Yemen Journal of Medicine

https://yemenjmed.com

Article information

DOI: 10.63475/yjm.v5i1.0312

Article history:
Received: 05 March 2026
Accepted: 25 April 2026
Published: 30 April 2026

Correspondence to:
Theeb Osama Sulaiman
Email: Theebpalestine.21@gmail.com
ORCID: 0000-0003-4812-3459

How to cite this article
Sulaiman TO, Hassan EA. Mesothelial cell–rich tuberculous pleuroperitoneal effusion in an immunocompetent patient: a diagnostic pitfall. Yemen J Med. 2026;1(1):211-214.

Case Report

Mesothelial Cell–Rich Tuberculous Pleuroperitoneal Effusion in an Immunocompetent Patient: A Diagnostic Pitfall

Theeb Osama Sulaiman[1], Ebtihal Abdelmoneim Hassan[2]

1 Specialist, Pulmonology Department, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar
2 Consultant, Department of Accident and Emergency, Hamad General Hospital, Doha, Qatar

ABSTRACT

Tuberculous pleural effusion typically presents as an exudative effusion characterized by lymphocytic predominance and a notable scarcity of mesothelial cells, with studies confirming that pleural fluid from tuberculous patients rarely contains more than 5% mesothelial cells. We present a case of an immunocompetent patient who presented with concurrent ascites and right-sided exudative pleural effusion, both featuring elevated mesothelial cell counts. This finding represents a diagnostic pitfall, as high mesothelial cell counts often divert clinical suspicion toward malignancy or acute pleuritis rather than infection. Subsequent investigations, including peritoneal biopsy, confirmed the diagnosis of tuberculosis. The patient was started on anti-tuberculous drugs, resulting in significant clinical improvement and radiological resolution of the effusions.

Key words: Tuberculosis, tuberculous pleural effusion, mesothelial cells, ascites, diagnostic pitfall

INTRODUCTION

Tuberculosis (TB) remains a pervasive public health challenge, particularly in developing nations. While the majority of TB cases manifest as pulmonary tuberculosis, approximately 25% of adult patients exhibit extrapulmonary TB, affecting organs such as the lymph nodes, pleura, central nervous system, and gastrointestinal tract. [1]

Tuberculous pleural effusion (TPE) is the second most common form of extrapulmonary TB. [2] It typically results in an exudative effusion characterized by a lymphocytic predominance, traditionally accompanied by a scarcity of mesothelial cells in the pleural fluid. This paucity of mesothelial cells is attributed to the thick fibrin layer covering the pleura and the destruction of mesothelial cells by the intense inflammatory process. [3] Consequently, mesothelial cell counts have become a standard diagnostic parameter, with studies confirming that pleural fluid from TB patients rarely contains more than 5% mesothelial cells. [4, 5] However, reported cases of TPE with elevated mesothelial cells, particularly in HIV-positive patients, emphasize the variability of presentations and the potential for diagnostic confusion. [6] In this report, we present a rare case of an immunocompetent patient with tuberculous pleuroperitoneal effusion featuring significantly elevated mesothelial cell counts, highlighting the importance of not excluding TB based on cytological findings alone.

CASE REPORT

A 33-year-old male with an unremarkable past medical history presented with a 3-month history of asthenia, anorexia, and a 10 kg weight loss, accompanied by abdominal

distension. He reported an intermittent dry cough but denied fever, dyspnea, hemoptysis, or lower extremity edema. The patient was a former smoker (10-pack-year history) who had quit 1 year prior, and he had a negative family history of TB or malignancies.

Upon initial evaluation, vital signs were stable except for a heart rate of 110 beats/min. Oxygen saturation was 98% on room air. Physical examination revealed reduced air entry and dullness on percussion at the right lung base. Abdominal examination showed tense ascites with positive shifting dullness, but no organomegaly or lower limb edema was noted.

Laboratory investigations showed mild anemia (hemoglobin, 11.2 g/dL), hypoalbuminemia (albumin, 2.7 g/dL), and an elevated C-reactive protein (48 mg/L). HIV testing was negative. An abdominal ultrasound demonstrated mild ascites without organomegaly. A chest X-ray showed blunting of the right costophrenic angle (**Figure 1**), and a chest ultrasound confirmed a moderate right-sided pleural effusion.

Diagnostic thoracentesis and paracentesis were performed. Ascitic fluid analysis revealed a Serum Ascites Albumin Gradient of 0.23 g/dL, consistent with an exudate. The ascitic differential showed a lymphocyte predominance (72%) with 3% neutrophils and numerous mesothelial cells. Pleural fluid analysis was also exudative; however, the cytology showed a lymphocyte percentage of only 26%, neutrophils of 2%, and a striking mesothelial cell percentage of 31%. The remaining differential consisted predominantly of macrophages.

The initial TB workup, including acid-fast bacillus smear, GeneXpert MTB/RIF PCR, and culture, was negative in both ascitic and pleural fluid samples. Due to the posterior location of the pleural fluid and the feasibility of accessing the ascitic fluid, a medical thoracoscopy was deferred in favor of a laparoscopic approach. A laparoscopic peritoneal biopsy was performed, and histopathology revealed necrotizing granulomatous inflammation, consistent with RB.

The patient was initiated on standard anti-tuberculous therapy (isoniazid, rifampicin, pyrazinamide, and ethambutol). He tolerated the treatment well and showed significant clinical improvement. A follow-up chest X-ray 2 months later demonstrated complete resolution of the pleural effusion (**Figure 2**).

DISCUSSION

The differential diagnosis of an exudative pleural effusion is broad, encompassing malignancy, parapneumonic effusion, TB, pulmonary embolism, and rheumatologic diseases. [7] In the context of TB, TPE typically exhibits a lymphocytic predominance due to a delayed hypersensitivity reaction mediated by T lymphocytes. [4] Although TPE may initially present with neutrophilic predominance during the early acute phase (usually within the first 2 weeks), the combination observed in our case—relatively low lymphocyte proportion (26%) alongside a markedly elevated mesothelial cell count (31%)—is distinctly atypical and presents a significant diagnostic challenge. [8]

Importantly, the cytological profile of TPE is not static but evolves. In the early phase of pleural involvement, effusions may demonstrate a neutrophil-predominant or mixed cellular pattern, reflecting an acute inflammatory response, before transitioning to the more characteristic lymphocyte-rich profile as cell-mediated immunity becomes established. During this dynamic process, mesothelial cell representation may also fluctuate, particularly before the formation of the dense fibrinous layer that typically limits mesothelial exfoliation in established TPE. Thus, the unusual cytological findings in this case may represent a transitional phase captured at a specific

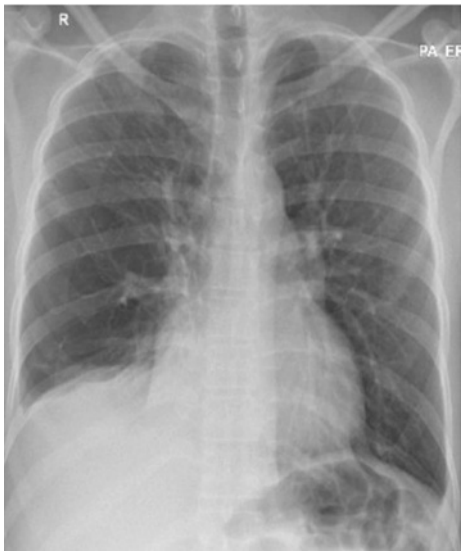

Figure 1: Chest X-ray (posteroanterior view) showing mild blunting of the right costophrenic angle. Subsequent chest ultrasound revealed a moderate right-sided pleural effusion that was predominantly posterior and loculated, indicating that plain radiography significantly underestimated effusion size.

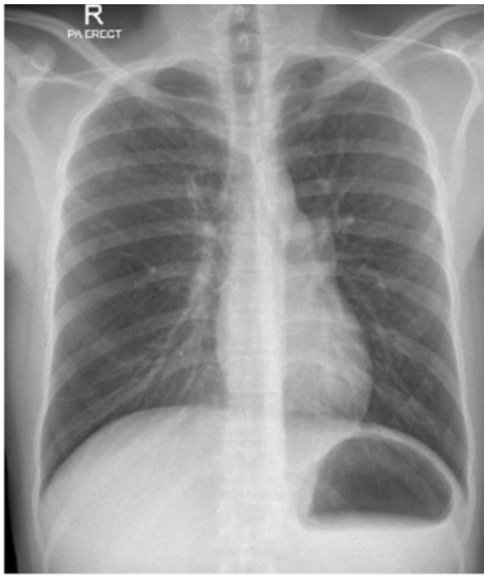

Figure 2: Chest X-ray 2 months after initiation of anti-tuberculous therapy, demonstrating complete resolution of the right pleural effusion with sharp and clear costophrenic angles. No residual parenchymal abnormality is seen.

point in disease evolution rather than a true deviation from classical pathophysiology. This temporal variability highlights the limitations of relying on a single cytological snapshot and reinforces the importance of integrating clinical suspicion with further diagnostic evaluation, particularly when TB remains a consideration.

The pleural mesothelium, derived from embryonic mesoderm, forms a monolayer lining both parietal and visceral pleural surfaces and plays a key role in maintaining pleural homeostasis and responding to injury. [9] The relative absence of mesothelial cells in classical TPE, first described in 1960, has been attributed to extensive chronic inflammation, which either destroys the mesothelium or covers it with a thick fibrinous exudate. [5] Consequently, a mesothelial cell proportion exceeding 5% has traditionally been used to argue against a diagnosis of TB and to favor alternative etiologies such as malignancy or viral pleuritis. [7]

Nevertheless, elevated mesothelial cell counts in TPE have been documented, most commonly in HIV-infected individuals, where altered immune responses—particularly reduced expression of interferon-γ and macrophage inflammatory mediators—may promote increased mesothelial cell exfoliation into the pleural space. [10] Such findings in immunocompetent patients remain exceedingly rare. [6]

In this context, several contributing factors have been proposed, including hypoalbuminemia and systemic conditions such as congestive heart failure, liver cirrhosis, and renal dysfunction, which may alter pleural fluid dynamics and mesothelial integrity. [11] Our patient, while immunocompetent, demonstrated moderate hypoalbuminemia (27 g/L), which may have facilitated increased mesothelial shedding and contributed to the atypical cytological profile.

Furthermore, the discordance observed between pleural and ascitic fluid cytology in this case—with lymphocyte predominance in ascitic fluid but not in pleural fluid—highlights the heterogeneous nature of TB-related serosal involvement within the same individual. [12] This variability may reflect differences in local immune responses, disease chronicity, or microenvironmental factors across serosal compartments.

Limitations

Several limitations merit consideration. First, pleural biopsy—the gold standard for confirming tuberculous pleuritis—was not performed due to technical constraints related to posterior loculation of the effusion, limiting histopathological confirmation of pleural involvement. Second, key adjunctive biomarkers such as adenosine deaminase levels in pleural and ascitic fluid were not reported, which could have provided additional supportive evidence. Third, while alternative diagnoses such as malignancy were considered less likely given the absence of supporting clinical, cytological, and radiological features, they were not definitively excluded by tissue diagnosis from the pleura. Finally, the atypically high mesothelial cell count introduces diagnostic ambiguity and underscores the potential for misclassification if reliance is placed solely on cytological patterns.

Overall, this case highlights the importance of integrating clinical features, fluid analysis, histopathology, and therapeutic response when evaluating atypical pleuroperitoneal effusions, particularly in TB-endemic settings where microbiological confirmation may be elusive.

CONCLUSIONS

The primary objective of this case report is to raise awareness that elevated mesothelial cell counts in pleural fluid do not categorically exclude the diagnosis of TB, even in immunocompetent individuals. While a lymphocyte-predominant exudate remains the standard presentation, clinicians must remain vigilant for atypical cytological patterns. In cases of diagnostic uncertainty, tissue diagnosis via biopsy (either pleural or peritoneal) remains the gold standard for confirming tuberculous etiology and ensuring appropriate management.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case report.

ETHICAL APPROVAL

The case report is approved by the Medical Research Center at Hamad Medical Corporation and the Hamad Institutional Review Board (IRB) under number MRC-04-24-439.

AUTHORS' CONTRIBUTION

All authors have significantly contributed to the work, whether by following the case at the bedside, conducting literature searches, drafting, revising, or critically reviewing the article. They have given their final approval of the version to be published, have agreed with the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

SOURCE OF FUNDING

None.

CONFLICT OF INTEREST

None.

REFERENCES

1. Lee JY. Diagnosis and treatment of extrapulmonary tuberculosis. Tuberc Respir Dis (Seoul). 2015;78(2):47-55. https://doi.org/10.4046/trd.2015.78.2.47
2. Vorster MJ, Allwood BW, Diacon AH, Koegelenberg CF. Tuberculous pleural effusions: advances and controversies. J Thorac Dis. 2015;7(6):981-991. https://doi.org/10.3978/j.issn.2072-1439.2015.02.18
3. Spriggs AI, Boddington MM. Absence of mesothelial cells from tuberculous pleural effusions. Thorax. 1960;15(2):169-171. https://doi.org/10.1136/thx.15.2.169
4. Cohen LA, Light RW. Tuberculous pleural effusion. Turk Thorac J. 2015;16(1):1-9. https://doi.org/10.5152/ttd.2014.001
5. Jany B, Welte T. Pleural effusion in adults-etiology, diagnosis, and treatment. Dtsch Arztebl Int. 2019;116(21):377-386. https://doi.org/10.3238/arztebl.2019.0377
6. Jones D, Lieb T, Narita M, Hollender ES, Pitchenik AE, Ashkin D. Mesothelial cells in tuberculous pleural effusions

of HIV-infected patients. Chest. 2000;117(1):289-291. https://doi.org/10.1378/chest.117.1.289
7. Light RW. Pleural effusions. Med Clin North Am. 1977;61(6):1339-1352. https://doi.org/10.1016/s0025-7125(16)31265-2
8. Batra H, Antony VB. Pleural mesothelial cells in pleural and lung diseases. J Thorac Dis. 2015;7(6):964-980. https://doi.org/10.3978/j.issn.2072-1439.2015.02.19
9. Batra H, Antony VB. The pleural mesothelium in development and disease. Front Physiol. 2014;5:284. https://doi.org/10.3389/fphys.2014.00284
10. Barnes PF, Fong SJ, Brennan PJ, Twomey PE, Mazumder A, Modlin RL. Local production of tumor necrosis factor and IFN-gamma in tuberculous pleuritis. J Immunol. 1990;145(1):149-154.
11. Cheng SL, Wang HC, Yang PC, Kuo SH. Risk factors for high mesothelial cell counts in HIV-negative patients with tuberculous pleural effusion. J Formos Med Assoc. 2010;109(6):456-462. https://doi.org/10.1016/s0929-6646(10)60077-0
12. Luzze H, Elliott AM, Joloba ML, Odida M, Oweka-Onyee J, Nakiyingi J, et al. Evaluation of suspected tuberculous pleurisy: clinical and diagnostic findings in HIV-1-positive and HIV-negative adults in Uganda. Int J Tuberc Lung Dis. 2001;5(8):746-753.