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Case Report

An Atypical presentation of pulmonary embolism in a critically ill patient☆,☆☆

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ABSTRACT

A pulmonary embolism (PE) occurs when a venous thrombotic material from the lower extremities embolizes to the pulmonary vasculature. Common presenting symptoms include shortness of breath and pleuritic chest pain with vital signs demonstrating hypoxia, tachycardia, and tachypnea. In this paper, we describe a unique presentation of a critically ill patient who developed a saddle pulmonary embolism despite being on prophylactic anticoagulation.

Introduction

A pulmonary embolism (PE) occurs when venous thrombi dislodge from the lower extremities and causes a downstream obstruction in the branching pulmonary vasculature. It is associated with a significant mortality burden and continues to be the third most common cause of death from cardiovascular disease [1]. The incidence of PE diagnoses has increased with increased use of computer tomographic pulmonary angiography, from 62 per 100,000 cases in 1998 to 120 per 100,000 cases in 2016 [2]. These deaths may be attributed to the fact that patients often have comorbidities, such as cancer or sepsis, as well as due to post-treatment complications such as bleeding from anticoagulation, recurrent venous thrombotic events, pulmonary hypertension, and long-term psychological stress [3]. In this case, we describe a unique presentation of pulmonary embolism, in which clinical suspicion was initially low, due to the presence of multiple seemingly plausible explanations (Fig. 1).

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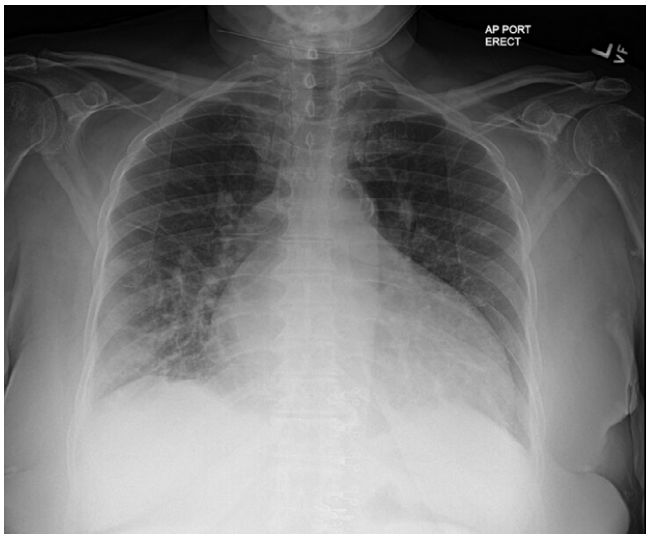

Fig. 1 – Chest radiograph (day 1 on admission) with ground glass air opacities in the right lung base.

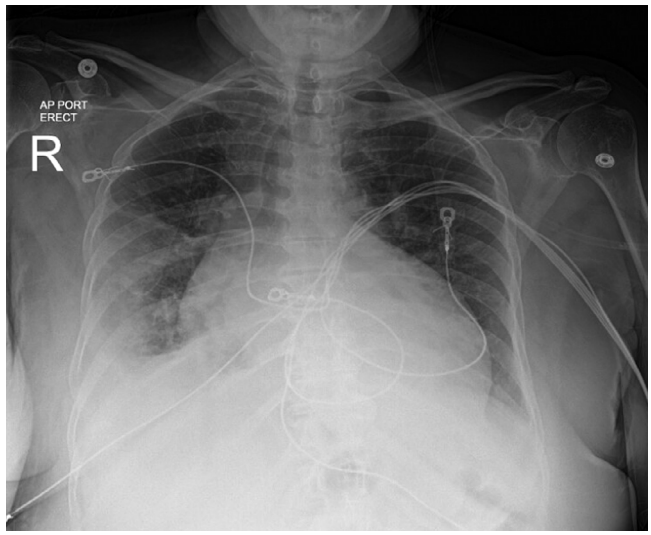

Fig. 2 – Repeat chest radiograph (day 4 of admission) demonstrating increased density of air opacities in the right lung base.

Case report

A 66-year-old female with a past medical history of hypertension, hyperlipidemia, diabetes mellitus type 2, and tobacco dependence presented to the emergency room complaining of shortness of breath, headache, productive cough with clear sputum and a small amount of bright red blood, as well as fever and nausea. On exam, her blood pressure was slightly elevated at 133 systolic and 80 diastolic, she was tachycardic at 130, she was febrile at 101.3°F (38.4°C), her respiratory rate was normal at 19 breaths per minute, and her oxygen saturation was normal at 98% on room air. She was ill appearing and had rhonchi and decreased breath sounds in the lower lung fields bilaterally. Her white blood cell count was elevated at 12 K/uL (normal range: 4-11 K/uL), hemoglobin was slightly low at 11.5 (normal range 11.7-16.1 g/dL), and proBNP was increased at 6344 (normal range: less than or equal to 125 pg/mL). Urinalysis demonstrated negative nitrites, moderate leukocyte esterase, 10-20 white blood cells, and rare bacteria. Chest radiograph revealed ground glass opacities in the right lung base (Fig. 1). The patient's presentation was attributed to sepsis secondary to urinary tract infection or pneumonia. Echocardiogram revealed an ejection fraction of 11% (normal range greater than 55%) and right ventricular systolic dysfunction. Her symptoms were then believed to be secondary to acute systolic heart failure rather than pneumonia and her antibiotic was modified to only treat possible urinary tract infection. The patient later developed altered mental status and arterial blood gas was ordered, which revealed respiratory alkalosis, with a pH of 7.467 (normal range: 7.35-7.45), a pCO2 of 31.5 mm Hg (normal range: 34-45 mm Hg), a pO2 of 49 mm Hg (normal range: 80-100 mm Hg), a HCO3- of 22.8 (normal range: 22-26 mm Hg), and an arterial O_2 saturation of 88%. Her symptoms improved on nasal cannula and cardiology planned for a coronary angiogram to evaluate for coronary artery disease, however this was canceled due to development of new onset atrial fibrillation. She was found to have a decreased thyroid stimulating hormone of less than 0.01 mcU/mL (normal range: 0.27-4.20 mcU/mL) and an increased free T4 of 3.5 ng/dL (normal range: 0.9-1.8 ng/dL). She was started on methimazole per endocrinology and beta blockers were started for rate control per cardiology. Her white blood cell count increased to 16.8 K/uL (normal range: 4-11 K/uL) and repeat chest radiograph demonstrated slightly increased opacity in the peripheral right midlung zone (Fig. 2). Therefore, her antibiotic regimen was further modified to again cover pneumonia. The patient remained persistently hypoxic and tachycardic, and repeat chest radiograph was obtained, which revealed a wedge-shaped peripheral opacity in the right middle lung, concerning for pneumonia or pulmonary embolism (Fig. 3). Computer tomography (CT) of the chest was ordered for further evaluation, which revealed dome shaped opacities in the right middle and right lower lobes, which could represent pneumonia or pulmonary infarction (Fig. 4). D-dimer performed at the time was 3.27 mg/L (normal range: 0-1.2 mg/L). Computer tomographic pulmonary angiogram (CTA) revealed saddle pulmonary embolus with lobar and segmental pulmonary emboli bilaterally, right greater than left (Fig. 5A–C).

Discussion

Pulmonary embolisms have diverse presentations, ranging from largely asymptomatic to the presence of more characteristic symptoms. It is crucial to consider pulmonary embolism in the presence of consistent symptoms, clinical findings, and risk of venous thromboembolic disease [4]. When considering the risk for venous thromboembolic disease, it is important to consider Virchow's triad of blood hypercoagulability, venous

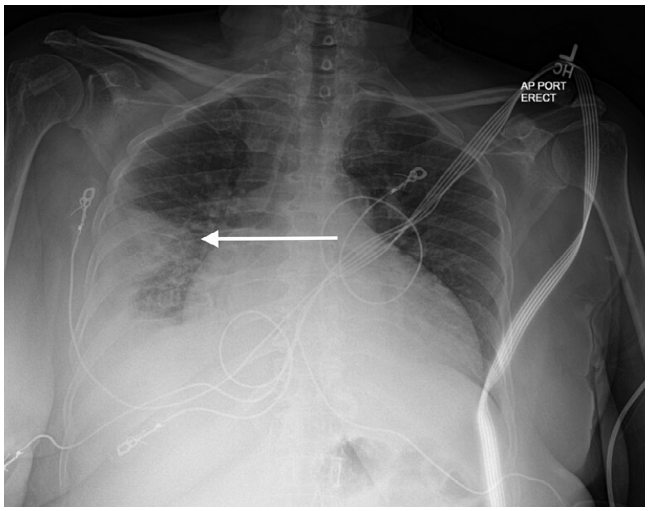

Fig. 3 – Follow up chest radiograph (day 5 of admission) showing wedge shaped peripheral opacity in the right middle lung (white arrow) with associated effusion and atelectasis, concerning for pneumonia or pulmonary embolism.

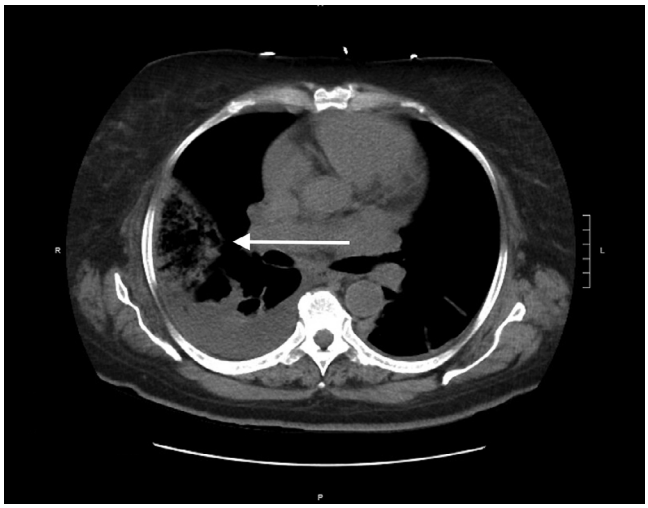

Fig. 4 – Computer tomography of the chest (day 5 of admission) revealing dome shaped opacities in the right middle and right lower lobes (white arrow), which could represent pneumonia or pulmonary infarction. Bilateral, right worse than left, pleural effusions are also noted.

stasis, and endothelial damage [5]. Common presentations include immobilized patients who are hypoxic, tachycardic, and complain of sudden onset severe pleuritic chest pain and dyspnea. Patients with high risk of death may also present with syncope, hemodynamic instability, cardiac arrest, shock, or hypotension [6].

Clinical consideration of PEs may be delayed if presentation is atypical, such as in our patient, who uniquely presented

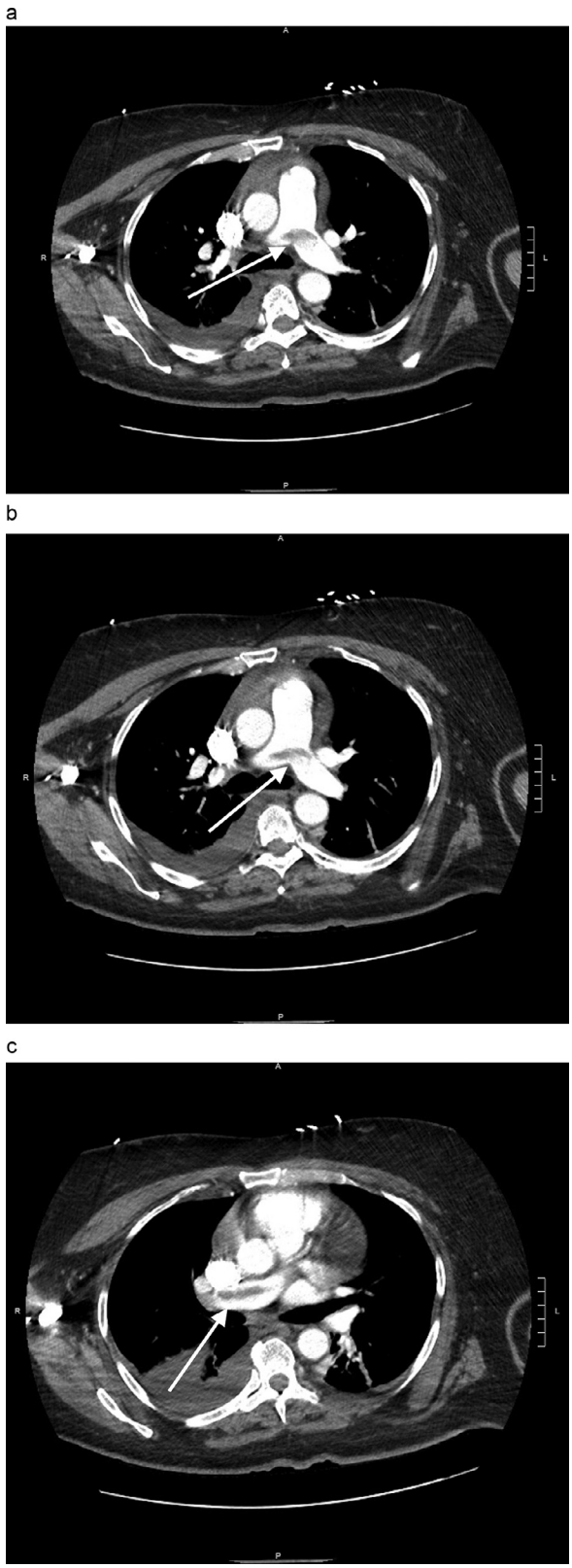

Fig. 5 – (A–C) Computer tomographic pulmonary angiography (day 5 of admission) revealing massive saddle pulmonary embolus (white arrow). Bilateral, right worse than left, pleural effusions are also noted.

with initial complaints of productive cough with leukocytosis and reduced ejection fraction, and developed altered mental status due to respiratory alkalosis, atrial fibrillation, persistent hypoxia, and tachycardia later in the course of her hospitalization. Diagnosis may further be delayed if multiple comorbidities exist, such as in our patient, where her systolic heart failure, underlying pneumonia, and hyperthyroidism seemed to plausibly explain her presentation, and further investigation was prompted by the patient's lack of clinical improvement despite treatment efforts. It is also important to note, that the absence of pleuritic chest pain should not prevent consideration of a PE, as seen by the complete absence of pleuritic chest pain throughout our patient's clinical course. Clinical suspicion of a PE was also low due to our patient being on prophylactic anticoagulation. However, patients may still develop PEs despite being anticoagulated, termed anticoagulation failure, and further hematological work up is indicated in these patients [7].

Chest radiographs, while nonspecific, may assist in excluding other causes of chest pain and dyspnea. In rare cases, such as in our patient, a wedge-shaped consolidation mimicking pneumonia may be seen. This is colloquially referred to as Hampton's Hump sign [8]. Approximately 25% of patients with PEs have normal EKG findings. However, those with EKG abnormalities typically have sinus tachycardia, P pulmonale, atrial fibrillation, or right axis deviation. S1Q3T3 may also be seen in some patients, which describes a large S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III, together which represent acute right heart strain [9].

D-dimer levels may exclude pulmonary embolism; however, it must be qualified in patients with baseline elevations. Echocardiograms may show evidence of right ventricular dysfunction; however, these findings may be attributed to other cardiovascular diseases or may be incidentally found in otherwise healthy patients. Specific echocardiographic findings, such as the 60/60 sign, McConnel sign or direct visualization of right heart thrombotic material are rarely seen, but when present, are diagnostic for PE [4]. CTA is the gold standard of diagnosis, as it allows direct visualization of the thrombosed vessels, while also ruling out aortic dissections and myocardial infarctions, which may present similarly. Ventilation perfusion scans examine lung areas that remain ventilated without adequate perfusion, but use is limited by high cost and frequency of inconclusive results [10].

Therapeutic anticoagulation is the primary treatment for patients with an acute PE, because it significantly improves patient outcomes. First line therapy typically involves anticoagulation with low molecular weight heparin followed by novel oral anticoagulants with apixaban or rivaroxaban for at least 3 months [10]. In patients with contraindications to thrombolysis, percutaneous mechanical thrombectomy may be considered [11].

Patient consent

The authors obtained informed consent from the patient whose case was discussed in this report.

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