



The Great Masquerader: Tuberculous Pleural Effusion Mimicking Rheumatoid Pleuritis in a Seropositive Rheumatoid Arthritis Patient

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Abstract

Rheumatoid arthritis (RA) is a chronic inflammatory disease with well-recognized extra-articular manifestations, including pleural involvement. Rheumatoid pleural effusion (RPE), although uncommon, is a known entity and may present as an exudative effusion with characteristic biochemical features. In countries with a high burden of tuberculosis (TB), tuberculous pleural effusion (TPE) remains one of the most frequent causes of lymphocytic exudative pleural effusion. The clinical and laboratory overlap between RPE and TPE often makes differentiation challenging. Patients with RA are frequently treated with immunosuppressive agents such as methotrexate and Janus kinase inhibitors like tofacitinib, which may increase susceptibility to infections, including TB. This overlap of disease manifestations and treatment-related risks can complicate clinical decision-making.

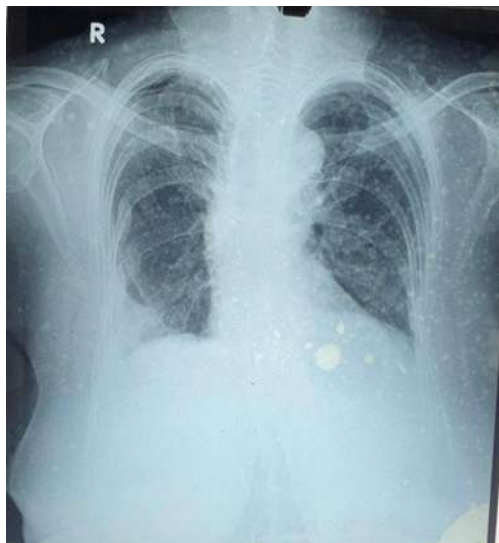
Keywords: NIL

Introduction

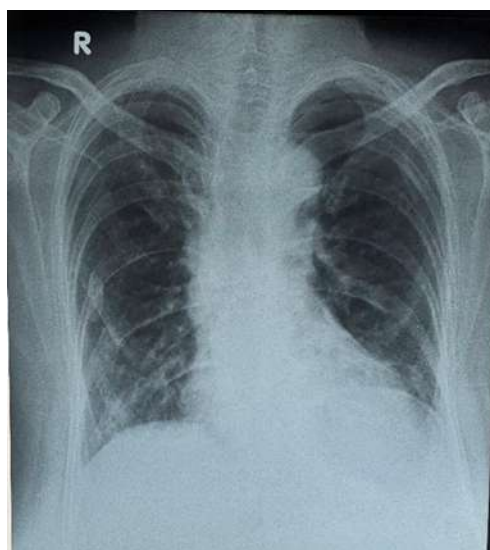
A middle-aged woman with a known history of seropositive rheumatoid arthritis i.e. RA (Rheumatoid Arthritis) factor & anti-CCP(anti-cyclic citrullinated peptide) positive, presented with progressive breathlessness and right-sided pleuritic chest pain of 4 weeks duration. She also reported constitutional symptoms like Fever with evening rise of temperature and loss of appetite, loss of weight which were suggestive of tuberculosis. She had been on treatment with methotrexate and tofacitinib for RA prior to presentation. On examination, findings were consistent with a right-sided pleural effusion. Chest imaging confirmed a unilateral moderate pleural effusion. Diagnostic thoracentesis revealed straw-coloured fluid. Pleural fluid analysis showed an

exudative effusion with 100% lymphocytic predominance. Adenosine deaminase (ADA) levels were markedly elevated at 94 U/L. Microbiological testing with GeneXpert was negative. However, the Mantoux test was positive. In view of the clinical presentation, pleural fluid characteristics, and supportive investigations, a working diagnosis of tuberculous pleural effusion was made. The patient was initiated on standard anti-tubercular therapy. Over the course of treatment, she showed steady symptomatic improvement. Follow-up imaging demonstrated significant resolution of the pleural effusion. After completing 6 months of therapy, treatment was discontinued with satisfactory clinic-radiological recovery.

CXR AT THE TIME OF DIAGNOSIS



CXR AFTER COMPLETING 6 MONTHS OF ANTI-TB TREATMENT



Discussion

Pleural effusion in patients with rheumatoid arthritis presents a well-recognized diagnostic dilemma. Rheumatoid pleural effusion and tuberculous pleural effusion share several overlapping features, including lymphocytic predominance and exudative nature, making differentiation difficult on routine analysis alone. Rheumatoid pleural effusions are typically associated with very low glucose levels, low pH, and high lactate dehydrogenase, whereas TPE often demonstrates elevated ADA levels and lymphocyte predominance. However, these distinctions are not always absolute, particularly in real-world clinical settings. In this case, the markedly elevated ADA level

(94 U/L), lymphocytic predominance, and positive Mantoux test strongly supported a tuberculous etiology, despite a negative GeneXpert result. It is well recognized that microbiological confirmation in pleural TB can be limited due to the paucibacillary nature of the disease. Another important consideration is the patient's background of immunosuppressive therapy. Both methotrexate and tofacitinib are associated with an increased risk of infections, including reactivation of latent tuberculosis. This further strengthens the likelihood of TB as the underlying cause in this patient. The case underscores the importance of maintaining a high index of suspicion for TB in endemic regions and avoiding premature attribution of pleural effusion to rheumatoid

disease alone. Empirical treatment based on a composite clinical assessment may be justified when microbiological confirmation is lacking but suspicion remains high.

Conclusion

Tuberculous pleural effusion should be carefully considered in patients with rheumatoid arthritis presenting with pleural effusion, particularly in TB-endemic settings. Overlapping clinical and laboratory features may obscure the diagnosis. A combination of clinical judgment, pleural fluid analysis, and supportive tests is often required to guide management when definitive microbiological evidence is not available.

Keywords

Rheumatoid arthritis; Tuberculous pleural effusion; Adenosine deaminase; Methotrexate; Tofacitinib; Pleural effusion.

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