International Journal of Advances in Medicine
Kadir Y et al. Int J Adv Med. 2024 Mar;11(2):104-108
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20240315

Case Report

Spontaneous tension pneumothorax: a devastating consequence of pulmonary tuberculosis

Yolanda Kadir[1]*, Elok Ariyani Safitri[2]

[1]Department of Emergency, Charitas Hospital, Palembang, South Sumatera, Indonesia
[2]Department of Pulmonology, Charitas Hospital, Palembang, South Sumatera, Indonesia

Received: 20 January 2024
Accepted: 05 February 2024

***Correspondence:**
Dr. Maya Vikas Kshirsagar,
E-mail: yolandakadir@gmail.com

ABSTRACT

Tuberculosis (TB) was the world's second leading cause of death from a single infectious agent, with mortality reaching 50% in untreated cases. It has been acknowledged as a potential cause of secondary spontaneous pneumothorax. A 57-year-old male presented to emergency department due to shortness of breath since 12 hours before admission. He had a history of chronic cough along with significant weight loss. He was cachexic and fully alert, with normal blood pressure, tachypnea, and slight tachycardia. Decreased breath sounds on the right hemithorax was noted. Chest x-ray revealed right tension pneumothorax and active pulmonary TB. Emergency needle decompression was performed with 14-gauge intravenous catheter. Unfortunately, he died eight hours later following a cardiac arrest. Occurrence of spontaneous tension pneumothorax in TB involves several mechanisms such as pleural porosity, chronic inflammation, and alveolar rupture. Compensatory mechanisms including gradual tachycardia, respiratory rate elevation, along with increasingly negative contralateral intrathoracic pressures could preserve venous return, serving as protective factors against hypotension until the late stages. Late presentation of 12 hours after the first onset might contribute to enormous air leak that could not be effectively managed by needle decompression. Undiagnosed and untreated TB could lead to morbid consequence such as tension pneumothorax, highlighting the importance of TB detection within the community. Clinicians should be aware of variations in the clinical presentation of tension pneumothorax as compensatory mechanisms may hinder the diagnosis at initial presentation. Strategic approaches are imperative to reinforce our commitment to eliminate TB by 2030.

Keywords: Decompression, Tension pneumothorax, TB

INTRODUCTION

Pneumothorax refers to an abnormal accumulation of air or gas in the pleural space between the visceral and parietal pleura that can adversely affect ventilation, oxygenation, or both. The manifestation of symptoms varies based on the underlying cause and the extent of the pneumothorax. Predominant symptoms often include chest pain and shortness of breath, reported in 64 to 85% of cases.[1] The gradual accumulation of air within the pleural cavity can escalate to a life-threatening condition known as tension pneumothorax. In this scenario, there is a displacement of the mediastinum to opposite side, resulting in compression of the vena cava and other major vessels, diminished diastolic filling, and eventually compromised cardiac output. Tension pneumothorax exhibits more severe signs and symptoms, underscoring critical importance of prompt diagnosis and intervention for patient's survival.[2,3]

TB has long been recognized and documented as a cause to secondary spontaneous pneumothorax, occurring in approximately 1.3-5% of individuals with pulmonary TB.[4-6] Despite being preventable and often curable illness, in 2022 TB was world's 2nd leading cause of death from single infectious agent, surpassed only by coronavirus disease (COVID-19). TB caused nearly double number of

deaths compared to HIV/AIDS. Each year, over 10 million people contract TB, and without intervention, mortality rate for TB is estimated to reach 50%.[7]

In this report, we present a devastating case of spontaneous tension pneumothorax associated with undiagnosed and untreated pulmonary TB.

CASE REPORT

A 57-year-old male was brought into our emergency department due to sudden shortness of breath since 12 hours before admission. He had a history of chronic dry cough with scanty sputum for around 6 months that became more productive in recent weeks. He also complained about decreased appetite and significant weight loss, along with fatigue and occasional chest discomfort. Expectoration of blood-tinged sputum was not reported. He did not seek treatment for his condition.

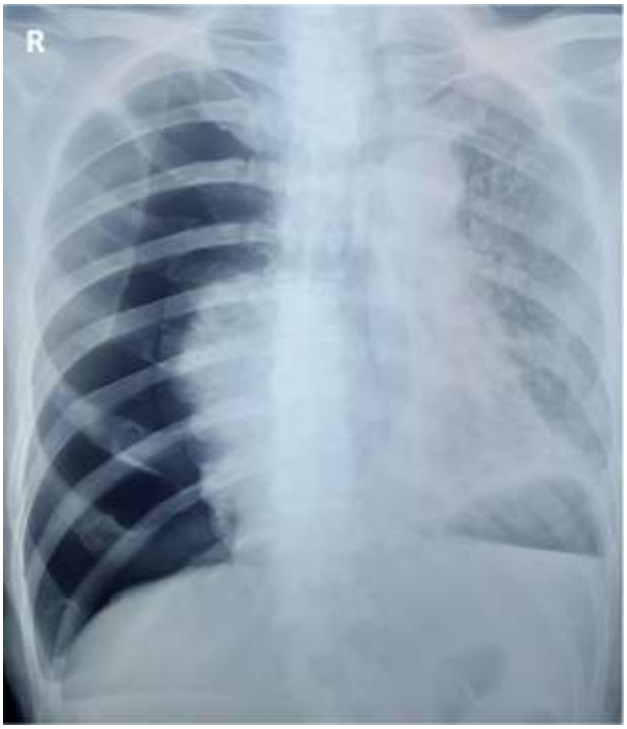

Figure 1: Chest X-ray of right tension pneumothorax and active pulmonary TB. Yellow arrow points to visceral pleural edge of collapsed right lung.

On initial evaluation, the patient was cachexic and fully alert, with slight tachycardia (heart rate of 104 bpm), tachypnea (respiratory rate of 34x/minute), blood pressure of 130/80 mmHg, and normal temperature (36.8ºC). Oxygen saturation was 81% with room air, increasing to 92% with 15 liters/minute oxygen via non-rebreathing mask (NRM). Physical examination revealed signs of respiratory distress, with decreased breath sounds on the right hemithorax and use of accessory muscles for respiration. Chest x-ray was promptly obtained, demonstrating a collapsed right lung with visible visceral pleural edge, seen as a thin and sharp white line, with no lung markings peripheral to this line. Trachea was deviated to the left side, along with depression of the right hemidiaphragm, and ipsilateral increased of intercostal spaces. These findings were consistent with tension pneumothorax. On the left lung, infiltrations were seen on the upper and middle zone, along with focal consolidations on basal zone, suggestive of active pulmonary TB. Laboratory results were within normal range.

History of traumatic injury was denied. Due to personal reasons, the patient refused to be referred to a larger hospital for chest tube drainage insertion. Shortly afterwards, he developed substantial deterioration with decreased level of consciousness and worsening cardiovascular status (blood pressure of 78/56 mmHg, heart rate of 139 bpm, respiratory rate of 44x/minute, oxygen saturation of 60% on 15 lpm NRM). His extremities were cold, with weak pulse and diaphoresis. Immediate needle decompression was performed by inserting a 14-gauge intravenous catheter into the second intercostal space in the midclavicular line (2nd ICS-MCL) of the right hemithorax. Thirty minutes after needle decompression, the patient went into cardiac arrest but was able to be resuscitated. Subsequently, the patient's family opted for a DNR (do-not-resuscitate) status. The patient died eight hours later following a second cardiac arrest.

DISCUSSION

Tension pneumothorax, a relatively rare yet potentially devastating clinical condition, is most frequently encountered in pre-hospital, emergency department (ED), and intensive care unit (ICU) settings.[3] This condition arises when there is disruption to the visceral pleura, parietal pleura, or the tracheobronchial tree, resulting in the formation of a one-way valve. This valve allows air to enter the pleural space but prevents its outward flow. With each inhalation, the volume of nonabsorbable intrapleural air increases, elevating pressure within the affected hemithorax. This leads to the collapse of the ipsilateral lung, inducing hypoxia. As pressure continues to build, the mediastinum shifts toward the contralateral side, compressing both the contralateral lung and the vasculature entering the right atrium of the heart. Consequently, worsening hypoxia and compromised venous return ensue.[8,9]

During lung auscultation, the presence of decreased or absent breath sounds on the ipsilateral side, diminished tactile fremitus, hyper-resonant percussion sounds, and potential asymmetrical lung expansion indicates the likelihood of pneumothorax. In cases of tension pneumothorax, patients typically exhibit signs of hemodynamic instability, such as hypotension and tachycardia. Additional indicators may include cyanosis and jugular venous distension.[2] However, despite tension pneumothorax being commonly viewed as a clinical diagnosis, there is a discrepancy in the descriptions of its clinical manifestations in the literature and guidelines.

This discrepancy raises concerns, as delayed or missed diagnoses have been reported in patients lacking the classic findings associated with the disorder.[3]

Our patient presented to the emergency department with respiratory distress and classic signs of pneumothorax. However, he was alert with normal blood pressure and only slight tachycardia, with no history of traumatic injury, thus hindering our immediate diagnosis of tension pneumothorax at initial presentation. Previous systematic review by Roberts et al. demonstrated that hypotension were reported among only 16% of cases.[10] The observed phenomenon may be elucidated by several compensatory mechanisms that evolve during tension pneumothorax, potentially serving as protective factors against the onset of hypotension until the late stages of the disorder. These compensatory mechanisms involve a gradual increase in tachycardia, an elevation in respiratory rate and tidal volume, and a progressive enhancement in contralateral chest excursions. These mechanisms may sustain arterial blood pressure by preserving cardiac venous return through heightened spontaneous respiratory efforts, resulting in increasingly negative contralateral intrathoracic pressures during inspiration. Additionally, a significant rise in heart rate may occur due to baroreceptor reflexes and/or the effects of catecholamines released onto the heart. The authors argue that individuals breathing unassisted (not on ventilatory support) rarely exhibit sudden hemodynamic compromise. Therefore, they propose that obtaining a chest radiograph in a monitored setting to confirm the diagnosis and localize the disease might be more appropriate than immediately resorting to urgent thoracic decompression for patients who are not in an extreme condition.[10,11]

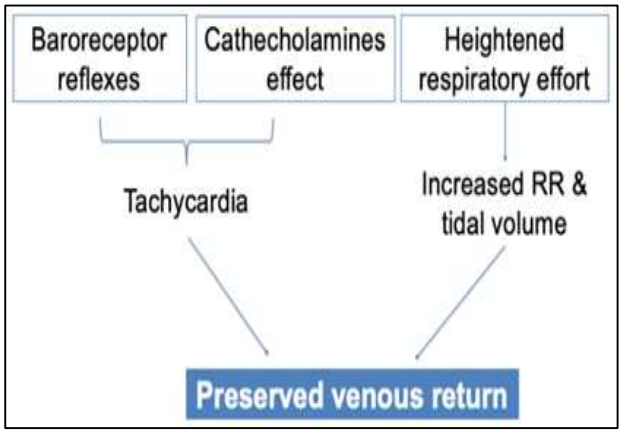

Figure 2: Compensatory mechanisms during tension pneumothorax.

Our case involved a secondary spontaneous pneumothorax, likely instigated by an underlying TB infection. The pathogenesis of spontaneous tension pneumothorax in pulmonary TB involves pleura necrosis and the formation of pleural-pulmonary fibrotic adhesions, termed 'pleural porosity'.[4] This condition is characterized by areas of disrupted mesothelial cells at the visceral pleura, replaced by an inflammatory elastofibrotic layer with increased porosity, allowing air leakage into the pleural space.[12] It is also suspected that *Mycobacterium tuberculosis* induces chronic inflammation through macrophage activation, leading to obstruction, hyperinflation, and alveolar rupture.[13]

Miliary TB represents a potentially lethal manifestation of disseminated disease, stemming from the hematogenous spread of tubercle bacilli to the lungs and other organs. It leads to the development of tiny tuberculous foci, each approximately the size of millet seeds (1 to 2 mm).[14] Several mechanisms have been proposed to elucidate the pathogenesis of pneumothorax in miliary TB. One hypothesis suggests the formation of a small area of confluent subpleural miliary nodules, which undergoes caseation and necrosis, eventually rupturing into the pleural space and causing pneumothorax. Another mechanism involves increased intra-alveolar pressure resulting from excessive coughing, leading to the rupture of intra-alveolar septa and subsequent pneumomediastinum. Pneumothorax in this scenario occurs due to the escape of air through the mediastinal pleura. Additionally, bullous or emphysematous lesions may form near miliary tubercles, and their rupture could contribute to the development of pneumothorax.[15,16]

Pleural infection may arise from the rupture of subpleural caseous lesions, leading to the buildup of chronic empyema. A bronchopleural fistula, characterized as a sinus tract connecting the main stem, lobar, or segmental bronchus to the pleural space, can spontaneously occur as a complication during the progression of TB. Both chronic empyema and bronchopleural fistula have the potential to cause spontaneous and subsequent tension pneumothorax.[6,17]

In this particular case, we performed emergency needle decompression in the 2nd ICS-MCL following progressive deterioration of patient's clinical status after chest x-ray was taken. We confirmed the removal of air in the form of air bubbles in sterile infusion fluid. The primary objective of needle decompression is to remove ectopic air from the pleural space, allowing the lung to re-expand and relieving associated tension physiology. However, reported success rates vary widely, ranging from 5% to 96%. Studies have shown that needle thoracocentesis often fails to effectively evacuate an active, ongoing air leak.[18] Cullinane et al reported a 50% failure rate in chest decompression despite seemingly adequate catheter length.[19] Lack of clinical improvement and subsequent cardiac arrest in our patient raised concerns about a potential failure in evacuating the pneumothorax. Various factors may contribute to this failure, including inadequate catheter length, incorrect placement technique, clot formation within the catheter, kinking or compression of the catheter, and an air leak rate surpassing the needle's evacuation capacity.[20] In our case, late presentation of 12 hours after the first symptom onset might contribute to the enormous air leak that could not be effectively managed by needle decompression.

Despite rigorous measures enforced to eliminate TB, Indonesia is ranked as the 2nd country with the highest TB burden based on the world health organization (WHO) estimation. In 2021, TB incidence rate is 354 per 100,000 populations with mortality rate reaching 52 per 100,000 populations.[21,22] In the same year, a study estimated that Indonesia's TB incidence rate is 759 per 100,000 populations, significantly higher than the figure estimated by the WHO.[23] Pulmonary TB accounts for approximately 91% of TB cases in Indonesia, posing a heightened risk of transmission between individuals and underscoring the need for effective screening and prevention measures. According to the 2022 global TB report, the ministry of health noted that only 45% of TB cases in Indonesia had been diagnosed. Furthermore, preventative measures, including TB prevention therapy for family members or close contacts (0.3%) and other high-risk groups (0.2%), have fallen short of their targeted goals, reaching only 29% and 10%, respectively.[24]

In this case, our patient had chronic cough and other typical symptoms of TB, but he was never diagnosed and treated accordingly. Early diagnosis is a pivotal strategy in the fight against TB. Delays in diagnosis are often attributed to either patients or the health system. Patient-related delays, as observed in our case, result from individuals not seeking medical care promptly. Health-system delays, on the other hand, are primarily due to healthcare professionals not considering TB during patient care.[25] Suboptimal detection performance is further compounded by inadequate access to advanced diagnostic tests. The resulting delay in diagnosis and instances of misdiagnosis contribute to heightened morbidity and mortality in patients and perpetuate the transmission of TB.[23] Addressing these challenges requires the implementation of strategic approaches, including improvement of access to patient-centered TB services, intensifying health measures for TB prevention and control, strengthening TB program management, and fostering collaboration among communities, stakeholders, and other multisectoral partners in TB control.[26]

We report a case of 57-year-old man with a history of chronic cough, who came to our emergency department due to severe respiratory distress. Timely diagnosis and treatment of tension pneumothorax is critical due to the emergency nature of this condition. Clinicians should be aware of variations in the clinical presentation as compensatory mechanisms may hinder the diagnosis at initial presentation. This case report also highlights the importance of TB detection in our community, as delay in diagnosis and treatment could lead to morbid consequence such as tension pneumothorax in our case. On that account, strategic approaches are imperative to reinforce our commitment to eliminate TB by 2030.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Sajadi-Ernazarova KR, Martin J, Gupta N. Acute Pneumothorax Evaluation and Treatment, 2022. Available at: http://www.ncbi.nlm.nih.gov/books/NBK538316/. Accessed on 15 December 2023.
2. Jalota Sahota R, Sayad E. Tension Pneumothorax, 2023. Available at: http://www.ncbi.nlm.nih.gov/books/NBK559090/. Accessed on 15 December 2023.
3. Roberts DJ, Leigh-Smith S, Faris PD, Ball CG, Robertson HL, Blackmore C et al. Clinical manifestations of tension pneumothorax: protocol for a systematic review and meta-analysis. Syst Rev. 2014;3:3.
4. Pradana AD. Spontaneous Tuberculosis-Associated Tension Pneumothorax: A Case Report and Literature Review. Case Rep Acute Med. 2020;3(2):35-9.
5. Briones-Claudett KH, Briones-Claudett MH, Moreno AP, Vargas DE, Martinez Alvarez ME, Andrade MG. Spontaneous Pneumothorax After Rupture of the Cavity as the Initial Presentation of Tuberculosis in the Emergency Department. Am J Case Rep. 2020;21:e920393-1-e920393-6.
6. Grossman DB, Nasrallah E. Pneumothorax in Liberia: Complications of Tuberculosis. West J Emerg Med. 2013;14(3):233-5.
7. World Health Organization. Global Tuberculosis Report, 2023. Available at: https://www.who.int/teams/global-tuberculosis-programme/tb-reports. Accessed on 18 December 2023.
8. Sharma A, Jindal P. Principles of diagnosis and management of traumatic pneumothorax. J Emerg Trauma Shock. 2008;1(1):34-41.
9. Zarogoulidis P, Kioumis I, Pitsiou G, Porpodis K, Lampaki S, Papaiwannou A et al. Pneumothorax: from definition to diagnosis and treatment. J Thorac Dis. 2014;6(4):S372-6.
10. Roberts DJ, Leigh-Smith S, Faris PD, Blackmore C, Ball CG, Robertson HL et al. Clinical Presentation of Patients with Tension Pneumothorax: A Systematic Review. Ann Surg. 2015;261(6):1068-78.
11. Leigh-Smith S, Harris T. Tension pneumothorax-time for a re-think? Emerg Med J. 2005;22(1):8-16.
12. Noppen M, De Keukeleire T. Pneumothorax. Respiration. 2008;76(2):121-7.
13. Nugroho AG, Edijono E, Marthaty SSI. Simultaneous Bilateral Spontaneous Pneumothorax in an HIV Positive Tuberculosis Patient. J Respirol Indon. 2023;43(2):116-20.
14. Vohra S, Dhaliwal HS. Miliary Tuberculosis, 2023. Available at: http://www.ncbi.nlm.nih.gov/books/NBK562300/. Accessed on 20 December 2023.
15. Dhamgaye T, Mishra G, Deokar K. Miliary tuberculosis with bilateral pneumothorax-A case report. Asian Pacific J Trop Dis. 2012;2(6):492-4.
16. Singh AS, Atam V, Das L. Secondary spontaneous pneumothorax complicating miliary tuberculosis in a young woman. Case Rep. 2014;2014:bcr2013201109.
17. Salik I, Vashisht R, Abramowicz AE. Bronchopleural Fistula, 2023. Available at: http://www.ncbi.

nlm.nih.gov/books/NBK534765/. Accessed on 21 December 2023.
18. Kaserer A, Stein P, Simmen HP, Spahn DR, Neuhaus V. Failure rate of prehospital chest decompression after severe thoracic trauma. Am J Emergency Med. 2017;35(3):469-74.
19. Cullinane DC, Morris JA, Bass JG, Rutherford EJ. Needle thoracostomy may not be indicated in the trauma patient. Injury. 2001;32(10):749-52.
20. Wernick B, Hon HH, Mubang RN, Cipriano A, Hughes R, Rankin DD et al. Complications of needle thoracostomy: A comprehensive clinical review. Int J Crit Illn Inj Sci. 2015;5(3):160-9.
21. World Health Organization. Global Tuberculosis Report, 2022. Available at https://www.who.int/teams/global-tuberculosis-programme/tb-reports/global-tuberculosis-report-2022. Accessed on 20 December 2023.
22. Directorate General of Prevention and Disease Control. Tuberculosis Control in Indonesia 2022. Ministry of Health Indonesia. 2023.
23. Noviyani A, Nopsopon T, Pongpirul K. Variation of tuberculosis prevalence across diagnostic approaches and geographical areas of Indonesia. PLoS One. 2021;16(10):e0258809.
24. Wahid W, Arsy F, Setiawati N, Vicri R, Miranda A, Lestari B et al. Policy Brief: ncreasing Financing for Tuberculosis Programs in Indonesia. Resilience Development Initiative. 2023.
25. Miller AC, Polgreen LA, Cavanaugh JE, Hornick DB, Polgreen PM. Missed Opportunities to Diagnose Tuberculosis Are Common Among Hospitalized Patients and Patients Seen in Emergency Departments. Open Forum Infect Dis. 2015;2(4):ofv171.
26. World Health Organization. Indonesia commitment to eliminate TB by 2030 supported by the highest-level government, 2021. Available at: https://www.who.int/indonesia/news/detail/28-11-2021-indonesia-commitment-to-eliminate-tb-by-2030-supported-by-the-highest-level-governmen. Accessed on 20 December 2023.

Cite this article as: Kadir Y, Safitri EA. Spontaneous tension pneumothorax: a devastating consequence of pulmonary tuberculosis. Int J Adv Med 2024;11:104-8.