

Case Report

Pneumothorax as an atypical initial presentation of tuberculosis in non-endemic Australia: A case report

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Introduction

Tuberculosis (TB)-related pneumothorax is a rare but serious complication, especially in low-incidence settings such as Australia. We report the case of a 21-year-old indigenous female who presented with respiratory distress following a two-week history of low-grade fever and pleuritic chest pain. Initial imaging revealed a right-sided hydropneumothorax, managed with emergency pigtail catheter insertion and empirical antibiotics. Persistent fever and radiological findings of loculated pleural effusion and cavitary lung disease prompted further evaluation. She underwent video-assisted thoracoscopic surgery (VATS) with decortication and biopsy, which confirmed tuberculosis via nucleic acid amplification testing, despite negative acid-fast bacilli on smear. Anti-tubercular therapy was initiated, resulting in rapid clinical improvement and complete radiological resolution by two months. This case highlights the diagnostic challenges of atypical TB presentations in low-endemic regions and underscores the importance of early surgical intervention and molecular diagnostics in managing TB-related pleural disease.

Case Report

We report the case of a 21-year-old indigenous female who presented with a two-week history of low-grade fever, non-productive cough, and right-sided pleuritic chest pain. She attended the Emergency Department with sudden-onset worsening of right-sided chest pain associated with shortness of breath. On examination, she was febrile at 38.7°C. Her cardiovascular parameters were stable, with a heart rate of 98 beats per minute and

blood pressure of 120/80 mmHg. She was in respiratory distress with a respiratory rate of 28 breaths per minute. Respiratory examination revealed reduced chest expansion and air entry on the right hemithorax, a hyper resonant percussion note and a midline trachea. Her oxygen saturation on room air was 88%.

Basic blood investigations are given in Table 1.

Table 1: Blood investigations

Investigation	Value	Normal Range
White Cell Count	24 * 10 ³ /ml	4-11
Haemoglobin	114 g/dl	110-140
Platelet	456 * 10 ³ / ml	150-450
C- Reactive Protein	230 mg/dl	<5
Erythrocyte Sediment Rate	110 mm/hr	<15
Sodium	138 mmol/L	135-145
Potassium	4.9 mmol/L	3.5-5.2
Chloride	103 mmol/L	95-110
Bicarbonate	30 mmol/L	22-32
Glucose	4.7 mmol/L	3.0-6.0
Urea	5.9 mmol/L	2.1-7.1
Creatinine	48 umol/L	45-90
Protein (Total)	72 g/L	60-80
Albumin	27 g/L	35-50
Globulin	46 g/L	25-45
Bilirubin (Total)	5 umol/L	<20
Alkaline Phosphatase	114 U/L	30-110
Gamma-GT	40 U/L	<38
Alanine Transaminase	47 U/L	<34
Aspartate Transaminase	28 U/L	<31
Lactate Dehydrogenase	444 U/L	120-250
Calcium	2.27 mmol/L	2.10-2.60
Phosphate	1.53 mmol/L	0.75-1.50
Magnesium	0.72 mmol/L	0.70-1.10
Mycoplasma Antibody	Negative	
CMV IgG and IgM	Negative	
Retroviral Antibody	Negative	
EBV IgG and IgM	Negative	
Hepatitis B C antibody	Negative	

A chest X-ray revealed a right-sided hydropneumothorax (Figure 1).



Figure 1: Chest Xray – Right side hydropneumothorax

An emergency pigtail catheter was inserted to relieve the pneumothorax. Empirical intravenous antibiotic therapy was commenced with ceftriaxone 2 g daily and vancomycin 1g twice daily, given the patient's background from an indigenous community and the associated risk of MRSA infection. The intercostal catheter was removed on the second day post-insertion once the air leak stopped. Despite 96 hours of intravenous antibiotic therapy, the patient remained febrile. A contrast-enhanced CT scan of the chest showed a persistent loculated pneumothorax and loculated right pleural effusion with residual fluid suggestive of a fibrinopurulent phase empyema. Additionally, nodular infiltrates and cavitary changes were noted in the right lower lobe, raising suspicion of a granulomatous infection.



Figure 2: Computed Tomography of chest. Green arrows point to loculated right pleural effusion, nodular infiltrates and cavitary changes in the right lower lobe.

A series of investigations was undertaken to determine the underlying etiology, including sputum and pleural fluid studies.

The results of the sputum and pleural fluid studies are given in Table 2.

Table 2: Sputum and Pleural fluid investigations

Sputum	
Sputum microscopy and culture	Normal respiratory flora 1+
Sputum Acid Fast Bacilli	No acid-fast bacilli (AFB) were seen
Pleural Fluid Aspirate	
Appearance	Straw colored
Cell Count : WBC	370 $\times 10^6/L$
RBC	760 $\times 10^6/L$
Differential: Polymorphs	38% (<10%)
Mononuclear	62% (<10%)
Protein	60 g/L (<30g/L))
Albumin	20 g/L (<10g/L)
pH	7.2 (>7.4)
Adenosine Deaminase	61 U/L (<50U/L)
ACID - FAST MICROSCOPY [Standard Ziehl-Neelsen]	No acid-fast bacilli (AFB) were seen

She was transferred to a cardiothoracic unit where she underwent video-assisted thoracoscopic surgery (VATS) with pleural washout, decortication, and pleural biopsy. A suction drain was inserted intraoperatively and was removed 24 hours post-surgery. Right pleural biopsy specimen report is given in Table 3.

Table 3: Right Pleural Biopsy Report

Gram Stain	Leucocytes – Nil, Epithelial – Nil, No organisms seen
Bacterial and mycology Culture	Negative
ACID - FAST MICROSCOPY [Standard Ziehl-Neelsen]	No acid-fast bacilli (AFB) were seen
MYCOBACTERIAL NUCLEIC ACID AMPLIFICATION TEST Method: GeneXpert ULTRA Assay (Direct)	DNA specific for <i>Mycobacterium tuberculosis</i> complex was detected
Tuberculosis culture	Positive for <i>Mycobacterium tuberculosis</i>

While acid-fast bacilli were not identified on smear microscopy, nucleic acid amplification testing of the pleural tissue detected DNA specific for the *Mycobacterium tuberculosis* complex. Given the presence of parenchymal nodular infiltrates and cavitary changes in the right lower lobe, along with biopsy-confirmed pleural tuberculosis, a diagnosis of concomitant pulmonary and pleural tuberculosis complicated by hydropneumothorax was made. Initial anti-tubercular therapy included rifampicin, ethambutol, isoniazid, and pyrazinamide. Following the development of hepatic dysfunction, isoniazid and pyrazinamide were withheld and levofloxacin was introduced. She was referred to the local infectious diseases service and enrolled in directly observed therapy (DOT) under the care of the TB nursing team.

She made an uneventful recovery, with defervescence and marked clinical improvement observed within one week of commencing anti-tuberculosis therapy. A repeat chest X-ray performed two months into treatment showed resolution with minimal residual pleural collection.



Figure 3: Follow up Chest Xray – Radiological resolution with minimal residual pleural collection.

Discussion

Tuberculosis (TB), caused by *Mycobacterium tuberculosis*, remains a significant global health issue, with variable prevalence across regions. Australia is considered a low-incidence country; however, certain populations and regions continue to experience disproportionately higher TB rates (1).

According to the Communicable Diseases Intelligence reports from the Australian Government Department of Health, Queensland consistently reports one of the higher TB notification rates among Australian states, although still within low-endemic thresholds (approximately 5–7 cases per 100,000 population annually (2,3).

Notably, the burden of TB in Queensland is not evenly distributed across the population. Higher notification rates are observed among overseas-born individuals, particularly those originating from high-prevalence countries such as India, Papua New Guinea, the Philippines, and nations in Southeast Asia (2,4). Aboriginal and Torres Strait Islander populations are also disproportionately affected, especially in Far North Queensland and the Torres Strait regions, where healthcare access and socioeconomic factors contribute to increased vulnerability (2,5). Additionally, Queensland's cross-border regions near Papua New Guinea present unique public health challenges due to geographic proximity, population mobility, and cross-border transmission, necessitating ongoing surveillance and collaborative control efforts (6).

TB in Queensland is often presented in a pulmonary form, consistent with global patterns. However, a significant proportion of patients present with non-specific or

atypical symptoms, which can delay diagnosis, especially in populations without traditional risk factors (7). Common clinical presentations include chronic cough, weight loss, night sweats, and hemoptysis (pulmonary TB). Lymphadenitis (most common form of extrapulmonary TB), pleural effusion, skeletal TB, and genitourinary TB are also reported (7,8). Rare presentations such as spontaneous pneumothorax, miliary TB, or CNS involvement can occur, often posing diagnostic challenges in low-incidence settings (9,10).

Pneumothorax is a rare but recognized complication of pulmonary tuberculosis (TB), with incidence estimates ranging from 1% to 2% among hospitalized patients with active disease, though rates may be higher in subgroups such as those with cavitary TB or HIV co-infection (11,12). Girling and Lyle reported spontaneous pneumothorax in approximately 1.5% of TB cases, highlighting its low frequency even in earlier clinical eras (13). With advancements in early diagnosis and effective anti-tubercular therapy, the incidence appears to be even lower in high-resource settings (14).

The pathophysiology of pneumothorax in TB commonly involves rupture of subpleural tuberculous cavities or bullae into the pleural space, caseous necrosis eroding the lung parenchyma near the pleura, or post-infective scarring that leads to bullous lung disease and eventual rupture (15). Iatrogenic causes, including complications from thoracentesis or mechanical ventilation in severely ill patients, can also contribute (16).

Clinically, TB-related pneumothorax often presents acutely with sudden onset of dyspnea, pleuritic chest pain, and hypoxia. It may be unilateral or, rarely, bilateral, the latter associated with high morbidity and mortality (17). Risk is further increased in patients with HIV or multidrug-resistant TB (MDR-TB), and outcomes are often poor if diagnosis and intervention are delayed (18).

In a study done by Shamai et al in 2011, of 53 patients with tuberculosis related pneumothorax, 64% were male, and the mean age was 34 years. Most patients (68%) were newly diagnosed with tuberculosis at the time they presented with pneumothorax. There was no significant association between pneumothorax and factors such as sex, smoking, or drug use. However, pneumothorax was significantly more common in patients younger than 30 years. Radiological findings showed that cavitary lesions were present in 38% of the patients and were significantly more frequent among those with pneumothorax ($P = .006$). Pulmonary infiltration and effusion were also common, observed in 36% and 32% of cases, respectively (19).

The management of tuberculosis (TB)-related pneumothorax involves a combination of supportive care and targeted treatment of the underlying mycobacterial infection. Immediate management typically includes intercostal chest tube drainage (ICD), which is the mainstay for moderate to large pneumothoraxes, especially when symptomatic (16). Needle aspiration may be used in selected small, first-time pneumothoraxes, but it is generally avoided in TB cases due to higher recurrence and complexity associated with cavitary lesions and bronchopleural communications. In cases of recurrent

pneumothorax or persistent air leaks, chemical pleurodesis may be indicated to obliterate the pleural space and prevent recurrence (23).

Effective control of the underlying TB infection is essential. Anti-tubercular therapy (ATT) should be promptly initiated or continued as per standard treatment guidelines. The World Health Organization (WHO) recommends the use of a two-month intensive phase consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol (2HRZE), followed by a four-month continuation phase with isoniazid and rifampicin (4HR), unless resistance is suspected (21). In patients with drug-resistant TB, second-line regimens should be used according to susceptibility patterns.

Surgical intervention may be required in complicated cases. Indications for surgery include persistent air leak beyond 5–7 days, non-resolving pneumothorax, bronchopleural fistula, or recurrent pneumothorax. Video-assisted thoracoscopic surgery (VATS) is often preferred over open thoracotomy due to its minimally invasive nature and efficacy. Surgical procedures may include bullectomy, decortication, fistula closure, or pleurectomy with pleurodesis (22).

Patients co-infected with HIV are more susceptible to pneumothorax due to greater pulmonary tissue damage and higher prevalence of disseminated disease. These patients experience more complications and have a significantly higher mortality risk (20).

Despite the seriousness of TB-related pneumothorax, the prognosis is generally favorable when managed promptly and effectively. However, outcomes are worsened by delayed initiation of ATT, extensive parenchymal destruction, HIV co-infection, presence of bronchopleural fistula, and multidrug-resistant TB (23). In resource-limited settings, a lack of surgical facilities and delayed diagnosis may further worsen prognosis.

Recent developments in management include the use of endobronchial valves to treat persistent pulmonary air leaks, especially in patients unfit for surgery. This minimally invasive approach has shown promising outcomes in select cases (24). Additionally, research is ongoing into minimally invasive bronchoscopy techniques for managing bronchopleural fistulas, offering hope for less invasive treatment alternatives.

Conclusion

This case illustrates the importance of considering tuberculosis in the differential diagnosis of spontaneous hydropneumothorax, especially in vulnerable populations such as Indigenous Australians. Although rare, TB-related pneumothorax can be initial manifestation of active infection and requires a high index of suspicion for timely diagnosis. Molecular diagnostic tools such as nucleic acid amplification testing can confirm the diagnosis even in smear-negative cases, facilitating early initiation of appropriate therapy. Surgical intervention, including VATS and decortication, remains an important component in managing loculated empyema and optimizing outcomes.

Prompt recognition and multidisciplinary care are essential for favorable prognosis in TB-related pleural complications.

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