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Not All Bilateral Exudative Effusions Are Tuberculosis or Heart Failure

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CASE VIGNETTE

A 62-year-old female with a background of Hashimoto's thyroiditis presented with 14 days of pleuritic chest pain followed by an acute-onset non-productive cough and progressive exertional dyspnea. Examination revealed reduced air entry and decreased vocal fremitus over the left infra-axillary and infra-scapular regions. Inflammatory markers were markedly elevated (CRP 186 mg/L), with no documented fever.

Chest radiograph showed bilateral pleural effusion (L > R). Pleural fluid analysis revealed an exudative effusion (protein 4.1 g/dL) with mononuclear predominance (80%), low ADA, and sterile microbiology (negative Gram stain, KOH, AFB, CBNAAT, and cultures). Cardiac evaluation (2D-ECHO, CT-CAG, cardiac biomarkers) was unremarkable. HRCT thorax demonstrated fatty proliferation with pericardial thickening, suggestive of a chronic inflammatory process.

The patient showed clinical and radiological improvement with NSAIDs alone, along with a declining CRP trend.

Ques. What is the most likely driver of this patient's bilateral exudative pleural effusion with pericardial thickening?

A. Tuberculous pleurisy

B. Congestive heart failure

C. Acute bacterial mediastinitis

D. Post-viral inflammatory mediastinitis with reactive pleuro-pericarditis

ANSWER: D. Post-viral inflammatory mediastinitis with reactive pleuro-pericarditis.

COMMENTS

This case highlights a non-infectious, inflammatory presentation of mediastinitis with reactive pleural involvement. The alternative diagnoses are less likely for the following reasons:

Option A: Tuberculous pleurisy is unlikely because the pleural fluid had low ADA, sterile microbiology, absence of constitutional symptoms, and showed rapid clinical and radiological improvement with NSAIDs rather than anti-tubercular therapy.

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Option B: Congestive heart failure is improbable as it typically produces transudative pleural effusions and this patient had a clear exudate with lymphocyte predominance and a completely normal cardiac evaluation

Option C: Acute bacterial mediastinitis is unlikely because it usually presents with fever, systemic toxicity, neutrophil-predominant inflammation, and progressive clinical deterioration, none of which were seen, and such cases would not respond to NSAIDs alone..

While bacterial mediastinitis and tuberculous pleurisy are important differentials in endemic settings, the presence of a sterile, lymphocyte-predominant exudative pleural effusion, low ADA, absence of fever, and a marked clinical and radiological response to NSAIDs rather than antibiotics strongly support a post-viral or post-infectious reactive pleural process. Such sterile exudative effusions are well described in post-viral inflammatory states and often resolve with supportive or anti-inflammatory therapy without the need for prolonged antimicrobial treatment.[12]

The HRCT finding of fatty proliferation with pericardial thickening further suggests a subacute inflammatory involvement of mediastinal structures rather than an active infective pathology.

Distinguishing post-viral inflammatory conditions from true infectious aetiologies is a clinically critical step. Infectious pleural effusions — including tuberculous pleurisy — typically demonstrate lymphocyte predominance with elevated ADA, arise in the context of constitutional symptoms (fever, night sweats, weight loss), and do not improve with anti-inflammatory therapy alone. By contrast, post-viral or post-infectious reactive effusions are characterised by sterile cultures, low or normal ADA, temporal association with a preceding viral illness, and prompt resolution with NSAIDs or corticosteroids without any antimicrobial agent. The key distinguishing features in this case — low ADA, sterile microbiology across multiple modalities (Gram stain, KOH, AFB smear, CBNAAT, and cultures), absence of fever, mononuclear-predominant exudate, and rapid NSAID response — collectively and robustly exclude an infectious aetiology.

The decision to withhold both antibiotics and anti-tubercular therapy was reached through careful, stepwise clinical reassessment rather than reflexive empiric treatment. The treating team recognised that initiating anti-tubercular therapy without microbiological confirmation carries a substantial patient burden: a minimum six-month regimen, significant hepatotoxicity risk, potential for drug resistance, and unnecessary patient stigmatisation. Similarly, broad-spectrum antibiotics were not initiated because the clinical picture — haemodynamic stability, absence of systemic sepsis, sterile effusions on comprehensive testing, and a coherent alternative explanation — did not support infection. This case underscores that clinical confidence to withhold antimicrobials, grounded in systematic diagnostic evaluation, is itself an important stewardship act.



Follow-up at 2 weeks: marked resolution of pleural effusion

ANTIMICROBIAL STEWARDSHIP PERSPECTIVE

This case carries important lessons for antimicrobial stewardship, particularly in tuberculosis-endemic regions where the threshold for empiric anti-tubercular therapy is often low. Three stewardship principles are illustrated here. First, avoidance of unnecessary anti-tubercular therapy and antibiotics: the diagnosis was reached through careful synthesis

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of negative microbiological evidence and a supportive clinical narrative, allowing the team to confidently defer antimicrobial treatment. Second, the role of diagnostic stewardship: sequential and appropriate use of ADA measurement, CBNAAT, microbiology cultures (including AFB), and cross-sectional imaging enabled a definitive non-infectious diagnosis without the need for surgical biopsy or empiric therapeutic trials — each test contributed incrementally to narrowing the differential. Third, the importance of clinical reassessment before initiating empiric therapy: rather than committing to treatment based solely on the initial presentation in a high TB-prevalence setting, the team re-evaluated the cumulative diagnostic picture before any antimicrobial was prescribed. This reassessment — integrating effusion characteristics, the inflammatory marker trend, HRCT findings, and the therapeutic response to NSAIDs — was the cornerstone of responsible clinical decision-making and successful stewardship.

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