e-ISSN (online): 2986-853X

Sriwijaya Journal of Radiology and Imaging Research (SJRIR)

Journal website: https:// phlox.or.id/index.php/sjrir

Loculated Right-Sided Hydropneumothorax Mimicking Giant Pulmonary Bullae in a Post-Tuberculosis Patient: A Multimodality Imaging Diagnostic Challenge

Sidik Teghar Sanyadi[1*], Bernard Sujijanto Suwito[2], Gandhi Estrada Atmanto[3]

[1]General Practitioner, Emergency Department, Aminah Islamic Hospital, Blitar, Indonesia

[2]Radiologist, Aminah Islamic Hospital, Blitar, Indonesia

[3]Pulmonologist Specialist at Aminah Islamic Hospital, Blitar, Indonesia

ARTICLE INFO

Keywords:
Computed tomography
Hydropneumothorax
Multimodality imaging
Post-tuberculosis
Thoracic ultrasound

***Corresponding author:**
Sidik Teghar Sanyadi

E-mail address:
sanyadisidik@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.59345/sjrir/v4i1.245

A B S T R A C T

Introduction: Post-tuberculosis lung disease remains a significant public health challenge affecting millions of individuals globally, representing a substantial health burden in tuberculosis-endemic regions and in developed countries with immigration from endemic areas. Loculated hydropneumothorax as a late complication of successfully treated pulmonary tuberculosis is a rare but diagnostically challenging entity, particularly when imaging findings suggest alternative pathology such as giant pulmonary bullae. This case illustrates the complexity of post-tuberculosis complications and the essential role of multimodality imaging. **Case presentation:** A 63-year-old retired woman presented to the emergency department with three days of progressive dyspnea accompanied by a productive cough with yellowish-white sputum. Physical examination revealed severe tachypnea (41 breaths per minute), clinically significant hypoxemia (SpO_2 88 percent on room air), and diminished breath sounds over the right hemithorax with crackles in the right upper lobe. Chest radiography demonstrated a large thin-walled cavity (18 by 9.5 by 14 centimeters) with a horizontal air-fluid level in the right hemithorax, mediastinal leftward shift, and right costophrenic sinus obliteration. Thoracic point-of-care ultrasound revealed predominant gas throughout the right hemithorax with minimal pleural fluid in dependent zones and absence of identifiable lung tissue above the hemidiaphragm. Contrast-enhanced computed tomography definitively identified a loculated right-sided hydropneumothorax with a thin-walled pleural compartment, an air-fluid level, compressive atelectasis of the right lower and middle lobes, and post-tuberculosis fibrotic sequelae. This case illustrates the critical importance of multimodality imaging integration in differentiating loculated hydropneumothorax from mimicking entities, particularly giant pulmonary bullae. Individual imaging modalities—radiography, ultrasound, and computed tomography—each contributed essential diagnostic information, demonstrating that none is sufficient in isolation. **Conclusion:** Loculated hydropneumothorax must be considered in the differential diagnosis of large cavitary lesions in post-tuberculosis patients. A multimodality imaging approach is essential for achieving diagnostic certainty and preventing unnecessary surgical intervention.

1. Introduction

Post-tuberculosis lung disease has become increasingly recognized as a significant global health burden, affecting millions of individuals worldwide in both high-tuberculosis-burden endemic regions and in developed countries with immigration from endemic areas. The World Health Organization estimates approximately 15 million individuals have post-tuberculosis lung disease globally, yet this remains an under-recognized clinical entity in many healthcare

settings. Following documented microbiological cure of active pulmonary tuberculosis, a substantial proportion of patients—estimates range from 15 to 50 percent depending on severity of initial disease—develop chronic pulmonary sequelae that persist and sometimes worsen over time. These complications include pulmonary fibrosis and secondary pulmonary hypertension, bronchiectasis (bronchial dilation and progressive destruction), emphysematous changes with formation of pulmonary bullae, and serious pleural abnormalities. The pathophysiological basis involves irreversible destruction of normal lung architecture from caseous necrosis, vascular injury from granulomatous inflammation, and fibrotic healing that causes permanent distortion of normal anatomy. Even years after a successful microbiological cure is documented, the chronic structural changes persist indefinitely.[1,2]

Pleural involvement represents a particularly serious manifestation of post-tuberculosis disease, and various pleural complications have been documented as late sequelae occurring months to years after microbiological cure is achieved.[3,4] Hydropneumothorax, characterized by the simultaneous presence of both air and fluid within the pleural space, represents an uncommon but potentially life-threatening complication in post-tuberculosis patients. This condition arises through several distinct pathophysiological mechanisms: development of bronchopleural fistulas from rupture of tuberculous cavities with erosion into bronchial structures allowing air entry, spontaneous rupture of tuberculous pleural involvement leading directly to atmospheric air entering the pleural space, or drainage of infected material from subpleural caseous lesions into the pleural space combined with air entry. The pleural space becomes divided into compartments or becomes loculated when organization of pleural inflammation results in adhesions and fibrotic bands, which compartmentalize the air and fluid into confined spaces that may progressively grow and expand over time.[5,6]

Diagnostic differentiation between loculated hydropneumothorax and giant pulmonary bullae presents a substantial clinical challenge because both entities present with large, thin-walled, air-containing structures visible on imaging studies. Bullae are large (typically exceeding 20 millimeters) air-filled spaces that develop within lung parenchyma from emphysematous destruction of alveolar walls and progressive pathological dilation of distal air spaces.[7,8] Giant bullae, particularly when large and causing significant mass effect, may present clinically with dyspnea and hypoxemia similar to that caused by pleural effusions or pneumothoraces.[9,10] Misidentification carries significant clinical consequences: giant bullae are typically managed either conservatively with close monitoring or surgically if causing substantial respiratory symptoms or pulmonary function deterioration, whereas loculated hydropneumothorax typically requires interventional drainage through tube thoracostomy or other drainage procedures to relieve respiratory compromise and prevent secondary infection.[11,12] The presence or suspicion of bronchopleural fistula adds further complexity, requiring bronchoscopic evaluation and possibly thoracic surgical intervention.

Multimodality imaging—systematically integrating chest radiography, thoracic point-of-care ultrasound, and high-resolution computed tomography—provides complementary diagnostic information and significantly enhances diagnostic accuracy in pleural pathology. Each modality offers distinct advantages: chest radiography provides a rapid overview and initial detection within minutes, thoracic ultrasound provides real-time bedside assessment with portability and capability for repeated examinations for monitoring, and computed tomography offers superior anatomical detail, precise pleural characterization, and mediastinal assessment. Despite substantial advances in imaging technology and radiological expertise, achieving diagnostic certainty in complex post-tuberculosis presentations remains challenging, requiring careful synthesis of clinical findings, imaging data, and clinical reasoning.[9,13]

This case report presents a detailed diagnostic odyssey of loculated right-sided hydropneumothorax in a 63-year-old post-tuberculosis patient, systematically demonstrating the critical complementary role of multimodality imaging in

achieving diagnostic certainty and differentiating this entity from mimicking pathologies such as giant pulmonary bullae. We illustrate the clinical value of systematic and comprehensive imaging analysis in this challenging presentation, document the diagnostic limitations of individual imaging modalities when used in isolation, and demonstrate the diagnostic certainty and clinical confidence achieved through an integrated multimodality approach. This case contributes important insights to the medical literature on post-tuberculosis complications and emphasizes best practices in diagnostic imaging of complex pleural pathology.

2. Case Presentation

This case report was conducted in accordance with the principles of the Declaration of Helsinki. Informed written consent was obtained from the patient for publication of clinical and imaging findings. The patient was fully informed about the research use of her medical information and consented to the case report with understanding that her privacy would be protected through appropriate anonymization and de-identification.

A 63-year-old retired woman with a significant medical history of pulmonary tuberculosis, successfully treated, presented to the emergency department with acute-onset dyspnea and productive cough. The patient had completed standard anti-tuberculosis therapy two years prior, receiving the Category I regimen consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol for 6 months total duration. Microbiological documentation of cure was confirmed with sputum smear conversion and negative sputum cultures obtained on repeated testing at completion of therapy. The demographic and clinical characteristics are summarized in Table 1.

Table 1. Patient demographics and clinical characteristics.

Variable	Value
Age (years)	63
Gender	Female
Occupation	Retired teacher
Prior TB diagnosis and treatment	Category I, 4-drug fixed-dose combination, 6 months total
Time since TB cure	2 years prior with documented sputum conversion
TB treatment outcome	Cured status with negative follow-up sputum cultures
Documented comorbidities	None reported or documented
Current home medications	None on regular medication regimen
Duration of current illness	3 days of progressive symptoms
Primary presenting complaint	Progressive dyspnea and productive cough

The patient reported that her symptoms began three days prior to hospital presentation. The dyspnea was progressive in nature, worsening daily over the three-day period before seeking medical care. The cough was productive, with sputum described as yellowish-white in color, suggesting a possible infectious component or inflammatory response. The patient specifically denied hemoptysis (absence of blood in sputum), pleuritic chest pain exacerbated by breathing, fever, and any recalled recent trauma to the chest wall or penetrating injury. She reported that her dyspnea was particularly notable on exertion, with routine activities of daily living becoming limited by shortness of breath, requiring her to stop and rest frequently. She could only walk short distances before needing to pause. She denied recent travel to areas with high tuberculosis prevalence and denied household sick contacts or family members with recent respiratory illness. Comprehensive family history revealed no documented tuberculosis among first-degree or second-degree relatives and no genetic pulmonary diseases such as alpha-1 antitrypsin deficiency or inherited emphysema syndromes.

At presentation to the emergency department, a comprehensive vital sign assessment with clinical significance interpretation is detailed in Table 2. The

patient displayed objective signs of moderate to severe respiratory compromise requiring immediate clinical evaluation and therapeutic intervention. The respiratory rate of 41 breaths per minute was substantially elevated above the normal range of 12 to 20 breaths per minute, indicating severe tachypnea with obvious evidence of increased work of breathing evident on observation. Oxygen saturation measured at 88 percent on room air was clinically significantly below the normal value of 95 to 100 percent, representing moderate hypoxemia that was clearly contributing to tissue hypoxia and risk of organ dysfunction. The blood pressure of 128/67 mmHg showed borderline elevation in systolic pressure, likely reflecting a physiological stress response to acute respiratory compromise.

Table 2. Vital signs and clinical assessment.

Parameter	Value	Clinical significance and assessment
Respiratory rate	41 breaths/min	CRITICAL - Severe tachypnea, substantially elevated
Oxygen saturation	88% room air	CRITICAL - Moderate hypoxemia, tissue hypoxia risk
Blood pressure	128/67 mmHg	BORDERLINE - Elevated systolic, stress response
Heart rate	87 beats/min	NORMAL - Regular sinus rhythm, appropriate response
Body temperature	36.8°C (98.2°F)	NORMAL - Afebrile, no fever present
Glasgow Coma Scale	Alert and fully oriented	NORMAL - Full consciousness and orientation

Physical examination revealed multiple findings consistent with significant right-sided pulmonary or pleural pathology affecting respiratory mechanics. Observation of the patient at rest showed nasal flaring (dilation of the nares or nostrils), a compensatory sign of increased work of breathing. Visible intercostal and subcostal chest wall retractions were prominent, indicating substantially increased work of breathing and active recruitment of accessory respiratory muscles including the scalene and sternocleidomastoid muscles. Auscultation of the thorax demonstrated completely absent breath sounds over the right middle and lower lung zones, suggesting either absence of air entry to those regions or presence of fluid-filled spaces. Fine crackles (rales), described as discontinuous, popping sounds, were distinctly audible in the right upper lobe region, suggesting the presence of secretions, fluid, or partial consolidation. Percussion over the right hemithorax demonstrated dullness at the base, consistent with fluid or solid material replacing air-filled lung. Cardiovascular examination revealed a regular heart rate without murmurs or additional sounds, and no evidence of apical displacement, suggesting pericardial involvement. Abdominal examination was unremarkable without organomegaly or peritoneal signs. Lower extremity examination showed no edema or clinical signs of venous thrombosis.

Posteroanterior and lateral chest radiographs were obtained in the emergency department as the initial diagnostic imaging study in the clinical workup. The radiographic findings are presented in Table 3. The radiographs demonstrated a large thin-walled cavity in the right hemithorax measuring approximately 18 by 9.5 by 14 centimeters with a clear horizontal air-fluid interface. The mediastinum was shifted significantly to the left, indicating substantial mass effect from the right-sided pathology. The right costophrenic sinus was completely obscured, indicating fluid or consolidation filling that normal space. Linear enhancement of the right minor fissure was noted consistent with fibrotic changes from prior tuberculosis. The cardiac silhouette remained normal in size, excluding primary cardiac pathology as contributing to respiratory compromise.

Table 3. Chest radiography findings.

Radiographic finding	Detailed observation	Anatomical location and extent
Cardiac silhouette	Normal size and configuration	Central mediastinum, no enlargement
Mediastinal position	Leftward displacement	Marked displacement indicating mass effect
Primary pathology	Large thin-walled cavity	18 × 9.5 × 14 centimeters right hemithorax
Fluid-air interface	Clear horizontal interface	Well-defined air-fluid level visualized
Right costophrenic sinus	Completely obliterated	Complete loss of normal visualization
Right minor fissure	Thickened linear density	Linear enhancement with fibrotic appearance
Additional radiographic findings	Suspected paratracheal adenopathy	Right side, mild in extent

Thoracic point-of-care ultrasound was performed at the patient's bedside using a multi-frequency linear transducer operating in the 5 to 12 megahertz frequency range. The transducer was systematically positioned at six standard anatomical landmarks on the right hemithorax as recommended by international guidelines for comprehensive thoracic ultrasound assessment. The systematic ultrasound findings are detailed in Table 4. Predominant echogenic shadowing from air accumulation was consistently observed across all scanning positions, with minimal anechoic (fluid-filled) collections identified in posterior dependent zones. Critically, no identifiable consolidated or atelectatic lung tissue could be visualized above the right hemidiaphragm on any scanning position, strongly suggesting mechanical compression of lung parenchyma by external pressure from a space-occupying process, consistent with pleural rather than primary pulmonary pathology.

Table 4. Thoracic ultrasound findings by anatomical line.

Anatomical position	Gas	Pleural fluid	Lung tissue	Notes and artifacts
Scapular line (posterior)	Dominant	Minimal	Absent above the diaphragm	Liver echoes visible below
Posterior midclavicular	Dominant pattern	Trace fluid	Absent	Shadowing artifact prominent
Posterior axillary	Dominant echogenicity	Small collection	Absent	Hyperechoic artifacts present
Mid-axillary	Minimal gas	Minimal effusion	No solid tissue	Transition zone between air and fluid
Anterior axillary line	Gas predominantly	None visualized	Absent	Anterior pneumothorax pattern
Anterior midclavicular	Gas only	No fluid	Absent	Characteristic barcode sign

Contrast-enhanced computed tomography of the thorax was performed with 1-millimeter slice thickness and multiplanar reformatting to provide a comprehensive anatomical assessment. The computed tomography imaging revealed a large loculated air-filled cavity in the right hemithorax with a thin but well-defined pleural wall demonstrating subtle contrast enhancement indicative of pleural inflammation and reactive enhancement from inflammatory response. Fluid accumulated within this cavity in the dependent portions, with a clear horizontal air-fluid interface separating the gas from the liquid phase. The mediastinum and heart were displaced substantially to the left, indicating

significant mass effect from the large pleural collection. The right lower lobe and middle lobe demonstrated compressive atelectasis with characteristic ground-glass opacification pattern consistent with collapsed but perfused lung tissue. The right upper lobe appeared compressed but retained some degree of aeration, suggesting it was less severely compressed. Post-tuberculosis fibrotic changes were evident throughout the right lung, including bronchiectasis (bronchial dilation and destruction) and bronchial wall thickening characteristic of sequelae from previous tuberculosis. The left lung parenchyma appeared entirely normal in size, density, and appearance without infiltrates. No pulmonary embolism was identified on the arterial phase imaging. The pleural cavity showed no obvious bronchopleural fistula tract, although small fistulous connections can be difficult to visualize even with high-resolution computed tomography.

Patient perspective

The patient reported significant distress from progressive dyspnea and was deeply concerned about the findings on initial chest radiograph examination. She was relieved to learn that the condition, while serious, could be identified through systematic imaging assessment and did not require immediate surgical intervention. The patient appreciated the thorough diagnostic approach and the detailed explanation of her clinical condition and imaging findings. She understood the importance of follow-up imaging and close clinical monitoring for signs of deterioration. The patient expressed gratitude for the multidisciplinary team approach and felt confident in the diagnostic and management plan.

3. Discussion

Post-tuberculosis lung disease encompasses a broad spectrum of chronic pulmonary abnormalities that develop or persist following documented microbiological cure of active tuberculosis, even years after successful completion of anti-tuberculosis chemotherapy. The underlying pathophysiology involves irreversible destruction of normal lung parenchyma, extensive pleural fibrosis, bronchial damage and remodeling, vascular changes and pulmonary hypertension, all caused by inflammation and tissue necrosis during the active infection phase. When Mycobacterium tuberculosis infects lung tissue, it induces a robust granulomatous inflammatory response including epithelioid histiocytes, multinucleated giant cells, and lymphocytes surrounding the bacilli. This process results in caseous necrosis of affected lung tissue, cavitation with necrotic debris, and subsequent organization and fibrotic healing that causes permanent scarring and architectural distortion. Even after microbiological cure is achieved through appropriate chemotherapy, this extensive fibrotic scarring persists permanently, leading to chronic functional impairment and predisposition to secondary complications. Pleural complications represent a particularly serious and potentially life-threatening manifestation of post-tuberculosis lung disease, arising when rupture of subpleural caseous lesions or development of bronchopleural fistulas allow abnormal communication between the pleural space and either the atmosphere (causing pneumothorax) or infected cavities (causing hydrothorax with infected fluid). Loculation of hydropneumothorax occurs when organization of pleural inflammation results in adhesions and fibrotic bands that compartmentalize and contain the air and fluid to limited spaces, creating confined collections that can progressively grow and cause compression of adjacent vital structures.[6,7,17]

A comparative analysis of imaging modalities in this case is presented in Table 5. Chest radiography remains the essential first-line imaging study in respiratory emergencies, providing rapid assessment of the thoracic cavity and detection of gross abnormalities within minutes of examination. In this case, radiography successfully identified the large cavity with an air-fluid level and mediastinal shift, establishing the presence of significant pleural pathology requiring further investigation. However, radiography alone could not definitively characterize whether this represented a pneumothorax, hydropneumothorax, bullae, or other cavity-containing lesion. The inherent limitations of

radiography stem from the superimposition of anatomical structures, the inability to assess tissue planes and pleural boundaries in cross-section, and the two-dimensional representation of three-dimensional anatomy, which limit diagnostic specificity.[9,13] Thoracic ultrasound provided real-time, portable, bedside assessment of pleural pathology without radiation exposure, making it ideal for critically ill patients and allowing serial examinations. The systematic scanning of six anatomical landmarks revealed predominant gas throughout the right hemithorax with minimal posterior pleural fluid accumulation. Critically, the complete absence of identifiable lung tissue above the hemidiaphragm indicated mechanical lung compression from external pressure. The characteristic "barcode sign" (alternating lines of acoustic shadowing and reverberation artifacts from air-pleural interfaces) is highly specific for pneumothorax or hydropneumothorax when identified.[11,12] However, ultrasound cannot definitively distinguish between primary pneumothorax, hydropneumothorax, and giant bullae, nor can it adequately characterize the pleural lining and surrounding mediastinal structures. Computed tomography provided superior anatomical detail and definitive characterization of pleural pathology. The thin, enhancing pleural wall visible on computed tomography confirmed that the air-containing cavity represented a pleural compartment rather than intraparenchymal bullae. The presence of fluid within this cavity (visualized as dependent material with different attenuation from air) established the hydro-component of the diagnosis. Compressive atelectasis of the ipsilateral lower and middle lobes and leftward mediastinal shift further supported the diagnosis of significant pleural pathology causing mechanical compromise.[9,13]

The key differentiating features between loculated hydropneumothorax and giant pulmonary bullae are outlined in Table 6. Giant bullae are large (typically exceeding 20 millimeters), thin-walled, air-filled cavities that form within lung parenchyma from emphysematous destruction of alveolar walls or progressive pathological dilatation of distal alveolar spaces. They are bounded by visceral pleura as the inner boundary and may be surrounded by compressed lung tissue. The pleural boundary of a bulla is single-layered visceral pleura, whereas hydropneumothorax is bounded by visceral pleura on the lung side and parietal pleura on the chest wall side, creating a double-layered boundary.[10,14] Air-fluid levels can occur in both entities if fluid is present from superimposed infection or bleeding, making this finding non-specific.[15,16] However, bullae arise from intraparenchymal air trapping, whereas hydropneumothorax results from pleural space pathology. Bullae typically develop in patients with chronic obstructive pulmonary disease or inherited emphysema, whereas hydropneumothorax in this clinical context results from post-tuberculous pleural disease and bronchopleural communication.[7,17] Computed tomography can definitively differentiate these entities by demonstrating the exact location of the lesion (intraparenchymal with surrounding lung tissue for bullae, versus pleural space compartmentalization for hydropneumothorax), the nature of the boundary (thin visceral pleura alone for bullae, versus dual visceral and parietal pleural layers for hydropneumothorax), and the presence of associated compressive effects on surrounding structures. In this case, the visualization of compressive atelectasis of the lower and middle lobes, mediastinal shift, and the thin double pleural boundary on computed tomography favored hydropneumothorax over giant bullae as the correct diagnosis.[9,10]

A comparison with similar published case reports is presented in Table 7. Hydropneumothorax complicating post-tuberculosis lung disease remains uncommon, with limited case reports in the medical literature. Al-Neyadi and colleagues reported a case of hydropneumothorax with bronchopleural fistula following tuberculosis reactivation, similarly demonstrating the substantial diagnostic challenge of distinguishing this entity from other cavitary lesions.[17] Piao and colleagues described giant bullous lung disease that was initially misdiagnosed as pneumothorax, highlighting the diagnostic difficulty and clinical implications of differentiating between large air-containing lesions.[18-20] El Husseini and

colleagues recently published a prospective multicenter study mapping bullous emphysema with lung ultrasound, demonstrating that lung ultrasound can identify bullae but requires comparison with computed tomography for definitive diagnosis.[21-25] These published reports collectively underscore the importance of multimodality imaging and the diagnostic uncertainty that can arise when individual imaging modalities are used in isolation. The present case adds significantly to the literature by demonstrating the complete diagnostic pathway in a post-tuberculosis patient and emphasizing how systematic ultrasound assessment, when integrated with radiography and computed tomography, achieves diagnostic certainty.[1,2,7] This case illustrates several important clinical principles: loculated hydropneumothorax should be included in the differential diagnosis of large cavitary lesions in patients with post-tuberculosis lung disease; chest radiography provides valuable initial assessment but has limitations; thoracic ultrasound offers rapid bedside assessment but cannot definitively characterize pleural lining; computed tomography provides definitive anatomical characterization; integration of clinical presentation, imaging findings, and radiological diagnosis guides appropriate management; and post-tuberculosis patients remain at risk for serious complications years after successful treatment.[1,5,15]

Table 5. Imaging modality comparison.

Parameter	Chest radiography	Thoracic ultrasound	Computed tomography
Sensitivity for pathology	High for cavitary lesion	High for gas and fluid	Very high for anatomy
Specificity	Low for characterization	Low without correlative	Very high for diagnosis
Primary clinical role	Initial screening, rapid assessment	Bedside assessment, portable	Definitive diagnosis, anatomical detail
Major limitations	Cannot characterize the pleural	Cannot visualize tissue above the lesion	Radiation exposure, cost, and availability
Key findings in this case	Cavity with air-fluid level	Dominant gas, minimal fluid	Thin pleural wall, compressed lung
Characteristic imaging sign	Air-fluid level	Barcode sign artifact	Compressive atelectasis pattern

Table 6. Differential diagnosis - Loculated hydropneumothorax versus giant pulmonary bullae.

Feature	Loculated hydropneumothorax	Giant pulmonary bullae
Origin and location	Pleural space (extra-pulmonary)	Intraparenchymal (within lung tissue)
Wall composition	Visceral and parietal pleura	Visceral pleura alone
Wall thickness and enhancement	Often thicker, enhances contrast	Thin wall, minimal enhancement
Air-fluid level	Common (hydro component)	Less common if present
Mediastinal shift	Common from mass effect	Uncommon unless very large
Compressive atelectasis	Present (lung pushed inward)	Minimal (surrounded by air)
Ultrasound findings	Barcode sign, posterior fluid	Barcode sign, no fluid
Computed tomography	Pleural boundary visible, fluid dependent	Intraparenchymal location, the lung around it
Underlying pathology	TB history, pleural disease, bronchopleural fistula	COPD, emphysema, genetic disease
Typical treatment approach	Drainage, antibiotics if infected	Observation, surgery if symptomatic

Table 7. Literature comparison - Similar case reports.

Parameter	Present case	Al-Neyadi 2023	Piao 2023	El Husseini 2025
Age and gender	63-year-old female	Adult, gender not reported	Adult, gender not reported	Prospective series
Tuberculosis history	Post-TB, cured 2 years prior	TB reactivation during the course	Non-TB etiology	Non-TB etiology
Clinical presentation	Dyspnea, productive cough	Similar respiratory symptoms	Pneumothorax-like presentation	Varied presentations
Initial diagnostic impression	Suspected giant pulmonary bullae	Suspected pneumothorax	Giant bullae/pneumothorax	Bullae
Imaging modalities employed	Chest radiography, ultrasound, CT	CXR, ultrasound, CT	CXR, CT	Ultrasound and CT
Final confirmed diagnosis	Loculated hydropneumothorax	Hydropneumothorax with BPF	Giant pulmonary bullae	Bullae with ultrasound mapping
Key clinical learning point	Multimodality essential for certainty	Bronchopleural fistula challenge	CT essential for diagnosis	Ultrasound useful with CT

4. Conclusion

This case of loculated right-sided hydropneumothorax in a 63-year-old post-tuberculosis patient highlights the diagnostic complexity of pleural complications arising as late sequelae of successfully treated mycobacterial infection. The clinical presentation of progressive dyspnea and hypoxemia, combined with radiological findings of a large cavitary lesion with air-fluid level and mediastinal shift, created initial diagnostic uncertainty regarding whether this represented a simple pneumothorax, hydropneumothorax, or giant pulmonary bullae. Systematic application of multimodality imaging—including chest radiography for initial rapid detection, thoracic ultrasound for real-time pleural assessment with portability, and contrast-enhanced computed tomography for definitive anatomical characterization—successfully established the diagnosis of loculated hydropneumothorax. Each imaging modality contributed essential diagnostic information; none alone was sufficient for complete and confident characterization. This case adds significantly to the medical literature concerning post-tuberculosis lung disease and emphasizes that loculated hydropneumothorax should be considered in the differential diagnosis of large cavitary lesions in patients with prior tuberculosis history. Furthermore, it demonstrates that multimodality imaging integration is the appropriate diagnostic strategy in complex thoracic pathology, allowing for confident diagnosis and guiding subsequent clinical management decisions with minimal diagnostic delay and without unnecessary surgical intervention.

5. References

1. Allwood BW, Byrne AL, Meghji J, et al. Post-tuberculosis lung disease: clinical review of an under-recognised global challenge. Lancet Respir Med. 2021; 9(12): 1243-55.
2. Meghji J, Simpson H, Squire SB, et al. A systematic review of the prevalence and pattern of imaging defined post-tuberculosis lung disease. PLoS One. 2021; 16(8): e0254475.
3. Byrne AL, Marais BJ, Mitnick CD, et al.Tuberculosis and chronic respiratory disease: a systematic review. Int J Infect Dis. 2015; 32: 138-46.
4. Amaral AFS, Coton S, Kato B, et al. Tuberculosis associates with both airflow obstruction and low lung function: BOLD results. Eur Respir J. 2015; 46(4): 1104-12.
5. Ravimohan S, Kornfeld H, Weissman D, et al. Tuberculosis and lung damage: from epidemiology to pathophysiology. Eur Respir

Rev. 2018; 27(147): 170077.
6. Adegunsoye A, Strek ME. Therapeutic approach to adult fibrotic lung diseases. Chest. 2016; 150(6): 1371-86.
7. Kim HY, Song KS, Goo JM, et al. Thoracic sequelae and complications of tuberculosis. Radiographics. 2001; 21(4): 839-58.
8. Skoura E, Zumla A, Bomanji J. Imaging in tuberculosis. Int J Infect Dis. 2015; 32: 87-93.
9. Choi H, Chon HR, Kim K, et al. Imaging of pleural diseases: a comparison of recommendations and practice. Diagnostics. 2021; 11(7): 1282.
10. Mirsadraee S, Oswal D, Alizadeh Y, et al. The 7th lung cancer T staging classification and TNM system: imaging implications. Clin Radiol. 2012; 67(8): e57-e63.
11. Volpicelli G, Elbarbary M, Blaivas M, et al. International evidence-based recommendations for point-of-care lung ultrasound. Intensive Care Med. 2012; 38(4): 577-91.
12. Staub LJ, Biscaro RRM, Kaszubowski E, et al. Chest ultrasonography for the emergency diagnosis of traumatic pneumothorax and haemothorax: a systematic review and meta-analysis. Injury. 2018; 49(3): 457-66.
13. Hansell DM, Bankier AA, MacMahon H, et al. Fleischner Society: glossary of terms for thoracic imaging. Radiology. 2008; 246(3): 697-722.
14. Hnizdo E, Singh T, Churchyard G. Chronic pulmonary function impairment caused by initial and recurrent pulmonary tuberculosis following treatment. Thorax. 2000; 55(1): 32-8.
15. Datta S, Evans CA. Healthy survival after tuberculosis. Lancet Infect Dis. 2019; 19(10): 1060-1.
16. Auld SC, Barczak AK, Bishai WR, et al. Pathogenesis of post-tuberculosis lung disease: defining knowledge gaps and research priorities at the Second International Post-Tuberculosis Symposium. Am J Respir Crit Care Med. 2024; 210(8): 979-93.
17. Al-Neyadi M, Alghfeli S, Dukandar M. Hydropneumothorax with bronchopleural fistula following the activation of Mycobacterium tuberculosis: a case report. Cureus. 2023; 15(6): e40844.
18. Mahalingam S, Rajendran G, Nathan B, et al. Hydro-point—the forgotten and unspoken entity in hydropneumothorax. Australas J Ultrasound Med. 2021; 24(4): 246-8.
19. Santosh KC, Allu S, Rajaraman S, et al. Advances in deep learning for tuberculosis screening using chest X-rays: the last 5 years review. J Med Syst. 2022; 46(11): 82.
20. Piao X, Alyass F, Yousuf A. Cardiothoracic surgery management of giant bullous lung disease initially misdiagnosed as pneumothorax: a case report. Cureus. 2023; 15(3): e36313.
21. El Husseini K, Flament T, Laroumagne S, et al. Mapping bullous emphysema with lung ultrasound: a prospective multicentre study. Respirology. 2025; 30(7): 633-43.
22. Roberts ME, Rahman NM, Maskell NA, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023; 78(Suppl 3): s1-s42.
23. Soldati G, Demi M, Smargiassi A, Inchingolo R, Demi L. The role of ultrasound lung artifacts in the diagnosis of respiratory diseases. Expert Rev Respir Med. 2019; 13(2): 163-72.
24. Desai H, Agrawal A. Pulmonary emergencies: pneumothorax, acute chest syndrome, and massive hemoptysis. Med Clin North Am. 2012; 96(6): 1127-48.
25. Zhai K, Lu Y, Shi HZ. Tuberculous pleural effusion. J Thorac Dis. 2016; 8(7): E486-E494.