

Pulmonary artery thrombosis in a patient with right-sided heart failure

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Abstract

Pulmonary thromboembolism occurring either abruptly or insidious poses a greater challenge in diagnosis. A high index of suspicion is required to proceed with proper investigations in patients with nonspecific cardiac or respiratory presentation to make the diagnosis of pulmonary embolism (PE). Early diagnosis of PE with prompt initiating of anticoagulation therapy has been proven to have a positive impact in mortality reduction associated with recurrent episodes of this condition. We present a case of a 76-year-old man, known to have cardiac failure on regular treatment who presented with predominant features of right-sided heart failure accompanied with dizziness. He was diagnosed to have pulmonary artery thrombosis by computerized tomography. Anticoagulation therapy was initiated with marked clinical improvement.

Key words: Chronic pulmonary artery thrombosis, computerized tomography, right-sided heart failure

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Introduction

Pulmonary embolism (PE) is a very common condition associated with lethal outcomes in all age groups.^[1] Untreated around one-third of patients who survive initial PE succumb to future embolic events. Most of the patients with PE are initially completely asymptomatic, and those with symptoms tend to have atypical presentation.

The classic triad of symptoms and signs in patients with PE (hemoptysis, dyspnea, and chest pain) are neither sensitive nor specific. They occur in fewer than 20% of patients with such clinical manifestations, and most often there is another diagnosis to account for them.^[1]

Studies have shown a significant improvement in mortality outcomes in patients with PE from approximately 30% to around 2–8% when accurate diagnosis is made, followed by effective anticoagulation therapy.^[2,3]

Deep venous thrombosis (DVT) of the lower extremities is the primary source of thrombi in up to 90% of cases of PE. Thrombi may also originate in the pelvic, renal, upper extremity, and in the right heart.^[4]

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This case is presented due to its uniqueness compared to other cases reported in Africa and rest of the world.^[5-7] Majority of previously reported cases presented acutely and had a DVT as a primary source of pulmonary thromboembolism. The patient in this case report had predominant features of right-sided heart failure over a period of 3 months. The aim of authors is to highlight factors other than DVT that may attribute to pulmonary thromboembolism, emphasizing need for high index of suspicion to enhance evaluation with computerized tomography (CT) chest which has a higher diagnostic value than echocardiogram in heart failure patients.

Case Report

A 76-year-old man known to have chronic heart failure since 2 years ago was admitted with a history of 3 months dizziness. The dizziness was reported to be postural (experienced sitting up from bed) with occasional palpitations, there

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was no history of vertigo. He denied history of orthopnea or paroxysmal nocturnal dyspnea in the past 3 months. Patient's functional status was the same as prior to current symptoms (New York Heart Association (NYHA) II).

There was no history of cough or chest pain. He also noted that his lower limbs were progressively swelling over the past 3 months. He denied history of fevers and night sweats; there was no history of contact with a person with chronic cough or a person known to him to have tuberculosis.

His past medical history was significant for suffering from pulmonary tuberculosis in 1995 from which he completed a 6-month course and declared cured, no history of hypertension, diabetes mellitus, or asthma. He was a reformed smoker and heavy alcohol drinker of more than 20 years who quit both alcohol drinking and cigarette smoking 7 years ago. Drug history included antifailure medications; furosemide 40 mg BID, enalapril 5 mg OD, spironolactone 25 mg OD, and carvedilol 6.25 mg BID. He was adhering well to his heart failure medications.

General examination was not significant for jaundice, cyanosis, or any evidence of anemia, he had bilateral lower limb pitting edema that extended to the mid-tibia. There was no obvious respiratory distress. He was afebrile, respiratory rate-19 breath/min, pulse rate-95 bpm, regular with good volume, and blood pressure was 121/63 mmHg sitting and 99/51 mmHg standing. Systemic examination was significant for raised jugular venous pressure (6 cmH₂O), displaced apex at sixth intercostal space anterior-axillary line, left-sided middle and upper zones coarse inspiratory crackles with reduced breath sounds on the same areas.

Abdominal examination was consistent with a smooth tender hepatomegaly with moderate ascites. The rest of other systems examined were unremarkable.

Laboratory investigations showed; hemoglobin = 11 g/dl, mean corpuscular volume (MCV) = 88.5 fl, white blood cell (WBC) = 4.06 k/UL, platelet = 302 × 10⁹/l, human immunodeficiency virus (HIV) serology = negative. Liver enzymes and renal functions were within normal limits. Coagulation profile: Prothrombin time = 14.6 s (11-16 s), partial thromboplastin time = 46.5 s (25-45 s), international normalized ratio (INR) = 1.32. Patient's lipid profiles were consistent with normal triglycerides and total cholesterol. High density lipoprotein (HDL) levels was low at 0.56 (normal > 1 mmol/l).

Protein C, protein S, Leiden V factor, anticardiolipin antibodies, and B-type natriuretic peptide (BNP) are not routinely done at our facility. DVT of both lower and upper extremities was ruled out by Doppler scans. Chest X-ray revealed opacity in the left upper and mid-zone [Figure 1].

Electrocardiogram (ECG): Sinus rhythm, normal axis, negative T waves in lead II, III, avF, and V4-V6. Atrial and ventricular ectopic [Figure 2].

Echocardiogram revealed bilateral atrial and ventricular chamber dilatation, with moderate tricuspid regurgitation, mild mitral regurgitation with left ventricular (LV) ejection fraction of 46%. Pulmonary artery systolic pressure was within normal limit. CT of the chest showed a large isodense poorly enhancing mass in the lumen of the left pulmonary artery causing luminal narrowing. There was left upper lobe collapse secondary to narrowing of the left main bronchus, marked left lung volume loss secondary to fibrosis, and left upper lobe collapse.

Conclusion

Pulmonary artery thrombus and fibrosis left lung secondary to old tuberculosis [Figures 3 and 4].



Figure 1: Chest X-ray, posterior-anterior view showing opacity in the left middle and upper zones



Figure 2: Electrocardiogram of the patient



Figure 3: Non-contrast computerized tomography showing left lung collapse and fibrosis

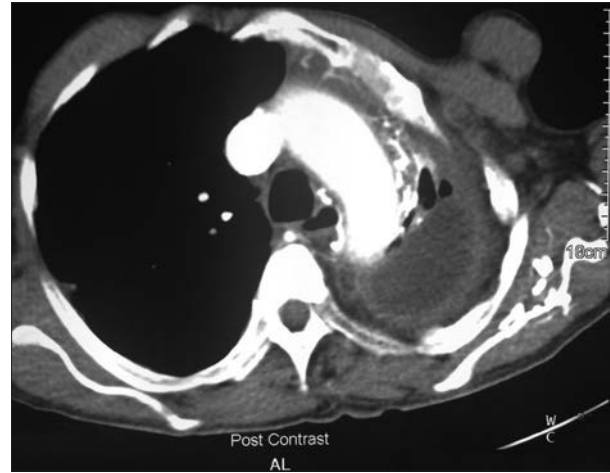


Figure 4: Contrast enhanced CT showing pulmonary arterial thrombus

Patient treatment was optimized on antifailure regimen and anticoagulated with clexane 60 mg subcutaneous (SC) BID initially followed by warfarin, his condition improved gradually with features of right-sided heart failure subsiding. He was discharged with INR of 2.6 on warfarin 5 mg OD after 2 weeks of hospital stay to be followed at medical clinic.

Discussion

Several risk factors have been attributed for both DVT and PE; the most important ones being a prior history of DVT or PE, recent surgery, prolonged immobilization, or underlying malignancy.^[1]

Other recognized risk factors for thromboembolic disease are chronic heart failure, old age, reduced high density lipoprotein (HDL), and parenchymal lung disease;^[1,8,9] all of which were present in our patient.

The relative risk of PE in heart failure patients is at least double that of patients without heart failure and increases as LV systolic function declines.^[10]

In conclusion, clinicians should be alerted to patients with new or worsened right more than left heart failure such as lower limb edema, hepatomegaly, and raised jugular venous pressure in absence of significant basal

crepitations as these may indicate presence of subacute or chronic PE.

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