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(CASE REPORT)

Organizing Pneumonia Revealing Mixed Connective Tissue Disease: A Case Report

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Abstract

Organizing pneumonia is a pulmonary condition characterized by inflammation of the alveoli and the development of connective tissue in the lungs. It can be triggered by infections, autoimmune diseases, or medications. Mixed connective tissue disease, on the other hand, is an autoimmune disorder that combines features of several diseases, such as lupus and Sjögren's syndrome.

This case report describes a 24-year-old female patient, initially misdiagnosed with pleuropulmonary tuberculosis after presenting with respiratory symptoms and general signs. Failure of antituberculous treatment led to a re-evaluation of the diagnosis, ultimately revealing organizing pneumonia related to mixed connective tissue disease. Immunological testing and salivary gland biopsy confirmed the diagnosis of mixed connective tissue disease, including systemic lupus erythematosus and Sjögren's syndrome. The patient was treated with corticosteroids and immunosuppressive agents, with significant clinical improvement, highlighting the diagnostic challenges and the importance of a multidisciplinary approach.

Keywords: Organizing Pneumonia; Mixed Connective Tissue Disease; Multidisciplinary Approach; Corticosteroids; Azathioprine.

1. Introduction

Mixed connective tissue diseases are complex autoimmune disorders that often present with a variety of clinical manifestations, making their diagnosis challenging. They can mimic other conditions, including pulmonary infections such as tuberculosis (TB), which further complicates management. This similarity in clinical presentation can lead to diagnostic errors. Particular vigilance and a multidisciplinary approach are essential to avoid confusion and ensure appropriate treatment.

2. Case report

We report the case of a 24-year-old female patient, who had been followed for pleuropulmonary tuberculosis for 52 days, receiving anti-bacillary treatment (2RHZE/4RH). Two months prior to her admission, she developed progressive dyspnea (Sadoul stage II), accompanied by productive cough with whitish sputum, occasionally hemoptoic. Left-sided lateral chest pain was also reported, without other systemic manifestations such as arthralgia or ocular and oral symptoms. This occurred in the context of general malaise, including anorexia, fatigue, and a weight loss of 7 kg over two months, along with night sweats.

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On clinical examination, the patient was alert (Glasgow Coma Score: 15/15) and in good general condition. Vital signs were normal: respiratory rate of 18 breaths per minute, oxygen saturation of 98% on room air, heart rate was 80 bpm, and normotension (120/65 mmHg). Pulmonary examination revealed basal right-sided crackles.

The chest X-ray **(Figure 1)** showed pleuropneumopathy with migrating opacities.

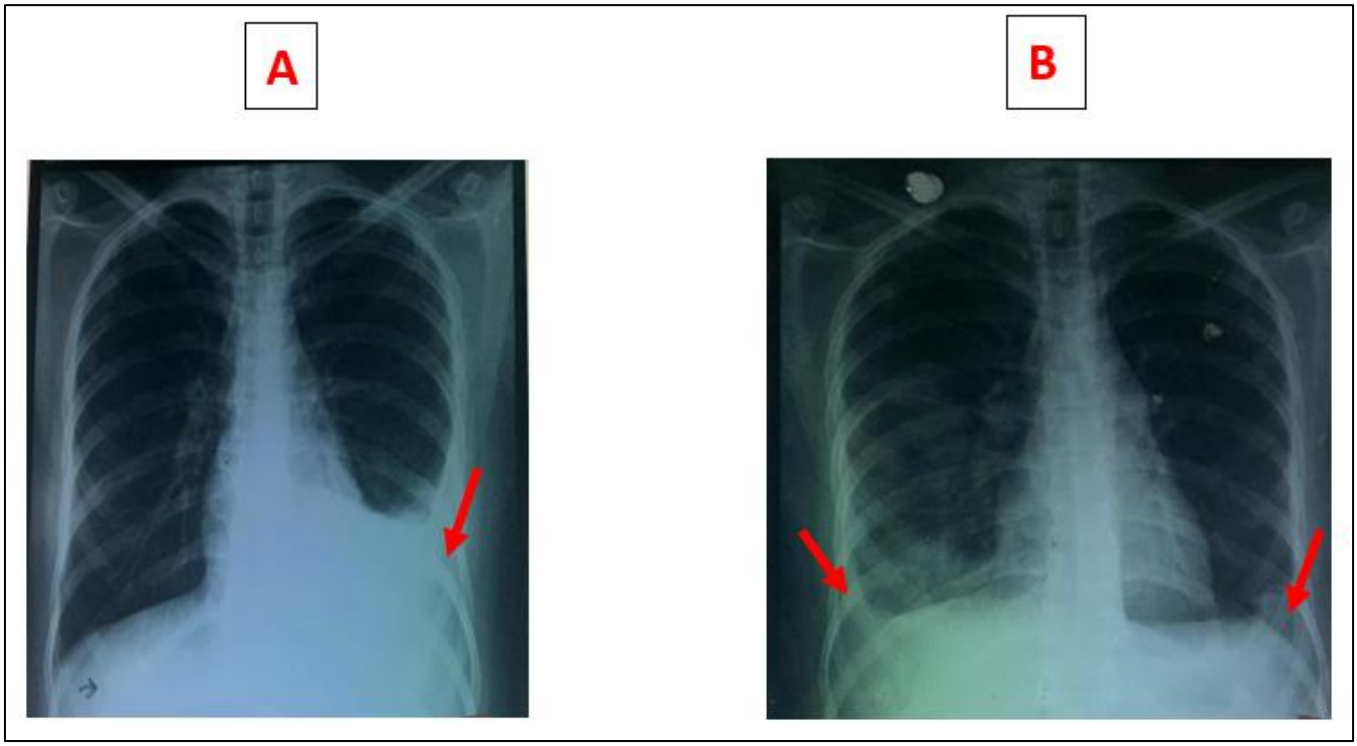

Figure 1 (A) Chest X-ray (frontal view) at the time of TB diagnosis: showing a dense homogeneous opacity with a watery tone in the left basithorax, obliterating the left diaphragm dome and the corresponding pleural recesses, with a heterogeneous opacity above. (B) Chest X-ray on day 52 of treatment: Bilateral alveolar opacities, predominantly in the subpleural regions (migratory pattern).

The chest CT scan **(Figure 2)** revealed an organizing pneumonia pattern with bilateral consolidation foci and intra-parenchymal micronodules, suggesting a non-infectious etiology .

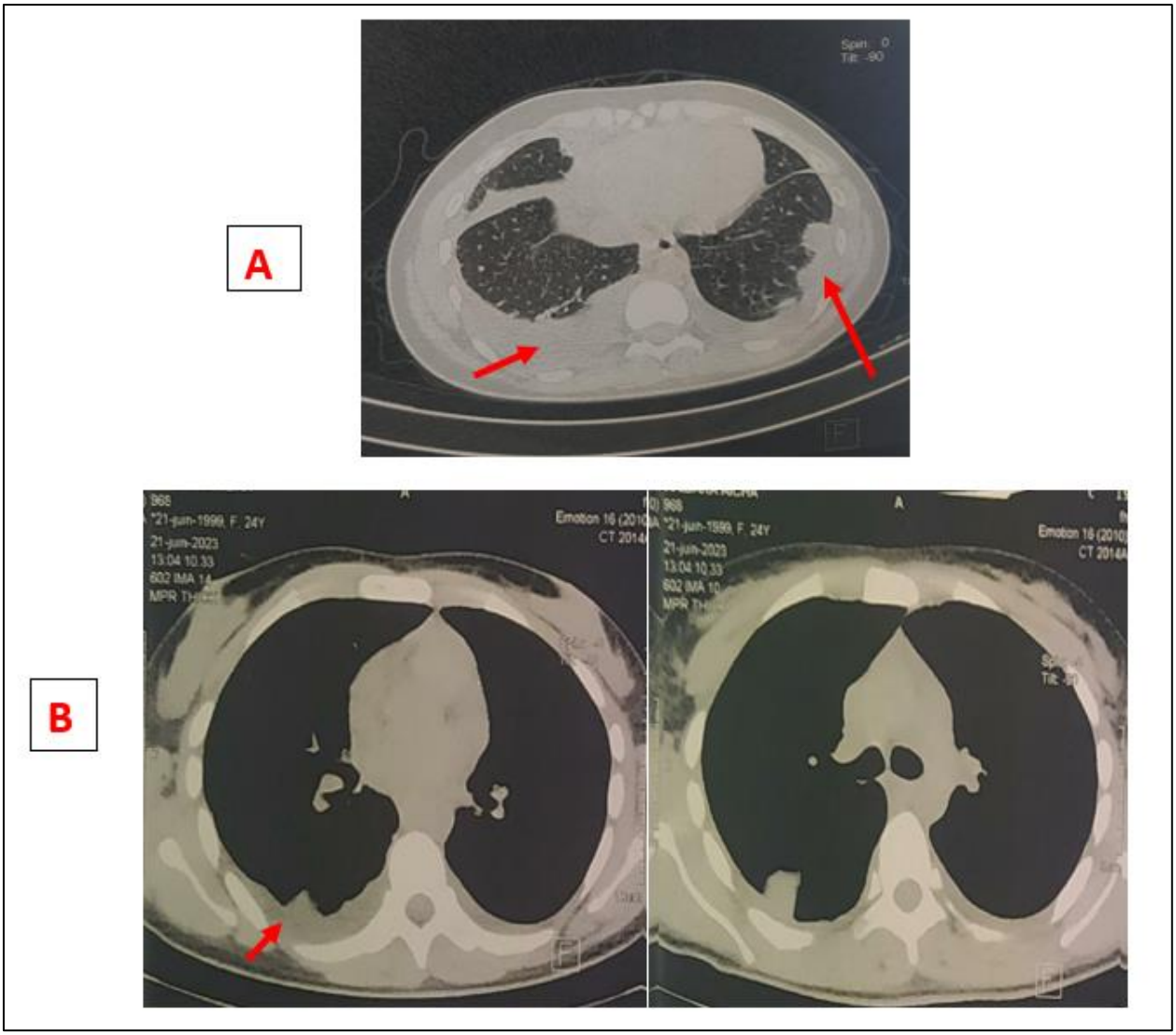

Figure 2 (A) The parenchymal window shows multiple alveolar opacities in bilateral patchy areas of varying sizes, predominantly in the subpleural and peripheral regions, confluent in some areas. **(B)** The mediastinal window reveals the presence of a small right pleural effusion, with no mediastinal lymphadenopathy

A bronchoscopy was performed, revealing a diffuse inflammatory condition of grade 1, primarily on the left. Bronchial biopsies showed subacute and chronic inflammatory changes, without specific characteristics. Bronchoalveolar lavage revealed alveolitis with a predominance of histiocytes. Tests for Mycobacterium tuberculosis (TB) and Xpert MTB/RIF on bronchial aspirates were negative. The biological assessment showed an inflammatory syndrome with hypochromic microcytic anemia and an elevated erythrocyte sedimentation rate (ESR). The immunological workup revealed positive antinuclear antibodies (ANA), along with anti-native DNA and anti-SSA/Ro antibodies, confirming an autoimmune profile. A salivary gland biopsy showed focal lymphocytic sialadenitis of grade 4 according to Chisholm and Mason.

After a thorough analysis of the clinical results and additional tests, a diagnosis of mixed connective tissue disease, including systemic lupus erythematosus and Sjögren's syndrome, was made.

The treatment was modified to include corticosteroids (1 mg/kg), along with adjuvant therapy, Plaquenil (hydroxychloroquine) (200 mg twice daily) and Imurel (azathioprine) (50 mg/day for five days, then 2 tablets/day). Anti-bacillary treatment was discontinued after the confirmation of the mixed connective tissue disease diagnosis.

The clinical and radiological outcomes were favorable. A follow-up chest CT scan (Figure 3) showed radiological clearance.

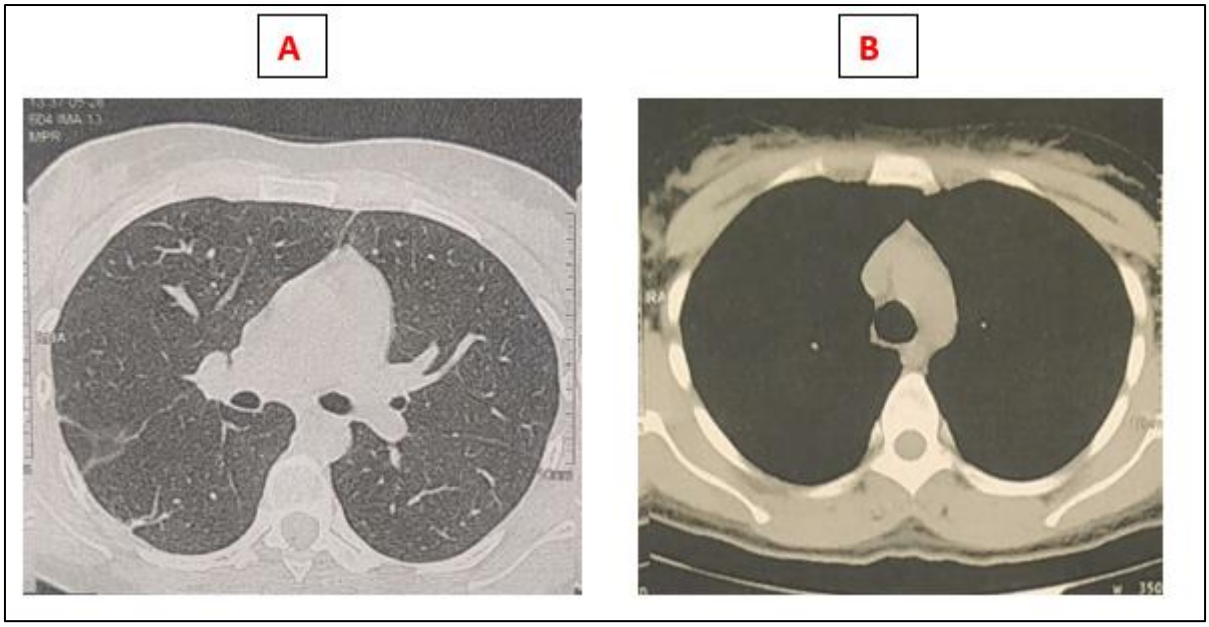

Figure 3 After 3 months of treatment, The parenchymal window (A) shows radiological resolution . The mediastinal window (B) shows no pleuropericardial effusion

3. Discussion

The presented case illustrates the diagnostic challenges when mixed connective tissue disease (MCTD) initially presents with respiratory symptoms mimicking an infectious pathology, particularly pleuropulmonary tuberculosis (TB). Misinterpretation of the initial symptoms, misleading imaging results, and the presence of atypical biological manifestations led to an incorrect diagnosis, delaying appropriate therapeutic management. Accurate diagnosis in this context relies on a thorough evaluation and multidisciplinary collaboration to avoid inappropriate treatments.

Mixed connective tissue diseases, such as systemic lupus erythematosus (SLE) and Sjögren's syndrome (SS), can present with a variety of pulmonary signs, including diffuse interstitial pneumonias, organizing pneumonias, or restrictive pulmonary syndromes. In this case, the patient initially presented with respiratory symptoms consistent with TB: dyspnea, productive cough, hemoptysis, and chest pain. Moreover, the general symptoms, including night sweats, anorexia, and a 7 kg weight loss over two months, suggested an infectious etiology. This clinical context led to the immediate initiation of anti-bacillary treatment [1]. However, the lack of clinical improvement after 52 days of anti-tuberculous therapy raised doubts about the initial diagnosis. The failure to respond to treatment prompted further investigation for an alternative etiology, including autoimmune diseases like SLE, which can mimic pulmonary infections due to their atypical clinical and radiological presentation [2].

The chest CT scan was pivotal in re-evaluating the diagnosis. The images revealed an organizing pneumonia pattern, characterized by bilateral consolidation foci, ground-glass opacities, and traction bronchiectasis, typical of interstitial lung diseases associated with connective tissue diseases [3] . The lesion distribution in the lower lobes, absence of cavitations, and intra-parenchymal micronodules are distinguishing features of connective tissue diseases, in contrast to the classic radiological patterns of TB, such as pulmonary cavities and hilar lymphadenopathy.

The immunological profile was decisive in differentiating between an infectious pathology and mixed connective tissue disease. The positive antinuclear antibodies (ANA) at a high titer (1/320), along with anti-native DNA and anti-SSA/Ro antibodies, suggested SLE. The concomitant presence of anti-SSB/Ro 52 and anti-Ku antibodies indicated mixed connective tissue disease or an overlap syndrome, which combines multiple autoimmune entities such as SS and SLE [4]. This serological feature emphasizes the importance of a comprehensive immunological assessment in the presence of atypical pulmonary symptoms and unexpected therapeutic responses.

The biopsy of the accessory salivary glands revealed focal lymphocytic sialadenitis of grade 4 according to Chisholm and Mason, a key histopathological criterion for the diagnosis of SS [5]. Focal lymphocytic infiltration is typical of primary or secondary SS associated with other connective tissue diseases, as in this case. This histological result,

combined with the systemic manifestations and immunological profile, confirmed the diagnosis of mixed connective tissue disease with pulmonary involvement.

The decision to discontinue anti-bacillary treatment was made after the confirmation of mixed connective tissue disease. The shift to immunosuppressive therapy, including corticosteroids, Plaquenil, and Imurel, led to significant clinical improvement. However, this therapeutic change must be carefully considered in regions where tuberculosis is endemic. Indeed, latent tuberculosis may be reactivated under immunosuppression, increasing the risk of morbidity [6]. Close monitoring of these patients is therefore crucial, including latent tuberculosis screening tests (e.g., IGRA or tuberculin skin test) before initiating any immunosuppressive treatment [7].

Recent studies have shown that mixed connective tissue diseases often present with multisystem involvement, and pulmonary involvement is seen in 20% to 30% of cases of combined SLE and SS [8]. Approximately 10% of patients with autoimmune pulmonary involvement are initially treated for infections due to clinical and radiological similarities. This diagnostic misinterpretation is particularly common in settings where tuberculosis and other pulmonary infections are prevalent [2].

Studies have also highlighted the importance of using biomarkers in the evaluation of mixed connective tissue diseases. The serological profile, including ANA, anti-DNA, anti-SSA, and anti-SSB antibodies, allows for early and accurate differentiation, thus preventing delays in diagnosis and therapeutic management [9].

4. Conclusion

This case highlights the challenges of differential diagnosis between an infectious pathology and mixed connective tissue disease. A multidisciplinary approach, including a comprehensive immunological workup, high-quality thoracic imaging, and targeted histopathological examination, is essential to avoid diagnostic errors and inappropriate treatments. Clinical vigilance remains crucial in managing connective tissue diseases, especially in the presence of atypical respiratory symptoms or unexpected therapeutic responses.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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