CASE REPORT

Pulmonary Empyema with Possible Tuberculosis Infection: A Case Report

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ABSTRACT

Introduction: Pulmonary empyema is an infectious disease with mortality and morbidity rates ranging between 3% and 33%. It occurs in approximately 8.9% of patients with tuberculosis (TB), and the associated mortality rate is approximately 20%. If not treated promptly following diagnosis, empyema may result in a poor prognosis. Therefore, this case report presented a patient with pulmonary empyema caused by a possible TB infection.

Case: A 57-year-old female presented with a two-week history of coughing up yellow phlegm and a three-day history of fever. The patient reported an untreated dental cavity in the right lower tooth for the past three weeks. Physical examination revealed decreased vesicular breath sounds in the right hemithorax's fifth to sixth intercostal space (ICS). Chest X-ray examination suggested pneumonia and right pleural effusion. Laboratory examinations revealed leukocytosis, thrombocytosis, and an increased neutrophil-to-lymphocyte ratio (NLR). The adenosine deaminase (ADA) level was 89 U/L. Ultrasonography (USG) of the right hemithorax confirmed right pleural effusion. A pleural puncture was performed, and a thoracic drainage was inserted. The procedure yielded approximately 1,700 cc of cloudy brown pus. The patient was diagnosed with right pulmonary empyema caused by tuberculous pleurisy. She received antibiotic therapy and a four-drug fixed-dose combination (FDC) of anti-TB therapy. After nine days of treatment, the patient's symptoms showed improvement.

Conclusion: Empyema is a complex disease with diverse etiologies and multifactorial pathogenesis. Early detection and prompt treatment are essential to minimize the risk of further complications.

INTRODUCTION

Pulmonary empyema is an infectious condition characterized by pus in the pleural cavity.[1] Empyema is a complex disease with multifactorial pathogenesis and diverse etiologies.[2] The mortality and morbidity rates range between 3% and 33%, with the hospital mortality rate in the adult population (over the age of 65 years old) at approximately 16.1%.[2,3]

Empyema has been reported to occur in 8.9% of patients with tuberculosis (TB).[4] A previous study suggested that the mortality rate among TB empyema patients is approximately 20%, with 20% requiring surgical intervention within one year of the initial infection to recover.[5] Tuberculosis empyema is a rare complication characterized by the accumulation of intrathoracic pus, which may decompress and extend through the parietal pleura and weakened chest wall, resulting in the accumulation of pus in the extrathoracic soft tissues due to *Mycobacterium tuberculosis* (MTB) infection. In addition, this complication presents a clinical challenge because it is frequently underdiagnosed. Tuberculosis empyema is commonly reported in older people with a mean age of 35.9±15.8 years old.[6]

Pleural aspiration, antibiotic therapy, and physiotherapy are standard treatment plans for empyema patients. If empyema is not promptly treated following diagnosis, it may result in a poor prognosis.[7,8] Although the majority of patients recover, clinical outcomes remain suboptimal. Approximately 20% of patients may die within the first year after

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diagnosis, and one in five patients may require surgical intervention. Patients may experience severe sepsis, septic shock, or even death as a result of the underlying pathological process of empyema.[9]

Therefore, in this case report, clinical data from the patient suggested a possible TB infection. However, this diagnosis was initially considered among differential diagnoses, which was confirmed through appropriate examinations. The therapeutic approach adopted in this case was based on established guidelines, expert judgement, and contextual considerations, particularly the high prevalence of TB in Indonesia, which influenced the management of this case.

CASE

A 57-year-old female was referred from an outside clinic complaining of coughing up brown-yellowish phlegm for two weeks and fever for approximately three days. The fever was described as coming and going, occasionally accompanied by chills that resolved spontaneously. The patient also reported occasional shortness of breath, which worsened during physical exertion, such as climbing steep stairs for more than five minutes. Her daily activities were mostly limited to staying at home, with a preference for sleeping. She reported an uncertain degree of weight loss and a decreased appetite. She denied any history of smoking.

According to the patient, there were elderly people in her home environment, either family members or close relatives, with a history of chronic cough, sometimes coughing up phlegm, and a history of smoking. The patient frequently interacted with them, such as during religious gatherings. However, she was uncertain whether they were undergoing or had previously received TB treatment. Moreover, the patient lived with her adult daughter. Her daughter reported that they lived in a cluster home with limited space. Their house had ventilation, and the patient's room was specifically equipped with a window allowing adequate circulation. There was no prior history of pulmonary disease. However, the patient had a history of heart disease and hypertension, for which she regularly took candesartan 8 mg.

Physical examination revealed a heart rate (HR) of 90 beats/minute, blood pressure of 144/70 mmHg, axillary temperature of 37.5°C, respiratory rate (RR) of 22 breaths/minute, and oxygen saturation of 98% with a nasal cannula at 2 lpm. An additional finding was the cavity in the lower right tooth, which had developed three weeks prior and had not been treated by a dental professional. Physical examination of the lungs showed symmetrical results, normal vocal fremitus, sonorous percussion, and decreased vesicular breath sounds in the fifth to sixth intercostal space (ICS) of the right hemithorax. Minimum rhonchi were heard in the upper and middle parts of the right lung.

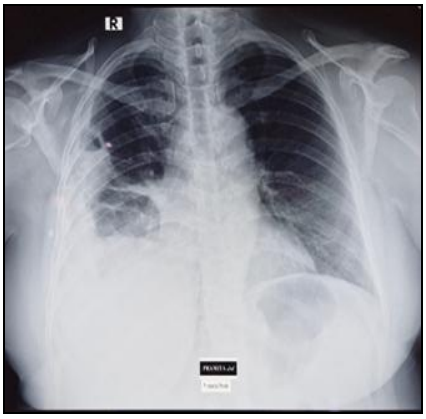

Figure 1. Chest X-ray showing right pleural effusion

Chest X-ray examination suggested suspected pulmonary TB with cavitation, with differential diagnoses including lung abscess. Additional impressions included pneumonia and early thickening of the minor fissure, which may indicate right-pocketed and right pleural effusion.

Table 1. Results of laboratory examinations

	Day 1	Day 4	Unit
White blood cell	12,300	5,130	/µL
Hemoglobin	12.9	12.0	%
Platelet	412,000	443,000	/µL
Neutrophil-to-lymphocyte ratio	11.46	8.14	
Serum glutamic oxaloacetic transaminase	63	30	µ/L
Serum glutamic pyruvic transaminase	116	67	µ/L
Urea	65	46	mg/dL
Creatinine	1.4	1.3	mg/dL
Hepatitis B surface antigen	Negative	-	
Anti-hepatitis C virus rapid test	Negative	-	
Tuberculosis polymerase chain reaction	Negative	-	
Pleural fluid culture	No growth of specific pathogenic bacteria	-	
Adenosine deaminase test	-	89	U/L

Laboratory examinations revealed leukocytosis and elevated levels of serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), urea, creatinine, platelet count, and an increased neutrophil-to-lymphocyte ratio (NLR). The adenosine deaminase (ADA) level was 89 U/L.

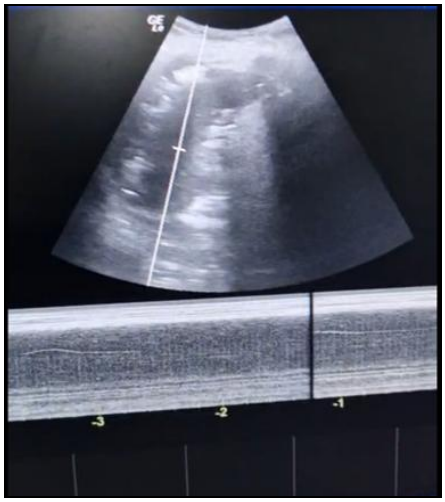

Figure 2. Ultrasonography of the right hemithorax

Ultrasonography (USG) of the right hemithorax showed a hypoechoic area at the fifth ICS along the right posterior axillary line (PAL), with an impression of right pleural effusion. On the first day of treatment, pleural fluid was evacuated via pleural puncture, yielding 750 cc of cloudy brown pus. A thoracic drainage was subsequently inserted. On the second day, 700 cc of cloudy brown pus was drained. Over the following days, the drainage continued, with a progressive decrease in volume, totaling less than 60 cc by the sixth day. The results of sterile fluid culture from the pleura during the initial pleural puncture showed no growth of specific pathogenic bacteria. GeneXpert (rapid molecular test/RMT) testing of the pleural fluid also did not detect MTB bacteria.

Figure 3. Evacuation of pleural fluid

Pleural fluid analysis revealed a white blood cell (WBC) count of 209,037/mm³, with 172,050/mm³ polymorphonuclear cells (PMN) and 36,987/mm³ mononuclear cells (MN), positive Rivalta, 2 mg/dL glucose, 2,386 IU/L lactate dehydrogenase (LDH), and 0.70 g/dL albumin. The patient was diagnosed with right pulmonary empyema caused by TB pleurisy, transaminitis, and stage II chronic kidney disease (CKD). The patient received folic acid therapy (2 tablets twice daily), curcuma (1 tablet twice daily), levofloxacin 750 mg once daily for seven days, ceftriaxone 1 g twice daily for three days, followed by meropenem 1 g three times daily for seven days, clindamycin 300 mg three times daily for three days, and fartison (2 vials daily) for nine days.

On the fifth day of treatment, a four-drug fixed-dose combination (FDC) anti-TB therapy was initiated at a dose of five tablets once daily. After nine days of inpatient treatment, the patient showed clinical improvement. Upon discharge, she was prescribed levofloxacin 750 mg orally once daily for five days, along with the continuation of anti-TB therapy. At a follow-up visit six days later in the outpatient department, the patient reported significant improvement and was no longer experiencing fever, coughing up phlegm, or shortness of breath.

DISCUSSION

The presence of pus in the pleural cavity, or purulent pleural effusion, is a clinical sign of pulmonary empyema. Empyema persisting for more than four weeks may be categorized as chronic empyema. The disease progresses through three stages: acute exudative phase (stage I), subacute fibrinopurulent phase (stage II), and chronic organizing phase (stage III). Stage I is characterized by elastic visceral pleura, while stage II is characterized by the formation of multiple loculations and the accumulation of murky, infected fluid with fibrin deposits, which are separated by septations dividing the effusion. In Stage III, granulation tissue replaces these structures. Although surgical intervention is rarely required to treat the acute phase of empyema, management typically includes chest tubes, antibiotics, and intrapleural fibrinolytics.[8]

Empyema shares many clinical characteristics with bacterial pneumonia, making differentiation challenging. Common symptoms include fever, cough, dyspnea, and pleuritic chest pain.[2] Previous case reports described a 59-year-old male presenting with pain and swelling on the left side of his back, accompanied by productive cough, fatigue, and poor oral intake for one week.[3,10] In comparison, overlapping symptoms were observed, including cough, fever, and dyspnea, which align with the general symptoms of empyema.[3,10] The

patient, in this case, a 57-year-old female, exhibited clinical symptoms suggestive of respiratory infections, including TB, pneumonia, or bronchitis. However, TB was strongly suspected as the underlying cause. This suspicion was supported not only by the clinical presentation but also by contextual risk factors, including the high prevalence of TB and the patient's potential exposure to individuals within her household and environment. These increased the likelihood of TB transmission and supported the diagnosis.

The patient in this case had an untreated cavity in the lower right tooth that had developed three weeks prior. A study suggested that independent risk factors for empyema include age under 60 years old and poor oral hygiene.[11] Another study has identified *Campylobacter rectus*, a predominant periodontal pathogen, as a potential cause of empyema, although it is rarely found in extraoral sites.[12] In some cases, *C. rectus* was isolated from pleural fluid samples drained from empyema patients. Although poor dental hygiene appears to be a significant risk factor for systemic infections, positive oral cultures are often unlikely in patients who have already received multiple courses of antibiotics. Therefore, the most likely clinical diagnosis was either a periodontal or a lung infection.[12]

Oral health, including the quantity of oral bacteria, is associated with bacterial infections, among the most common causes of empyema. These microorganisms can lead to infected parapneumonic effusions by infiltrating the visceral pleura, ultimately resulting in empyema. A previous study identified oral bacteria, such as *Fusobacterium spp.*, *Streptococcus spp.*, and *Staphylococcus aureus*, as primary pathogens in the development of empyema.[13] Consequently, poor dental hygiene may influence the development or outcome of empyema.[13]

Iwata, *et al.* (2023) suggested two ways to develop empyema.[14] The first is through descending mediastinitis, where a dental infection spreads directly into the thoracic cavity. The second pathway involves hematogenous dissemination, where bacteria travel to the thoracic cavity via the bloodstream. A study reported that among patients who survived, 15 out of 27 (55.6%) had their teeth extracted, while two of the nine patients (22.2%) who did not survive had teeth with a poor prognosis.[15] Compared to patients who survived, those who did not typically had fewer dental examinations.[14]

A previous case report described a 53-year-old male with recurrent periodontitis who underwent treatment for an unidentified space-occupying lesion in the lung.[15] Biopsy of the lesion revealed solid and necrotic areas with liquefaction, and metagenomic next-generation sequencing (mNGS) identified the presence of *Porphyromonas endodontalis* and *Parvimonas micra*.[15] Additionally, the patient reported a history of periodontal disease before the onset of respiratory symptoms that could pose a risk for aspiration.[15] He was diagnosed with an aspiration-related lung abscess and treated with appropriate antibacterial therapy before being discharged.[15] A study by Yuanjun, *et al.* (2024) highlighted the role of *Porphyromonas gingivalis*, a gram-negative oral anaerobe commonly found in the oral cavity and considered a key pathogen in the polybacterial consortia involved in periodontitis.[16] Although *P. gingivalis* is primarily associated with oral infections, it can occasionally be detected in extraoral conditions. In one case, the bacterium was identified in the bloodstream of a 43-year-old male with underlying diabetes who developed severe pneumonia through hematogenous dissemination.[16] The infection improved following specific antimicrobial treatment.[16] This case underscores how poor oral hygiene in patients with periodontal disease can facilitate the translocation of oral bacteria, leading to severe pneumonia.[16]

Periodontal disease, particularly periodontitis, has been identified as a rare cause of septic pulmonary embolism (SPE). One study reported the case of an 85-year-old Japanese male who was admitted to the hospital with persistent fever and malaise.[17] A computed tomography (CT) scan revealed multiple bilateral subpleural lung nodules. *Parvimonas micra*, a bacterium primarily associated with infections in the head and neck region, was isolated from blood cultures following treatment. The source of the SPE was traced to an infratemporal fossa abscess and apical periodontitis. Although *P. micra* is commonly found in the root canals of teeth with chronic apical periodontitis, it is rarely documented as the causative organism in SPE associated with periodontal disease.[17] The oral cavity is generally resistant to bacterial invasion, such as MTB. Oral manifestations of TB are typically the result of hematogenous spread from a primary infection site elsewhere in the body. Several previous cases have reported oral involvement in TB patients, such as the presence of nodules, ulceration, mucosal proliferation, granulomas, gingivitis, and periodontitis, which tend to be more severe in individuals suffering from TB.[18–20]

In this case, physical examination of the lungs revealed decreased vesicular breath sounds in the fifth to sixth ICS of the right hemithorax. This finding is consistent with a previous study, which reported that the physical examination of empyema patients revealed dullness to percussion, decreased breath sounds, and reduced tactile fremitus on the side of the effusion.[2]

Chest X-ray examination in this case suggested possible lung TB with cavitation, with differential diagnoses including lung abscess. Additional impressions included pneumonia and the thickening of the minor fissure, which may indicate a right-pocketed

and right pleural effusion. A previous study described empyema as presenting with a lenticular shape on chest X-rays and forming an obtuse angle with the surrounding wall.[21] Thinner walls and a smoother lumen may serve as early indicators of empyema. Empyema is also known to exert compressive effects on the lung parenchyma. A distinctive radiologic sign, the split pleural sign, may be observed, reflecting the accumulation of empyema-induced fluid in the pleural cavity, thickening and separating the two pleural layers.[22]

Leukocytosis was identified in the patient's laboratory examination. The WBC count was elevated at 12.08×109/L (normal range: 4.0–10.0×109/L), with neutrophils accounting for 11.6×109/L. C-reactive protein (CRP) levels rose significantly to 123.79 mg/L, while procalcitonin levels slightly increased to 0.26 ng/ml. These findings align with previous studies, which suggested that leukocytosis indicates an ongoing infection process.[23,24] Similarly, in the case report by Brims, *et al*. (2010), patients with empyema commonly present with leukocytosis and elevated inflammatory markers, including CRP.[25]

In this case, the ADA test yielded a result of 89 U/L. Adenosine deaminase is a purine-degrading enzyme produced in significant amounts in TB pleural effusions and is commonly assessed using a rapid and cost-effective method. Even in patients with low CD4 counts, ADA levels above 40 U/L in pleural fluid are frequently employed as a diagnostic threshold for pleural TB and appear to be unaffected by human immunodeficiency virus (HIV) status. The patient's ADA level, being well above this threshold, strengthened the clinical suspicion of a TB infection. Moreover, a pleural LDH-to-ADA ratio of less than 16.2 was found to be a reliable indicator of TB effusions, with a sensitivity and specificity of 93.6% and 93.1%, respectively, in a retrospective study involving 72 TB effusions and 47 parapneumonic effusions in an area with a high TB endemicity.[26] It has also been suggested that tests targeting the ADA isoenzyme elevated in TB effusions may offer greater diagnostic specificity for pleural TB.[26]

Although TB empyema can also result from caseous material from parenchymal foci, bronchopleural fistula by direct extension from adjacent nodes, hematogenous dissemination, or surgical procedures such as pneumonectomy, it remains a rare condition. It is typically characterized by coarse, purulent fluid containing numerous bacterial microbes. In patients with TB pleurisy, memory T cells in the pleural fluid express some crucial homing factors, including CD11a, CCR5, and RANTES. They also generate elevated levels of pro-inflammatory cytokines, such as interferon-γ (IFN-γ), compared to those found in peripheral blood, significantly impacting the immune response. However, the pleural cavity of TB patients contains fewer Th2 cells producing interleukin-4 (IL-4) than the peripheral blood. This increased capillary permeability induced by pro-inflammatory cytokines allows fluid to enter the pleural cavity, while lymphocytic pleurisy and granuloma development interfere with the normal reabsorption of pleural fluid.[26] Tuberculosis empyema typically results from primary TB effusion and is associated with the thickening and calcification of the pleural skin and radiologically evident thickening of the rib cage. This process may lead to pleural fibrosis and fusion, resulting in fibrothorax, a chest wall malformation.[26]

Empyema frequently necessitates an interventional drainage approach, typically involving the insertion of a chest tube under ultrasound guidance. According to previous studies, the primary objective of this procedure is to evacuate the pleural cavity, thereby allowing the compressed lung to expand fully.[10,27] Thoracocentesis is a basic procedure used not only to obtain fluid samples to differentiate between transudates and exudates but also to remove fluid in patients with large effusions to relieve symptoms. A lateral decubitus chest radiograph revealing pleural fluid of unclear etiology in the pleural cavity with a thickness greater than 10 mm is the most common indication for diagnostic thoracentesis.

In this case, the patient underwent pleural fluid evacuation (pleural puncture) at ICS 5 PAL dextra on the first day of treatment, yielding 750 cc of cloudy brown pus. A thoracic drainage was subsequently inserted, yielding an additional 700 cc of cloudy brown fluid. Thoracentesis may be postponed if an identified underlying condition is causing the effusion. For instance, in a patient with known congestive heart failure presenting with bilaterally symmetric pleural effusions but without fever or chest pain, treatment with diuretics should be initiated before considering thoracentesis. Ultrasound guidance is recommended to obtain fluid samples from localized effusions and minimize the risk of potential complications.[28]

In addition to thoracic drainage, an intercostal catheter can treat empyema. Imaging-guided chest tube placement is preferred over blind bedside insertion, as it allows for optimal positioning of the drainage tube within the collection of infected material, particularly in patients with multiseptated pleural collections. Although large-diameter chest tubes are frequently recommended for pus drainage, several observational studies indicate that small-diameter catheters may also be employed.[25,29,30] Several studies suggest that multiple catheters may be necessary for sufficient pus evacuation. However, small-diameter drains are more

prone to blockages, necessitating frequent flushing.[25,29,30]

Liu, *et al*. (2024) reported numerous adhesions, purulent discharge, and necrotic material during thoracoscopy.[23] In their study, pleural fluid analysis revealed an exudative pleural effusion, indicated by a nucleated cell count of 6.2×109/L, glucose level of 0.74 mmol/L, total protein concentration of 36.4 g/L, albumin concentration of 20.2 g/L, LDH level of 4,032 U/L, and ADA level of 16.1 U/L.[23] Although Hankins, *et al*. (2023) noted that lymphocyte-dominated pleural fluid is traditionally considered one of the diagnostic criteria for pleural TB, their findings also indicated that patients with neutrophil-predominant effusions exhibited a more severe inflammatory response and had TB more frequently confirmed in pleural fluid samples.[31] They further reported that the pleural effusions were exudative, with high protein levels, neutrophil predominance, and high LDH levels.[31] These findings are in line with this case, in which pleural fluid analysis revealed exudative characteristics: positive Rivalta test, WBC count of 209,037/mm³, PMN count of 172,050 /mm³, MN count of 36,987 /mm³, glucose level of 2 mg/dL, LDH level of 2386 IU/L, and albumin level of 0.70 g/dL.

Effective treatment of empyema depends on the early and complete draining of infected fluid, in conjunction with antibiotic therapy and management of the underlying disease. A large drainage catheter (22F-34F) is typically inserted into the pleural cavity to treat empyema. However, empyema has been effectively treated with tiny drainage catheters. In a study involving 103 empyema patients, 80 were successfully treated using the small drainage catheters (less than 14F) inserted under CT or ultrasound guidance.[2] These findings are similar to a previous study that used large catheters to treat empyema.[2]

It is generally agreed that thoracentesis or additional drainage is necessary for treating empyema, while antibiotic therapy is typically sufficient for treating pneumonia with parapneumonic effusion. Studies indicate that laboratory investigation of pleural fluid may be helpful in more complex cases where the necessity for drainage cannot be ascertained.[31] Established parameters suggesting the necessity for drainage include a pH level of less than 7.2, a glucose level of less than 60 mg/dL, and an LDH level of more than 1,000 units/L.[31]

Treatment for empyema included antibiotic administration. Antibiotic regimens for patients with community-acquired empyema should focus on common oropharyngeal infections, such as aerobic and anaerobic species of *Streptococcus* and *Staphylococcus*. Suitable antibiotic options include third-generation cephalosporins, metronidazole, or a combination of beta-lactam/beta-lactamase inhibitors.

Antimicrobial therapy for hospital-acquired empyema patients should target protection against methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas*, in addition to typical organisms and anaerobes. Recommended regimens for such cases include vancomycin combined with metronidazole and antipseudomonal cephalosporins. Anaerobic and antipseudomonal activities are provided by vancomycin combined with piperacillin/tazobactam, a broad-spectrum beta-lactam/beta-lactamase inhibitor. The use of aminoglycosides is generally discouraged because of poor pleural penetration and limited efficacy in treating empyema.[2]

The patient in this case received antibiotic therapy consisting of levofloxacin, ceftriaxone, and meropenem. Levofloxacin, which is included in the fluoroquinolone antibiotic group, and a combination of ceftriaxone, which is included in the third-generation cephalosporin group, were administered to achieve broad-spectrum bacterial eradication. Although the culture examination did not yield the growth of specific bacteria, it is strongly suspected that common causative bacteria include *Streptococcus* and *Staphylococcus* species. These bacteria are often associated with independent risk factors, such as poor dental and oral hygiene, which in this case was the presence of untreated cavities. Poor oral health may increase the risk of bacterial spread through the bloodstream or lymphatic circulation, leading to clinical lung infections complicated by pulmonary empyema. Furthermore, meropenem was introduced on the third day of treatment. Based on clinical findings, the pleural fluid remained thick despite a significant reduction in volume compared to the first and second day. At that time, the patient continued to experience symptoms such as shortness of breath and fever, which began to improve significantly by the fifth day of treatment. Therefore, the antibiotics administered in this case align with current recommendations for treating empyema.[2]

In this case, the administration of anti-TB therapy was based on clinical considerations derived from the patient's history, supporting examination results, and characteristic findings suggestive of TB infection. External risk factors were identified, including potential exposure to a high-risk environment and contact with at-risk populations. Notably, several close relatives of the patient had a history of prolonged coughing up phlegm, and close physical interactions with each other were still ongoing. Although some of the aforementioned factors were difficult to eliminate, the decision to initiate anti-TB therapy remained a subject for continued clinical discussion, particularly considering the absence of clear, strong evidence that

the patient was infected with TB. However, based on expert clinical judgement and medical experience, the administration of anti-TB therapy was deemed appropriate because the possibility of TB infection could not be ruled out and should not be underestimated. Given the potential risk of disease progression and further complications in the absence of treatment, anti-TB drugs were initiated to support recovery and minimize the risk of deterioration in the patient's condition.

The penetration and activity of anti-TB therapy in the pleural cavity, as well as its high coexistence and risk of progression to parenchymal disease, remain poorly understood. As a result, the anti-TB regimen recommended for TB empyema follows the same protocol as that indicated for pulmonary TB. The anti-TB drugs administered to TB patients include isoniazid (H or INH), rifampicin (R), pyrazinamide (Z), streptomycin (S), and ethambutol (E).[32,33] If there are concerns regarding possible noncompliance, the typical six-month regimen is administered under directly monitored therapy.[26] In one case reporting a 26-year-old patient with TB empyema, anti-TB therapy was initiated following the World Health Organization (WHO) guidelines.[34] By the end of the intensive phase, the patient experienced a 6% increase in body weight, and by the end of the continuation phase, a total weight gain of approximately 15% was observed.[34] Chest radiographs indicated improvement, chest pain subsided, and no recurrence was noted.[34] The patient was considered clinically recovered.[34]

CONCLUSION

Pulmonary empyema is a serious complication that requires prompt and accurate treatment. Identifying risk factors and possible causes could help diagnose a condition that remains clinically ambiguous because clinical symptoms can resemble those of other diseases, such as pneumonia. In addition, it can help create an appropriate therapeutic strategy, enabling the delivery of optimal treatment and reducing the risk of complications associated with pulmonary empyema. Clinical evaluation should be guided by patient history and presenting symptoms in cases of empyema with suspected TB infection. This consideration is especially relevant in Indonesia, where the incidence of TB remains high. Patients diagnosed with pulmonary empyema may continue to experience recurrent symptoms up to one year after the initial diagnosis. In some cases, invasive interventions such as surgery may be necessary. Therefore, routine examination is recommended.

Consent

Written informed consent was obtained from the patient.

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Conflict of Interest

The authors declared there is no conflict of interest.

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Authors' Contributions

Data collection, analysis, and discussion: IGPACP, RS, WWSP. All authors contributed to and approved the final version of the manuscript.

REFERENCES

1. Imburgio S, Tavakolian K, Mararenko A, *et al*. Empyema Versus Lung Abscess: A Case Report. *J Investig Med High Impact Case Reports* 2022; 10: 23247096221139268. [PubMed]
2. Iguina MM, Sharma S, Danckers M. Thoracic Empyema. Treasure Island (FL), 2025. [PubMed]
3. Karim SHA, Zain WZW, Hashim MNM, *et al*. Empyema Thoracis Presented as Giant Back Abscess. *Radiol Case Reports* 2021; 16: 1061–1064. [PubMed]
4. Wu YH, Wang JL, Wang MS. Factors Associated with the Presence of Tuberculous Empyema in Children with Pleural Tuberculosis. *Front Pediatr* 2021; 9: 751386. [PubMed]
5. Wen P, Wei M, Han C, *et al*. Risk Factors for Tuberculous Empyema in Pleural Tuberculosis Patients. *Sci Rep* 2019; 9: 19569. [PubMed]
6. Silva S, Waduge L. Empyema Necessitans due to Tuberculosis in a Patient with Poorly Controlled Diabetes Mellitus Type 2: A Case Report. *Cureus* 2024; 16: e73919. [PubMed]
7. Kwon YS. Pleural Infection and Empyema. *Tuberc Respir Dis (Seoul)* 2014; 76: 160–162. [PubMed]
8. Biswas A, Jantz MA, Penley AM, *et al*. Management of Chronic Empyema with Unexpandable Lung in Poor Surgical Risk Patients Using an Empyema Tube. *Lung India* 2016; 33: 267–271. [PubMed]
9. Bandaru S, Manthri S, Sundareshan V, *et al*. Empyema Necessitans in the Setting of Methicillin-Susceptible *Staphylococcus aureus* Causing Pneumonia and Bacteremia. *Case Rep Infect Dis* 2018; 2018: 4906547. [PubMed]
10. Semenkovich TR, Olsen MA, Puri V, *et al*. Current State of Empyema Management. *Ann Thorac Surg* 2018; 105: 1589–1596. [PubMed]
11. Petrusevska-Marinkovic S, Kondova-Topuzovska I, Milenkovic Z, *et al*. Clinical, Laboratory and Radiographic Features of Patients with Pneumonia and Parapneumonic Effusions. *Open Access Maced J Med Sci* 2016; 4: 428–434. [PubMed]

12. Figueiredo L, Ferrão J, Ferreira C, *et al*. A Rare Case of Pleural Empyema Caused by *Campylobacter rectus*. *Cureus* 2022; 14: e23205. [PubMed]
13. Nomoto A, Shimizu A, Ohno T, *et al*. Poor Oral Health and Anorexia in Older Rehabilitation Patients. *Gerodontology* 2022; 39: 59–66. [PubMed]
14. Iwata E, Nishiuma T, Hori S, *et al*. Relationship between Oral Health and Prognosis in Patients with Empyema: Single Center Retrospective Study with Propensity Score Matching Analysis. *PLoS One* 2023; 18: e0282191. [PubMed]
15. Li Y, Shi P, Zhu R. A Pulmonary Abscess Caused by *Porphyromonas endodontalis* Infection: A Case Report and Literature Review. *Diagn Microbiol Infect Dis* 2024; 108: 116126. [PubMed]
16. Yuanjun Z, Yan C, Qingyan Z, *et al*. *Porphyromonas gingivalis*-Induced Hematogenous Disseminated Severe Pneumonia: A Case Report. *J Int Med Res* 2024; 52: 03000605231213760. [Journal]
17. Watanabe T, Yokoe M, Noguchi Y. Septic Pulmonary Embolism associated with Periodontal Disease: A Case Report and Literature Review. *BMC Infect Dis* 2019; 19: 74. [PubMed]
18. Parmasari WD. Hubungan antara Tuberkulosis Paru dengan Terjadinya Kelainan pada Jaringan Lunak Rongga Mulut berdasarkan Lama Pengobatan. *Sinnun Maxillofac J* 2024; 6: 56–61. [Journal]
19. Amperawati M, Said F, Suryana E. Gambaran Kondisi Klinis Rongga Mulut pada Penderita TB Paru di Kecamatan Cerbon Kabupaten Barito Kuala Provinsi Kalimantan Selatan. *J Skala Kesehat*; 7. Epub ahead of print 25 October 2018. [Journal]
20. Ernawati DS, Parmadiati AE, Soebadi B, *et al*. Prevalence of Oral Tuberculosis Ulcer in DOTS Polyclinic of Dr. Soetomo General Hospital Surabaya. *Biochem Cell Arch* 2020; 20: 2845–2848. [Journal]
21. Hassan M, Asciak R, Rizk R, *et al*. Lung Abscess or Empyema? Taking a Closer Look. *Thorax* 2018; 73: 887–889. [PubMed]
22. Kumaresh A, Kumar M, Dev B, *et al*. Back to Basics - 'Must Know' Classical Signs in Thoracic Radiology. *J Clin Imaging Sci* 2015; 5: 43. [PubMed]
23. Liu M, Liu J, Wu J, *et al*. A Case Report of Empyema Caused by *Enterococcus gallinarum*. *BMC Infect Dis* 2024; 24: 769. [PubMed]
24. Viner E, Berger J, Bengualid V. Etiologies of Extreme Leukocytosis. *Cureus* 2023; 15: e38062. [PubMed]
25. Brims FJH, Lansley SM, Waterer GW, *et al*. Empyema Thoracis: New Insights into an Old Disease. *Eur Respir Rev* 2010; 19: 220–228. [PubMed]
26. McNally E, Ross C, Gleeson LE. The Tuberculous Pleural Effusion. *Breathe* 2023; 19: 230143. [PubMed]
27. Heimes J, Copeland H, Lulla A, *et al*. The Use of Thrombolytics in the Management of Complex Pleural Fluid Collections. *J Thorac Dis* 2017; 9: 1310–1316. [PubMed]
28. Yu H. Management of Pleural Effusion, Empyema, and Lung Abscess. *Semin Intervent Radiol* 2011; 28: 75–86. [PubMed]
29. Nakada T, Ohtsuka T. Thoracic Drain Management Using a Digital System. *Journal of Thoracic Disease* 2023; 15: 219–222. [PubMed]
30. Anand K, Kaufman CS, Quencer KB. Thoracentesis, Chest Tubes, and Tunneled Chest Drains. *Semin Intervent Radiol* 2022; 39: 348–354. [PubMed]
31. Hankins E, Khvolis D, Spigos JT, *et al*. Acute Presentation of Tuberculosis Empyema in a Healthy Adolescent. *Am J Case Rep* 2023; 24: e939419. [PubMed]
32. Ahmed O, Zangan S. Emergent Management of Empyema. *Semin Intervent Radiol* 2012; 29: 226–230. [PubMed]
33. Suprayitno E, Hannan M, Afifah U. Konsep Diri Penderita TB Paru dengan Karies Gigi di Wilayah Kerja UPT Puskesmas Ambunten Kecamatan Ambunten Kabupaten Sumenep. *STOMATOGNATIC - J Kedokt Gigi* 2020; 17: 41. [Journal]
34. Tufail M. A Case Report of Tuberculous Empyema: A Tricky Disease. *Int J Mycobacteriology* 2019; 8: 302–304. [PubMed]