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Original Article

Case Report on Right Pulmonary Embolism with Pulmonary Hypertension and Cor Pulmonale with a Differential Diagnosis of Pulmonary Tuberculosis

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released into the lung vasculature. In patients receiving medical care, the frequency of pulmonary embolisms (PE) ranges from 0.14% to 61.5%, and the death rate from PE is between 40% and 69.5% (2). Pulmonary embolism is most frequently caused by thrombi from deep veins, especially in the lower extremities. Additional factors that might lead to pulmonary embolism include air embolus, amniotic fluid embolus, fat embolus, which is typically connected to the fracture of big bones, and deep vein thrombosis of the upper limbs, pelvis, renal, and from the right. The disease normally affects the lungs and may lead to Cor pulmonale and pulmonary hypertension. This short case report presents a 43-year-old male with a chronic history of cough productive of bloody sputum, weight loss, and a recent history of syncope. History and clinical examination findings were suggestive of pulmonary tuberculosis complicated by pulmonary

thromboembolism. Investigations done were suggestive of pulmonary embolism. Conclusion: An unusual presentation of tuberculosis and pulmonary embolism in

A pulmonary embolism is an acute emergency resulting from blood clots being

Keywords:

Pulmonary Embolism, Thrombus, Tuberculosis, Amniotic Fluid, Venous Thrombosis.

APA CITATION

a 43-year-old male in a tertiary hospital in Ghana.

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INTRODUCTION

Pulmonary embolism occurs when emboli, which are formed from blood clots in the veins, travel and block the arteries in the lungs. It is a life-threatening condition associated with venous thromboembolism. Failure to diagnose or treat pulmonary embolism promptly can result in experiencing mortality. **Patients** pulmonary embolism typically exhibit symptoms such as sudden chest pain, difficulty breathing, or fainting. According to CCM Ghana, the country's tuberculosis (TB) prevalence, estimated at 290 cases per 100,000 population, suggests a significantly higher disease burden compared to the World Health Organization's (WHO) estimated rate of 71 cases per 100,000 population for the same year. Utilising these revised figures, the calculated national TB case detection rate for 2013 is 20.7%(1). Paul Poku Sampene Ossei conducted a post-mortem study regarding venous thromboembolism, which showed a total of 150 cases of deep vein thrombosis and pulmonary embolism were included. The findings revealed that the average age of the patients was 45.3 years, with a standard deviation of 19.96. The age range varied from 3 to 96 years, with the most common age group being 31 to 40 years. Among the cases, males accounted for the highest number, with 92 (59.35%), while females comprised 63 cases (40.65%)(2).

Cor pulmonale, commonly known as pulmonary heart disease, is characterised by the enlargement and impaired function of the right ventricle due to underlying pulmonary conditions(3). This condition is a type of right-sided heart failure. Under normal circumstances, the right ventricle pumps deoxygenated blood into the pulmonary artery for

oxygenation in the lungs(3). However, various pulmonary disorders can cause an increase in pressure within the pulmonary artery, hindering the right ventricle's ability to transport blood efficiently to the lungs(3). This can be likened to the challenge of opening a car door in strong winds, where additional effort is required(3). As the right ventricle experiences this heightened workload, it may undergo hypertrophy, ultimately leading to a decline in its functional capacity(3). Cor pulmonale is estimated to comprise about 6% to 7% of all cases of heart disease among adults in the United States(4). The prevalence of cor pulmonale differs notably between various countries(4). Our team reports a case of pulmonary embolism with pulmonary hypertension and cor pulmonale, with a differential diagnosis of pulmonary tuberculosis

METHODOLOGY

This case report was conducted in the internal medicine ward at Komfo Anokye Teaching Hospital. Chest x-ray revealed homogeneous reticular opacifications. A venous Doppler ultrasound of both lower limbs showed no deep vein thrombosis. CT scan of the chest revealed a right pulmonary embolism with pulmonary hypertension. The patient was treated with enoxaparin, IV cefuroxime, tab azithromycin, and 2L IVF. Further treatment modalities administered during the patient's stay at the ward were anti-tuberculosis medication, specifically HRZE, pyridoxine, tab rivaroxaban (Xarelto) 15 mg BD, and IV furosemide.

CASE REPORT

A 43-year-old male with no known chronic illness was well until about 4 years ago when he started coughing. The cough was productive of blood-

stained sputum. The cough was consistent throughout the 4-year period and was mostly worse, especially when he talked. On account of the blood-stained sputum cough of about 4 years, he had been visiting a peripheral hospital for management and treatment until three weeks ago, when he was referred to the tertiary hospital on account of tuberculosis. The patient was placed on antituberculosis medications (rifampicin, isoniazid, pyrazinamide, ethambutol), IV rocephin 2 grams twice daily, and tab azithromycin 500mg once daily. He collapsed at home and was rushed to the peripheral hospital, where he was subsequently referred to KATH for the continuation of medications.

The patient revealed that he had developed a fever and lost weight, but he could not specify the duration of the symptoms. There were no night sweats, chest pain, palpitations, dizziness, or other symptoms or signs of clinical relevance. There was no family history of any chronic illness. On examination, the patient was conscious and alert and had a GCS of 15/15. The patient looked chronically ill and was on a non-rebreather mask. He was not pale, anicteric, or afebrile, but he had developed bilateral pitting pedal edema up to the level of both knees. Other significant examination findings were reduced air entry on both lungs with bronchial breath sounds accompanied by coarse crepitations at the right upper and left lower lung zones, with rhonchi generally heard. The first and second heart sounds were heard in all auscultatory areas, with no murmurs heard. The heart rate was 128 cycles per minute, and no abnormal rhythm was noticed. The abdomen was scaphoid, moved with respiration, soft, non-tender, and had no organomegaly. Chest x-ray revealed homogeneous reticular opacifications.

A venous Doppler ultrasound of both lower limbs showed no deep vein thrombosis. CT scan of the chest revealed a right pulmonary embolism with pulmonary hypertension. An impression of acute respiratory distress with cough, with a differential diagnosis of pulmonary tuberculosis and pulmonary embolism with pulmonary hypertension, was made. The patient was treated with enoxaparin, IV cefuroxime, tab azithromycin, and 2L IVF. Further treatment modalities administered during the patient's stay at the ward were anti-tuberculosis medication, specifically HRZE, pyridoxine, tab rivaroxaban (Xarelto) 15 mg BD, and IV furosemide. On admission, the patient's oxygen concentration was 78% on room air, on oxygen via a non-rebreather mask at 15L/min, and oxygen therapy was provided. Further investigations revealed a HB-13.0g/dL MCV-75.3 MCH-25.7 MCHC-34.1 Platelet-248 (10³/uL) WBC-7.56 (10³/uL) Lymph-1.32 (17.5%) Mono-0.53(7.0%) Neutrophils-5.63 (74.4%) ESR-13mmfall/hr. The renal function test showed urea-6.80mmol/L creatinine- 64mmol/L bun/creatinine ratio-49.6 sodium-137mmol/L potassium-4.6mmol/L chloride-102mmol/L and liver function test revealed AST-51.8u/l ALT-40.4u/l ALP-134u/l GGT-44u/l total protein-57.8g/l albumin- 27.3 g/l globulin-30.5g/l bilirubin-4.3umol/L. All serology (HIV, HEPB, HCV) tests were negative. On the sixth day of admission, the patient continuously had respiratory distress; oxygen concentration was 79% on room air, and Saturation was 79% on oxygen. On examination, air entry was reduced, breath sound intensity was bronchial, and there were no added sounds. The day the patient was noticed to have developed cardiorespiratory arrest, and on the 7th day, was not responsive to treatment.

DISCUSSION

This is the first known reported case of pulmonary embolism in a patient with chronic lung infection (TB) seen in our hospital. It is an unexpected finding of tuberculosis and pulmonary embolism in a patient who had only TB as a risk factor and did not have other classical risk factors for pulmonary embolism. The patient, though chronically ill, was ambulant and was considered to be unlikely to develop pulmonary embolism despite the presence of TB. The clinical clues that supported PE in the

patient who potentially could suggest PE were the occurrence of syncope and lower limb edema. Although the Doppler ultrasound of the lower limbs did not reveal deep vein thrombosis, the findings of an embolus in the pulmonary vascular system signalled that a thrombus had formed and was released into the pulmonary vasculature, leading to sudden shortness of breath and syncope. The shortfall in the case was the failure to perform a post-mortem to appreciate the pathological changes in the lungs and also to ascertain the thrombus revealed on the CT scan.

Delays in diagnosing pulmonary tuberculosis in a 43-year-old adult with tuberculosis, however, presenting to a peripheral hospital with chronic cough and weight loss, were sufficient to suspect chronic infections like TB. There are global reports of tuberculosis and pulmonary embolism, and these cases reinforce the occurrence of the disease. Amitesh Gupta et al. reported three cases of pulmonary tuberculosis in patients with pulmonary embolism. Further reported that about 25%-50% of patients with first-time venous thromboembolism (VTE) have an idiopathic condition, without a readily identifiable risk factor. Further stated that the occurrence is rare in patients with tuberculosis (TB), stating studies have reported TB and Deep venous thrombosis incidence between 1.5%-3.4%. Early diagnosis and prompt initiation of anti-TB treatment, along with anticoagulant therapy, potentially reduce the risk of mortality. Our patient was a chronically sick and delayed-diagnosed TB patient who developed VTE in the early phase of treatment.

Bishav Mohan reported pulmonary embolism in patients with pulmonary tuberculosis. A total of 5 cases were studied. Venous Doppler was done in all 5 cases, and two of the five cases had developed a large thrombus in the left iliofemoral vein and a large femoral venous thrombus in the left leg. An echocardiography was done in three case studies, the first case revealed a dilated right atrium & ventricle, moderate tricuspid regurgitation,

pulmonary artery hypertension, and an intracardiac vermicular echogenic with a mass attached to the interatrial septum. The second case had a dilated right atrium & ventricle, Pulmonary artery hypertension, and the third case had a dilated right atrium & ventricle, Pulmonary artery hypertension. A pulmonary angiography was done in all five cases, and results revealed that a thrombus was found in the arteries, and one case had a bilateral pulmonary embolism. All the cases involved in this were also confirmed diagnoses tuberculosis. In one of the studies, a left-sided pleural effusion was seen, and in another case, the patient developed cervical lymphadenopathy.

The other three cases involved in this case report diagnosed with miliary tuberculosis, pulmonary tuberculosis. and pulmonary tuberculosis with right-sided pleural effusion(5). Nkoke Clovis Ekukwe et al. reported a case in Cameroon involving a 52-year-old male patient with no identifiable risk factors for thromboembolic disease(6). He was referred for a detailed clinical assessment due to cardiomegaly and exertional dyspnea(6). Echocardiography and computed tomography (CT) scans revealed dilated heart chambers and bilateral proximal pulmonary emboli, along with a cavitary lesion in the apical region of the right lung(6). Bronchial aspiration and culture identified Mycobacterium tuberculosis, and no evidence of malignancy was found(6). A clinical review and a lower limb ultrasound ruled out deep vein thrombosis(6). Following the initiation of antituberculosis and anticoagulant therapies, the patient exhibited a favourable clinical response(6). This case underscores the importance for clinicians to be aware of the potential for thromboembolic events in patients being treated for tuberculosis, especially in areas with high prevalence(6). Amitesh Gupta et al. performed a similar study with three case reports that were diagnosed with pulmonary tuberculosis with venous thromboembolism.

A Doppler ultrasound was done for the first case, revealing deep venous thrombosis in the left

saphenofemoral vein. The second case showed a thrombosis of the popliteal vein of the right lower limb, and the third case had a thrombus in the right popliteal vein with a thickening of venous valves in lower limbs(7). Kulatunga Mudiyanselage Pramitha Prabhashini Kumarihamy et al. presented a case report on a rare instance of severe pulmonary tuberculosis. This report highlights the potential risk of thromboembolism development associated with the disease(8). Further, a study by Hyerim Ha examined a cohort of 7905 patients with tuberculosis. Among these patients, pulmonary thromboembolism and deep vein thrombosis were observed. Notably, approximately 44% of the patients in the study had evidence of thrombosis at the time of tuberculosis diagnosis(9). Haris S conducted a case study of a 55-year-old female patient who exhibited symptoms of cor pulmonale with a background of chronic obstructive pulmonary disease (COPD) and recent severe pulmonary artery hypertension(10).

The patient presented with anasarca and elevated brain natriuretic peptide (BNP) levels, indicating right ventricular failure related cor pulmonale(10). The treatment regimen included diuretics, phosphodiesterase-5 (PDE-5) inhibitors, anticoagulants, bronchodilators, oxygen therapy, and antibiotics(10). The patient's condition improved significantly, leading to her discharge from the hospital(10). Jussara de Almeida Bruno reported a case study of a 28-year-old female patient who presented with dyspnea and a dry cough after being diagnosed with pulmonary thromboembolism (PTE) one year earlier(11). Initially symptom-free, she later experienced syncope, prompting her admission for PTE treatment(11). Imaging findings revealed significant occlusion of the pulmonary artery and indications of right ventricular failure, including edema and low oxygen saturation(11). Despite receiving anticoagulant therapy, her condition worsened, resulting in notable weight loss and increased dyspnea(11). She ultimately required orotracheal intubation due to severe hypoxemia(11). Echocardiographic evaluations indicated right ventricular dysfunction and elevated pulmonary artery pressure(11).

Unfortunately, the patient suffered cardiac arrest shortly after intubation and did not survive(11). This case illustrates the complications associated with chronic PTE in younger individuals and emphasises the importance of early surgical assessment and management of ongoing pulmonary hypertension(11). Ana M. Aristizabal reported a case of a 17-year-old male with no significant medical history who experienced two years of progressive exertional dyspnea, hemoptysis, and palpitations(12). Echocardiography moderate right atrial dilation and interventricular septum deviation, indicating elevated ventricular pressure (112 mmHg) and mild pulmonary hypertension (mean pressure of 20-25 mmHg)(12). Computed tomography angiography showed a chronic thrombus in the main pulmonary scintigraphy artery, confirmed by angiography(12). Diagnostic cardiac catheterisation demonstrated increased pulmonary vascular resistance and systemic pulmonary hypertension(12).

After developing acute respiratory distress postcatheterisation. the patient underwent thromboendarterectomy, revealing multiple thrombi in the pulmonary branches(12). surgery, the patient was discharged without supplemental oxygen and improved to NYHA functional class II(12). This case underscores the rarity of chronic thromboembolic pulmonary hypertension (CTEPH) in pediatric patients and highlights the need for early diagnosis and intervention(12). Despite no identifiable risk factors, it emphasises maintaining a high suspicion for pulmonary vascular disease in similar cases(12). Yvonne Walker et al. reported a case involving a female patient in her forties who presented with a sudden onset of continuous dyspnea that intensified over four hours(13). This dyspnea was associated with palpitations and diaphoresis(13). Initial assessments using clinical scoring tools suggested a

low pre-test probability for pulmonary embolism (PE)(13). The patient had no significant risk factors for PE other than obesity(13). Laboratory findings indicated a troponin level of 0.10 ng/mL, a D-dimer level of 8.10 µg/mL, and a B-type natriuretic peptide (BNP) level of 1,160 pg/mL(13). Computed tomography angiography (CTA) revealed extensive pulmonary emboli in the main and segmental pulmonary arteries, accompanied by evidence of right heart strain(13). The patient was started on unfractionated heparin with a loading dose in the emergency department, followed by a continuous infusion(13). She was then transferred to a tertiary care centre for further management(13). Early identification and treatment of submassive PE are essential for improving patient outcomes regarding mortality and morbidity(13).

CONCLUSION

In summary, this case marks the first reported occurrence of pulmonary embolism in a patient with chronic tuberculosis, revealing an unexpected link between these two conditions. Although the patient lacked conventional risk factors for pulmonary embolism, her chronic illness and symptoms, such as syncope and lower limb edema, warranted further examination. The discovery of extensive pulmonary emboli and elevated biomarker levels highlights the importance of vigilance in patients with chronic lung infections. While the association between venous thromboembolism and tuberculosis is acknowledged, this case underscores the critical need for early diagnosis and treatment. Initiating anticoagulation and anti-TB therapy promptly can help reduce mortality and morbidity associated with these complications. The lack of a post-mortem examination represents a missed opportunity to clarify the underlying pathological changes, emphasising the need for thorough diagnostic evaluations in similar situations. Overall, this case underscores the necessity for increased awareness of thromboembolic risks in tuberculosis patients and the importance of timely interventions to enhance patient outcomes.

Recommendation

Regular screenings for pulmonary embolism in patients with tuberculosis, particularly those showing respiratory issues. Develop prompt imaging protocols and encourage collaboration among healthcare specialists for effective management. Inform patients about the signs of PE and support post-mortem studies to enhance understanding and treatment approaches.

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