Archives of the Balkan Medical Union
vol. 61, no. 1, pp. 118-125
March 2026
Copyright © 2026 Balkan Medical Union

CASE REPORT

PROGRESSIVE FIBROSING INTERSTITIAL LUNG DISEASE IN RHEUMATOID ARTHRITIS WITH ANTISYNTHETASE AUTOIMMUNE OVERLAP

Stefan SANDULACHE[1]✉

[1] ELYTIS Hospital, Iasi, Romania

Received 2nd Febr 2026, Accepted 15th Febr 2026
https://doi.org/10.31688/ABMU.2026.61.1.15

Abstract

Introduction. Fibrosing interstitial lung diseases (fILDs) are a severe pulmonary manifestation of systemic autoimmune disorders. Autoimmune overlap syndromes, including rheumatoid arthritis (RA) associated with Sjögren's syndrome and antisynthetase syndrome, are uncommon and confer an increased risk of progressive fILD.
Case presentation. A 56-year-old Caucasian man, with a 20-year history of RA and no previous respiratory assessment, presented with progressive dyspnoea. High-resolution computed tomography (HRCT) demonstrated an usual interstitial pneumonia (UIP) pattern. Pulmonary function tests revealed initially preserved spirometry with impaired gas transfer, followed by a mild restrictive pattern during follow-up. Immunological profiling identified anti–PL-12 antibodies together with anti–Ro52 and anti–SS-A and anti–SS-B antibodies, consistent with an autoimmune overlap phenotype. The multidisciplinary management included antifibrotic therapy with nintedanib combined with immunosuppressive treatment, leading to radiological and functional stabilisation at 6 months.
Conclusions. This case highlights that long-standing RA may mask a rare antisynthetase overlap (anti–PL-12/Ro52), in which preserved spirometry does

Résumé

Maladie pulmonaire interstitielle fibrosante progressive dans la polyarthrite rhumatoïde avec chevauchement auto-immun antisynthétase

Introduction. Les maladies pulmonaires interstitielles fibrosantes (MPIf) constituent une manifestation pulmonaire sévère des maladies auto-immunes systémiques. Les syndromes de chevauchement auto-immuns, incluant l'association de la polyarthrite rhumatoïde (PR) avec le syndrome de Sjögren et le syndrome des antisynthétases, sont rares mais associés à un risque accru de fibrose pulmonaire progressive.
Rapport du cas. Nous rapportons le cas d'un homme caucasien de 56 ans, suivi pour une polyarthrite rhumatoïde évoluant depuis 20 ans, sans évaluation respiratoire antérieure, admis pour une dyspnée progressivement aggravée. La tomodensitométrie thoracique à haute résolution (TDM-HR) a mis en évidence un aspect de pneumopathie interstitielle commune (PIC). Les explorations fonctionnelles respiratoires montraient initialement une spirométrie conservée avec une altération de la capacité de diffusion, suivie de l'apparition d'un trouble ventilatoire restrictif léger au cours du suivi. Le bilan immunologique a identifié la présence d'anticorps anti-PL-12 associés aux anticorps

✉ Address for correspondence: Stefan SANDULACHE
Pneumology Department, Elytis Hospital Iasi
Adddress: 43A Gheorghe Saulescu Str., 700010, Iasi, Romania
E-mail: dr.stefansandulache@gmail.com

not exclude advanced or progressive UIP, underscoring the need for early HRCT and extended immunological profiling. Early screening and systematic monitoring for interstitial lung disease in patients with RA, prompt recognition of autoimmune overlap syndromes, and a multidisciplinary therapeutic approach are essential to optimise outcomes in progressive fILD, in accordance with current recommendations from the European Respiratory Society (ERS)/ European Alliance of Associations for Rheumatology (EULAR), the American College of Rheumatology (ACR), the American Thoracic Society (ATS) and the American College of Chest Physicians (CHEST).

Keywords: rheumatoid arthritis, fibrosing interstitial lung disease, usual interstitial pneumonia, antisynthetase syndrome, nintedanib.

List of abbreviations:

6MWT – six-minute walk test
ACR – American College of Rheumatology
ANA – antinuclear antibodies
ANMDM – National Agency for Medicines and Medical Devices (Romania)
anti-CCP – anti–cyclic citrullinated peptide sntibodies
ATS – American Thoracic Society
BAAR – acid-fast bacilli
CHEST – American College of Chest Physicians
COPD – chronic obstructive pulmonary disease
CRP – C-reactive protein
CT – computed tomography
CTD – connective tissue disease
DLCO – diffusing capacity of the lung for carbon monoxide
ERS – European Respiratory Society
ESR – erythrocyte sedimentation rate
EULAR – European Alliance of Associations for Rheumatology
FEV_1 – forced expiratory volume in one second
fILDs –fibrosing interstitial lung diseases
FVC – forced vital capacity
HR – heart rate
HRCT – high-resolution computed tomography
ILD – interstitial lung disease
INBUILD – INBUILD Trial (Nintedanib in Progressive Fibrosing Interstitial Lung Disease)
KCO – carbon monoxide transfer coefficient
NSIP – nonspecific interstitial pneumonia
pMDI – pressurized metered-dose inhaler
RA – rheumatoid arthritis
RA-ILD – rheumatoid arthritis–associated interstitial lung disease
SARS-CoV-2 – severe acute respiratory syndrome coronavirus 2
SpO_2 – peripheral oxygen saturation
UIP – usual interstitial pneumonia
WBC – white blood cell count

anti-Ro52, anti-SS-A et anti-SS-B, compatible avec un syndrome de chevauchement auto-immun. Une prise en charge multidisciplinaire a été instaurée, associant un traitement antifibrosant par nintédanib à une immunosuppression, permettant une stabilisation clinique, fonctionnelle et radiologique à six mois.

Conclusions. Cette observation souligne qu'une polyarthrite rhumatoïde ancienne peut masquer un syndrome de chevauchement rare de type antisynthétase (anti-PL-12/Ro52). Une spirométrie conservée n'exclut pas une atteinte interstitielle pulmonaire avancée ou évolutive. Une évaluation précoce par TDM-HR, un bilan immunologique étendu, ainsi qu'un dépistage et un suivi systématiques des pneumopathies interstitielles chez les patients atteints de PR sont essentiels. La reconnaissance rapide des syndromes de chevauchement auto-immuns et une approche thérapeutique multidisciplinaire sont indispensables pour optimiser le pronostic des MPIf progressives.

Mots-clés: polyarthrite rhumatoïde, maladie pulmonaire interstitielle fibrosante, pneumopathie interstitielle commune, syndrome des antisynthétases; nintédanib

Introduction

Fibrosing interstitial lung diseases (fILDs) represent a major pulmonary manifestation of systemic autoimmune disorders. Rare autoimmune overlap syndromes can accelerate fibrotic progression, underscoring the need for early recognition and multidisciplinary management[1,2].

Rheumatoid arthritis (RA) can involve the lungs, with interstitial lung disease (RA-ILD) affecting 5–10% of patients and significantly increasing morbidity and mortality. The usual interstitial pneumonia (UIP) pattern is most frequent in RA-ILD and is associated with progressive fibrosis and poor prognosis[5]. Respiratory symptoms are often subtle, delaying the diagnosis[3-5].

Current international guidelines—including European Respiratory Society (ERS)/ European Alliance of Associations for Rheumatology (EULAR) 2025, American College of Rheumatology (ACR) 2023, and American College of Chest Physicians (CHEST) 2022—recommend systematic screening for ILD in at-risk patients using high-resolution computed tomography (HRCT) and pulmonary function tests, integrated multidisciplinary care, and early identification of high-risk phenotypes[6,7]. Identification of specific autoantibodies, such as anti–PL-12 and anti–Ro52, is essential to recognize rare autoimmune overlap syndromes that may require tailored therapy[8].

This report presents a patient with long-standing RA in whom preserved spirometry masked a progressive UIP-pattern ILD associated with a rare antisynthetase overlap (anti–PL-12 and anti–Ro52), demonstrating that normal baseline spirometry does not preclude advanced or rapidly progressive fibrosis and emphasizing the need for early HRCT and comprehensive functional assessment, including diffusing capacity for carbon monoxide (DLCO) and six-minute walk testing (6MWT).

Case presentation

A 56-year-old Caucasian man, former smoker (10 pack-years, quit 20 years ago), with significant occupational exposure, having worked for 15 years as a shipyard mechanic with chronic exposure to industrial dust, vapors, and oils, placing him at increased risk for pulmonary disease, presented for progressive dyspnoea. His medical history is notable for secondary pulmonary tuberculosis in 1996, localized to the left upper lobe, bacteriologically confirmed (BAAR 1+), treated completely and declared cured.

In 2005, he was diagnosed with RA and received sequential treatments: sulfasalazine for 10 years, methotrexate (20 mg/week, discontinued after one year because of gastrointestinal intolerance), and leflunomide (10 mg/day for 5 years, increased to 20 mg/day from 2021 to present).

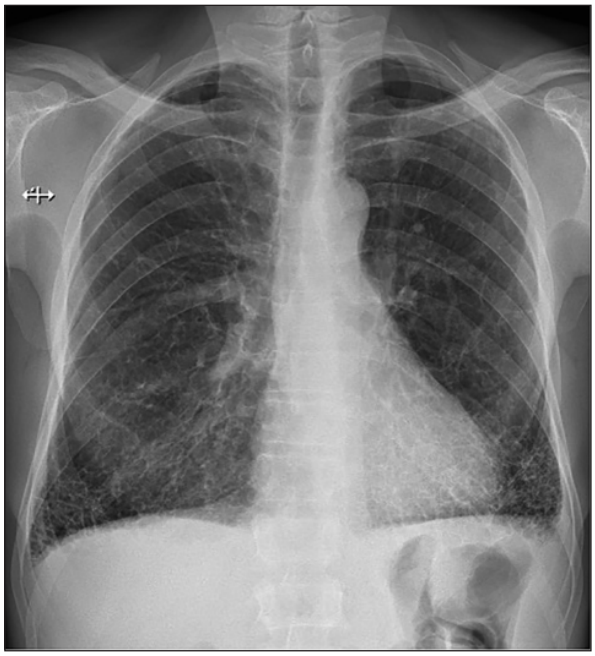


Figure 1. Chest X-ray demonstrating fibrotic interstitial changes with coarse reticulation, most pronounced in the lower lobes, consistent with traction bronchiectasis and honeycombing observed on HRCT. Scattered residual micronodules are noted in the upper lobes [Source: Author's personal archive].

In 2022, he was diagnosed with chronic obstructive pulmonary disease (COPD) and treated with inhaled tiotropium/olodaterol respimat 2.5/2.5µg (2 inhalations/day) and salbutamol pMDI 100µg, 1–2 puffs as needed, although spirometry showed a mild restrictive pattern without obstructive or mixed features. This comorbidity confounded the evaluation of his progressive dyspnoea.

In May 2025, he was first evaluated at our dedicated ILD center, referred from the infectious diseases department following hospitalization for an acute respiratory infection. Chest X-ray (Figure 1) revealed diffuse interstitial changes. On clinical examination, he exhibited progressive exertional dyspnoea over the preceding three years, Raynaud phenomenon, small-joint arthralgia in the hands, gonalgia, morning stiffness >30 minutes, xerostomia, and xerophthalmia. The vital signs were: SpO_2 98% on room air, heart rate 88 bpm, blood pressure 122/85 mmHg. The pulmonary auscultation revealed "Velcro" crackles, predominantly in the bilateral mid-lower lung fields.

Non-contrast HRCT (Figures 2–4) demonstrated a definitive fibrotic UIP pattern, involving >20% of the lung parenchyma. The disease distribution exhibited a classic apicobasal gradient, with severe

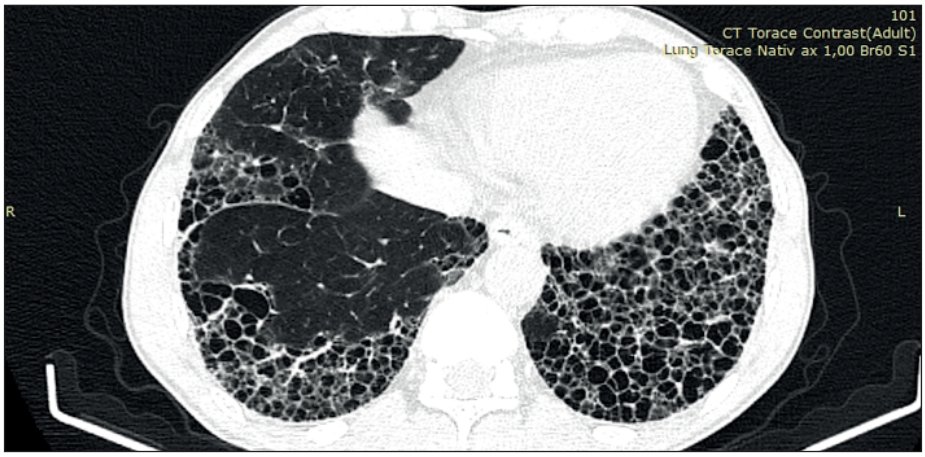


Figure 2. Axial HRCT section at the lung bases. High-resolution axial section demonstrating the hallmark features of honeycombing, characterized by clustered, multi-layered subpleural cystic airspaces with thickened, well-defined walls. The image highlights the peripheral distributionof the fibrotic process and the complete replacement of normal lung architecture by coarse reticular opacities, consistent with a definitive UIP diagnosis.



Figure 3. Sagittal HRCT reconstruction of the right lung. The image displays a definitive UIP pattern with a marked apicobasal gradient. Key findings include relative sparing of the superior segments and terminal subpleural honeycombing at the lung bases. Associated traction bronchiectasis and significant architectural distortion reflect advanced parenchymal fibrosis and secondary lobar volume loss.

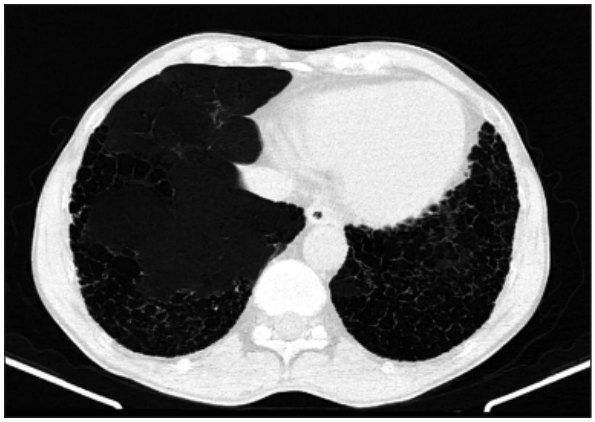


Figure 4. Axial HRCT section with minimum intensity projection (MinIP) reconstruction. The MinIP technique enhances the visualization of low-attenuation structures, clearly delineating the multi-layered subpleural honeycombingand air columns within dilated bronchi. This highlights the severe architectural distortion and confirms the definitive UIP pattern by emphasizing the cystic parenchymal replacement at the lung bases.

architectural distortion, multi-layered honeycombing, and traction bronchiectasis predominantly localized within the lower lobes, resulting in significant volume loss. Additional findings included perivascular micronodules in the upper lobes, focal irregular pleural thickening, and subpleural microcystic conglomerates, all consistent with established, advanced pulmonary fibrosis.

At initial pulmonary evaluation in May 2025, pulmonary function testing demonstrated preserved spirometry, with forced vital capacity (FVC) 5.05 L (101% predicted), forced expiratory volume in one second (FEV_1) 3.88 L (98% predicted), and FEV_1/FVC ratio of 77%. However, gas transfer was already impaired, with DLCO 71% predicted and carbon monoxide transfer coefficient (KCO) 82%, indicating early functional involvement despite normal lung volumes.



Figure 5. Criteria for progressive fibrosing phenotype and initiation of nintedanib therapy, based on the Romanian national protocol. Functional decline, radiologic progression, and symptom worsening guide treatment decisions [Source: Author's original work].

The 6MWT showed 400 m achieved out of 739 m predicted (54%), with desaturation from 98% to 90% and a Borg dyspnoea score increase from 1 to 4.

Given the extent of fibrosis and clinical symptoms, a comprehensive immunological assessment was recommended, not performed for approximately 5 years. Immunological profiling revealed elevated antinuclear antibodies (ANA) (1:1280, normal <1:80), anti-CCP (>340 U/mL, normal <5), rheumatoid factor (172.3 IU/mL, normal <14), anti–SS-A (100 U, normal <5), anti–SS-B (100 U, normal <5), anti–Ro52 (97 U, normal <5), and anti–PL-12 (56 U, normal <5), consistent with an autoimmune overlap phenotype with features of Sjögren disease and antisynthetase syndromes. These findings supported the classification of the patient as having RA-ILD with autoimmune overlap, at high risk of fibrosing progression.

At 3-month pulmonary follow-up in August 2025, the case was reviewed by the multidisciplinary ILD team (pulmonologist, radiologist, and rheumatologist). Repeat HRCT was not performed because of the established UIP pattern with extensive honeycombing and minimal inflammatory activity (isolated ground-glass opacities), making short-term radiological progression difficult to assess. Compared with baseline pulmonary function testing performed at initial evaluation in May 2025, the patient fulfilled criteria for a progressive fibrosing phenotype (Figure 5)[9], based on clinical and functional deterioration, including worsening dyspnoea, a relative decline in FVC of approximately 15%, a DLCO decrease of 9%, a reduction in 6-minute walk distance by 70 m, and exertional desaturation from 98% to 82%. Based on multidisciplinary consensus, antifibrotic therapy with nintedanib 150 mg twice daily was initiated, in combination with ongoing leflunomide 20 mg/day, and planning for biologic therapy with rituximab or tocilizumab was initiated. This combination of antifibrotic therapy and ongoing immunosuppressive treatment was planned to target both fibrotic progression and autoimmune activity, reflecting an individualized, phenotype-driven approach.

Three months following the initiation of antifibrotic treatment (November 2025), the patient experienced an acute infectious episode: productive mucopurulent cough, fever 39.5°C, chills, and right posterior thoracic pain. Laboratory workup revealed leukocytosis (WBC 16,350/mm^3, 91% neutrophils)

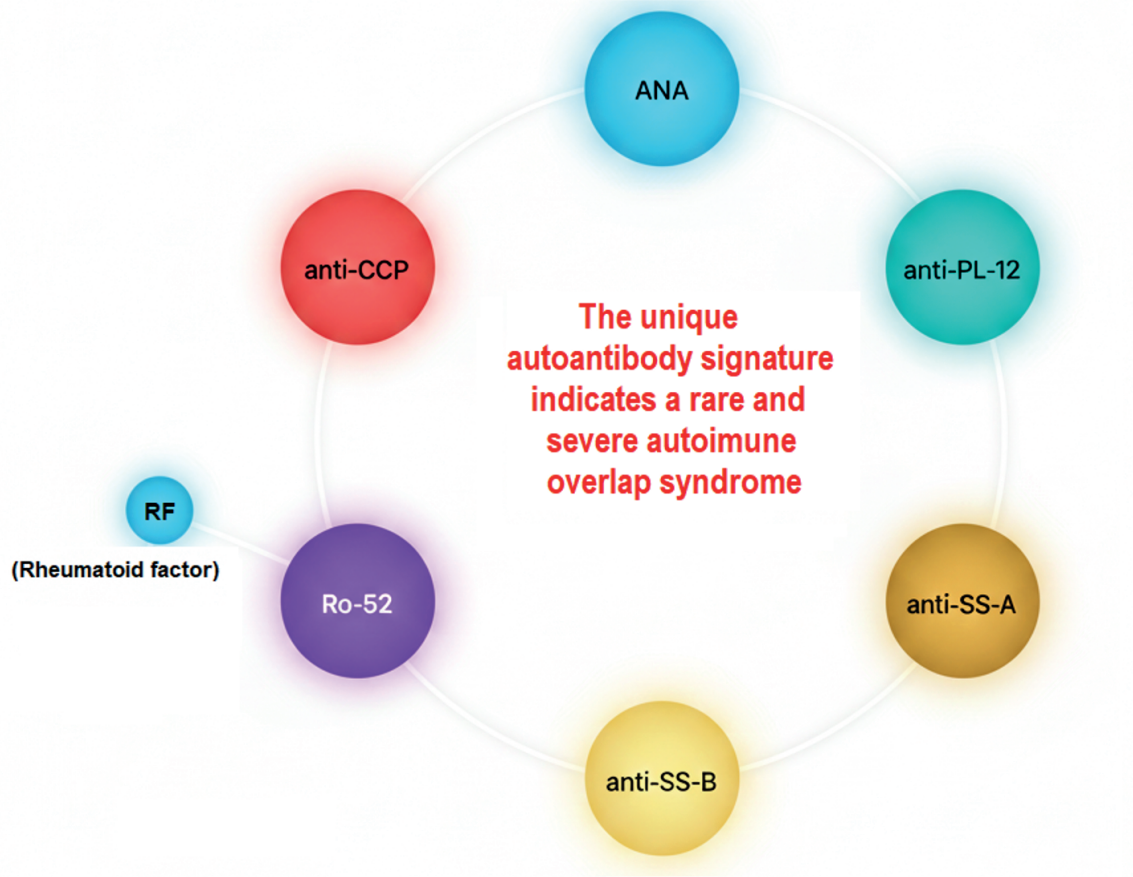


Figure 6. Overlap syndrome:
rheumatoid arthritis – Sjögren's disease – antisynthetase syndrome
[Source: Author's original work]

and C-reactive protein (CRP) 152 mg/L (normal<5) and erythrocyte sedimentation rate (ESR) of 102 mm in the first hour (normal<15). Respiratory cultures were negative for *Mycobacterium tuberculosis*; cytobacteriologic testing showed mixed flora, and influenza, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), and respiratory syncytial virus tests were negative. Combined antibiotic therapy with levofloxacin 500 mg/day and cefixime 400 mg/day for 10 days was administered, alongside symptomatic treatment. Leflunomide was temporarily suspended, and pulmonary function reassessments were postponed until resolution of the infection.

The 6-month follow-up HRCT demonstrated radiological stability: fibrotic UIP pattern with honeycombing, without significant progression, and non-fibrotic lesions remained unchanged.

At pulmonary follow-up in December 2025, spirometry showed a FVC 3.6 L (72% predicted), FEV_1 2.93 L (74% predicted), FEV_1/FVC 81 and DLCO 55%, indicating a mild restrictive pattern with functional stabilization, reflecting the positive effect of antifibrotic therapy and highlighting the need for ongoing monitoring of fibrotic progression.

Discussion

This case illustrates the complexity of diagnosing and managing RA-ILD in the setting of a rare autoimmune overlap syndrome, including features of Sjögren's disease and antisynthetase syndrome with positive anti-PL-12 and anti-Ro52 antibodies (Figure 6). This constellation of autoimmune diseases is rarely reported and defines a high-risk mixed phenotype prone to progressive fibrosis[6,7]. Evidence indicates that RA-ILD presents with variable HRCT patterns, most commonly UIP or nonspecific interstitial pneumonia (NSIP). The presence of antisynthetase syndrome features, even in the absence of overt myopathy, confers additional risk for rapid fibrotic progression[10,11].

A remarkable aspect of this case is the diagnostic delay, partly due to a history of COPD and a subtle restrictive pattern on spirometry, masking the evolution of RA-ILD. Despite RA for over 20 years, the patient had never undergone HRCT, resulting in late recognition of pulmonary fibrosis after symptom onset. This underscores the need for early and systematic ILD screening in RA patients, especially in

the presence of risk factors such as male sex, smoking history, seropositivity for rheumatoid factor, and anti-CCP antibodies[12,13].

Current international recommendations—including ACR 2023, CHEST 2022, and the latest ERS/EULAR 2025 guidelines for connective tissue disease-associated ILD—emphasize a structured approach: baseline or symptom-triggered HRCT, routine functional assessment (spirometry, DLCO), and regular multidisciplinary review to identify progressive phenotypes[1,7,14,15]. HRCT remains the gold standard for early detection and fibrosis severity stratification, allowing identification of UIP with multilayered honeycombing, traction bronchiectasis, and minor inflammatory changes such as isolated ground-glass opacities[16,17].

Functional assessment—including spirometry, DLCO, and the 6MWT—provided a comprehensive evaluation of both initial impairment and treatment response. Although FVC and FEV_1 were initially normal, reduced DLCO and exertional desaturation proved sensitive for early pulmonary involvement, confirming the importance of integrated functional monitoring. DLCO and the 6MWT are established tools for assessing disease progression and functional impairment in RA-ILD, correlating with imaging findings and patient prognosis[18,19]. Importantly, this case demonstrates that a normal FVC at baseline does not preclude advanced fibrotic disease or rapid progression in RA-associated UIP, particularly when DLCO reduction and exertional desaturation are present.

The multidisciplinary management played a critical role, enabling identification of a progressive phenotype, optimal timing for initiating antifibrotic therapy (nintedanib), and adjustment of immunosuppressive treatment (leflunomide). Multidisciplinary discussion is strongly recommended by ERS/EULAR 2025 and CHEST, demonstrating improved functional outcomes and reduced risk of rapid fibrotic progression[1,14,15].

Antifibrotic therapy led to radiological and functional stabilization, maintaining FVC and DLCO within a mild restrictive range, consistent with findings from INBUILD (Nintedanib in Progressive Fibrosing Interstitial Lung Disease) trial and other recent trials showing nintedanib slows functional decline in progressive fibrosing ILD, including RA-ILD[20,21,22]. The acute infectious episode three months after antifibrotic initiation was effectively managed with temporary leflunomide cessation, tailored antibiotics, and close follow-up, illustrating the need for vigilant monitoring of intercurrent events in this vulnerable population[23].

The extended immunological profile (anti-SS-A, anti-SS-B, Ro52, and anti-PL-12 positivity) supports classification as an overlap syndrome involving RA, Sjögren's disease, and antisynthetase syndrome. These autoantibodies have been linked to accelerated fibrotic progression and variable responses to immunosuppressive therapy, underscoring the need for individualized monitoring and periodic reassessment of biologic treatment[22,24].

In line with ERS/EULAR 2025, ACR 2023, and CHEST 2022 guidelines, monitoring should include periodic clinical assessments, spirometry, DLCO and 6MWT every 3–6 months, with HRCT performed at individualized intervals based on symptoms and functional changes. Integrating clinical, imaging, and immunological data remains the recommended standard for managing RA-ILD and autoimmune overlap syndromes.

Key lessons from this case include: (1) early ILD screening should include HRCT and extended immunological testing even in long-standing RA patients with preserved FVC; (2) systematic functional monitoring using FVC trends, DLCO, and 6MWT is critical to detect rapid progression; (3) multidisciplinary evaluation allows timely initiation of antifibrotic therapy; (4) combined antifibrotic and immunomodulatory therapy may stabilize disease in high-risk overlap phenotypes.

Conclusions

This case illustrates a rare autoimmune overlap phenotype in RA–associated ILD, combining features of Sjögren's disease and anti-PL-12 antisynthetase syndrome. Delayed thoracic imaging for over 20 years highlights the critical need for systematic pulmonary screening in long-standing RA, even in asymptomatic patients. Early detection through HRCT, functional testing, and comprehensive immunological profiling enables timely initiation of antifibrotic therapy. Disease stabilization under nintedanib within a multidisciplinary approach underscores the value of integrated management for preventing fibrotic progression and preserving quality of life. Clinicians should recognize overlap syndromes and combine imaging, functional, and immunological data to guide individualized care.

Author Contributions:

S.S. was responsible for the conception of the case report, data collection, literature review, and manuscript preparation.

Compliance with Ethics Requirements:

"The author declares no conflict of interest regarding this article"

"The author declares that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from the patient included in the study"

"No funding for this study"

Acknowledgements:

None

References

1. Sambataro G, Maher TM, Flaherty KR, et al.; ERS/EULAR Task Force. ERS/EULAR clinical practice guidelines for connective tissue disease–associated interstitial lung disease. *Eur Respir J.* 2025; doi:10.1183/13993003.02533-2024.
2. Cottin V, Brown KK. Interstitial lung disease associated with systemic autoimmune diseases. *Lancet*. 2019;394(10201):1344–1356. doi:10.1016/S0140-6736(19)30979
3. Dinache G, Popescu CM, Mogosan C, et al. Rheumatoid arthritis-associated interstitial lung disease. *Rom J Rheumatol.* 2025;34(2):204–213. doi:10.1183/13993003.02533-2024
4. Kelly CA, Saravanan V, Nisar M, et al. Rheumatoid arthritis–related interstitial lung disease. *Rheumatology* (Oxford). 2014;53(9):1676–1682. doi:10.1093/rheumatology/keu135
5. Lee HK, Yoo JY, Kim TJ, et al. Impact of the pattern of interstitial lung disease on mortality in rheumatoid arthritis. *Respir Med*. 2019;150:62–68. doi:10.1016/j.rmed.2019.01.009
6. Johnson SR, Bernstein EJ, Bolster MB, et al. 2023 American College of Rheumatology/American College of Chest Physicians guideline for screening and monitoring of interstitial lung disease in systemic autoimmune rheumatic diseases. *Arthritis Rheumatol*. 2024;76(8):1201–1213. doi:10.1002/art.42860
7. Johnson SR, Bernstein EJ, Bolster MB, et al. 2023 ACR/CHEST guideline for treatment of interstitial lung disease in systemic autoimmune rheumatic diseases. *Arthritis Rheumatol*. 2024;76(8):1182–1200. doi:10.1002/art.42861
8. Solomon JJ, Swigris JJ, Brown KK. Antisynthetase syndrome and interstitial lung disease. *J Clin Rheumatol.* 2016;22(2):65–72. doi:10.1097/RHU.0000000000000334
9. Romanian Ministry of Health / National Agency for Medicines and Medical Devices (ANMDM). Therapeutic protocol for DCI Nintedanibum (OFEV): Diffuse interstitial lung disease – progressive fibrosing phenotype (PID FFP). Bucharest: Ministry of Health / ANMDM; 2023. Available at: https://legislatie.just.ro/Public/DetaliiDocumentAfis/252606
10. Solomon JJ, Danoff SK, Cosgrove GP, et al. Rheumatoid arthritis-associated interstitial lung disease: predictors of mortality and pattern distribution. *Eur Respir J*. 2016;47(2):588–596. doi:10.1183/13993003.00253-2015
11. Kim EJ, Elicker BM, Maldonado F, et al. Usual interstitial pneumonia in rheumatoid arthritis-associated interstitial lung disease. *Eur Respir J*. 2010;35(6):1322–1328. doi:10.1183/09031936.00013009
12. Bongartz T, Nannini C, Medina-Velasquez YF, et al. Incidence and mortality of interstitial lung disease in rheumatoid arthritis: a population-based study. *Arthritis Rheum*. 2010;62(6):1583–1591. doi:10.1002/art.27475
13. Matson SM, Baqir M, Moua T, et al. Treatment outcomes for rheumatoid arthritis-associated interstitial lung disease: real-world multisite study of immunosuppression impact on pulmonary function. *Chest*. 2023;163(4):861–869. doi:10.1016/j.chest.2022.09.020
14. Flaherty KR, Wells AU, Cottin V, et al. Nintedanib in progressive fibrosing interstitial lung diseases. *N Engl J Med*. 2019;381(18):1718–1727. doi:10.1056/NEJMoa1908681
15. Wells AU, Flaherty KR, Brown KK, et al. Nintedanib in patients with progressive fibrosing ILDs: subgroup analyses by ILD diagnosis. *Lancet Respir Med*. 2020;8(5):453–460. doi:10.1016/S2213-2600(20)30062-6
16. Cottin V, Richeldi L, Selman M, et al. Interstitial lung disease in rheumatoid arthritis: a practical review. *Front Med (Lausanne)*. 2022;9:837133. doi:10.3389/fmed.2022.837133
17. Distler O, Highland KB, Goh NSL, et al. Screening for rheumatoid arthritis-associated ILD: Delphi consensus for early detection. *Z Rheumatol*. 2024;83:160–168. doi:10.1007/s00393-023-01464-w
18. Fischer A, Brown KK, du Bois RM, et al. Detection and management of ILDs associated with connective tissue diseases. *Am J Respir Crit Care Med*. 2021;203(1):55–65. doi:10.1164/rccm.202010-3873CI
19. Juge P-A, Lee JS, Ebstein E, et al. Shared genetic predisposition in RA-ILD and familial pulmonary fibrosis: MUC5B promoter variant study. *Eur Respir J*. 2017;49(5):1602314. doi:10.1183/13993003.02314-2016
20. Myasoedova E, Crowson CS, Turesson C, et al. Incidence and mortality of extra-articular RA and association with ILD. *J Rheumatol*. 2011;38(6):983–989. doi:10.3899/jrheum.101043
21. Assayag D, Lubin M, Lee JS, et al. Predictors of mortality in rheumatoid arthritis-related ILD. *Respirology*. 2014;19(4):493–500. doi:10.1111/resp.12282
22. Maher TM, Corte TJ, Richeldi L, et al. Efficacy and safety of antifibrotic agents in connective tissue disease-associated ILD and RA-ILD: systematic review and meta-analysis. *Pulmonology*. 2023;[epub ahead]; doi:10.1016/j.pulmoe.2023.07.002
23. Raimundo K, Solomon JJ, Olson AL, et al. RA-ILD in the US: prevalence, incidence, and healthcare costs. *J Rheumatol*. 2019;46(4):360–369. doi:10.3899/jrheum.180771
24. Yunt ZX, Chung JH, Hobbs S, et al. High-resolution CT pattern of UIP in RA-ILD: relationship to survival. *Respir Med*. 2017;126:100–104. doi:10.1016/j.rmed.2017.03.010