CLINX RHEUMATOLOGY
Case Discussions

2026 BUDAPEST 6-8 March

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Invitation

Committees

Scientific Program

Abstracts

Dear Esteemed Professors and Colleagues,

It is with great pleasure and honor that I invite you to the Budapest Rheumatology Congress, an international scientific meeting dedicated to sharing the latest developments, clinical experiences, and advanced therapeutic approaches in the field of rheumatology.

As one of Europe's most important centers of science and culture, Budapest—renowned for its rich historical and cultural heritage—provides an exceptional setting for this congress, which offers a valuable platform for the exchange of scientific knowledge and clinical experience.The scientific program will encompass a wide range of topics, including the pathogenesis of rheumatic diseases, advanced diagnostic approaches, biologic and targeted therapies, and emerging innovative treatment modalities. In addition, the congress aims to promote interdisciplinary dialogue and to foster national and international collaborations that will contribute to the advancement of clinical practice and patient care.

With the contributions of leading experts in the field, the presentation of up-to-date research findings, and interactive scientific sessions, I believe this congress will provide a strong foundation for groundbreaking discussions in both basic science and clinical applications. I am confident that the scientific synergy arising from the collaboration of young researchers and international centers will offer meaningful and long-term contributions to rheumatology practice.

I would like to sincerely thank the members of the organizing committee, all participants, and everyone involved for their efforts in bringing this meaningful event to life. I am confident that the Budapest Rheumatology Congress will be a scientifically enriching, professionally rewarding, and socially memorable experience for all attendees.
With my kind regards and best wishes to you all,

Oğuz Gürler, MD
VM MedicalPark, Samsun

Endre Péter, MD
Liv Duna Medical Center

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- Réka Selyem, Hungary

**The names above are listed in alphabetical order by surname.*

Scientific Program

6 MARCH 2026, FRIDAY

08:50-09:00 Opening Session Oğuz Gürler, Endre Péter

09:00-10:30 SESSION 1

Rheumatoid Arthritis (RA) From Bench to Bedside

Chair: ***İhsan Ertenli, Gábor Hartman***

09:00-09:20 Extra Articular Features of RA - ***Ediz Dalkılıç***

09:20-09:40 Summary of Treatment Guidelines - ***Büşra Fırlatan***

09:40-10:00 Biologic Treatment: Cycling or Switching - ***Nevsun İnanç***

10:00-10:20 Treatment of RA Associated Interstitial Lung Disease - ***Levent Kılıç***

10:20-10:30 Discussion

10:30-11:00 Coffee Break

11:00-12:30 SESSION 2

Psoriatic Arthritis

Chair: ***Duygu Ersözlü, Endre Péter***

11:00-11:20 Pathogenesis of Psoriatic Arthritis - ***Umut Kalyoncu***

11:20-11:40 Pharmacoeconomic Acquisitions with Biosimilars - ***Nida Konacaklı***

11:40-12:00 EULAR 2025 Guideline - ***Ali Aytuğ Kuştaş***

12:00-12:20 TNFİ & Jaki: Which Treatment for Which Patient? - ***Büşra Fırlatan***

12:20-12:30 Discussion

12:30-13:30 Lunch

13:30-15:00 SESSION 3

Appoach to Autoinflammatory Diseases

Chair: ***Yusuf Karabulut, Réka Selyem***

13:30-13:50 Clinical Features of Familial Mediterranean Fever - ***Şeyda Zengin Acemoğlu***

13:50-14:10 Treatment of FMF: IL-1 Inhibitors and Colchicine - ***Orhan Zengin***

14:10-14:30 Update on Still's Disease - ***Emrah Koç***

14:30-15:00 Coffee Break

15:00-16:30 SESSION 4

Safety Issues in Rheumatology

Chair: ***Sercan Gücenmez, Suade Özlem Badak, Dezső Dávid***

15:00-15:20 Cardiovascular Safety of Jak inhibitors - ***Barış Gündoğdu***

15:20-15:40 Safety of Biological and tsDmards in Pregnancy and Lactation - ***Ali Aytuğ Kuştaş***

15:40-16:00 Malignancy Screening: Turkish Rheumatology Association Guideline - ***Buğu Bulat***

16:00-16:20 Screening of Tbc and Hepatitis - ***Zehra Özsoy***

16:20-16:30 Discussion

7 MARCH 2026, SATURDAY

09:00-10:30 SESSION 5

Axial Spondyloarthritis

Chair: ***Timur Pırıldar, Kamil Nas***

09:00-09:20 Update on Pathogenesis - ***Ali Şahin***

09:20-09:40 Treatment Targets - ***Servet Akar***

09:40-10:00 Imaging Modalities - ***Atalay Doğru***

10:00-10:20 New Treatment Guidelines: ACR & EULAR & APLAR 2025 - ***Hasan Satış***

10:20-10:30 Discussion

10:30-11:00 Coffee Break

11:00-12:30 Session 6

Biosmilars in Rheumatology

Chairs: ***İhsan Ertenli, Zevcet Yılmaz, Pál Géher***

11:00-11:20 Biosmilars: Development Steps - ***Hatice Ecem Konak***

11:20-11:40 Extrapolation & Interchangeability and Substitution Concepts - ***Duygu Kurtuluş***

11:40-12:00 Clinical Subtypes and Treatment Selection - ***Sibel Bakırcı***

12:00-12:20 Real World Data - ***Hakan Apaydın***

12:20-12:30 Discussion

12:30-13:30 Lunch

13:30-15:00 SESSION 7

Recent Developments in Connective Tissue Diseases

Chair: ***Süleyman Özbek, Ömer Kuru, György Nagy***

13:30-13:50 SLE: Organ Specific Treatment Approach-New EULAR Guideline – ***Mete Kara***

13:50-14:10 Sjogren Syndrome: Treatment Update and New Treatments for Near Future - ***Fatih Sarıtaş***

14:10-14:30 Systemic Sclerosis: Treatment of IS Lung Disease - ***Duygu Tecer***

14:30-14:50 Clinical Significance of Myositis Specific Autoantibodies - ***Berkan Armağan***

14:50-15:00 Discussion

15:00-15:30 Coffee Break

15:30-17:00 SESSION 8

Large Vessel Vasculitis

Chair: ***Göksel Keskin, Dezső Dávid***

15:30-15:50 Diagnosis and Treatment Update on PMR & GCA - ***Müge Aydın Tufan***

15:50-16:10 Imaging Modalities in Large Vessel Vasculitis - ***Gökhan Sargın***

16:10-16:30 Tnfi in the Treatment of LVV - ***Özgül Soysal Gündüz***

16:30-16:40 Discussion

8 MARCH 2026, SUNDAY

09:00-10:30 SESSION 9

Oral Presentations

Chairs: ***Özlem Kılıç, Pınar Talu, Sevtap Şimşek, Şeyda Zengin Acemoğlu***

10:30-11:00 Coffee Break

11:00-12:30 SESSION 10

Poster Presentations

Chair: ***Adem Küçük, Sevinç Can Sandıkçı, Volkan Yılmaz***

12:30-13:30 Lunch

Abstracts

PP-1

TNFRSF11A (RANK) Exon 5 Variants Define a Milder Clinical Phenotype and Favorable Methotrexate Response in Early Rheumatoid Arthritis

Gizem Nur Onay Celik[1], Ozgur Erkal[2], Ulku Ucar[3], Oznur Kutluk[3], Gokhan Sargin[4], Sibel Bakirci[5]

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Background

Rheumatoid arthritis (RA) is a clinically heterogeneous disease in which early identification of prognostic biomarkers is essential for precision management. Beyond HLA-associated risk, non-HLA genetic pathways—particularly the RANK/RANKL axis—play a central role in osteoclastogenesis, structural damage, and inflammatory regulation. However, the clinical implications of TNFRSF11A (RANK) polymorphisms in early RA remain insufficiently characterized.

Objectives

To investigate the association between TNFRSF11A exon 5 variants and disease phenotype, serological status, and methotrexate (MTX) response in treatment-naïve early RA patients.

Methods

This prospective cohort study included 57 newly diagnosed, DMARD-naïve RA patients fulfilling 2019 ACR/EULAR criteria. TNFRSF11A exon 5 variants (c.521+235T>C, c.428-106C>T, c.428-338G>A) were identified by PCR and direct sequencing. Disease activity (DAS28-CRP, CDAI, SDAI), serological markers, and inflammatory parameters were assessed at baseline and after six months of MTX therapy. Effect size (Cohen's d) and post-hoc power analyses were applied to support interpretation of low-frequency variants.

Results

TNFRSF11A exon 5 variants were detected in 18% of patients (n=10). Variant carriers demonstrated a distinct clinical profile characterized by a higher proportion of anti-CCP seronegativity (50% vs. 27.7%) and numerically lower baseline disease activity (mean DAS28-CRP 4.16 vs. 4.35). Both groups achieved significant improvement with MTX; however, variant-positive patients showed a consistently lower disease activity at six months and a reduced need for biologic escalation (20% vs. 27.7%). The effect size for DAS28-CRP change (Cohen's d = 0.11) suggested a subtle but coherent phenotypic modulation.

Conclusion

TNFRSF11A exon 5 variants may delineate a milder, predominantly seronegative RA phenotype with favorable response to conventional therapy. These findings support the integration of RANK-related genomic markers into early RA stratification strategies and reinforce the broader precision-medicine framework in inflammatory rheumatic diseases.

Keywords

Rheumatoid arthritis; TNFRSF11A; RANK; genetic polymorphism; seronegative RA; precision medicine

PP-2

A Case of 'The Stiff Skin Syndrome' Presenting with Scleroderma-Like Skin Findings

Ismail Ucar

Mersin City Education and Research Hospital

INTRODUCTION:

"Stiff Skin Syndrome" is a condition resembling scleroderma, beginning in infancy or early childhood, characterized by stiff skin, limited joint mobility, and moderate hypertrichosis, with no organ or muscle involvement or immunological changes, primarily affecting the skin. The extra-cutaneous finding is joint contractures, particularly affecting the large joints. Consequently, patients may develop scoliosis, toe-walking, and chest wall narrowing. Restrictive pulmonary changes and growth retardation may be seen in patients. The disease is slow-progressing and not fatal. We present a case treated with a diagnosis of scleroderma at other centers but whose clinical history and findings are consistent with "The Stiff Skin Syndrome."

CASE:

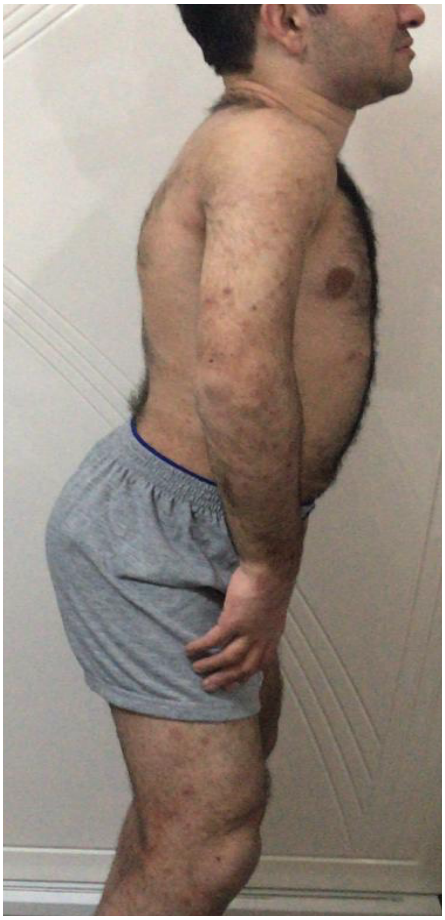
A 28-year-old male civil servant presented with complaints of skin stiffness that began primarily in the shoulders, waist, and hands at the age of 5 and subsequently developed in the trunk, neck, face, and lower extremities. The patient, who had previously been followed up by dermatology, first consulted rheumatology approximately 8 years ago and was diagnosed with systemic sclerosis. Due to the widespread nature of the skin findings, the patient received 12 courses of cyclophosphamide treatment and was followed up for 1 year with azathioprine treatment. Subsequently, the patient, who did not undergo regular follow-ups, was referred to our clinic for further treatment due to joint limitations and progression of skin hardening. The patient did not report Raynaud's phenomenon or digital ulcers during questioning. Physical examination revealed thickening and hardening of the skin on the patient's upper and lower extremities, face, and trunk. Respiratory and cardiac examinations were normal. Laboratory tests showed negative results for ANA, Scl-70, and Anticentromere Ab.

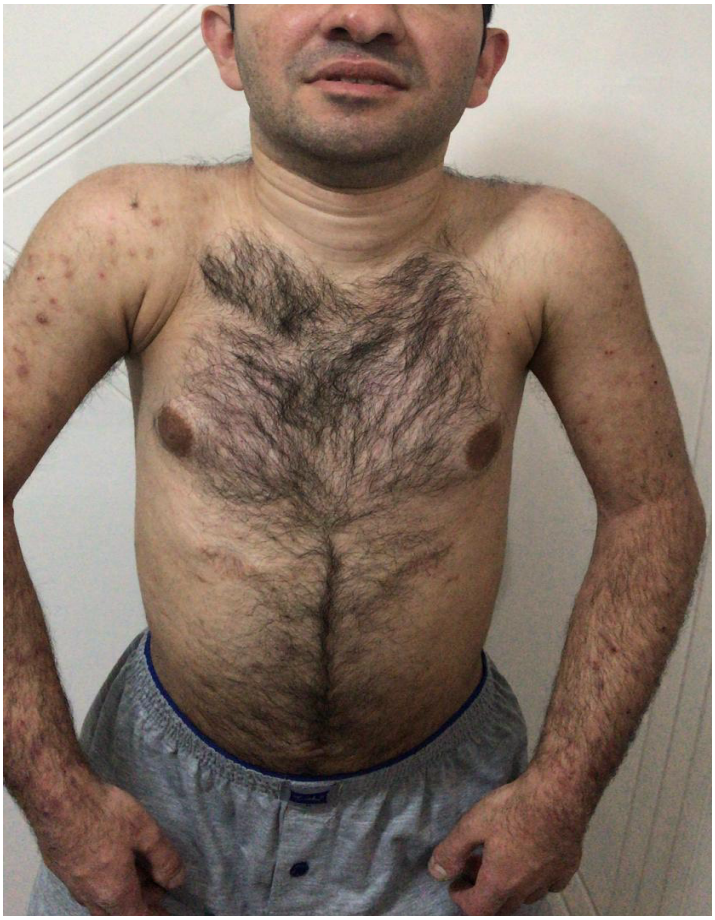
Scans were performed to check for organ involvement in systemic sclerosis. No pathology was detected on chest CT. Upper GI endoscopy performed on the patient complaining of difficulty swallowing revealed no pathology other than gastritis. Transthoracic ECHO showed no valve lesion or PAB elevation.

Despite skin changes on physical examination, systemic sclerosis was not considered due to the absence of findings of vasculopathy, inflammation, and autoimmunity, and because the skin changes were more proximal than distal. Given the patient's early onset history and the absence of visceral organ involvement, ‘The Stiff Skin Syndrome’ was considered.

DISCUSSION:

Stiff skin syndrome is a rare skin disease characterized by thickening of the fascia. Clinical symptoms include thickening of the skin, increased hair growth, and limited joint movement, with findings appearing at birth or in early childhood. The condition usually begins in the hips and slowly spreads to the extremities and trunk. Skin biopsy reveals noninflammatory, thickened, and hyalinized fascia. Due to overlapping clinical features, scleroderma is the first differential diagnosis that comes to mind. Scleroderma is a disease characterized by fibrosis in the connective tissues of the skin and internal organs. The presence of Raynaud's phenomenon, nail bed changes on capillaroscopy, organ involvement, and immunological changes are important in the differential diagnosis.





PP-3

Title

İnterstitial Lung Disease as a Presenting Feature of Sjögren's syndrome:The Importance of Integrating Clinical,Laboratory,and Radiological Findings

Authors

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Aim

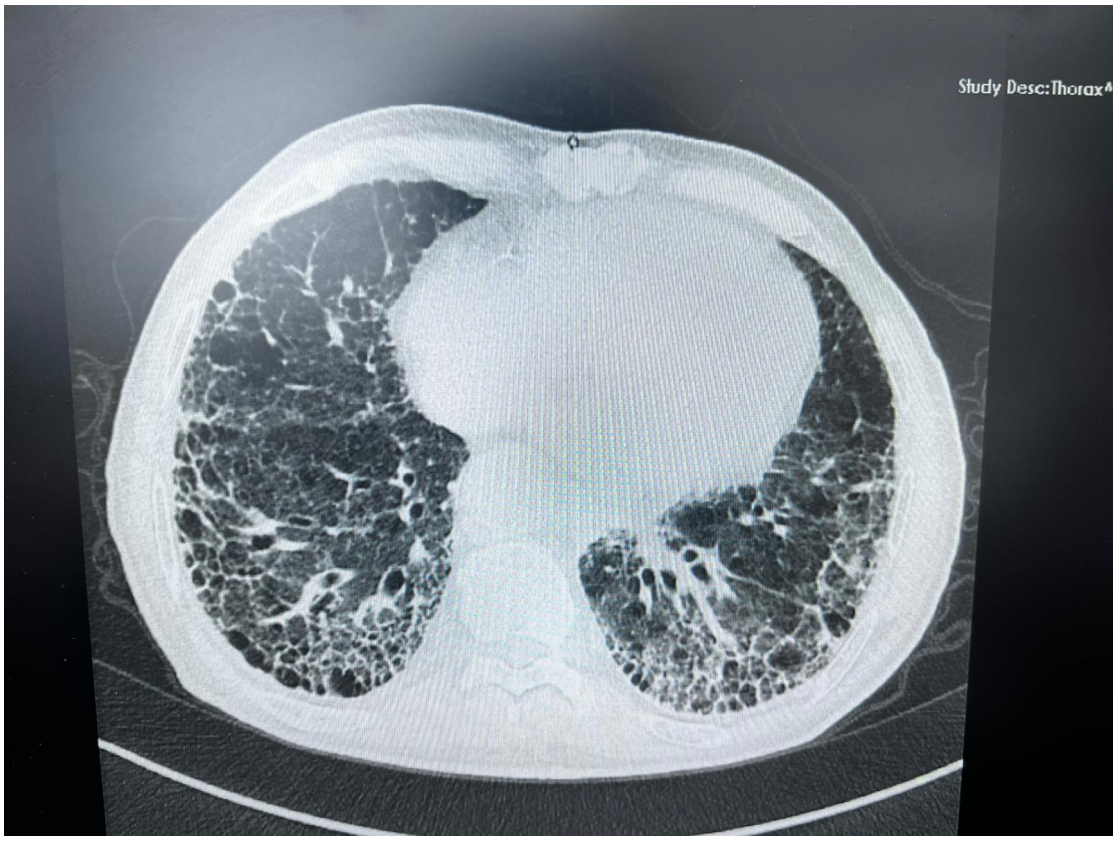
To highlight the importance of integrating clinical findings with laboratory parameters in the diagnosis of Rheumatologic syndromes, such as Sjogren's syndrome.

Introduction

Sjögren's syndrome is a systemic autoimmune disease that can involve multiple organs, including the lungs. Interstitial lung disease may precede or overshadow classical sicca symptoms, making diagnosis challenging. This case demonstrates the role of clinical judgment and radiological findings in guiding diagnosis and management beyond serological results alone.

Case Discussion

We describe a 63-year-old patient who presented with shortness of breath. Her medical history was significant for COVID-19 infection that had resolved and rheumatoid arthritis. Treatment included methotrexate, folic acid (Folbion), and sulfasalazine. Hydroxychloroquine (Plaquenil) had been previously attempted but discontinued due to adverse effects.The patient's Spirometry revealed a restrictive pattern. Laboratory evaluation showed elevated CRP ,and erythrocyte sedimentation rate. Serological testing showed positivity for rheumatoid factor(RF), anti-SSA (Ro), and anti-SSB (La) antibodies. On physical examination, the patient had joint tenderness without swelling, and rheumatoid arthritis was not initially considered clinically.Thoracic computed tomography revealed a honeycomb pattern consistent with interstitial lung disease. Based on the radiological findings, serological profile, and clinical presentation, Sjögren's syndrome was considered as a diagnosis. The patient was treated with mycophenolate mofetil and rituximab, targeting interstitial lung involvement.



Conclusion

Patients should be evaluated not only based on laboratory parameters but also with clinical and radiological findings. In this case, pulmonary involvement played a key role in suggesting Sjögren's syndrome. Treatment was initiated with consideration of interstitial lung disease.

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PP-4

Ankylosing spondylitis and anterior uveitis: single-center data

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Objectives: Acute anterior uveitis is the most common extra-articular manifestation of ankylosing spondylitis (AS). It occurs in up to 30% of patients during the disease course, and this rate can rise to 50% with longer follow-up. (1) The incidence of uveitis increases with the duration of the disease. Uveitis can be the first sign of spondyloarthritis (SpA), and studies have shown that previously undiagnosed SpA is present in 40-50% of patients who present with uveitis. (2,3) AS tends to occur earlier in patients who are HLA-B27 positive, and this condition is more prevalent in men. Uveitis is usually unilateral, non-granulomatous, acute anterior uveitis. HLA-B27 positivity is associated with an increased frequency of exacerbations. It is important to note that uveitis is not associated with the severity or progression of joint involvement. (4) Patients usually experience sudden eye pain, redness, sensitivity to light (photophobia), and decreased vision. Acute anterior uveitis attacks related to AS often respond well to short-term treatment with topical corticosteroids and cycloplegic agents. Most patients achieve full vision recovery within 2 months after the exacerbation resolves. (1) There is no correlation between ocular involvement and joint symptoms. Short-term treatment with topical corticosteroids and cycloplegic agents can effectively control uveitis attacks. For cases that are resistant to these treatments, local or systemic corticosteroid therapy is recommended. Nonsteroidal anti-inflammatory drugs (NSAIDs), disease-modifying antirheumatic drugs (DMARDs), methotrexate, azathioprine, anti-IL-17A monoclonal antibodies, and TNF-α antagonists are all effective treatments for both the ocular and systemic manifestations of AS.

Patients and methods: The study involved 20 patients with ankylosing spondylitis and anterior uveitis at our center, and data were reviewed retrospectively. All patients were over 18 years of age, and those with uveitis due to causes other than AS were excluded.

Results: The study included 5 female patients (%25) and 15 male patients (%75). The average age of the participants was 41 ± 9 years. The mean age at diagnosis for ankylosing spondylitis (AS) was 37.8 ± 8.1 years, and for uveitis, 37.8 ± 8.7 years. The average age at disease onset was 4.9 years. HLA-B27 and ANA were positive in %25 of the patients. All patients had abnormal sacroiliac MRI, with %80 exhibiting signs of active sacroiliitis and %20 showing signs of chronic sacroiliitis. None of the patients exhibited other extra-articular manifestations. One patient presented with enthesitis, while 14 patients (%70) reported peripheral arthralgia. Regarding medications used before uveitis diagnosis, 13 patients (%65) had been treated with sulfasalazine, 3 (%15) with methotrexate, and 4 (%20) with leflunomide. Regarding biological therapies, 3 patients (%15) had received adalimumab, 2 patients (%10) had received certolizumab, and 2 patients (%10) had received secukinumab. At the time of uveitis diagnosis, 7 patients (%35) were on adalimumab, 1 patient (%5) on infliximab, 2 patients (%10) on certolizumab, and 1 patient (%5) on golimumab. The remaining patients had received local treatments. At the last follow-up visit, 1 patient (%5) had active uveitis, and 18 patients (%90) were inactive. The current status of one patient is unknown due to discontinuation of follow-up. At the last visit, the patients' baseline mean score was 3.2 ± 0.7.

Discussion: Delayed diagnosis of spondyloarthritis (spA) is linked to severe functional impairment, rapid radiographic progression, poor quality of life, and a decreased response to treatment. (5) From this perspective, although our sample size was small, we observed that patients experienced delays in receiving their AS diagnosis. Almost all patients were diagnosed with AS during an episode of uveitis. In contrast to the existing literature, we found HLA-B27 positivity in a smaller percentage of patients, but the limited sample size hindered any meaningful comparison with the general population. Additionally, sulfasalazine has been shown to reduce both the frequency and severity of uveitis relapses. (6) In our study, a group of patients initially received SLZ but were later switched to biological agents. Anti-TNF-α therapy is recommended for severe cases. All four TNF-α antagonists are similarly effective in managing spinal manifestations and uveitis associated with ankylosing spondylitis (AS). However, infliximab and adalimumab are more effective in treating acute uveitis. In fact, these two agents, especially infliximab, have been shown to significantly reduce the frequency of uveitis flare-ups. (7) Although etanercept is part of the same drug class, it is ineffective for treating ocular inflammation and, paradoxically, has been reported to cause uveitis. (8) In our study, 11 patients (55%) were started on anti-TNF therapy, primarily with adalimumab. The remaining patients received topical treatments. None of the patients had used etanercept before or after developing uveitis. Only one patient experienced refractory uveitis and had been treated with both adalimumab and infliximab. All other patients were in remission.

In conclusion, early diagnosis and prompt initiation of treatment for ankylosing spondylitis are essential for managing extra-articular manifestations. While the prognosis for anterior uveitis is generally favorable, uveitis flare-ups can significantly impact patients' quality of life. Therefore, early diagnosis, effective treatment, and collaboration among specialists are crucial for managing this disease.

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