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Case Report

Internal Medicine Section

Tuberculous Pleural Effusion Complicated by Empyema Thoracis in a Newly Diagnosed PLWHA: A Case Report

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ABSTRACT

The co-existence of Pulmonary Tuberculosis (PTB), empyema thoracis, and Human Immunodeficiency Virus (HIV) infection constitutes a formidable diagnostic and therapeutic challenge. In immunocompromised individuals, such triple pathology can evolve rapidly and is associated with significant morbidity and mortality, particularly in TB-endemic regions. A 32-year-old male with a past history of pulmonary TB, 15 years prior presented with progressive exertional dyspnoea, dry cough, right-sided pleuritic chest pain, low-grade fever for seven days, and significant weight loss of one month duration. SpO_2 was 90% on room air. Chest X-ray demonstrated complete opacification of the right hemithorax without tracheal deviation. HRCT thorax revealed gross multiloculated pleural effusion with underlying lung collapse and smooth interlobular septal thickening. Pleural fluid analysis confirmed empyema. HIV serology was reactive and pleural fluid Cartridge-Based Nucleic Acid Amplification Test (CB-NAAT) was positive for Mycobacterium tuberculosis (MTB). Sputum culture identified Klebsiella pneumoniae sensitive to amikacin and tigecycline. The patient was managed with intercostal drain insertion, broad-spectrum antibiotics (Piperacillin-Tazobactam and Levofloxacin), Anti-Koch's Therapy (AKT), and amikacin, with gradual clinical and biochemical improvement. This case underscores the critical importance of systematic microbiological workup including CB-NAAT, pleural fluid Adenosine Deaminase (ADA), and HIV screening in all patients presenting with pleural effusion in endemic regions. The triad of TB, empyema, and HIV demands a multidisciplinary approach with concurrent management of all three conditions for favourable outcomes.

Keywords: Adenosine deaminase, Anti-Koch's therapy, CB-NAAT, Multiloculated effusion, Person living with HIV/AIDS

CASE REPORT

A 32-year-old male presented to the emergency department with a chief complaint of progressively worsening breathlessness on exertion of 10 days duration. The breathlessness was of gradual onset, beginning insidiously and worsening over the course of 10 days, modified Medical Research Council (mMRC) Dyspnoea Scale Grade 2, with the patient experiencing breathlessness when walking on level ground or climbing a gentle slope. This was associated with a dry cough and right-sided chest pain that was sharp and pleuritic in nature, of gradual onset, worsening progressively over the same 10-day period, and consistently aggravated on deep inspiration and coughing. He additionally reported mild diffuse abdominal pain, nausea, and a significant unintentional weight loss of approximately 4 kg over the preceding two months. A 7-day history of low-grade fever was also noted.

The patient had no known comorbid illness at the time of presentation. However, he had a significant history of PTB 15 years prior, for which he had completed a full course of anti-tubercular therapy. HIV status was found positive on screening at admission. On detailed history taking, the patient had received intravenous fluids and injectable medications on multiple occasions in past for recurrent episodes of fever and generalised weakness, with possibility of parenteral exposure risk factor for HIV transmission, likely via non-sterile or inadequately sterilised equipment in informal healthcare settings.

Physical Examination

On examination, temperature 100°F (37.8°C), pulse rate 112 beats/minute, blood pressure 140/70 mmHg, Glasgow Coma Scale (GCS) E4M6V5 (15/15), SpO2 90% on room air. General examination revealed a lean, ill-appearing male in respiratory distress. The chest was ellipsoid and symmetrical, with drooping of the right shoulder. Bilateral hollow supraclavicular fossae were noted with active use of accessory respiratory muscles.

Respiratory system examination suggested dull note on percussion over the right mammary, axillary, infraaxillary, interscapular, and infrascapular areas. Auscultation demonstrated decreased air entry over the right infraaxillary and infrascapular regions with bilateral generalised rhonchi. No tracheal deviation was detected. Abdominal examination revealed mild tenderness without clinically apparent organomegaly.

Investigations

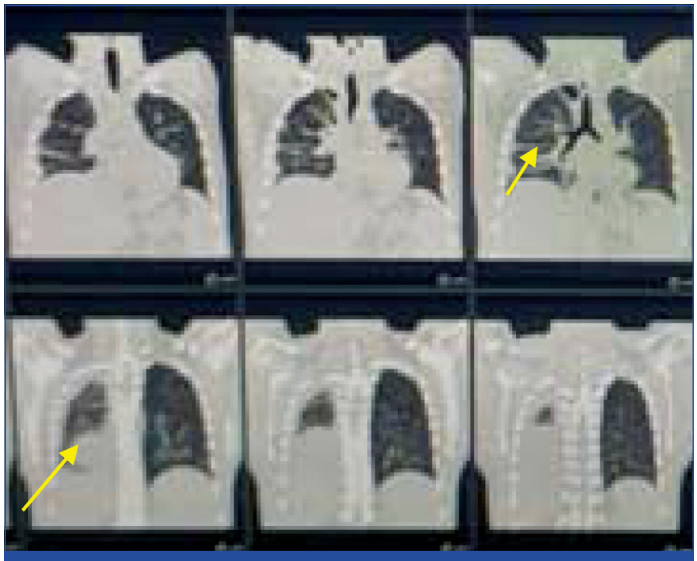
Haematological investigations revealed mild anaemia, mild leucocytosis, and thrombocytopenia, with markedly elevated inflammatory markers indicative of a significant systemic inflammatory response with concurrent bacterial infection. Liver function tests showed mild transaminitis with an SGOT: SGPT ratio of >1 and a predominantly indirect hyperbilirubinaemia, suggestive of hepatic involvement in the setting of TB-HIV co-infection, while renal parameters and electrolytes remained within normal limits [Table/Fig-1].

Chest X-ray (PA view) showed complete whiteout of the right lung field without tracheal deviation. HRCT thorax confirmed a gross multiloculated pleural effusion involving diaphragmatic, costal, and mediastinal pleura on the right side with underlying lung collapse and smooth interlobular septal thickening [Table/Fig-2-5]. Ultrasound thorax demonstrated gross echogenic right pleural effusion. Ultrasound abdomen revealed hepatomegaly (17 cm span) with Grade 1 fatty liver and mild splenomegaly (14.6 cm).

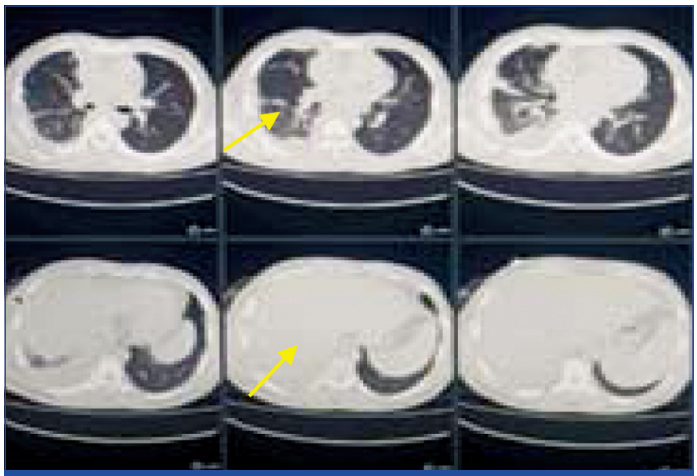
Diagnostic and therapeutic pleural fluid aspiration was performed. Pleural fluid analysis revealed a turbid fluid with a total leucocyte count of 20,000 cells/cumm, demonstrating a predominance of polymorphonuclear cells (70%) and 10% lymphocytes. Biochemical

Parameter (Normal Range)	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Hb (g/dL) (13.5-17.5)	10.9	9.6	9.0	9.0	8.9	9.1	9.7	9.0
TLC (/cumm) (4,000-11,000)	11,200	19,500	14,500	16,000	22,300	14,640	12,000	10,580
Platelets (lac/cumm) (1.5-4.5)	0.77	0.82	1.52	2.26	3.12	4.5	4.36	3.56
PT/INR (11-13.5s/ <1.1)	15.1/1.07	16/1.1	18.9/1.35	–	16/1.14	–	15/1.07	–
APTT (sec) (25-35)	28.9	36	37.7	–	34	–	44	–
Creatinine (mg/dL) (0.7-1.2)	0.7	0.8	0.8	0.9	1.2	1.0	0.8	0.8
Bilirubin (mg/dL) (<1.2) (D/I)	2.0 (0.7/1.3)	2.5 (1.6/0.9)	2.3 (1.3/1.0)	1.4 (0.8/0.6)	1.2 (0.7/0.5)	0.8 (0.4/0.4)	0.6 (0.3/0.3)	–
Serum Protein (g/dL)	6.3	–	–	–	–	–	–	–
SGOT (IU/L) (<40)	155	105	91	88	86	90	–	80
SGPT (IU/L) (<41)	73	69	50	41	44	54	40	–
LDH (IU/L) (140-280)	441	–	865	–	–	–	–	–
CRP (mg/L) (<5)	124	–	102	–	50	–	20	–
PCT (ng/mL) (<0.5)	1.7	–	–	–	1.0	–	–	–
Na (mEq/L) (136-145)	137	140	142	146	138	139	142	–
K (mEq/L) (3.5-5.0)	3.9	4.0	4.5	3.4	4.0	5.0	4.4	–
ESR (mm/hr)	108	–	–	–	–	–	–	–

[Table/Fig-1]: Serial haematological and biochemical parameters during hospital stay.
Hb: Haemoglobin; TLC: Total leucocyte count; PT: Prothrombin time; INR: International normalised ratio; APTT: Activated partial thromboplastin time; SGOT: Serum glutamate oxaloacetate transaminase; SGPT: Serum glutamate pyruvate transaminase LDH: Lactate dehydrogenase; CRP: C-Reactive protein; PCT: Procalcitonin; Na: Sodium; K: Potassium; ESR: Erythrocyte sedimentation rate D: Direct; I: Indirect. (–) denotes not tested on that day.

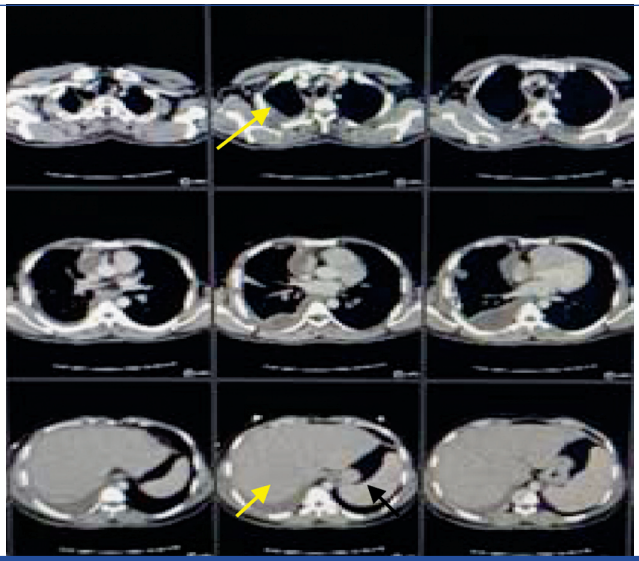


[Table/Fig-2]: Coronal CECT Thorax shows the gross right-sided white-out with multiloculated effusion, underlying lung collapse, and mediastinal pleural involvement.

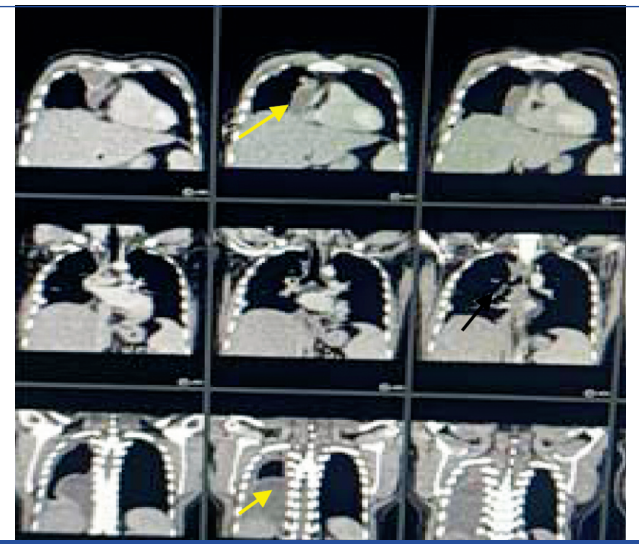


[Table/Fig-3]: HRCT Thorax lung window (axial) Top row: upper lobe cuts- bilateral lung parenchyma with right-side opacification beginning. Middle row: mid-zone cuts- right lung collapse with interlobular septal thickening clearly visible. Bottom row: lower zone/basal cuts- near-complete right opacification, left lung clear.

analysis showed a Lactate Dehydrogenase (LDH) of 2,000 IU/L against a serum LDH of 441 IU/L, pleural fluid protein of 4.6 g/dL against a serum protein of 6.3 g/dL, and a markedly elevated



[Table/Fig-4]: Axial CECT of the thorax and upper abdomen. Upper rows demonstrate massive right-sided pleural effusion causing compression of the right lung with mediastinal shift. Lower rows reveal liver and spleen enlargement.



[Table/Fig-5]: HRCT Thorax coronal and saggital lung window reconstructions Top row: coronal cuts showing right-sided effusion with left lung aeration preserved. Middle row: deeper coronal cuts showing collapsed right lung with pleural effusion. Bottom row: near-sagittal/oblique reconstructions showing spinal anatomy and bilateral lung fields.

Adenosine Deaminase (ADA) of 100 U/L. By Light's criteria, the fluid was classified as an exudate. The turbid macroscopic appearance in conjunction with marked polymorphonuclear predominance was consistent with a diagnosis of empyema thoracis. Pleural fluid AFB smear was negative; however, pleural fluid Cartridge-Based Nucleic Acid Amplification Test (CB-NAAT) returned positive for MTB, confirming tuberculous empyema as the underlying aetiology.

Sputum Gram stain revealed 10-25 epithelial cells, moderate pus cells, and gram-positive cocci arranged in short chains and pairs. Sputum AFB smear was negative. Sputum culture and sensitivity identified the growth of Klebsiella pneumoniae, which demonstrated sensitivity to amikacin and tigecycline.

Treatment Course

Days 1-3: The patient was admitted to the Medical Intensive Care Unit (MICU) and commenced on supplemental oxygen via non-rebreather mask. Empirical broad-spectrum antibiotics Inj. Piperacillin–Tazobactam 4.5 g IV every 8 hours and Inj. Levofloxacin 750 mg IV once daily were administered. On Day 1, a diagnostic pleural tap confirmed empyema biochemically; in view of the massive multiloculated effusion on HRCT, an intercostal drain (28F) was inserted under aseptic precautions using the Seldinger technique in the right fifth intercostal space mid-axillary line and connected to a water-seal system. Approximately 800 mL of turbid, yellowish-brown fluid drained on Day 1, with immediate symptomatic relief. Drainage continued at 400–500 mL/day over Days 2–3; SpO2 improved to 94–95% on 4L/min nasal cannula. Platelet counts began recovering from 0.77 to 1.52 lacs/cumm by Day 3.

Days 3-5: Pleural fluid CB-NAAT (GeneXpert MTB/RIF) returned positive for MTB with no rifampicin resistance on Day 3. Antitubercular Therapy (AKT) was initiated as per National Tuberculosis Elimination Programme (NTEP) guidelines. (extrapulmonary TB, new case) with the four-drug intensive phase regimen: Isoniazid 300 mg, Rifampicin 450 mg, Pyrazinamide 1,500 mg, and Ethambutol 800 mg- all once daily under DOT- along with Pyridoxine 40 mg OD. Sputum culture sensitivity (Day 4) identified Klebsiella pneumoniae sensitive to amikacin; Inj. Amikacin 15 mg/kg i.v. OD was added and Levofloxacin discontinued to rationalise the regimen and minimise hepatic enzyme interactions with AKT. By day 5, the patient was afebrile with an SpO_2 of 94% on 2 L/min oxygen support. The ICD output was approximately 200–250 mL over 24 hours, C-reactive protein levels showed a declining trend (124 to 50 mg/L), and the platelet count improved to 3.12 lakhs/cumm. The infectious disease team deferred ART pending CD4 count and viral load results, as per national HIV-TB co-treatment guidelines.

Days 6-7: ICD Removal and Step-Down to Ward

By Day 6, ICD output had fallen to <50 mL/24 h. Bedside ultrasound confirmed no residual drainable collection. The drain was clamped for 12 hours without clinical deterioration and removed on Day 7; a post-removal chest X-ray showed no pneumothorax and partial right lung re-expansion. The patient was simultaneously stepped down from MICU to the general ward, meeting all criteria: sustained haemodynamic stability, SpO2 >95% on room air, afebrile >24 hours, and tolerating oral medications; Amikacin i.v. was continued with creatinine monitoring along with piperacillin tazobactam for 14 days. AKT continued under DOT. Physiotherapy with incentive spirometry was initiated to aid lung re-expansion.

Days 8-14: Ward Consolidation and Discharge

The patient remained afebrile, fully ambulatory, and maintaining SpO2 97% on room air. Chest X-ray on Day 9 demonstrated significant right lung re-expansion with a small residual basal opacity consistent with post-empyema fibrotic change; repeat ultrasound confirmed no collection. CRP declined to 20 mg/L, platelets 3.56 lacs/cumm, and Hb stable at 9.0 g/dL (oral iron supplementation added). The patient was discharged on Day 14 on: AKT (HRZE) under DOT for 2 months intensive phase followed by HRE for four months, Pyridoxine 40 mg OD, Iron-Folic Acid OD, and multivitamins. He was counselled on AKT hepatotoxicity warning signs and enrolled with ICTC and the ART centre.

Follow-Up after 15 Days Post-Discharge

After 15-days OPD follow-up (Day 25 from admission), the patient reported resolution of dyspnoea, cough, and fever, improved appetite, and a 1.5 kg weight gain. He had maintained 100% DOT compliance. Vitals were normal: SpO2 98% on room air. Respiratory examination was clear bilaterally; the ICD wound site was well-healed. Investigations showed Hb 10.2 g/dL, TLC 8,400/cumm, platelets 3.8 lacs/cumm, CRP 12 mg/L, bilirubin 0.5 mg/dL, and mildly elevated SGOT/SGPT (52/38 IU/L- within acceptable <3× ULN range per NTEP guidelines; AKT continued unchanged). Chest X-ray demonstrated near-complete right lung re-expansion with a small residual pleuro-parenchymal scar. CD4 count was 180 cells/µL; ART was planned at the 4-week mark per national guidelines (CD4 >50 cells/µL, clinically stable at 2 weeks of AKT). The patient was counselled on Immune Reconstitution Inflammatory Syndrome (IRIS) and scheduled for further follow-up at one and two months post-discharge. On first month follow-up, the patient showed marked clinical improvement with no dyspnoea, cough, or fever with 100% DOT compliance, 2 kg weight gain from baseline , and ART (Tenofovir + Lamivudine + Dolutegravir-TLD) initiated; chest X-ray demonstrated near-complete resolution of pleural effusion with a small residual basal pleural thickening. On second month follow-up, the patient was fully ambulatory with a total weight gain of 4.5 kg; AKT was transitioned to the continuation phase (HRE) per NTEP guidelines and ART was well tolerated with no IRIS features.

DISCUSSION

Pleural Effusion in a Person Living with HIV/AIDS (PLWHA) carries a broad differential diagnosis encompassing infectious, malignant, and immunological aetiologies, with TB remaining the most prevalent cause in endemic countries [1,2]. The co-occurrence of tuberculous pleuritis with bacterial superinfection leading to empyema represents a particularly complex clinical scenario, as empyema thoracis- defined by the presence of pus in the pleural space or a positive bacterial culture from pleural fluid- when superimposed upon pre-existing tuberculous pleuritis in an immunocompromised host, renders the clinical presentation atypical and microbiological identification indispensable [3,4]. HIV-associated immunosuppression further predisposes such patients to concurrent infections by multiple pathogens, including both MTB and gram-negative organisms such as Klebsiella pneumonia- a notoriously drug-resistant pathogen of increasing clinical relevance [5]. The simultaneous occurrence of tuberculous empyema, Klebsiella pneumoniae superinfection, and newly detected HIV infection in a patient with prior tubercular history, as illustrated in the present case, is exceedingly rare and diagnostically challenging.

The present case describes a 32-year-old HIV-positive male with a prior history of PTB who developed a right-sided tuberculous empyema confirmed by CB-NAAT, with concurrent Klebsiella pneumoniae identified on sputum culture- sensitive only to amikacin and tigecycline, consistent with an Extended-Spectrum Beta-Lactamase (ESBL)-producing resistant phenotype [5]. The clinical presentation of progressive exertional dyspnoea, pleuritic chest pain, low-grade fever, and significant weight loss is consistent with the well-established syndromic profile of HIV-TB co-infection with pleural involvement. The pleural fluid demonstrated markedly atypical PMN predominance (70%), TLC of 20,000 cells/cumm, LDH of 2,000 IU/L, and an elevated ADA

of 100 U/L- findings that, together with a positive CB-NAAT and turbid macroscopic appearance, were diagnostic of tuberculous empyema with superimposed bacterial co-infection. This case further illustrates three concurrent pathological processes: reactivation of TB-associated pleural disease in a patient with past PTB; bacterial superinfection with Klebsiella pneumoniae causing empyema thoracis; and newly detected HIV infection facilitating both processes through profound immunosuppression.

The TB is the dominant aetiology of pleural effusion in HIV-positive patients across endemic regions, a finding robustly corroborated by multiple studies [6]. Joshi N et al., in their prospective evaluation of 150 HIV-infected patients from India, reported pleural effusion in 32 (22.6%) cases, with TB accounting for 28 (82.3%) cases; empyema thoracis was documented in 4 (11.7%) cases and all effusions were exudative in nature [7]. The landmark retrospective study by Relkin F et al., identified 70 patients with pleural TB over five years, of whom 43 were HIV-positive and 27 were HIV-negative, with HIV-positive patients being significantly younger at a mean age of 38 years compared to 52 years in HIV-negative patients [8]- a demographic profile entirely consistent with the present patient aged 32 years. Luzze H et al., in their comparative evaluation of 156 adults with suspected tuberculous pleurisy in Uganda- of whom 142 had confirmed TB and 80% were HIV-positive- further demonstrated that HIV-positive patients experienced a more severe and prolonged illness, with lower absolute pleural fluid lymphocyte counts, more frequently positive pleural fluid cultures, and a higher proportion of concurrent parenchymal infiltrates compared to HIV-negative patients, reflecting impaired immune containment and higher mycobacterial burden [9]. These observations are closely mirrored in the present case, where HIV-positive status was associated with an atypical and aggressive pleural fluid profile, underscoring the immunopathological complexity of pleural TB in the context of HIV co-infection.

The occurrence of thoracic empyema as both a primary presenting illness and a diagnostic trigger for HIV identification is well documented [6]. Hernández Borge J et al., in their nine-year retrospective study of 23 HIV-infected patients with thoracic empyema, reported a mean age of 28.7 years, with empyema representing the first cause of medical consultation in 65% of cases and leading to a new HIV diagnosis in 48% [10]. This pattern bears a direct parallel to the present case, where empyema was the index clinical event prompting HIV diagnosis at admission, and the risk factor profile- parenteral exposure through repeated intravenous injections- aligns with the parenteral transmission pathway documented in that cohort. The study further established that prompt chest tube drainage with targeted antibiotic therapy, without necessitating aggressive surgical intervention, resulted in favourable outcomes- a management principle directly applicable to the current case. Thrombocytopenia at presentation (platelets 0.77 lacs/cumm) in the present patient was likely multifactorial, attributable to HIV-associated immune thrombocytopenic purpura, sepsis-related consumptive thrombocytopenia, or bone marrow suppression, with gradual recovery parallel to clinical improvement on antimicrobial therapy suggesting an infective-reactive aetiology [11]. Elevated transaminases with indirect hyperbilirubinaemia were consistent with systemic sepsis, TB dissemination, or HIV-related hepatic involvement, all of which normalised during the hospital course.

The atypical PMN predominance in the present case is mechanistically explained by a triple-hit immunopathological process: acute neutrophilic influx triggered by rupture of a subpleural caseous focus, amplification via Toll-Like Receptor (TLR)-mediated cytokine release (Interleukin-8 [IL-8], IL-1β, Tumour Necrosis Factor-Alpha [TNF-α]) driven by concurrent Klebsiella pneumoniae infection, and HIV-mediated CD4+ T-cell depletion impairing the Th1-driven lymphocytic transition that would ordinarily follow in an immunocompetent host- resulting in a self-perpetuating neutrophil-dominant pleural microenvironment atypical for isolated tuberculous pleuritis alone [12]. Despite this atypical cytology, the markedly elevated ADA of 100 U/L alongside a positive CB-NAAT enabled definitive diagnosis, reinforcing that ADA remains a robust diagnostic marker in HIV-positive patients even in the context of impaired T-lymphocyte response, with levels above 40 IU/L carrying high sensitivity and specificity for tuberculous pleuritis in endemic populations [13,14]. CB-NAAT on pleural fluid provided rapid confirmation of MTB and is increasingly recommended as the test of choice in resource-limited settings, offering high specificity despite moderate sensitivity in pleural samples [15]. Vorster MJ et al., (2015) further corroborated this diagnostic paradigm, emphasising that in high-burden settings, elevated ADA combined with clinical pre-test probability justifies treatment initiation, while the gold standard remains microbiological or histological confirmation; the authors also highlighted that combining ADA with closed pleural biopsy approaches the diagnostic accuracy of thoracoscopy. Collectively, these findings affirm that in HIV-positive patients presenting with PMN-predominant exudative effusion, TB must never be excluded on cytological grounds alone, and that a multimodal diagnostic strategy incorporating ADA, CB-NAAT, and targeted microbiological workup remains the definitive clinical cornerstone [16].

CONCLUSION(S)

The triad of empyema thoracis, tuberculous pleural effusion, and newly detected HIV infection in a young male with prior TB represents a rare, diagnostically challenging, and potentially life-threatening presentation. Elevated ADA with CB-NAAT positivity is the most reliable diagnostic combination when AFB smear is negative; that multiloculated effusion mandates ICD insertion; and that concurrent Klebsiella superinfection, while uncommon, must be actively excluded. A systematic approach encompassing comprehensive microbiological workup, early intercostal drainage, concurrent anti-tubercular and targeted antibiotic therapy, and planned ART initiation constitutes the pillars of successful management. This case reinforces the need for a high index of suspicion for multimicrobial aetiology in immunocompromised patients presenting with pleural effusion in TB-endemic regions.

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