp psoriasis, in disease remission under monotherapy with secukinumab (SEC) 150mg for 4 years. At age 38, after dose interval extension and becoming a father, patient presented with asymmetric oligoarthritis and dactylitis, enthesitis and two episodes of anterior uveitis, as well as psoriasis exacerbation. SEC was increased to PsA dose and subcutaneous MTX 20mg was initiated, with no response obtained. Switch to ADA 40mg and leflunomide (LEF) 20mg showed initial response but loss of efficacy at 7 months and patient has recently started tofacitinib, maintaining LEF 20mg. Case 3: 46-year-old male with distal interphalangeal predominant PsA, nail dystrophy and skin psoriasis that, after 5 years responding to SEC in PsA dose adjusted to weight, changed phenotype to asymmetric oligoarthritis with dactylitis, enthesitis and skin psoriasis. At this point, switch to certolizumab (CZP) in psoriasis dose was made considering articular involvement was the main issue and patient's intolerance to MTX and LEF. At 6 months evaluation, partial articular response was obtained but psoriasis showed no response.

Discussion: Young age, HLA-B27 antigen and post-partum hormonal variations present in the female patient are described in literature as risk factors to change into axial disease and uveitis occurrence.

However, other variables present in our patients as psychologic stress, type of bDMARD and bDMARD discontinuation or increase in dose interval do not influence phenotype changes. In the female patient, ETN was discontinued because there were yet no international recommendations supporting its safety in pregnancy and choice of anti-TNF for bDMARD reinitiation was due to its efficacy in uveitis and axial involvement. In case 2 first choice of SEC 150mg was due to axial disease and no relevant skin involvement. After that, peripheral involvement and psoriasis exacerbation prompt dose increase of SEC to 300mg and start of MTX, uveitis and enthesitis motivated ADA as first switch choice and second switch choice was tofacitinib as other classes failed. Case 3 patient was switched to CZP due to no clinically relevant psoriasis at the time and preference of CZP for monotherapy in a patient with csDMARD intolerance. Recognition of different PsA phenotypes and manifestations is key to adequate treatment of each patient.

PO CC 30 - RS3PE SECONDARY TO METASTATIC BREAST CANCER

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Introduction: Remitting, seronegative, symmetric synovitis with pitting edema (RS3PE) is a syndrome with an acute onset and benign course characterized by rheumatoid factor seronegativity, pitting edema on the dorsum of the hands and feet, symmetric distal synovitis, and flexor tendinitis on the fingers. An important characteristic of RS3PE is the association with hematological and solid malignancies, as well as

rheumatic diseases. PRESENTATION OF CASE: An 80-year old woman was referred to the Rheumatology appointment for a two-month history of inflammatory arthralgia predominantly involving the small joints of hands and wrists, as well as bilateral dorsal hand edema. Laboratory investigations revealed normocytic, normochromic anemia, elevated erythrocyte sedimentation rate of 60 mm/h, and a C-reactive protein level of 0.28 mg/dL. Immunological studies were negative for rheumatoid factor, anti-cyclic citrullinated peptide antibodies, antinuclear antibodies, and extractable nuclear antigens. Complement levels (C3 and C4) were within normal limits. Empiric treatment with prednisolone and methotrexate was initiated.

Approximately one month after the initial rheumatologic assessment, screening mammography and breast ultrasound revealed right axillary lymphadenopathy. Histopathological analysis of a lymph node biopsy demonstrated metastatic poorly differentiated invasive carcinoma, grade 3, immunohistochemically consistent with a breast primary cancer (positive for GATA-3 and E-cadherin), estrogen receptor negative, HER2 positive (3+ by immunohistochemistry, confirmed by FISH), with a Ki-67 50%.

Breast MRI did not demonstrate any suspicious enhancement in the mammary parenchyma, while abdominal MRI revealed findings compatible with diffuse hepatic metastases. The patient began systemic therapy with paclitaxel, pertuzumab, and trastuzumab. DISCUSSION: The present clinical case demonstrates that RS3PE can associate with an underlying tumor. In the presence of this rare syndrome, a high level of clinical suspicion is necessary to identify patients at risk. Although the pathogenic mechanism is not clear, it could involve a paraneoplastic polyarthritis linked to the synthesis of humoral factors such as IL-6. The association between this syndrome and malignancy is seen in approximately 6% to 20% of patients, most frequently adenocarcinomas (prostate, stomach, colon and lung), non-Hodgkin lymphomas and chronic lymphocytic leukemia. Our case shares multiple features with those described in the literature, including age, clinical presentation and association with underlying malignancy. The interval between onset of RS3PE and diagnosis of cancer was fairly long (2 months to 14 years) suggesting that these patients should be screened with appropriate age and sex specific surveillance for an extended period, if not indefinitely. **Conclusions:** In conclusion, RS3PE is a syndrome with a benign course. Patients with specific features, such as lack of response to corticosteroids, systemic symptoms (weight loss, fever) and unexplained anemia, should raise the suspicion for an underlying malignant disease. Correct identification of these cases and patient follow-up after diagnosis are important.

Prospective follow up studies on larger cohorts would help clarify long-term risk of neoplasia in patients with RS3PE syndrome.

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PO 31 - SEXUAL DYSFUNCTION IN FEMALE PATIENTS WITH SYSTEMIC SCLEROSIS: PRELIMINARY DATA FROM A MULTICENTRE STUDY

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Background: Sexual dysfunction (SD) is common in female patients with systemic sclerosis (SSc), with a reported prevalence of up to 86.6%. [1-10] Several factors may contribute to SD in those patients. Sexual distress in patients with SSc is often not adress and can affect more than a third of patients. [11]

Aim: To evaluate and compare SD and sexual distress in women with SSc to age-matched healthy controls (HC) and to determine the potential impact of clinical features on sexual function and sexual distress.

Methods: We performed a multicentre cross-sectional study including women (29-83 years-old) with a clinical diagnosis of SSc and HC (23-68 year-old) who attended general practitioner. Participants were invited to complete an anonymous paper questionnaire assessing demographics, sexual function [Female Sexual Function Index (FSFI)], sexual distress [Female Sexual Distress Scale-Revised (FSDS-R)], gynecological/obstetric history, and symptoms of anxiety/depression [Hospital Anxiety and Depression Scale (HADS)]. Additionally, in the SSc group, disease characteristics, quality of life, symptoms of depression and anxiety [Hospital Anxiety and Depression Scale], and additional questions regarding sexual life were collected.

SD was defined as FSFI<26.5 and sexual distress was defined as FSDS-R≥11.

Comparisons were made using T-tests, Mann-Whitney and Chi-squared tests with a significance threshold of $p<0.05$.

Results: A total of 49 SSc female patients [mean age±SD: 58.41±13.87 years] and 12 HC [47.33± 2.46 years; p=0.015] were included. Among SSc patients, 46 (94%) fulfilled the 2013 ACR/EULAR classification criteria, with a mean disease duration of 16.67±11.60 years. Clinical subtypes were: limited cutaneous SSc in 26 (53%), diffuse cutaneous SSc in 15 (31%), SSc sine scleroderma in 3 (6%), Very Early Diagnosis of Systemic Sclerosis in 2 (4%), and SSc-overlap syndromes in 3 (6%) patients.

SSc patients had fewer years of education [median (IQR): 12.00 (6.00) vs 14.50 (6.00); p=0.009]. No significant differences were found in obstetric history or marital status between the groups (see Table 1). SSc patients had significantly lower FSFI scores [11.40 (16.60) vs 31.40 (4.65); p<0.001], with lower scores across all six domains. SD (FSFI<26.55) was present in 42/46 (91%) of SSc patients vs 0/10 of the HC (p < 0.001). Sexual distress (FSDS-R≥11) was common in both groups [23/42 (55%) vs 5/12 (42%); p=0.423].

Among SSc patients, 19/45 (42%) reported disease-related impact on their sexuality. The main reasons for reduced sexual activity were SSc-related symptoms (12/37 patients, 32%), lack of a partner (30%), and personal choice (19%). Of those reporting SSc-related causes, most cited vaginal or oral dryness (16/35

patients, 46%), followed by fatigue (29%), dyspareunia (17%), dyspnoea (17%), reduced pelvic mobility (14%), hand disability or digital ulcers (14%), depression (14%),

altered body image (11%), vaginal stenosis (6%), and abdominal pain (3%); and 5/37 (14%) revealed other reasons. Only 2/46 (4%) SSc patients had ever discussed sexual issues with their rheumatologist, although 22/38 (58%) expressed a desire to do so.

Conclusion SD is highly prevalent in women with SSc, with significantly lower sexual function scores across all domains compared to HC. Although sexual distress was also common, no significant difference was found between groups. These findings underscore the need for clinicians to address sexual health in SSc care to ensure a holistic approach.

Study limitations include small sample size and demographic diferences between groups, further studies are needed to confirm these results..

	All patients (N=61)	SSc patients (N=49)	Healthy-control patients (N=12)	*p-value**
Demographics				
Current age, years - mean±SD	56.23±14.21	58.41±13.87	47.33±12.46	**0.015**
BMI, Kg/m^2 - median (IQR)	23.73 (4.74)	23.66 (4.64)	24.39 (6.69)	0.293
Education$^{\Delta}$, years - median (IQR)	12.00 (7.00)	12.00 (6.00)	14.50 (6.00)	**0.009**
Current employment status, n (%)				**0.002**
Student	1 (2)	0	1 (8)	
Housemaid	2 (3)	2 (4)	0 (0)	
Employed full-time	26 (43)	15 (31)	11 (92)	
Employed part-time	1 (2)	1 (2)	0 (0)	
Prolonged medical leave	2 (3)	2 (4)	0 (0)	
Unemployed	9 (15)	9 (18)	0 (0)	
Retired	20 (33)	20 (41)	0 (0)	
Marital status, n (%)				0.346
Single	9 (15)	7 (14)	2 (17)	
Married/cohabitation	37 (61)	28 (57)	9 (75)	
Divorced	4 (7)	3 (6)	1 (8)	
Widow	11 (18)	11 (22)	0 (0)	
Habits				
Physical activity$^{\Delta}$, n (%)				**0.001**
Sedentary	25 (43)	25 (54)	0 (0)	
Once a week	20 (34)	15 (33)	5 (42)	
2 to 3 times a week	8 (14)	4 (9)	4 (33)	

4 or more times a week	5 (9)	2 (4)	3 (25)	
Gynecological and obstetric history				
Postmenopausal status, n (%)	40 (66)	35 (71)	5 (42)	0.088
Age at menopause$^{\Delta}$, years – median (IQR)	50.00 (5.00)	49.00 (5.00)	54.00 (6.00)	**0.025**
Number of pregnancies – median (IQR)	2.00 (1.00)	2.00 (6.00)	2.00 (2.00)	0.489
Number of deliveries – median (IQR)	1 (2.00)	2 (2.00)	0 (2.00)	0.075
Number of caesarean sections - median (IQR)	0 (1.00)	0 (1.00)	0 (1.00)	0.567
Number of episiotomies – median (IQR)	0 (1.00)	0 (1.00)	0 (1.00)	0.938
Sexual health history				
FSFI$^{\beta}$ - median (IQR)	15.50 (23.25)	11.40 (16.60)	31.4 (4.65)	**<0.001**
Desire	2.40 (2.40)	1.60 (1.80)	3.60 (1.35)	**0.008**
Arousal	1.95 (4.05)	1.20 (3.40)	5.40 (1.28)	**<0.001**
Lubrification	2.55 (5.40)	0.95 (3.30)	6.00 (0.15)	**<0.001**
Orgasm	2.40 (5.20)	1.20 (3.60)	6.00 (0.50)	**<0.001**
Satisfaction	4.00 (3.30)	3.20 (2.80)	5.60 (0.90)	**<0.001**
Pain	2.00 (5.60)	1.20 (3.30)	6.00 (0.50)	**<0.001**
FSFI< 26.55$^{\beta}$ - n(%)	42 (75)	42 (91)	0 (0)	**<0.001**
FSDS-R$^{\beta}$ - median (IQR)	12 (21.00)	13 (24.00)	10 (11.00)	0.504
FSDS-R ≥11$^{\beta}$- n (%)	28 (52)	23 (55)	5 (42)	0.423

$^{\Delta}$Missing data <5%;$^{\beta}$Missing data <20%.

*Independent samples t-test or Mann-Whitney test for continuous variables, and Chi-square test for categorical variables. p-value considered significant if < 0.05.

Exclusion criteria: pregnancy, <6 months post-partum, nursing/breastfeeding, congenital or iatrogenic female genital tract diseases (e.g. pelvic radiotherapy), endocrine dysfunction not related to SSc (e.g. hypogonadism, hypopituitarism), active gynecological and/or breast cancer, cardiac failure NHYA grade III or higher, hepatic failure, unstable angina pectoris or any other inflammatory rheumatic disease in the control group.

BMI, Body Mass Index; FSDS-R, Female Sexual Distress Scale-Revised; FSFI, Female Sexual Function Index; IQR, Interquartile Range; SD, Standard Deviation; SSc, Systemic Sclerosis.

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PO CC 32 - DOENÇA RELACIONADA COM IGG4 - ATÉ ONDE VÃO AS SUAS COMPLICAÇÕES?

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ULS Cova da Beira

Introdução : A doença relacionada com IgG4 é uma patologia inflamatória sistémica rara, capaz de mimetizar diversas doenças, nomeadamente neoplasias pancreáticas, e conduzir a intervenções cirúrgicas com potenciais complicações. O reconhecimento e diagnóstico precoces são cruciais para uma abordagem terapêutica adequada, permitindo reduzir significativamente a morbimortalidade associada.

Caso clínico: Homem de 70 anos, com antecedentes de hipertensão arterial e doença relacionada com IgG4 com envolvimento multiorgânico (pancreatite autoimune, colangite, envolvimento renal e ganglionar abdominal) diagnosticada após pancreatectomia cefálica por suspeita de neoplasia pancreática. Consequentemente desenvolveu diabetes mellitus tipo 3c insulinotratada.

Com seguimento prévio noutra instituição, foi observado pela primeira vez em Reumatologia com um quadro com 3 meses de evolução caracterizado por anasarca progressiva, diarreia crónica (cerca de 5 dejeções diárias, de coloração amarelada) e queixas sicca em agravamento. O estudo em internamento revelou anemia macrocítica, trombocitopenia (Hb 9.5 g/dL, VCM 102.7 fL, Plaq 141 x 10^3/uL), défices iónicos e vitamínicos (hipocalcémia, hipomagnesémia e hipovitaminose D), insuficiência pancreática exócrina grave (elastase fecal <15 ug/g), hipoalbuminémia marcada (Alb 1.8 g/dL), elevação ligeira de VS e PCR (25 mm/H e 2.05 mg/dL) e proteinúria 24h de 120 mg/24h. TC TAP prévia revelava espessamento parietal do ramo biliar principal e ramos segmentares. Considerando o diagnóstico de base, clínica e ECD's atuais, assumida doença em atividade. Iniciou prednisolona 25mg id, terapêutica diurética e albumina endovenosas, suplementação enzimática pancreática, correção iónica e vitamínica, com evolução clínica favorável e perda ponderal significativa (~16Kg). Teve alta a aguardar realização de exames endoscópicos para esclarecimento adicional de anemia e hipoalbuminémia observados.

Um mês depois, foi reinternado por agravamento de anemia (Hb 6.5 g/dL), agudização de doença renal crónica (creat 2.16 mg/dL, ureia 51 mg/dL) e citocolestase de novo (AST 93 U/L, ALT 56 U/L, GGT 487 U/L, FA 570 U/L), associada a descrição de fezes negras. O estudo endoscópico evidenciou varizes gástricas exuberantes, com erosões na mucosa gástrica e restos dehematina. A AngioTC abdominal revelou um marcado espessamento parietal gástrico e edema parietal, com trombose completa da veia esplénica e circulação colateral venosa gástrica consequente. Por suspeita de hemorragia intermitente, decidiu-se em conjunto com Imunohemoterapia não introduzir hipocoagulação.

Posteriormente, iniciou quadro de hematoquézias abundantes. Realizou enteroscopia por videocápsula que revelou uma possível variz jejunal com ponto de rotura e nova AngioTC abominopélvica demonstrou potencial conteúdo hemático nas ansas jejunais. O doente evoluiu com alteração do estado de consciência, em contexto de hematemeses maciças, a condicionar queda de hemoglobina de 9.6 para 6.8 g/dL em 24h, o que motivou a transferência urgente para um centro diferenciado para orientação por Radiologia de Intervenção. Foi submetido a embolização da artéria esplénica com estabilização clínica subsequente. O doente encontra-se atualmente em acompanhamento multidisciplinar e a tolerar desmame progressivo de corticoterapia.

Discussão e conclusões: Este caso sublinha, por um lado, a importância de considerar ab initio a doença relacionada com IgG4 no diagnóstico diferencial perante a presença de uma massa pancreática e, por outro lado, a necessidade de reconhecer que nem todas as complicações observadas durante o follow-up são secundárias à doença de base. Perante inconsistência de sintomas e ausência de resposta à terapêutica instituída, impõe-se uma avaliação abrangente e multidisciplinar, por forma a evitar atrasos diagnósticos e prevenir complicações pós-cirúrgicas como as observadas neste doente.

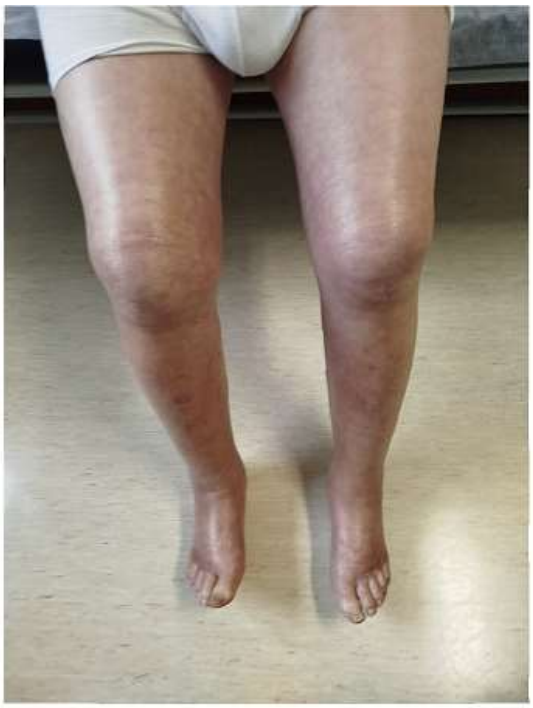

Figura 1: Doente em anasarca, aquando da primeira observação por Reumatologia.

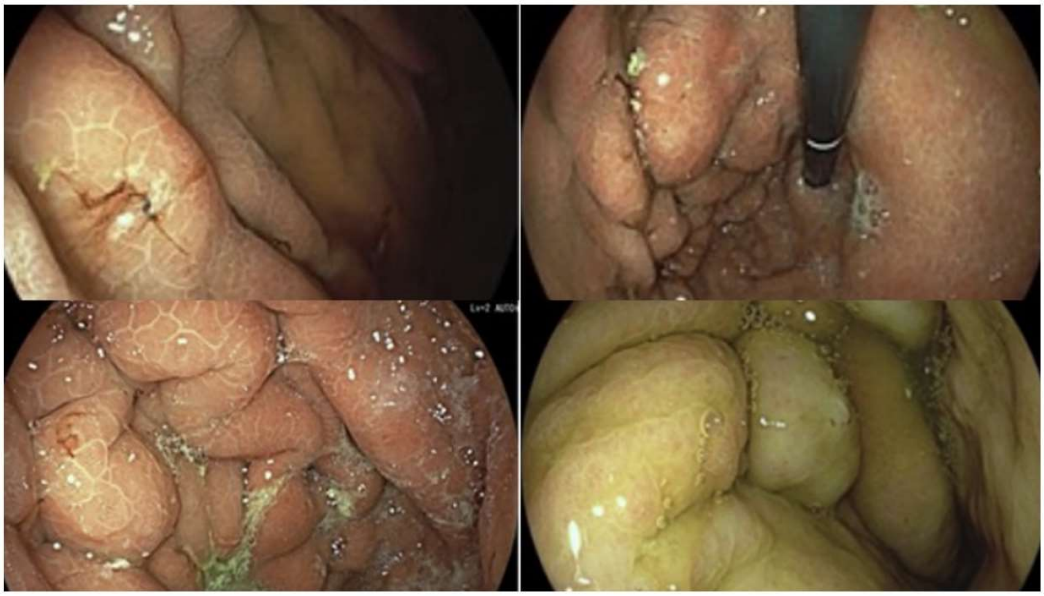

Figura 2: Imagens de Endoscopia Digestiva Alta, a revelar varizes gástricas exuberantes, com erosões na mucosa gástrica com restos de hematina.

PO CC 33 - HEMOPHAGOCYTIC SYNDROME TRIGGERED BY VIRAL INFECTION IN A PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a severe and potentially life-threatening hyperinflammatory syndrome, characterized by hypercytokinemia and excessive activation of macrophages, cytotoxic T cells and natural killer cells, leading to inflammation, organ dysfunction and, ultimately, multi-organ failure and

death (1,2). HLH may be a primary disease of genetic

underground, or secondary, triggered by infections, drugs, neoplasms or rheumatic diseases such as systemic lupus erythematosus (SLE) (2,3). Its prevalence in SLE is estimated at 0.9% and 4.6% (1,4). HLH manifestations often overlap with lupus flares, with fever, hepatosplenomegaly, and cytopenias being typical; other signs and symptoms are hypertriglyceridemia, hypofibrinogenemia, liver dysfunction, elevated ferritin levels, lymphadenopathy, skin rash and neurological symptoms (5,6). Distinguishing lupus flare from HLH is therefore particularly challenging (3). We report a case of SLE complicated by HLH, successfully treated with interleukin (IL) 1 blockade.

Case report: A 41-year-old woman with SLE since age 28, with previous cutaneous, articular e serosae involvement, on baseline therapy with hydroxychloroquine 200 mg/day and prednisolone 10 mg/day due to recent flare with pericarditis and pleural effusion, presented, in July 2025, odynophagia, severe headache, myalgia and high fever, general malaise and asthenia. She had no other accompanying symptoms, such as cutaneous lesions, cough, dyspnoea, chest pain, or urinary symptoms.

Initial laboratory findings revealed liver cytolysis and cholestasis, elevation of C-reactive protein (13.5 mg/dL), low erythrocyte sedimentation rate (17mm), and normal blood counts. Thoracic and abdominal computer tomography (CT) showed multiple adenopathies and mild hepatomegaly; cerebral CT did not show any brain lesions or thrombosis. Cerebrospinal fluid (CSF) had mild protein elevation, without pleocytosis or glucose consumption. Blood cultures and CSF viral antigen search and cultures were negative. Viral serologies were negative for hepatitis B and C virus, human immunodeficiency virus, parvovirus B19 and Epstein-barr virus and positive for cytomegalovirus (CMV) IgG/IgM, though without detectable viral load.

During hospitalization, she developed thrombocytopenia (97000 platelets/µL), progressive liver enzyme dysfunction (aspartate and alanine aminotransferase of 547 and 147UI/L, respectively, total and direct bilirubin of 2.3 and 1.79mg/dl, respectively), hyperferritinemia (>10,000 ng/mL) and hypertriglyceridemia (232 mg/dL). She also presented an acute confusional state. Brain MRI revealed nonspecific changes.

Suspicion of HLH arise and fibrinogen level was low (188 mg/dL), HScore was 194 points, supporting HLH diagnosis.

Treatment with IL-1 antagonist anakinra 8mg/kg/day for 10 consecutive days led to impressive improvement, namely rapid defervescence, improvement of adenophaties, resolution of constitutional and neurological symptoms, and progressive normalization of laboratory parameters.

Conclusions: HLH is a rare but life-threatening complication in SLE, often triggered by infections and difficult to distinguish

from lupus flare (3,5). Early recognition and prompt therapy, such as IL-1 blockade, can result in rapid recovery. This case underscores the importance of systematic evaluation of persistent fever, cytopenias, hyperferritinemia, and organ dysfunction in SLE patients. In our patient, although no CMV viral load was detected, positive IgM/IgG suggested recent CMV infection and was considered a most likely trigger of HLH, highlighting the need to actively investigate viral infections in this context.

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PO 34 - ROMOSOZUMAB DOES NOT IMPROVE VOLUMETRIC BONE MINERAL DENSITY AT DISTAL RADIUS BUT INCREASES BONE STRENGTH: A PROSPECTIVE STUDY

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Introduction: Romosozumab (ROMO) is a monoclonal antibody that inhibits sclerostin, promoting bone formation and suppressing resorption. While ROMO leads to substantial BMD gains at the lumbar spine and the hip, its effects on distal radius volumetric BMD (vBMD) remain unclear. High-resolution peripheral Quantitative Computed Tomography (HR-pQCT) allows for detailed assessment of bone microarchitecture and strength at peripheral regions. To our knowledge, no published clinical studies evaluated the response to ROMO with HR-pQCT at the radius.

Objectives: The aim of this study was to evaluate the impact of 12 months of ROMO treatment on volumetric BMD, bone microarchitecture, and biomechanical ability at the distal radius using HR-pQCT.

Materials and Methods: We did a prospective study on 49 postmenopausal women with osteoporosis treated with ROMO for 12 months who underwent HR-pQCT with microfinite element analysis (uFEA) assessments at baseline, 3, 6 and 12 months. We also performed dual-energy X-ray absorptiometry (DXA) at lumbar spine, femoral neck and total hip. HR-pQCT parameters trajectories were analyzed using mixed-effects modeling.

Results: A total of 49 postmenopausal women were included, with a mean age of 73.4±10.1 years old. Mean BMI was 23.1±3.4 kg/m2 and most patients were no smokers (n=45, 91.8%). The majority of the patients had a previous vertebral fracture (n=41, 83.7%) and a smaller percentage had sustained previous hip (n= 9, 18.4%) and wrist fractures (n= 16, 32.7%).

Bone microstructure parameters measured with HR-pQCT did not change over the 12-month treatment period (Table 1, Figure 1). Trabecular vBMD remained stable throughout follow-up (p=0.383), as did trabecular thickness (p=0.868) and spacing (p=0.234). Similarly, BV/TV showed a mild numerical increase at month 3 and month 12, but this was not significant (p=0.614 and p=0.058, respectively). Cortical parameters, including cortical vBMD and cortical thickness, did not exhibit significant changes over time (p=0.905 and p=0.463, respectively). Parameters of uFEA demonstrated early and significant changes (Table 1, Figure 2). Cortical failure load increased at both 3 and 6 months (p=0.018 and p=0.049, respectively) but was stable when considering the overall period of treatment (p=0.102). Consistent with these findings, shear strength increased at 3 and 6 months, with significant changes at month 6 (p=0.008). Stiffness remained relatively stable until 12 months (p=0.288). As expected, lumbar spine, femoral neck, and total hip areal BMD evaluated with DXA significantly increased by 11.6%, 8.3%, and 3.1%, respectively (all p<0.001).

Discussion: In conclusion, ROMO may enhance bone mechanical properties, particularly within the cortical compartment in non-load-bearing sites, such as distal radius. The preserved mechanical integrity despite modest or absent vBMD gains underscores the value of incorporating HR-pQCT and uFEA in evaluating therapeutic response in patients treated with ROMO.

	M0	M3	M6	M12	p-value
Tot.vBMD (mgHA/cm^3)	263.245 (222.426-303.816)	256.903 (280.720-)	275.159 (225.079-310.731)	271.625 (227.468-313.298)	0.960
Ct.vBMD (mgHA/cm^3)	778.401 (718.450-844.725)	755.957 (691.460-805.905)	767.680 (719.022-836.766)	763.181 (711.32-801.153)	0.905
Tb.vBMD (mgHA/cm^3)	120.161 (82.706-149.975)	121.449 (90.851-143.075)	122.648 (86.439-163.279)	129.967 (103.441-148.317)	0.383
Ct.Th (mm)	0.716 (0.668-0.781)	0.697 (0.653-0.770)	0.733 (0.665-0.784)	0.721 (0.662-0.764)	0.463
Tb.BV/TV (%)	10.284 (8.642-12.015)	11.241 (9.202-12.204)	10.986 (8.943-12.226)	10.842 (9.319-12.136)	0.137
Tb.Th (mm)	0.269 (0.256-0.278)	0.271 (0.263-0.273)	0.265 (0.257-0.273)	0.268 (0.253-0.273)	0.868
Tb.Sp (mm)	0.910 (0.860-0.971)	0.878 (0.846-0.988)	0.894 (0.841-0.960)	0.905 (0.851-0.959)	0.617
Stiffness (kN/mm)	37.542 (36.909-38.087)	37.408 (39.969-37.746)	37.508 (37.222-37.677)	37.198 (36.771-38.022)	0.288
Shear (KN/m^2)	118639.3 (107618.6-137550.1)	151812.7 (110633.125-180862.150)	143614.3 (11.4588.4-170783.8)	137988.2 (119754.5-151843-3)	**0.019**
% Cortical Break Bricks	8.108 (6.283-10.351)	6.299 (4.777-9.100)	7.303 (4.646-9.423)	7.336 (5.749-9.185)	0.102
% Trabecular Break Bricks	6.510 (3.054-10.502)	4.229 (2.496-10.007)	5.734 (3.403-11.429)	5.276 (2.237-8.549)	0.052

Figure 1. High-resolution peripheral quantitative computed tomography (HR-pQCT) microarchitecture parameters changes at distal radius in patients treated with romosozumab

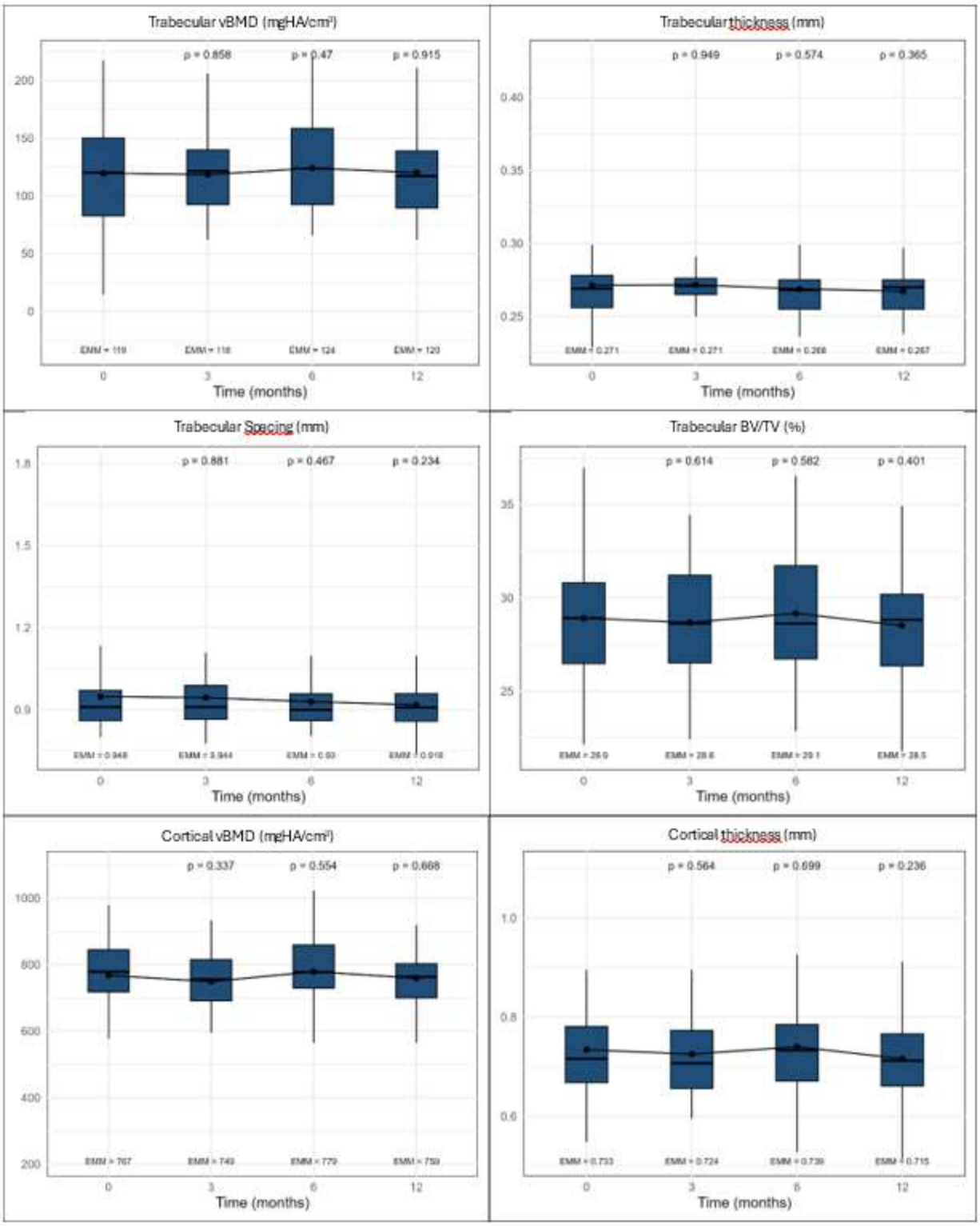
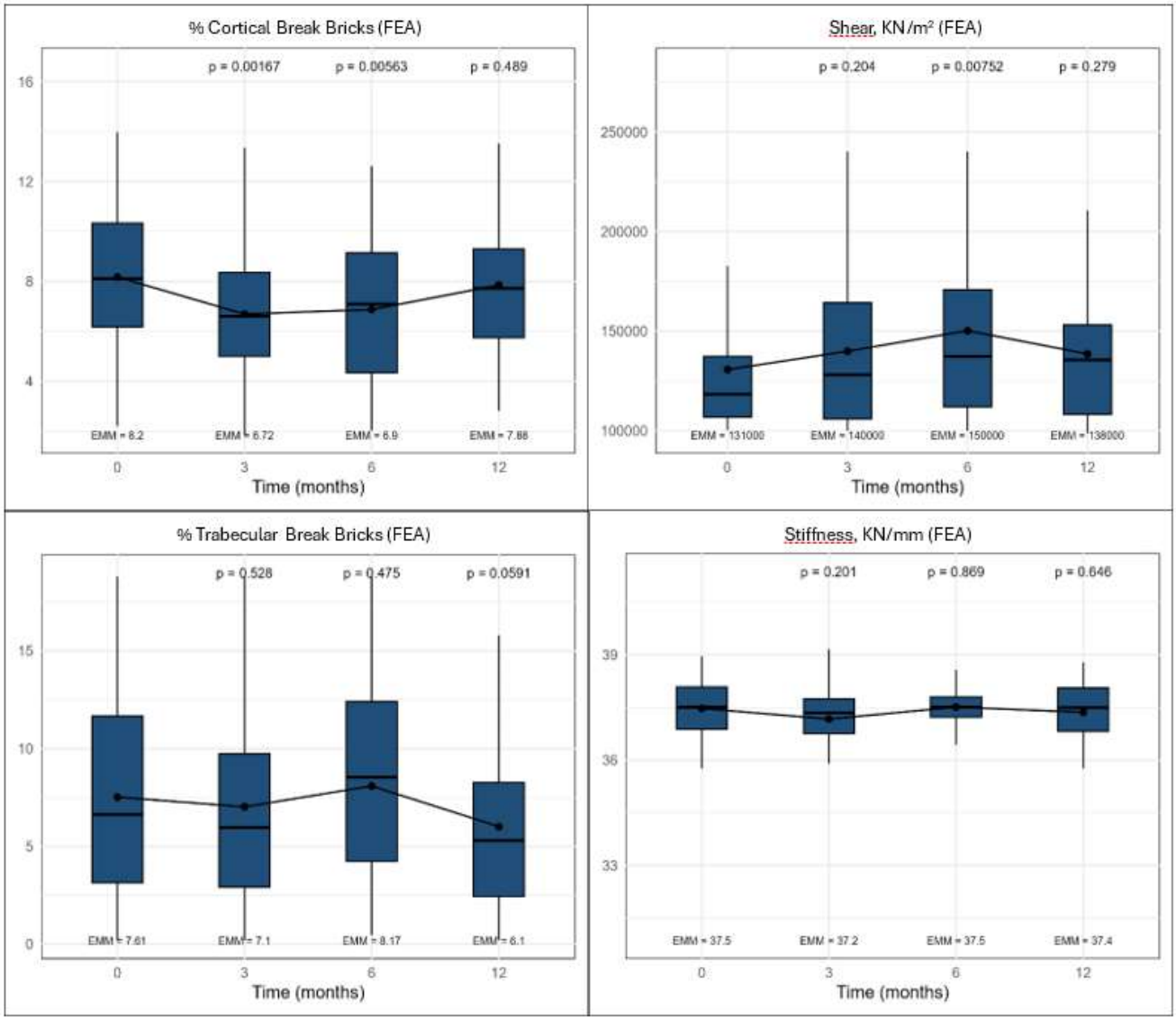

Trabecular vBMD (mgHA/cm³)
p = 0.858
p = 0.47
p = 0.915
Time (months)
Trabecular thickness (mm)
p = 0.949
p = 0.574
p = 0.365
Time (months)
Trabecular Spacing (mm)
p = 0.881
p = 0.467
p = 0.234
Time (months)
Trabecular BV/TV (%)
p = 0.614
p = 0.582
p = 0.401
Time (months)
Cortical vBMD (mgHA/cm³)
p = 0.337
p = 0.554
p = 0.668
Time (months)
Cortical thickness (mm)
p = 0.564
p = 0.699
p = 0.236
Time (months)

Figure 2. Parameters of microfinite element analysis (uFEA) from high-resolution peripheral quantitative computed tomography (HR-pQCT) evolution over 12 months of treatment with romosozumab



PO CC 35 - DERMATOMYOSITIS IN THE ELDERLY: KEEPING ALL EYES ON CANCER

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**Contribuíram igualmente para o desenvolvimento do trabalho*

Introdução: A dermatomiosite é uma miopatia inflamatória sistémica que pode representar uma manifestação paraneoplásica em até 40% dos casos, sobretudo em idosos. A presença de anticorpos anti-TIF1-gama associa-se a maior risco de neoplasia, exigindo vigilância clínica apertada.

Caso clínico: Mulher de 86 anos, previamente autónoma, diagnosticada em 2024 com dermatomiosite, caracterizada por fraqueza proximal, mialgias e exantema cutâneo típico. Apresentava ANA positivos (padrão AC-4, 1/1280) e anticorpos anti-TIF1-gama; a biópsia cutânea e a eletromiografia confirmaram o diagnóstico. O estudo inicial excluiu malignidade, revelando apenas envolvimento pulmonar intersticial. Após resposta parcial à terapêutica imunossupressora, verificou-se agravamento clínico com declínio funcional (MMT8 68/150), disfagia, rouquidão e toracalgia. Analiticamente: PCR 9,76 mg/dL, aldolase 12,1 U/L, CK 109 U/L e troponina 490 ng/L, sugerindo envolvimento digestivo, cardíaco e respiratório. Após confirmação de miocardite, foram iniciados pulsos de metilprednisolona. A PET-CT revelou lesões hepáticas, ganglionares e ósseas hipermetabólicas compatíveis com doença linfoproliferativa metastizada. Apesar da terapêutica instituída, houve deterioração progressiva, tendo sido instituída abordagem paliativa, com falecimento poucos dias depois.

Discussão: Este caso ilustra a forte associação entre dermatomiosite anti-TIF1-gama e neoplasias ocultas em doentes idosos, reforçando a necessidade de reavaliação periódica mesmo após rastreio inicial negativo. A rápida evolução clínica e o envolvimento multissistémico sublinham a importância de uma abordagem precoce e multidisciplinar.

Conclusão: A dermatomiosite com positividade para anti-TIF1-gama exige elevado índice de suspeição para neoplasia subjacente. Pode apresentar um curso agressivo e rapidamente progressivo, com prognóstico reservado. A integração precoce dos Cuidados Paliativos promove uma abordagem centrada no doente e humanizada em fases avançadas da doença, quando o tratamento curativo já não é viável.

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PO CC 36 - SACROILEÍTE INFECIOSA - UMA CAUSA RARA DE LOMBALGIA INFLAMATÓRIA

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Introdução: A sacroileíte infecciosa é uma causa rara de artrite séptica, geralmente subdiagnosticada devido à inespecificidade das suas manifestações. Tipicamente apresenta-se com lombalgia aguda, com irradiação para o abdómen ou coxa, o que pode simular outras patologias. Embora mais frequente em doentes com fatores predisponentes, também pode surgir em indivíduos previamente saudáveis. O diagnóstico precoce depende de um elevado grau de suspeição clínica.

Descrição do caso: Mulher, 26 anos, autónoma, com antecedentes de síndrome do ovário poliquístico, G0P0. Negava hábitos tabágicos, alcoólicos, uso de drogas endovenosas, viagens recentes ou contactos de risco epidemiológico. Desde há 3 dias com lombalgia intensa, rigidez matinal > 1 hora, com irradiação para face anterior da coxa direita, sem fator desencadeante, associada a incapacidade de suportar a carga corporal no membro inferior direito. Já teria recorrido ao serviço de urgência por múltiplas vezes, sem melhoria com as medidas instituídas, o que a levou a recorrer à consulta de Medicina Física e de Reabilitação. Ao exame objetivo, destacava-se o fácies de dor, febre (38 ºC), marcha claudicante por incapacidade em fletir a anca direita por inibição álgica. Foi medicada com analgesia, colheu estudo analítico (leucocitose (12.4x109/L) com neutrofilia (90,5%) e PCR 21 mg/dL) e fez tomografia computorizada (TC) da coluna lombar que não demonstrou alterações relevantes. Foi novamente referenciada ao serviço de urgência, tendo sido internada para esclarecimento do quadro clínico. As serologias para VIH, hepatites, *Brucella* e tuberculose foram negativas. As hemoculturas foram positivas para *Staphylococcus aureus* sensível à meticilina (MSSA), tendo sido excluída endocardite por ecocardiograma transtorácico. A ressonância magnética (RMN) mostrou alterações compatíveis com artrite séptica da sacroilíaca direita, evidenciando derrame articular, erosão da superfície óssea adjacente, edema medular local, e a presença de duas colecções abcedadas em continuidade com a articulação (uma no músculo psoas-ilíaco com 49 x 23 x 31 mm e outra no piriforme direito com 15 x 11 x 22mm). Perante os achados clínicos, analíticos e imagiológicos, estabeleceu-se o diagnóstico de artrite séptica da articulação sacro-ilíaca direita com extensão óssea e formação de abcessos periarticulares. Foi instituída antibioterapia intravenosa com doxiciclina (6 dias) e ceftriaxone (38 dias), e, posteriormente, oral com trimetropim-sulfametoxazol (30 dias), com boa resposta clínica, sem necessidade de drenagem.

Discussão: A artrite séptica da sacroilíaca representa menos de 2 % das artrites sépticas periféricas, sendo o *S. aureus* o agente mais comum[1,3,4]. A apresentação clínica com lombalgia inflamatória e limitação funcional pode ser confundida com patologia músculo-esquelética inflamatória, como espondilartrite axial, ou patologias abdominais ou ginecológicas, o que pode atrasar o diagnóstico. Os exames de imagem como a TC podem não detectar alterações em estádios iniciais da doença, enquanto a RMN permite um diagnóstico precoce e avaliação da extensão da infecção (edema medular, erosões e colecções abcedadas)[1,2,3]. O tratamento baseia-se em antibioterapia durante 4 a 6 semanas, com drenagem percutânea ou cirúrgica em caso de falência ou coleções acessíveis[3]. No presente caso, a boa resposta clínica permitiu evitar procedimentos invasivos.

Conclusão: Este caso mostra como a lombalgia inflamatória refratária a anti-inflamatórios, num adulto jovem, pode mimetizar uma espondiloartrite. O aparecimento de febre e inflamação sistémica deve levantar a suspeita de etiologia infecciosa, tornando essencial a realização precoce de RMN e hemoculturas, neste contexto. O diagnóstico precoce e a introdução de antibioterapia dirigida são fundamentais para reduzir a morbilidade e prevenir sequelas estruturais.

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PO CC 37 - LATE DIAGNOSIS OF AXIAL SPONDYLOARTHRITIS IN PATIENTS WITH GIANT CELL ARTERITIS

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Introduction: Giant cell arteritis (GCA) is a large vessel vasculitis (LVV) that affects individuals over 50 years old. Coexisting LVV has previously been seldom reported in cases of established spondyloarthritis (SpA). Overlapping immunopathogenic pathways may allow for this coexistence. Herein we describe two patients with GCA and late diagnosis of axial SpA.

Case 1: A 75-year-old man presented with shoulder and pelvic girdle pain, prolonged morning stiffness and elevated ESR (81 mm/h) and CRP (15.5 mg/dL). A diagnosis of polymyalgia rheumatica (PMR) was established and treatment with prednisolone (15 mg qd) ensured clinical remission, without recurrence after glucocorticoid (GC) withdrawal.

Five years later, he presented with sudden unilateral vision loss, frontal headache and elevated ESR/CRP. Ophthalmologic evaluation suggested arteritic anterior ischemic optic neuropathy and, given the high index suspicion for GCA, pulse treatment with methylprednisolone (1g qd for 3 days) followed by prednisolone (60 mg qd) was started. Temporal artery ultrasound revealed nonspecific intima-media thickening that normalized on follow-up.

During GC tapering, despite methotrexate therapy, the patient experienced PMR-like recurrences with predominantly pelvic girdle pain and ankle arthritis. PET-CT excluded active LVV. Unexpectedly, pelvic radiography revealed partial sacroiliac joint ankylosis and chronic active sacroiliitis was confirmed by MRI.

After the diagnosis of axial SpA (HLA-B27 positive) with high-disease activity (ASDAS-CRP 4,6), treatment with adalimumab combined with methotrexate ensured sustained GC-free clinical remission.

Case 2: A 78-year-old woman presented with a 2-year diagnosis of relapsing LV-GCA. She first presented with a history of pelvic girdle and low back pain, morning stiffness and elevated ESR (81 mm/h) and CRP (9,2 mg/dL). Presence of the ultrasonographic halo sign of the axillary and subclavian by US confirmed

the diagnosis and treatment with prednisolone (60mg qd) was started. After failure to taper GC below 20 mg qd, methotrexate was initiated with partial benefit.

On clinical review, the patient reported long standing low back pain with mixed rhythm. Follow-up vascular US revealed chronic halo of the subclavian, axillary and left temporal arteries. PET-CT excluded active LVV but exposed chronic sacroiliitis, confirmed by MRI. After the diagnosis of axial SpA (HLA-B27 negative) treatment with adalimumab was started with clinical improvement and GC tapering (5 mg qd). ESR and CRP

remained mildly elevated and PET-CT reassessment showed mild-to-moderate FDG uptake in the thoracic aorta, suggestive of subclinical aortitis.

Conclusion: Evidence suggesting an association between LVV and SpA is emerging, although GCA-SpA coexistence remains rarely reported. We describe two additional cases, adding to the few published to date.

Both conditions may share genetic and immunological pathways. While HLA-B27 is commonly associated with SpA, possible links with HLA-DRB104 and HLA-DRB101—both strongly related to GCA—have been reported. Th1/Th17-mediated inflammation with elevated TNF-α, IL-6, IL-17, and IL-23 also plays a key role in both diseases.

These cases highlight the need to consider SpA in GCA patients with persistent inflammation or atypical musculoskeletal symptoms, particularly in elderly patients with incomplete treatment response.

PO CC 38 - REATIVAÇÃO DA GRANULOMATOSE COM POLIANGEÍTE: DO ENVOLVIMENTO CUTÂNEO À HEMORRAGIA ALVEOLAR DIFUSA — CASO CLÍNICO

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Introdução: A granulomatose com poliangeíte (GPA) é uma vasculite sistémica necrotizante, associada a anticorpos anti citoplasma de neutrófilos (ANCA), que afeta vasos de pequeno a médio calibre (1, 2). Apresenta-se, habitualmente, na sexta ou sétima décadas de vida (2, 3), com predileção pelo pulmão e rim (1, 4). Uma das complicações mais graves da GPA é a hemorragia alveolar difusa (HAD), associada a uma mortalidade que pode atingir os 60% (3).

Caso clínico: Relata-se o caso de um homem de 63 anos com vasculite associada aos ANCA sob forma de GPA. A doença iniciou-se com polipose nasal e surdez neurossensorial. Três anos antes da apresentação atual, o doente desenvolveu hemoptises e anemia, associados a elevados títulos de ANCA-PR3, tendo o lavado broncoalveolar confirmado o diagnóstico de hemorragia alveolar. Foi submetido a terapêutica de indução com corticoterapia (em pulsos e oral) e ciclofosfamida, com boa resposta clínica e normalização dos títulos de ANCA-PR3. Iniciou, depois, terapêutica de manutenção com azatioprina, que manteve 18 meses, suspensa por mielotoxicidade com pancitopenia. Permaneceu estável, sem imunossupressão, até janeiro de 2025, quando desenvolveu púrpura palpável nos membros superiores e inferiores, com evolução de algumas lesões para formas bolhosas e ulceradas. Pela suspeita de vasculite cutânea, foi internado, realizou biópsia cutânea e iniciou corticoterapia em baixa dose, com melhoria cutânea progressiva. Durante o internamento, desenvolveu dispneia súbita e

insuficiência respiratória aguda, com necessidade de oxigenoterapia em alto débito, associada a queda acentuada de hemoglobina de 3g/dL. Neste contexto, realizou tomografia computorizada (TC) torácica que revelou consolidações bilaterais, compatíveis com HAD. A posteriori, o aparecimento de hemoptises e elevação dos ANCA-PR3 (>200 U) reforçaram o diagnóstico de HAD no contexto da GPA. Foi iniciada terapêutica de indução com pulsos de metilprednisolona, mas o quadro clínico evoluiu de forma desfavorável, com o falecimento do doente. A biópsia cutânea confirmou vasculite leucocitoclástica com necrose e extravasamento eritrocitário.

Discussão: A HAD é uma complicação grave da GPA, com uma incidência reportada entre 5% e 45% (1), caracterizando-se, tipicamente, pela tríade de hemoptise, infiltrados pulmonares e anemia (2). O diagnóstico baseia-se na integração dos dados clínicos, analíticos, imagiológicos, histopatológicos e do lavado broncoalveolar (2, 3). Importa salientar que, em cerca de 30% dos casos, a hemoptise pode estar ausente na apresentação inicial, uma vez que o sangue pode permanecer contido nos alvéolos (1). Nestes casos, a

associação de anemia aguda e insuficiência respiratória hipoxémica deve levantar a suspeita clínica de HAD. A

TC torácica foi essencial na confirmação do diagnóstico (1, 3).

Conclusão: Deve-se suspeitar de HAD em doentes com GPA que apresentem anemia aguda e insuficiência respiratória hipoxémica, mesmo na ausência inicial de hemoptise, uma vez que o diagnóstico e início precoce de imunossupressão são fundamentais para induzir a remissão e melhorar o prognóstico.

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PO CC 39 - BETWEEN OSTEOPOROSIS AND OSTEONECROSIS: A CASE OF CHALLENGING THERAPEUTIC BALANCE

Miguel Correia Natal[1], Bárbara Fernandes Esteves[1], Teresa Burnay[1], Lúcia Dias Costa[1], Georgina Terroso[1]

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Introduction: Medication-related osteonecrosis of the jaw (MRONJ) is a rare but potentially serious

complication described in patients receiving bisphosphonates or denosumab. Denosumab, a monoclonal antibody that inhibits RANKL, is currently a first-line therapeutic option for the treatment of severe osteoporosis (OP) in patients with contraindications to bisphosphonates, particularly in the presence of advanced chronic kidney disease (CKD). The risk of osteonecrosis is generally low at doses used for OP, but increases in the presence of local risk factors (dental extractions, infection, or poor oral hygiene) and systemic risk factors (immunosuppression, corticosteroid therapy, advanced CKD). Management of these cases is particularly challenging due to the scarcity of safe and effective therapeutic alternatives in this population.

Case Description: A 73-year-old woman with a history of pulmonary lymphangiomatosis, who underwent left lung transplantation in 2013 and has stage 2b chronic kidney disease (CKD) of unknown etiology, was referred to the Rheumatology clinic in 2015 for evaluation of severe osteoporosis (bone densitometry with T-score of -4.2 at the lumbar spine and -3.6 at the femoral neck), without known history of fracture. Laboratory tests showed parathyroid hormone, 25(OH)vitamin D, phosphorus, and calcium within reference values; thoracolumbar radiography showed D7 flattening suggestive of vertebral fracture. At that time, she had been on alendronate therapy for approximately two years; given worsening renal dysfunction, densitometry compatible with severe osteoporosis, and detection of vertebral fracture, transition to denosumab was chosen.

She maintained regular treatment until 2023, with stable densitometry (T-score of -3.5 at the lumbar spine and -3.6 at the femoral neck). In December 2023, she reported gingival pain, with examination revealing gingival recession but no other alterations. Following dental extraction in May 2024, she developed a painful lesion with progressive swelling of the right hemimandible, associated with purulent exudate. She was urgently evaluated at the Maxillofacial Surgery clinic, where bone exposure was observed, and was

admitted in December 2024 for probable MRONJ.

Imaging studies (CT and MRI) showed extensive mandibular and maxillary alterations compatible with medication-related osteonecrosis, with possible osteomyelitis. Microbiological analysis revealed mandibular actinomycosis with superinfection by Staphylococcus warneri. She completed 14 days of intravenous amoxicillin/clavulanic acid, followed by prolonged oral therapy, with favorable clinical evolution.

Denosumab was definitively discontinued, and she currently remains without pharmacological anti-osteoporotic therapy, given the limitation imposed by CKD, currently at stage 4 (transitioning to stage 5), and the absence of currently available pharmacological alternatives.

Discussion/Conclusions: This case illustrates a rare but clinically relevant complication of denosumab treatment in a patient with advanced CKD and a history of lung transplantation. Despite the drug's proven benefit in fracture prevention, the presence of multiple predisposing factors – prolonged treatment, renal insufficiency, chronic immunosuppression, and recent dental procedure – likely contributed to the development of mandibular osteonecrosis. Definitive discontinuation of denosumab was necessary, leaving the patient without active anti-osteoporotic therapy, which highlights the therapeutic gap existing in this clinical context.

This case reinforces the importance of oral surveillance before and during treatment with anti-resorptive agents, multidisciplinary coordination, and the urgent need for safe and effective therapeutic options for the treatment of osteoporosis in patients with advanced CKD.

PO CC 40 - FROM NORMAL TO ACTIVE SCLERODERMA PATTERN: NAILFOLD VIDEOCAPILLAROSCOPY AS A MIRROR OF DISEASE PROGRESSION IN SYSTEMIC SCLEROSIS

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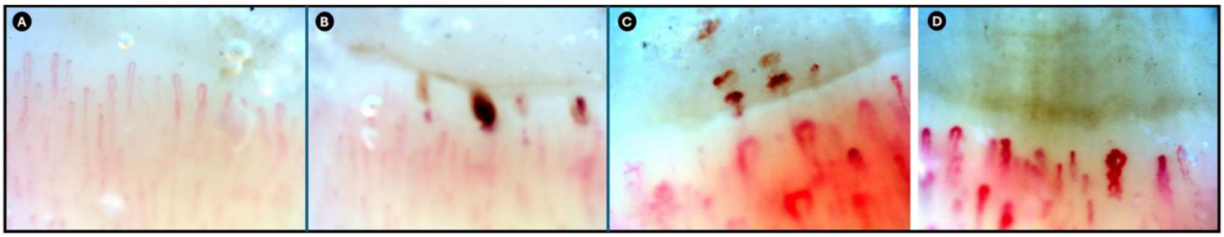
[1]ULS S. João

Introduction: Systemic sclerosis (SSc) is a complex autoimmune connective tissue disorder characterized by vascular dysfunction, immune activation, and progressive fibrosis. of the skin and internal organs. Despite advances in pathophysiological understanding, predicting disease course and organ involvement remains a major challenge in clinical practice. Nailfold videocapillaroscopy (NVC) has emerged as a valuable diagnostic and monitoring tool, providing insights into microvascular changes that often precede clinical manifestations. The evolution of capillaroscopic patterns may reflect disease activity and predict organ involvement. We present a case of rapidly progressive SSc where serial NVC examinations closely paralleled clinical deterioration and immunological changes.

Case description: A 62-year-old man was referred to Rheumatology for a 2-month history of symmetric polyarthritis affecting wrists and small hand joints, partially responsive to high-dose corticosteroids, generalized hand swelling, and Raynaud's phenomenon. Physical examination revealed facial telangiectasias and minimal skin thickening distal to proximal interphalangeal joints. Initial investigations demonstrated mildly elevated erythrocyte sedimentation rate (41 mm/h) and ANA 1/640 speckled pattern, while extended autoimmune panel, including SSc-specific antibodies, was negative. Baseline NVC was normal. Treatment with oral methotrexate 10mg weekly and vasodilator therapy resulted in mild clinical improvement despite poor therapeutic compliance.

Approximately eighteen months later, the patient developed worsening hand swelling with more pronounced sclerodactyly, facial skin thickening, and microstomia. Repeat laboratory studies revealed strong anti-Scl70 antibody positivity, and concurrent NVC showed isolated haemorrhages in the right 4th finger.

Six months subsequently, skin thickening progressed to involve the forearms, accompanied by increased telangiectasias. The patient complained of fatigue, weight loss and worsening inflammatory arthralgias



Photographs from Inspectis© Nailfold Digital Capillaroscopy illustrating the evolution of videocapillaroscopic changes in the patient. From left to right: A – right 4th finger, without relevant abnormalities (May/2023); B – right 4th finger, with microhemorrhages (Dec/2024); C and D – right 4th finger and left 5th finger, with microhemorrhages, dilated capillaries, and megacapillaries (May/2025).

despite successive methotrexate dose escalations. Cardiopulmonary assessment revealed nonspecific interstitial pneumonia-pattern interstitial lung disease on high-resolution chest CT. Repeat NVC demonstrated megacapillaries in 4th and 5th fingers bilaterally and hemorrhages involving left 3rd to 5th fingers and right 4th and 5th fingers, consistent with active scleroderma pattern. Following multidisciplinary discussion, rituximab therapy was initiated.

Discussion/Conclusions: This case illustrates the dynamic interplay between immune activation, microvascular injury, and fibrotic progression in SSc. The rapid clinical and serological evolution from an initially seronegative presentation to strong anti-Scl70 positivity exemplifies the heterogeneous and unpredictable disease trajectory. Serial NVC examinations provided non-invasive and reproducible means of detecting early microvascular deterioration, transitioning from normal to active scleroderma pattern in parallel with clinical worsening and organ involvement. The close temporal relationship between capillaroscopic changes, antibody seroconversion, and clinical progression reinforces NVC's value as a non-invasive monitoring tool in SSc management, potentially guiding therapeutic decisions and predicting organ complications in rapidly progressive disease.

PO CC 41 - DOENTE QUE NÃO SE QUEIXA, MAS TEM UMA DOENÇA RARA – SÍNDROME DE PARSONAGE-TURNER

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[1]ULS Vale do Sorraia

Enquadramento: O Síndrome de Parsonage-Turner (SPT), também denominado como nevrite braquial ou amiotrofia nevrálgica, é uma doença rara do sistema nervoso periférico e frequentemente subdiagnosticada, caracterizada por dor súbita no ombro, seguida de défices motores e eventualmente sensitivos, podendo ser de comprometimento unilateral ou bilateral.

Objetivos: Apresentar um caso clínico de SPT em um idoso, destacando a importância do diagnóstico precoce sem queixas específicas e da intervenção adequada.

Material e Métodos: Homem de 73 anos, em consulta de vigilância de hipertensão, onde foi identificada uma parésia com 2 meses de evolução no ombro direito, comprometendo as atividades diárias, inicialmente com desvalorização das queixas por parte do doente. O exame neurológico e os estudos complementares foram realizados para confirmar o diagnóstico.

Resultados: O doente apresentava fraqueza na abdução do ombro, flexão e pronação do cotovelo, com omoplata alada e atrofia do deltóide. A sensibilidade e a pinça polegar-indicador estavam preservadas. A ecografia não revelou alterações estruturais, e as análises laboratoriais foram normais, exceto pela positividade para CMV-IgG. O estudo eletromiográfico evidenciou sinais de desnervação compatíveis com SPT, afetando o tronco superior do plexo braquial.

Discussão/Conclusões: O paciente iniciou um curto curso de corticoterapia e foi encaminhado para fisioterapia para apoio à recuperação motora e realização de ressonância magnética. Este caso destaca a importância da relação próxima e contínua entre os médicos de família e os seus doentes, permitindo a deteção de doenças raras, especialmente em idosos,

onde as apresentações podem ser atípicas (neste caso: dor ligeira, omoplata alada). Nos Cuidados de Saúde Primários, o conhecimento da função e comportamento do doente é fundamental para reconhecer alterações subtis, possibilitando um diagnóstico atempado e referenciação adequada, resultando em melhores resultados através de intervenção e reabilitação precoces.

PO CC 42 - O DESAFIO – A MELHORIA DA QUALIDADE DE VIDA DO DOENTE COM GOTA TOFÁCEA CRÓNICA

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Introdução: A gota é uma artrite inflamatória comum causada por hiperuricemia e episódios recorrentes de artrite aguda monoarticular. A gota tofácea crónica pode apresentar envolvimento poliarticular, representando desafios significativos nos cuidados de saúde primários (CSP), especialmente em pacientes com comorbidades e medicamentos que elevam o ácido úrico.

Apresentação do Caso: Apresentamos o caso de um homem de 62 anos com gota de longa data, afetando as mãos e os pés, com dor debilitante e tofos exuberantes afetando cerca de 30 pequenas articulações. Apesar de estar em tratamento com febuxostat 80 mg nos últimos 4 anos, relatou crises frequentes e demonstrou conhecimento limitado sobre a sua própria doença. Os seus problemas de saúde incluem hipertensão, doença renal (estádio 3b) e síndrome metabólica. Na sua medicação crónica antihipertensiva incluía azilsartan/clorotalidona, que poderia estar a contribuir para a hiperuricemia persistente.

Resultados: O doente integrou uma específica de Consulta de Gota no CSP articulando com a reumatologia: abordagem multidisciplinar, incluindo aconselhamento dietético e modificações no estilo de vida, aumentado a literacia do doente. A otimização terapêutica incluiu a troca dos antihipertensivos para losartan/amlodipina e a titulação do febuxostat para 120mg/dia, com melhora nos níveis de ácido úrico (de 9.8mg/dl para 6.4mg/dl em apenas 3 meses). No entanto, o paciente ainda relatou dor articular debilitante e infecções recorrentes de tofos, apresentando deformidade articular, além de "erosões em mordida de rato"/esclerose na radiografia simples com demostração de fracturas a vários níveis. O doente foi encaminhado para consultas de Nefrologia, Reumatologia e Cirurgia Plástica para remoção cirúrgica dos tofos e eventual otimização terapêutica adicional.

Discussão/Conclusão: Este caso ilustra a importância da educação centrada no paciente e destaca estratégias de gestão da doença no doente com gota: otimização de medicamentos hipourecimiantes e modificação de medicamentos que hiperurecimiantes (por exemplo, escolha de antihipertensivos e hipolipídicos apropriados). Abordagens multidisciplinares melhoram os resultados clínicos com impacto crucial na qualidade de vida do doente.

PO CC 43 - DOS CRISTAIS À INFEÇÃO – UM DIAGNÓSTICO INESPERADO

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Introdução: A gota e a osteomielite são 2 entidades nosológicas que, pela sua diferente etiologia, fisiopatologia, apresentação clínica e abordagem terapêutica, raramente são consideradas, em simultâneo, como diagnósticos diferenciais plausíveis. Apesar disso, e mesmo considerando que a artropatia gotosa pode cursar com destruição/deformação articular significativas, sobretudo nas fases mais tardias da doença, convém realçar que a osteomielite pode, nas circunstâncias “adequadas”, ser fatal. Por esse

motivo, a sua distinção é essencial, mas nem sempre óbvia, como descrito no caso adiante.

Caso Clínico: Doente do sexo masculino, de 28 anos, referenciado à consulta de Reumatologia por episódios que descrevia como tumefação do hálux direito, de início súbito, com pelo menos 6 meses de evolução, à data da primeira avaliação. Apesar da descrição sugestiva de podagra e, por consequência, artropatia gotosa, não apresentava história pessoal prévia de hiperuricemia nem biótipo sugestivo de consumo excessivo de purinas. Apresentava, em 2º grau, história familiar de Artrite Reumatoide (AR) e Espondilartropatia Indiferenciada. Descrevia vários episódios semelhantes no período decorrido, com melhoria significativa verificada apenas após ciclo de Corticoterapia (CCT) sistémica e Antibioterapia (ATB). Teria já tentado múltiplos AINE e até terapêutica antifúngica, dadas alterações do leito ungueal (figura 1), contudo sem sucesso.

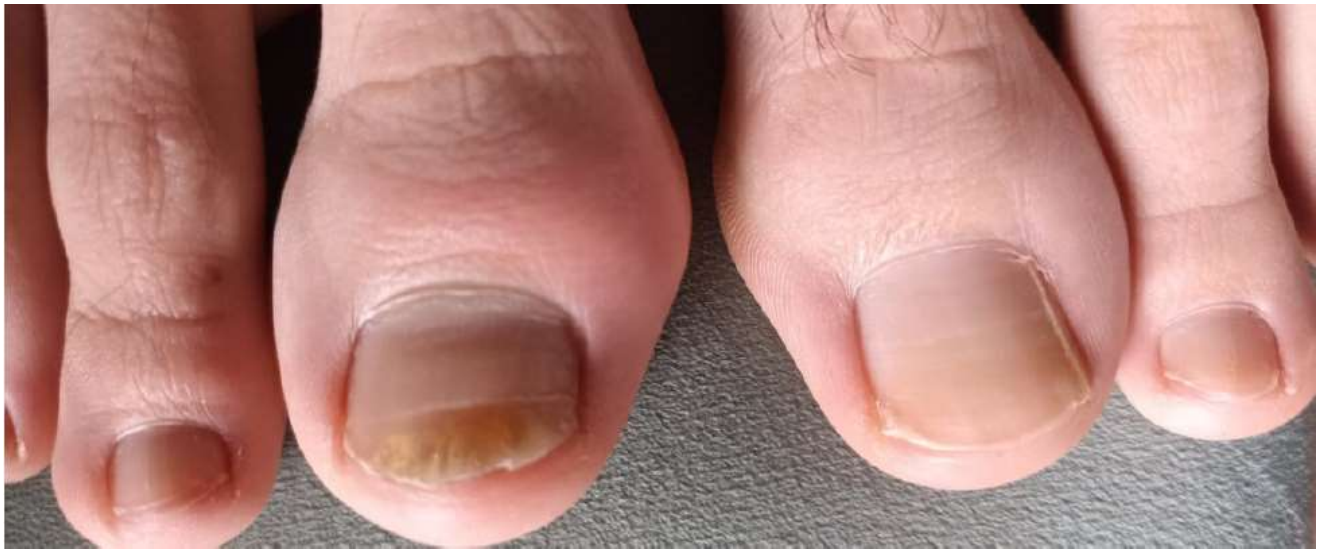

Figura 1 - achados à inspeção, de onicodistrofia do leito ungueal do hálux direito, bem como proeminência óssea adjacente

Objetivamente, foi identificada proeminência óssea (figura 1) no território identificado, caracterizada radiograficamente (figura 2) como calo ósseo na vertente medial da falange distal do primeiro dedo dos pés.

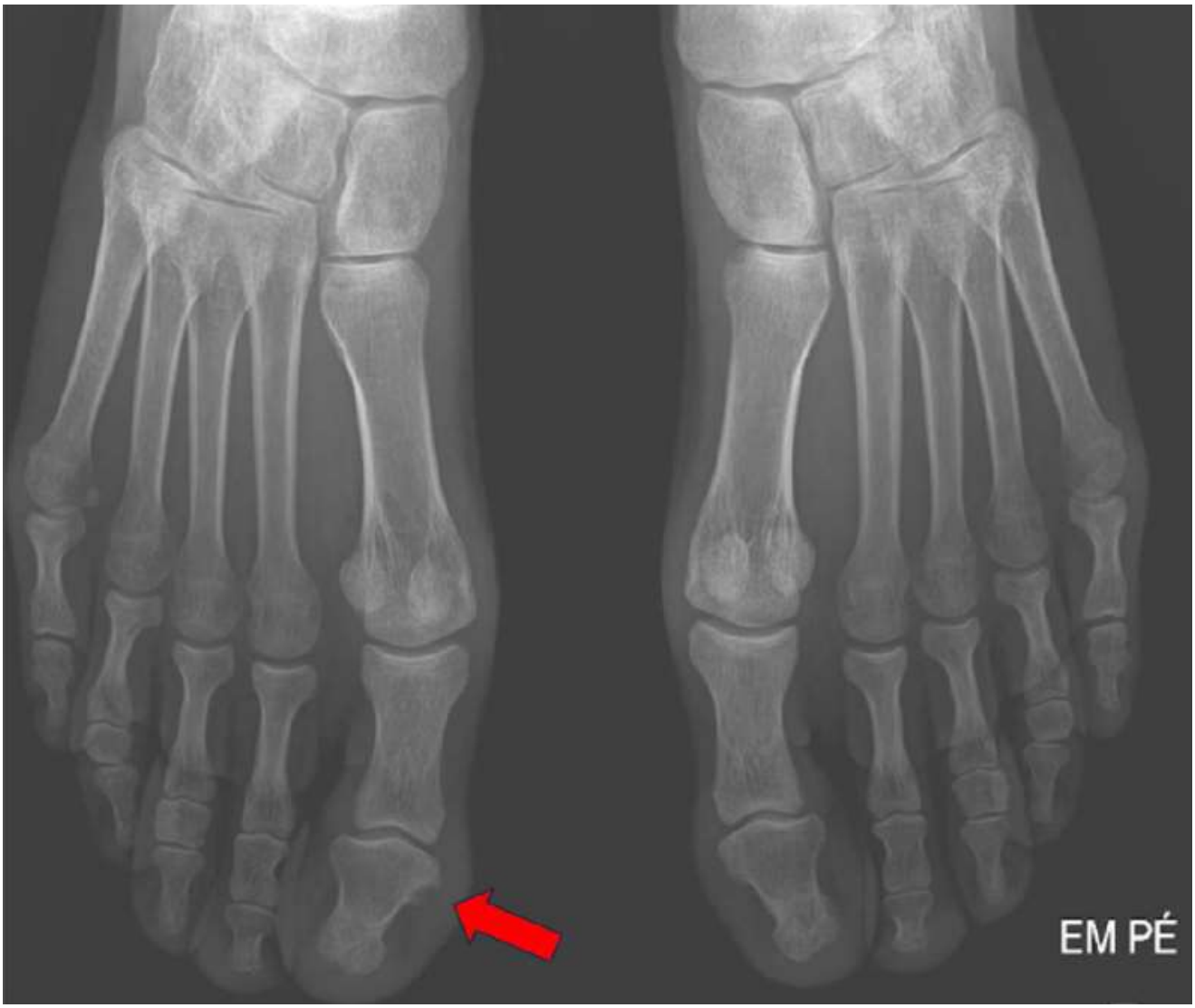

Figura 2 - calo ósseo no hálux direito, caracterizado em radiografia dos pés. Sem outras alterações relevantes.

A investigação requisitada inicialmente, com estudo analítico alargado (Fator Reumatoide, Anti-CCP e HLA B27 ausentes; reagentes de fase aguda dentro do intervalo de normalidade, ausência de leucocitose/neutrofilia) não permitiu esclarecer etiologia, pelo que manteve vigilância em consulta de Reumatologia até esclarecimento etiológico. Durante seguimento, referia manter episódios de agudização a cada 1-2 meses, que pareciam intensificar-se em número e intensidade. Num destes, por refratariedade a analgesia convencional, foi avaliado por Ortopedia num SU, que optou pela realização de RMN (figura 3) deste segmento, que revelou alterações sugestivas de Osteomielite que, por sua vez, motivaram desbridamento cirúrgico e novo ciclo de ATB sistémica.

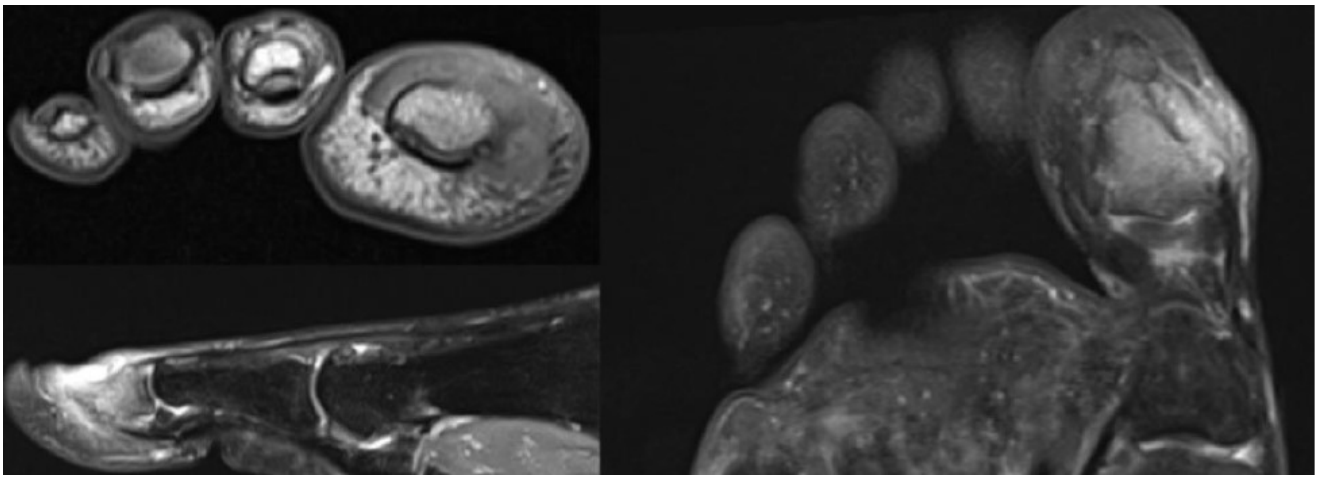

Figura 3 - RMN do hálux direito nos vários planos, onde é visível hipersinal em T2/STIR dos tecidos periungueais e falange distal, mais sugestiva de osteomielite.

Após intervenção, manteve vigilância em consulta de Reumatologia, contudo sem novos episódios de agudização, pelo que acabou por ter alta da mesma.

Conclusão:

A osteomielite é um raro, mas essencial diagnóstico diferencial de artropatia microcristalina, não só pela diferente abordagem diagnóstica, mas também pelo prognóstico, que tende a ser mais agressivo na doença infeciosa, com elevado potencial de destruição óssea e, eventualmente, disseminação hematogénea que, em última instância, pode revelar-se fatal. Além da atipia no diagnóstico diferencial, vários fatores contribuem para o especial interesse neste caso, nomeadamente a apresentação atípica de osteomielite (quer pela localização, quer pela evolução episódica), os múltiplos fatores confundidores, nomeadamente clínica mais sugestiva de gota, antecedentes familiares de AR e Espondilartropatia, faixa etária pouco coincidente, e também a ausência de uma porta de entrada identificada para agentes infeciosos, bem como a ausência de fatores de risco predisponentes a infeção, como é o caso da imunossupressão, traumatismo prévio e doença arterial periférica. No global, estes fatores contribuíram para o atraso diagnóstico de uma entidade já de si, difícil de identificar.

PO CC 44 - ERYTHEMA NODOSUM IN A PATIENT WITH CROHN'S DISEASE ON VEDOLIZUMAB

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Introduction: Erythema nodosum (EN) is the most common form of septal panniculitis and the most frequent skin manifestation associated with inflammatory bowel disease (IBD), affecting up to 15% of Crohn's disease (CD) patients. The most common site of onset is the pretibial region, but it can occur elsewhere. In IBD patients, EN lesions exacerbate during flare-ups of colitis and are correlated with the activity of the underlying disease (extra-intestinal manifestation) but it can be also associated with infections such as bacteria, fungi, and viruses, sarcoidosis, certain medications such as antibiotics, oral contraceptives and malignancies. It is commonly treated with corticosteroids and can be also treated with biologic treatment. The author reports a 34-year-old woman with CD presenting with EN in the setting of vedolizumab therapy.

Case report: A 34-year-old woman, with a 13-year history of CD which required ileocecal resection and a history of poorly controlled intestinal disease (with previous infliximab in 2013 and vedolizumab since 2017), presented with complaints of erythematous, painful bilateral lesions on her lower extremities, and pain in both ankles. Initially, she was treated by her gastroenterologist with anti-inflammatory drug (etoricoxib 90mg id at least 20 days) but without resolution of the problem. On physical examination, she had several tender erythematous nodules on the anterior and posterior aspects of both lower legs, synovitis of her left ankle, synovitis of some proximal interphalangeal joints and presented also with right aquilian enthesitis. Investigations showed leucocytosis (11250), elevation of erythrocyte sedimentary rate (105mm), C-Reactive Protein (90.5mg/l), faecal calprotectin (1033mg/Kg). She started corticosteroids and stopped oral contraceptive, coxib and vedolizumab with substantial improvement. At third week she had no complaints at all. However, in third month of follow-up, she had recurring of similar nodular lesions at both legs again. At that time, again with her IBD not controlled, she had some difficulty to walk because of a painful vulvar nodule in the right vulvar region. A vulva abcess was supected but MRI only detected a Bartholin's cyst, 2cm in diameter, with inflammation. She started amoxiciline that worsens the cutaneous lesions. She was maintained without biologic treatment, restart corticosteroids and sent to Gynaecology Department for surgical intervention.

Discussion / Conclusion: In this case, the relationship of EN was correlated with multiple factors: the patient experiencing CD symptons, because disease was not well controlled (even under vedolizumab), anticoncepcional drug, infectious disease, anti-inflammatory drug and antibiotic. Since the other two biologic treatments (infliximab and vedolizumab) were not effective to treat CD and specifically vedolizumab was not capable to treat or improve cutaneous problem (even with treatment persistence), she was advised to switch to another biologic treatment after the surgical procedure.

In conclusion, this case demonstrates that EN can occur during follow-up of CD and under biologic treatment.

PO 45- MUSCULOSKELETAL DISORDERS AMONG PEOPLE LIVING WITH DIABETES IN A FAMILY HEALTH UNIT

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Context: Diabetes mellitus (DM) is one of the most common diseases worldwide. Patients with DM are prone to develop multiple musculoskeletal (MSK) manifestations, which may lead to disability and an impact on patients' quality of life.

Aims: Assess the prevalence of MSK disorders in diabetic patients in a Family Health Unit (FHU) of Beira Interior-Portugal.

Methods and Materials: This is a retrospective, transversal and descriptive study carried out between 2023 and 2024 involving all patients with DM (type I, type II and LADA), between the age of 10 and 93, looking for patients who were diagnosed with MSK manifestations.

Results: A total of 518 patients were found to have DM but only 482 were included int the study (476 type II, 5 type I and 1 LADA). 54.1% were men and 45.9% women. In this group, 65.4% had MSK manifestations. The average age of patients was 70.5 years old ranging from (10-93). 80.3% had IMC≥25 (2.9% with obesity grade III). The mean reading of A1C was 7.14%. Axial and peripheral osteoarthritis (OA) were the most prevalent pathologies, followed by periarticular pathology (tendinopathy). Carpal tunnel syndrome (CTS) was found only in 8 patients (1.7%). Clinical findings may be more associated with excess weight, the time of disease progression and poor metabolic control.

Conclusions: MSK manifestations are common among diabetic patients. OA was the most frequently seen disorder. Physicians should be aware of the high prevalence and the huge impact on patients when treating patients with type II DM. The early recognition and management can improve quality of life and physical function.

PO 46 - TRATAMENTO MULTIDISCIPLINAR – CASUÍSTICA DA COLABORAÇÃO ENTRE O INSTITUTO PORTUGUÊS DE REUMATOLOGIA E O SERVIÇO DE CIRURGIA PLÁSTICA DA UNIDADE LOCAL DE SAÚDE DE SÃO JOSÉ NOS ÚLTIMOS 2 ANOS E MEIO

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Introdução: A complexidade das doenças reumatológicas frequentemente implica manifestações clínicas com impacto funcional, estético e psicológico significativos para os doentes. Nestes contextos, a colaboração interdisciplinar assume um papel determinante na resposta integrada ao doente. A parceria entre o Instituto Português de Reumatologia (IPR) e o Serviço de Cirurgia Plástica e Reconstrutiva (CPR) da Unidade Local de Saúde (ULS) São José surge como um modelo de cooperação que visa otimizar resultados clínicos, melhorar a qualidade de vida e garantir uma abordagem holística e centrada no doente. Este trabalho apresenta a experiência desta colaboração nos últimos 2 anos e meio, destacando a importância da articulação entre especialidades médicas e cirúrgicas no tratamento de casos complexos.

Objetivos: Caraterizar a colaboração entre o IPR e o Serviço de CPR da ULS São José, através da análise do

número de consultas realizadas, bem como do número e tipo de cirurgias efetuadas, neste contexto.

Descrever o perfil dos doentes acompanhados nesta colaboração, incluindo dados epidemiológicos, patologias mais frequentes e intervenções cirúrgicas realizadas.

Material e Métodos: Estudo retrospetivo dos doentes observados em contexto da colaboração entre o IPR e o Serviço de CPR da ULS São José entre janeiro de 2023 e maio de 2025. Os dados foram obtidos através da consulta dos processos clínicos. As variáveis estudadas incluíram a idade, sexo, patologia, presença de indicação cirúrgica, número e tipo de cirurgias realizadas.

Resultados: Durante o período analisado foram realizadas 142 consultas no IPR (135 primeiras consultas e 7 subsequentes).

Verificou-se uma preponderância de doentes do sexo feminino (82,3%) e de doentes entre os 40 e 64 anos de idade (6,4% com < 40 anos; 53,2% dos 40-64 anos; 23,4% dos 65-74 anos e 12,8% com >/= 75 anos).

As patologias mais frequentes foram por ordem decrescente, a artrite reumatoide (19,1%), a síndrome do túnel cárpico (18,4%), a rizartrose (13,5%) e a osteoartrose de outras localizações da mão (8,5%).

Dos doentes observados, 91 apresentavam indicação cirúrgica (64,5%). Destes, 35 não quiseram ser intervencionados (38,5%), 48 já foram intervencionados (52,7%) e 8 encontram-se em lista de espera (8,8%).

As cirurgias mais frequentemente realizadas foram a abertura do ligamento anular anterior do carpo e neurólise do nervo mediano para tratamento da síndrome do túnel cárpico, abertura da poleia A1 para tratamento de tenossinovite estenosante e correção cirúrgica de rizartrose através da técnica de *Burton-Pellegrini*.

Conclusão: A colaboração entre o IPR e o Serviço de CPR da ULS São José demonstrou elevado impacto assistencial, traduzido num número significativo de consultas e intervenções cirúrgicas. Verificou-se uma predominância de doentes do sexo feminino e em idade ativa, com destaque para a artrite reumatoide,

síndrome do túnel cárpico e rizartrose como principais patologias abordadas. A maioria dos doentes apresentou indicação cirúrgica, sendo que mais de metade foi já submetida a intervenção. Estes resultados reforçam a importância da articulação multidisciplinar na abordagem de doentes com patologia reumatológica da mão, permitindo otimizar o tratamento e melhorar a qualidade de vida.

PO 47 NAILFOLD CAPILLAROSCOPY IN MIXED CONNECTIVE TISSUE DISEASE: INSIGHTS FROM THE CAPRAS REGISTRY

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Background: Mixed connective tissue disease (MCTD) is a rare systemic autoimmune disorder with overlapping features mostly of systemic lupus erythematosus (SLE), systemic sclerosis (SSc), and inflammatory myopathies; Raynaud's phenomenon (RP) is a prominent feature. Nailfold capillaroscopy assesses RP, distinguishes primary from secondary forms, aids early SSc recognition, provides prognostic insights, and monitors microvascular changes. However, in MCTD, microvascular changes are poorly defined.

Objective: To compare nailfold capillaroscopy findings among patients with MCTD, SSc, and controls without systemic autoimmune rheumatic disease (SARD) with RP, and to explore associations between capillaroscopic patterns and predominant MCTD phenotypes.

Methods: We performed a cross-sectional study with patients registered in the CAPillaroscopy Registry Almada-Seixal (CAPRAS), a database with nailfold capillaroscopies performed in our center since 2014. Capillaroscopies were conducted using a digital microscope (Dino-Lite CapillaryScope 200 pro). All adult patients with a clinical diagnosis of MCTD were included, as well as randomly selected controls (SSc and non-SARD patients). Descriptive statistics were calculated for all variables. Continuous data were presented as mean ± SD or median (IQR) per Shapiro–Wilk test; categorical data as frequencies and percentages. Group comparisons (MCTD, SSc, non-SARD) used ANOVA or Kruskal–Wallis for continuous variables, and chi-square or Fisher's exact for categorical, with Bonferroni-corrected post hoc tests when $p<0.05$. Analyses were performed in Python® 3.13.

Results: A total of 112 patients were included (32 MCTD, 40 SSc, 40 non-SARD). MCTD patients were younger than SSc and non-SARD (43.6 ± 16.6 vs 58.4 ± 14.6 vs 50.0 ± 17.9 years; $p=0.001$), with no differences in sex or smoking. Capillaroscopy patterns differed significantly ($p<0.001$): SSc predominantly exhibited scleroderma pattern (75%), MCTD heterogeneous (scleroderma 28.1%, scleroderma-like 28.1%, non-specific 34.4%), non-SARD mainly non-specific (65%) or normal (35%).

Megacapillaries and decreased capillary density were most prevalent in SSc (62.5% each), intermediate in MCTD (34.4%), and lowest in non-SARD (0% and 2.5%, respectively; $p<0.001$). Capillary dilations were frequent in SSc (69.2%) and MCTD (68.8%), contrasting with the non-SARD group (40.0%; $p=0.012$). Hemorrhages were higher in SSc (72.5%) than non-SARD (42.5%; $p=0.024$). Neoangiogenesis occurred in SSc (35%) and MCTD (28.1%) but not in non-SARD.

Among MCTD patients, scleroderma and scleroderma-like patterns tended to align with SSc-like phenotype (n=5, 55.6% and n=6, 66.7%, respectively), while normal/non-specific patterns aligned more often with

SLE-like/other phenotypes, without reaching statistical significance (p=0.078).

Discussion: This study shows a capillaroscopic gradient: SSc patients exhibit marked microvascular damage, non-SARD controls mostly normal or non-specific findings, and MCTD patients show intermediate, heterogeneous abnormalities reflecting variable "scleroderma burden." MCTD patients were younger than SSc in our sample, consistent with known epidemiology. However, these results should be interpreted with caution, as the cross-sectional design captures only a snapshot of a dynamic process. Thus, age and unrecorded disease duration may confound findings. Within MCTD, the trend toward alignment of scleroderma/scleroderma-like capillaroscopic patterns with SSc-like clinical phenotypes did not reach statistical significance, likely reflecting the limited sample size. Scleroderma patterns could indicate greater SSc-related burden and justify closer surveillance. However, further studies with larger cohorts and longitudinal follow-up are needed to better correlate capillaroscopic patterns with clinical disease manifestations.

Table 5 – Demographic and capillaroscopic findings comparison between MCTD, SSc and patients without SARD.

Variable	MCTD (n=32)	SSc (n=40)	No SARD (n=40)	p-value	p (post-hoc)
Demographic characteristics					
Age (years), mean ± SD	43.6 ± 16.6	58.4 ± 14.6	50.0 ± 17.9	0.001	
Female sex, n (%)	29 (90.6)	36 (90.0)	31 (77.5)	0.18	
Smoker (current/past), n/N (%)	7/29 (24.1)	7/31 (22.6)	8/37 (21.6)	0.971	
Capillaroscopic findings					
Capillaroscopic pattern, n (%)				<0.001	MCTD vs SSc: <0.001 MCTD vs No SARD: <0.001 SSc vs No SARD: <0.001
Normal	3 (9.4)	4 (10.0)	14 (35.0)		
Non-specific changes	11 (34.4)	5 (12.5)	26 (65.0)		
Scleroderma pattern*	9 (28.1)	30 (75.0)	0		
Scleroderma-like	9 (28.1)	1 (2.5)	0		
Hemorrhages, n (%)	19 (59.4)	29 (72.5)	17 (42.5)	0.024	MCTD vs SSc: 0.471 MCTD vs No SARD: 0.471 SSc vs No SARD: 0.037
Dilations (20-50 µm), n (%)	22 (68.8)	27 (69.2)	16 (40.0)	0.012	MCTD vs SSc: 1.0 MCTD vs No SARD: 0.039 SSc vs No SARD: 0.039
Megacapillaries (>50 µm), n (%)	11 (34.4)	25 (62.5)	0		MCTD vs SSc: 0.032
Decreased density (≤7 capillaries/mm), n (%)	11 (34.4)	25 (62.5)	1 (2.5)	<0.001	MCTD vs SSc: 0.032 MCTD vs No SARD: 0.001 SSc vs No SARD: <0.001

Neoangiogenesis, n (%)	9 (28.1)	14 (35.0)	0		MCTD vs SSc: 0.616

MCTD, mixed connective tissue disease; SARD, systemic autoimmune rheumatic disease; SSc, systemic sclerosis; SD, standard deviation

*includes early, active and late patterns

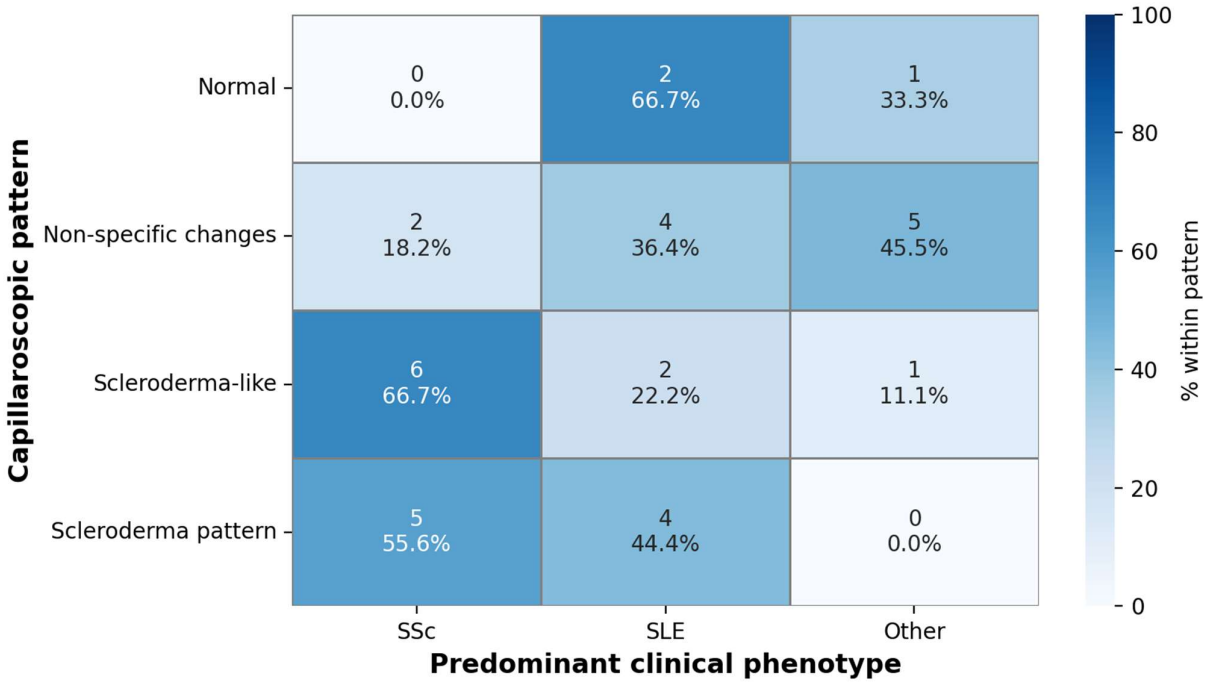

Figure 1 – Distribution of clinical phenotypes by capillaroscopy pattern in MCTD patients.

PO 48 - SHOULD PAIN ASSOCIATED WITH FIBROMYALGIA BE CONSIDERED AN INDICATION TO SUBSIDIZED BALNEOTHERAPY IN PORTUGAL? AN EVIDENCE-BASED REVIEW

Sandra Torres, Bruno Daniel Carneiro, Vera Costa, Daniela Santos Faria

Introduction: Fibromyalgia (FM) is a chronic, heterogeneous syndrome marked by widespread musculoskeletal pain, often associated with fatigue, sleep, mood, and cognitive disturbances. Predominantly affecting women, it has a major impact on quality of life and socioeconomic burden. Diagnosis relies on clinical criteria due to symptom overlap and lack of biomarkers. Its pathophysiology involves central sensitization, requiring a multidimensional treatment approach. Among non-pharmacological options, balneotherapy (BT) shows promising benefits through thermal, mechanical, and neuroendocrine effects, though it is not yet reimbursed for FM by the Portuguese National Health Service.

Objectives: This review aims to assess the available evidence on the effectiveness of BT in reducing pain in FM and to support its potential inclusion in the list of reimbursable indications.

Methods: Search for clinical guidelines (CG), systematic reviews (SR) with or without meta-analyses (MA), and randomized controlled trials (RCT), published between 01/01/2004 and 31/12/2024, in Portuguese, Spanish, French and English languages, using the MeSH terms "fibromyalgia", "hydrotherapy" and "balneology" were performed. The American Family Physician's Strength of Recommendation Taxonomy (SORT) scale was used to assign levels of evidence (LE) and strengths of recommendation (SR).

Results: A total of 234 articles were identified, and 9 met the inclusion criteria: 1 CG, 2 MA, and 6 RCT. The European League Against Rheumatism (EULAR) guideline states that, despite low-quality evidence, consistent findings indicate that BT significantly reduces pain in FM, with effects lasting up to 14 weeks, recommending BT as an adjunctive treatment (LE 2). The MA by García-López (2024) found large and significant pain reductions at all time points ($p = 0.04$ immediately; $p < 0.001$ at 1 month; $p = 0.003$ at 3 months; $p = 0.002$ at 6 months). Similarly, the MA by Naumann et al. (2014) showed significant pain reduction after treatment ($p = 0.002$) and a smaller but maintained effect at follow-up ($p = 0.01$). Both MA concluded that BT is effective, safe, and well tolerated (LE 1). Among the RCT, Pérez-Fernández et al. (2019) reported significant FIQ score reductions after treatment and at 1 and 3 months ($p < 0.001$); Fioravanti et al. (2018) found significant pain improvement at 2 weeks, 3, and 6 months ($p < 0.05$); and Fioravanti et al. (2007) observed significant improvement in all parameters after treatment and at 16 weeks ($p < 0.0001$). These RCT provide high-quality evidence supporting BT efficacy (LE 1). Additional trials – Dönmez et al. (2005), Bağdatlı et al. (2015), and Koçyiğit et al. (2016) – also demonstrated significant and sustained pain reduction ($p \leq 0.045$; $p < 0.001$; $p < 0.05$, respectively), reinforcing BT's benefits though with less robust evidence (LE 2).

Conclusion: BT proves to be a safe, well-tolerated, and clinically effective NP intervention in reducing pain in FM (SR A). Given its demonstrated benefits, current evidence supports broader accessibility, including consideration of reimbursement policies, and it is reasonable that FM be included among the recognized indications for subsidized BT in Portugal.

PO CC 49 - UNCOMMON PRESENTATION OF POPLITEAL HEMATOMA MIMICKING BAKER'S CYST

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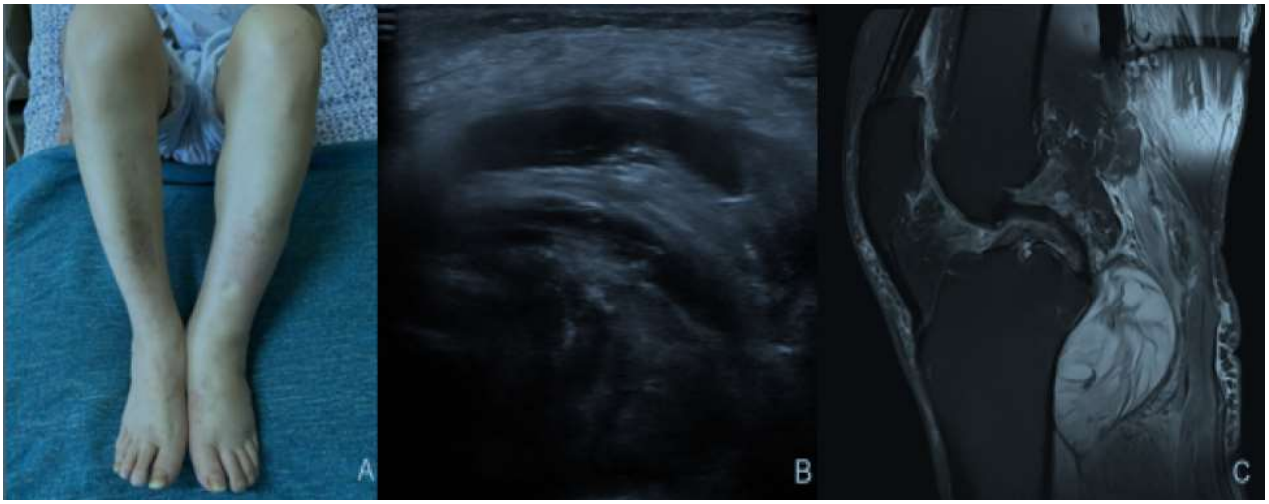
Clinical case: A 75-year-old male presents with one month of asymmetric oedema and neuropathic pain in the left leg. He had no history of recent trauma or surgery. On physical examination, he presented with an antalgic gait, with positive godet oedema of the left leg, up to the knee (A), with a 5º flexion deformity and no apparent effusion. The distal pulses were palpable, but he had a pattern of hypoesthesia in the external face of the leg and left foot, without other focal neurological or muscle strength deficits.

Doppler ultrasound excluded deep vein thrombosis. Left knee ultrasound revealed extensive synovitis, characterized by significant synovial hypertrophy and a grade II power Doppler signal. Additionally, a heterogeneous, hypoechoic, and poorly compressible formation, lacking Doppler signal, was identified in the posterior knee (B). While the findings raised suspicion for a Baker's cyst, its atypical location in the posterolateral knee— deep to the tibial nerve and popliteal artery, with suspected neurovascular compression, and extension into the lower leg— raised concern and prompted consideration of alternative diagnoses.

A magnetic resonance imaging (MRI) scan was requested, which revealed findings compatible with a hematoma secondary to rupture of the popliteal muscle, measuring 66 x 46 x 98 mm. This hematoma

was causing compression of the popliteal vessels and the left tibial nerve, along with significant gonarthrosis, medial meniscal maceration, and synovitis, with surrounding soft tissue oedema (C). Ultrasound guided aspiration resulted in rapid symptom relief. The patient's neuropathic pain improved significantly within a few months with physical therapy and rehabilitation.



Discussion and conclusion: Not all masses in the popliteal region are Baker's cysts, the differential diagnosis includes abscess, deep vein thrombosis, hematoma, solid tumour masses, among others. Clinically, the rupture of a Baker's cyst can also lead to compression of the posterior tibial nerve and occlusion of the popliteal artery, which can present with symptoms similar to those seen in this case.1

A thorough evaluation with ultrasound is essential to confirm the typical features of a Baker's cyst, including its characteristic location in the posteromedial aspect of the knee joint, situated between the medial head of the gastrocnemius muscle and the semimembranosus tendon, often communicating with the knee joint. Additionally, the absence of colour Doppler signal can help differentiate it from other conditions.

Although rupture of the popliteus muscle or tendon is very rare, it should be considered in the differential diagnosis of atypical popliteal fossa lesions. Most reported cases occur in young athletes or following high impact trauma. In older patients, spontaneous or atraumatic ruptures, as in this case, are exceptional and can mimic more frequent conditions such as Baker's cysts.2,3 In complex cases, namely when neurovascular compression is suspected, the use of MRI may be pivotal in establishing the diagnosis and help guiding the treatment.

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PO 50 - PRIMARY SJÖGREN'S SYNDROME: A COHORT STUDY FROM A PORTUGUESE TERTIARY CENTER

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** Contributed equally to the development of this work.*

Introduction: Primary Sjögren's syndrome (pSS) is a systemic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands, resulting in sicca symptoms and a wide range of systemic manifestations.1 The aim of this study is to characterize the cohort of pSS patients followed at the Rheumatology Department of Coimbra's Local Health

Unit, Portugal.

Methods: This retrospective, observational study included adult patients identified from our electronic clinical files and registry fulfilling the ACR/EULAR 2016 classification criteria for pSS. Demographic, clinical, serological, and therapeutic data were collected. Statistical analysis was performed using SPSS software (version 29.0), with results expressed as frequencies, medians, and interquartile ranges (IQR) as appropriate.

Results: A total of 151 patients with pSS were included: 93.4% female, with a median age of 65.0 years (IQR: 20.0), and a median age at diagnosis of 52.4 years (IQR: 10.2). Detailed characterization of this pSS cohort is provided in table 1.

Among these patients, 94.7% presented xerostomia and 98.7% xerophthalmia, with 85.9% having a positive Schirmer's test. Extra-glandular involvement was observed in 84.8% of patients. The most frequent was musculoskeletal (33.8%), mainly with inflammatory arthralgias, followed by Raynaud's phenomenon (23.8%). Neurological involvement was identified in 4.6%, including both peripheral and central nervous system manifestations (most frequently, with sensitive polyneuropathy). Renal involvement occurred in 4.0%, half of them with membranoproliferative glomerulonephritis. Cutaneous manifestations were present in 18.5% (from these, 53.6% presented vascular lesions and 46.4% had photosensitive rash). Pulmonary involvement was reported in 9.3%, with interstitial lung disease being the most frequent. Hematologic abnormalities comprised lymphopenia (40.4%), leukopenia (39.1%), and anemia (32.5%).

Immunological features included positive ANA (99.3%), with high titers (≥1:1280) observed in 27.2%. Anti-Ro/SSA antibodies were positive in 90.1%, anti-SSA52 in 25.8%, anti-La/SSB antibodies in 47.7%, and rheumatoid factor in 50.3%. Hypocomplementemia was reported in 23.2%, mainly low C3.

The most frequent comorbidities were hypertension (47.7%), osteoarthritis (45.7%), depression (39.1%) and fibromyalgia (34.4%). Seven patients (4.6%) had a history of lymphoma.

Treatment included ophthalmic solutions (88.1%), pilocarpine (23.8%), hydroxychloroquine (35.1%), and glucocorticoids (9.9%) with a median prednisolone dose of 5.0 mg (IQR 2.5).

Discussion and Conclusion: The pSS patients in this cohort present clinical, analytical and immunological features that are concordant with other reference cohorts in Europe. Over one third of these patients presented multisystemic features of pSS and over 40% required treatment with immunosuppressants or immunomodulators.

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Table 1. Demographic, Clinical, and Immunological Features of Primary Sjögren's Syndrome.

Variables	Whole Cohort, n=151 (100%)
Demographic features	
Current age, median (IQR)	65.0 (20.0)
Age at pSS diagnosis, median (IQR)	52.4 (10.2)
Female sex (n [%])	141 (93.4)
Clinical features [n (%)]	
- Xerophthalmia	149 (98.7)
.: Positive Schirmer test	128 (85.9)
- Xerostomia	143 (94.7)
- Parotitis	23 (15.2)
- Parotid gland enlargement	16 (10.6)
- Lymphadenopathies	8 (5.3)
- Raynaud's phenomenon	36 (23.8)
- Articular involvement	51 (33.8)
.: Inflammatory arthralgias	42 (82.4)
.: Arthritis	21 (41.2)
- Neurological involvement	7 (4.6)
.: Central Nervous System	3 (42.9)
.: Peripheral Nervous System	4 (57.1)
- Renal involvement	6 (4.0)
.: Glomerular	3 (50.0)
.: Tubulointerstitial nephritis	2 (33.0)
.: Distal renal tubular acidosis	1 (17.0)
.: Autoimmune Cystitis	1 (17.0)
- Cutaneous involvement	28 (18.5)
.: Vascular Manifestations	15 (53.6)
.: Photosensitive / Lupus-like rash	13 (46.4)
.: Other	10 (35.7)
- Pulmonary involvement	14 (9.3)
.: Interstitial lung disease	(71.4)
.: Bronchial involvement	(28.6)
- Miositis	1 (0.7)
- Serositis	2 (1.3)

Previous or current treatments [n (%)]	
- Ophthalmic solution	133 (88.1)
- Oral spray	15 (9.9)
- Pilocarpine	36 (23.8)
- Non-steroidal anti-inflammatory drugs	26 (17.2)
- Immunosuppressants/ immunomodulators	64 (42.4)
- Hydroxychloroquine	53 (35.1)
- Methotrexate	3 (2.0)
- Leflunomide	1 (0.7)
- Azathioprine	10 (6.6)
- Mycophenolate mofetil	3 (2.0)
- Prednisolone	15 (9.9)
.: Prednisolone dose (median (IQR))	5.0 (2.5)
- Inpatient admission	32 (21.2)
Common comorbidities [n (%)]	
- Fibromyalgia	47 (34.4)
- Chronic fatigue	52 (34.2)
- Depression	59 (39.1)
- Osteoarthritis	69 (45.7)
- Osteoporosis	31 (20.5)
- Hypertension	72 (47.7)
- Diabetes	15 (9.9)
- Dyslipidemia	47 (31.1)
- Heart failure	5 (3.3)
- Chronic obstructive pulmonary disease	3 (2.0)
- Neoplasia	28 (18.5)
.: Hematologic	10 (7.0)
* Lymphoma	7 (4.6)
.: Breast	8 (5.3)
.: Skin	3 (2.1)
.: Genitourinary tract	4 (2.8)
.: Gastrointestinal tract	2 (1.4)
.: Thyroid	2 (1.4)
.: Musculoskeletal	1 (0.7)
- Autoimmune hepatitis	3 (2.0)
- Autoimmune thyroiditis	20 (13.2)
Laboratory features [n (%)]	

- Anemia (<11.8 g/ dL)	49 (32.5)
- Leukopenia (<3.6 x10^{3}L)	59 (39.1)
- Neutropenia (<1.5 x10^{3}L)	38 (25.2)
- Lymphopenia (<1.1 x10^{3}L)	61 (40.4)
- Thrombocytopenia (<140 x10^{3}L)	26 (17.2)
- Hypergammaglobulinemia	93 (61.6)
- Hypocomplementemia	35 (23.2)
.: Hypocomplementemia C3	25 (71.4)
.: Hypocomplementemia C4	19 (54.3)
- Anti-dsDNA	6 (4.0)
- Antinuclear antibodies	150 (99.3)
- Titer >1/1280	41 (27.2)
- Anti-Ro/SSA positive	136 (90.1)
- Anti-SSA52	39 (25.8)
- Anti-La/SSB positive	72 (47.7)
- Anti-RNP positive	2 (1.3)
- Rheumatoid factor	76 (50.3)

IQR: Interquartile range

PO CC 51 - ESCLEROSE CUTÂNEA: A IMPORTÂNCIA DA REUMATOLOGIA NA ABORDAGEM DIAGNÓSTICA

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INTRODUÇÃO: A esclerose cutânea é uma manifestação clínica que se caracteriza pelo espessamento da pele como consequência do aumento da produção de colagénio. É uma manifestação típica da esclerose sistémica, doença que se associa à presença de autoanticorpos e na qual o fenómeno de Raynaud é um sintoma clássico, podendo ocorrer o envolvimento de outros sistemas orgânicos.

Na esclerose cutânea localizada, verifica-se um espessamento cutâneo sem envolvimento de outros órgãos, de que são exemplos a morfeia (placas de espessamento cutâneo) e a esclerodermia linear (atingimento em bandas lineares). No entanto, em alguns casos, o atingimento pode ser mais extenso e profundo e pode originar manifestações como a limitação na mobilidade articular, mimetizando um atingimento cutâneo mais típico de esclerose sistémica.

Caso clínico: Doente do sexo masculino de 30 anos, referenciado à consulta de Reumatologia em agosto/2024, por quadro com 4 anos de evolução de lesões cutâneas hiperpigmentadas na região do tronco e cotovelos e limitação da mobilidade ao nível dos dedos das mãos e cotovelos. Tinha sido previamente observado em hospital privado onde realizou videocapilaroscopia do leito ungueal com descrição sugestiva de padrão esclerodérmico em fase ativa (capilares dilatados e megacapilares, desestruturação arquitetura, micro-hemorragias e neoangiogénese), sendo referenciado com suspeita de esclerose sistémica.

Na anamnese, o doente referiu as lesões cutâneas e a limitação na mobilidade previamente descritas e negou queixas sugestivas de conectivite, nomeadamente fenómeno de Raynaud ou outras queixas sistémicas.

Objetivamente, apresentava esclerose cutânea ao nível dos dedos das mãos até à região das metacarpofalângicas com flexo dos dedos e lesões cutâneas espessadas com áreas de hipo e hiperpigmentação ao nível da face extensora dos cotovelos, região abdominal (principalmente nas fossas ilíacas) e dorsal (imagem 1). Não se observou artrite periférica, úlceras digitais, pitting scars, telangiectasias ou microstomia.

Do estudo complementar pedido, de realçar estudo analítico normal (incluindo proteína C-reativa e velocidade de sedimentação normais e estudo imunológico negativo, nomeadamente anticorpos anti-nucleares e painel de anticorpos de esclerose sistémica). A videocapilaroscopia do leito ungueal realizada no serviço de Reumatologia foi normal.

Tendo em conta os achados, o doente foi referenciado à consulta de Dermatologia em novembro/2024, tendo realizado biópsia cutânea (de lesão cutânea da fossa ilíaca) com o resultado a corroborar o diagnóstico de morfeia. Em fevereiro/2025 iniciou tratamento por Dermatologia com corticosteróides e tacrolimus tópicos, prednisolona (10mg/dia) e azatioprina (em dose crescente) per os.

Conclusão: O diagnóstico de esclerose cutânea localizada pode ser desafiante quando as manifestações cutâneas não são evidentes à apresentação da doença. No caso de estarmos perante flexos articulares irredutíveis sem
artrite objetivável ou manifestações clínicas de conectivite, devemos pensar numa patologia que atinja apenas a pele e tecidos moles subjacentes, como é o caso da morfeia. Tal como se descreve no caso clínico, é importante a distinção entre morfeia e esclerose sistémica, tendo em conta a diferença nas manifestações clínicas, prognóstico e orientação das duas entidades. Além disso, não há um risco aumentado de esclerose sistémica nos doentes com morfeia.

A evolução das lesões de morfeia é variável e as opções terapêuticas têm evidência limitada, incluindo desde tratamentos tópicos a imunossupressores sistémicos.

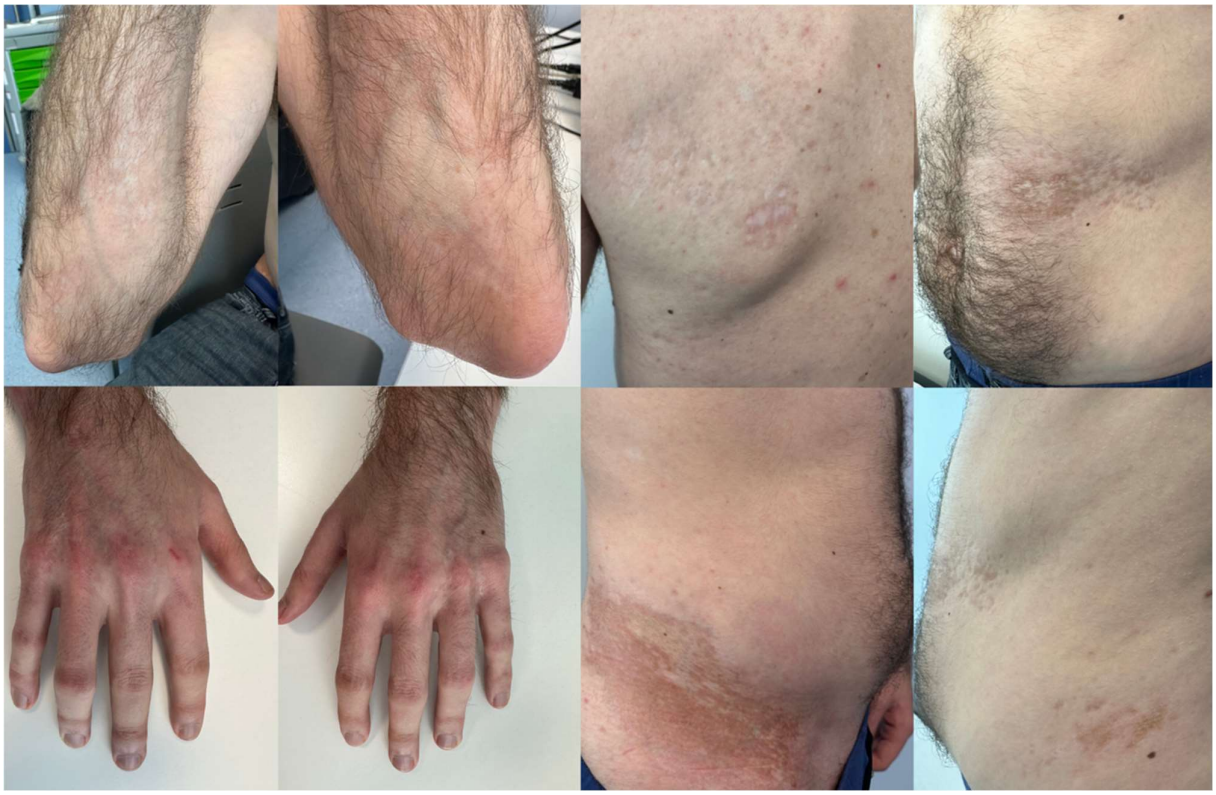

IMAGEM 1: Lesões de esclerose cutânea localizada ao nível dos cotovelos, tronco e face dorsal das mãos.

PO CC 52 - UMA EXTENSÃO DOLOROSA - SÍNDROME DE SINDING-LARSEN-JOHANSSON

Tiago Rei Miranda, Marta Lopes, Diogo Cardadeiro, Ana Pereira, Ana Rita Pessoa, Fábio Pinho, Rui Santos, Pedro Soares Branco

ULS São José

A Síndrome de Sinding-Larsen-Johansson (SLJ) é uma osteocondrose por tração que afeta o polo inferior da rótula, no ponto de inserção proximal do tendão rotuliano. Ocorre tipicamente em crianças e adolescentes ativos, entre os 10 e 14 anos de idade, sendo mais frequente em praticantes de desportos com saltos e movimentos repetidos de flexão e extensão do joelho (como basquetebol, futebol, voleibol e atletismo). A dor localiza-se habitualmente na região inferior da rótula, agravando-se com atividades físicas (como correr, saltar ou subir escadas) e alivia com o repouso. O diagnóstico é essencialmente clínico, apoiado pela história do doente e exame físico, podendo ser confirmado por radiografia evidenciando fragmentação, irregularidade ou esclerose do polo inferior da rótula.

O objetivo deste trabalho passa por apresentar um caso clínico de SLJ num adolescente atleta, descrevendo o quadro clínico, diagnóstico, abordagem terapêutica e evolução, de modo a ilustrar o curso típico e o bom prognóstico desta patologia.

Trata-se de um doente do sexo masculino, de 13 anos, jogador federado de basquetebol, com gonalgia bilateral com 5 meses de evolução. O estudo baseou-se na observação clínica, exame físico detalhado e exame de imagem (radiografia dos joelhos). Posteriormente, foi implementado um programa de reabilitação, com acompanhamento evolutivo e reavaliação sintomática.

O doente referido apresentava gonalgia bilateral, mais intensa à esquerda, com 5 meses de evolução, de ritmo mecânico e intensidade moderada a intensa. Referia agravamento da dor ao subir e descer escadas, correr, saltar e ao mudar de direção, com END basal de 0/10 e END pico de 8/10. Ao exame objetivo, constatou-se dor à palpação do polo inferior da rótula bilateralmente, amplitudes articulares preservadas, com dor na flexão máxima e dor à extensão resistida dos joelhos bilateralmente. A radiografia dos joelhos confirmou a suspeita diagnóstica de SLJ. O paciente iniciou programa de reabilitação, com melhoria sintomática significativa, apresentando atualmente END 3/10 no pico da dor, sem necessidade de medicação analgésica.

A SLJ representa uma entidade benigna e autolimitada, relacionada com a tração repetida do tendão rotuliano sobre o polo inferior da rótula durante o crescimento. O tratamento é conservador, baseado na educação para a saúde e repouso relativo, com redução ou suspensão de atividades dolorosas, aplicação de gelo e eventual uso de anti-inflamatórios e um programa de reabilitação, focado no alongamento e fortalecimento muscular da musculatura da perna e do joelho. O prognóstico é bom, sendo uma condição autolimitada, que tende a resolver-se completamente com o fim do crescimento, sem deixar sequelas. O reconhecimento precoce e a abordagem adequada permitem a recuperação funcional completa e o retorno seguro à prática desportiva.

PO 53 WHEN SPONDYLOARTHRITIS TURNS TO VASCULITIS

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Introduction: Immunoglobulin A (IgA) vasculitis is a small-vessel vasculitis characterized by IgA1-dominant immune complex deposition, typically presents with palpable purpura, arthralgia, abdominal pain,

and renal involvement1, 2. Although mainly paediatric, adult-onset cases can be more severe and atypical, often mimicking other inflammatory disorders such as inflammatory bowel disease (IBD)or spondyloarthritis. We report a case initially diagnosed as Crohn's disease with enteropathic spondyloarthritis, later reclassified as IgA vasculitis after multisystem involvement.

Clinical case: A 54-year-old man presented in November 2024 with two days of colicky abdominal pain and vomiting. He denied fever, diarrhea, or hematochezia. Examination revealed diffuse abdominal tenderness without peritoneal signs. C-reactive protein was elevated (7.66mg/dL), with no otherrelevant abnormalities. Abdominal CT showed parietal thickening of multiple ileal loops with prominent mesenteric vessels and interloop fat stranding. He was admitted for further evaluationand started on ciprofloxacin and metronidazole. Abdominal ultrasound showed inflammatory changes in the cecal region, and colonoscopy revealed ulcerated, friable mucosa causing narrowing of the ileal lumen. Biopsies showed ulcerated mucosa.

Suspecting IBD, particularly Crohn's disease, prednisolone 40 mg/day was started. Recurrent abdominal distension prompted repeat abdominal CT, showing thickened, hyperenhancing smallbowel loops with several air–fluid levels and moderate colonic dilatation. Azathioprine 50 mg/day and infliximab 5 mg/kg were added for immunosuppression. During hospitalization, he developed bilateral knee and ankle pain and a purpuric dermatosis consisting of millimetric, non-blanching papules on the feet, legs, thighs, and abdomen.

Rheumatology confirmed oligoarthritis of the knees and ankles, with Achilles enthesitis and calf pain. Extensive laboratory testing—including autoantibodies, complement, immunoglobulins, cryoglobulins, and urinary sediment—was unremarkable. A cutaneous small- and medium-vessel vasculitis was diagnosed by Dermatology, and initially considered drug-induced. Subsequent skin biopsy revealed capillaritis, and prednisolone 0.5 mg/kg was initiated.

A diagnosis of enteropathic spondyloarthritis was made, and infliximab and azathioprine were resumed, leading to resolution of arthritis, skin lesions, and intestinal symptoms. He was discharged in mid-December 2024Three days later, he returned with recurrent purpura aftersteroid tapering and haematuria. Prednisolone 60 mg/day was restarted with partial improvement.

In January 2025, he presented with right foot drop, dorsal foot hypoesthesia, testicular pain, and calf discomfort. Electroneuromyography showed a conduction block with focal axonal lesion of the right peroneal nerve. The diagnosis of IgA vasculitis was established.

Azathioprine and infliximab were discontinued, and rituximab 1 g was initiated. After two infusions, neurological recovery was complete, prednisolone was successfully tapered, and follow-up electromyography was normal. Nephrology evaluation revealed elevated albumin–creatinine and protein–creatinine ratios, confirming minor renal involvement.

The patient maintained remission with no evidence of active articular, gastrointestinal, cutaneous, or neurological disease.

Discussion: This case highlights the diagnostic overlap between IBD-associated inflammation and IgA vasculitis. Gastrointestinal and articular symptoms mimicked Crohn's disease with enteropathic spondyloarthritis, but subsequent cutaneous, neurological, and renal involvement established the diagnosis of IgA vasculitis. Rituximab led to full recovery and sustained remission.

Conclusion: Adult-onset IgA vasculitis can masquerade as IBD or spondyloarthritis. Diagnostic reassessment is essential when systemic features evolve. Early recognition of multisystem disease and timely rituximab therapy were crucial for a favourable outcome.

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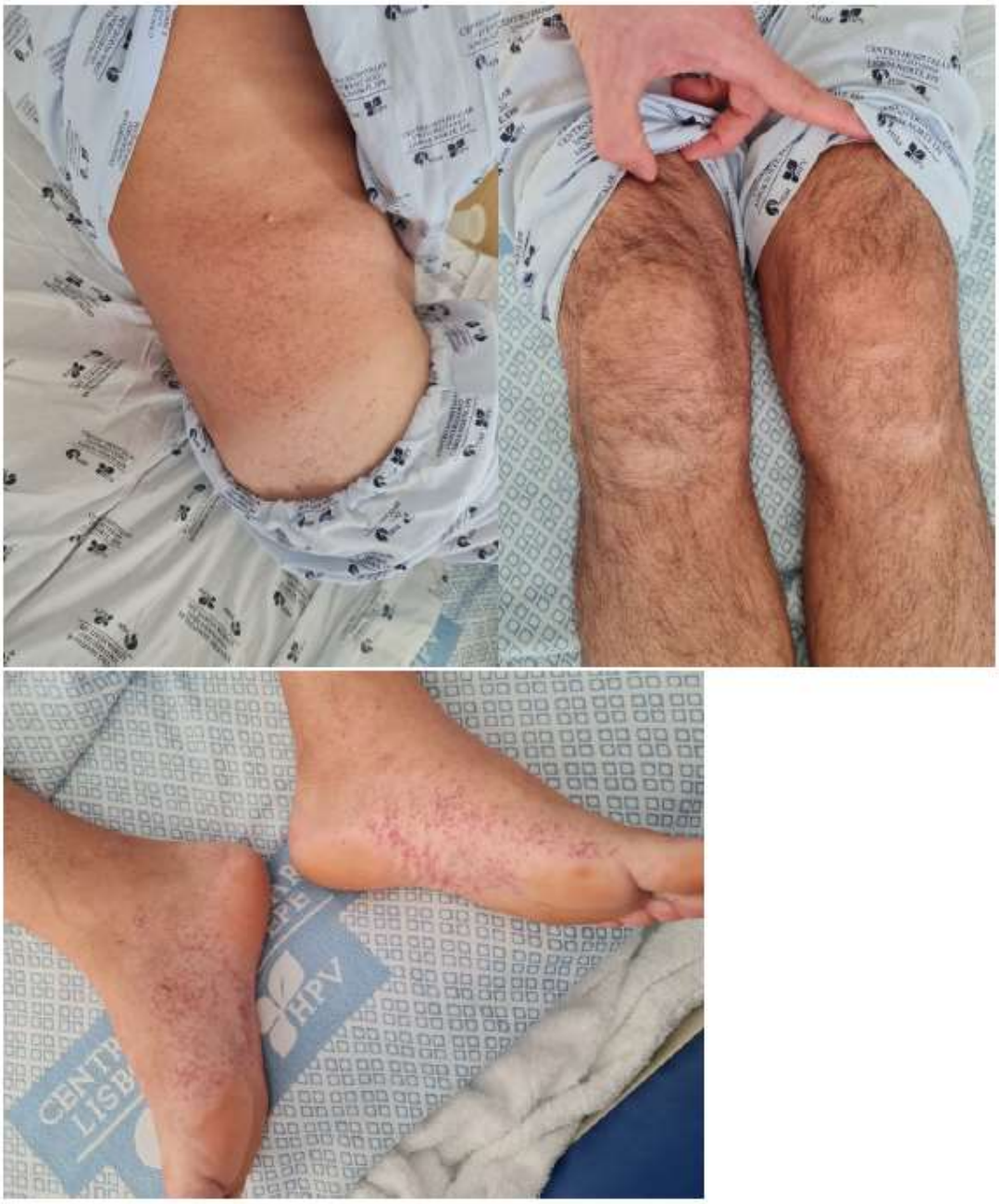
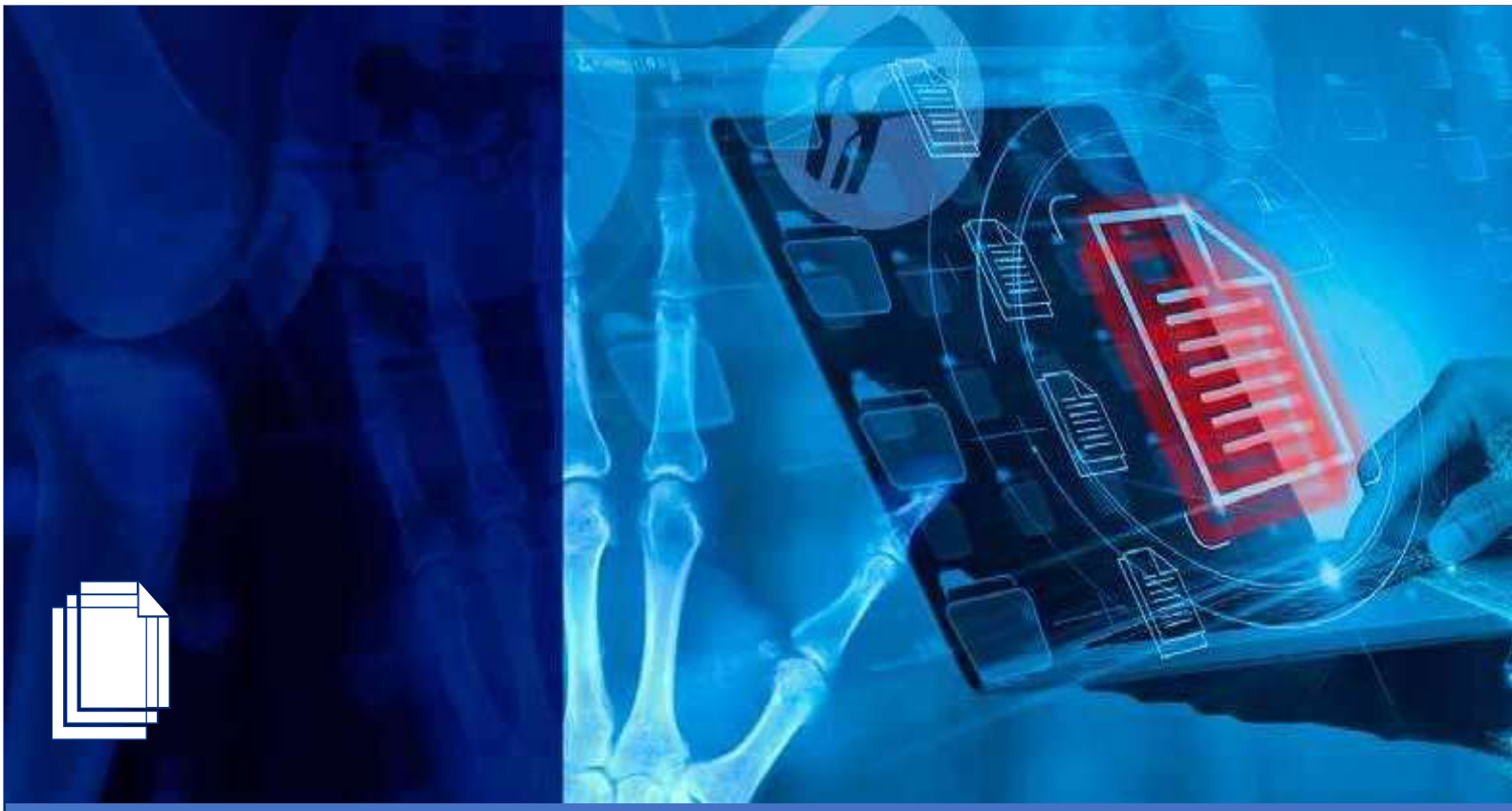
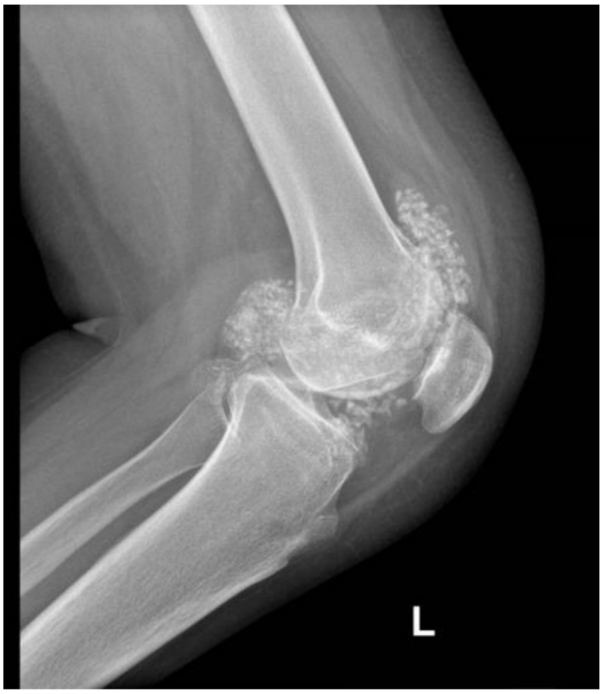




CONCURSO IMAGENS

C IM 01 - GONALGIA ATRAUMÁTICA: DIAGNÓSTICO IMAGIOLÓGICO DE CONDROMATOSE SINOVIAL

Bárbara Monteiro, Pedro Lemos, Renata Vaz, Fernando Amaral



Caso clínico: Sexo feminino, 26 anos, saudável, recorre ao serviço de urgência por gonalgia esquerda atraumática com 1 mês de evolução. Objetivamente com marcha claudicante, edema ligeiro, sem sinais inflamatórios e dor à palpação difusamente por todo o joelho. ROM diminuído ~ 0-90º, sem instabilidade ou défice aparente.

A condromatose sinovial é uma patologia rara, benigna e idiopática, mais frequente em articulações de carga, usualmente monoarticular, sendo mais frequentemente afetado a articulação do joelho, seguido pela articulação do ombro. É uma lesão cartilagínea proliferativa por metaplasia do tecido sinovial ou bursa, havendo o desenvolvimento de corpos livres que podem migrar e mineralizar para o interior da articulação.

O diagnóstico pode ser atrasado pela falta de especificidade dos sintomas, que por vezes protelam a realização de exames de imagem.

O raio x é frequentemente patognomónico e faz o diagnóstico.

Devido ao risco, apesar de baixo, descrito de transformação maligna para condrossarcoma, esta patologia deve ser tratada atempadamente. O tratamento recomendado passa pela remoção dos corpos livres, com ou sem sinovectomia complementar, de forma a prevenir evolução da destruição articular e promover a melhoria sintomática.

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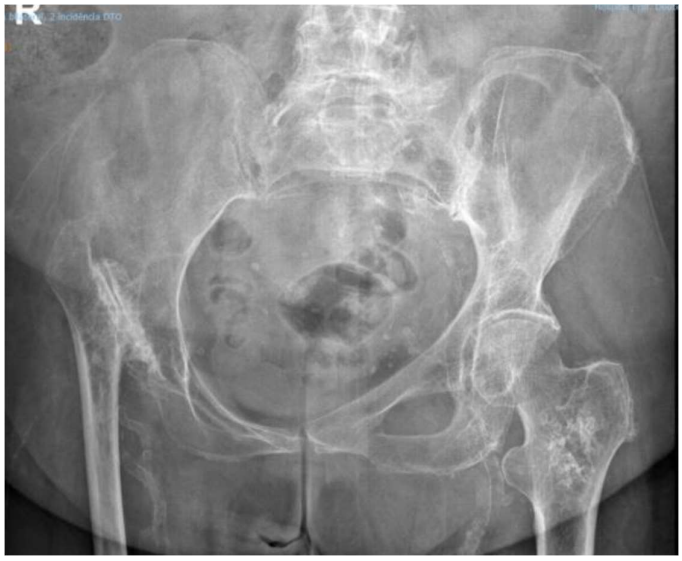
C IM 02 - SEQUELAS RARAS DE TUBERCULOSE ÓSSEA INFANTIL

Bárbara Monteiro, Pedro Lemos, Renata Vaz, Fernando Amaral



Caso clínico: Sexo feminino, 83 anos, com antecendentes de fibrilhação auricular anticoagulada, obesidade, DM, HTA, status pós-neoplasia da mama + RT há 3 anos e história prévia de tuberculose tratada na infância. Recorreu ao serviço de urgência por queda da própria altura com trauma da articulação coxo femoral direita. Refere dor cronica e limitação funcional da anca direita, que terá piorado com queda. Objetivamente limitação franca do arco de movimento da anca, com dor associada.

A tuberculose óssea na articulação da anca é o 2º tipo de tuberculose osteoarticular mais frequente (entre 10-15% dos casos de tuberculose osteoarticular). Pode ter implicações destrutivas e um grande impacto na qualidade de vida do doente.

Quando o diagnóstico é feito em estadios iniciais e o tratamento é prontamente iniciado, a maioria dos doentes consegue manter uma boa função articular (cerca de 95%). O padrão progressivo e destrutivo da anca ocorre nos doentes que não recebem o tratamento até fases mais adiantadas da doença, provocando sintomas como dor crónica, deformidade e instabilidade articular causadas pela destruição cartilagínea e óssea. O diagnóstico é frequentemente atrasado pela falta de especificidade dos sintomas e pela falta de repercussão imagiológica inicial, não sendo infrequente o aparecimento de doentes com destruição articular completa, nomeadamente em países em desenvolvimento.

Apesar dos desenvolvimentos feitos na área da terapêutica farmacológica da tuberculose, a resposta ao tratamento nem sempre é favorável, pelo que o tratamento cirúrgico deve ser realizado antes da destruição completa da articulação. Entre as opções cirúrgicas está a artrodese da anca e a artroplastia total da anca, sendo que esta última implica risco de reativação da tuberculose.

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C IM 03 - WHEN THE BAMBOO SPINE BREAKS: AN ATYPICAL CERVICAL FRACTURE IN SPONDYLOARTHRITIS

Maria Pontes Ferreira[1], Anita Cunha[1], Susana Almeida[1], Diana Barros[1], Duarte Augusto[2], José Tavares-Costa[1]

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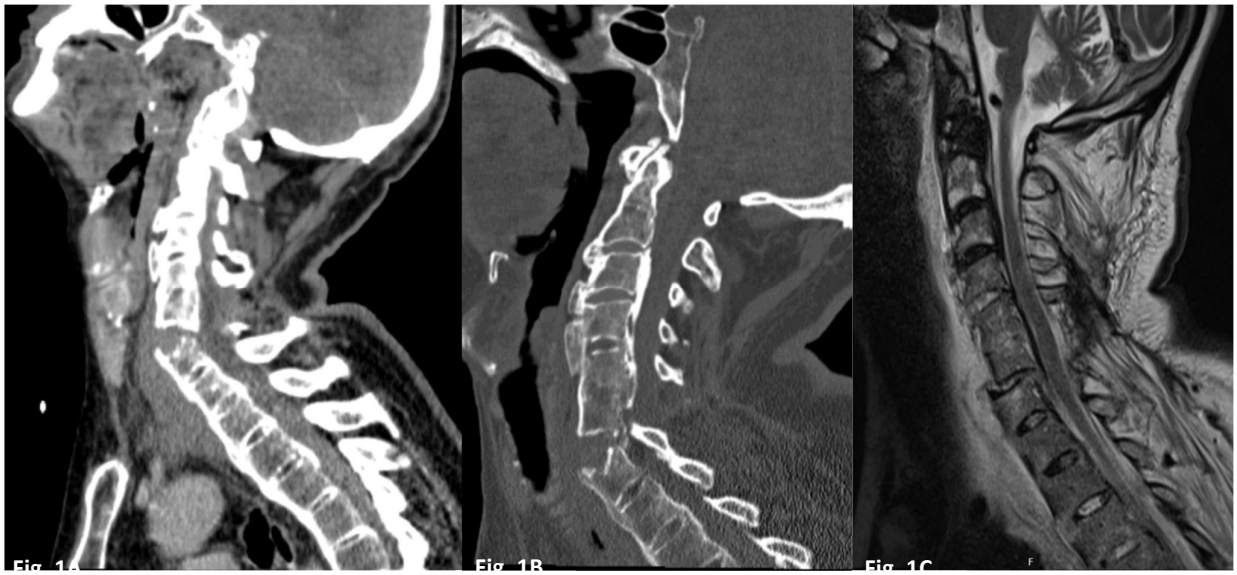


An 82-years-old man with advanced spondyloarthritis with spinal fusion suffered trauma due to tractor rollover in January 2025.

On admission, he presented severe cervical pain and oedema, and paraesthesia of both arms, without motor deficit.

Computer tomography (Fig. 1A-B) revealed C6–C7 fracture-dislocation, confirmed by magnetic resonance imaging (Fig. 1C).

The patient underwent an anterior cervical discectomy and fusion, with no neurological deficits.

C IM 04 - METASTATIC BONE DISEASE OR PAGET'S DISEASE – REVISITING THE DIFFERENTIAL DIAGNOSIS OF IVORY VERTEBRAE

Hugo Gonçalves, Paulo Pereira, Carla Campinho-Ferreira, Ana Margarida Correia, Diogo Esperança Almeida, Ana Roxo Ribeiro

Introduction: Paget's bone disease is a metabolic disorder characterized by abnormal bone remodeling often involving both lytic and sclerotic phases simultaneously. In the spine, it may lead to vertebral expansion, cortical thickening and coarse trabeculation, resulting in the classic late *ivory vertebra* appearance.

Clinical Case: A 68 years old woman presented with chronic lower back pain, prompting imaging evaluation of the lumbar spine. Lumbar CT and MRI demonstrated L4 vertebral body with low signal on T1 and T2/STIR, coarse vertical trabeculation, mild vertebral expansion, diffuse cortical thickening. This constellation of findings was suggestive of an *ivory vertebra*, and a Rheumatology consultation was requested for further evaluation with the initial suspicion of Paget's bone disease.

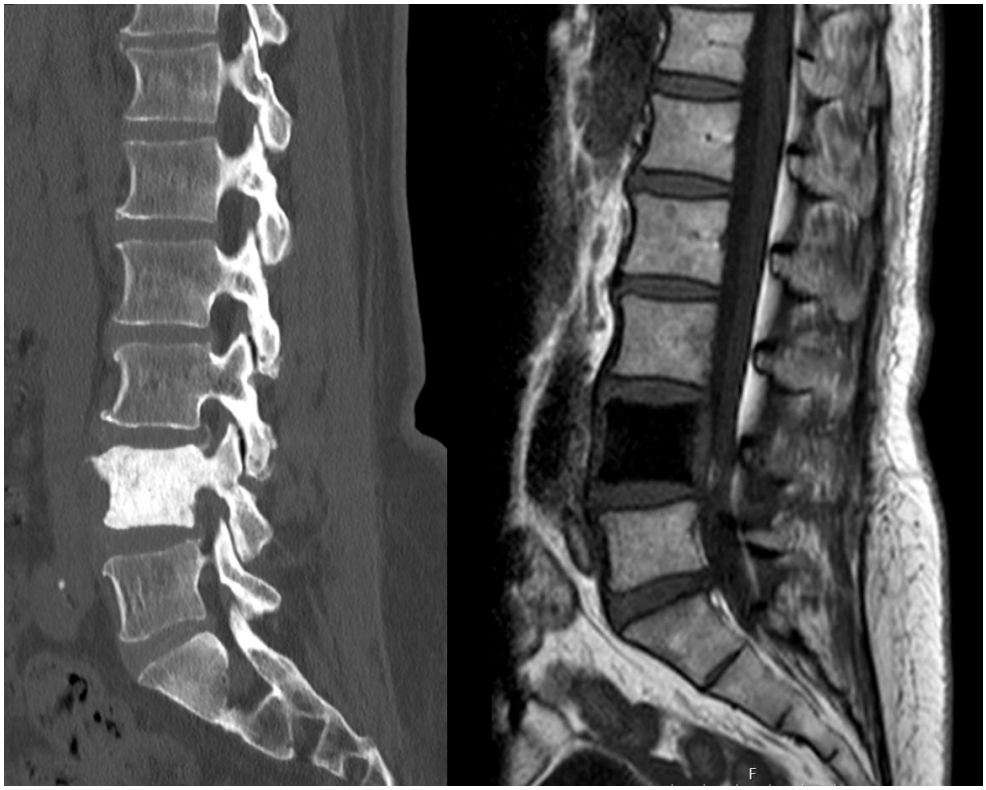


Figure 1 – (A) Sagittal CT showing L4 vertebral body with diffuse sclerosis, cortical thickening and coarse vertical trabeculation, with the typical *ivory vertebra* appearance. **(B)** Saggital T1-weighed MRI showing low signal intensity of L4, with cortical thickening producing the typical *picture frame* appearance, corresponding to the sclerotic signs observed on CT.

Prior to the Rheumatology consultation, the patient was hospitalized for evaluation of recent developed constitutional symptoms and pancytopenia. During this admission, thoraco-abdomino-pelvic CT revealed extensive hepatic and skeletal lesions, a large nodular hepatic mass and multiple sclerotic bone metastases and a breast nodule was identified and biopsied with histopathology confirming luminal B, HER-2 negative breast adenocarcinoma.

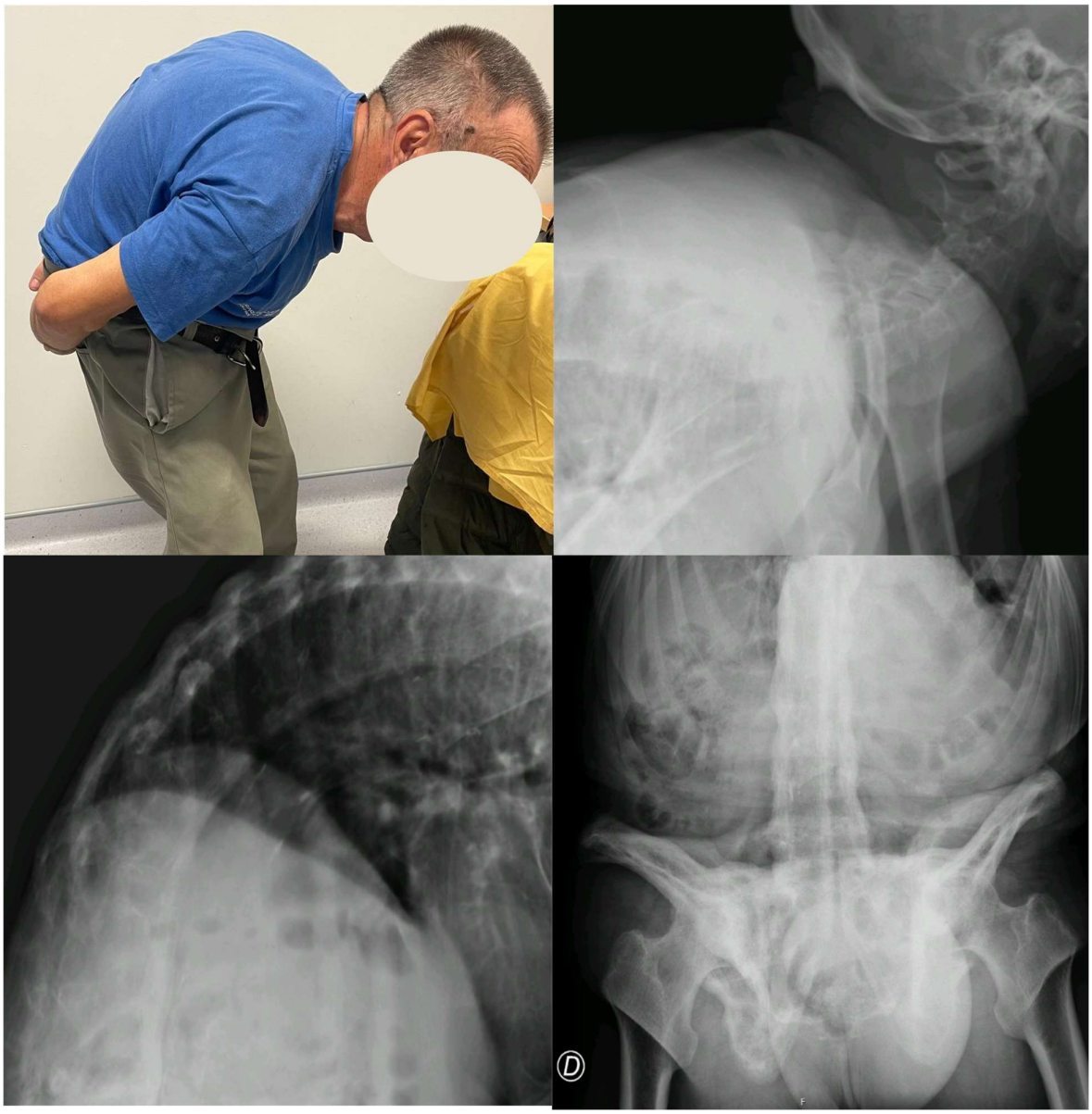
Given these findings, the initial suspicion of Paget's disease was reconsidered, and a metastatic process was considered the more likely diagnosis.

Conclusion: Although the vertebral lesion initially suggested Paget's disease, the systemic findings confirmed diffuse metastatic breast cancer as the underlying cause. This case highlights the importance of considering differential diagnoses in ivory vertebrae, which may include osteoblastic bone metastases, chronic osteomyelitis and SAPHO syndrome

C IM 05 - O PESO DO TEMPO NA COLUNA - UM CASO GRAVE DE ESPONDILITE ANQUILOSANTE

Catarina Rua[1], Tiago Beirão[1], Catarina Silva[1], Mariana Patela[1], Tiago Meirinhos[1] ,
Flávio Costa[1] Diogo Fonseca[2]

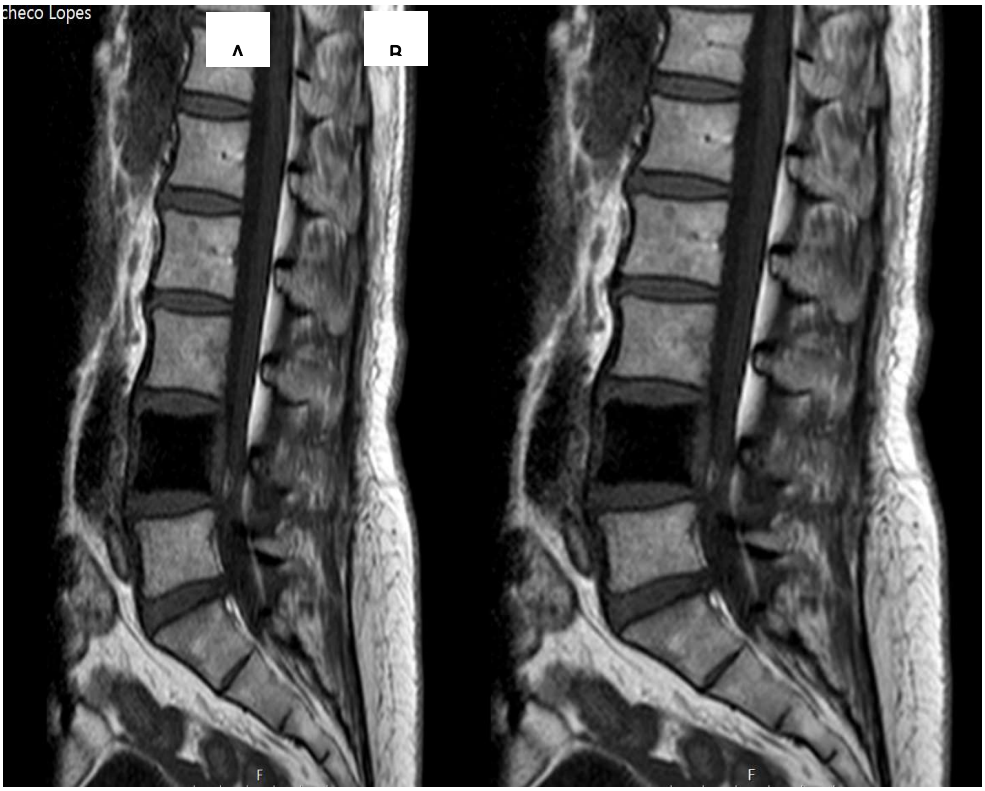
1. Serviço de Reumatologia, ULS Gaia e Espinho
2. Reumatologista, Hospital da Boa Nova



Homem, 79 anos, com espondilite anquilosante de 51 anos sem seguimento. Ao exame objetivo: hipercifose dorso-lombar grave (~90°), flexão cervical e impingement costelas–bacia. A Radiografia coluna com Triple Track Sign; bacia com anquilose das sacroilíacas. Atualmente medicado com AINE on-demand e sob cinesioterapia respiratória, sem indicação para correção cirúrgica.

C IM 06 - METASTATIC BONE DISEASE OR PAGET'S DISEASE – REVISITING THE DIFFERENTIAL DIAGNOSIS OF IVORY VERTEBRAE

Hugo Gonçalves, Paulo Pereira, Carla Campinho-Ferreira, Ana Margarida Correia, Diogo Esperança Almeida, Emanuel Costa, Ana Roxo Ribeiro



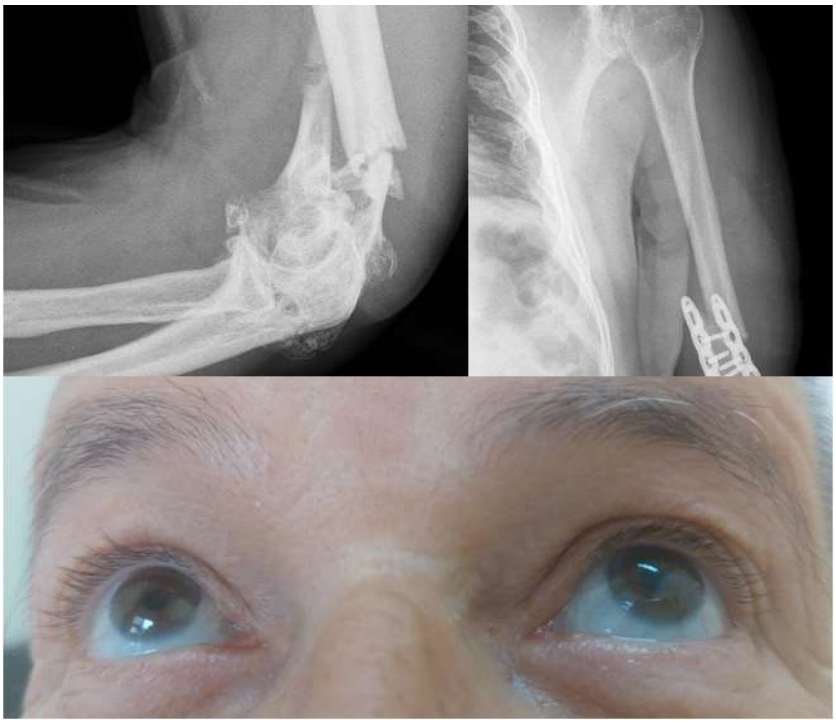
Sagittal lumbar CT **(A)** and T1-weighed MRI **(B)** showing diffuse sclerosis, cortical thickening and coarse vertical trabeculation in L4 vertebra (*ivory vertebra*) MRI shows low signal intensity with cortical thickening (*picture frame*). The patient was initially referred for suspected Paget's disease; later confirmed blastic metastasis from luminal HER2-negative breast carcinoma.

C IM 07 - FRATURAS EM SÉRIE E UM OLHAR CARACTERÍSTICO – UM CASO DE OSTEOGÉNESE IMPERFEITA

Guilherme Santos Luís, Luísa Brites, Alexandra Daniel

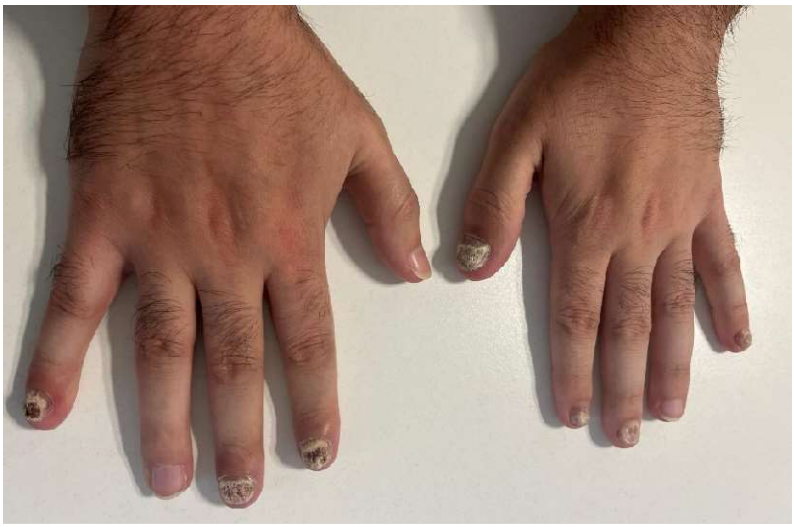
Legenda da imagem (caso clínico):

Homem de 62 anos com múltiplas fraturas de fragilidade: fratura supracondiliana do úmero esquerdo, da diáfise umeral esquerda e de L2. Ao exame objetivo, a destacar baixa estatura e escleróticas azuladas. Estudo fosfocálcico normal. T score lombar total -2.1 em DEXA. Referenciado a consulta de genética, sendo identificada a variante familiar do COL1A1, que confirma osteogénese imperfeita tipo I.



C IM 08 - REFRACTORY NAIL DISEASE IN PSORIATIC ARTHRITIS

Hugo Gonçalves, Paulo Pereira, Carla Campinho-Ferreira, Ana Margarida Correia, Diogo Esperança Almeida, Emanuel Costa, Ana Roxo Ribeiro



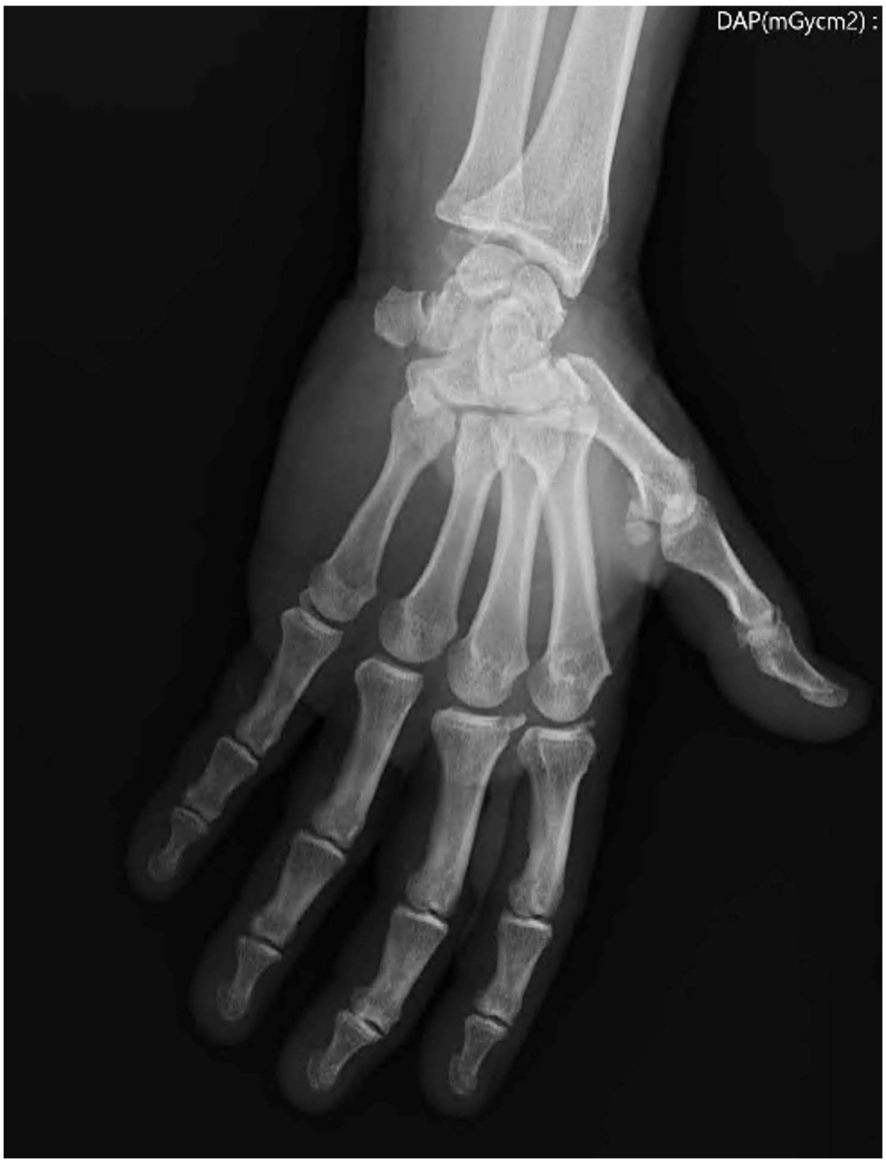
Severe nail dystrophy in a patient with psoriatic arthritis, refractory to steroids local nail injection. Nail lesions persisted despite arthritis remission with methotrexate and tofacitinib (previous failure with Adalimumab, Golimumab, Secukinumab and Brodalumab). This highlights the extremely refractory nature of psoriatic nail involvement.

C IM 09 - DOENÇA POR DEPOSIÇÃO DE PIROFOSFATO DE CÁLCIO COM ENVOLVIMENTO METACARPOFALÂNGICO

Tremoceiro J, Rodrigues SD, Moniz AC, Emília M, Silva I

Serviço de Reumatologia, Unidade Local de Saúde de Lisboa Ocidental

Homem de 76 anos com doença por deposição de pirofosfato de cálcio (DPPC), com condrocalcinose da fibrocartilagem do carpo e das cartilagens hialinas das metacarpofalângicas do 2.º e 3.º dedos da mão esquerda, apresentando osteófitos característicos em gancho na vertente radial. Apesar de mais frequente em joelhos e punhos, a DPPC avançada pode envolver as metacarpofalângicas.

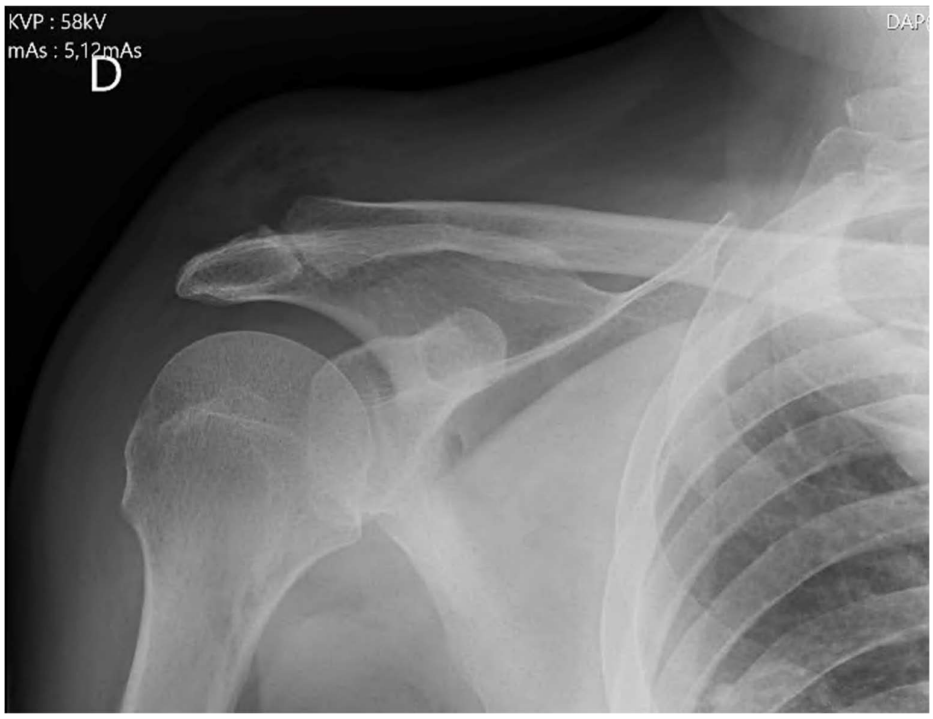


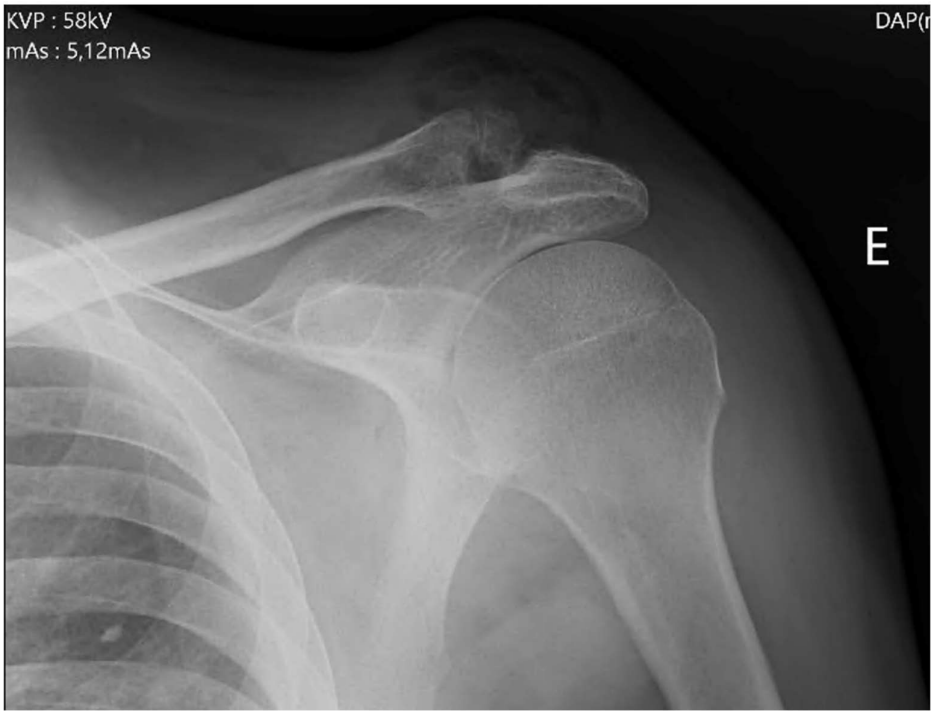
C IM 10 - ENVOLVIMENTO EXTENSO DOS OMBROS POR TOFOS GOTOSOS

Tremoceiro J, Rodrigues SD, Moniz AC, Emília M, Silva I

Serviço de Reumatologia, Unidade Local de Saúde de Lisboa Ocidental

Homem de 63 anos com história prolongada de gota não tratada. Apresentava nódulos duros e irregulares na face anterossuperior de ambos os ombros. Radiografia com agregados sobre a articulação acromioclavicular e erosão em sacabocados na clavícula esquerda. Ecografia confirma agregados circunscritos, heterogéneos e hiperecogénicos intra-articulares, sem sombra acústica, rodeados por orla anecoica.

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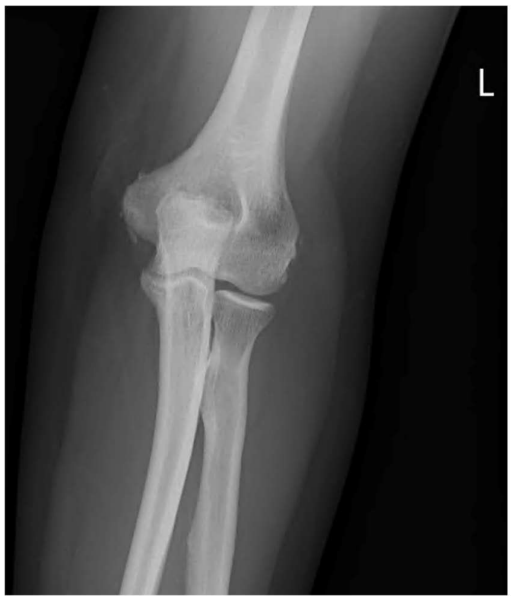
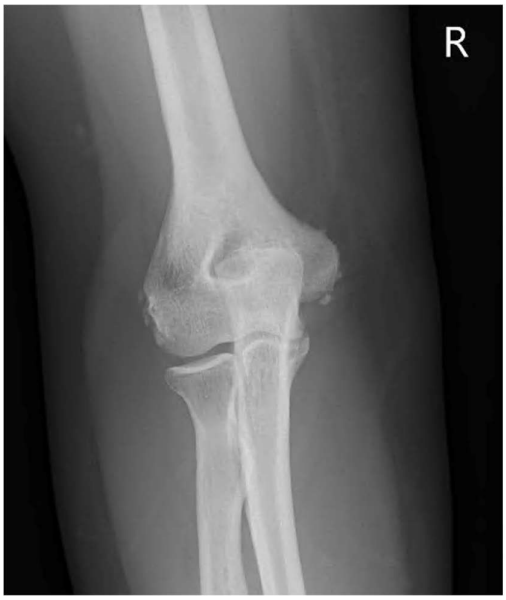
C IM 11 - CALCIFICAÇÕES METASTÁTICAS NA DOENÇA RENAL CRÓNICA

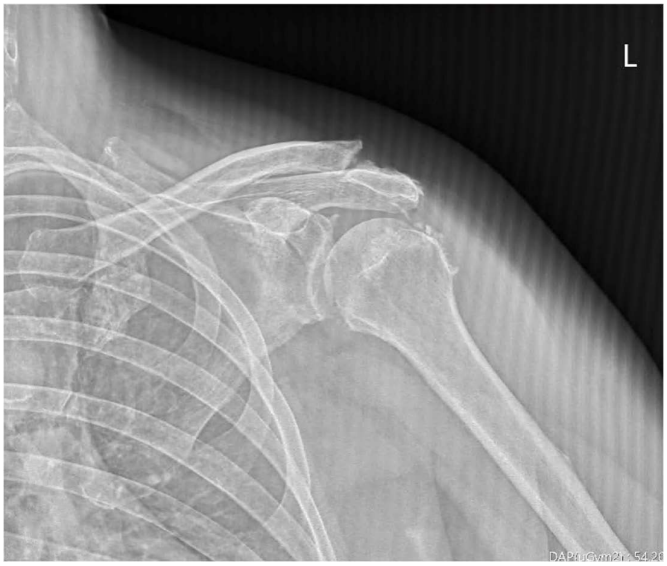
Tremoceiro J, Rodrigues SD, Moniz AC, Emília M, Silva I

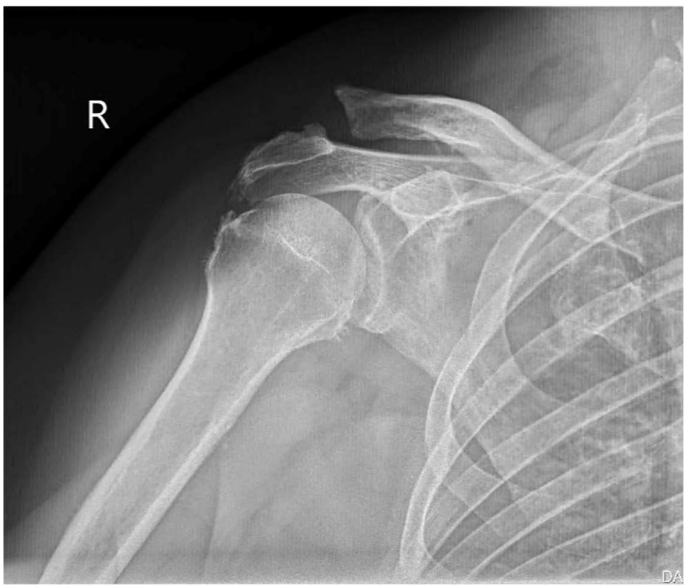
Serviço de Reumatologia, Unidade Local de Saúde de Lisboa Ocidental

Homem de 50 anos, com doença renal crónica estadio 5 e quadro de poliartralgias de ritmo inflamatório. As radiografias evidenciam depósitos radiopacos periarticulares nas enteses da coifa dos rotadores e cápsula articular, enteses dos tendões flexores e extensores comuns dos dedos, e ligamentos colaterais das interfalângicas proximais e das suas cápsulas articulares.





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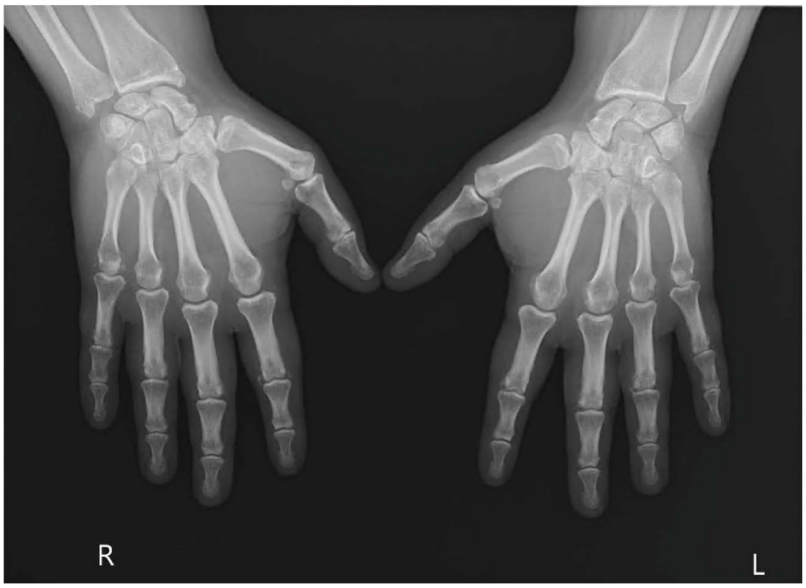
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C IM 12 - A RARE CUTANEOUS TWIST IN RHEUMATOID ARTHRITIS PALISADED NEUTROPHILIC DERMATOSIS UNVEILED

Rodrigo Rei1,2, José Ramos3, Joana Nogueira4, Tomás Stein Novais1, Ana Catarina Duarte1, Pedro Gonçalves1, Maria José Santos1,5

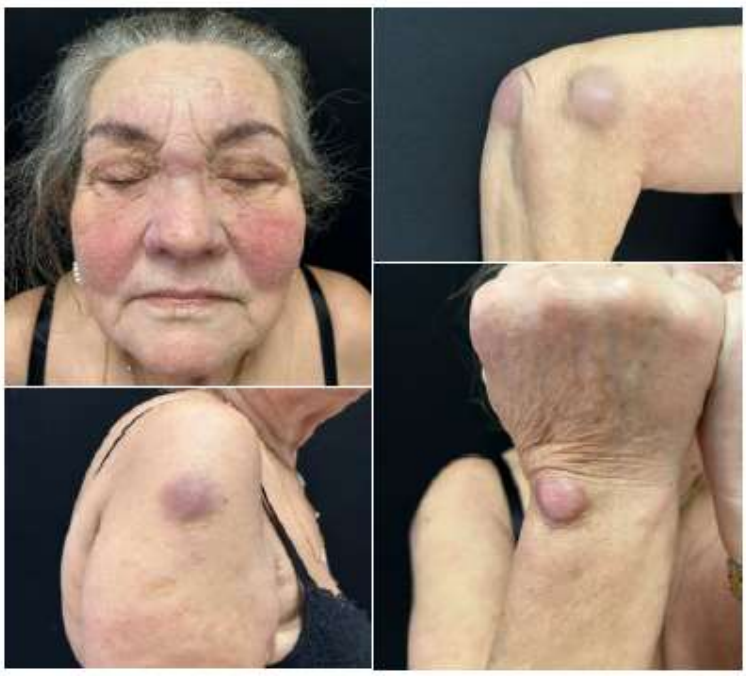
1 – Serviço de Reumatologia, Hospital Garcia de Orta

2 – Serviço de Reumatologia, Hospital de Faro

3 – Serviço de Dermatologia, Hospital Garcia de Orta

4 – Serviço de Anatomia Patológica, Hospital Garcia de Orta

5 – Faculdade de Medicina da Universidade de Lisboa



A 71-year-old woman developed violaceous nodules on the right wrist, elbow, shoulder, and glabellar region. Four months later, she presented with polyarthritis and was diagnosed with seropositive rheumatoid arthritis (RA). Skin biopsy showed features of palisaded neutrophilic and granulomatous dermatitis, a rare dermatosis associated with autoimmune diseases, particularly RA.

C IM 13 - BONE SARCOIDOSIS UNCOMMON IMAGING PRESENTATION

Susana Almeida1, Anita Cunha1, Diana Barros1, Maria Pontes Ferreira1, Duarte Augusto2, José Tavares-Costa1, Ana Rita Gigante3, Filipa Teixeira1

1 Rheumatology department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal

2 Rheumatology department, Unidade Local de Saúde da Guarda, Guarda, Portugal

3 Pneumology department, Unidade Local de Saúde do Alto Minho, Viana do Castelo, Portugal

A 47-year-old male with ganglionar and pulmonary sarcoidosis, without musculoskeletal symptoms, was referred to Rheumatology due to imaging abnormalities. PET revealed multiple areas of uptake, including bone (Fig.1), and MRI showed sacral, iliac and peritrochanteric lesions (Fig. 2). The imaging findings, in the context of known sarcoidosis, supported the diagnosis of osseous sarcoidosis.

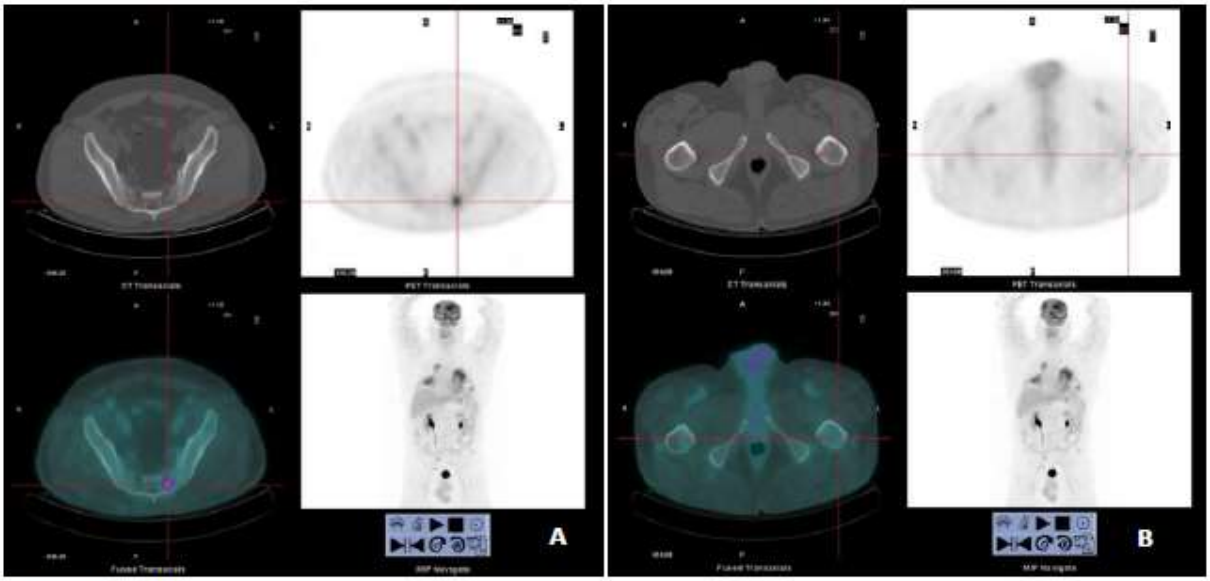


Fig.1 (A and B)

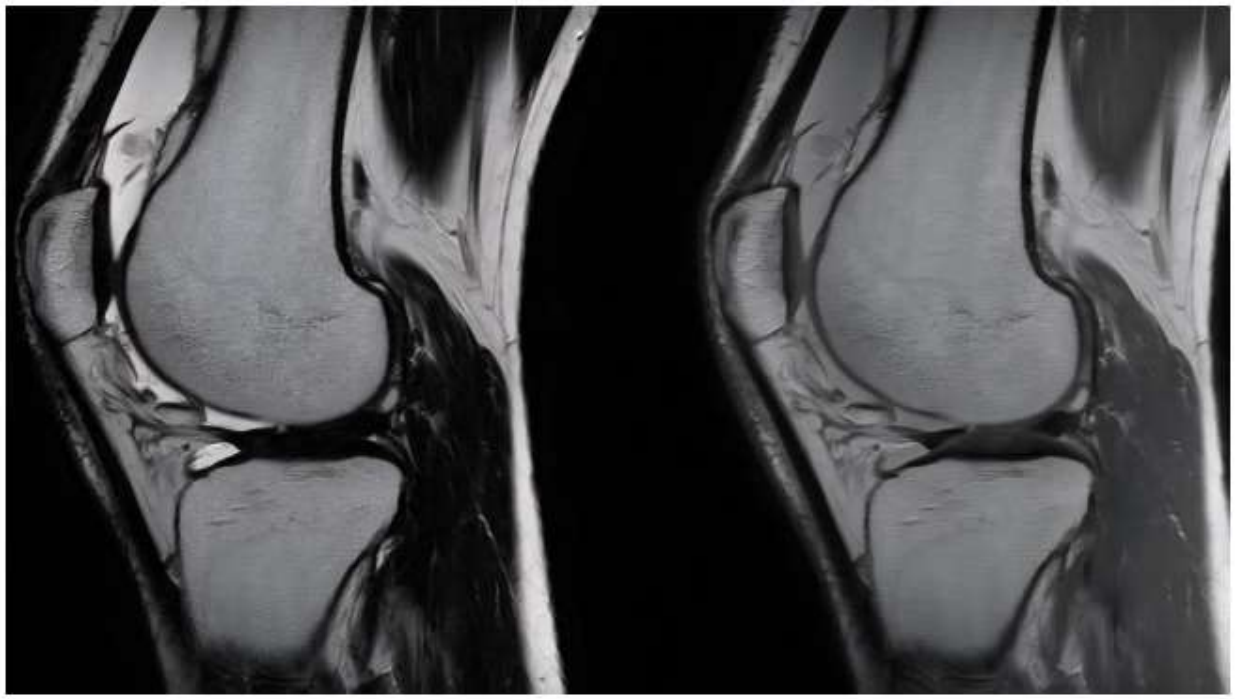
C IM 14 - LIPOMA ARBORESCENS IN SPONDYLOARTHRITIS

João Aguiar 1, 2, Joaquim Pereira1, 2

1. Serviço de Reumatologia, Unidade Local de Saúde de Santa Maria (ULSSM), Centro Académico de Medicina de Lisboa.

2. Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa.

A 25-year-old patient with peripheral spondyloarthritis persisted with knee monoarthritis despite optimized methotrexate and glucocorticoid injections. MRI revealed moderate joint effusion with mild diffuse synovial thickening and multiple small finger-like projections, suggestive of lipoma arborescens.

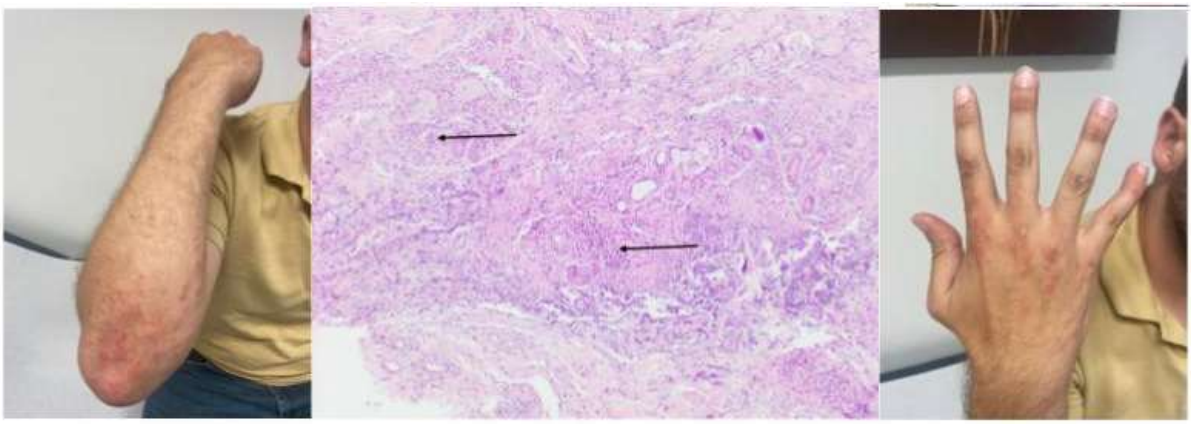


C IM 15 - ARTRITE REUMATÓIDE: INFLAMAÇÃO À FLOR DA PELE

Patela M, Beirão T1, S, Rua C1 , Silva C1, Ramos AC2, Meirinhos T1, Pinto AS1

1. Serviço de Reumatologia da Unidade Local de Saúde Gaia/Espinho

2 Serviço de Anatomia Patológica da Unidade Local de Saúde Gaia/Espinho



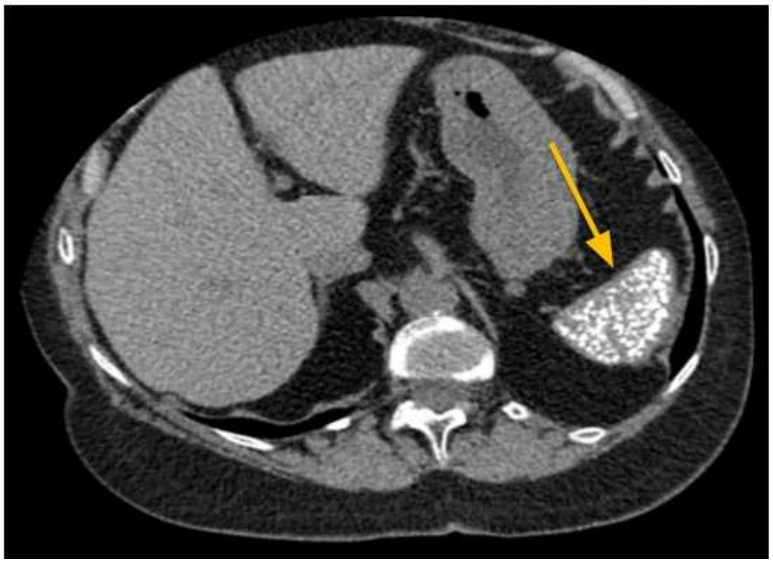
Homem de 38 anos, com artrite reumatoide FR e anti-CCP positivos, apresenta placas eritematosas, dolorosas e mal delimitadas na superfície extensora do cotovelo e na face dorsal das articulações metacarpofalângicas. O estudo histológico mostra infiltrado dérmico difuso rico em neutrófilos, com leucocitoclasia marcada, compatível com dermatite neutrofílica.

C IM 16 - CALCIFICAÇÕES ESPLÉNICAS NO LÚPUS UM ACHADO RARO E ENIGMÁTICO

Patela M, Beirão T1, S, Rua C1 , Silva C1, Meirinhos T1, Pinto AS1, Vieira R1

1. Unidade Local de Saúde Gaia/Espinho

Mulher de 62 anos com lúpus eritematoso sistémico com envolvimento articular, cutâneo e nefrite classe IV, tratada inicialmente com ciclofosfamida (esquema Eurolupus) e atualmente com micofenolato de mofetil (2 g/dia). Tomografia abdominal mostrou calcificações difusas no baço, atribuídas ao lúpus após exclusão de outras causas. Achado raro, com poucos casos descritos e significado clínico ainda incerto.

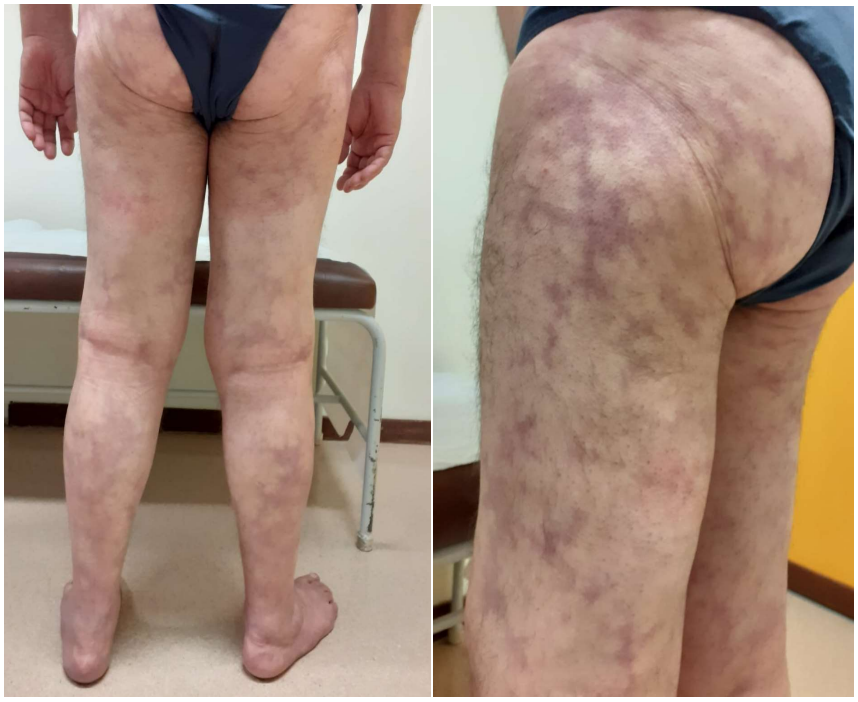


C IM 17 - LIVEDO RACEMOSA: A REDE VISÍVEL DA SÍNDROME ANTIFOSFOLIPÍDICA

Filipa Canhão André, Beatriz Mendes, Fabiana Gouveia, João Oliveira, André Saraiva, Margarida Coutinho

Legenda:

Homem de 60 anos, com poliartrite crónica simétrica e aditiva com 10 anos de evolução. Aparecimento de **livedo racemosa** e úlceras espontâneas dos membros inferiores 1 ano depois. O estudo laboratorial revelou inibidor tipo lúpus persistentemente positivo (restante estudo de autoimunidade negativo). A biópsia cutânea revelou vasculopatia oclusiva, sugestiva de **SAF com envolvimento microvascular**.



C IM 19 - DERMATOFIBROMAS MÚLTIPLOS APÓS ADALIMUMAB: UMA MANIFESTAÇÃO CUTÂNEA RARA

Catarina Rua1, Matilde Monteiro2, Tiago Beirão1, Catarina Silva1, Mariana Patela1, Tiago Meirinhos1, Catarina Queirós2 e Flávio Costa1

1. Serviço de Reumatologia, ULS Gaia e Espinho

2. Serviço de Dermatologia, ULS Gaia e Espinho

Homem de 49 anos, seguido em Reumatologia por espondiloartrite axial, que desenvolveu múltiplas lesões papulares firmes e pruriginosas nos membros superiores e inferiores (setas vermelhas), uma semana após o início de adalimumab 40 mg SC de 15/15 dias. A biópsia excisional e a análise histológica confirmaram a suspeita de dermatofibromas. Dada a sua benignidade, não está recomendado o seu tratamento cirúrgico.

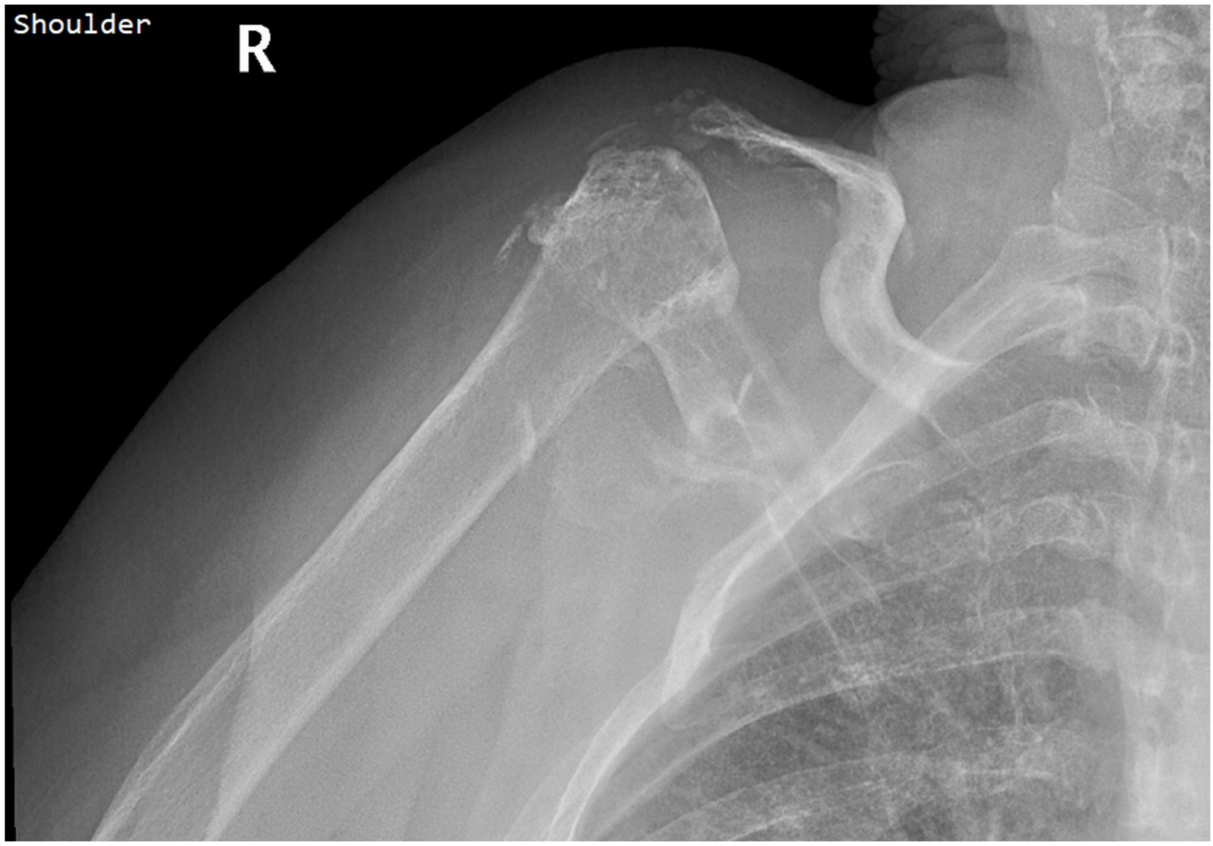
C IM 20 - RADIOGRAPHIC FINDINGS IN SEVERE EROSIVE AND DEFORMING RHEUMATOID ARTHRITIS

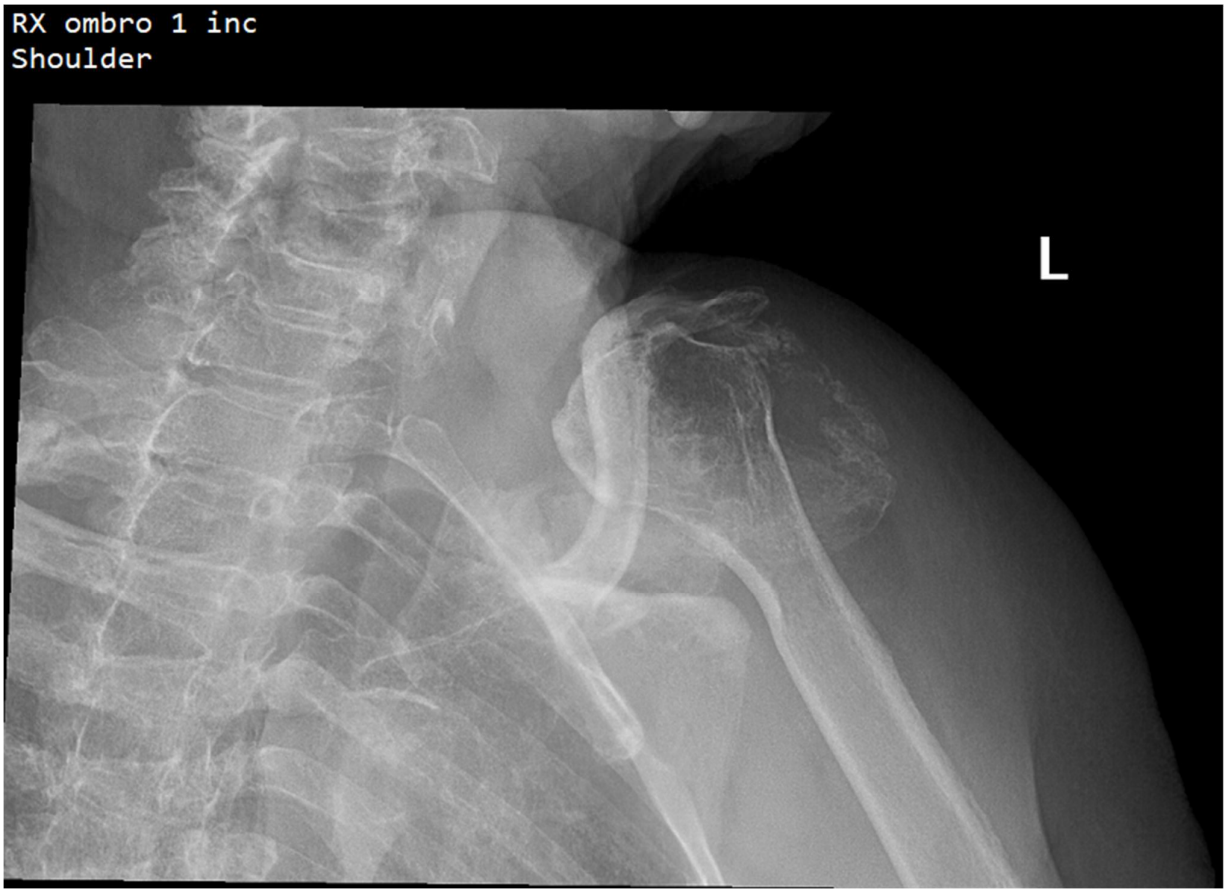
Ana Bispo Leão1, Leonor Reynolds1, Rita Silva-Vieira1, Beatriz de Carvalho Mendonça1, Bárbara Lobão1, Beatriz Santos1, Manuela Parente1, Helena Santos1, 2

1Rheumatology Department, Instituto Português de Reumatologia, Lisboa, Portugal

2Comprehensive Health Research Center (CHRC), NOVA Medical School, University of Lisbon, Lisboa, Portugal

We present the radiographic shoulder evaluation of an 80-year-old female with long-lasting, erosive Rheumatoid Arthritis. Bilateral X-ray shows complete loss of glenohumeral joint architecture, diffuse calcifications, erosions, destruction and subluxation of the humeral head (due to rotator cuff dysfunction and joint deformity), consistent with a concomitant diagnosis of Milwaukee shoulder.





Rheumatoid arthritis (RA) is a systemic autoimmune disorder characterized by persistent synovial inflammation, leading to progressive joint destruction. In severe cases, RA manifests as an erosive and deforming disease, resulting in significant functional impairment and reduced quality of life. Radiographic imaging remains a cornerstone in the assessment of joint damage and its progression. Here, we present a radiographic image of the shoulders of a 80 years old, female patient, with a past medical history notable for a long-lasting, erosive and deformant RA. Concurrently, the patient also had multiple chronic tendinopathies of shoulders, with complete rupture of the long head of the biceps tendon, subscapularis, and the infraspinatus, bilaterally. On previously performed ultrasound, was also evident cortical changes of the humeral head and degenerative changes of the acromioclavicular joint, consistent with a 'Milwaukee shoulder". In this context, the patient was previously referred to Orthopaedic Surgery, with no indication for surgery.

Currently, due to worsening, painful, shoulder disability, a radiographic reevaluation of the shoulders was performed. We therefore present the X-ray result, which strikingly demonstrates complete loss of glenohumeral joint architecture, marked erosion and destruction of the humeral head, and its subluxation due to rotator cuff dysfunction and joint deformity.

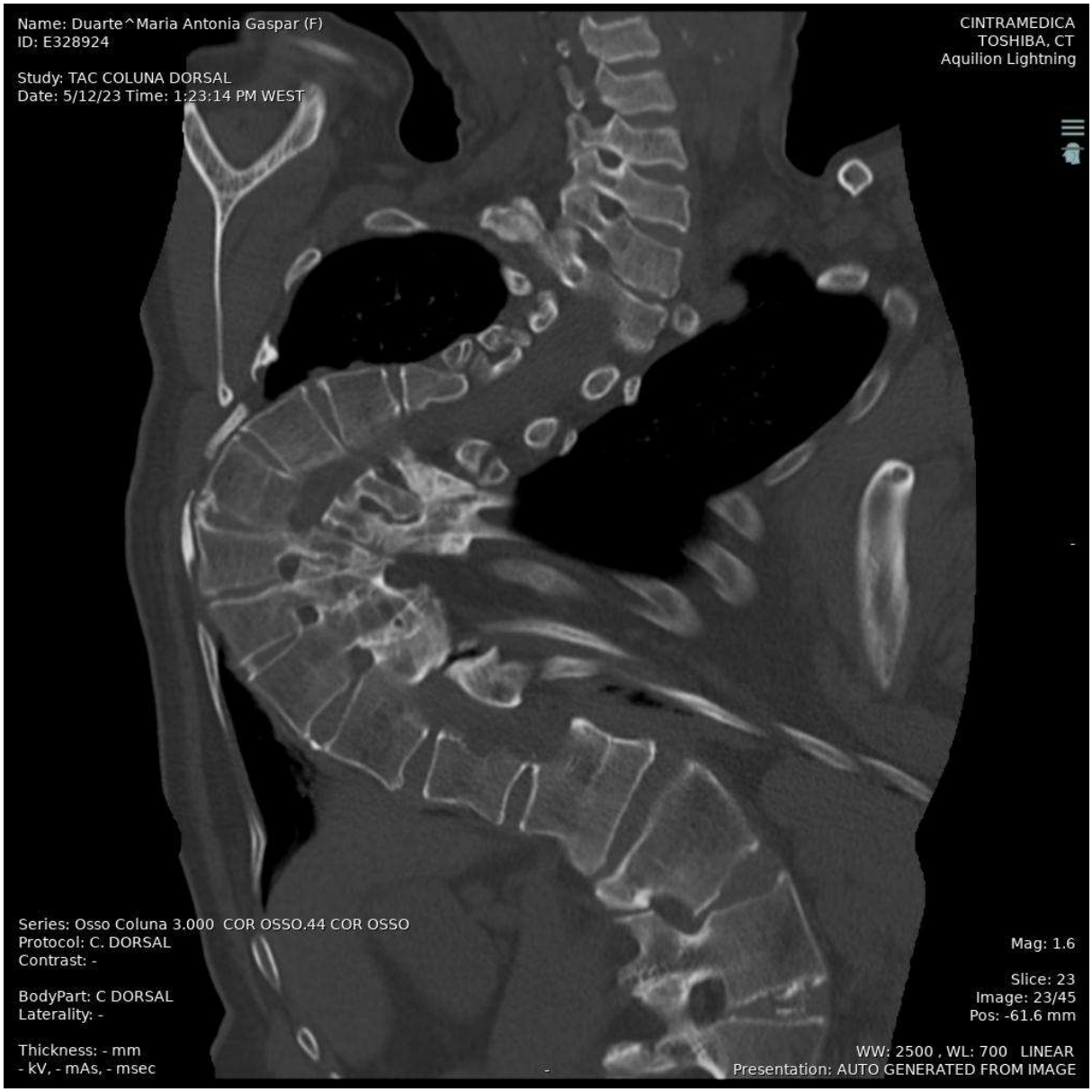
C IM 21 - THREE-DIMENSIONAL IMAGING OF SEVERE THORACIC AND LUMBAR SCOLIOSIS

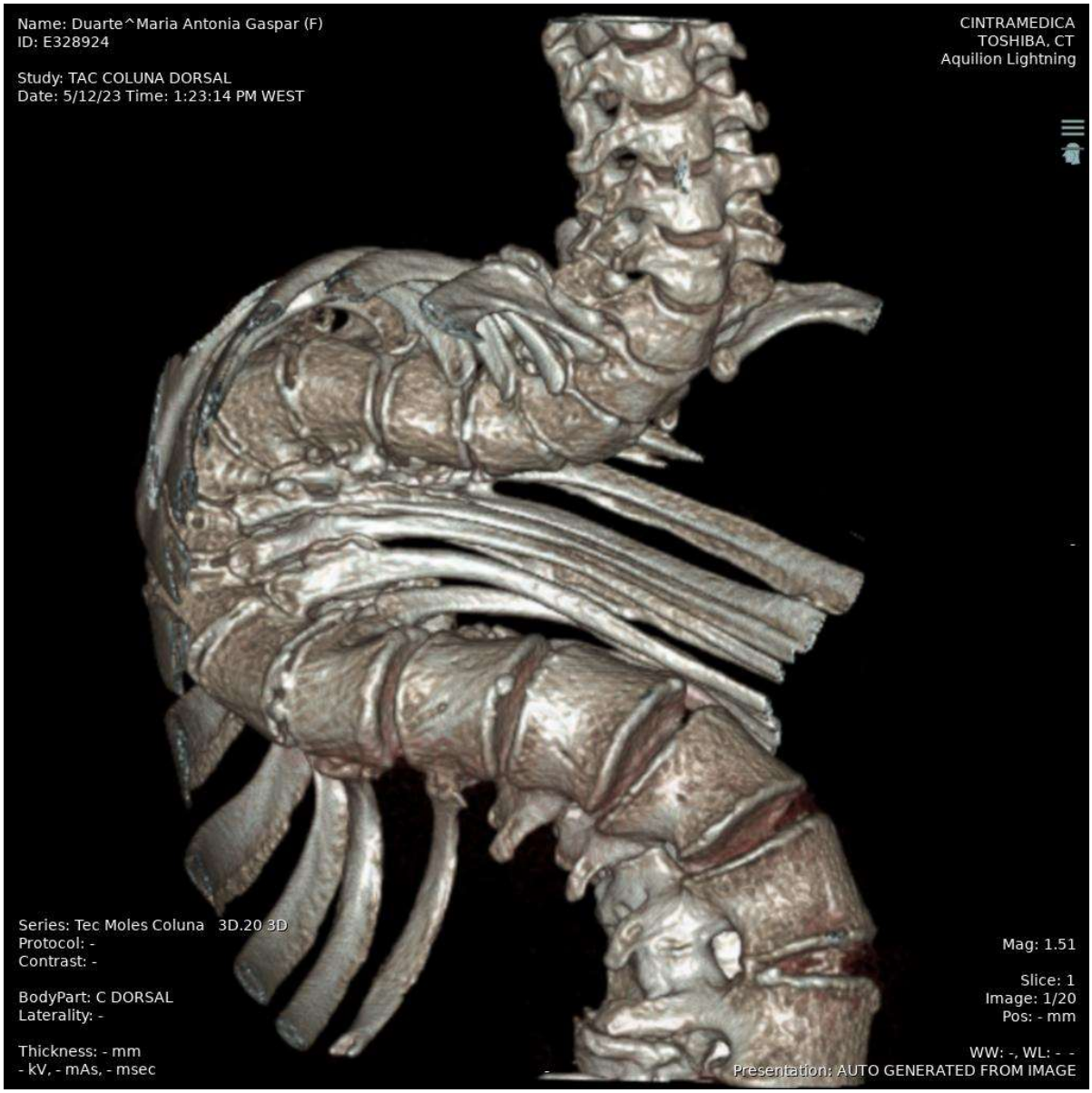
Ana Bispo Leão1, Susana Fernandes1, Helena Santos1, 2

1Rheumatology Department, Instituto Português de Reumatologia, Lisboa, Portugal

2Comprehensive Health Research Center (CHRC), NOVA Medical School, University of Lisbon, Lisboa, Portugal

We present a computed tomography scan with three-dimensional reconstruction of the thoracolumbar spine of a 69-year-old female patient. The imaging reveals severe right-convex thoracic scoliosis and left-convex lumbar scoliosis, findings that correlate clinically with respiratory insufficiency (restrictive-pattern) and chronic pain.

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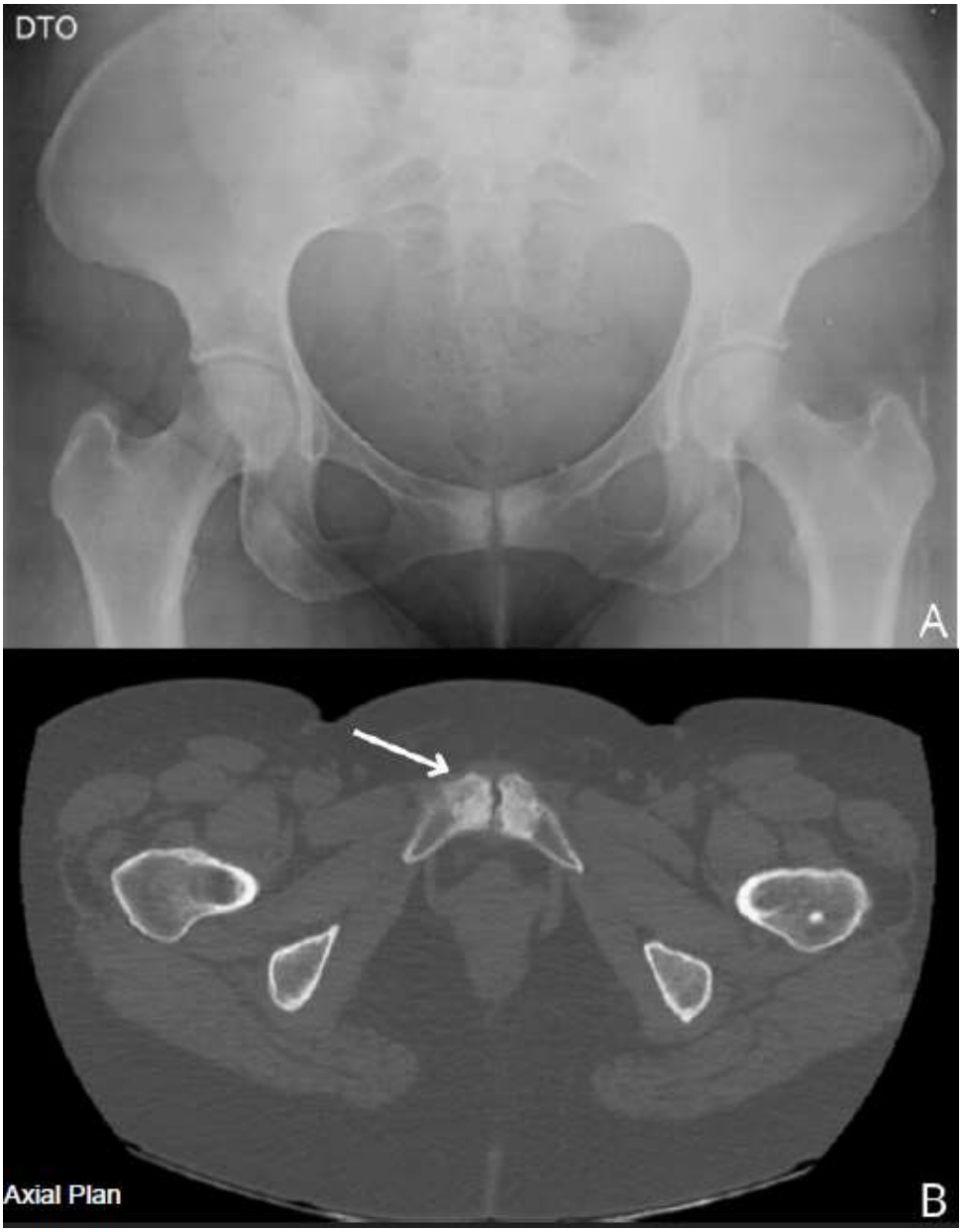
C IM 22 - WHEN AXIAL SPONDYLOARTHRITIS HITS BELOW THE BELT

Sara Alves Costa1, Filipa Canhão André1, Mariana Rodrigues1, Fabiana Gouveia1, Maria João Cadório1, João Oliveira1, Marcelo Neto1, Fernando Albuquerque1, Sara Serra1

1. Rheumatology Department, Local Health Unit of Coimbra



Female patient, 48 years, with a newly diagnosed axial spondyloarthritis, presenting with grade IV sacroiliitis and pubic symphysitis, characterised by marked sclerosis, irregularity, and erosive changes of the joint visualised on the radiograph (A).

Computed tomography revealed sclerosis of the pubic symphysis with ground-glass densification of the proximal segments and irregularity of the contour (B, white arrow).

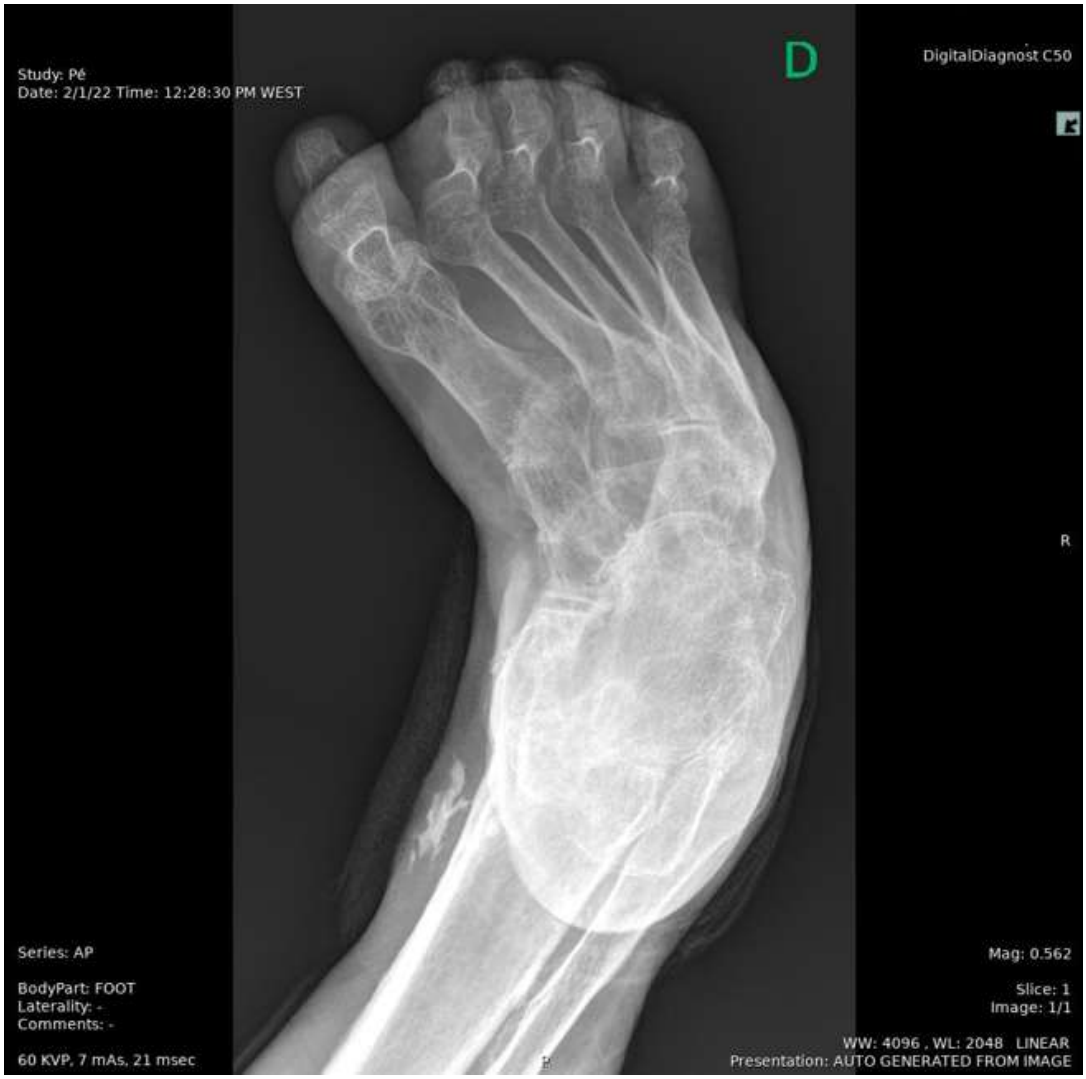
C IM 23 - IMAGING FEATURES OF AN OLD SNAKEBITE

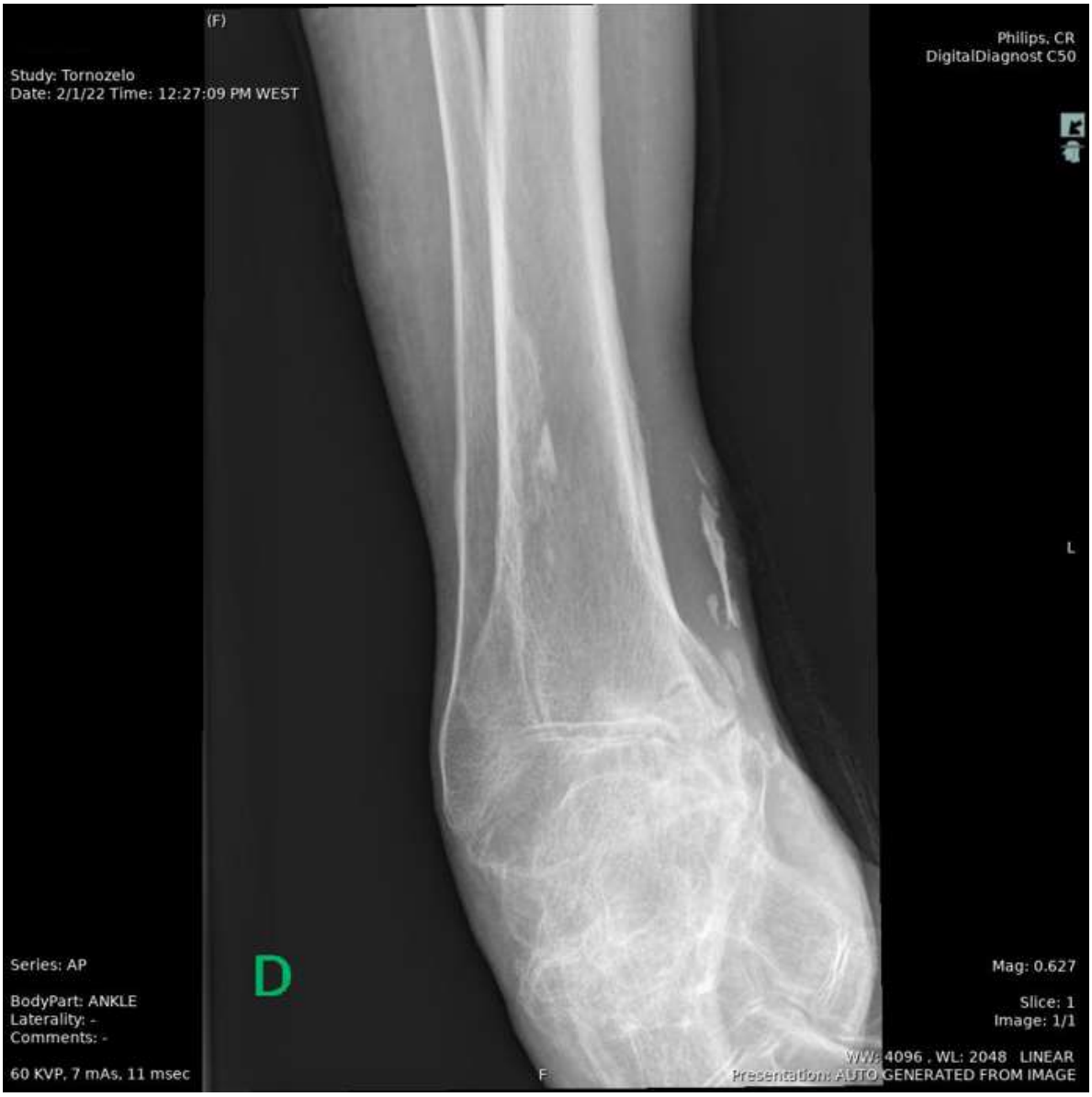
Ana Bispo Leão[1], **Susana Fernandes**[1], **Helena Santos**[1,2]

1Rheumatology Department, Instituto Português de Reumatologia, Lisboa, Portugal

2Comprehensive Health Research Center (CHRC), NOVA Medical School, University of Lisbon, Lisboa, Portugal

We present a radiographic image of the right ankle and foot of a 75-year-old patient who suffered a snakebite at the age of 12. The x-ray image shows articular deformities with Volkmann contracture of the foot, with ankle inversion, pes cavus and claw fingers. There is also evident an area of dystrophic calcification secondary to local soft tissue necrosis.

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Mag: 0.627
BodyPart: ANKLE
Laterality: -
Comments: -
Slice: 1
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60 KVP, 7 mAs, 11 msec
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WW: 4096 , WL: 2048 LINEAR
Presentation: AUTO GENERATED FROM IMAGE

C IM 24 - HIPERPLASIA DE CORPÚSCULOS DE PACINI E DOENÇA MICROGEÓDICA DAS FALANGES: CONDIÇÕES INCOMUNS DAS MÃOS

Sara Amaro Lopes[1], Ana da Rocha Sá[1], Bruno Miguel Fernandes[1], Miguel Castro[2], Lúcia Costa[1]

1 - Rheumatology Department, ULS São João, Porto, Portugal

2- Radiology Department, ULS São João, Portugal

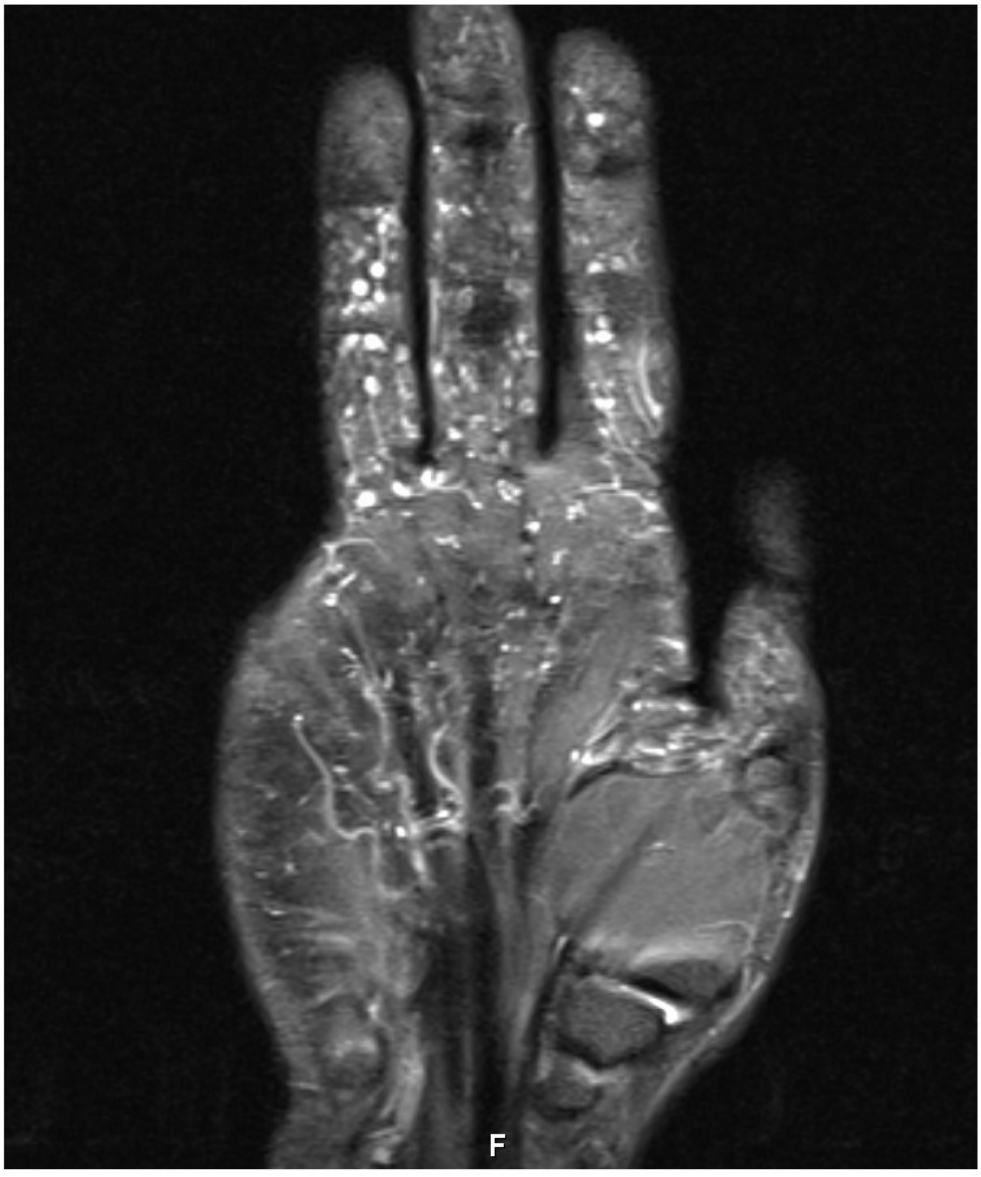
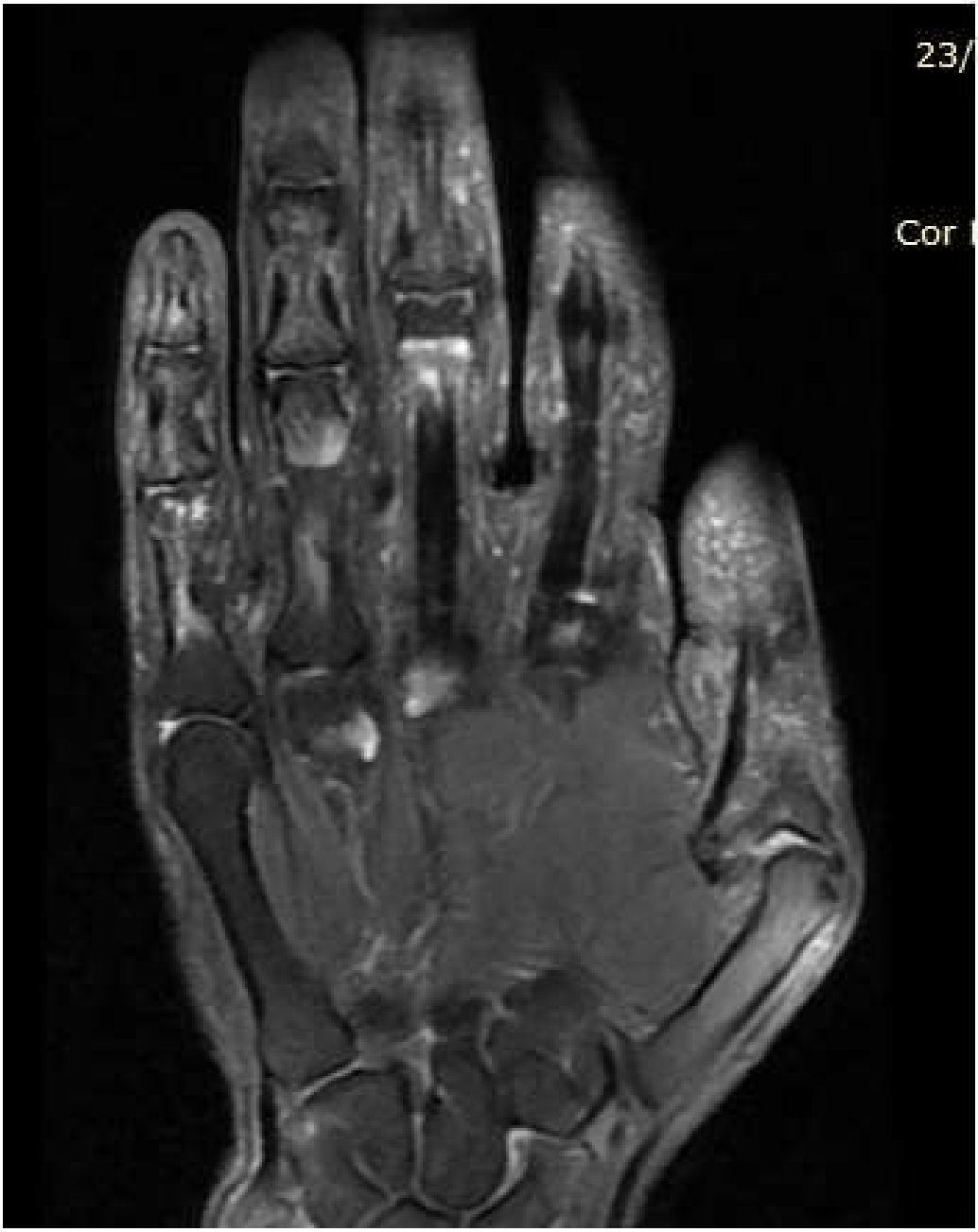


IMAGEM 1: Hiperplasia de corpúsculos de *Pacini*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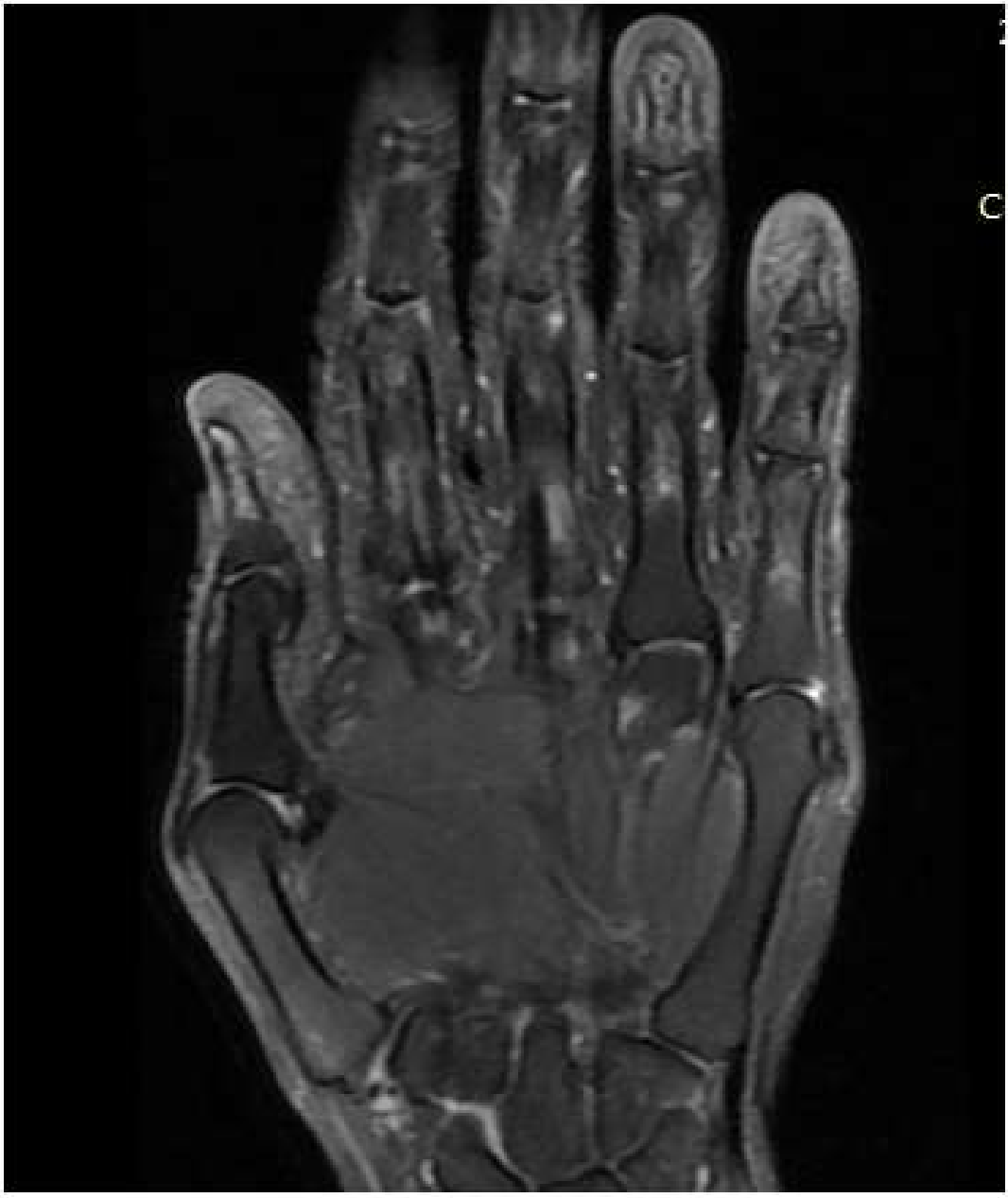


IMAGENS 2 e 3: Doença microgeódica das falanges

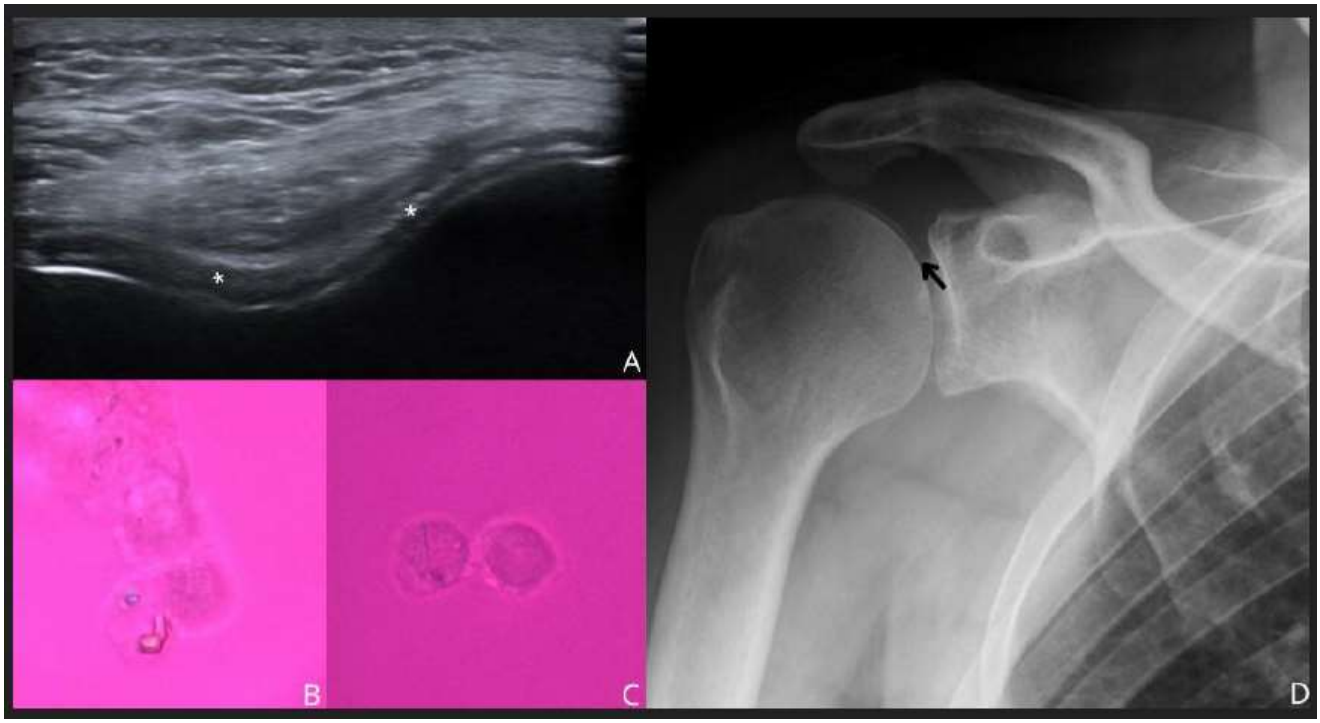
1) Mulher, 58 anos, com nódulos dolorosos. RM com alterações sugestivas de hiperplasia de corpúsculos de Pacini. 2) Mulher, 58 anos, com artralgias e edema dactilar. RM compatível com doença microgeódica das falanges. Pode associar-se a algumas doenças imunomediadas e o tratamento é conservador.

C IM 25 - DOUBLE TROUBLE IN THE KNEE: WHO TO BLAME?

Sara Alves Costa1, Tânia Cardoso2, Cristiana Canha2, Sara Serra1

1- Rheumatology Department, Coimbra´s Local Health Unit

2- Clinical Pathology Department, Coimbra´s Local Health Unit



A 63-year-old man presents with an monoarthritis of the right knee. US examination(A) revealed chondrocalcinosis within the femorotibial cartilage (white asterisks).

Synovial fluid examination confirmed these findings, but also demonstrated the presence of monosodium urate crystals (B), rather than just calcium pyrophosphate crystals (C).

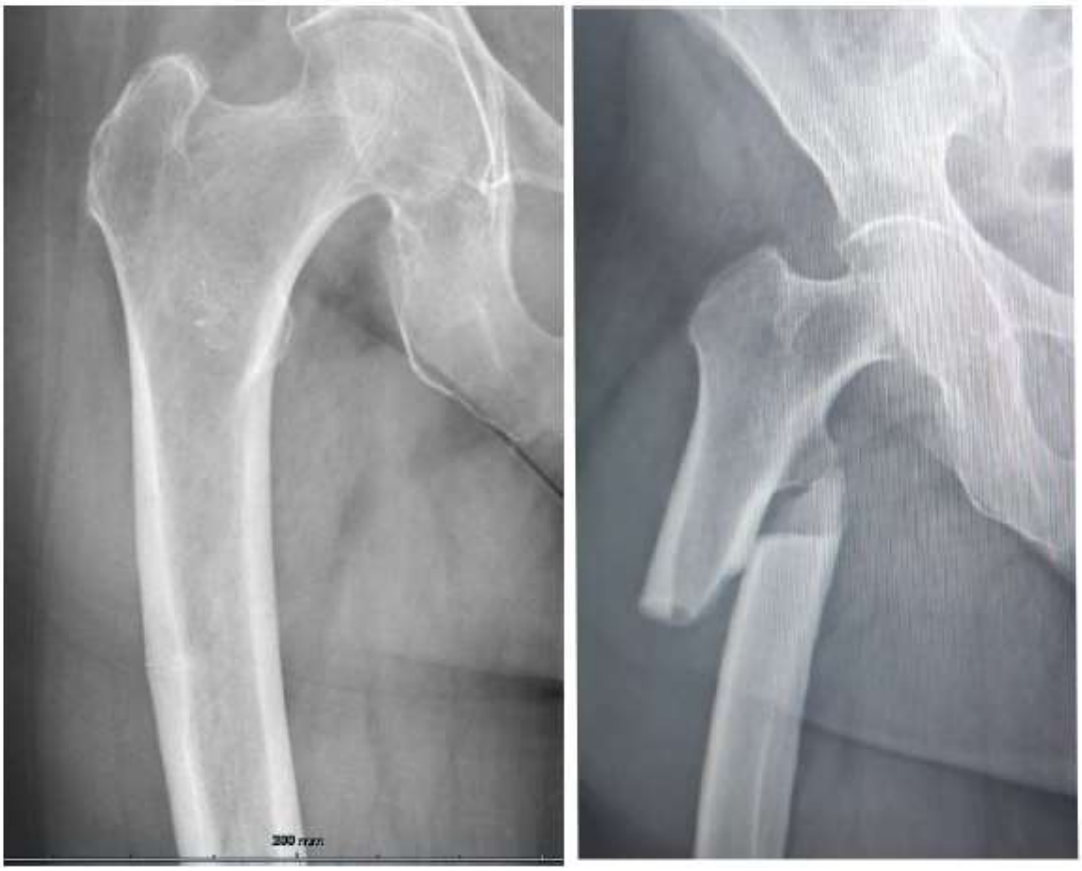
We can also observe, on the x-ray (D), the presence of calcification parallel to the contour of the humeral head (black arrow).

C IM 26 - ATYPICAL FRACTURE AFTER LONG BIPHOSPHONATE USE

Leonor Reynolds

Instituto Português de Reumatologia

76-years-old female, on ibandronate for 5 years after a 4-year drug holiday and previous treatment with 5 years of risedronate, presents with right hip pain while hanging her clothes conditioning a fall. X-ray revealed femoral shaft fracture. Previous X- ray showed atypical femoral fracture. She underwent surgery and started teriparatide.



C IM 27 - RAPIDLY PROGRESSIVE DIGITAL ISCHEMIA

Leonor Reynolds Sousa1, Susana Fernandes1, Helena Santos1,2

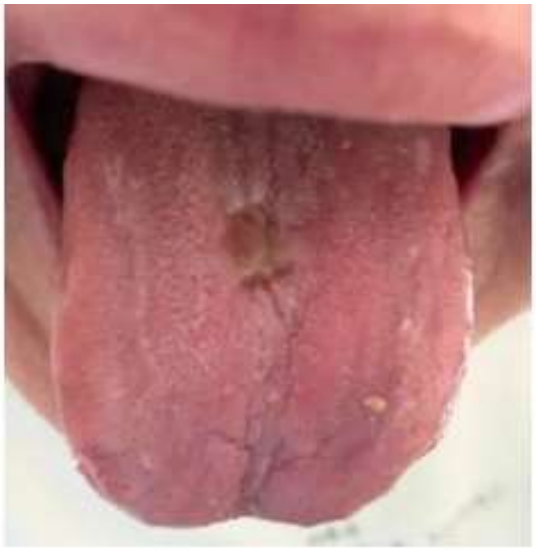
1- Instituto Português de Reumatologia

2- NOVA Medical School, University of Lisbon

72-years-old male with new-onset Raynaud phenomenon and rapidly progressive distal ischemia with necrosis of several fingers. Work-up showed interstitial lung disease, positive anti-SSA, and enterococcus faecalis bacteremia. Patient died of multiorgan failure three weeks after, before diagnosis was confirmed.

C IM 28 - TONGUE ULCER AS AN ATYPICAL MANIFESTATION OF CYTOMEGALOVIRUS INFECTION

Fabiana Gouveia, João Oliveira, Mariana Rodrigues, Filipa André, Sara Costa, Maria João Cadório, Fernando Albuquerque, Marcelo Neto, André Saraiva, Mariana Luís



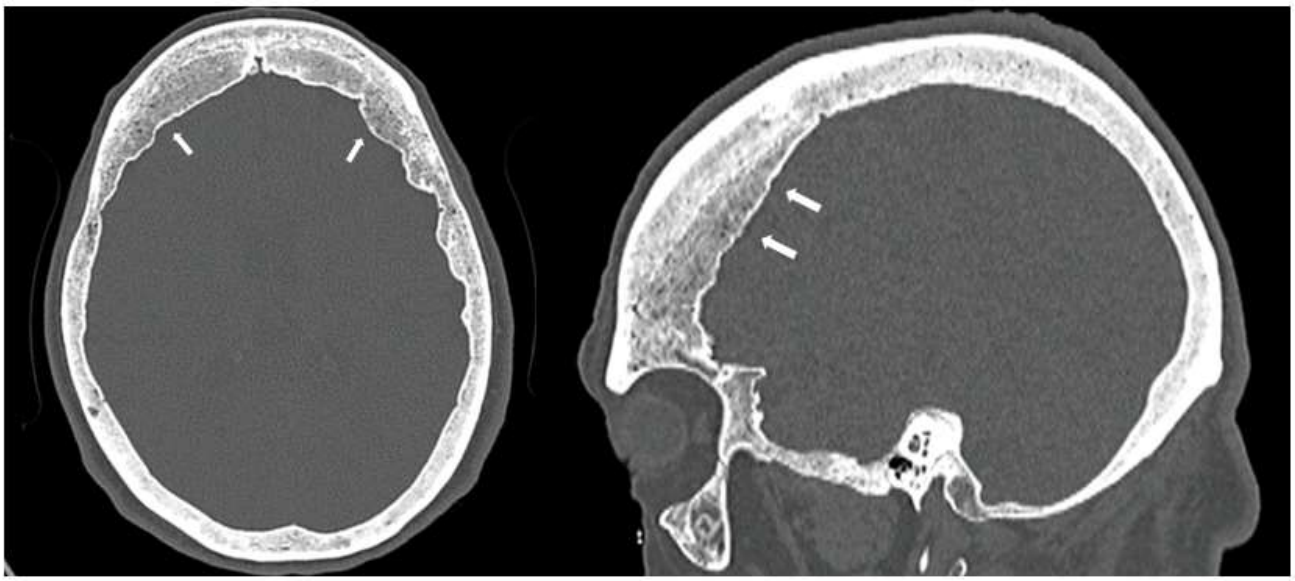
Tongue ulcer as an atypical manifestation of Cytomegalovirus infection in a patient with Polyarteritis Nodosa (on cyclophosphamide and high-dose corticosteroids). Involvement confirmed by histopathological analysis: cells with cytoplasmic and nuclear enlargement with eosinophilic nuclear and cytoplasmic inclusions. Cyclophosphamide was stopped until a 3-week course of ganciclovir was completed.

C IM 29 - MIMETISMO ÓSSEO: ENTRE PAGET E HIPEROSTOSE

Silva C[1], Fernandes J[2], Beirão T[1], Rua C[1], Patela M[1], Pinto P[1], Meirinhos T[1,]

[1] *Serviço de Reumatologia da Unidade Local de Saúde Gaia e Espinho*

[2] *Serviço de Radiologia da Unidade Local de Saúde Gaia e Espinho*



Mulher de 82 anos, previamente seguida noutra instituição com diagnóstico de doença de Paget monostótica craniana, assintomática, sem alterações da fosfatase alcalina ou achados radiográficos sugestivos. Na cintigrafia óssea de reavaliação observou-se atividade osteoblástica difusamente aumentada e heterogénea no osso frontal, em padrão mais compatível com hiperostose frontal interna do que com doença de Paget, achados corroborados por tomografia computorizada.

C IM 30 - SUDECK'S SYNDROME: AN IMPORTANT DIFFERENTIAL DIAGNOSIS IN SEVERE OSTEOPENIA

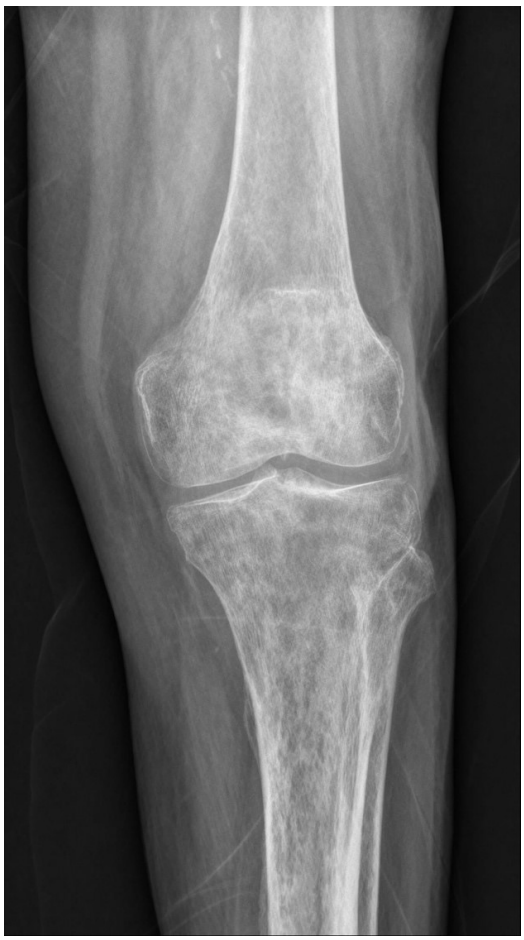
Carlota Nóbrega[1], Tiago Pereira[2], Fernando Luz Campina[1], Miguel Pádua Figueiredo[1]

1. Serviço de Ortopedia - Hospital de Cascais Dr. José de Almeida

2. Serviço de Ortopedia - Hospital de Santa Maria

Case presentation: An 81-year-old man, six months after a tibial tuberosity fracture, with a history of prostate carcinoma and negative CT for bone metastases.

Image description: Radiograph of the left knee shows severe diffuse osteopenia with preserved joint surfaces. Differential diagnoses include metastatic disease, Sudeck's atrophy, disuse osteoporosis, and inflammatory arthropathies.



C IM 31 - UM DIAGNÓSTICO À VISTA DESARMADA

Maria João Cadório*[1], Mariana Mendes Rodrigues*[1], João Teixeira[2], João Oliveira[1], Fabiana Gouveia[1], Filipa Canhão André[1], Sara Costa[1], Fernando Albuquerque[1], Marcelo Neto[1], André Saraiva[1]

1. Serviço de Reumatologia, Unidade Local de Saúde de Coimbra

2. Serviço de Dermatologia e Venereologia, Unidade Local de Saúde de Coimbra

**Contribuíram igualmente para o desenvolvimento do trabalho*

Mulher, 86 anos. Edema infra-palpebral exuberante com 6 meses. Evoluiu com sinal do decote, xaile, coldre e pápulas de Gottron ulceradas, síndrome constitucional, mialgias e fraqueza muscular proximal. O estudo revelou CK 2748 U/L, ANA AC4 1/1280, anti-TIF1γ+, eletromiografia e biópsia cutânea compatíveis com dermatomiosite, associado a envolvimento multissistémico e neoplasia oculta metastizada.

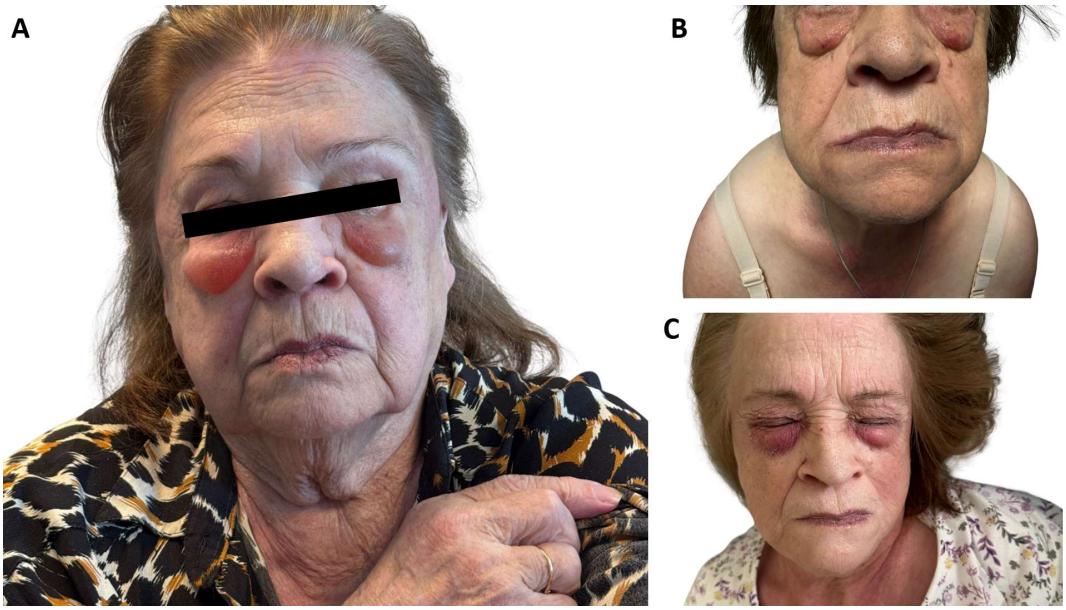


Figura 1. Dermatomiosite, manifestando-se com eritema heliotropo. Evolução cronológica das lesões cutâneas: eritema violáceo e edema infra e periorbitário exuberante (A), seguido de persistência do eritema com descamação fina (B) e posterior atenuação do edema com hiperpigmentação residual após terapêutica imunossupressora (C).

C IM 32 - O PESO DOS CRISTAIS: GOTA TOFÁCEA NÂO TRATADA

Maria João Cadório[1], João Oliveira[1], Mariana Mendes Rodrigues[1], Fabiana Gouveia[1], Filipa Canhão André[1], Sara Costa[1], Fernando Albuquerque[1], Marcelo Neto[1], Cátia Duarte[1]

1. Serviço de Reumatologia, Unidade Local de Saúde de Coimbra

Homem, 85 anos. Crises de monoartrite aguda recorrentes e intervalos intercríticos assintomáticos, com uricémia > 10mg/dL há mais de 30 anos, sem qualquer tratamento hipouricemiante prévio. Atualmente, marcada incapacidade funcional e doença renal crónica. Medicado com febuxostate com ótima resposta clínica e analítica.

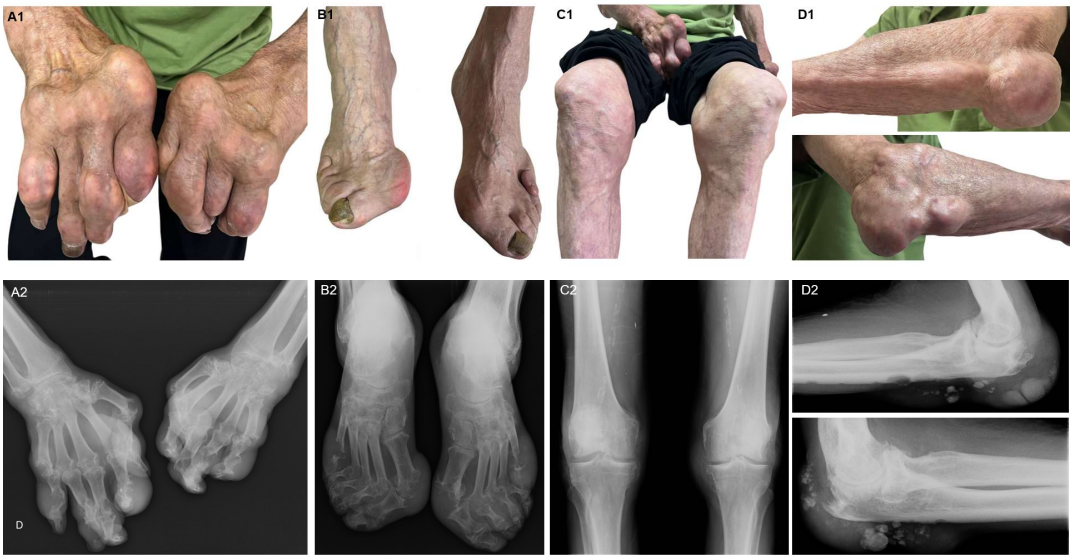
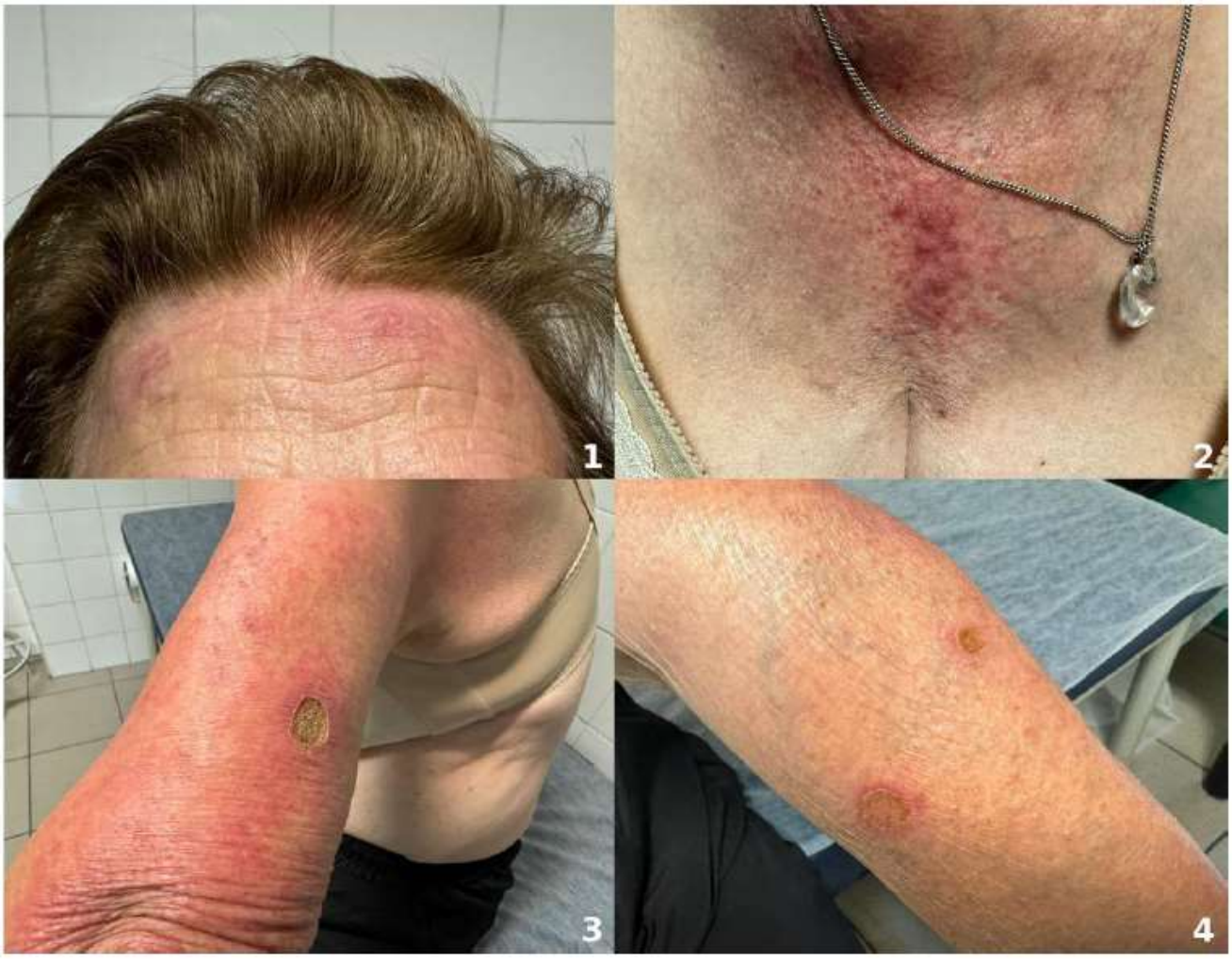


Figura 1. Múltiplos tofos subcutâneos e deformidades articulares graves em doente com gota tofácea crónica não tratada. Deformidade articular acentuada e volumosos tofos sobre as pequenas articulações das mãos **(A1)**, pés **(B1)**, joelhos **(C1)** e cotovelos **(D1)**. As radiografias correspondentes **(A2–D2)** evidenciam marcada destruição e erosões justarticulares em "saca bocados", características da gota tofácea crónica.

C IM 33 - LESÕES ULCERADAS EM DOENTE COM DERMATOMIOSITE EM CONTEXTO PARANEOPLÁSICO

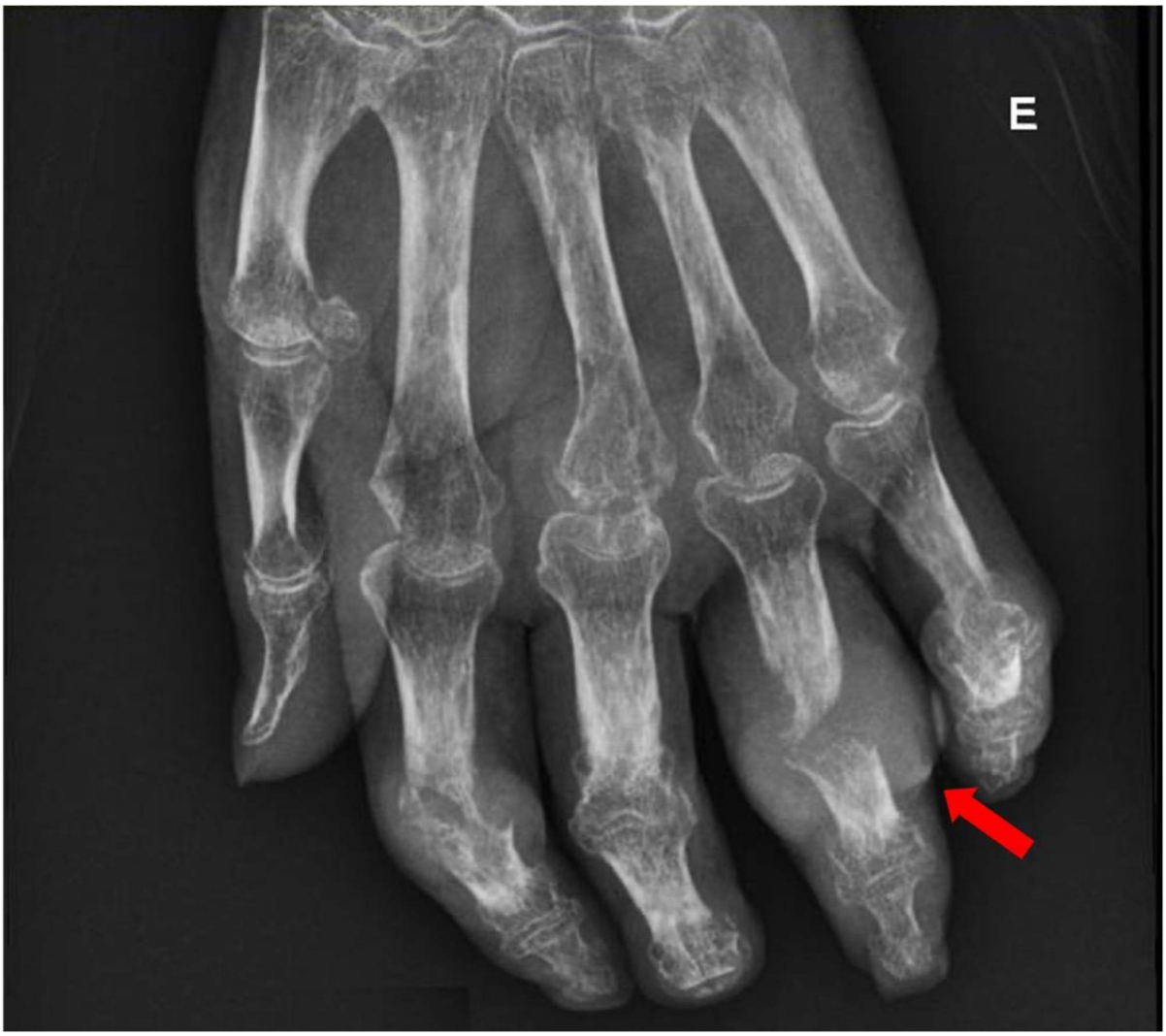
Maria de Sá Pacheco, Nuno Delgado, Ana Águeda, Miguel Guerra, Rita Pinheiro Torres, Joana Ramos Rodrigues, Margarida Alexandre Oliveira



Mulher de 86 anos com quadro constitucional e lesões cutâneas de novo caracterizadas por dermatite descamativa do couro cabeludo e fronte(Fig.1), eritema heliotropo, sinal emV(Fig.2) e do xaile, eritema poiquilodérmico e lesões ulceradas de fundo fibrinoso dos membros superiores (Fig.3,4) e pápulas de Gottron. Diagnosticada dermatomiosite em contexto paraneoplásico (ac anti-Tif1 gama positivo).

C IM 34 - DESTRUIÇÃO ARTICULAR COMPLETA EM DOENTE COM GOTA TOFÁCEA

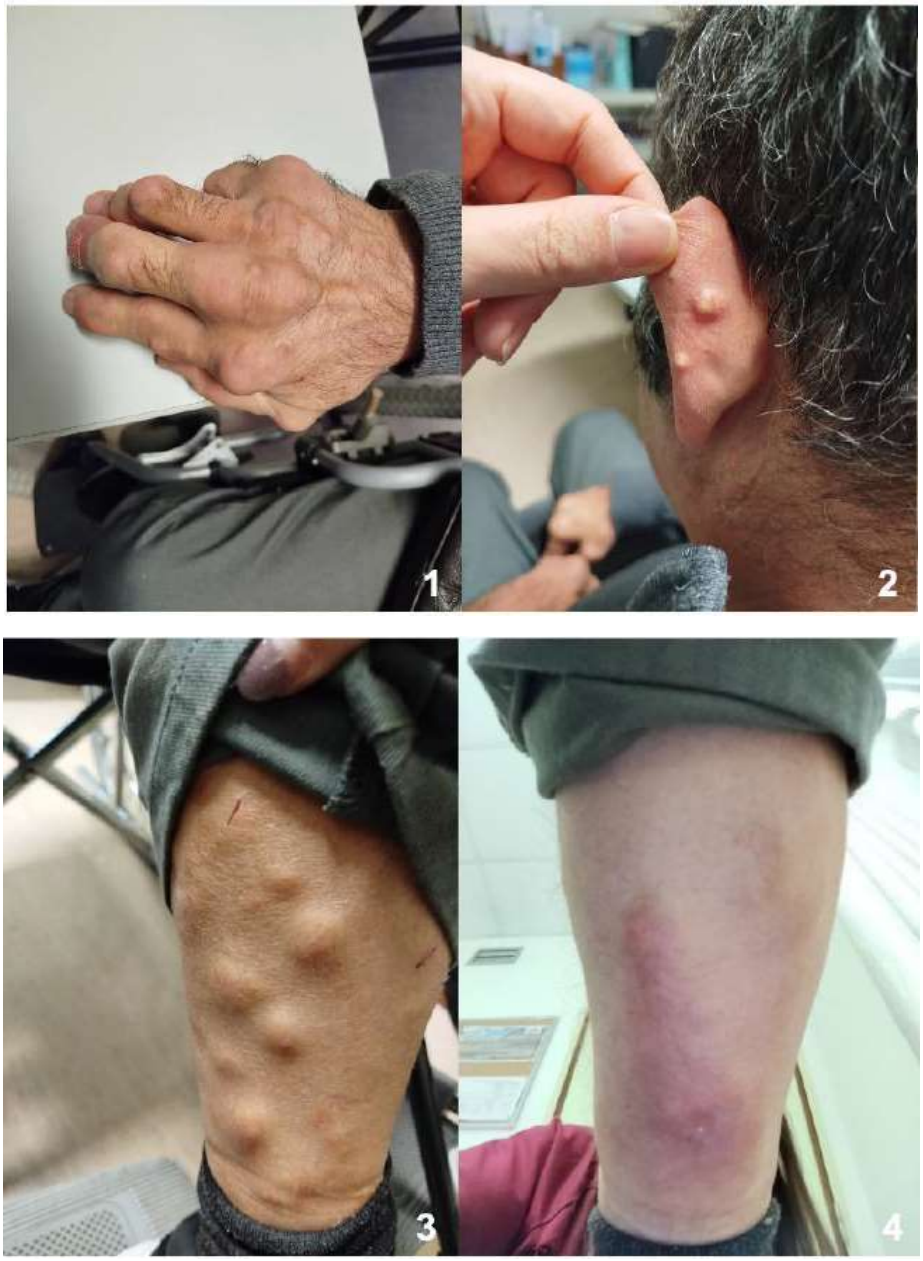
Maria de Sá Pacheco, Nuno Delgado, João Corrêa, Catarina Lourenço Rodrigues, Ana Águeda, Miguel Guerra, Rita Pinheiro Torres, Joana Ramos Rodrigues, Margarida Alexandre Oliveira



Homem de 93 anos com gota tofácea não tratada. Quadro de dor de início súbito, edema e impotência funcional ao nível do 4º dedo da mão esquerda, no qual apresentava ainda hematoma com drenagem de conteúdo sero-hemático. Sem trauma conhecido. Radiografia a demonstrar reabsorção completada 4ª IFP, por destruição óssea articular generalizada. Realizada imobilização o e analgesia.

C IM 35 - GOTA TOFÁCEA COM LOCALIZAÇÃO ATÍPICA

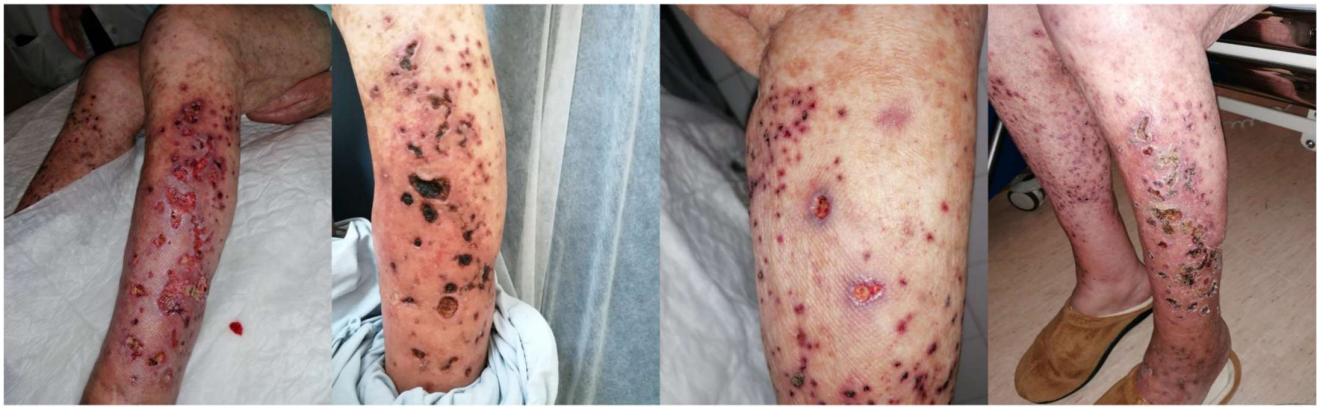
Maria de Sá Pacheco, Nuno Delgado, Ana Águeda, Miguel Guerra, Rita Pinheiro Torres, Joana Ramos Rodrigues, Margarida Alexandre Oliveira



Homem de 51 anos com gota tofácea, doença renal crónica e paraplegia sequelar pós-AVC. À observação com presença de tofos gotosos em localizações típicas, nomeadamente articulações das mãos (Fig.1) e pavilhões auriculares(Fig.2), mas também com formações nodulares ao nível dos membros inferiores na região prétibial(Fig.3) e na região posterior com ponto de drenagem em resolução(Fig.4).

C IM 36 - LESÕES VASCULÍTICAS SOBREINFETADAS EM DOENTE COM ARTRITE PSORIÁTICA SOB METOTREXATO

Maria de Sá Pacheco, Nuno Delgado, Ana Águeda, Miguel Guerra, Rita Pinheiro Torres, Joana Ramos Rodrigues, Margarida Alexandre Oliveira



Mulher de 91 anos, com artrite psoriática sob metotrexato. Quadro de lesões purpúricas nos membros inferiores, com mais de 3 meses de evolução, histologicamente compatível com vasculite leucocitoclástica. Agravamento posterior com presença de lesões vesiculares, ulceradas e necrosadas secundárias a reativação por citomegalovírus sobreinfetadas com *Proteus mirabilis* e *Epidermophyton floccosum.*

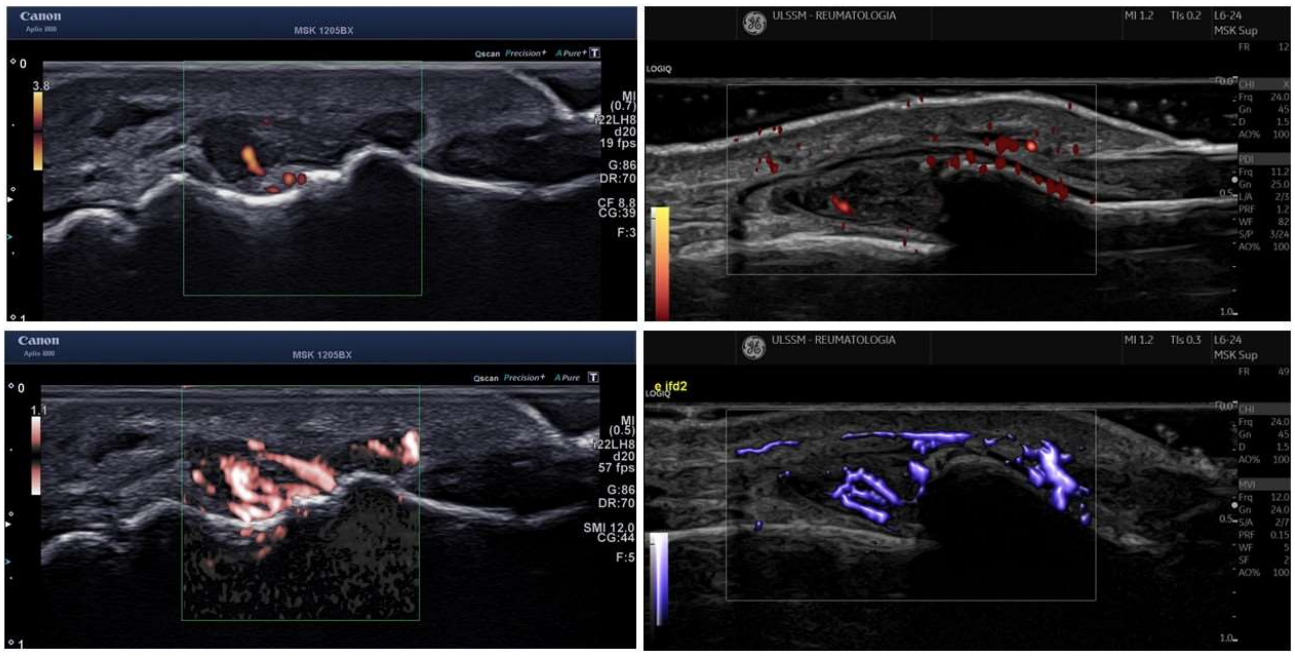
C IM 37 - BEYOND POWER DOPPLER: SIDE-BY-SIDE MICROVASCULAR IMAGING IN JOINT ASSESSMENT

Carolina Vilafanha*[1,2], João Aguiar* [3,4], Joaquim Polido Pereira [3,4]

[1]*Serviço de Reumatologia, Unidade Local de Saúde da Região de Aveiro (ULSRA), Aveiro;*

[2]*Centro de Investigação em Reumatologia de Aveiro (CIRA), Centro Académico Clínico Egas Moniz Health Alliance;* [3]*Serviço de Reumatologia, Unidade Local de Saúde de Santa Maria (ULSSM), Centro Académico de Medicina de Lisboa;* [4]*Unidade de Investigação em Reumatologia, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa.*

** Both contributed equally*



Ultrasound showing exuberant osteophytosis with grade 2–3 synovitis and grade 1 Power Doppler (PD) signal in the second distal interphalangeal joint. Side-by-side view: Canon (left) – PD vs Superb Microvascular Imaging; GE (right) – PD vs Microvascular Imaging. Both techniques showed greater sensitivity than PD, though the clinical significance of the additional signals detected remains uncertain.

C IM 38 - POST-TRAUMATIC SPONDYLOARTHRITIS-LIKE ANKYLOSIS AND NEUROGENIC HETEROTOPIC OSSIFICATION IN A YOUNG TETRAPLEGIC MAN

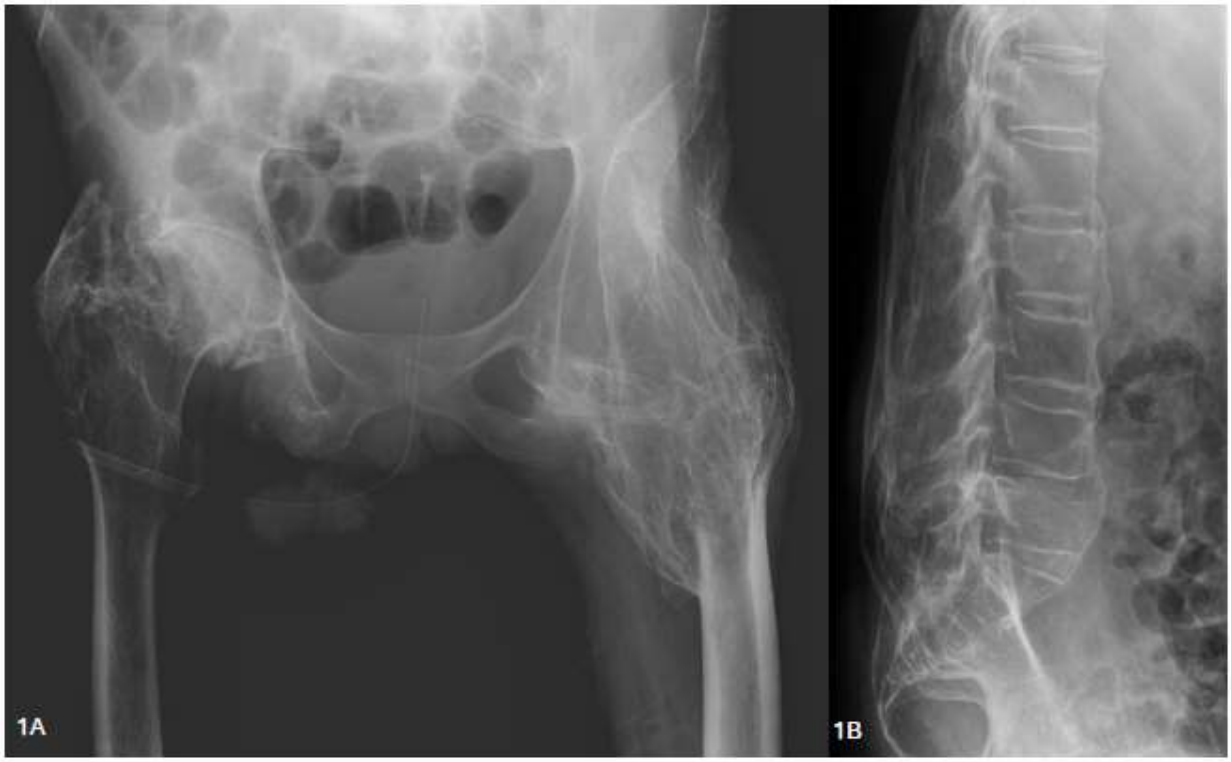
Bianca Paulo Correia1,2, Gonçalo Boleto1,2, Joana Martins-Martinho1,2, Susana Capela1,2, Ricardo Henriques2,3, João Eurico Fonseca1,2, Elsa Vieira-Sousa1,2

1. Rheumatology Department, Unidade Local de Saúde de Santa Maria (ULSSM), Centro Académico de Medicina de Lisboa, Lisbon, Portugal

2. Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisbon, Portugal

3. Physical Medicine and Rehabilitation Department, Unidade Local de Saúde de Santa Maria (ULSSM), Lisbon, Portugal

A 33-year-old man with spastic tetraplegia after C4 spinal injury was referred for progressive joint stiffness. Imaging showed widespread ankylosis, neurogenic heterotopic ossification of hips, and sacroiliac involvement, suggesting a post-traumatic spondyloarthritis-like phenotype.



C IM 39 - TATTOO REVEALING SARCOIDOSIS

Anita Cunha[1], Maria Pontes Ferreira[1], Susana Almeida[1], Diana Barros[1], Duarte Augusto[2], José Tavares-Costa[1], Daniela Peixoto[1]

[1] Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal

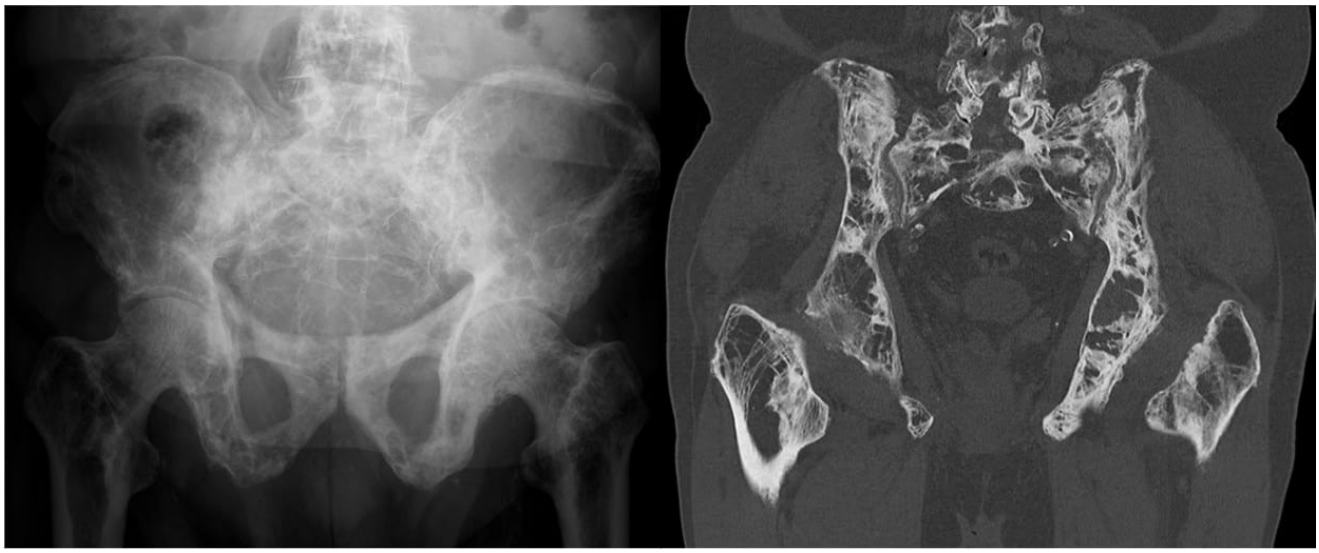
[2] Rheumatology Department, Unidade Local de Saúde da Guarda, Guarda, Portugal

Cutaneous sarcoidosis may arise in areas of prior trauma, such as scars or tattoos, and serve as an important diagnostic clue.

We present a 53-year-old woman with oligoarthritis, erythema nodosum and hilar lymphadenopathy. Further work-up confirmed sarcoidosis, and the concomitant appearance of nodules over an old tattoo, a well-recognized cutaneous feature, was interpreted as part of the disease spectrum.

C IM 40 - POLYOSTOTIC PAGET'S DISEASE

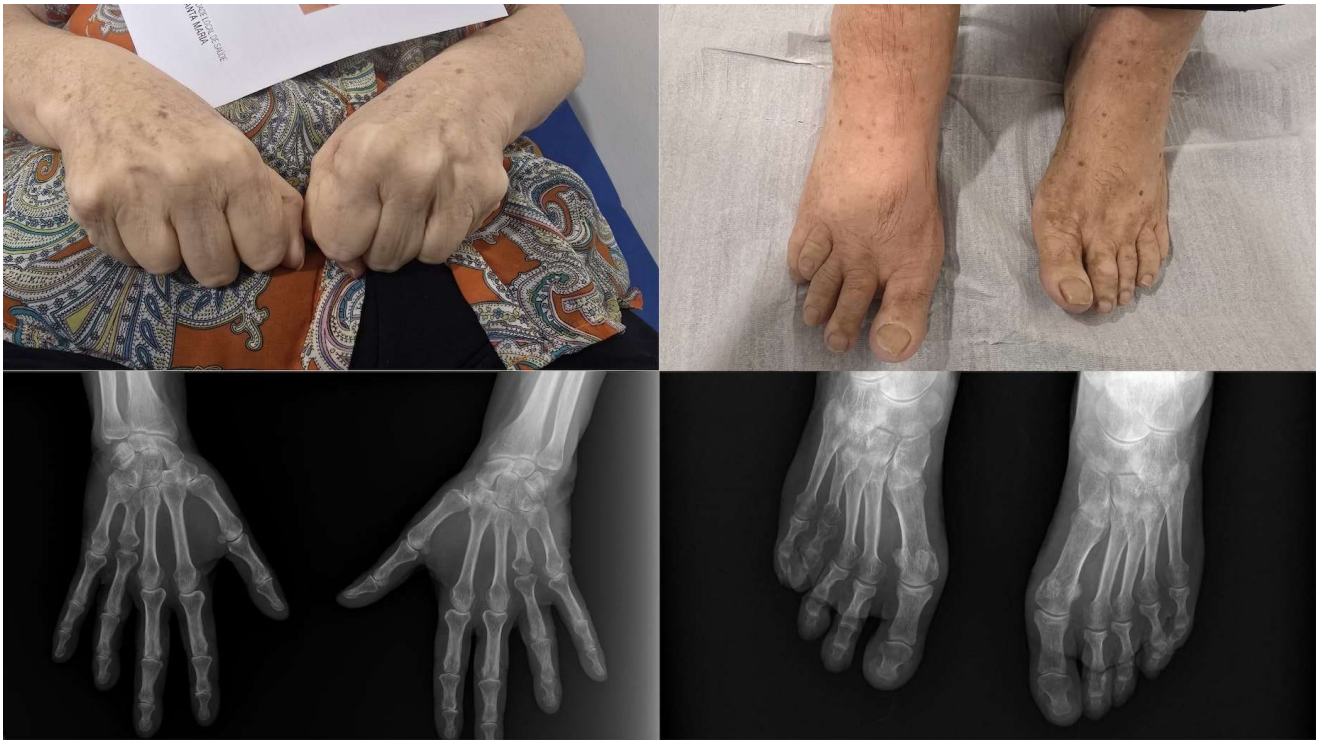
João Aguiar, Augusto Silva, Joaquim Pereira



An 88-year-old man with polyostotic Paget's disease affecting the spine, sacrum, iliac bone, and femur. Extensive bone remodeling caused lumbar stenosis and multisegmental radiculopathy. He was treated with denosumab due to chronic kidney disease, achieving symptomatic and biochemical improvement. Left: Pelvic X-ray; Right: Lumbar CT.

C IM 41 - BRACHYMETACARPIA AND BRACHYMETATARSIA A RARE FINDING

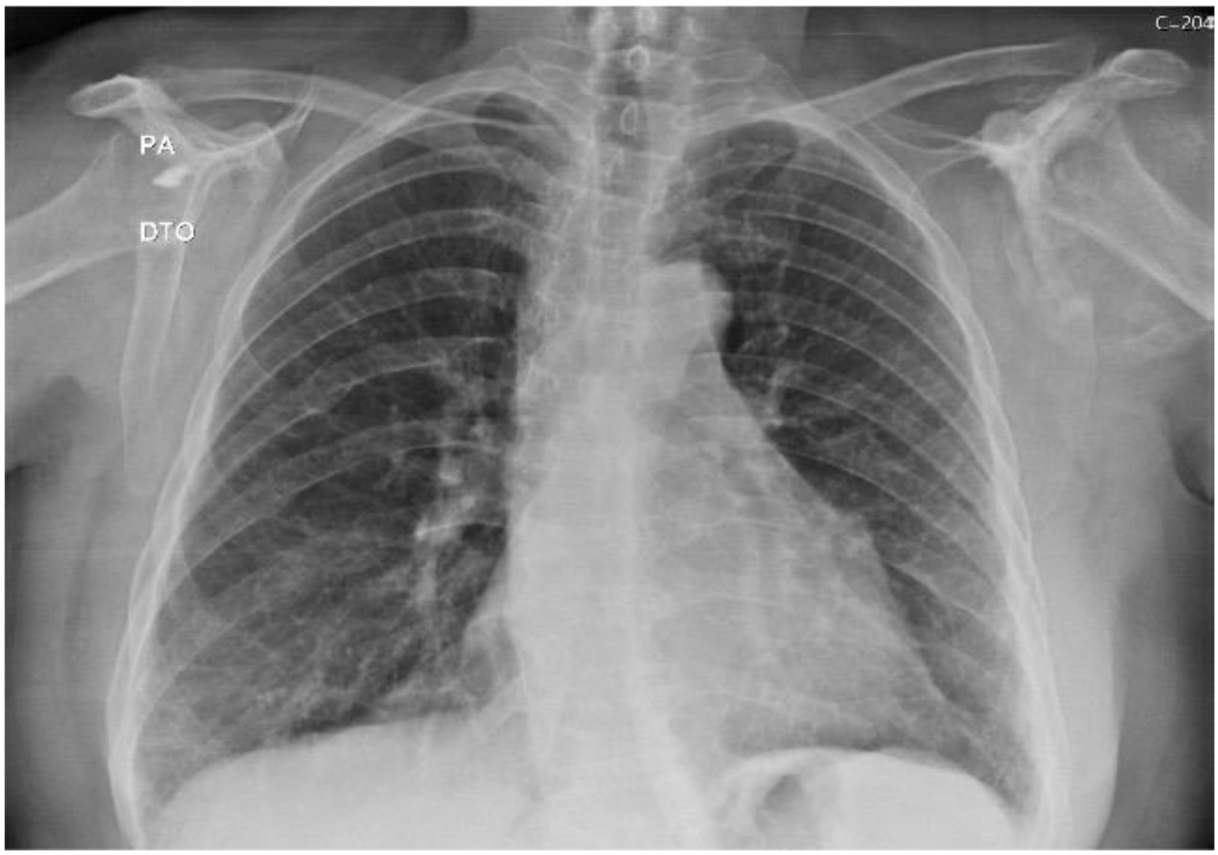
João Aguiar, Carolina Vilafanha, Joaquim Polido Pereira



A 71-year-old woman presented for hand and foot ultrasound, revealing brachymetacarpia and brachymetatarsia, characterized by shortening of the metacarpal and metatarsal bones due to premature epiphyseal closure. These rare anomalies may appear isolated or as part of syndromic conditions such as Turner or pseudohypoparathyroidism.

C IM 44 - "OMBRO DE CHARCOT" – UMA ARTROPATIA RARA, MAS ALTAMENTE INCAPACITANTE

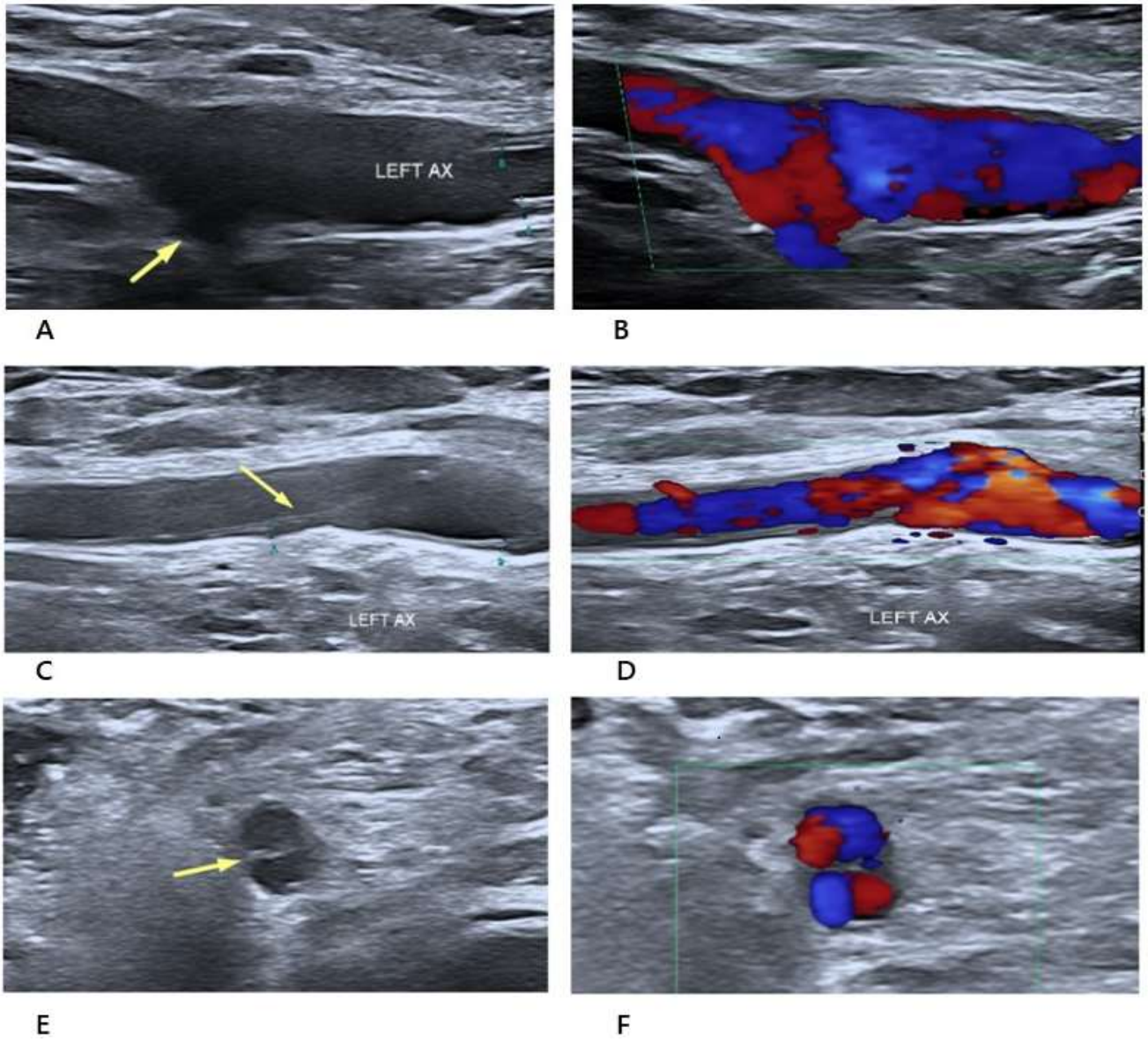
Paulo Jorge Pereira, Hugo Gonçalves, Carla Campinho Ferreira, Ana Margarida Correia, Emanuel Costa, Diogo Esperança Almeida, Joana Leite Silva, Marcos Cerqueira, Ana Ribeiro, Joana Sousa Neves



Mulher, 61 anos, operária fabril, com antecedentes de siringomielia e tendinopatia da coifa. Descrevia omalgia mecânica bilateral estável há vários anos. Nos 2 meses prévios, referia agravamento à esquerda após queda no domicílio, com lesão do plexo braquial por estiramento do membro superior. Em imagem apresentava destruição da normal estrutura umeral à esquerda, sugestiva de Ombro de Charcot.

C IM 45 - SILENT AXILLARY ARTERY COMPLICATIONS IN GIANT CELL ARTERITIS

Mariana Pereira Silva, Nikita Khmelinskii



Aortic complications in GCA are well known. We report two GCA cases with axillary artery complications of subclinical arteritis. In one case, follow-up axillary artery ultrasound (AAUS) showed a chronic halo and aneurysmal dilatation (A and B); in the other, AAUS revealed a double lumen consistent with arterial dissection (C-F).

C IM 46 - FRATURA DO ESCAFÓIDE PROXIMAL COM NECROSE AVASCULAR ASSOCIADA – UMA CAUSA RARA DE DOR DO PUNHO

Nuno Delgado; Maria Pacheco; Miguel Guerra; Ana Águeda; Joana Rodrigues; Rita Torres; Margarida Oliveira

Doente do sexo masculino, 26 anos, com doença inflamatória intestinal sob mesalazina, com dor persistente no punho direito após movimento de hiperextensão forçada, desde há 7 meses. A radiografia (fig. 1) evidenciou alteração da trabeculação do osso escafóide. RM confirmou fratura do polo proximal, com sinais de necrose avascular associada (hipossinal em T1) (fig. 2).

Fig. 1

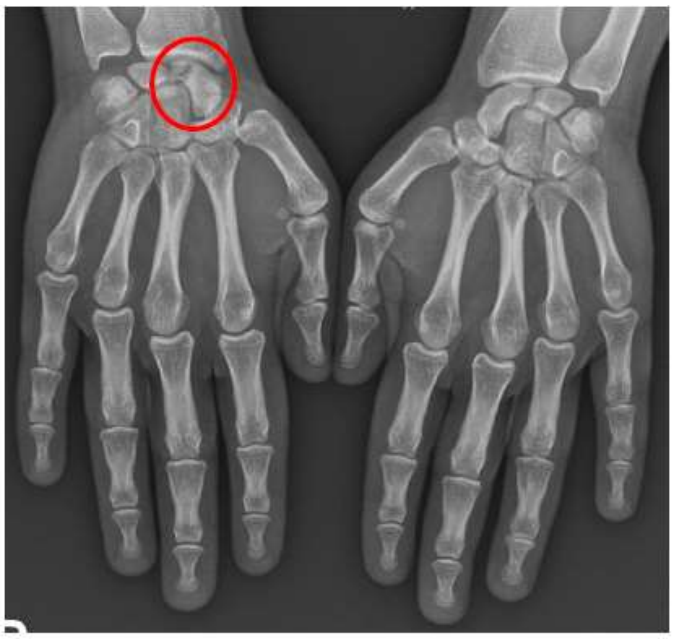
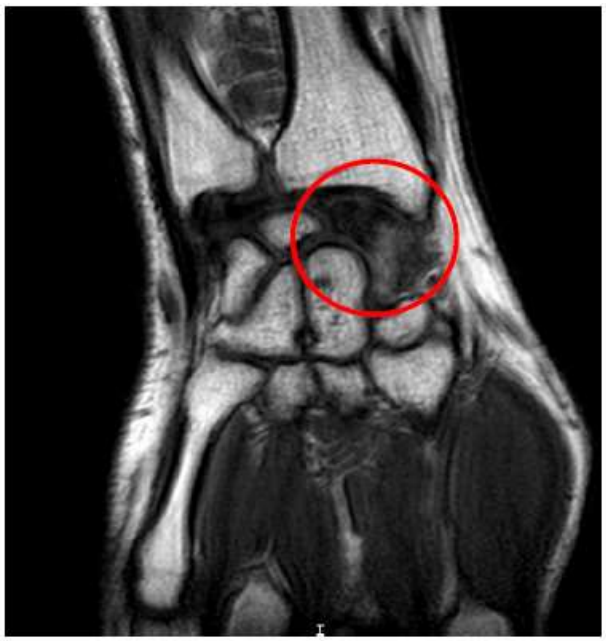


Fig. 2

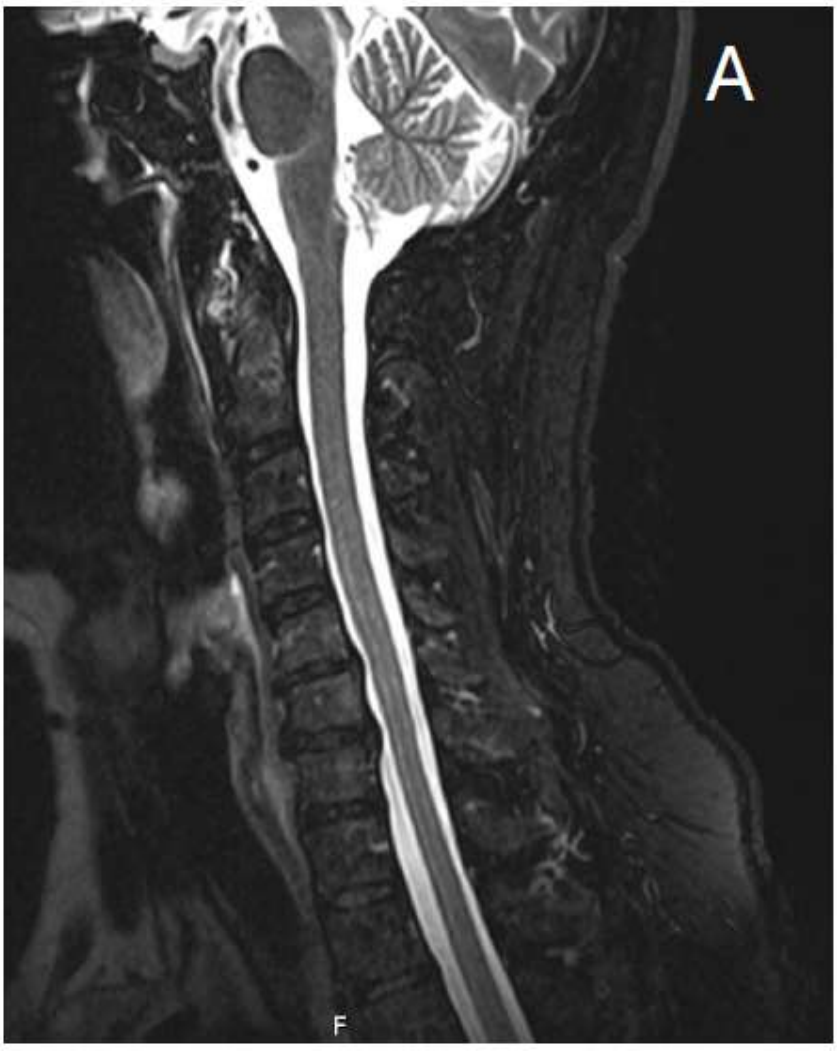


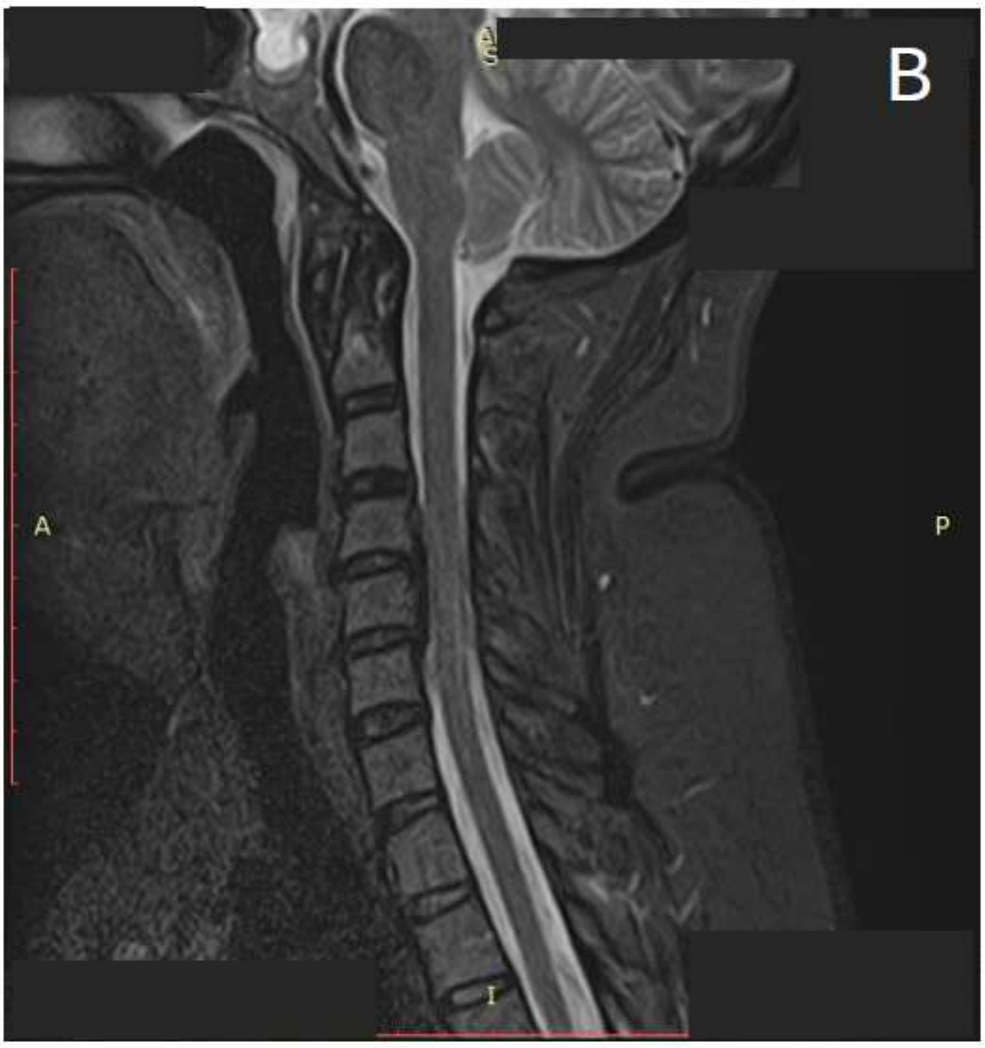
C IM 47 - ATENÇÃO À CERVICAL: ATINGIMENTO ATLANTO-AXIAL EM ESPONDILARTRITES

Ana da Rocha Sá1, Mariana Diz Lopes1, Sara Helena Amaro Lopes1, Salomé Garcia1,2, Lúcia Costa1, Teresa Martins da Rocha,1,2

1. Serviço de Reumatologia, ULS São João, Porto

2. Departamento de Reumatologia, Faculdade de Medicina da Universidade do Porto





Ressonâncias magnéticas cervicais de dois pacientes com cervicalgia recente de ritmo misto.

A) Mulher de 52 anos com espondilartrite axial radiográfica com inflamação atlanto-axial, erosões, com hipersinal em T2 STIR.

B) Homem com artrite psoriática, edema medular ósseo e artropatia erosiva atlanto-axial em T2 STIR.

C IM 48 - OBSCURED VISION: MARKED PERIORBITAL OEDEMA IN ANTI-TIF1-Y DERMATOMYOSITIS

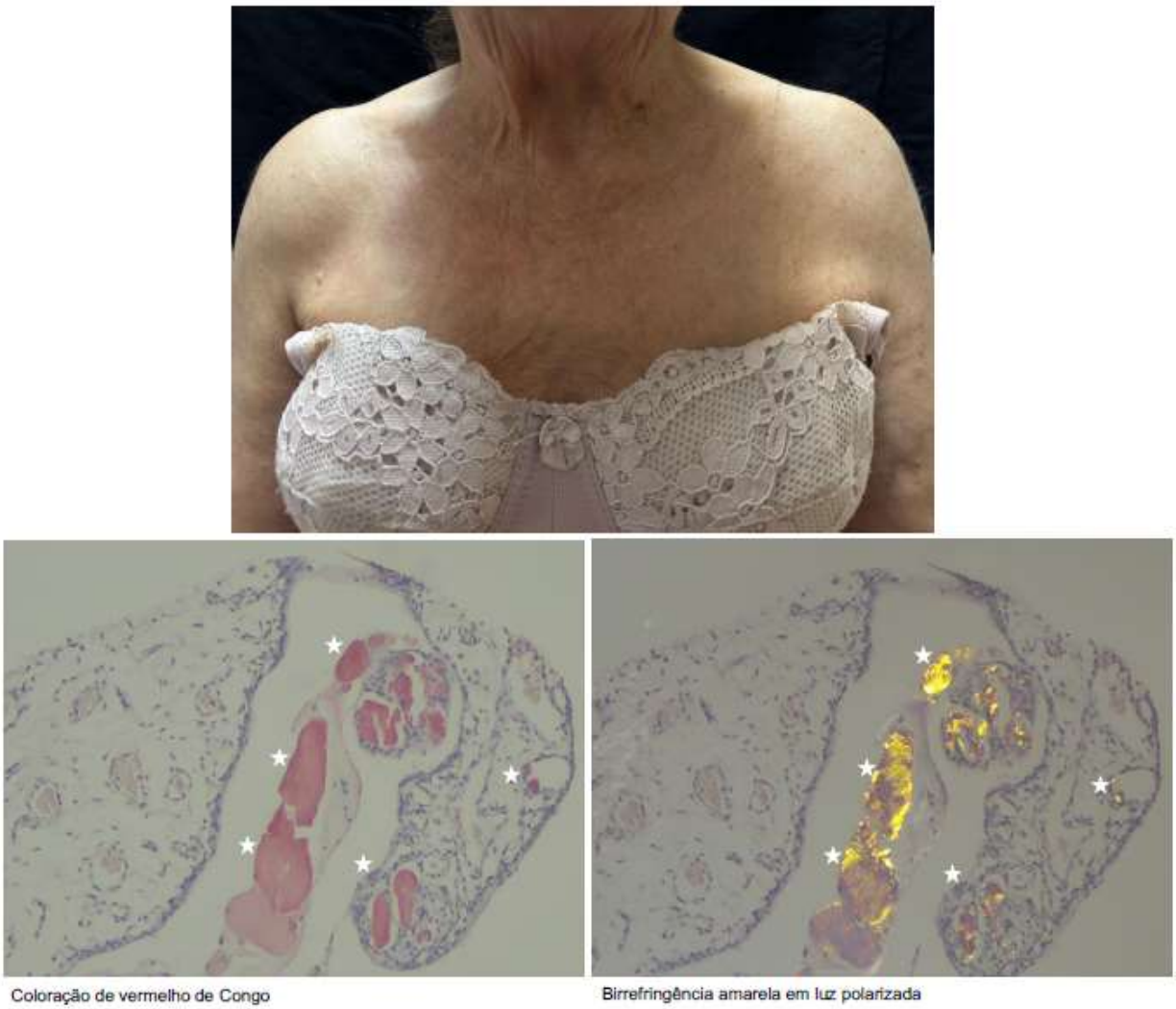
Daniel Melim1, Daniel Carvalho1, Margarida Faria1, Jorge Lopes1, Lídia Teixeira1, Mónica Franco2, Ricardo Figueira1

1. *Rheumatology Department, Centro Hospitalar do Funchal, SESARAM, Madeira, Portugal*
2. *Ophthalmology Department, Centro Hospitalar do Funchal, SESARAM, Madeira, Portugal*

Figure 1. A: 64-year-old woman with anti-TIF1-γ dermatomyositis showing severe left periorbital oedema causing visual obstruction. Infectious, allergic, vascular, and neoplastic causes were excluded. B: Marked improvement after treatment with prednisolone and IVIG. Periorbital oedema, though a classic feature of DM, is often underrecognized or misinterpreted.

C IM 49 - SHOULDER PAD SIGN

Filipa Canhão André*, Marcelo Neto*, Fabiana Gouveia, João Oliveira, André Saraiva, Mariana Santiago, Margarida Coutinho (*Os autores contribuíram de forma igual para o manuscrito)



Legenda:

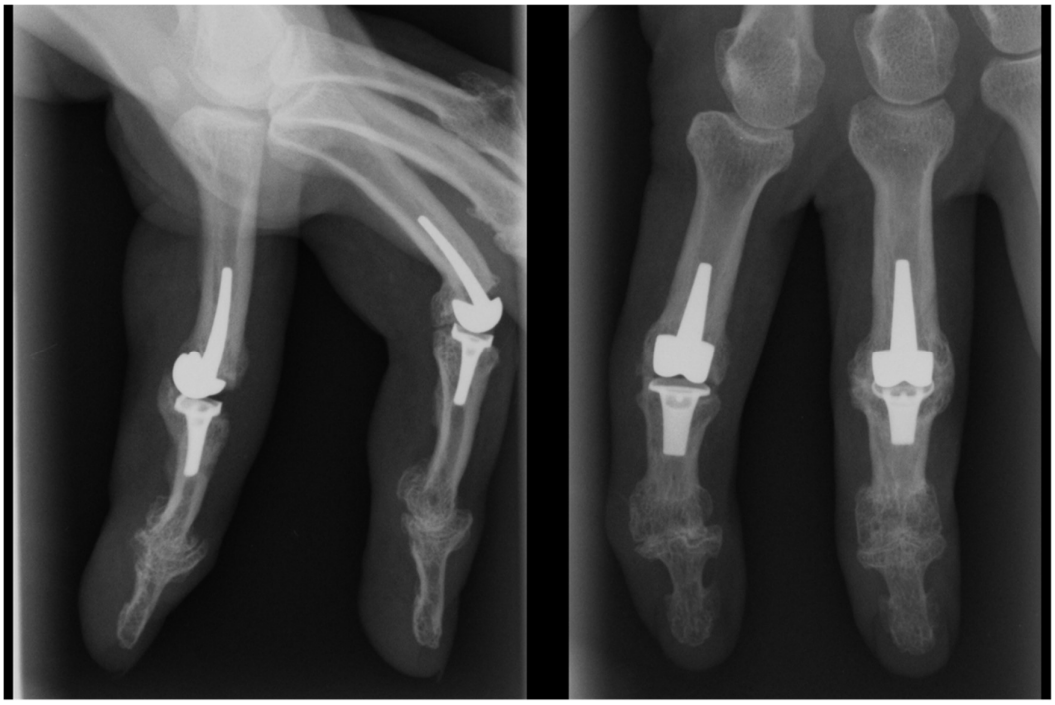
Mulher, 73 anos, com antecedentes de Mieloma Múltiplo sem seguimento e transplante renal por DRC (causa indeterminada). Desenvolveu omalgia bilateral de ritmo misto e tumefação exuberante. A ecografia evidenciou bursite subacromiodeltoideia e sinovite glenoumeral. Foram aspirados 150 cc de líquido sinovial mecânico, com cultura negativa e sem cristais. A biópsia revelou sinovite por Amiloidose.

C IM 50 - UM JOELHO EM CADA DEDO

Nuno Freire[1], Mariana Mendes Rodrigues[2], João Oliveira[2]

[1]IFE em Medicina Física e Reabilitação, ULS de Coimbra

[2] IFE em Reumatologia, ULS de Coimbra



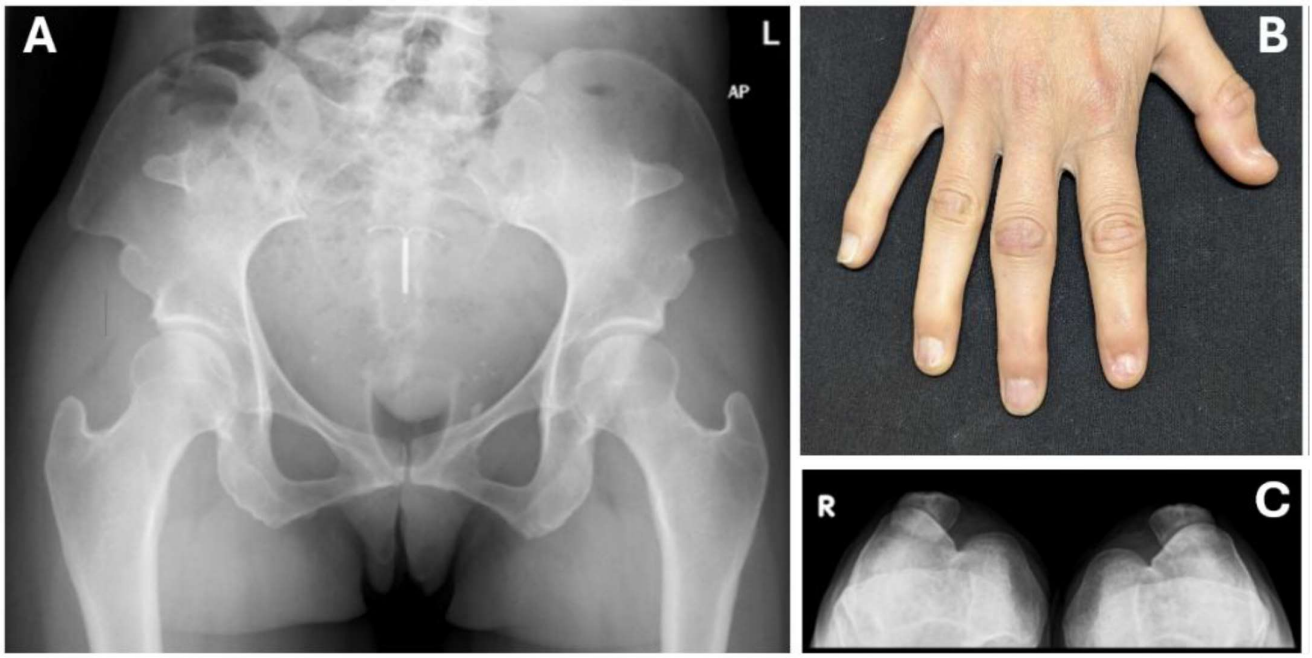
Artroplastia total das 2ª e 3ª IFP em mulher de 62 anos, com osteoartrose nodal exuberante nas mãos desde os 35 anos.

C IM 51 - THE HORNS DON'T LIE: CLASSIC RADIOGRAPHIC CLUES IN NAIL–PATELLA SYNDROME

Mariana Mendes Rodrigues1*, Camila Sousa1*, Sara Alves Costa1, André Saraiva1

1 Serviço de Reumatologia, Unidade Local de Saúde de Coimbra

**Contribuíram igualmente para o trabalho*



A Síndrome Unha-Patela é uma doença autossómica dominante causada por variantes patogénicas no gene LMX1B. A imagem apresenta achados imagiológicos típicos: cornos ilíacos, considerados patognomónicos (A); hipoplasia ungueal e ausência de pregas sobre as articulações interfalângicas distais (B); patelas hipoplásicas (C). A presença destes achados é muito sugestiva do diagnóstico (1).

Referências:

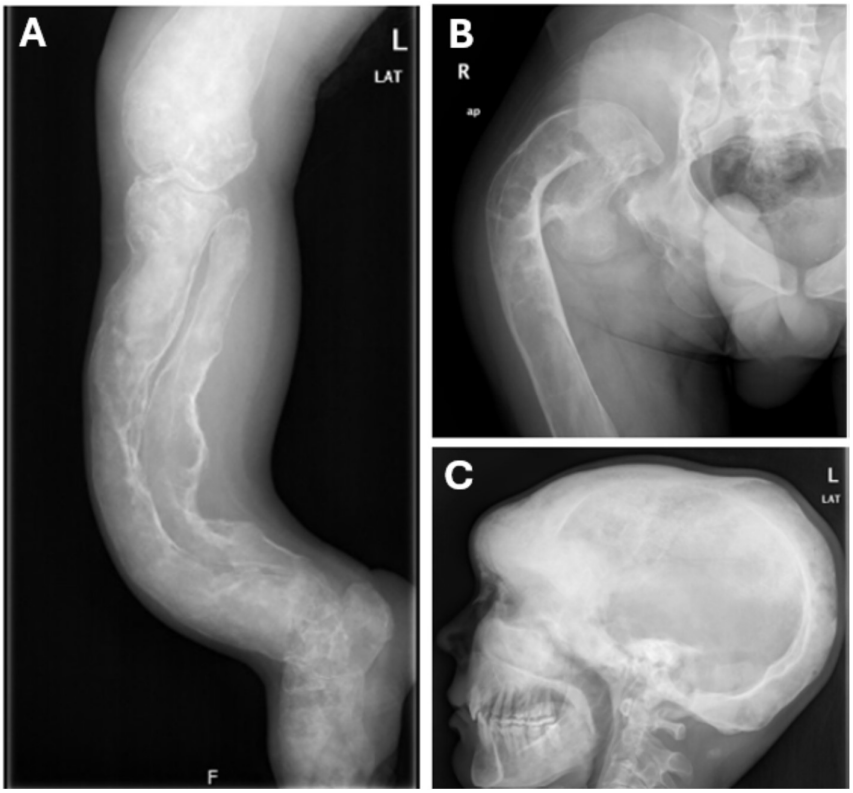
1. Sweeney E, Hoover-Fong JE, McIntosh I. Nail-Patella Syndrome. 2003 May 31 [Updated 2023 Dec 14]. In: Adam MP, Feldman J, Mirzaa GM, et al., editors. GeneReviews® [Internet]. Seattle (WA): University of Washingto@n, Seattle; 1993-2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK1132/, último acesso a 08/10/2025.

C IM 52 - CURVED AND CLOUDED: RADIOGRAPHIC HALLMARKS OF MCCUNE–ALBRIGHT SYNDROME

Camila Sousa1*, Mariana Mendes Rodrigues1*, André Saraiva1

1 Serviço de Reumatologia, Unidade Local de Saúde de Coimbra

**Contribuíram igualmente para o trabalho*



A imagem evidencia achados radiográficos característicos da Síndrome de McCune-Albright. Observa-se no fémur proximal deformidade em *cajado de pastor* (A); na tíbia arqueamento anterior em *sabre* (B); e no crânio espessamento e esclerose óssea com padrão em vidro fosco (C). Estes padrões sugerem fortemente o diagnóstico que poderá ser confirmado em estudo genético realizado em biópsia óssea (1,2).

Referências:

1. Kushchayeva YS, Kushchayev SV, Glushko TY, Collins MT, Boyce AM. Fibrous dysplasia for radiologists: beyond ground glass bone matrix. *Insights Imaging.* 2018;9(6):1035–1056.

2. GeneReviews [Internet]. Seattle (WA): University of Washington, Seattle; 1993–2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK274564/, *último acesso a 8/10/2025.*

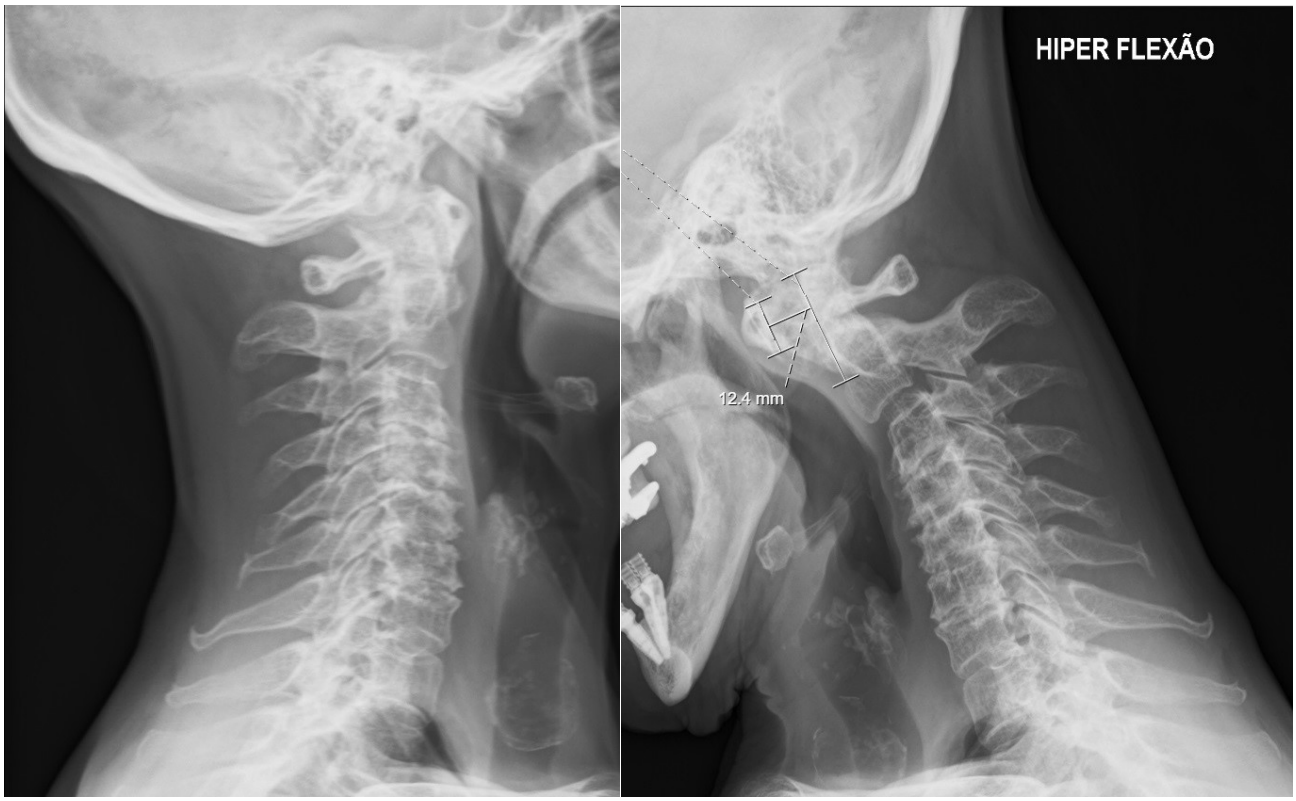
C IM 53 - ATLANTO-AXIAL SUBLUXATION IN RHEUMATOID ARTHRITIS THE ROLE OF DYNAMIC X-RAYS

Duarte Augusto[1], Maria Pontes Ferreira[2], Anita Cunha[2], Susana Almeida[2], Diana Barros[2], José Tavares-Costa[2], Daniela Faria[2], Francisca Guimarães[2]

1. Rheumatology Department, Unidade Local de Saúde da Guarda, Guarda, Portugal

2. Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal

A 62-year-old female with a long-standing history of rheumatoid arthritis presented with new-onset cervical pain. Lateral cervical spine radiography (X-ray) (Fig. 1-A) revealed no changes. Dynamic X-ray with hyperflexion of the neck (Fig. 1-B) revealed atlanto-axial subluxation, with an anterior atlantodental interval of 12.4mm. Magnetic resonance imaging excluded myelopathy. Dynamic X-rays may show atlanto-axial instability when not apparent on neutral imaging.



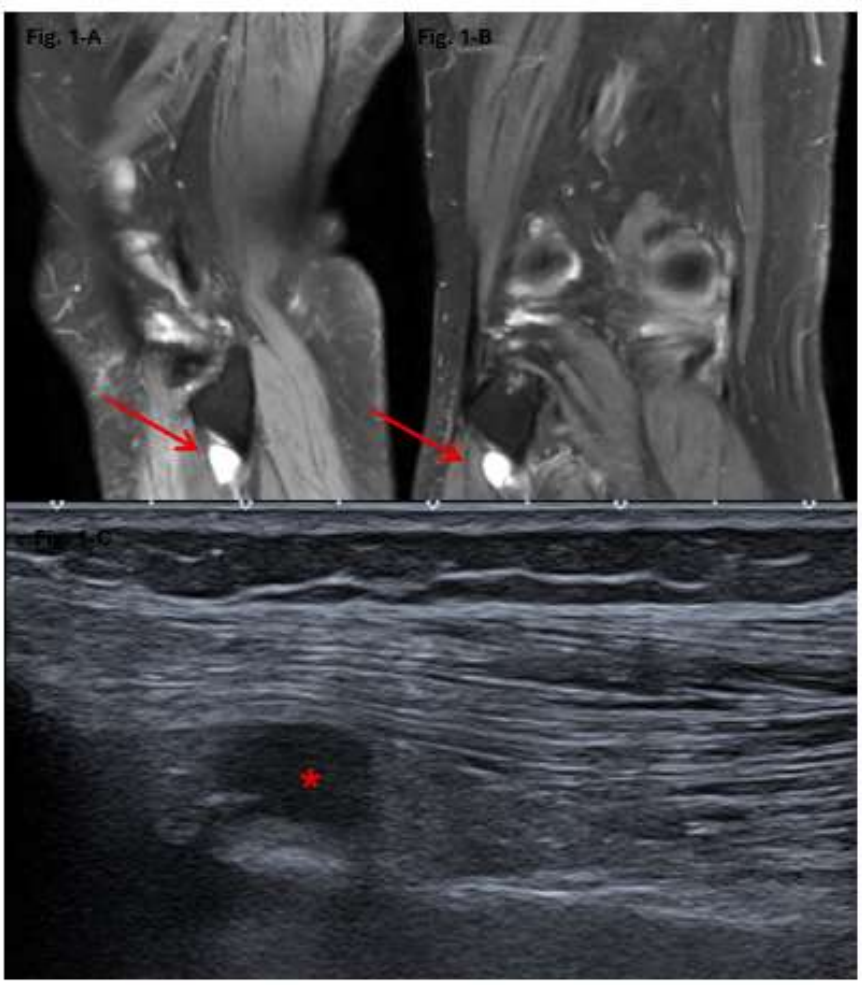
C IM 54 - COMPRESSION NEUROPATHY OF THE COMMON FIBULAR NERVE CAUSED BY A TIBIOFIBULAR JOINT CYST

Duarte Augusto[1], Maria Pontes Ferreira[2], Anita Cunha[2], Susana Almeida[2], Diana Barros[2], José Tavares-Costa[2], Filipa Teixeira[2], Daniela Faria[2]

1. Rheumatology Department, Unidade Local de Saúde da Guarda, Guarda, Portugal

2. Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal

A 67-year-old woman presented with knee pain and distal muscle weakness, with foot drop on examination. Electromyography and MRI (Fig. A-B) performed at presentation demonstrated a subacute lesion of the common fibular nerve and revealed a cystic lesion arising from the proximal tibiofibular joint, likely responsible for fibular nerve palsy. Follow-up ultrasound (Fig. 1C) (*) performed several months later after clinical improvement showed a reduction in cyst size.



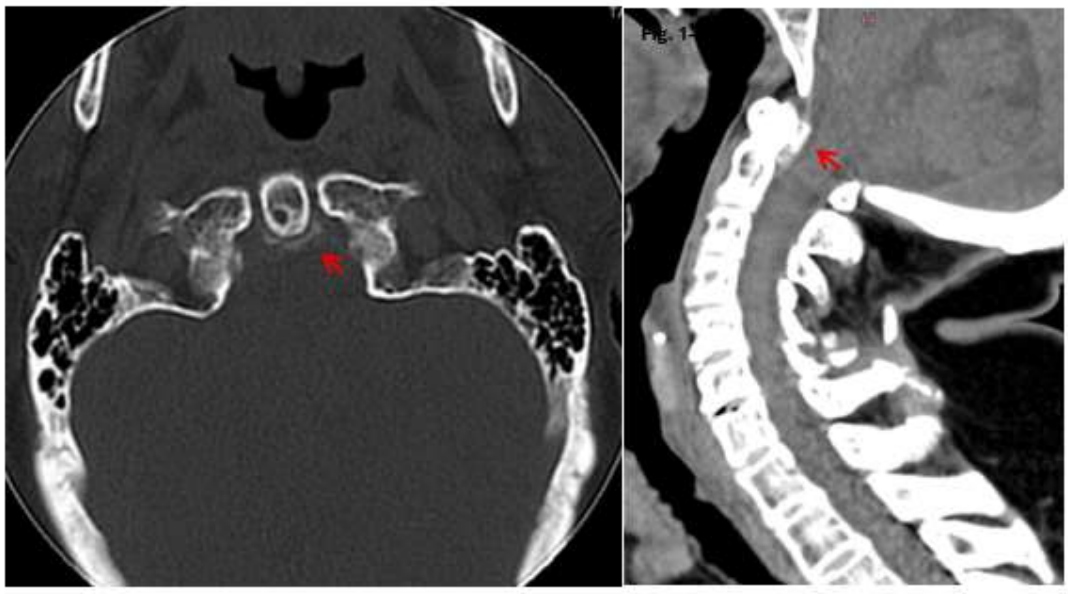
C IM 55 - COLCHICINE SHORTAGE UNMASKING CROWNED DENS SYNDROME

Duarte Augusto[1]; Francisca Magalhães[1] , Filipe C. Santos[1]; Joana F. Ferreira[1,2]; Cláudia C. Vaz [1,2], Nathalie Madeira[1,2]

[1] *Rheumatology Department, Local Health Unit of Guarda, Guarda, Portugal*

[2] *Faculty of Health Sciences, Centro Académico Clínico das Beiras, Covilhã, Portugal*

A 73-year-old woman with a history of peripheral calcium pyrophosphate deposition disease presented with a one-week history of inflammatory cervical pain. She reported interruption of colchicine therapy due to a national shortage. Cervical computed tomography (Fig. 1A-B) showed calcification of the retro-odontoid ligaments. This case stresses the relevance of colchicine in flare prevention and illustrates crowned dens syndrome as a distinctive presentation.



C IM 56 - UNEXPECTED GHOSTS

Diana Belchior Raimundo[1,2], Carolina Ochôa Matos[1,3], Augusto Silva[1,3], João Janeiro[4], Elsa Vieira-Sousa[1,3]

[1] *Serviço de Reumatologia, Unidade Local de Saúde de Santa Maria, Lisboa*

[2] *Serviço de Reumatologia, Unidade Local de Saúde de Loures-Odivelas, Loures*

[3] *Faculdade de Medicina, Universidade de Lisboa, Lisboa*

[4] *Serviço de Imagiologia Geral, Unidade Local de Saúde de Santa Maria, Lisboa*

A routine pelvic radiograph was performed on a 64-year-old man that showed sacroiliac joint fusion - “ghost sign” (Fig 1A). The MRI (Fig 1B-C) showed subchondral irregularities and ankylosis, suggestive of chronic sacroiliitis. A review of all studies, including a CT scan (Fig 1D), supported a diagnosis of diffuse idiopathic skeletal hyperostosis, in an atypical location, not found elsewhere.

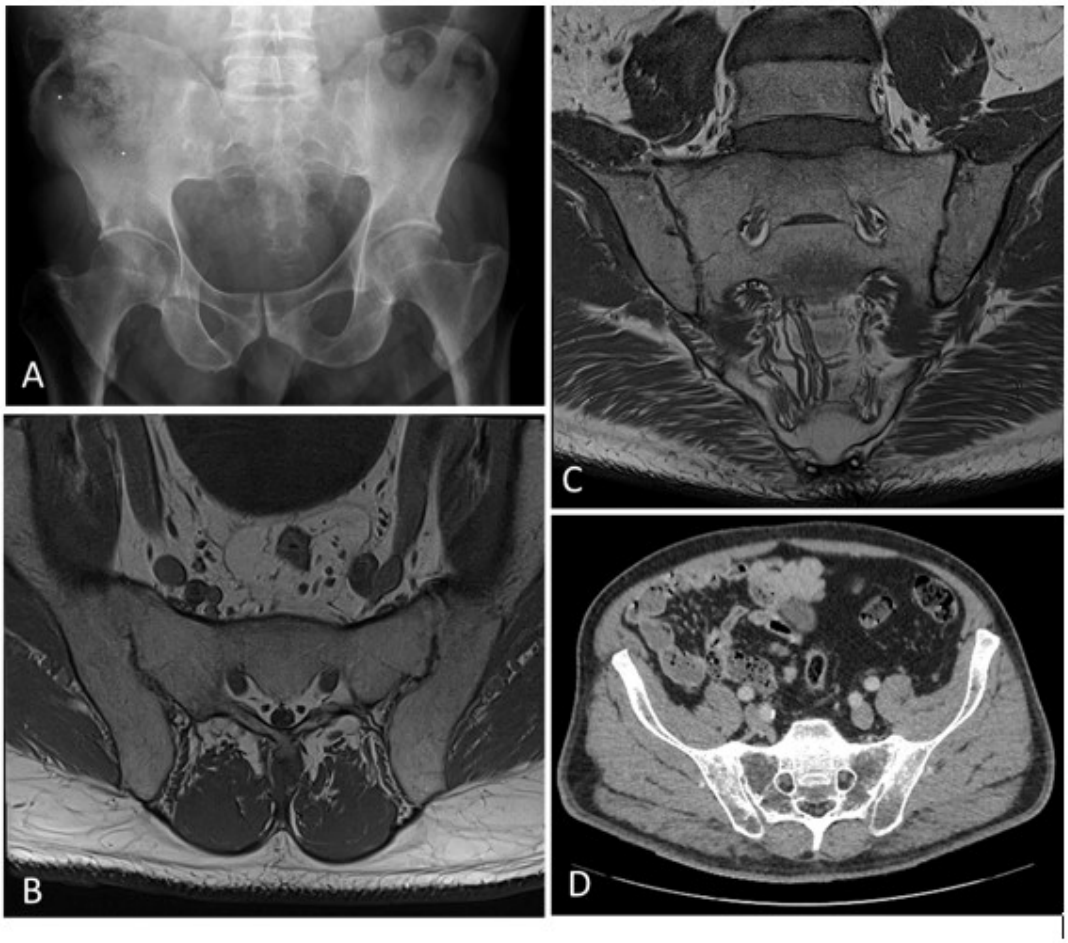
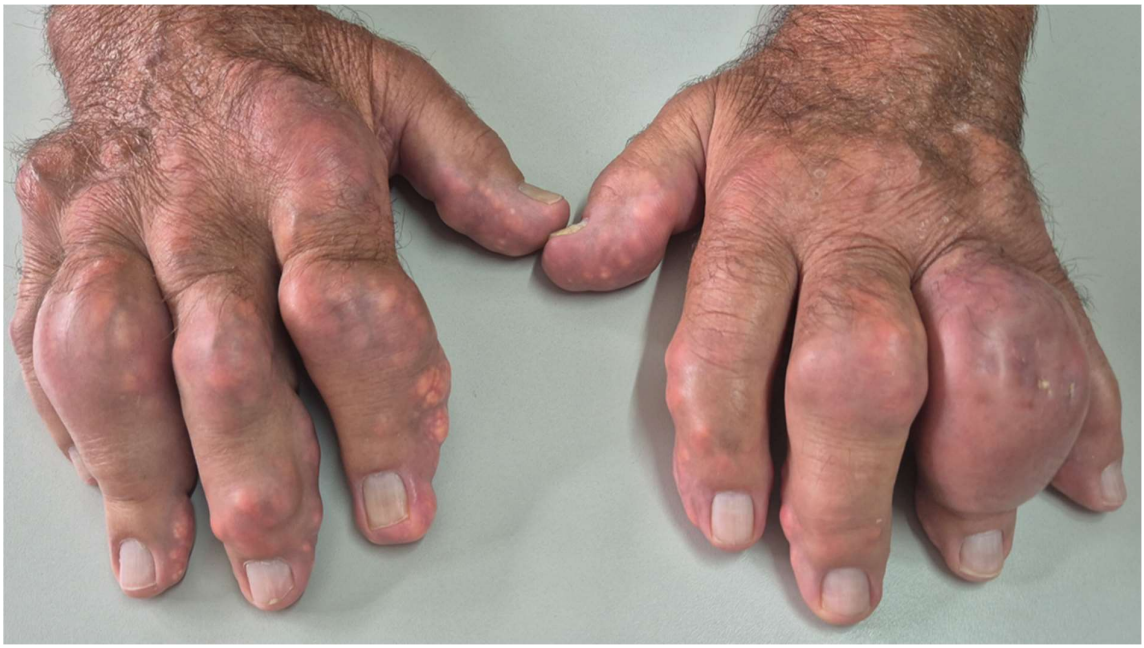


Figure 1. Pelvic imaging of the patient. A: plain radiograph of the pelvis, B & C: MRI images of the sacroiliac joints, T1 sequence with fat suppression in semi-axial (B) and semi-coronal (C) planes; D: abdominopelvic CT scan.

C IM 57 - MÃOS DE DOENTE COM MAIS DE 30 ANOS COM GOTA

Mileta Gomes, Margarida Barroso

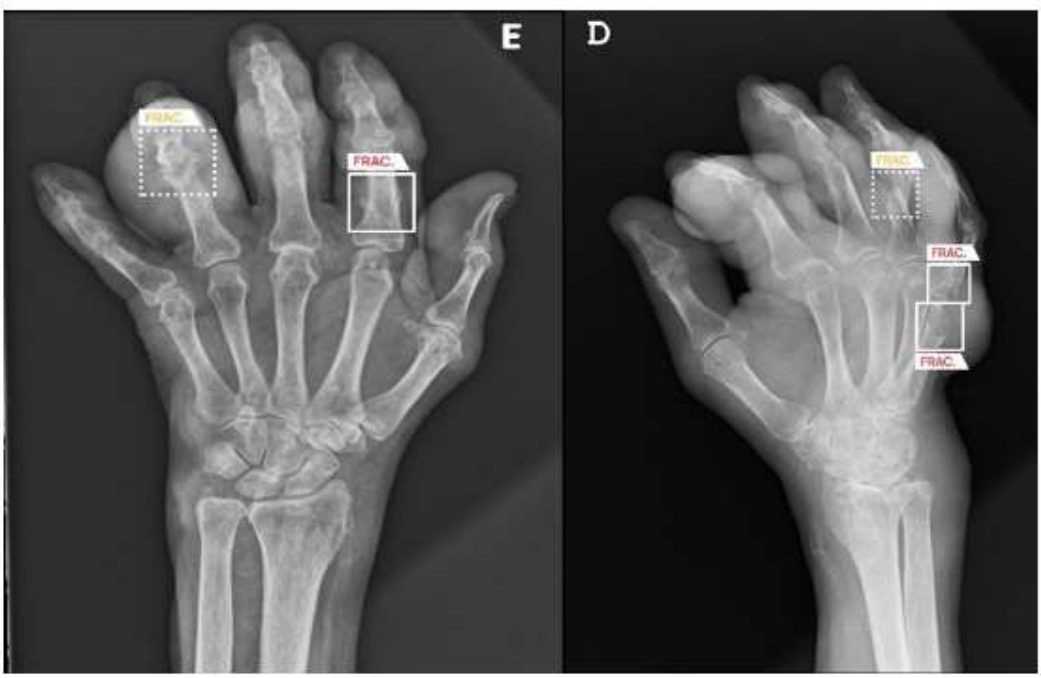
Doente com atingimentos de dezenas de articulações, tofos de grandes dimensões nas mãos de doente com gota tofácea crónica.



C IM 58 - IMAGEM DE RX DE MÃOS DE DOENTE COM GOTA CRÓNICA

Mileta Gomes, Margarida Barroso, Carina Dias

Homem com gota tofácea crónica e com identificação por inteligência articificial de fracturas de diversas falanges e metacarpos em radiografia simples



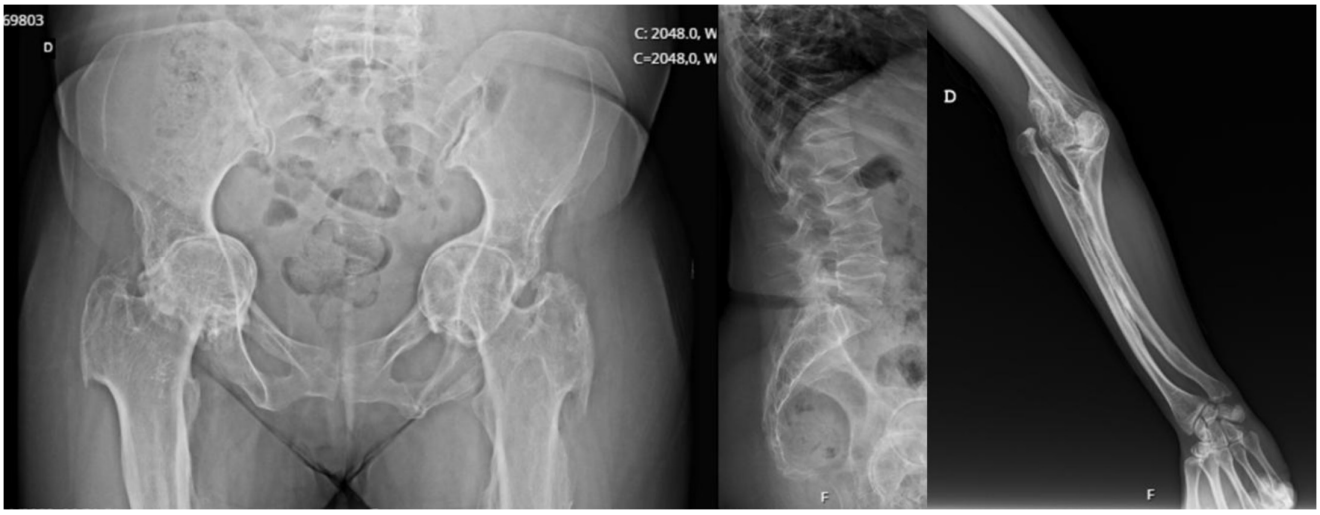
C IM 59 - PROTUSÃO ACETABULAR COMO PISTA RADIOLÓGICA PARA A OSTEOGÉNESE IMPERFEITA (OI) TIPO V

Marina Oliveira1,2, Lúcia Costa1, Maria Rato1

1Serviço de Reumatologia da Unidade Local de Saúde de São João, Porto, Portugal

2Serviço de Reumatologia do Hospital do Divino Espírito Santo, E.P.E.R., Ponta Delgada, Portugal

Mulher de 42 anos referenciada por dor e limitação da mobilidade das articulações coxofemorais. As radiografias mostraram protrusão acetabular bilateral acentuada, vértebras com deformidade em espinha de peixe e calcificação das membranas interósseas dos antebraços. Estes achados levantaram a suspeita de OI Tipo V, um subtipo raro da doença, confirmada posteriormente por análise molecular.

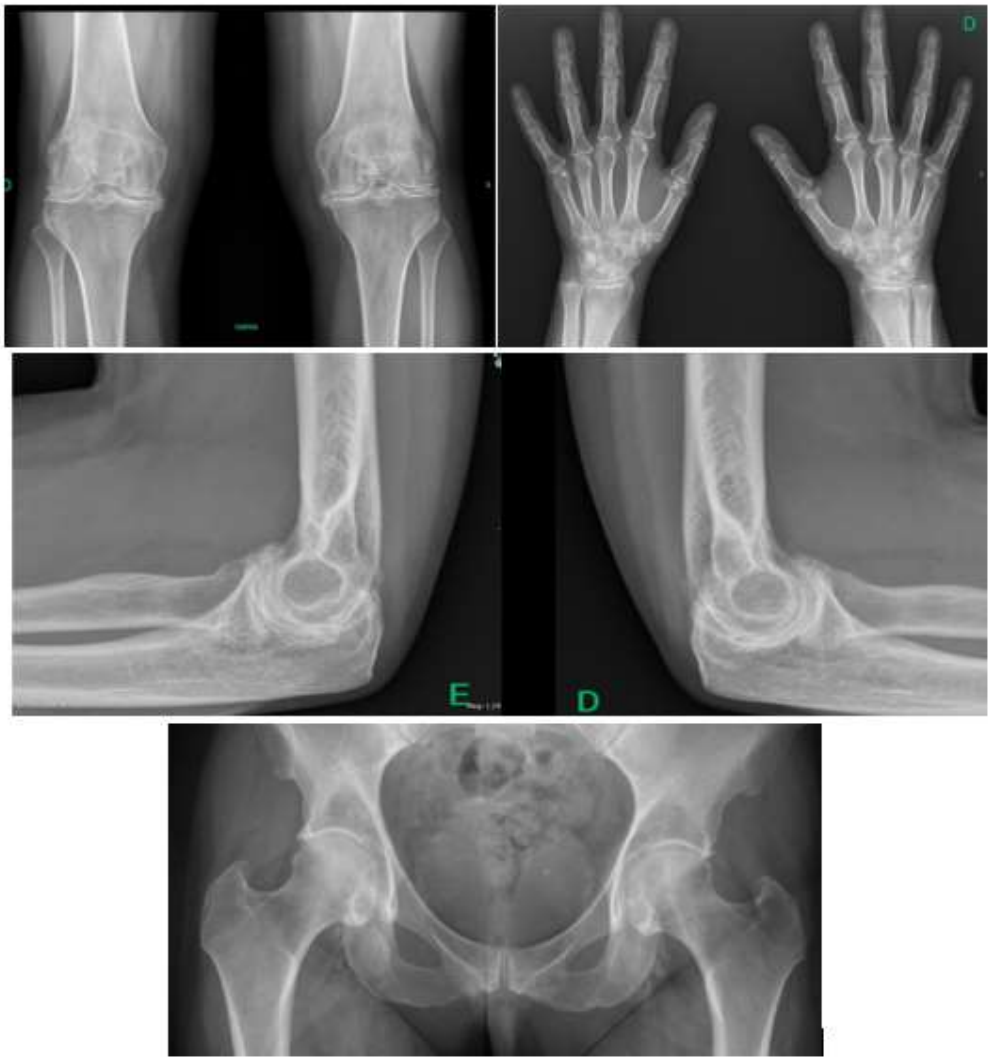


C IM 60 - APRESENTAÇÃO POLIARTICULAR DE DOENÇA DE DEPOSIÇÃO DE CRISTAIS DE PIROFOSFATO DE CÁLCIO

Bárbara Lobão[1], Ana Bispo Leão[1], Leonor Reynolds[1], Susana Fernandes[1], Helena Santos[1,2]

[1]Instituto Português de Reumatologia, [2]Universidade NOVA de Lisboa (Compreensive Health Research Centre)

Género feminino, 56 anos, seguida em Reumatologia por doença de deposição de cristais de pirofosfato de cálcio poliarticular e osteoartrose secundária. As radiografias evidenciam exuberante condrocalcinose envolvendo múltiplas articulações, incluindo punhos, joelhos, cotovelos, coxo-femurais e sínfise púbica.



C IM 61 - ARTROPLASTIA TOTAL DO PUNHO REUMATÓIDE : O ÚLTIMO RECURSO ?

Tiago M. Pereira[1]; Carlota Nóbrega[2]; Fabíola Ferreira[3]

[1]*ULSSM*

[2] *H. Cascais*

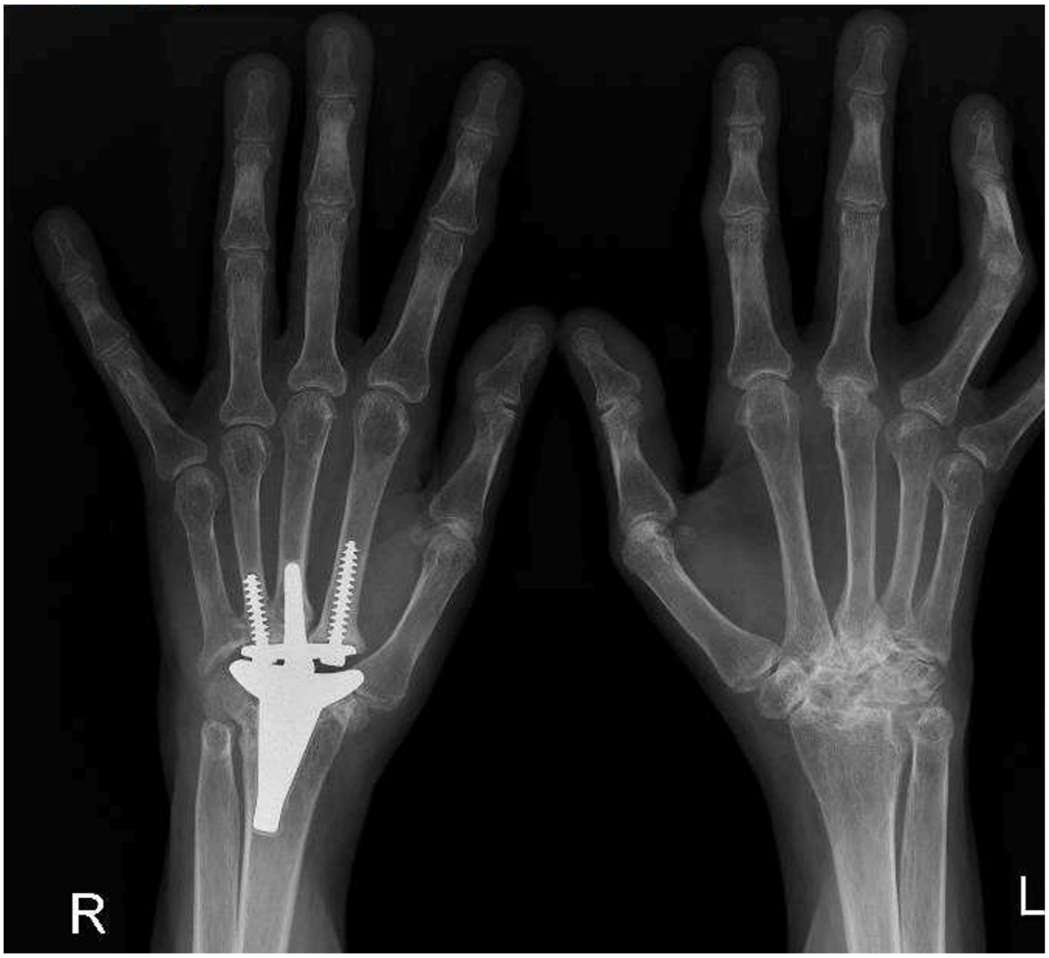
[3] *C.H. Funchal*

Apresentação do caso:

F 54 anos. Dano articular severo nos punhos em contexto de Artrite Reumatóide. Dada a dor e limitação funcional optou-se pelo tratamento cirúrgico – artroplastia total do punho – permitindo mobilidade indolor

Descrição da imagem:

Radiografia AP, de ambos os punhos da paciente, com alterações características da Artrite Reumatóide. Está presente uma prótese total do punho direito



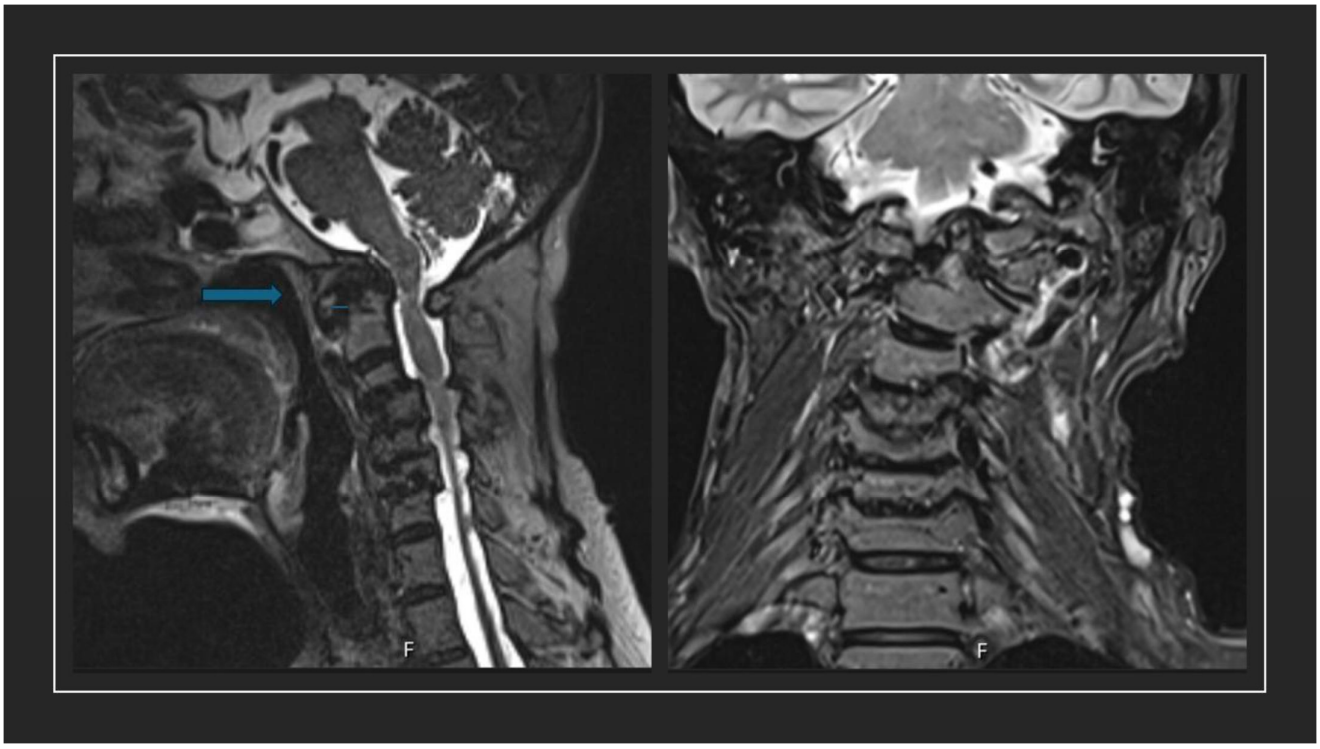
C IM 62 - LOSING HER HEAD OVER RHEUMATOID ARTHRITIS: A CERVICAL PLOT TWIST

Mariana Mendes Rodrigues[1]*, Maria João Cadório[1]*, Inês Serra[2], Fabiana Gouveia[1], Sara Alves Costa[1], Filipa Canhão André[1], João Oliveira[1], Fernando Albuquerque[1], Marcelo Neto[1], André Saraiva[1]

[1]*Serviço de Reumatologia, Unidade Local de Saúde de Coimbra*

[2]*Serviço de Neurocirurgia, Unidade Local de Saúde de Coimbra*

*Contribuíram igualmente para o trabalho



Mulher de 65 anos, diagnóstico de artrite reumatóide seropositiva erosiva há 29 anos. Realizou RM vertebromedular cervical por cefaleias occipitais e TC cervical prévia que revelara luxação C1–C2 com suspeita de invaginação basilar. Exame neurológico normal. A RM confirmou luxação atlanto-axial e impressão basilar com erosões em C1 e C2, sugestivas de envolvimento axial pela doença.

C IM 63 - MUITO ALÉM DA PELE: QUANDO A PSORÍASE É IGNORADA.

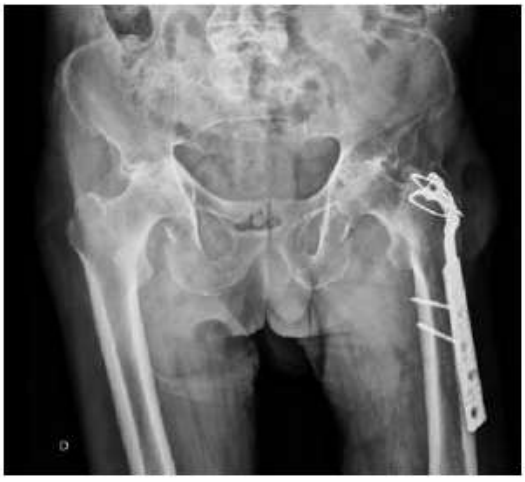
Ana Guida Freitas[1], Pedro Abreu[2,3]

[1]*USF Beira Saúde, ULS Castelo Branco*

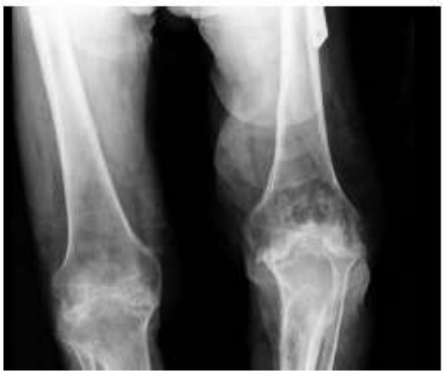
[2]*Unidade de Reumatologia da ULS Castelo Branco*

[3]*Faculdade de Ciências da Saúde, UBI*

Homem, 68 anos, com antecedentes de psoríase sob tratamento tópico desde o seu diagnóstico. Foi internado no Serviço de Cirurgia por agravamento de úlceras, tendo-se constatado um agravamento significativo e fulminante da sua patologia articular identificando-se coxartrose bilateral grau IV (A), sacroileíte grau IV (B) e Gonartrose Grau IV (C), com destruição óssea. Ao exame objetivo apresentava lesões de psoríase no tronco, Hiperqueratose ungueal / micose ungueal, joelho esquerdo em flexão, anquilosado, múltiplas lesões na pele de membro inferior esquerdo (não psoriáticas) com perda de integridade cutânea e no pé esquerdo possível dactilite 2º dedo (D).



A



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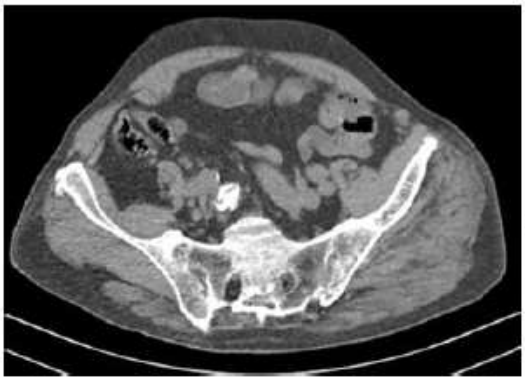

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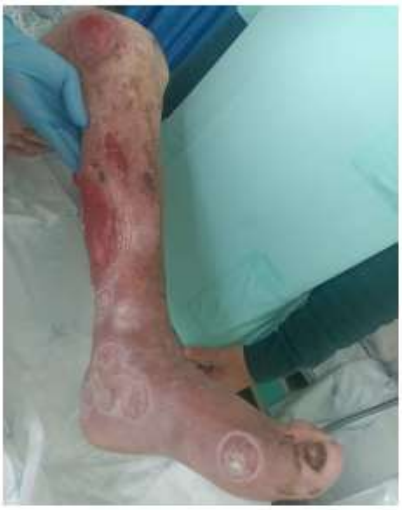

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