Navigate Autoimmunity

1st International Conference

WARSAW, MARCH 14, 2026

ABSTRACTS OF ORAL AND POSTER PRESENTATIONS

The papers have been qualified by the Scientific Committee of the Conference

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Dear Students, Interns, Rheumatologists, and all Doctors Interested in Autoimmunity,

The "Navigate Autoimmunity" Conference is dedicated to you. Its goal is to engage all the interested in a discussion on whether we can navigate autoimmune processes and what challenges face the modern medicine. Thanks to the involvement of many of you, who joined our efforts and of your mentors, the abstracts presented in this supplement were created, reflecting today's difficulties and challenges concerning all fields of medicine, not just rheumatology. Thanks to the invited guests, we were able to delve into various issues pertaining to the domain of autoimmunity – e.g. the genetics of autoimmune diseases, the role of adjuvants and cannabinoids in the development of autoimmunity, and the perception of difficult-to-treat diseases, using the RA as an example. I would like to thank all the Authors, lecturers, reviewers, members of the Scientific Committee, and the Organising Committee for their efforts and enthusiasm, thanks to which we were able to organise the conference and create this supplement. I would also like to thank all the Conference patrons, thanks to whom we were able to recognise and award the authors. Finally, trying to answer the question: "can we navigate autoimmunity?" It can be said that even if we succeed to some extent, the phenomenon of autoimmunity still surprises us in many ways and brings with it new challenges, as well as new discoveries, including drugs that act more and more precisely on the immune system.

I strongly encourage you to take a look at the presented abstracts so to better understand and navigate the issues of autoimmunity.

Maria Maślińska
Editor-in-chief
Reumatologia Journal
Head of the Scientific Committee

Warsaw, March 14, 2026

Dear Colleagues!

Navigare necesse est, states the ancient Latin wisdom. Thus, let us set sail and embark towards the uncharted, rough seas to fill in the blank spots on the map of medical knowledge.

May we discover places appearing distant, threatening, and unrecognised. These lands of mystery during medical studies are often autoimmune diseases – rare, with vague, atypical symptoms.

A successful voyage depends on a skilled crew. For this reason, we are particularly pleased with the interdisciplinary nature of our meeting, as each of us is interested in different areas of medical research to which we want to devote our professional future as healthcare professionals.

As future physicians aspiring to specialise in rheumatology, respiratory medicine, nephrology, cardiology, internal medicine, or general practice, we want to speak a common language, based on one, most important principle: *salus aegroti suprema lex esto.*

On behalf of the Navigate Autoimmunity 2026 Organising Committee, I cordially thank you for taking part in the conference and wish you every success in your professional and academic endeavors. We hope this will also be an opportunity for you to establish new contacts and fruitful cooperation.

Bon voyage!

Paweł Piluch
Head of the Organising Committee

Warsaw, March 14, 2026

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Agnieszka Cudnoch-Jędrzejewska, Michał Kowara

Department of Experimental and Clinical Physiology, Medical University of Warsaw, Poland

Genetic factors in autoimmunity: the common and the uncommon

Carlo Perricone

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Autoimmune diseases arise from a complex interplay between genetic predisposition and environmental influences. As famously summarized by Fathman, autoimmunity can be considered a combination of "genetics and bad luck." Advances in genomic technologies have significantly improved our understanding of the genetic architecture underlying these conditions. Family aggregation, segregation, linkage, and association studies – culminating in genome-wide association studies (GWAS) – have identified numerous susceptibility loci involved in innate immunity, T- and B-cell signaling, apoptosis, autophagy, ubiquitination, and phagocytosis. Many of these genetic variants are shared across multiple autoimmune diseases, highlighting common pathogenic pathways.

Among genetic determinants, the human leukocyte antigen (HLA) region remains the strongest contributor to disease susceptibility, explaining up to 50% of the genetic risk in some disorders. Nevertheless, HLA alleles are neither necessary nor sufficient for disease development, indicating the involvement of additional genetic factors and gene–gene interactions. Structural variations, such as complement gene copy number differences (particularly C4A and C4B), have also been associated with diseases like systemic lupus erythematosus. Moreover, next-generation sequencing and phenome-wide association studies are expanding our capacity to identify rare variants and genotype–phenotype correlations.

Genetic predisposition is further modulated by sex-related differences, endogenous retroelements, and interactions with environmental factors such as smoking and the microbiome. Together, these elements form a multifactorial "mosaic" that explains the heterogeneity, clinical manifestations, and prognostic variability.

Difficult-to-treat rheumatoid arthritis

György Nagy

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Rheumatoid arthritis (RA) remains a complex systemic disease, with a significant subset of patients reaching a "difficult-to-treat" (D2T) state characterized by persistent inflammation despite conventional and biological DMARD therapies. This presentation explores the multi-faceted nature of D2T RA, emphasizing that it is not merely a failure of pharmacological intervention but a complex psycho-neuro-immunological state. Key contributing factors include multi-morbidity (such as cardiovascular and lung disease), non-inflammatory conditions like fibromyalgia and depression, and behavioral factors including medication non-adherence and poor coping strategies. Recent research highlights the critical role of central nervous system (CNS) involvement, where altered brain connectivity and "morbid adaptation" – marked by hopelessness and cognitive rigidity – contribute to poor outcomes. Advanced imaging and transcriptomic analyses suggest that controlling peripheral inflammation alone is often insufficient, necessitating the consideration of central mechanisms.

Ultimately, the management of D2T RA requires a shift toward a multidisciplinary approach and personalized, patient-tailored treatment strategies.

Why we develop autoimmunity

Yehuda Shoenfeld

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Autoimmune syndrome induced by adjuvants

Maria Maślińska

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ABSTRACTS ACCEPTED FOR ORAL PRESENTATION

Online oral session

News from the world – international session

N_01

DOI: https://doi.org/10.5114/reum/219207

Selective immunoglobulin A deficiency associated with autoimmune diseases

Lilla Andó, Szilvia Szamosi, Anita Pusztai, Monika Bodoki, Dóra Csige, Gabriella Szűcs

Department of Rheumatology, Faculty of Medicine, University of Debrecen, Hungary

Key words: IgA deficiency, autoimmunity, common variable immune deficiency

Introduction: Selective immunoglobulin A deficiency (SIgAD) is the most common primary immunodeficiency. Although most patients remain asymptomatic, SIgAD is frequently associated with autoimmune and allergic diseases, while infections occur considerably less often than in common variable immunodeficiency (CVID).

The aim of the study was to examine the prevalence of SIgAD among patients with inflammatory and autoimmune diseases, to characterise their infectious risk and immunological abnormalities, and to compare their clinical and laboratory parameters with those observed in CVID. The SIgAD was diagnosed when the serum sensitive IgA level was 0.7 g/l.

Material and methods: Among patients treated for autoimmune/inflammatory conditions at the Rheumatology and Immunology Clinic of the University of Debrecen, SIgAD was confirmed in 33 patients (27 women, 6 men; average age 54.15 ±13.4 years, and average sensitive IgA 0.31 g/l). The CVID (defined as IgG 6 g/l with low IgA, IgM levels) was diagnosed in 10 patients (8 women, 2 men; average age 60.6 ±15.6 years).

Results: In the SIgAD group, recurrent non-severe infections (mainly upper respiratory and urinary tract infections) occurred in 11 patients (33.3%). Allergic diseases (asthma, pollen allergy, atopic dermatitis) were present in 19 cases (57.6%), and concomitant malignancy in 4 patients (12.1%). The underlying autoimmune diseases included rheumatoid arthritis (n = 12), systemic lupus erythematosus or rheumatoid arthritis–systemic lupus erythematosus overlap syndrome (n = 7), undifferentiated connective tissue disease (n = 8), spondyloarthropathy (n = 2), myositis (n = 1), polymyalgia rheumatica (n = 2), and scleromyxedema (n = 1). Allergic and celiac diseases occurred significantly more frequently in SIgAD than in CVID, whereas malignant diseases were significantly less common. Immunophenotypic analysis revealed that the proportion of CD8+ naïve T cells (40.44%) was significantly higher in SIgAD compared to CVID (34.4%, p = 0.05). The proportion of CD19 naïve B cells was significantly lower and showed a strong positive correlation with decreased IgA levels (R = 0.52, p = 0.003). In contrast, CD19+ IgM memory B cells were significantly increased and were associated with the more frequent occurrence of allergic and neoplastic diseases.

Discussion: Patients with SIgAD exhibit significant abnormalities in both humoral and cellular immune responses.

Conclusions: Determining the subtypes of T- and B-cells may contribute to a better understanding of disease pathogenesis and may help identify patients at increased risk for additional comorbidities.

N_02 DOI: https://doi.org/10.5114/reum/219193

Microplastics as emerging environmental cofactors in autoimmune rheumatic diseases

Iryna Dovbnia, Bohdana Doskaliuk

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Key words: microplastics, autoimmune rheumatic diseases, immunopathogenic mechanisms

Introduction: Microplastics (MPs) are ubiquitous environmental particles with emerging immunotoxic and pro-inflammatory potential. Their persistence in water, food chains and air leads to continuous low-dose human exposure. Increasing evidence suggests MPs may act as environmental cofactors capable of triggering immune dysregulation, oxidative stress and chronic low-grade inflammation. These mechanisms raise concern that MPs could contribute to the initiation or exacerbation of autoimmune inflammation.

The study aims to synthesise peer-reviewed evidence on MPs exposure and its potential role in the development and worsening of autoimmune pathology, with a focus on rheumatic diseases and their underlying immunopathogenic mechanisms.

Material and methods: Narrative review based on structured searches of PubMed/MEDLINE, Scopus and Web of Science, enhanced by manual reference screening. Experimental, translational and clinical studies evaluating MPs exposure in relation to rheumatic outcomes were qualitatively integrated. Exclusion criteria included non-peer-reviewed publications, conference abstracts, and studies lacking immune or clinical outcome assessment.

Results: In murine models, oral MPs exposure induced lupus-like manifestations in C57BL/6 mice and aggravated spontaneous lupus in MRL/lpr mice. These effects were associated with expansion of splenic double-negative T cells and plasma cells, increased anti-dsDNA and anti-nuclear antibodies titres, elevated interleukin-6 and tumour necrosis factor levels, and renal injury with proteomic signatures consistent with lupus-associated pathways and complement-mediated damage. In rheumatoid arthritis models, MPs were internalised by fibroblast-like synoviocytes, enhancing proliferation, migration and invasion, increasing inflammatory mediator release, and promoting cartilage damage.

Discussion: Across experimental systems, MPs appear to function as pro-inflammatory adjuvant-like particles, activating stromal effector cells, amplifying cytokine and protease cascades, and potentially enhancing complement-driven tissue injury. However, human causality remains uncertain due to heterogeneity in exposure assessment, particle composition and size variability, and predominance of high-dose experimental models.

Conclusions: Current evidence, largely preclinical, supports a plausible role of MPs in promoting initiation and progression of autoimmune rheumatic phenotypes. Standardised exposure assessment, biomonitoring strategies and prospective human studies are required to define attributable risk and identify preventive public health interventions.

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Botulinum toxin for microstomia in systemic sclerosis: a literature review

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Key words: systemic sclerosis, scleroderma, microstomia, botulinum toxin, oral aperture

Introduction: Systemic sclerosis (SSc) often causes perioral fibrosis leading to microstomia, which impairs speech, nutrition, and oral hygiene. Conventional stretching, physiotherapy, and surgery provide limited and inconsistent benefit. Botulinum toxin (BoNT) has been proposed to relax perioral muscles and improve mouth opening.

The study aims to review published clinical trials, prospective studies, case series, and case reports on the use of BoNT for SSc-related microstomia, summarise, and to critically appraise the available evidence regarding its efficacy and safety.

Material and methods: A literature review was conducted in major biomedical databases and a clinical trial registry (inception – 2023). Eligible reports included clinical trials, prospective studies, case series, and case reports evaluating BoNT injections with outcomes related to mouth opening and/or oral function in patients with SSc.

Results: Of 677 records identified, four studies met the predefined inclusion criteria, namely: studies involving patients with systemic sclerosis; administration of BoNT as an intervention; evaluation of outcomes related to microstomia or oral aperture; and original clinical study design (clinical trials, prospective studies, or case series). These comprised one small prospective interventional study, one registered clinical trial with unpublished results, one case series, and one case report. Across studies, perioral BoNT type A (most commonly 16–20 units) was associated with short-term increases in maximal interincisal distance and improvements in patient-reported oral function. Reported adverse events were minimal and transient, with no serious complications described.

Discussion: The evidence suggests a consistent signal of short-term benefit, but certainly is low due to small samples, heterogeneity, and limited controlled data. Optimal formulation, dosage, injection mapping, and retreatment intervals remain unclear, and durability beyond about 3 months is insufficiently studied.

Conclusion: Botulinum toxin appears to be a promising adjunctive option for SSc-related microstomia; however, adequately powered randomised trials with standardised outcomes are needed before routine clinical use can be recommended.

N_04 DOI: https://doi.org/10.5114/reum/219195

Functional impairment in patients with systemic sclerosis: relation to disease peculiarities and pain

Yaroslava Krasienko, Tetiana Karasevska

Department of Internal Medicine with a course of Cardiology and Rheumatology, Bogomolets National Medical University, Kyiv, Ukraine

Key words: systemic sclerosis, pain syndrome, mRSS, HAQ-DI

Introduction: Systemic sclerosis (SSc) is characterised by microvascular abnormalities affecting multiple organs. Pain is a prevalent and debilitating symptom of SSc that can have a significant impact on patients' quality of life and physical function.

The study aims to evaluate functional impairment in patients with SSc and to investigate their association with disease peculiarities and pain.

Material and methods: A retrospective analysis of medical records from the European Alliance of Associations for Rheumatology (EULAR) Research Voucher grant "The bone and muscle state in patients with systemic sclerosis" included 32 patients with SSc (mean age: 57.1 ±12.5 years; body mass index: 24.6 ±4.6 kg/m²; disease duration 8.4 ±5.9 years). Functional status was assessed using HAQ-DI, and patients were stratified into 3 groups: G1 minimal (score 0–1), G2 moderate (1–2), and G3 severe (2–3) disability. We analysed pain in patients using the Visual Analogue Scale (VAS) and the VAS physician global score. A hand-grip dynamometer assessed muscle strength. The analysis included laboratory markers (C-reactive protein, erythrocyte sedimentation rate, interleukin-6), skin involvement evaluated by modified Rodnan skin score (mRSS), the presence of digital ulcers (DU) and interstitial lung disease (ILD). Statistical analysis was performed on the Med-Stat software.

Results: Patients in G1 exhibited significantly lower pain scores than those in G2, as measured by both the patient VAS (30.1 ±18.1 vs. 59.0 ±18.5; $p = 0.05$) and the physician VAS (26.9 ±16.0 vs. 50.5 ±19.2; $p = 0.05$). As far as it was in G3: VAS patient (63.3 ±15.1 vs. 30.1 ±18.1; $p = 0.05$), and VAS physician (69.3 ±34.4 vs. 26.9 ±16.0; $p = 0.05$). Among G3 patients in comparison with G1, significantly higher mRSS scores (27.3 ±7.9 vs. 16.7 ±7.8; $p = 0.05$) and reduced hand grip strength (6.33 ±6.1 vs. 16.7 ±7.8; $p = 0.05$) were demonstrated. No significant associations were observed between HAQ-DI and age, disease duration, laboratory parameters, DU or ILD.

Discussion: These findings highlight the importance of a thorough evaluation of functional limitations and pain in SSc. Our observations emphasise that functional impairments are closely related to patients' reported pain and the severity of the disease as assessed by physicians. In our study, decreased hand strength and skin involvement were found to be risk factors for functional impairment.

Conclusions: In patients with SSc, functional impairment correlates with pain intensity, muscle strength, extent of skin involvement, and global disease activity assessed by physicians.

N_05

DOI: https://doi.org/10.5114/reum/219206

Investigation of the molecular background of early skeletal involvement in systemic sclerosis by serum osteoimmune mediators

Sára Lipták-Lukácsik, Dóra Csige, Levente Bodoki

Division of Rheumatology, Department of Internal Medicine, University of Debrecen, Hungary

Key words: osteoimmunology, bone markers, SSc, biomarkers

Introduction: Systemic sclerosis (SSc) is a complex, autoimmune disease associated with reduced bone density and increased fragility.

The study aims to investigate the molecular background of early bone alterations in SSc using dual X-ray absorptiometry (DXA)-derived bone mineral density (BMD) parameters and their associations with osteoimmunological biomarkers.

Material and methods: The prospective cross-sectional study included 40 women with SSc of relatively short disease duration (≤ 7 years from diagnosis) and 45 age-, sex- and bone status-matched non-immune controls at the Department of Rheumatology and Immunology, Clinical Centre, University of Debrecen, Hungary. Bone status was assessed by DXA at the lumbar spine and femoral neck. Routine bone and mineral metabolism markers were measured, as well as a targeted panel of osteoimmunological mediators (osteoprotegerin [OPG], osteopontin [OPN], platelet-derived growth factor-BB [PDGF-BB], tartrate-resistance acid-phosphatase [ACP5], receptor activator of NF-κB ligand [RANKL], tumour necrosis factor [TNF], interleukin-6 [IL-6], IL-1β, Dickkopf-1 [DKK-1]), which were quantified by multiplex immunoassay. Ten-year probabilities of major osteoporotic fracture and hip fracture were calculated using the FRAX® algorithm.

Results: The DXA-defined bone status distribution did not differ between SSc patients and controls, yet FRAX estimates were numerically higher in SSc. Serum OC levels were significantly lower, while 25-hydroxy vitamin D levels were significantly higher in SSc compared to non-SSc group, reflecting more frequent supplementation (Mann-Whitney U test, $p = 0,05$). Whereas SSc exhibited a distinct osteoimmune serum profile, characterised by significantly elevated OPG, ACP5, RANKL, IL-6, IL-1β, DKK-1, and an increased RANKL/OPG ratio, remaining significant after false discovery rate correction. Subgroup analyses across osteoporosis, osteopenia, and normal bone status showed consistent elevation of DKK-1 in SSc patients, with additional subgroup-specific differences of OPG, ACP5, RANKL, IL-6, and IL-1β levels, which were preferentially featured with osteoporosis and osteopenia.

Discussion: Early diagnosis and molecular characterisation of bone metabolism alterations may be crucial for optimising therapeutic strategies and potentially reducing the extent of bone loss in patients with SSc.

Conclusion: In women with relatively early SSc, a distinct osteoimmunological biomarker profile differentiated SSc from controls and was associated with DXA-derived BMD parameters, supporting a link between osteoimmunological alterations and skeletal involvement in the first years of SSc.

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Follow-up of three Janus kinase inhibitors in rheumatoid arthritis patients

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Key words: rheumatoid arthritis, follow-up study, JAK inhibitors

Introduction: Targeted synthetic disease-modifying drugs (tsDMARDs), Janus kinase (JAK) inhibitors, have become an important treatment option in different indications, including rheumatoid arthritis (RA). The patients are also receptive to these drugs due to the route of administration.

The study aimed to conduct an observational follow-up study monitoring patients receiving JAK inhibitor therapy to assess the potential correlations with clinical parameters and their change over specific time points. The primary objective was to characterise the study population, while the secondary objective was to assess changes in studied parameters over time.

Material and methods: Fifty-five RA patients treated with tofacitinib, baricitinib, or upadacitinib at the Department of Rheumatology and Immunology were included in a one-year follow-up study. Clinical assessments were carried out at baseline (T0), after 6 (T6) and 12 months (T12). We evaluated Disease Activity Score 28-joints and the inflammatory markers, C-reactive protein (CRP) and erythrocyte sedimentation rate. Patients' history and discontinuation before T12 were also recorded, together with the reason for stopping treatment. Statistical analysis was carried out, where *p*-values 0.05 were considered significant.

Results: Thirty-five out of 55 RA patients completed the 12-month follow-up study, while 20 patients discontinued due to ineffectiveness or side effects. The JAK inhibitor therapy led to a significant reduction in disease activity by month 6 ($p = 0.001$) and month 12 ($p = 0.001$), and CRP by T6 ($p = 0.016$). Glucocorticosteroid need showed a significant decline by month 6 compared with baseline ($p = 0.006$). Looking at each therapy, disease activity was also found to be significantly reduced; CRP levels did not show a significant reduction, which could be explained by the smaller sample size (13 tofacitinib, 11 baricitinib, 11 upadacitinib).

Discussion: The study provided further evidence for the efficacy of the agents, coming from a real-world experience, clinical setting, where, although contraindications, patient history and local protocol are considered and followed, there are no strict inclusion criteria when starting therapy like in clinical trials. Some patients stopped treatment because of side effects, which were in line with the known safety profiles of these drugs.

Conclusions: Overall, our findings support the effectiveness of JAK inhibitors in routine practice and generally reflect what has been seen in earlier clinical trials.

N_07

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Personality alterations in systemic lupus erythematosus identified by psychiatric and psychological assessment

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Key words: SLE, cognitive dysfunction, neuropsychiatric lupus, DSM-5 personality disorders

Introduction: Systemic lupus erythematosus (SLE) is a complex, multisystem autoimmune disorder that is frequently associated with psychological and cognitive disturbances. Depression, fatigue, and anxiety occur considerably more often among individuals with SLE than in the general population; however, the mechanisms underlying these manifestations remain insufficiently understood.

This study aims to examine the personality profiles of patients with SLE using a comprehensive, multidimensional assessment that integrates clinical, psychological, and biological perspectives.

Material and methods: The current cohort consists of 11 SLE participants and 10 age- and sex-matched healthy controls. Participants went through a detailed assessment process that included a clinical evaluation, psychological assessment, and a psychiatric examination, as illustrated in Figure 1.

Results: Psychological and psychiatric assessments suggest that mental health changes in SLE are heterogeneous and multidimensional, frequently overlapping with neuropsychiatric manifestations of the disease and thereby complicating differential diagnosis. Psychological evaluation identified emotional restriction, difficulties with interpersonal attachment, increased level of anxiety and depressive symptomatology, self-destructive ideations, and indications of delayed psychological development across the SLE group. Furthermore, DSM-5-based psychiatric diagnoses were established for all patients with SLE. Within the spectrum of DSM-5 personality disorders, traits aligning predominantly with Cluster B features – particularly schizoid patterns – were more evident in a subset of patients. The distribution of these psychiatric diagnoses is illustrated in Figure 2. Regarding cognitive functioning, "brain fog" emerged as the most prominent complaint, associated

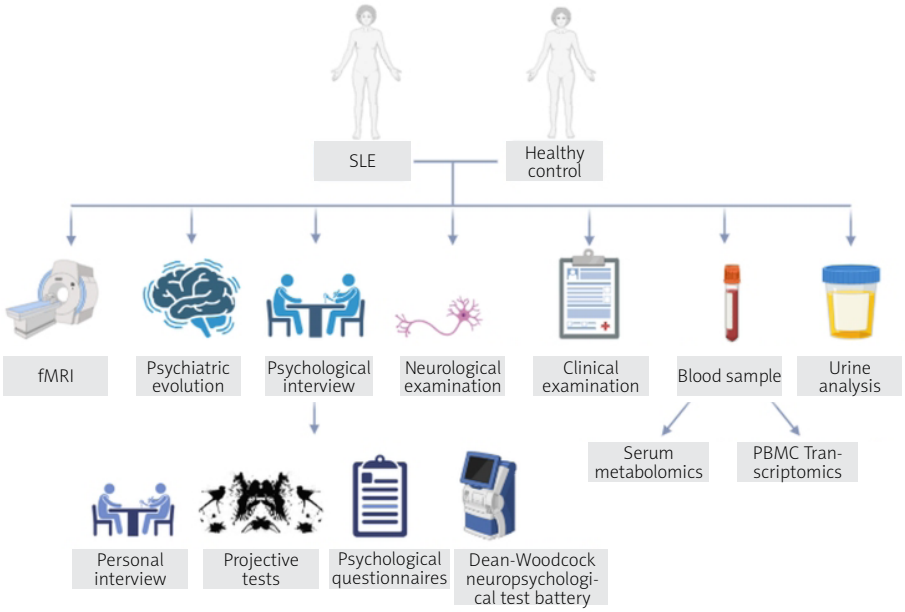

Fig. 1. Study design. Participants with systemic lupus erythematosus (SLE) and healthy controls were enrolled and underwent a comprehensive evaluation including neuroimaging (fMRI), psychiatric and psychological assessments, and neurological and clinical examinations. Psychological evaluation involved personal interviews, projective tests, and standardized questionnaires, complemented by the Dean-Woodcock neuropsychological test battery. In parallel, blood samples were collected for molecular analyses, including serum metabolomics and PBMC transcriptomic profiling.

with subjective memory difficulties, diminished attention, and slower information processing.

Discussion: The findings suggest that psychological functioning in SLE is complex and influenced by interacting biological and psychosocial factors. The observed personality features and psychiatric comorbidities may both reflect and contribute to the challenges of living with a chronic autoimmune disease.

Conclusions: Personality patterns observed in SLE appear diverse and likely arise from multifactorial interactions involving autoimmune activity, psychosocial stressors, and neuropsychiatric symptoms. Improved recognition of personality patterns specific to SLE may facilitate more precisely targeted psychological interventions and enable personalized long-term management strategies for affected individuals.

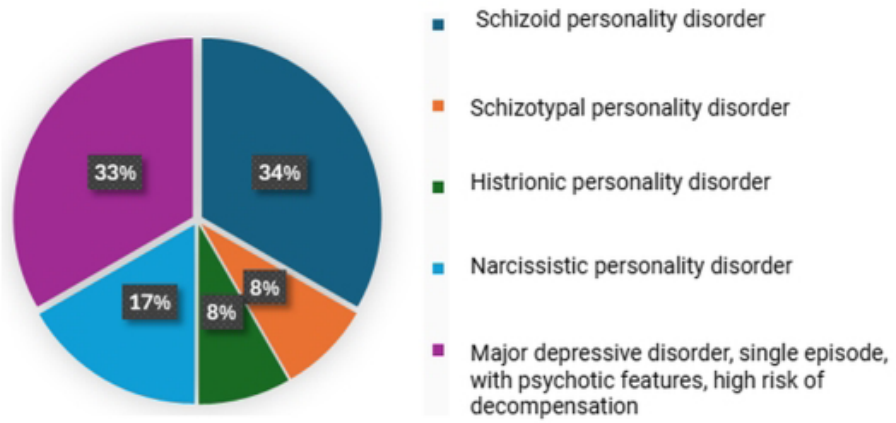

Fig. 2. Distribution of psychiatric diagnoses among patients with SLE. The pie chart depicts the prevalence of DSM-5-based psychiatric diagnoses identified in the SLE cohort. Schizoid personality disorder was the most frequent (34%), followed by major depressive disorder, single episode, with psychotic features and high risk of decompensation (33%), narcissistic personality disorder (17%), and schizotypal and histrionic personality disorders (8%).

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Preliminary results of real-world whole-blood hydroxychloroquine level monitoring in systemic lupus erythematosus: low target attainment and key pharmacokinetic determinants

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Key words: hydroxychloroquine, systemic lupus erythematosus (SLE), therapeutic drug monitoring (TDM), medication adherence, blood concentration

Introduction: Hydroxychloroquine (HCQ) is a cornerstone therapy in systemic lupus erythematosus (SLE), yet treatment benefit depends on adequate and sustained drug exposure. Whole-blood HCQ level measurement may provide an objective approach to detect subtherapeutic exposure and could potentially support individualised clinical decision-making.

The study aims to determine the proportion of SLE patients reaching a literature-defined therapeutic HCQ range and to identify independent pharmacokinetic determinants of measured HCQ concentrations in routine care.

Material and methods: In a cross-sectional observational study, consecutive adult SLE outpatients were recruited at Semmelweis University between November 1 and December 17, 2025. Whole-blood HCQ levels were quantified using a validated liquid chromatography coupled to tandem mass spectrometry (LC–MS/MS) method. Of 35 enrolled patients, 32 were included in the final analysis. Target attainment was defined as 750–1,200 ng/ml. A one-sided exact binomial test evaluated whether ≥ 50% of patients were within range. Multivariable linear regression modelled HCQ levels using weight-adjusted daily dose, estimated glomerular filtration rate (eGFR), and time from last intake to blood draw.

Results: Only 6/32 patients (18.8%) achieved the 750–1,200 ng/ml therapeutic range; this proportion was significantly below 50% ($p = 0.00027$; exact 95% CI: 0.000–0.337). In the regression model ($F = 8.98$, $p = 0.001$; adjusted $R^2 = 0.51$), higher eGFR was independently associated with lower HCQ levels ($\beta = -13.49$; 95% CI: from −23.58 to −3.40; $p = 0.011$), and longer time since last intake was also associated with lower levels ($\beta = -14.79$ per hour; 95% CI: from −26.80 to −2.78; $p = 0.018$). Weight-adjusted dose showed a positive but non-significant association ($\beta = 64.64$; $p = 0.237$).

Discussion: These preliminary findings suggest that subtherapeutic HCQ exposure is common in routine care despite ongoing treatment. The results highlight the limitations of dose-based prescribing, while emphasising the clinical relevance of pharmacokinetic factors such as renal function.

Conclusion: In this preliminary real-world cohort, most SLE patients did not achieve the predefined therapeutic HCQ exposure range despite ongoing treatment. These findings suggest that dose-based prescribing alone may be insufficient to ensure adequate drug exposure. Whole-blood HCQ monitoring could help identify patients with subtherapeutic levels and potentially support a more individualised therapeutic approach by distinguishing nonadherence from pharmacokinetic variability.

Oral session 1

Autoimmunity of the respiratory system

O_01 DOI: https://doi.org/10.5114/reum/219192

Ocrelizumab-associated organising pneumonia in a patient with multiple sclerosis: case report

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Key words: ocrelizumab, multiple sclerosis, organising pneumonia, drug-induced lung disease

Introduction: Ocrelizumab is a humanised anti-CD20 monoclonal antibody used in the treatment of multiple sclerosis (MS) by targeting B-cells to reduce autoimmune demyelination. It is generally well tolerated, although rare pulmonary toxicities, including interstitial lung disease and organising pneumonia (OP) have been reported. Migratory ground-glass opacities and consolidations are common radiological features in OP, with systemic symptoms such as fever. Drug-induced OP should be suspected when infectious and autoimmune causes are excluded, and radiological abnormalities improve after drug withdrawal.

Case description: A 51-year-old man with MS treated with ocrelizumab presented with recurrent fever up to 39°C since 8 weeks and night sweats, without cough nor dyspnea. Chest computed tomography scan revealed diffuse ground-glass opacities mainly in the upper lobes. Fever recurred despite antibiotic treatment and low-dose glucocorticosteroids. Laboratory investigations revealed elevated C-reactive protein with procalcitonin level within normal range, normocytic anaemia, and elevated liver enzymes. High-resolution computed tomography (HRCT) demonstrated migratory peripheral ground-glass opacities with consolidations, reverse halo sign, and air bronchograms, which may be compatible with OP. Autoimmune serology (antinuclear antibodies, anti-neutrophilic cytoplasmic autoantibodies, anti-cyclic citrullinated peptide antibodies) and viral tests (cytomegalovirus, Epstein-Barr virus) were negative. Pulmonary function tests found normal lung volumes and low diffusing capacity of lung for carbon monoxide (DLCO, 55% predicted). Bronchoalveolar lavage cultures excluded bacterial, viral, and fungal infections. Ocrelizumab therapy was stopped. Three months after cessation on ocrelizumab, HRCT confirmed almost complete resolution of pulmonary infiltrates and improvement in DLCO (67% predicted). No different etiological mechanism was detected, and MS remained clinically stable.

Conclusions: This case highlights the importance of maintaining a high index of suspicion for drug-induced pulmonary toxicity in patients receiving anti-CD20. Although ocrelizumab is generally well tolerated, awareness of rare but potentially reversible complications such as OP is essential to ensure prompt diagnosis, appropriate management, and favourable clinical outcomes.

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Clinical, functional, and microvascular predictors of interstitial lung disease in systemic sclerosis

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Key words: systemic sclerosis, interstitial lung disease, nailfold capillaroscopy

Introduction: Systemic sclerosis (SSc) is a chronic autoimmune disease characterised by immune dysregulation, vasculopathy, and progressive fibrosis. Systemic sclerosis-associated interstitial lung disease (SSc-ILD) is a leading cause of morbidity and mortality. Early identification of clinical and microvascular markers linked to ILD is crucial for risk stratification and timely intervention.

The study aims to evaluate risk factors associated with the presence of SSc-ILD through comparative analysis of clinical, functional, serological, echocardiographic and microvascular parameters.

Material and methods: This study analysed 41 adult patients with SSc (American College of Rheumatology/European Alliance of Associations for Rheumatology 2013) treated at the EUSTAR centre in Kyiv (2022–2024). Patients were stratified into ILD and non-ILD groups based on high-resolution computed tomography. Variables assessed: SSc subtype, modified Rodnan Skin Score (mRSS), autoantibodies (anti-Scl-70, ACA), pulmonary function (forced vital capacity [FVC], diffusion capacity of the lungs for carbon monoxide), 6-minute walk test (6MWT), and echocardiographic indices (left ventricular ejection fraction, E/A ratio). Microvascular damage was evaluated via nailfold videocapillaroscopy (NVC) using Cutolo patterns. Statistical analysis employed Mann-Whitney U and Fisher's exact tests ($p = 0.05$).

Results: The cohort was predominantly female (mean age 56.2 ± 12.4 years; disease duration 8.9 ±6.4 years). Interstitial lung disease was present in 63.4% of patients and was significantly associated with diffuse cutaneous SSc (96.2% vs. 26.7%; $p = 0.001$), higher mRSS (22.9 ±8.9 vs. 15.5 ±7.4; $p = 0.007$), and anti-Scl-70 positivity (57.7% vs. 19.2%; $p = 0.024$). The ILD patients showed lower FVC (74.9% vs. 96.5%; $p = 0.010$) and shorter 6MWT distance (281.5 ±64.5 m vs. 396.7 ±59.5 m; $p = 0.001$). An E/A ratio 0.8 was more frequent in ILD (57.7% vs. 20.0%; $p = 0.025$). Interstitial lung disease was also associated with advanced NVC patterns, reduced capillary density, and more frequent digital ulcers (69.2% vs. 33.3%; $p = 0.049$).

Discussion: The SSc-ILD is associated with a severe systemic phenotype marked by skin fibrosis and anti-Scl-70 positivity. Reduced FVC and 6MWT indicate functional impairment, while the higher prevalence of E/A 0.8 suggests early diastolic dysfunction may contribute to exercise intolerance. The association between advanced NVC patterns and ILD supports the link between microvascular damage and internal organ involvement.

Conclusion: In SSc, ILD identifies a high-risk subgroup with functional decline and advanced vasculopathy. Integrated assessment, including pulmonary, cardiac, and microvascular evaluation, is crucial for early risk stratification and personalised management.

O_03

DOI: https://doi.org/10.5114/reum/219164

Pulmonary nodules in a 29-year-old patient: a case report of pulmonary manifestations in rheumatoid arthritis

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Key words: pathology, joints, pulmonary, rheumatoid arthritis

Introduction: Rheumatoid arthritis (RA) is a systemic autoimmune disease that primarily affects connective tissue. Its development has been strongly associated with immunological factors, particularly certain major histocompatibility complex (MHC) class II molecules such as HLA-DRB1 alleles (including DRB101 and DRB104 variants). However, genetic susceptibility alone is not sufficient; most patients develop RA due to a combination of environmental exposures, infections, and underlying immune dysregulation. In many cases, RA has also been linked to abnormal protein citrullination, a biochemical process that alters protein structure and triggers autoimmune responses against immunologically significant molecules.

Case description: This case study describes a 29-year-old man whose disease course is followed over many years, starting with his first hospitalisation in 2013 due to further diagnosis regarding pulmonary lesions found in computed tomography. His medical history and physical examination findings led to an initial diagnosis of RA with the presence of pulmonary lesions. After a long follow-up period, the patient is reassessed again in 2023 due to evolving symptoms, which prompts a pathomorphological (histopathological) examination to clarify the diagnosis. The final findings integrate the clinical evolution with tissue-based findings, highlighting how the diagnosis and understanding of the disease developed over time and emphasising the importance of long-term monitoring and reassessment of the complexity of these pulmonary lesions.

Conclusions: Rheumatoid arthritis requires a comprehensive, multidisciplinary approach to diagnosis and management. Pathological manifestations observed in patients with RA often necessitate a thorough histopathological assessment to clarify the underlying aetiology, differentiate between disease-related and comorbid processes, and complete the clinical interpretation of observed findings.

O_04 DOI: https://doi.org/10.5114/reum/219163

Shrinking lung syndrome – a rare manifestation of systemic lupus erythematosus

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Key words: SLS, shrinking lung syndrome, systemic lupus erythematosus, dyspnea

Introduction: Systemic lupus erythematosus (SLE) is a heterogeneous autoimmune disease. Shrinking lung syndrome (SLS) is a rare pulmonary complication of SLE and other connective tissue diseases – e.g. Sjögren's disease, characterised by dyspnea, restrictive ventilatory defect, and diaphragmatic dome elevation, mimicking e.g. infections.

Case description: In 2022, a 32-year-old man had a severe left-sided pneumonia with pleural abscess, treated with ciprofloxacin and therapeutic video-assisted thoracoscopy, with good clinical response. Pleural fluid cultures were negative, including for *Mycobacterium tuberculosis*.

In June 2024, the patient developed arthritis, myalgia, and progressive exertional dyspnea with elevated inflammatory markers (C-reactive protein: 85 mg/l, erythrocyte sedimentation rate: 50 mm/h), high-titer antinuclear antibodies (ANA 1 : 1,280, homogenous pattern), and borderline proteinuria (0.51 g/24 h). Glucocorticosteroid (GC) therapy (prednisone 0.5 mg/kg/day) led to a transient clinical improvement.

The patient was admitted to the Department of Connective Tissue Diseases at the National Institute of Geriatrics, Rheumatology and Rehabilitation in October 2024 for suspected SLE, reporting progressive dyspnea, unintentional weight loss (10 kg in 4 months), and myalgia. Diagnosis of SLE was confirmed based on 2019 American College of Rheumatology/European Alliance of Associations for Rheumatology criteria (23 points): ANA > 1 : 80, arthritis, pleural and pericardial effusion, presence of lupus anticoagulant, proteinuria, and anti-dsDNA antibodies. High disease activity was assessed (SELENA-SLEDAI 12).

Due to progressive dyspnea, severe restrictive ventilatory defect (forced vital capacity [FVC]: 28%, total lung capacity [TLC]: 37%), and right pleural effusion with hemidiaphragm elevation, SLS associated with SLE was suspected. The patient received methylprednisolone pulses (in total 5 g i.v.), followed by oral prednisone (40 mg/day) and hydroxychloroquine (400 mg/day). After infection exclusion, treatment with anti-CD20 monoclonal antibody rituximab (2 × 1,000 mg, 14-day interval, first treatment cycle) and mycophenolate mofetil (p.o. 2 g/day) was initiated in March 2025. This led to a significant clinical improvement (SELENA-SLEDAI 0) and pulmonary recovery (FVC 65%, TLC 58%) by August 2025. In November 2025, GCs were discontinued.

Conclusions: This case shows that SLE can have atypical organ manifestations, complicating diagnosis. A rare autoimmune rheumatic complication – SLS, should be considered in progressive dyspnea with restrictive lung deficit. Prompt immunosuppressive treatment, including B-cell depleting therapy, can improve lung function and induce remission.

O_05 DOI: https://doi.org/10.5114/reum/219173

A rare pulmonary manifestation of immunoglobulin G4-related disease refractory to standard treatment

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Key words: IgG4-related disease, pulmonary manifestations, treatment resistance, lymphadenopathy

Introduction: Immunoglobulin G4-related disease (IgG4-RD) is a chronic immune-mediated disease that typically affects organs such as the pancreas, bile ducts, salivary glands, and lymph nodes. Involvement of the lungs was also reported. The disease usually responds well to standard immunosuppressive and glucocorticosteroid (GC) therapy, and, in refractory cases, biologic treatment, as in the case of the presented patient.

Case description: A 48-year-old patient presented with lymphadenopathy persisting for one year, located in the head, neck, and supraclavicular areas, accompanied by increased sweating and weight loss. Oncological and infectious diseases were investigated, with no identifiable cause of the reported symptoms. A cervical lymph node biopsy revealed: plasma cell density of 150–200 focal cells/HPF, predominant CylgG(+) cells with 40–50% IgG4(+) subclass content, and typical storiform fibrosis pattern. This, along with elevated serum IgG4 concentrations, led to the diagnosis of IgG4-RD disease in October 2023. Treatment with methylprednisolone and methotrexate was initiated, but due to the lack of improvement after 2 months, cyclosporine was added (to a maximum dose of 5 mg/kg), without achieving remission. The patient's clinical condition did not improve; significantly elevated acute-phase markers persisted in the serum. During GC dose reduction, new foci of lymphadenopathy (mainly paraaortic nodes and along the iliac vessels) and fibrotic nodules in the lungs appeared. A verification biopsy of the inguinal lymph node was performed, confirming the diagnosis of IgG4-RD. Due to the ineffectiveness of standard treatment and exhaustion of therapeutic options, rituximab (RTX) therapy was decided upon. Three cycles of RTX were administered: in January and July 2025, and in January 2026. Between cycles of RTX administration, clinical improvement was observed, along with a reduction in the size of the cervical, axillary, inguinal, and abdominal lymph nodes, but with persistent progression of pulmonary lesions (intensification of nodular lesions and atelectasis of the right middle lobe).

Conclusions: The presented case report illustrates the course of IgG4-RD refractory to standard treatment that poses a clinical challenge. The anti-CD19 antibody, inebilizumab, recently recognised as a breakthrough in the treatment of this disease, may be a new therapeutic proposition for such patients.

O_06 DOI: https://doi.org/10.5114/reum/219175

Diagnostic challenge in treating patients with pulmonary involvement in rheumatoid arthritis: a case report

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Key words: rheumatoid arthritis, lung cancer screening, lepidic adenocarcinoma

Introduction: Rheumatoid arthritis (RA) is a type of autoimmune disorder that can manifest itself by affecting various organs, predominantly joints. Pulmonary involvement is being defined as highly associated with substantial morbidity and mortality, with male sex, late onset, smoking history and high cyclic citrullinated peptide antibodies count being the main risk factors. Respiratory manifestations are usually present in patients already diagnosed with RA; however, they can also be the presenting feature. The spectrum of lung involvement is vast, with the most common being interstitial lung disease (ILD), especially usual interstitial pneumonia, pleuritis, rheumatoid nodules and airway disease. It is estimated that 10% of RA patients will suffer from significant ILD, and up to 60% will develop subclinical lung disease. Notably, patients with RA have an increased risk of lung cancer, which poses a significant diagnostic challenge.

Case report: A 70-year-old male with a 30-pack-year smoking history, arterial hypertension and rheumatoid arthritis was referred to the Institute of Tuberculosis and Lung Disease after multiple pulmonary nodules were detected on a chest computed tomography (CT) before initiation of immunosuppressive therapy with leflunomide. The most prominent nodule containing necrosis and measuring 13 × 21 mm was biopsied and diagnosed as a RA-nodule. The patient subsequently underwent follow-up CTs at least once a year. In which multiple scattered nodules, intermittent pleural effusions mainly on the left and ground-glass opacities (GGOs) predominantly in the right lower lobe (RLL) were identified. After 3 years of observation, in 2018, a progressive increase in the volume of GGO in RLL was noticed, with a growing solid component of the lesion reaching 64 × 27 mm by 2019, raising suspicion of cancerous growth, which was later confirmed in 2020. After the diagnosis, the patient underwent a wedge resection of the right lower lobe and was lost to follow-up in 2022.

Conclusions: Cancer is one of the leading causes of death in rheumatoid patients. The hazard ratio of lung cancer in RA patients revolves around 1.5 compared with the non-RA population. Thus, RA patients with risk factors such as male sex and a history of smoking should be screened for lung cancer. The guidelines of the American College of Rheumatology indicate that high-resolution chest CT may be used for screening patients for lung cancer, besides other pulmonary involvement, with the frequency being guided by individual clinical symptoms.

O_07

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A diagnostic and therapeutic challenge of rapidly progressive fibrosing interstitial lung disease: a case report

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Key words: interstitial lung disease, rapidly progressive interstitial lung disease, smoking-related interstitial fibrosis, desquamative interstitial pneumonia

Introduction: The differentiation between smoking-related interstitial lung diseases (SR-ILD) and interstitial pneumonia with autoimmune features (IPAF) represents a major diagnostic and therapeutic challenge. We present a complex case of a 46-year-old male with a dual phenotype of desquamative interstitial pneumonia (DIP) and smoking-related interstitial fibrosis (SRIF), complicated by high-titer autoantibodies and recurrent pulmonary embolism.

Case description: A 46-year-old male former smoker presented with progressive exertional dyspnea, dry cough, and recurrent fevers. Physical examination revealed digital clubbing and bilateral Velcro-like crackles. Initial high-resolution computed tomography showed diffuse ground-glass opacities (GGO) and lymphadenopathy. Over a three-year observation period, the patient experienced significant functional decline, reaching a restrictive pattern (total lung capacity: 55% predicted) and critical diffusion impairment (diffusion capacity of the lungs for carbon monoxide: 19% predicted). Echocardiography consistently showed no signs of pulmonary hypertension. Surgical lung biopsy confirmed a dual histological pattern of DIP and SRIF. Extensive immunological screening revealed high-titer anti-nuclear antibodies (1 : 2,560) and scleroderma-associated antibodies (anti-Th/To, anti-RP155), yet the patient did not meet the full American College of Rheumatology/European Alliance of Associations for Rheumatology criteria for systemic sclerosis. The clinical course was further exacerbated by recurrent pulmonary embolism and an admission to the Intensive Care Unit (ICU) and mechanical ventilation. Therapeutic strategies evolved from initial glucocorticosteroids to a combined regimen of mycophenolate mofetil, methylprednisolone pulses, and the addition of Nintedanib due to a progressive fibrotic phenotype. This multi-targeted approach was maintained throughout the patient's critical stabilisation following the ICU stay.

Conclusions: This case demonstrates that "overlap" ILD phenotypes can lead to life-threatening respiratory failure requiring intensive care intervention. However, the subsequent clinical improvement and partial radiological regression of GGO highlight the effectiveness of combining potent immunosuppression with antifibrotics. The recovery of a patient requiring intubation underscores the need for aggressive, multi-targeted treatment even in high-complexity, refractory cases.

Oral session 2

Vasculitides

O_08

DOI: https://doi.org/10.5114/reum/219184

Pituitary involvement in granulomatosis with polyangiitis: case report

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Key words: granulomatosis with polyangiitis, hypophysitis, hypopituitarism, ANCA-associated vasculitis, cyclophosphamide

Introduction: Granulomatosis with polyangiitis (GPA) is a multisystem disease characterised by necrotising small-vessel vasculitis, primarily affecting the upper respiratory tract, lungs, and kidneys. Pituitary involvement is reported in the literature in about 1% of all GPA cases. Pituitary GPA typically presents as a pituitary mass effect, resulting in symptoms such as headaches, visual disturbances, and anterior and posterior pituitary hormone deficiencies.

This report presents a rare case of pituitary GPA that was successfully treated with glucocorticosteroids (GCs) and cyclophosphamide.

Case description: A 22-year-old woman with a two-year history of headache, rhinitis, sinusitis, otitis media and cough. Sinus tomography revealed impaired ventilation of the middle ear, sphenoid and maxillary sinusitis. A chest computer tomography scans revealed multiple nodules. The diagnosis of GPA was confirmed by elevated anti-neutrophil cytoplasmic antibodies (c-ANCA) levels and the presence of ANCA directed to proteinase 3 antibodies. The initial treatment consisted of high-dose GCs and mycophenolate mofetil (2 g/day). After 6 months, clinical symptoms subsided. However, since May 2025, the patient experienced recurrent headache, general weakness, polyuria and polydipsia. The patient also had periodic nosebleeds and menstrual disorders. Biochemical tests revealed a deficiency of vasopressin, thyroid hormones and gonadotropins. The magnetic resonance imaging of the brain showed pituitary enlargement and thickening of the pituitary stalk – a typical symptom of pituitary inflammation. Treatment with GCs and cyclophosphamide in intravenous pulses was initiated. The headache resolved, and the patient underwent hormone replacement therapy.

Conclusions: To date, there are no established treatment guidelines of pituitary gland involvement in GPA. Several reports presented successful treatment with GCs and cyclophosphamide or rituximab. However, in most patients, pituitary gland dysfunction persists, necessitating long-term hormone replacement therapy.

It is important to be aware of the potential pituitary gland involvement in GPA, particularly in patients presenting with persistent headache and endocrine disorders.

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Difficulties in maintaining treatment of granulomatosis with polyangiitis

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Key words: vasculitis, complications, treatment, maintance therapy

Introduction: Granulomatosis with polyangiitis (GPA) is an autoimmune vasculitis which predominantly affects the respiratory system and kidneys. Immunosuppressive therapy effectively induces remission, but also may cause life-threatening complications. Consequently, maintaining balance between disease control and the risk of treatment-related adverse events is crucial in the therapeutic process.

Case description: In January 2020, a 62-year-old woman was admitted to a local hospital with unintentional weight loss, malaise, ascites and oliguria ongoing for several weeks. Chest high-resolution computed tomography revealed lung infiltrates with cavitary lesions, suggestive of *Staphylococcus aureus* pneumonia (Fig. 1). Unsuccessful treatment with antibiotics, together with oral and labial ulcerations, raised the suspicion of GPA. The diagnosis was confirmed by the presence of elevated levels of proteinase 3 antineutrophil cytoplasmic autoantibody (PR3-ANCA). Progressive renal failure required hemodialysis (HD) via a tunneled catheter. Plasmapheresis, intravenous prednisolone, and cyclophosphamide were initiated.

In June 2023, the patient was readmitted with suspected pneumonia and radiological progression of pulmonary lesions. Bronchioalveolar lavage cultures yielded *Acinetobacter baumannii* and *Candida krusei*, prompting initiation of targeted therapy with amikacin, colistin, and micafungin. Echocardiography revealed a thrombus in the right atrium and intravenous catheter distal tip thickening. Although positron emission tomography suggested fungal endocarditis, repeated blood and catheter cultures were negative. However, continuation of antifungal therapy resulted in clinical improvement. After creating an arteriovascular shunt on the right arm, HD was continued. Persistent pulmonary progression, elevated PR3-ANCA levels and hearing loss prompted qualification for rituximab.

In September 2023, the patient developed right arm cellulitis due to the right subclavian vein thrombosis, which was treated with piperacillin-tazobactam. Endovascular embolisation of the lateral rami of the cephalic vein contributed to the reduction of cellulitis. After subsidence of inflammation, rituximab treatment was initiated in December 2023.

Conclusions: The underlying disease and its treatment can cause severe infections and thrombotic events, contributing to the complexity of GPA management. This case shows that systematic reassessment and individualised therapy are essential to maintaining the effectiveness of the treatment, along with minimising treatment-related morbidity.

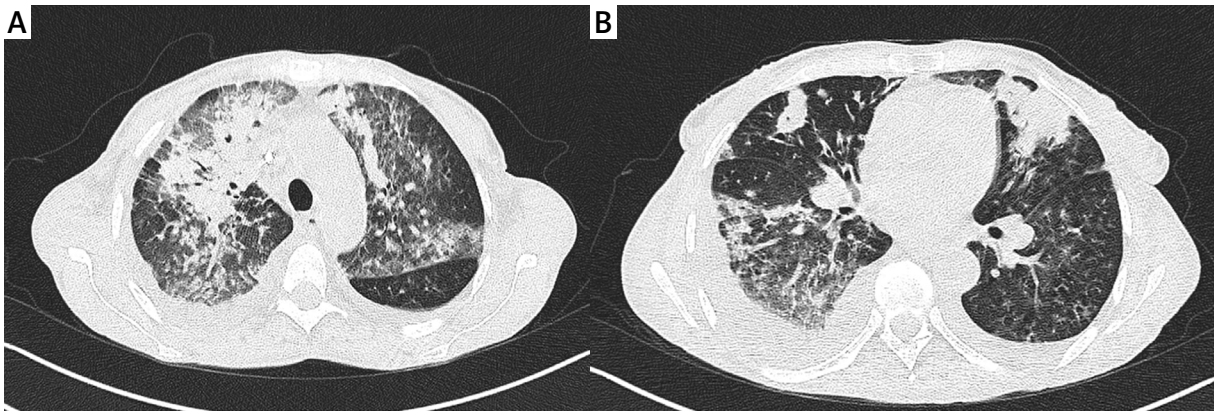

Fig. 1. High-resolution computed tomography scans of pulmonary lesions at the time of diagnosis in Ferbuary 2020.

O_10 DOI: https://doi.org/10.5114/reum/219176

Eosinophilic granulomatosis with polyangiitis – one disease with two overlapping mechanisms: a case report

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Key words: eosinophilic granulomatosis with poliangiitis, ANCA antibodies, mepolizumab

Introduction: Eosinophilic granulomatosis with polyangiitis (EPGA) is a rare multisystem inflammatory disease characterised by asthma with eosinophilia, polyneuropathy, and inflammation of small- and medium-sized blood vessels. The EGPA commonly progresses through three phases: allergic, eosinophilic, and vascular. We distinguish two clinical manifestations of Churg-Strauss syndrome: anti-neutrophil cytoplasmic antibodies (ANCA)-positive, typically associated with renal failure and neuropathy and ANCA-negative with heart and lung involvement. ANCA antibodies determined in EGPA can be both c- and p-ANCA. The ANCA-positive phenotype is observed in approximately 30–40% of patients with EGPA. First-line treatment consists of glucocorticosteroids (GCs) nonetheless, in severe cases, immunosuppressants such as cyclophosphamide in combination with immunotherapy targeting the interleukin-5 pathway, like mepolizumab, offer an effective treatment approach.

Case description: A 56-year-old male with a history of asthma presented with chronic cough lasting over 3 months, recent vomiting, peripheral blood hypereosinophilia, macular rash on lower extremities, and renal failure with eGFR 27 ml/min/1,73 m^2, accompanied by proteinuria. An enlarged hilum was found on a chest radiograph. Further diagnostics with computed tomography (CT) confirmed enlarged hilar lymph nodes. Moreover, it revealed subpleural nodules and ground-glass opacities in the apex of the right lung. Biopsy of the lymph nodes disclosed eosinophilic infiltrates. The CT scan of the paranasal sinuses showed thickening of the mucosal membranes. An extensive rheumatological workup was conducted, yielding negative results. Taking all the clinical manifestations into account, the diagnosis of seronegative EGPA was made. Initial treatment with GCs was insufficient. The patient was subsequently treated with cyclophosphamide in combination with mepolizumab, resulting in complete clinical remission.

Conclusions: We chose this case to highlight the overlapping manifestations of ANCA-negative and ANCA-positive EGPA. Although the patient was seronegative, he exhibited symptoms characteristic of both clinical manifestations, including lung and renal involvement. While the mechanisms of organ damage occurring in Churg-Strauss syndrome seem to be well described, this case is a clear example that we should keep looking for the underlying pathophysiological basis for overlapping features between the two clinical manifestations of this rare and heterogeneous disease.

O_11 DOI: https://doi.org/10.5114/reum/219196

A case of hypercalcemia and acute kidney injury caused by *Pneumocystis jirovecii* in an immunocompromised patient with granulomatosis with polyangiitis

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Key words: *Pneumocystis jirovecii*, hypercalcemia, acute kidney injury, granulomatosis with polyangiitis, rituximab

Introduction: We report the case of a patient with a history of granulomatosis with polyangiitis who developed severe *Pneumocystis jirovecii* pneumonia (PJP) with an atypical clinical presentation.

Case description: A 55-year-old male with a history of granulomatosis with polyangiitis (GPA) with lung and kidney involvement, treated with glucocorticosteroids, methotrexate and rituximab, presented to the rheumatology department with symptoms including generalised weakness, loss of appetite, weight loss, oral ulcers, and decline in renal function. Initial examination and laboratory tests revealed no signs of activity of the anti-neutrophil cytoplasmic antibodies (ANCA)-associated vasculitis, however they showed increased levels of serum creatinine (4.92 mg/dl, glomerular filtration rate: 13.1 ml/min/1.73 m^2) and hypercalcemia (total calcium: 3.58 mmol/l; normal range: 2.15–2.55 mmol/l). Symptomatic treatment with fluid therapy, diuretics and hydrocortisone was initiated. A comprehensive diagnostic work-up was performed, revealing low parathyroid hormone levels (11.1 pg/ml; normal range: 15.0–68.3 pg/ml), negative tumour markers, slightly elevated inflammatory marker levels and bilateral nephrocalcinosis on abdominal ultrasound. Patient's condition gradually worsened as he developed respiratory symptoms with simultaneous deterioration of pulmonary imaging. Due to rapid-onset severe pneumonia, the patient was temporarily transported to the intensive care unit. *Pneumocystis jirovecii* pneumonia was confirmed, and trimethoprim-sulfamethoxazole (TMP-SMX) treatment was initiated, resulting in both clinical and biochemical improvement. Rituximab treatment was temporarily discontinued after considering the risk-benefit balance, and chronic secondary prevention with TMP-SMX was implemented. The patient was discharged in good condition. A case of hypercalcemia secondary to *P. jirovecii* infection was concluded.

Conclusions: To our knowledge, this is the second reported case of hypercalcemia in a non-HIV, non-transplant immunosuppressed patient with ANCA-vasculitis. Hypercalcemia preceding clinical presentation of PJP in immunosuppressed patients is documented in medical literature and, along with this case study, underlines the importance of extended diagnostic work-up after the most common causes of laboratory hypercalcemia are excluded. Furthermore, considering that patients with rheumatic diseases, especially those receiving immunosuppressive therapy, are more prone to the *P. jirovecii* infection, an individual decision regarding chronic chemoprophylaxis needs to be made.

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Response to treatment of inflammatory pseudotumours in granulomatosis with polyangiitis based on case series

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Key words: granulomatosis with polyangiitis, ANCA-associated vasculitis, inflammatory pseudotumors, rituximab, cyclophosphamide

Introduction: Granulomatosis with polyangiitis (GPA) is a rare anti-neutrophil cytoplasmic antibody-associated vasculitis, characterised by necrotising inflammation of small- and medium-sized vessels. It typically involves the upper and lower respiratory tract and kidneys; however, virtually any organ may be affected. According to the 2022 European Alliance of Associations for Rheumatology (EULAR) recommendations, remission induction consists of glucocorticosteroids combined with rituximab or cyclophosphamide.
The study aims to evaluate the effectiveness of pharmacological treatment of solid inflammatory lesions located at atypical sites in patients with GPA.

Material and methods: Four hospitalised patients with GPA were included in this analysis. The diagnosis of GPA was established according to current EULAR classification criteria. The inclusion criterion was the presence of solid inflammatory lesions at atypical sites, despite typical respiratory tract involvement in GPA. Atypical lesions were located in the orbit, posterior mediastinum, and prostate and presented as a frontal sinus fistula. All patients received standard-of-care therapy in accordance with current guidelines, including rituximab (3 patients) or cyclophosphamide (1 patient). Treatment response was assessed at baseline and after 6 months based on inflammatory laboratory parameters, the Birmingham Vasculitis Activity Score (BVAS 3.0), and radiological evaluation of the lesions using computed tomography (CT).

Results: After 6 months of treatment, normalisation of C-reactive protein and decrease in disease activity within all BVAS 3.0 domains were observed (Table I). In all patients, we have observed a resolution of inflammatory changes in the respiratory tract and lungs in CT scans. Despite reaching clinical remission in all 4 cases, no radiological regression of atypically located lesions was noted, and all patients continued to present signs of local organ compression.

Discussion: All patients presented with typical involvement of the upper respiratory tract, and three also had lower respiratory tract involvement. The treatment outcomes observed in the studied patients are consistent with previous reports, indicating that solid lesions in GPA often demonstrate limited or no response to standard immunosuppressive therapy. Consequently, surgical intervention is currently considered in selected cases.

Conclusions: The heterogeneous localisation and treatment response observed among patients may reflect differences in underlying signaling pathways and pathogenetic mechanisms, warranting further research.

Table I. Characteristics and treatment of patients with GPA

Variable	Number			
	1	2	3	4
Age	65	39	59	48
Sex	Male	Female	Male	Male
CRP Before After	 66 < 1	 70 11	 146 2	 4 1
BVAS 3.0 Before After	 13 2	 16 1	 N/A 1	 8 1
Organ involvement	URT, LRT, CNS	URT, LRT, J, GIT	URT, LRT, REN	URT
Localisation of atypical lesions	Orbit	Posterior mediastinum	Prostate	Frontal sinus fistula
Atypical lesions response	No regression	No regression	No regression	No regression
Treatment	RTX	RTX	CYC	RTX

BVAS – Birmingham Vasculitis Activity Tract, CRP – C-reactive protein, CNS – central nervous system, CYC – cyclophosphamide, J – joints, GIT – gastrointestinal tract, LRT – lower respiratory tract, REN – renal involvement, RTX – rituximab, URT – upper respiratory tract.

Oral session 3

Autoimmunity in the course of other diseases

O_13

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Coexistence of juvenile systemic lupus erythematosus and Sjögren's disease in a 13-year-old girl

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Key words: juvenile systemic lupus erythematosus, Sjögren's disease, pediatric autoimmune disease

Introduction: Confirming systemic autoimmune rheumatic diseases in children is a challenge. Many diseases lack validated classification criteria, so we use criteria developed for adults.

Case description: A 13-year-old girl with an episode of parotitis, persistent fatigue, cervical lymphadenopathy, splenomegaly, malar rash, erythematous papules, photosensitivity, intermittent fever (≤ 38.5°C) without infection, prior arthralgia, and then arthritis of many joints. No clinical or subjective sicca symptoms. Bacterial, viral infections (human immunodeficiency virus, Epstein-Barr virus, cytomegalovirus, parvovirus B19), and toxoplasmosis were excluded. Laboratory tests revealed: leukopenia – 2.9 × 10^3/µl, anemia – Hb 10.9 g/dl with positive DAT, [a]hypergammaglobulinemia; low C3, C4 complement, positive rheumatoid factor, antibodies to citrullinated protein antigens (–), anti-nuclear antibodies (ANA) (HEp-2) high-titer 1 : 10,240 (homogeneous) and 1 : 320 (speckled); anti-dsDNA elevated (393.2 IU/ml); anti-Ro60/Ro52 high (quantitative method); anti-SSB/La and AMA-M2 (+++; semiquantitative method); negative antiphospholipid antibodies, circulating immune complexes present. Based on the cervical lymph node biopsy, a proliferative process was excluded – reactive lymphoid hyperplasia. The patient was diagnosed with juvenile systemic lupus erythematosus (28 points European Alliance of Associations for Rheumatology/American College of Rheumatology classification criteria for SLE). Sjögren's disease (SjD) diagnosis supported despite no minor salivary gland biopsy: typical Abs (anti-Ro60, anti-AMA-M2), SGUS score 2, recurrent sialadenitis. The SLEDAI 2K indicate high SLE activity (13 pts). Treatment with megadoses of glucocorticosteroid (GC) – methylprednisolone, maintenance therapy with oral GC (prednisone), methotrexate and hydroxychloroquine were introduced. Child lupus low disease activity was achieved. In 2024, GC was discontinued. The only clinical symptom was a discrete malar rash, peripheral blood counts, urinalysis were in norm, ANA titer 1 : 5,120 homogenous; 1 : 320 speckled pattern. Continued low C4 complement component and high Ro60 and Ro52 antibody concentrations without sicca symptoms.

Conclusions: Features of SLE appear to be identical to the adult form, but this is less obvious in children with SjD, especially with the absence of dryness symptoms. Recurrent parotitis, cutaneous manifestations, and similar laboratory abnormalities complicate the diagnostic process. Prior infections may act as a triggering factor, potentially explaining some atypical symptoms for both diseases (elevation of inflammatory markers). Treatment involves the same groups of drugs, and as in the case described, improvement was achieved in most domains.

O_14 DOI: https://doi.org/10.5114/reum/219191

Secondary Evans' syndrome as a first presentation of systemic lupus erythematosus: case study

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Key words: Evans' syndrome, SLE, nephrotic syndrome, AIHA, ITP

Introduction: Evans' syndrome is a rare condition defined as concurrent autoimmune hemolytic anaemia (AIHA) and immune thrombocytopenia (ITP). Distinction has been made between primary and secondary variant, which differentiate it on bases if condition is idiopathic or associated with another systemic disease. The most common causes of secondary variant include: haematological malignancies and autoimmune disease, especially systemic lupus erythematosus (SLE). This condition could sometimes precede the onset of the underlying disease.

Case description: A patient aged 43 years was admitted to the nephrology ward in March 2025 with massive oedema and acute kidney injury. She was treated since 2001, the first symptom was thrombocytopenia. Diagnosis of ITP was made, the first line of treatment was vincristine, stopped due to polyneuropathy. Then she was managed with glucocorticosteroids (GCs), due to resistance, the splenectomy was performed, after which the level of platelets of 50–90 G/l was achieved. In 2014, she developed AIHA and a recurrence of thrombocytopenia. The antinuclear, anti-GPIa/II and anti-GPIIb/IIa antibodies were detected. In bone marrow biopsy, hypoplasia was described corresponding to an autoimmune process. Concurrent infection of parvowirus B19 was detected. The patient was treated with GCs, intravenous immune globulin and multiple blood transfusions. Between 2015 to 2020, patient experience few recurrences, all treated with GCs and blood transfusions. As complications to coagulation disorders, the patient underwent paralytic ileus in 2015, ischemic stroke in 2018 and cardiac infarction in 2019. During neurological examination in 2017, anti-AQP4 antibodies were detected, disease from neuromyelitis optica spectrum disorders was diagnosed. The GCs were administrated with a good response. During hospitalisation in nephrology, in laboratory finding proteinuria in the nephrotic range was detected and estimated glomerular filtration rate of 50 ml/min/1.73 m^2. Renal biopsy was impossible due to coagulation disorders. The chronic kidney disease on the basis of lupus nephritis, was diagnosed. The patient was treated according to the EuroLUPUS scheme with 500 mg of cyclophosphamide. After 6 cycles, partial remission of nephrotic syndrome was achieved.

Conclusions: Systemic lupus erythematosus is a heterogeneous disease. Solely haematological manifestation preceding other symptoms posed a diagnostic challenge in this case. Renal involvement and good response to EuroLUPUS treatment support the diagnosis of SLE. The most important issue in this patient is treating the underlying disease.

O_15

DOI: https://doi.org/10.5114/reum/219170

Tocilizumab in refractory and glucocorticosteroid-dependent eosinophilic fasciitis: a case report

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Key words: eosinophilic fasciitis, tocilizumab, glucocorticosteroids, treatment failure, interleukin-6 inhibitors

Introduction: Eosinophilic fasciitis (EF) is a rare autoimmune connective tissue disease classified among scleroderma-like disorders. It is characterised by painful, woody induration of the skin of the extremities, peripheral eosinophilia, elevated erythrocyte sedimentation rate (ESR) and hypergammaglobulinemia.

Case description: A 28-year-old man was diagnosed with EF in August 2024 based on progressive, symmetrical induration of the skin and subcutaneous tissue of the forearms, hands, lower legs and feet, associated with restricted joint mobility. Raynaud's phenomenon was absent. Laboratory tests revealed elevated C-reactive protein (33 mg/l), ESR (22 mm/h), peripheral eosinophilia (12.5%), and hypergammaglobulinemia (1,44 g/dl). Magnetic resonance imaging (MRI) of the left lower leg showed inflammatory oedema of the muscular fascia (Fig. 1). Histopathology revealed inflammatory infiltrates composed predominantly of CD8+ > CD4+ T lymphocytes, CD68+ macrophages and scattered eosinophils.

Differential diagnosis included systemic sclerosis, other scleroderma-like syndromes, and a paraneoplastic process. Nailfold capillaroscopy was normal, antinuclear antibody test was negative, and oncological screening (computed tomography of the neck, chest, abdomen and pelvis) revealed no abnormalities. Additional investigations detected IgM antibodies in significant concentrations against *Borrelia burgdorferi*, and doxycycline therapy was administered for 28 days.

Initial treatment with oral glucocorticosteroids (GCs; prednisone equivalent 0.5 mg/kg/day) was ineffective. Subsequently, i.v. high-dose methylprednisolone pulse therapy (500 mg/day for 5 days) was administered, and methotrexate was introduced up to a target dose of 25 mg/week. Due to persistent disease activity and GC dependence, after reviewing the literature, treatment with the interleukin-6 (IL-6) inhibitor tocilizumab (162 mg s.c. once weekly) was initiated in January 2025. This resulted in improved joint mobility, stabilisation of skin induration, normalisation of eosinophil count and inflammatory markers, and allowed tapering of methylprednisolone to 6 mg/day. Follow-up MRI in January 2026 demonstrated near-complete resolution of fascial inflammatory changes (Fig. 2). Given the favourable response to anti-IL-6 therapy, treatment was continued.

Conclusions: This case highlights the efficacy of tocilizumab in refractory and GC-dependent EF, leading to clinical and laboratory remission as well as significant improvement on imaging. Interleukin-6 inhibitors may represent a valuable therapeutic option in severe forms of eosinophilic fasciitis.

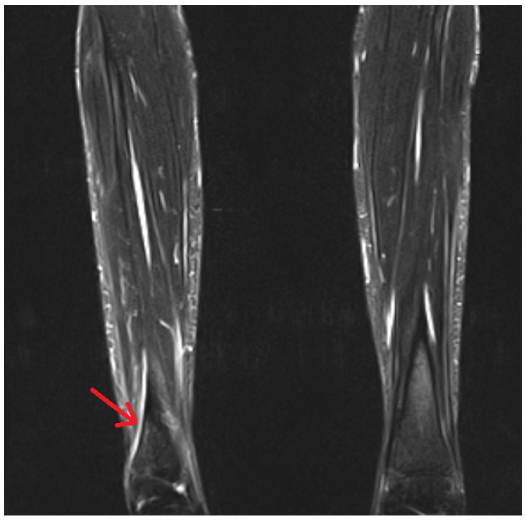

Fig. 1. Magnetic resonance imaging before treatment with tocilizumab.

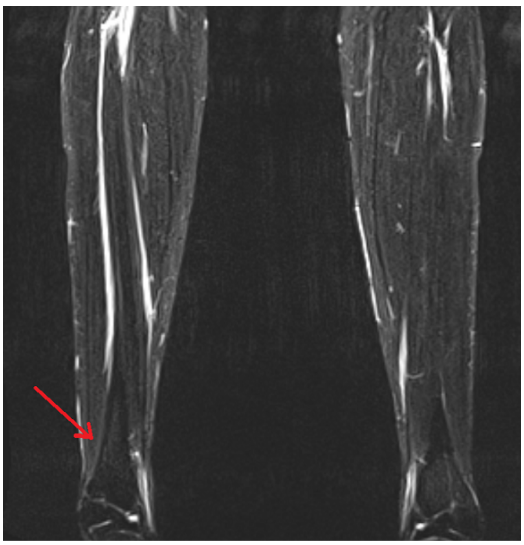

Fig. 2. Magnetic resonance imaging one year after starting treatment with tocilizumab.

O_16 DOI: https://doi.org/10.5114/reum/219166

Differentiating inflammatory from mechanical pathologies: the impact of rheumatologist-performed ultrasound on psoriatic arthritis diagnosis

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Key words: psoriatic arthritis, osteoarthritis, point-of-care ultrasound (POCUS), enthesitis

Introduction: Diagnosing autoimmune arthritis in patients with advanced osteoarthritis can be challenging, especially in the early stages of the disease. Overlapping symptoms often lead to delayed diagnosis and ineffective treatment focused solely on degenerative or mechanical changes.

Case description: We present the case of a 66-year-old female with a long-standing history of advanced osteoarthritis of the hands (Heberden's and Bouchard's nodes). The patient reported pain in the right heel (radiating to the Achilles tendon) for approximately 3 months and pain in the second left toe for 2 weeks. Laboratory tests, including rheumatoid factor (RF), antibodies to cyclic citrullinated peptide, and HLA-B27, were negative. A family history revealed psoriasis in the patient's mother.

Initially, the patient received orthopaedic treatment based on an ultrasound diagnosis of Achilles tendinopathy. Despite receiving a platelet-rich plasma injection and subsequent non-steroidal anti-inflammatory drugs therapy, no significant clinical improvement was observed.

During a rheumatological consultation, an ultrasound was performed. The examination revealed clear signs of enthesitis of the right Achilles tendon (increased Power Doppler signal and thickening) and inflammation of the MTP-2 flexor pulley, accompanied by subcutaneous tissue inflammation consistent with dactylitis. Based on the CASPAR criteria (3 points: negative RF, family history of psoriasis, and dactylitis), psoriatic arthritis was diagnosed. Treatment with methotrexate (20 mg/week) and prednisone (10 mg/day) was initiated, and the patient remains under regular rheumatological follow-up to monitor treatment efficacy.

Conclusions: This case illustrates that autoimmune arthritis can pose significant diagnostic difficulties in patients with pre-existing musculoskeletal disorders. It highlights the necessity for clinical vigilance whenever new joint pain patterns emerge. Rheumatologist-performed ultrasound at the point of care remains a crucial diagnostic tool for differentiating inflammatory from mechanical pathologies.

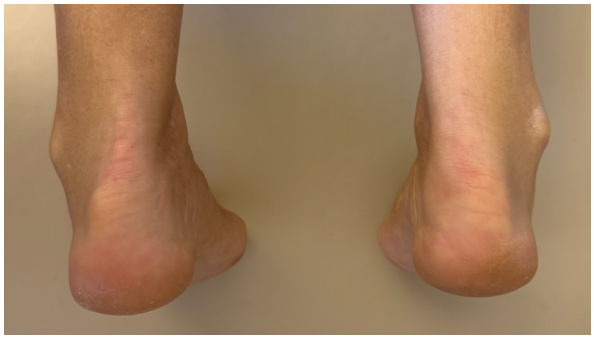

Fig. 1. Achilles oedema.

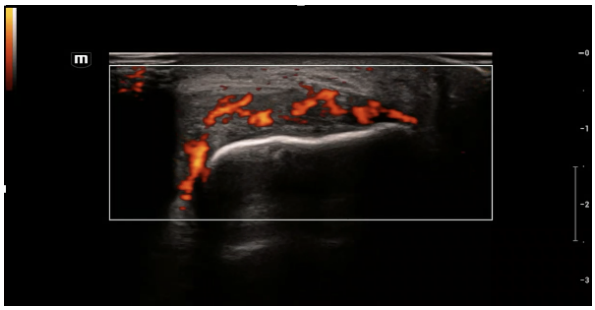

Fig. 2. Achilles enthesitis.

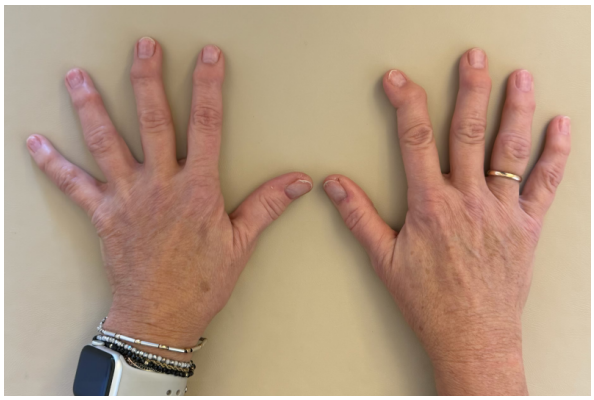

Fig. 3. Osteoarthritis of the hands.

O_17 DOI: https://doi.org/10.5114/reum/219201

When joints cost sight – vision loss in rheumatoid arthritis

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Key words: rheumatoid arthritis, dry eye syndrome, ophthalmic complications

Introduction: Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disease primarily affecting synovial joints. Ocular involvement is among the most important extra-articular manifestations of RA. The most common ophthalmic manifestation is dry eye syndrome in the course of secondary Sjögren's disease. Necrotic sclerosis and peripheral ulcerative keratitis are particularly aggressive inflammatory processes that may lead to corneal thinning, perforation and irreversible vision loss.

Case description: An 88-year-old woman has been under the care of the Department of Ophthalmology for 24 years. In 2002, she presented on an emergency duty for ophthalmologic consultation due to a corneal ulcer in the right eye complicated by subsequent corneal perforation. The condition developed in the course of severe dry eye syndrome secondary to RA.

During the course of her medical history, the patient underwent 5 procedures involving the transplantation of human amniotic membrane onto the cornea in the right eye and 3 in the left eye, as well as 5 penetrating corneal transplantation in the left eye and 2 in the right eye. Despite numerous attempts to improve visual acuity, a satisfactory visual outcome has not been achieved. However, preservation of both eyeballs and low visual acuity was maintained. Each surgical intervention was complicated by impaired wound healing and recurrent corneal ulcerations. The patient has been on long-term systemic treatment for rheumatoid arthritis and, following high-risk corneal grafts, required systemic glucocorticosteroid and immunosuppressive therapy. This may have contributed to other systemic complications, including myocardial infarction, mesenteric infarction with intestinal perforation, and lower limb ischemia, that occurred during the patients long term follow-up. The above-mentioned complications may also be attributable to advanced autoimmune disease.

Conclusions: Despite advances in disease-modifying drugs and biological drugs, severe ophthalmic complications remain a clinical challenge. Early and close cooperation between a rheumatologist and an ophthalmologist is crucial to prevent catastrophic outcomes. Rheumatoid arthritis is one of the autoimmune diseases that may lead to irreversible vision loss.

Oral session 4

Organ involvement in rheumatic diseases

O_18 DOI: https://doi.org/10.5114/reum/219169

Could acute pancreatitis develop in the course of the anti-melanoma differentiation-associated protein 5 antibody-positive dermatomyositis? Case reports of two patients

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Key words: dermatomyositis, anti-MDA5, acute pancreatitis, RP-ILD

Introduction: Anti-melanoma differentiation-associated protein 5 (MDA5) antibody-positive dermatomyositis (MDA 5 DM) is a subtype of a rare autoimmune disorder characterised by various skin manifestations and interstitial lung disease (ILD), which may progress rapidly. Most patients do not present significant muscle weakness (amyopathic dermatomyositis). Recent publications suggest a correlation between anti-MDA-5 antibodies and acute pancreatitis; thus, we present two case reports of adult patients with anti-MDA-5 dermatomyositis who also developed acute pancreatitis.

Case description: A 52-year-old woman was diagnosed with amyopathic anti-MDA-5 positive dermatomyositis with ILD of probable paraneoplastic origin due to suspected laryngeal carcinoma, which was later excluded. One month later, she was admitted to the Emergency Department, diagnosed with acute pancreatitis (abdominal pain, elevated pancreatic enzymes levels and typical radiological findings), and treated conservatively. Differential diagnosis excluded cholelithiasis, alcohol abuse, hypertriglyceridemia, and other causes. Unfortunately, she developed multiple organ failure and died after 24 days of Intensive Care Unit hospitalisation.

The second patient was a 40-year-old man also diagnosed with amyopathic anti-MDA-5 positive dermatomyositis (typical skin changes, arthritis, fever). Additionally, rapidly progressive interstitial lung disease was identified. Treatment was initiated with intravenous methylprednisolone and cyclophosphamide. However, a few days after discharge, he was re-admitted with the suspicion of acute pancreatitis (fever, vomiting, upper abdominal pain, elevated pancreatic enzymes levels) and treated conservatively. Other causes of pancreatitis were excluded. Due to the severe disease course and insufficient response to glucocorticosteroid therapy, intravenous immunoglobulins and cyclosporine A were implemented with a significant clinical improvement.

Conclusions: The MDA-5 DM was first described in 2005. Subsequently, it was reported that this DM subtype may be associated with an increased risk of developing acute pancreatitis. However, further long-term observational studies and extended, detailed follow-ups are necessary to confirm a potential causal relationship. Moreover, other acute pancreatitis causes, such as cholelithiasis, should be excluded. Eventually, it is important to include this rare but potentially life-threatening complication in MDA-5 DM patients.

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Dual autoantibody positivity in chronic kidney disease: a diagnostic consideration

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Key words: ANCA-associated vasculitis, glomerulonephritis, chronic kidney disease

Introduction: Co-presentation of perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA) and anti-glomerular basement membrane (anti-GBM) antibodies is considered relatively rare. Anti-GBM disease and ANCA-associated vasculitis (AAV) both can present with life-threatening manifestations such as rapidly progressive glomerulonephritis and alveolar haemorrhage. Studies of double-positive cohorts suggest a hybrid phenotype, combining features of AAV – such as older age at presentation, longer prodromal symptoms, chronic histologic changes, and a tendency to relapse – with the severe renal involvement and frequent pulmonary hemorrhage typical of anti-GBM disease.

Case description: An 82-year-old woman with a history of arterial hypertension, chronic heart failure, asthma, anaemia, and bronchiectasis was admitted to the nephrology unit in July 2025 due to deterioration of her general condition and rapidly progressing chronic kidney disease. Renal function had been normal in June 2024; however, elevated renal parameters were first documented in January 2025. On admission, laboratory tests revealed serum creatinine 4.2 mg/dl and estimated glomerular filtration rate (eGFR) of 10 ml/min/1.73 m^2, proteinuria, elevated inflammatory markers and positivity for p-ANCA and anti-GBM antibodies. Immunofixation electrophoresis was unremarkable. Imaging studies demonstrated interstitial lung disease and sinonasal polyps. In early August, the patient received three intravenous pulses of methylprednisolone (250 mg each), followed by oral prednisone 40 mg daily. She subsequently underwent four sessions of plasmapheresis until anti-GBM antibody negativity was achieved. Remission-induction therapy with cyclophosphamide was initiated; after eight pulses, until January 2026, serum creatinine decreased to 3 mg/dl, and eGFR increased to 15 ml/min/1.73 m^2.

Conclusions: This case shows that despite presenting with advanced chronic renal disease, this double-positive patient may recover renal function after aggressive therapy. These observations highlight the importance of early intensive diagnostics and treatment, including plasma exchange and immunosuppression, followed by careful long-term monitoring and consideration of maintenance immunosuppressive therapy.

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Lymphoma-associated hemophagocytic lymphohistiocytosis presenting as fever of unknown origin: a diagnostic challenge

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Key words: HLH, DLBCL, FUO, etoposide, R-CHOEP

Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a potentially fatal hyperinflammatory syndrome driven by uncontrolled immune activation. In adults, it is most commonly secondary to infections, autoimmune diseases, or hematologic malignancies.

Case description: In January 2020, a 55-year-old man was admitted to the hospital due to weight loss, weakness, and recurrent fevers lasting for about one month. Laboratory findings showed pancytopenia, while C-reactive protein was slightly elevated. A computed tomography (CT) scan revealed enlarged mediastinal lymph nodes and hepatosplenomegaly. Bone marrow biopsy immunophenotyping did not suggest lymphoma. Anti-nuclear antibodies at a titer of 1 : 160 (speckled pattern) and the presence of anti-RNP/Sm antibodies were found, causing a temporary transfer to the Rheumatology Department, where the extensive diagnostic process was inconclusive. Due to clinical features of HLH (6/8 HLH-2004 criteria), fever, hepatosplenomegaly, pancytopenia, hypertriglyceridemia, hypofibrinogenemia, ferritin at 9,000 ng/ml, and sIL-2R (sCD25) at 25,000 U/ml, i.v. dexamethasone was administered. Endobronchial ultrasound transbronchial needle aspiration, and the subcarinal lymph node from mediastinoscopy did not show lymphoma infiltration. At that time, the histopathologic results of the initial bone marrow biopsy revealed infiltration by aggressive B-cell lymphoma, most likely diffuse large B-cell lymphoma.

The patient received 8 cycles of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone)-based chemotherapy, initially without rituximab and including etoposide in the first 6 cycles (CHOEP), with subsequent modifications due to toxicities. After the first cycle of chemotherapy, the clinical symptoms of HLH began to subside, and after treatment, a complete metabolic response of lymphoma was observed on positron emission tomography/computed tomography (PET-CT). Patient remained in observation. In 2025, the patient presented with fever and, after examination, met 5 HLH-2004 criteria again. Although lymphoma relapse was suspected, it was not found in repeated biopsies (bone marrow, lymph node excision, liver) until the results of the spleen biopsy were obtained. The patient received second-line chemotherapy according to the R-ICE (rituximab, ifosfamide, carboplatin, etoposide) regimen. After 4 cycles, disease progression was detected on PET-CT. The patient is currently awaiting treatment with chimeric antigen receptor T-cell (CAR-T) therapy.

Conclusions: Hemophagocytic lymphohistiocytosis in adult patients necessitates thorough evaluation for possible underlying malignancy, especially lymphoma. Repeated biopsies of different organs may be required. Glucocorticosteroid treatment, although life-saving, can obscure the diagnosis of occult lymphoma.

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Sarcoidosis and ankylosing spondylitis – a coincidence or common ethiopathogenesis? Case series

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Key words: sarcoidosis, ankylosing spondylitis, mimicker

Introduction: Sarcoidosis is a chronic granulomatous disease affecting various organs and systems, most commonly the lungs and lymph nodes, but also the musculoskeletal system (4 – 38% of patients). Ankylosing spondylitis (AS) is the most common type of spondyloarthropathy, predominantly affecting the axial skeleton. It has been reported that AS can occur in patients with sarcoidosis almost two times more frequently than in the general population, we present three case reports of the coexistence of sarcoidosis and AS.

Case description: The first patient was a 52-year-old man diagnosed with sarcoidosis based on clinical manifestations, radiological findings and lymph node biopsy. Additionally, he suffered from back pain since he was 25 years old and a computed tomography (CT) scan depicted syndesmophytes in his spine and ankylosis of sacroiliac joints. The patient was referred to the rheumatologist, and based on modified New York Criteria, AS with positive HLA-B27 antigen was diagnosed. The patient has been treated with non-steroidal anti-inflammatory drugs (NSAIDs) and sulfasalazine due to an inflammatory bowel disease.

The second patient was a 56-year-old man diagnosed with AS with positive HLA-B27 antigen two years prior, treated with NSAIDs and upadacitinib. The patient reported a frequent productive cough, and his chest CT scan revealed enlarged bilateral mediastinal lymph nodes. Pulmonary functional tests were normal, and due to unclear histopathological results, a second lymph node biopsy was performed, and eventually, it confirmed the diagnosis of sarcoidosis.

The third patient was a 58-year-old woman also diagnosed with AS with positive HLA-B27 antigen eleven years prior, treated with certolizumab. Additionally, patient reported periodic dyspnoea, and based on a high-resolution CT scan, sarcoidosis was suspected. Lymph node biopsy was performed, and a diagnosis of sarcoidosis was established.

The first patient was treated with NSAIDs, the second and third with glucocorticosteroids and NSAIDs, with complete resolution of symptoms and lymphadenopathy in later observation.

Conclusions: Sarcoidosis and AS coexist, and this phenomenon might be explained by shared pathogenic mechanisms, including Th17 cells, tumour necrosis factor (TNF), interleukin-12 (IL-12), and IL-23 and – at least in some patients – sarcoidosis may occur during anti-TNF treatment. Further studies on this rare but intriguing association should be conducted.

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Gastric cancer mimicking systemic lupus erythematosus

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Key words: systemic lupus erythematosus, paraneoplastic rheumatic syndrome, gastric cancer, paraneoplastic syndrome

Introduction: Systemic lupus erythematosus (SLE) is a complex autoimmune disease. Its diagnosis is based on clinical and laboratory criteria, with mandatory exclusion of other differential diagnoses. Paraneoplastic syndrome is a rare disorder in which there is an underlying malignancy, causing symptoms not directly related to tumour metastasis or local invasions. Paraneoplastic rheumatic syndrome closely resembles the rheumatic disease and may be accompanied by the presence of autoantibodies.

Case description: A 45-year-old woman was admitted to the Rheumatology Ward following a previous suspicion of granulomatosis with polyangiitis (GPA) and an inconclusive diagnostic evaluation during outpatient treatment. Patient's medical history included generalised joint pain with oedema, weakness, occasional low-grade fever, and recurrent epistaxis with nasal perforation. Histopathological examination of the nasal mucosa excluded findings characteristic of GPA. The patient has been treated with methotrexate and glucocorticosteroids. During the current hospitalisation, physical examination revealed no joint or upper respiratory tract inflammation. However, supraclavicular lymph nodes were enlarged, and the patient reported newly onset of dysphagia. Laboratory tests showed mildly elevated C-reactive protein, lymphopenia, positive antinuclear antibody with a homogeneous pattern and a titre of 1 : 3,200, positive anti-dsDNA and mildly elevated carcinoembryonic antigen. The patient was referred for a lymph node biopsy, which concluded third-grade adenocarcinoma. Computed tomography showed gastric wall thickening and multiple enlarged lymph nodes in the abdominal area, followed by diffuse nodules in the lungs. Gastroscopy revealed a pathological, cauliflower-like infiltrative lesion involving a greater part of the gastric body. Histopathological examination of gastric biopsy specimens confirmed gastric adenocarcinoma. Due to the advanced stage of cancer, the patient was qualified for palliative treatment.

Conclusions: Paraneoplastic rheumatic syndromes pose a diagnostic challenge because they can closely mimic SLE while presenting with few unusual features. Therefore, the exclusion of other differential diagnoses is crucial. This case highlights the necessity to reconsider the diagnosis in patients with atypical presentations, even when other diagnostic criteria are met.

Oral session 5

Cardiovascular pathologies associated with autoimmunity

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A missed pulse and recurrent stroke: delayed diagnosis of Takayasu arteritis in a young soldier

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Key words: Takayasu arteritis, large-vessel vasculitis, ischemic stroke, diagnostic delay, immunosuppression.

Introduction: Takayasu arteritis (TA) is a rare, chronic large-vessel vasculitis predominantly affecting young women at the threshold of their professional careers. Due to its non-specific clinical presentation and evolving symptoms, TA is frequently misidentified as other rheumatological conditions. The coexistence of common health issues – such as skin lesions, musculoskeletal strain or anatomical anomalies like patent foramen ovale (PFO) – can further complicate diagnosis. Delayed recognition leads to irreversible vascular damage and recurrent ischemic strokes.

Case description: A female soldier in her 20s presented with joint pain, headaches and visual disturbances. Despite early documentation of an elevated ESR and a significant inter-arm blood pressure difference (> 50 mmHg), and an initial suspicion of vasculitis, these findings were overlooked during a months-long diagnostic process across multiple facilities. Clinical focus shifted to a positive rheumatoid factor and bone lesions, suggesting SAPHO (Synovitis-Acne-Pustulosis-Hyperostosis-Osteitis) syndrome or rheumatoid arthritis. Carotid artery narrowing on CT was misinterpreted as fibromuscular dysplasia. Low-dose glucocorticosteroids (GCs) failed to halt disease progression. Within 16 days, the patient suffered two consecutive ischemic strokes. The first was attributed to a known PFO, delaying correct recognition. Only advanced imaging revealed critical inflammatory stenosis of the right common carotid artery and its branches. After unsuccessful thrombolysis, mechanical thrombectomy improved her neurological status. Intensive antithrombotic and GC therapy did not prevent a second stroke. High-dose GCs led to metabolic complications. After failing sulfasalazine, cyclophosphamide, and infliximab, tocilizumab (TCZ, later combined with methotrexate [MTX]) was introduced. Clinical stabilization and GC reduction were achieved.

Conclusions: Autoimmune markers and osteoarticular lesions can lead to premature diagnostic closure. In young stroke patients with systemic inflammation, a PFO should not preclude searching for vasculitis. Bilateral blood pressure measurement is a fundamental yet often neglected screening tool; effective inter-specialist communication is vital to integrate disparate symptoms. The combination of TCZ and MTX is an effective strategy for refractory TA, enabling disease control despite metabolic complications. Diagnostic delays drastically prolong social and professional exclusion, particularly severe and potentially irreversible for high-performance individuals.

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Atypical hemolytic uremic syndrome with complement factor I variant triggered by malignant hypertension in a patient with breast cancer: case report and review of the literature

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Key words: malignant hypertension, atypical hemolytic uremic syndrome, anti-complement therapy, complement factor I, thrombotic microangiopathy

Introduction: Malignant hypertension (MHT) is a severe form of untreated hypertension, characterised by systolic blood pressure over 200 mmHg and diastolic pressure over 120 mmHg. The MHT is associated with a poorer prognosis and more severe consequences than common arterial hypertension. It is harmful for the endothelium, causing microcirculation damage, which leads to multi-organ ischemic dysfunction, affecting many organs, i.e., the kidneys, the heart, the brain, and the retina. This condition may trigger or accompany atypical hemolytic uremic syndrome (aHUS). We present diagnostic difficulties and therapeutic approach in a patient with MHT unmasking aHUS due to a complement factor I variant.

Case description: A 44-year-old woman with no previous medical history was admitted to a local emergency room with general malaise, dyspnea and one-week lasting hematuria. Physical examination revealed blood pressure 280/150 mmHg, pale skin, and oedema affecting the lower limbs. Laboratory tests showed severe normocytic anaemia, thrombocytopenia, schistocytosis, reticulocytosis, and elevated lactate dehydrogenase activity. Kidney function was poor – serum creatinine concentration (sCr) was 5.9 mg/dl, urinalysis was abnormal, active sediment with massive erythrocyturia and subnephrotic range of proteinuria were present. The Coombs' test was negative. Daily urine output was up to 3.5 l. Ultrasound ruled out urine obstruction, edematous renal parenchyma and renal artery stenosis. Brain computed tomography scan excluded intracranial bleeding. Echocardiography showed left ventricular hypertrophy. Hypertensive crisis was diagnosed, and antihypertensive treatment was administered with improvement.

Due to suspicion of aHUS patient was referred to our hospital for consideration of anti-complement therapy (anti-C5). Kidney biopsy revealed acute ischemic glomerulopathy without thrombotic microangiopathy (TMA) but with fibroelastosis. In the meantime, sCr decreased, so anti-C5 therapy was omitted and the patient was discharged home. Within a few months, kidney function improved. Genetic testing confirmed a pathogenic variant in CFI, indicating aHUS as the cause of kidney injury. Three years later, the patient was diagnosed with left breast invasive ductal carcinoma of no specific type, which did not trigger aHUS recurrence.

Conclusions: The MHT is a well-known trigger of aHUS. This case highlights the importance of early hypertension diagnosis and management to prevent complications such as MHT that might lead to aHUS in genetically predisposed patients.

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Hepatitis E virus: an underrecognized clinical challenge

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Key words: hepatitis E virus, immunosuppression, autoimmune diseases, chronic hepatitis

Introduction: Hepatitis E virus (HEV) is a widespread, while epidemiologically underassessed, single-stranded, positive-sense RNA virus. Of the four main genotypes, genotypes 1 and 2 occur rather in developing countries, genotype 3 is most common in Europe, and genotype 4 causes infections in East Asia. Risk factors for infection between genotypes include sanitary conditions and drinking contaminated water for genotypes 1 and 2, and consumption of undercooked pork or wild boar meat for genotypes 3 and 4. Receiving blood transfusions is also associated with the risk of transmission.

Hepatitis E virus has recently become the subject of interest beyond the field of infectious disease specialists, as the focus is gradually shifting towards its occurrence in the immunocompromised. It is important to raise awareness among healthcare professionals who may encounter patients with this condition. Therefore, this review aims to provide an up-to-date synthesis of the literature on the topic.

Material and methods: The Scopus database was queried with the following search terms: "hepatitis E virus", "HEV", "rheumatology", "connective tissue disease".

Results: Of the 277 results obtained, 105 articles were then selected to be included in the review.

Discussion: While HEV infection may be asymptomatic, the typical course involves acute hepatitis. Chronic infections also occur, especially in immunocompromised individuals, which also involves those treated for rheumatologic diseases. Genotype 3 and 4 infections are associated with extrahepatic manifestations, such as neurological symptoms, renal complications, acute pancreatitis, cryoglobulinemia, and vasculitis. Molecular mimicry is the proposed mechanism underlying these conditions. The infection is usually self-limiting and does not require treatment. However, patients undergoing immunosuppressive therapy may need a dose reduction or even complete withdrawal from the therapy. Off-label use of ribavirin may be considered.

Conclusion: The HEV infection in immunocompromised patients may be severe, leading to diagnostic challenges, severe complications, and treatment dilemmas. To establish optimal prevention and treatment strategies, further research is required.

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Atypical hemolytic uremic syndrome secondary to adjuvant therapy with gemcitabine

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Key words: gemcytabine, aHUS, hemolytic, uremic, HUS

Introduction: Hemolytic uremic syndrome (HUS) usually presents with a triad of symptoms: thrombocytopenia, hemolytic anaemia, and acute kidney injury. Currently, HUS is differentiated into HUS associated with *Escherichia coli* infection and Shiga toxin production, atypical HUS associated with complement system disorders, and secondary HUS associated with infection or drug-induced HUS, etc. During the differential diagnosis, thrombotic thrombocytopenic purpura (TTP), associated with reduced ADAMTS13 (a disintegrin and metalloprotease with thrombospondin type 1 motifs, member 13) activity or deficiency, should always be ruled out.

Case description: A 63-year-old woman with a history of ductal carcinoma of the pancreatic tail, surgically removed in February 2023, was undergoing adjuvant chemotherapy with gemcitabine. Additionally, patient had bronchial asthma, hypertension, and hypothyroidism treated with hormone replacement therapy. The patient was admitted to the emergency department due to severe anaemia requiring red blood cell transfusion and biochemical evidence of kidney damage. On admission, the patient reported weakness, intermittent shortness of breath, and palpitations, and denied nausea, vomiting, or bleeding symptoms. Laboratory tests revealed anaemia, thrombocytopenia, and markers of renal damage. Urinalysis revealed asymptomatic bacteruria, hematuria, and a small amount of protein. Abdominal ultrasound revealed post-resection changes of the pancreatic body and tail, a 19-mm hypoechoic right adrenal nodule, right renal cysts, and a non-dilated pelvicalyceal system. During hospitalisation, progressive deterioration of renal function was observed despite conservative treatment of acute kidney injury, as well as worsening blood pressure control despite gradual intensification of antihypertensive therapy. A suspicion was raised of the development of atypical hemolytic uremic syndrome during the course of adjuvant chemotherapy with gemcitabine. The ADAMTS13 activity was determined at 80.6%, and stool polymerase chain reaction for Shiga-toxigenic *Escherichia coli* was negative. Four therapeutic plasmapheresis sessions were performed, but they were ineffective. An application for eculizumab treatment was submitted under the drug program, which was rejected.

Conclusions: This case report illustrates a rare but severe complication of aHUS associated with gemcitabine treatment. Gemcitabine should be discontinued immediately if characteristic symptoms are observed. Plasmapheresis treatment was found to be ineffective in achieving significant clinical improvement, suggesting a potential need for complement inhibitor therapy, particularly eculizumab.

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From Raynaud's phenomenon to digital gangrene: progressive vasculopathy in mixed connective tissue disease

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Key words: MCTD, Raynaud's phenomenon, necrosis, surgical debridement

Introduction: The Raynaud's phenomenon (RP) is often an initial symptom of mixed connective tissue disease (MCTD), and its progression to skin necrosis and gangrene remains rare and life-threatening. This report presents a case of severe microvascular angiopathy in MCTD leading to phalangeal necrosis requiring vasodilator therapy and surgical intervention.

Case description: A 53-year-old patient with MCTD was admitted to the Rheumatology Department in Lodz for disease activity assessment and treatment modification. Diagnosed in 2020, the patient initially presented with mild skin fibrosis and severe RP without necrosis. Treatment included methylprednisolone (16 mg, tapering), methotrexate (25 mg s.c.), and pentoxifylline (600 mg).

In August 2025, the patient experienced a significant worsening of skin ulcers on both hands, with dry necrosis of the distal phalanges. On admission, inflammatory markers were moderately elevated, along with anaemia of chronic disease, thrombocytosis, and cyanotic, indurated skin on the hands, feet, and face. The patient exhibited features typical of systemic sclerosis and massive, hard oedema in the lower limbs, as well as stable pulmonary emphysema and dysphagia due to esophagitis. No other organ manifestations were noted.

Prostaglandin E1 infusions were initiated for five days, improving blood supply to the distal limbs. A calcium channel blocker was added, and targeted intravenous antibiotics were administered for infected ulcers on the right hand. The patient was discharged in satisfactory condition.

Four weeks later, the patient returned for another prostaglandin cycle. Significant improvement in the skin on the face and feet was observed, along with healing of the left hand ulcers. However, the right hand's necrosis deepened, a bacterial infection reactivated, and an abscess developed. Prostaglandin infusions continued, and surgical debridement of the right hand was performed, resulting in finger amputations.

The patient's general condition improved, inflammatory markers decreased, and a follow-up a month later showed significant skin improvement, no swelling, enhanced well-being, and normal healing of amputation wounds. Prostaglandin treatment will continue for another three months, with close monitoring of wound healing.

Conclusions: This case shows that angiopathy in MCTD may progress from RP to gangrene, and that critical digital ischemia requires multidisciplinary management.

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Fulminant myocarditis: a severe manifestation of systemic lupus erythematosus

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Key words: fulminant myocarditis, systemic lupus erythematosus, acute heart failure, cyclophosphamide

Introduction: Myocarditis is a rare manifestation of systemic lupus erythematosus (SLE), affecting 5–10% of patients. It is usually subclinical but fulminant cases have been reported. Common symptoms include fever, tachycardia, chest pain and dyspnoea. Diagnosis is based on clinical presentation combined with diagnostic imaging (echocardiography, cardiac magnetic resonance imaging) and elevated cardiac biomarkers after excluding other causes.

Case description: A 35-year-old man with a history of chronic discoid lupus erythematosus (DLE) was admitted to the Rheumatology Ward due to recurrent fever, unintentional weight loss, fatigue and alopecia lasting six months. Three weeks prior to hospitalization he developed aggravation of skin lesions, facial and lower limbs oedema, muscle weakness, morning joints stiffness, exertional dyspnoea and dry cough. Physical examination revealed decreased breath sounds at lung bases and tachycardia. Point-of-care ultrasound demonstrated myocardial hypokinesis with wall thickening and pleural effusion. Laboratory tests showed elevated aspartate transaminase, creatine kinase, troponin I (TnI) and N-terminal pro-B-type natriuretic peptide (NT-proBNP), as well as decreased complement components C3 and C4. Anti-double-stranded DNA (anti-dsDNA) antibodies were positive (49 IU/ml), while C-reactive protein was relatively low (16.3 mg/l). Hyperferritinemia and hypertriglyceridemia suggested macrophage activation syndrome (MAS), but other criteria were negative. The diagnosis of SLE was confirmed (cSLEDAI-2K = 57). Initial treatment with intravenous methylprednisolone and immunoglobulins (IVIG) was ineffective. A rise in TnI (31–141 ng/l) and markedly elevated NT-proBNP (> 35,000 pg/ml) correlated with a decline in left ventricular ejection fraction (LVEF) to 15%, indicating myocarditis. The patient was referred to the intensive care unit as he required mechanical ventilation and catecholamine support. The first dose of cyclophosphamide resulted in a significant clinical response, so after 10 days the patient returned to the Rheumatology Ward. After one month of hospitalization patient was discharged with mildly reduced LVEF (45%) and no severe symptoms.

Conclusions: This case highlights that acute heart failure in SLE patients should indicate evaluation for myocarditis. Broad immunological and clinical assessment enables timely initiation of immunosuppressive therapy, including high-dose glucocorticosteroids and cytotoxic agents such as cyclophosphamide, to control immune-mediated organ damage.

Abstracts accepted for poster presentation

P_01

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Clinically aggressive rheumatoid arthritis with minimal symptomatic inflammation: the importance of imaging in early disease

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Key words: ACPA (anti-citrullinated protein antibodies), rheumatoid arthritis, erosions

Introduction: Early-phase arthritis can manifest with significant clinical aggression despite minimal symptomatic inflammation. Intervention at an early stage is critical to prevent irreversible structural damage and preserve long-term joint function.

Case description: We present the case of a 41-year-old male referred to the Rheumatologic Outpatient Clinic with a one-month history of bilateral hand and feet arthralgia. Patient reported significant morning stiffness (lasting 30–60 minutes), although nocturnal pain was absent. Physical examination revealed tenderness in several metacarpophalangeal (MCP) and metatarsophalangeal (MTP) joints; joint swelling was not present at the time of the examination. A family history revealed an aggressive form of rheumatoid arthritis (RA) in the patient's mother. The laboratory tests indicated slightly elevated values of inflammatory markers and highly elevated anti-cyclic citrullinated peptide (anti-CCP) antibodies and rheumatoid factor. The ultrasonographic examination showed no synovial hypertrophy but identified swelling of the extensor tendon of the right second MCP joint (Fig. 1), bilateral tenosynovitis of the second finger flexors and the right hallux flexor. Based on the American College of Rheumatology/European Alliance of Associations for Rheumatology criteria, the patient was diagnosed with RA. The initial treatment was methotrexate (MTX) and prednisone. Due to the patients' intolerance, MTX was changed to hydroxychloroquine. The patient remained non-compliant due to reported malaise; therefore, remained on low-dose prednisone monotherapy. Arthralgia in the hands and feet persisted. With disease progression, clinically evident inflammatory changes developed in the hands, characterised by erythema and soft tissue swelling in the metacarpophalangeal joint region (Fig. 2). After three months of ineffective therapy, an ultrasound identified erosive changes in the right second MCP joint (Fig. 3) and left second proximal interphalangeal joint. Given the aggressive clinical course and objective evidence of joint destruction, initiation of biological therapy was indicated.

Conclusions: Patients presenting with high anti-CCP titers (ACPA-positive) require prompt and intensive treatment, along with frequent rheumatologic monitoring. This case indicates the importance of musculoskeletal ultrasonography in detecting early structural changes and monitoring subclinical activity that may be overlooked by conventional laboratory tests or physical assessments.

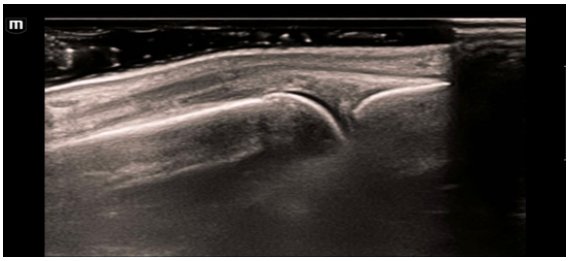

Fig. 1. Longitudinal scan of the right second metacarpophalangeal (MCP) joint from the dorsal aspect. Swelling of the extensor tendon overlying the joint is visible. No synovial hypertrophy.

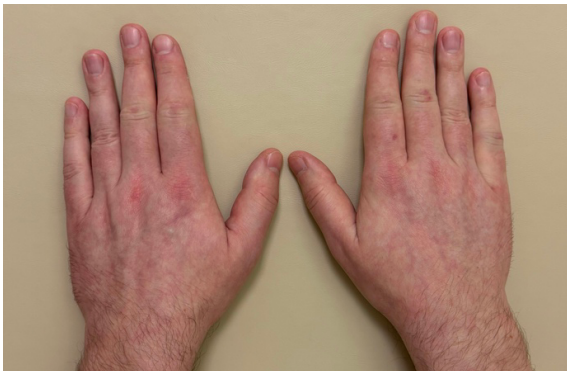

Fig. 2. Dorsal view of both hands in a patient with rheumatoid arthritis during a follow-up examination.

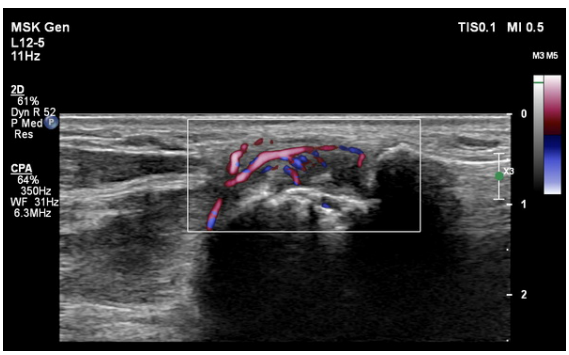

Fig. 3. Follow-up ultrasonographic examination after 3 months. Longitudinal dorsal scan of the right second MCP joint demonstrating synovitis with visible active inflammatory erosion.

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Rapidly progressive lymphocytic interstitial pneumonia with plasma cell predominance in Sjögren's disease

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Key words: interstitial lung disease, Sjögren's disease, lungs transplantation

Introduction: Pulmonary involvement is a major systemic manifestation of Sjögren's disease (SjD) and may present as interstitial lung disease (ILD) with various patterns. Lymphocytic interstitial pneumonia (LIP) is an uncommon but characteristic SjD-associated ILD, usually considered slowly progressive. However, LIP may occasionally lead to extensive cystic lung remodeling, severe impairment of gas exchange, and respiratory failure. Data on rapidly progressive, plasma cell-predominant and treatment-refractory LIP in SjD remain limited, and such cases may ultimately require lung transplantation.

Case description: A 47-year-old man with LIP and previously unexplained autoimmune features was subsequently found to have underlying SjD, followed by severe, rapidly progressive ILD. Within two years, serial high-resolution computed tomography (HRCT) scans demonstrated extensive progression to diffuse cystic remodeling with severe parenchymal destruction, affecting approximately 80% of both lungs (Fig. 1). Pulmonary function tests showed moderate restriction (forced vital capacity [FVC]: 54–60% predicted) and severely impaired diffusing capacity (diffusing lung capacity for carbon monoxide [DLCO]: 25–35% predicted, HGB = 14.7 g/dl), consistent with progressive respiratory failure. Clinically, the patient required long-term oxygen therapy and experienced marked exertional desaturation. The disease progressed despite treatment with hydroxychloroquine, cyclophosphamide, dose glucocorticosteroids, azathioprine and rituximab (anti-CD20 monoclonal antibodies). Bronchoscopy was unremarkable, and bronchoalveolar lavage revealed a low lymphocyte fraction. Histopathologic evaluation from CT – guided lung biopsy and mediastinal lymph node sampling excluded malignancy and immunoglobulin G4-related disease and supported the diagnosis of plasma cell-predominant LIP related to SjD. Due to progression and advanced hypoxemic respiratory failure, the patient was referred for lung transplantation evaluation.

Conclusions: This case demonstrates that SjD-associated LIP, although usually slowly progressive, may rarely progress into an aggressive cystic phenotype with profound diffusion defect despite immunosuppressive therapy. It also draws attention to the late diagnosis of SjD in men and confirms that men, although they are less likely to suffer from SjD, may present with a more severe course. In such patients, early recognition of treatment failure and timely referral for lung transplantation are essential to improve prognosis.

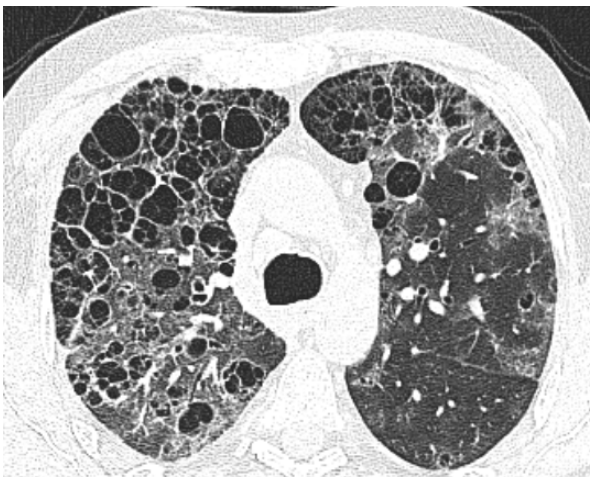

Fig. 1. High-resolution computed tomography showing diffuse cystic lung disease in Sjögren's disease-associated lymphocytic interstitial pneumonia.

Source: Department of Radiology, National Institute of Geriatrics, Rheumatology and Rehabilitation in Warsaw.

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Pulmonary hypertension and interstitial lung disease of organising pneumonia morphology as primary manifestations of seropositive rheumatoid arthritis

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Key words: rheumatoid arthritis, rheumatoid arthritis-associated interstitial lung disease, pulmonary arterial hypertension

Introduction: Pulmonary involvement is a frequent and prognostically significant extra-articular manifestation of rheumatoid arthritis (RA). Rheumatoid arthritis-associated interstitial lung disease (RA-ILD) is the most common respiratory presentation and a major contributor to morbidity and mortality. Growing evidence suggests the lungs may be a site for initiating autoimmune processes preceding clinically overt RA.

Case description: A 54-year-old female with a 14-year history of idiopathic pulmonary arterial hypertension (iPAH) presented with an acute onset of polyarthritis following an emotional stress accompanied by infection. She exhibited high disease activity (DAS28: 6.74), significant systemic inflammation (erythrocyte sedimentation rate: 96 mm/h, C-reactive protein: 105 mg/l), and high titers of autoantibodies (rheumatoid factor: 58.44 IU/ml, anti-citrullinated protein antibodies: > 500 U/ml). High-resolution computed tomography (HRCT) and lung cryobiopsy confirmed an organising pneumonia (OP) pattern (Fig. 1). Pulmonary function tests showed forced vital capacity 88% and reduced diffusion capacity of the lungs for carbon monoxide 58%. Based on clinical, serological, and radiological findings, a diagnosis of RA-ILD was established. Treatment was initiated with intravenous methylprednisolone, followed by a regimen of methotrexate, hydroxychloroquine and tapering dose of oral methylprednisolone.

Conclusions: This case highlights the diagnostic challenges of RA with pulmonary involvement. The patient's ILD, characterised by an inflammatory OP pattern on HRCT, was preceded by long-standing iPAH. This clinical sequence suggests a possible gradual evolution from a subclinical pulmonary state, such as interstitial pneumonia with autoimmune features, to clinically defined RA. Early recog-

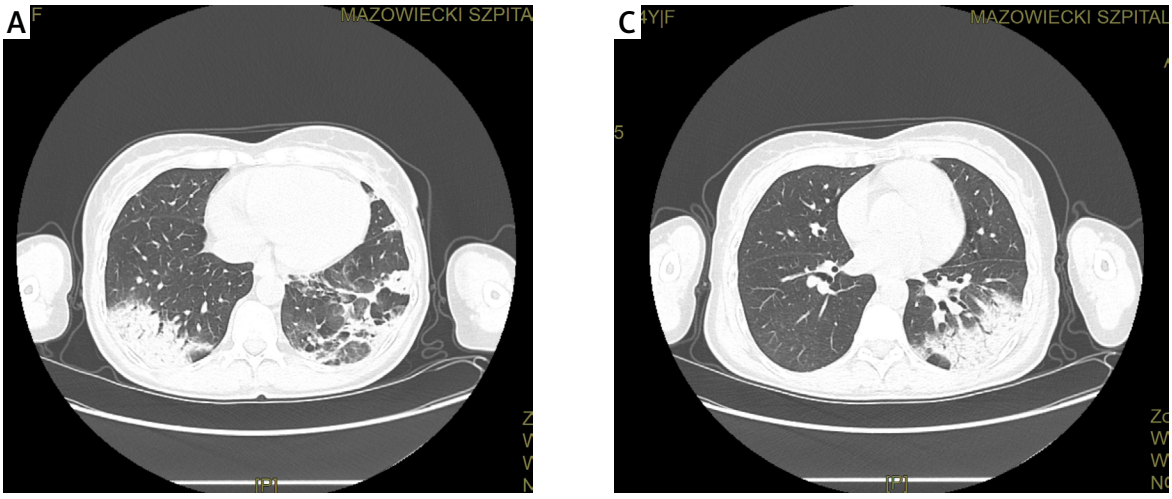

Fig. 1. High-resolution computed tomography of the chest showing extensive patchy consolidations and ground-glass opacities with air bronchograms accompanied by diffuse bronchial wall thickening and enlarged mediastinal and axillary lymph nodes.

nition of RA-ILD, enabling timely immunomodulatory and immunosuppressive treatment, is crucial for influencing the disease course and prognosis, particularly in patients with long-standing unexplained pulmonary disease.

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P_04

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Occupational silica exposure as a potential trigger of systemic sclerosis: a case report and diagnostic dilemma

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Key words: systemic sclerosis, silicosis, interstitial lung disease, Erasmus' syndrome, antifibrotic

Introduction: Systemic sclerosis (SSc) is an autoimmune connective tissue disease that may progress with multi-organ involvement. Both genetic predisposition and environmental factors are implicated in its pathogenesis. However, their exact roles remain incompletely understood.

Case description: A 59-year-old non-smoking man with a non-contributory family history who has worked as a foundry worker for 35 years, with exposure to silica-containing dust, was admitted to the clinic due to pain and swelling of the fingers, progressive skin tightness, Raynaud's phenomenon, pitted scars on the fingertips, exertional dyspnea and dysphagia. The patient reported symptom onset approximately 16 years earlier, with arrhythmia and conduction disorder requiring pacemaker implantation. One year earlier, the patient was diagnosed with dyspnea at the pulmonological department. Chest computed tomography (CT) revealed numerous enlarged, calcified mediastinal lymph nodes, pleural thickening at the lung apices and multiple, scattered, well-calcified perilymphatic nodules, predominantly in the upper lobes and subpleural regions, along with features of emphysema. Findings were consistent with silicosis given silica exposure. Bronchoscopy and lymph node cytology (BAC) were unremarkable; bronchial secretions and acid-fast bacilli microscopy were negative.

Rheumatological evaluation revealed markedly elevated inflammatory markers, normocytic anaemia, hypoalbuminemia with hypergammaglobulinemia, and an IgG κ protein with an acute-phase oligoclonal pattern. The levels of C3 and C4, as well as tumour, cardiac, and muscle injury markers, were normal. Antinuclear antibody titers were strongly positive (1 : 5,120) with the presence of anti-Scl-70 and anti-Pm-Scl-100 Abs. Urinalysis showed no proteinuria. Nailfold capillaroscopy indicated SSc, corresponding to the late phase according to Cutolo (Figs. 1,2). In addition to silicosis-related findings, CT demonstrated basal ground-glass opacities consistent with SSc-related non-specific interstitial pneumonia (Fig. 3). The patient met the 2013 American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria for the diagnosis of SSc.

Conclusions: Long-term occupational exposure, including high silicon content, may contribute to the development of SSc. Abnormalities in lung imaging may be both a consequence of SSc and a result of environmental factors. It remains unclear whether the clinical presentation is attributable to chemical exposure (Erasmus' syndrome) or represents pulmonary involvement in the course of overlap syndrome. Treatment decisions, including antifibrotic therapy, require prior evaluation of pulmonary changes.

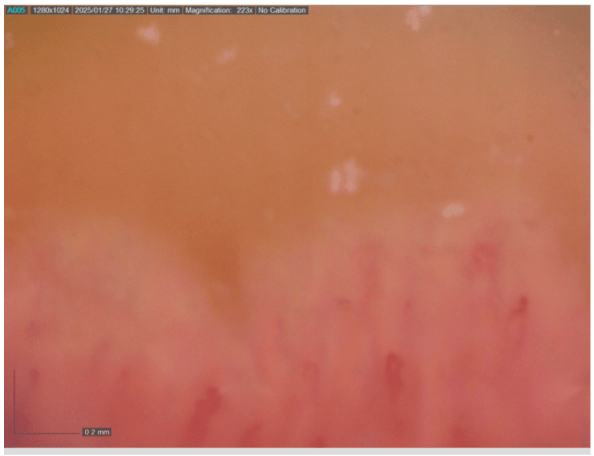

Fig. 1. Nailfold capillaroscopy demonstrating reduced capillary density and disorganised capillary architecture consistent with the late systemic sclerosis pattern.

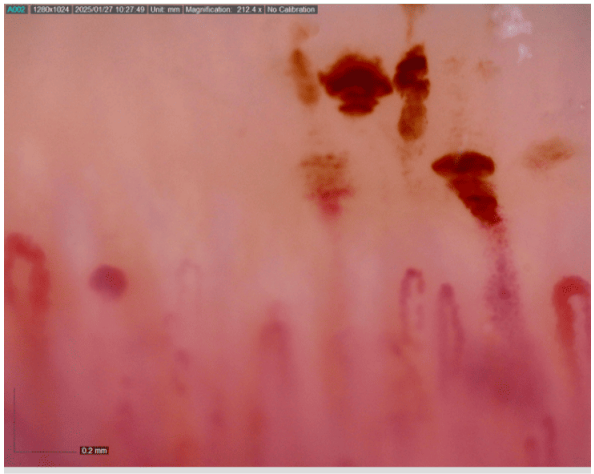

Fig. 2. Nailfold capillaroscopy showing giant capillaries and capillary haemorrhages.

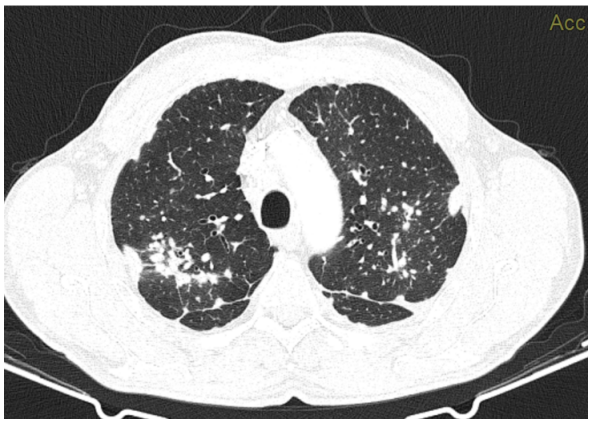

Fig. 3. Axial chest CT showing calcified perilymphatic nodules consistent with silicosis and basal ground-glass opacities suggestive of non-specific interstitial pneumonia.

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Therapeutic challenges of concurrent pneumonia and disease flare in a patient with Still's disease and recurrent macrophage activation syndrome

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Key words: Still's disease, MAS, AOSD, leflunomide, tocilizumab, pneumonia

Introduction: Still's disease belongs to non-familial autoinflammatory systemic disorders. Prior distinction of this condition into two separate entities: the systemic juvenile idiopathic arthritis (sJIA) and adult-onset Still's disease (AOSD), is obsolete, and European Alliance of Associations for Rheumatology recommends that it should be considered as one disease.

Case description: Patient at age 30, diagnosed with sJIA at 4. The first treatment was high doses of glucocorticosteroids (GCs). At that time, the patient 3 times develop macrophage activation syndrome (MAS). Since 2016, methotrexate (MTX) was prescribed, and in 2022, leflunomide was added as a second drug, but the patients took them irregularly. In 2022, due to a persistent subfebrile state and recurrent arthritis, he was qualified for treatment with tocilizumab at a dose of 400 mg applied in infusions every 4 weeks. Due to a remission state since 2023 multiple attempts to prolong the time between applications were made, but they failed. Finally, in 2025, the duration between dosages was successfully extended to 8 weeks. During therapy, the prior treatment with MTX was recommended but not taken by patients. In January 2026, the patient reported pain in multiple joints, fever, sore throat and rash on his trunk. In addition, patient complains of severe pain in his chest. In laboratory tests, the inflammatory indicators were very high. In the high-resolution computed tomography of the chest, the symptoms of pneumonia were described. Treatment with levofloxacin and a pulse of i.v. methylprednisolone has been introduced with subsequent treatment with prednisolone at a dose 1 mg/kg/day. The regression of changes in the lungs was achieved, the fever did not subside, and inflammatory indicators were still high, pointing to the development of MAS. After administration of tocilizumab, due anakinra was not available, complete remission was achieved. Although the interval from the end of pneumonia treatment was only one week. Due to the reluctance to use MTX, treatment with cyclosporine at a dose of 3 mg/kg/day was proposed.

Conclusions: The occurrence of severe pneumonia raises concerns about the safety of further tocilizumab treatment. A possible therapeutic option for this patient is interleukin-1 (IL-1) blockade, e.g. anakinra, which, according to the recommendations of the British Society for Rheumatology, is preferred in severe systemic disease over IL-6 blockade. In addition, it has been proven to be safe to use anakinra even in patients with sepsis, so it could be a therapeutic option in this case. Similar data for the use of tocilizumab in bacterial infection is lacking.

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A diagnostically challenging case of atypical hemolytic uremic syndrome in the course of severe hypertension

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Key words: aHUS, hypertension, AKI, hemolytic anaemia

Introduction: Atypical hemolytic uremic syndrome (aHUS) is a rare, progressive, often genetic disease that can be fatal if untreated. It may manifest with proteinuria, hematuria without acute kidney injury (AKI). It frequently leads to refractory hypertension, AKI and myocardial infarction or encephalopathy.

We present a case of a 51-year-old patient, treated twice in the Clinic of Internal Medicine, Nephrology and Dialysis Therapy of the Military Institute of Medicine – National Research Institute, with refractory hypertension and AKI, who developed a-HUS, in the course of hypertensive nephropathy with renal failure.

Case description: A 51-year-old patient presented to the Emergency Department with a 1-month history of abdominal pain and intermittent vomiting for 5 months. On admission, the patient was in good general condition but in hypertensive crisis (blood pressure: 248/145 mmHg, heart rate: 121 bpm) requiring intravenous urapidil infusion. Severe renal failure was identified with a creatinine level of 5.7 mg/dl, urea 140 mg/dl and hypokalemia K^+ 2.2 mmol/l. Furthermore, normocytic anaemia, thrombocytopenia, proteinuria and hematuria were observed. A multidrug antihypertensive regimen was initiated. The patient developed atrial fibrillation during hospitalisation, managed with amiodarone; echocardiography showed features of left ventricular hypertrophy, and head computed tomography showed diffuse white matter hypodensity. Labs indicated secondary hyperparathyroidism. A right kidney biopsy was performed, complicated by retroperitoneal hematoma with subsequent hypotension and anemization, however without signs of active bleeding. Amoxicillin with clavulanic acid was included in addition to 3 units of packed red blood cells. After radiologic consultation, angiography with additional renal artery embolization. The 50% hemolytic complement (CH50) and a disintegrin-like and metalloprotease with thrombospondin type 1 motif 13 (ADAMTS13) activity levels are not without abnormalities. Genetic screening for aHUS was initiated. Renal biopsy histopathology revealed arterionephrosclerosis, pointing to a hypertensive aetiology. Intravenous iron and folic acid supplementation were started for the mixed-aetiology anaemia. A month later readmission was due to high renal parameters, and acidosis was identified, along with anaemia and thrombocytopenia. Low complement C3 level 77 mg/dl. Genetic testing confirmed aHUS. However, the patient was not qualified for ravulizumab therapy by the National Commission.

Conclusions: The aHUS is a diagnostically challenging disease due to non-specific symptoms and multi-organ manifestations. This case illustrates one of the mechanisms in which aHUS may develop in the course of severe hypertension, which, in the setting of aHUS, was particularly refractory to treatment.

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Unmasking the hidden cause of muscle weakness in a patient with previously amyopathic dermatomyositis: a diagnostic and therapeutic challenge

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Key words: dermatomyositis, muscle weakness, nonspecific interstitial pneumonia

Introduction: Dermatomyositis (DM) is a systemic autoimmune disorder characterised by symmetrical muscle weakness and pathognomic skin lesions. Interstitial lung disease frequently coexists. Notably, in up to 20% of patients, DM can present as an amyopathic form, in which characteristic skin findings occur in the absence of clinical or laboratory evidence of muscle involvement.

Case description: A 62-year-old male was admitted to the Rheumatology Ward with decreasing exercise tolerance and cough. Dermatological findings included mechanic's hands, Gottron's papules over joints, and hyperkeratosis with fissuring of the fingertips. High-resolution computed tomography (HRCT) revealed fixed interstitial lung changes consistent with fibrosing nonspecific interstitial pneumonia. Pulmonary function tests showed significant impairment of the transfer factor of the lung for carbon monoxide (TLCO), measuring 48% of the expected value. Laboratory tests showed mildly elevated aldolase and C-reactive protein, with positive antinuclear antibodies (anti-MDA5, anti-PM-Scl-100, anti-PM-Scl-75, anti-Ro-52, anti-SS-B). A diagnosis of clinically amyopathic dermatomyositis was established. The patient received intravenous methylprednisolone and cyclophosphamide (6 monthly 1,000 mg pulses). On assessment after completing the cycle, he developed new-onset, rapidly progressing proximal muscle weakness. Cervical spine magnetic resonance imaging revealed severe C3–C4 stenosis with myelopathy and oedema of the nucleus pulposus. Pulmonary changes on HRCT improved, and TLCO function was 63% of the expected value. The patient subsequently underwent a laminectomy, followed by rehabilitation and continuation of his treatment. Methotrexate and nintedanib were initiated, and at a 1.5-year follow-up, the patient demonstrated further pulmonary and functional improvement (TLCO: 81% of the expected value). The patient was enrolled in a drug program and started on, which stabilised the disease course.

Conclusions: Rheumatological conditions can exhibit atypical or nonspecific presentations, often complicating timely and accurate diagnosis. In the described case, a comprehensive diagnostic evaluation enabled the implementation of targeted therapy, resulting in disease remission.

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Paraneoplastic systemic lupus erythematosus in association with colon cancer: case report

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Key words: systemic lupus erythematosus, colon cancer, paraneoplastic syndrome, autoimmune disease

Introduction: Systemic lupus erythematosus (SLE) is a chronic, multi-organ inflammatory disease of autoimmune origin. In some cases, SLE may be the first symptom of cancer. Paraneoplastic syndromes in rheumatology manifest as symptoms of inflammation of joints, muscles, and blood vessels caused by cancer, but not directly related to tumour invasion or the presence of metastases. Systemic lupus erythematosus as a paraneoplastic syndrome was observed in patients with lymphoma, breast cancer, lung cancer, and, rarely, gastrointestinal cancer.

Case description: We present the case of a 69-year-old man who was admitted to the rheumatology clinic in September 2025 with an inflammatory rash on his face, neck, upper limbs and back, as well as oral ulcers, arthritis and weight loss. Laboratory tests revealed pancytopenia, low complement C3 and C4, positive antinuclear antibodies, and positive anti-ribosomal protein P. The patient fulfilled the 2019 European Alliance of Associations for Rheumatology/American College of Rheumatology classification criteria for SLE. Thoracic, abdominal and pelvic computed tomography scans showed pleural effusion, a single enlarged mediastinal lymph node, irregular thickening of the splenic flexure of the colon, and splenomegaly. Colonoscopy revealed a polypoid lesion in the distal transverse colon and descending colon, measuring approximately 3 cm; histopathological examination showed a tubular adenoma of the large intestine with high-grade dysplasia and a focus of invasion (adenocarcinoma not otherwise specified [NOS], low grade).
The treatment consisted of glucocorticosteroids in high doses and hydroxychloroquine. As a result of the treatment, the skin and mucosal lesions, as well as arthritis, significantly decreased. In December 2025, a partial colon resection was performed. Since the operation, there have been no signs of SLE activity.

Conclusions: Any systemic connective tissue disease, including SLE, can be a paraneoplastic syndrome. Particular attention should be given to the possibility of cancer in cases involving severe skin lesions, general symptoms, and older age at the onset.

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