Case Report

Simultaneous Bilateral Spontaneous Pneumothorax in an HIV Positive Tuberculosis Patient

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Abstract

Background: Even though tuberculosis has been linked to pneumothorax for a long time and has caused significant morbidity and mortality in some patients, it has been the topic of few publications and analyses, thus very little study has been done to evaluate and review on this matter.

Case: In this article, we reported a 39-year-old male, presented to the ER with breathlessness for the last 3 days accompanied by increased sputum productivity. The patient had an active pulmonary tuberculosis taht was under treatment, as well as HIV. Physical examination showed low chest expansion, weakened breathing sounds on both lungs, and the use of accessory breathing muscles. The chest X-ray showed bilateral pneumothorax. The patient underwent emergency chest decompression with a 16-gauge needle on both sides, followed by the insertion of an IPC and chest tube. The patient's breathlessness got significantly better, and after 35 days, the IPC was removed.

Discussion: Pneumothorax is a frequent complication in Tuberculosis with HIV, with a prevalence of 6.8% compared to 0.95-1.4% in Tuberculosis without HIV. The progression of breathlessness in bilateral pneumothorax on HIV positive Tuberculosis patient is slower, up to 3 days since onset, compared to pneumothorax occured in other etiologies. Secondary pneumothorax usually occurs after extensive destruction of the lungs, leaving a little functionality and lower cardiopulmonary reserve, thus requiring prompt evaluation and more aggresive lifesaving treatment.

Conclusion: Based on this case, bilateral pneumothorax found in HIV-associated TB patients comes with an insidious onset but warrants immediate evaluation and aggressive treatment or surgery if necessary.

Keywords: HIV-associated tuberculosis, pneumothorax, pulmonary tuberculosis

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INTRODUCTION

Secondary spontaneous pneumothorax in HIV-associated tuberculosis (TB) patients is more prevalent compared to immunocompetent TB patients (6.8% versus 0.95–1.4%). In immunocompromised patients, many blebs form on the lungs, and pneumothoraxes can happen when they rupture.[1–5]

While most secondary spontaneous pneumothoraxes are found with a sudden onset of breathlessness, secondary spontaneous pneumothoraxes found in HIV-infected TB patients have an insidious symptom; many have days of breathlessness before presenting to the hospital, even if they had bilateral pneumothoraxes. Such distinct characteristics need to be known and anticipated when tending to patients with HIV-associated TB.[1–5]

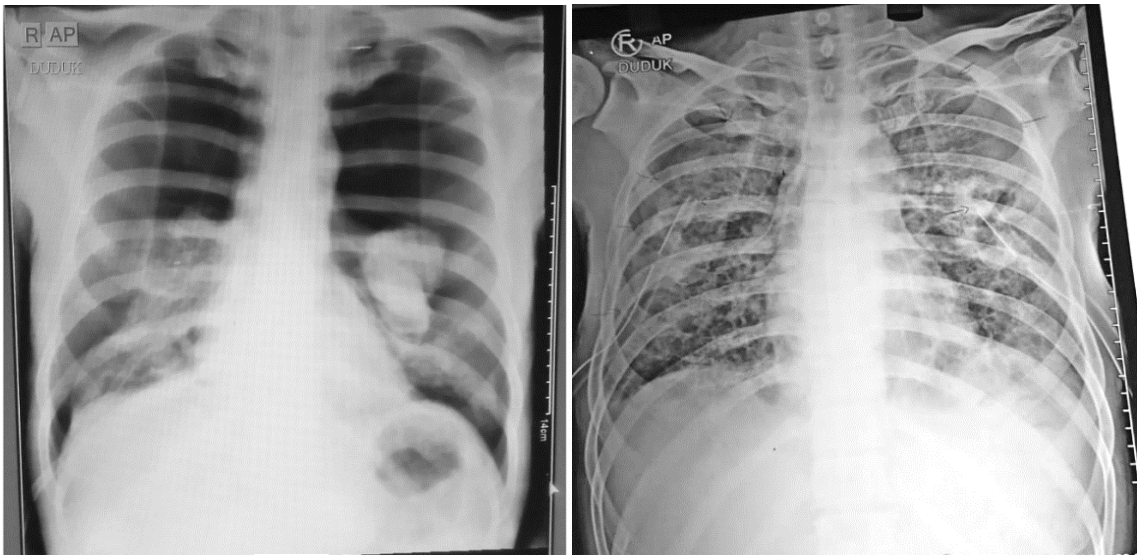
Tuberculosis has been linked to pneumothorax for a long time, but very few publications and analyses are performed on this subject. This case report discussed pneumothorax on TB, especially on people living with HIV.[1]

CASE

A 39-year-old male was presented to the ER with the chief complaint of breathlessness that started for the last 3 days and got worsened. The patient and his family said that he had never suffered from such a condition before. The patient had a productive cough for the last one month, which increased in production for the last 3 days. The patient had a history of having active pulmonary TB and had been undergoing treatment for the last 2 weeks with a fixed drug combination consisting of rifampicin, isoniazid, pyrazinamide, and ethambutol;

as well as being HIV positive, but had not received any treatment yet. No fever, difficulty swallowing, vomiting, nor any other symptom that was currently experienced.

Initial physical examination showed the patient was fully alert with GCS E4-V5-M6, blood pressure of 122/87 mmHg, heart rate of 105 bpm, body temperature of 36.5^{o}C, respiratory rate of 28/minute, and SpO_2 of 86% on room air. His chest was symmetrical, with low chest expansion, weakened breathing sounds on both lungs, and the usage of accessory breathing muscles. No other physical abnormalities were found.



(a) (b)

Figure 1. Chest X-Ray taken on (a) admission, radiological examination at the first time the patient went to the referral hospital, and (b) the fifth day, evaluation after IPC chest tube insertion and continuous suctions

The patient received oxygen supplementation, underwent emergency chest X-ray and blood laboratory examination. Chest X-ray showed bilateral pneumothorax (figure 1.a); no COVID-related laboratory abnormalities were found. The patient underwent emergency chest decompression with 16-gauge needle on the 2nd intercostal space midclavicular line on both side of his chest, followed by IPC insertion but no change in dyspnea. The IPC was changed to a chest tube with active suction for 15 minutes every 12 hours. The patient's breathlessness got significantly better, and the patient was admitted for further observation. Following 5 days of chest tube insertion, the patient felt better, and after confirming bilateral lung expansion (figure 1.b) on the 6th day, the chest tube was switched to IPC with occasional drainage whenever the patient had breathing difficulty. The patient was then discharged with stable hemodynamic and breathing.

On an outpatient visit after 35 days of IPC insertion, the patient had no breathing complaints, and the cough has subsided. The IPC was then removed, and since then the patient has been regularly treated and has not had a pneumothorax incident.

DISCUSSION

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis*. Depending on the infected organ, it is grouped into pulmonary TB, which is tuberculosis occurring in the lung parenchyma (constituting 80% of all cases), and extrapulmonary TB, which affects organs other than lungs, and is most common in the pleura, lymph nodes, spine, joints, genitourinary tract, central nervous system, abdomen, and other organs.[6]

Not everyone gets sick after becoming infected. Some people can get sick years after the infection; others whose immune systems got weakened by

other means may get sick just weeks after infection; while others may even never get sick. Only about 5–10% of all infected people get sick from *Mycobacterium tuberculosis* infection. Weakened immune systems strongly correlate with rate of TB disease development.[7]

There are two groups of people with higher rates of TB disease development. The first is people who have been recently infected, such as healthcare workers, people who have just traveled from high-risk areas, TB patient caregivers, and those who live in a high-TB transmission area. The second is those with lowered immunity, such as babies, children, or the elderly; an HIV-positive patient; drug abusers; people with diabetes mellitus, chronic kidney disease, low body weight; organ transplants; or head and neck cancer.[7]

People with TB most often come to their physician with a chief complaint of a chronic productive cough, sometimes with blood in their sputum. This symptom often comes with systemic symptoms, such as fever, night sweats, weight loss, and anorexia, while lymphadenopathy is a feature almost exclusively found in people with HIV infection.[8]

Cavitation is a major manifestation of human TB and closely related to poor prognosis, including delayed sputum conversion, infection relapse, and, arguably most avoided of all, the development of drug-resistant bacteria. If the cavity persists after 6 months of anti-tuberculosis therapy, the risk of relapse is doubled. Cavitation also increases transmission between humans since cavitation is attributable to a high bacterial burden and extensive disease. Relapse and the development of drug resistance are thought to be a result of poor drug penetration into the poorly vascularized cavity.[9]

Cavitation is found in 29% to 87% of all TB upon diagnosis. This could be higher than it actually is because cavitary TB has a higher bacterial load on its sputum, thus increasing the sensitivity of laboratory test. Lower immunity has a different effect on cavitation. Cavitation is found more often in people with diabetes mellitus but is significantly lower in people with untreated HIV, organ transplant recipients, and the elderly, although increased cavitation is seen after 6 months of ARV therapy.[9]

Cavitation most often occurs in the apices of the upper or lower lobes; once this happens, an exponential growth of bacteria in the lungs occurs, causing a higher bacterial burden and more bacteria being expectorated into the air through coughing. There are two ways the *Mycobacterium tuberculosis* can reach the apex of the lungs: first, through initial deposition in the apex; the other way is through the bloodborne phase in TB. This bloodborne phase is consistent with the way TB disseminates throughout the lungs in miliary TB and in positive TB blood culture in some HIV positive patients.[9]

Human lung parenchyma mainly consists of collagen fibers, specifically Type I, III, and IV. These collagens are very resistant to destruction and can only be degraded by specific enzymes. The aforementioned enzymes are found in leukocytes, which are recruited and activated by *Mycobacterium tuberculosis* to the lungs, consequently causing the destruction of lung parenchymal collagens. This release of protease enzymes activates particular matrix metalloproteinases (MMPs).[10]

MMP concentration correlates with the extent of lung tissue destruction. TB patients with more extensive tissue destruction have a significantly higher MMP yield in their sputum. Similarly, people with HIV and TB co-infection are more likely to have a lower MMP yield in their sputum, consistent with their anatomical finding that people with HIV and TB co-infection who have a lower CD4 count have lesser lung tissue destruction. This implies that in people with HIV and TB co-infection, the immune system is so weakened that it is insufficient to cause major lung destruction.[11]

Pneumothorax is a frequent complication found in TB patients with HIV infection. While TB alone is a frequent underlying cause of secondary spontaneous pneumothorax, consisting around 44.7–78% of pneumothorax patients, this seemingly high number consists only of about 0.95–1.4% of all active TB. Compared to pneumothorax in immunocompromised patients, the prevalence of pneumothorax in these patients goes up to 6.8%, which means around 20%

of all pneumothoraxes in immunocompromised patients are linked to the presence of TB.[1,2]

According to the WHO, in 2018, about 862,000 people living with HIV had tuberculosis co-infection, causing a third of AIDS deaths or about 251,000 deaths in 2018. Based on these numbers calculated with the pneumothorax incidents in HIV-infected TB patients, around 58,616 pneumothoraxes occurred in these patients, but many might not be documented due to the fact that up to 44% of all people with HIV-associated TB did not achieve medical care. [2,12]

Mechanism of pneumothorax in immunocompromised TB patients is still unclear, however, it is suspected that *Mycobacterium tuberculosis* induced a chronic inflammation through macrophage activation, causing obstruction, hyperinflation, and alveolar rupture. Other possible mechanisms are where a subpleural miliary nodule undergoes caseation and necrosis, followed by rupture, causing pneumothorax; and the formation of bullae or an emphysematous lesion, which then rupture.[2,3]

In the article published by Liu et al., several blebs were found on the lung surface during video assisted thoracoscopy (VAT), which supports the proposed mechanism where rupture of an emphysematous lesion causes pneumothorax, possibly even a bilateral pneumothorax in our case when a patient had multiple lesions on both lungs, which then rupture following a heavy cough induced by the first pneumothorax. The existence of multiple blebs also explains why patients with miliary TB often had recurrent pneumothoraxes, although this phenomenon was absent in our patient.[4]

Most secondary pneumothorax cases, such as secondary to COPD, TB, necrotizing pneumonia, *Pneumocystis carinii*, lung cancer, cystic fibrosis, acute severe asthma, and many others, had sudden onset of severe breathlessness as a primary symptom, accompanied with chest pain, hypoxemia, and hypercapnia. In contrast to other causes, pneumothorax secondary to TB in HIV patients has a different symptom: the onset of breathlessness is slower, even took 3 days in both our patient and the two patients mentioned in articles from Dhamgaye et al. and Liu et al., even though these 3 patients had bilateral pneumothorax, which arguably should present with more acute and severe breathlessness.[3–5]

Compared to primary spontaneous pneumothorax, secondary spontaneous pneumothorax bears a more severe complication to the patient; this is because of the underlying disease, be it TB, COPD, or HIV which compromises the patients' cardiopulmonary reserve, thus lowering their chance of survival. Added to this is the fact that most pneumothorax usually occurs after extensive destruction of the lungs, leaving only a little lung functionality. These premises warrant and prompt diagnosis from a precise and accurate history taking, physical diagnostic, chest radiography, and/or ultrasound which was found to be superior. Followed with aggressive lifesaving treatment such as chest tube, oxygen supplementation, or even a thoracotomy.[11,13]

In order to prevent the occurrence of recurrent secondary spontaneous pneumothorax, pleurodesis in form of thoracotomy surgery or Video Assisted Thoracoscopic Surgery (VATS) is considered the best solution, where identification and stapling of lesions are followed by pleurectomy and pleural abrasion to obliterate the pleural space. Although the recurrence of spontaneous pneumothorax following pleurodesis procedures is approximately 1%, pleurodesis in the form of VATS for secondary spontaneous pneumothorax is associated with higher morbidity compared to VATS for primary spontaneous pneumothorax, possibly due to the lower cardiopulmonary reserve observed in these patients; therefore, selective and strict patient evaluation before such procedure is essential to ensure patient safety and procedure benefit.[4,5]

LIMITATION

This study has potential limitations. The low number of patients currently documented with bilateral pneumothorax in Tuberculosis with HIV positive caused lack of generalizability and low level evidence. In this particular patient, late of treatment,

lack of medication history information may cause incomplete data and discussion.

CONCLUSION

This case report highlights insidious breathlessness onset in the case of bilateral pneumothorax in HIV positive patient, but due to more extensive pulmonary destruction that occured before pneumothorax and lower cardiorespiratory reserve, that requires more aggresive lifesaving treatment.

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CONFLICT OF INTEREST

None

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REFFERENCE

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