

Pulmonary tuberculosis presenting as spontaneous pneumothorax in a young Nigerian

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Introduction

Although a rare but well-recognised complication, spontaneous pneumothorax complicating pulmonary tuberculosis (PTB)¹ is scantily reported in the literature. This may account for the limited information on its epidemiology.² A pneumothorax implies the presence of air in the pleural space. A spontaneous pneumothorax occurs without antecedent trauma to the chest wall. It may be primary or secondary. Primary spontaneous pneumothorax occurs in the absence of underlying lung disease while a secondary spontaneous pneumothorax occurs in persons with significant pulmonary disease. PTB remains an important cause of secondary spontaneous pneumothorax especially in the developing world.³

We present the case report of a young Nigerian male with PTB presenting as secondary spontaneous pneumothorax.

Case report

Mr O K U, a 34-year-old single cobbler residing in a city in south-east Nigeria, presented on 3rd January 2012 with cough of 3 months and breathlessness of 3 days' duration.

Cough was initially dry but quickly became productive of mucoid sputum. There was no haemoptysis, chest pain, orthopnoea, paroxysmal nocturnal dyspnoea, or trauma to the chest wall. There was a history of fever, drenching night sweats and weight loss. There was no significant history of cigarette or alcohol use. He lives in a one-room apartment with his elder brother who had been previously treated for tuberculosis (TB).

Physical examination revealed a chronically ill-looking young man in severe respiratory distress. He was febrile (temperature =37.9°C) moderately pale, with no peripheral lymph node enlargement. Chest examination showed tachypnoea, (respiratory rate 48 cycles/minute), trachea was deviated to the left with reduction in chest excursion and tactile fremitus over all zones of the right lung field. Percussion note was hyper-resonant with loss of hepatic dullness and breath sounds on the same side. On the left

lung field, there was bronchial breath sounds and coarse crepitations posteriorly over the upper and mid zones. Vocal resonance was reduced over the right lung field and increased over the upper and mid zones on the left. Cardiovascular exam revealed tachycardia with a pulse of 128 beats per minute, regular and normal volume, Blood pressure was 100/60 mmHg, jugular venous pulse was not elevated and precordium was quiet with only the first and second heart sounds heard. Examination of the other systems revealed no abnormality.

An urgent chest radiograph was requested and showed hyper-inflation of the right lung field devoid of pulmonary vascular markings with mediastinal shift to the left. Heterogeneous opacities were noted on the left lung field (see Figure 1). Laboratory investigations showed a packed cell volume of 27%, leucocytosis with absolute neutrophilia, markedly elevated erythrocyte sedimentation rate of 135 mm/hour (Westergren). Three consecutive sputum smears were positive for acid fast bacilli. HIV serology was negative while other tests, including liver function test and urinalysis, were normal.

A diagnosis of large secondary spontaneous pneumothorax was made. Patient received high flow intra-nasal oxygen, and a wide bore needle was immediately inserted into the second right intercostal space in the mid-clavicular line. A closed thoracostomy drain was subsequently inserted into the fourth intercostal space in the mid-axillary line. He commenced on standard anti-Koch's treatment comprising daily doses of rifampicin 600 mg, isoniazid 300 mg, pyrazinamide 1.2 g and ethambutol 800 mg for 6 months. The chest tube was removed after 6 days and a post-extubation chest radiograph showed re-expansion of the right lung with reticulonodular shadows in the paratracheal region (see Figure 2).

The patient showed good clinical improvement and was discharged to the Endemic Disease Unit.

Discussion

Primary spontaneous pneumothorax usually occurs in young persons with significant history of cigarette smoking but without evidence of underlying lung parenchymal disease. Most often, it results from the rupture of a sub-pleural bleb. Secondary spontaneous pneumothorax on the other hand, is associated with underlying pulmonary disease.⁴ The incidence of primary spontaneous pneumothorax is 18–28 and 1.2–6/100 000

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Figure 1 Chest radiograph showing right sided pneumothorax

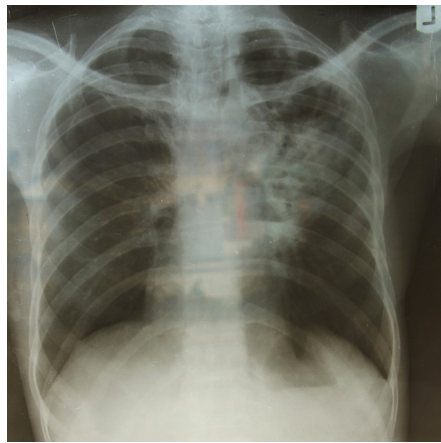


Figure 2 Chest X-ray after removal of chest tube

cases for males and females respectively, while that for secondary spontaneous pneumothorax is 5.8/100 000 cases for women and 16.7/100 000 cases for men.^{5,6} The male to female ratio is approximately 3.2: 1. Compared with primary spontaneous pneumothorax, secondary spontaneous pneumothorax occurs in the elderly, usually 60 years and above. This is explained by the presence of chronic airway diseases which are prevalent in that population. However, pneumothorax complicating PTB can occur at any age.⁷

PTB is the commonest cause of secondary spontaneous pneumothorax.⁷ Nigeria ranks fourth among the countries with the highest burden of PTB globally;⁸ nevertheless, there is scanty literature on the prevalence of secondary spontaneous pneumothorax. Spontaneous pneumothorax is thought to occur in 0.6–1.4% of patients presenting with pulmonary tuberculosis.⁹ Adebajo et al¹⁰ documented PTB as responsible for 90% of all non-traumatic pneumothorax over a 40-month period at University College Hospital, Ibadan, south-west Nigeria. This finding correlates with reports from other countries where PTB is endemic.¹¹

Several mechanisms have been postulated for the development of spontaneous pneumothorax in patients with TB.^{12,13} The commonest being rupture of a cavity into the pleural space. Occasionally, a broncho-pleural fistula may form following caseous necrosis and, rarely, a tubercular pneumatocele may rupture into the pleural space. Broncho-pleural fistula formation may be the most likely patho-physiological mechanism in our patient as a chest X-ray did not show cavitations and this has been a documented finding seen during surgery in Nigerian patients with tubercular pneumothorax.¹⁰

Secondary spontaneous pneumothorax usually occurs in association with active TB infection,¹² as was the case with our patient. They present with dyspnoea, cough, haemoptysis and are often malnourished. Sputum smear

for acid fast bacilli is often positive and cavitary lesions and reticulonodular opacities are seen on chest X-ray.

In spite of the acute and life-threatening presentation, patients respond favourably to treatment with high-flow oxygen, chemotherapy with anti-tuberculosis medications, surgical drainage with either needle insertion, or thoracostomy with chest tube drainage (or both) in line with the British Thoracic Society guidelines.⁶ Response to pleural drainage is as high as 85% and is the treatment of choice for symptomatic secondary spontaneous pneumothorax.¹⁴

In conclusion, PTB may present as spontaneous pneumothorax with acute

severe dyspnoea and may be missed with a catastrophic outcome. Although presentation may be confused with that of the other causes of acute dyspnoea, a high index of suspicion, thorough history and careful examination clinches the diagnosis and the response to treatment is often rewarding. Morbidity and mortality from this curable disease is thus reduced.

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