CASE REPORT

A Rare Coexistence of Pneumonia and Pulmonary Infarct with a Normal D-dimer Acute Pulmonary Thromboembolism: A Case Report

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Abstract

Introduction: Although pneumonia is associated with an increased risk of venous thromboembolism, patients with pulmonary embolism (PE) and concomitant pneumonia are uncommon. In this case report, we present a 40-year-old male patient with pneumonia and PE in whom pneumonia initially masked the diagnosis of PE. Moreover, pneumonia may occasionally mask PE, particularly in patients with predominant systemic symptoms such as fever, and with no evidence of deep vein thrombosis (DVT) or trauma.

Case presentation: This patient who is an industrial worker, smoker and alcoholic presented to the ER with acute left lower chest and upper abdominal pain, shortness of breath, high grade fever and cough with hemoptysis. On examination pulse rate was 118 and normal oxygen saturation. He was admitted to the intensive care unit (ICU) and managed conservatively. His ultrasound chest and chest X-ray showed left lower lobe consolidation with effusion. His Wells score is 2.5, and D-dimer is normal, but total leucocyte counts and hemoglobin are elevated. There no signs of DVT.

He was managed with intravenous antibiotics, antipyretics. He has no signs of hemodynamic instability or hypoxia. He had persistent blood clots in sputum, which warranted computed tomography (CT) pulmonary angiography. Acute pulmonary thromboembolism (PTE) with pulmonary infarct (PI) and concomitant consolidation with pleural effusion is reported. Venous Doppler study of both lower limbs was negative for deep venous thrombosis except for a slow flow vascular malformation in the left distal thigh. He was started on anticoagulation with low molecular weight heparin as per treatment guidelines for 6 days, later he improved and had no hemoptysis or any bleeding.

Further investigations for hypercoagulable state done that showed high levels of serum homocysteine and low folic acid. With the advice of oral anticoagulation and folic acid supplementation, he was discharged from the hospital in a stable condition. This case is a rare entity of normal D dimer PTE with PI.

This case highlights the importance of considering PE in patients with pneumonia when there would be an initial therapeutic response followed by a worsening of the condition during the treatment of pneumonia.

Conclusion: When approaching an otherwise young and healthy individual with unexplained pleuritic chest pain or hemoptysis in the emergency department, a pulmonary infarction (PI) complicating an acute PE should be considered as a possible diagnosis among other conditions (e.g., pneumonia, lung neoplasia, lung granulomatous disease), when there is a strong clinical suspicion.

Keywords: Case report, Cough, Effusion, Hemoptysis, Normal D-dimer, Pneumonia, Pulmonary embolism, Pulmonary infarct, Pleural effusion.

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Case Description

A 40-year-old male person who is a mechanical worker in industry setup came to the emergency room on 26th April 2023 with complaints of high-grade fever since afternoon, acute left lower chest and upper abdominal pain since morning, and shortness of breath since 1 day. He had blood in his sputum in the form of clots for 1 day and cough for 1 week.

His fever has been high grade since afternoon, continuous, not associated with chills, and no headache. The chest pain is catchy type, severe, and increases on taking a breath, moving, and coughing. His shortness of breath was since 1 day, sudden in onset, modified Medical Research Council Dyspnea Scale grade 4, persistent, not relieved by anything. His cough was since 1 week initially nonproductive, later associated with sputum with blood clots looking like red currant jelly, around 100 mL/day, and was not foul smelling in nature (Fig.1). He denied any history of travel, trauma, palpitations, giddiness, or sweating.

In the past history, there was no diabetes, hypertension, tuberculosis (TB), pneumonia, or coronavirus disease of 2019

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Conflict of interest: None

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Fig. 1: Clots mixed with sputum

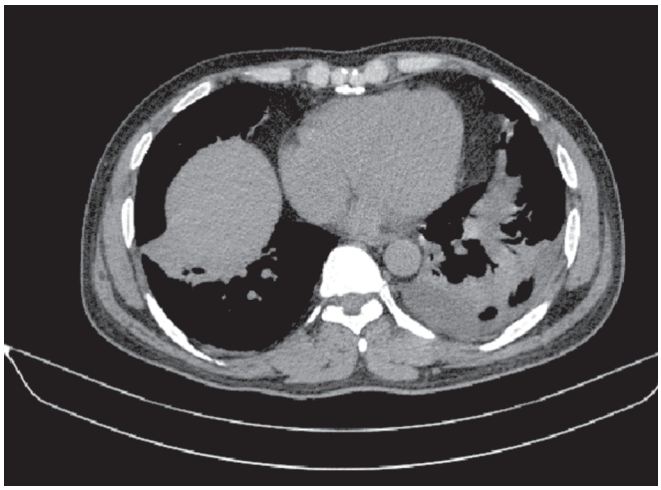

Fig. 3: High resolution computed tomography (HRCT) chest plain

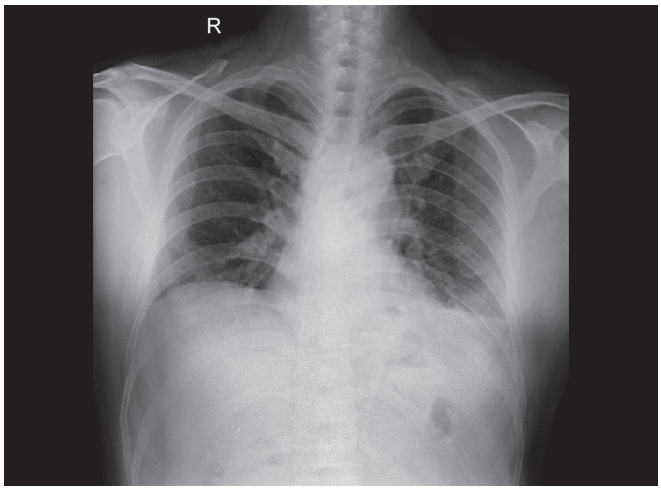

Fig. 2: Chest X-ray

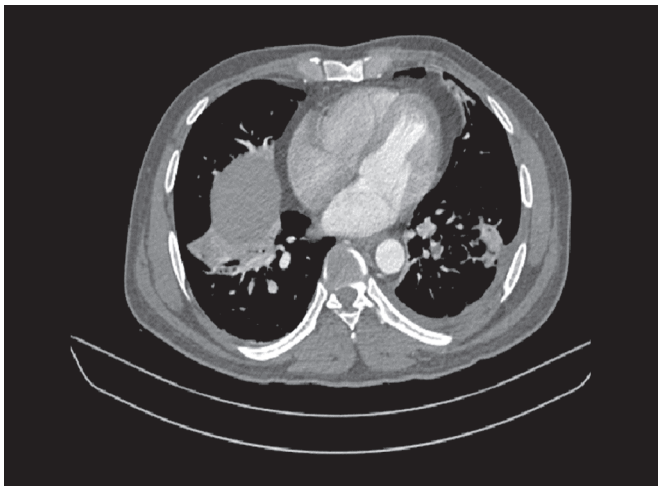

Fig. 4: CTPA with filling defects

(COVID-19). He has complained of mild left thigh pain while cycling and climbing stairs for 3–4 years. He has been a chronic smoker for 10 years, with 10 cigarettes per day and a chronic alcoholic. His bowel and bladder habits are regular.

On general examination, he was moderately built and well nourished, conscious, coherent, and oriented to place, person, and time. His pain score was 8. He had digital clubbing in grade 3. There was no pallor, jaundice, peripheral lymphadenopathy, and pedal edema. There are no signs of deep venous thrombosis. His wells score was 2.5, which suggested a low pretest probability of pulmonary embolism (PE). His temperature was 101.5°F, BP was 150/90 mm Hg, and pulse rate was 118 beats per minute.

On systemic examination, his respiratory system showed intercostal tenderness and increased vocal resonance with diminished breath sounds in the left infrascapular area. Hence, a provisional diagnosis of left lower lobar pneumonia was made.

On initial workup, his reverse transcription polymerase chain reaction for COVID-19 tested negative. His electrocardiogram showed tachycardia with T-wave inversion in lead III. His chest radiography revealed left lower zone consolidation with effusion, and the same was confirmed on ultrasonography chest (Fig. 2).

High-resolution computed tomography (HRCT) chest plain was done, which confirmed the left lower lobe segmental consolidation with pleural effusion (Fig. 3). A two-dimensional echocardiogram was done that showed a normal study except for mildly dilated inferior vena cava. All his blood parameters showed elevated hemoglobin of 19.8 gm/dL, red blood cell of 5.7 million cells/cumm, packed cell volume of 57.2%, and total leucocytes of 16,000 cells/cumm. His C-reactive protein (CRP) was 91.8 but with a normal D-dimer of 370 ng/mL (<500 on repeated occasions) and a normal serum procalcitonin of <0.1.

He was admitted to the intensive care unit (ICU) and initiated on intravenous broad-spectrum antibiotics and injection of tranexamic acid. The patient responded well and became afebrile in 48 hours. However, the persistence of blood clots in the sputum warranted the differential diagnosis of pulmonary infarct (PI). CT pulmonary angiogram was done, which confirmed the presence of acute PI of left lower lobe basal segments secondary to acute segmental pulmonary arterial thromboembolism. It also reported the presence of consolidation and pleural effusion (Fig. 4) (Video 1).

As he was hemodynamically stable, a therapeutic dosage of anticoagulation with low molecular weight heparin at 60 mg two times a day subcutaneously per day was immediately started.

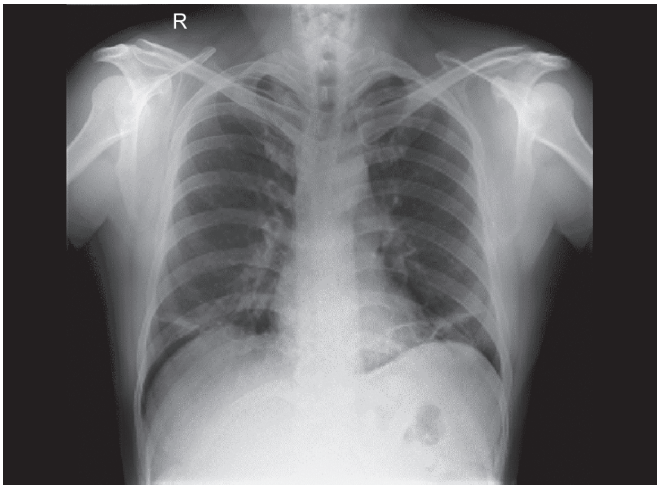

Fig. 5: A chest X-ray after anticoagulation and antibiotics

He was further worked up to identify the source of venous thromboembolism. Venous color Doppler of both lower limbs was negative for deep vein thrombosis (DVT) except for a slow flow vascular malformation in the left distal thigh. He has high serum homocysteine levels of 18.6 µmol/L (reference range 5.46–16.20) and folic acid deficiency of 3.6 ng/mL. He was supplemented with vitamin B_{12} and folic acid, and anticoagulation was continued.

After clinical improvement from symptoms of cough hemoptysis, he was discharged on 10th May 2023 with long-term anticoagulation for around 6 months, folic acid supplementation, cessation of smoking, and alcohol abstinence (Fig. 5).

Discussion

In our case report, we presented a young male with pneumonia and pulmonary thromboembolism (PTE) along with PI, in whom the presence of pneumonia masked the early diagnosis of pulmonary embolism (PE). The coexistence of PE and pneumonia can happen in some cases. The etiology of pneumonia remains unclear whether it is due to PE or community-acquired.[1] Consolidation itself masks the PE, particularly in patients who present with systemic symptoms like fever, chest pain, and cough without any signs and symptoms of lower limb deep venous thrombosis.[2]

Our case is one of the rare entities of normal D dimer PTE with PI. As per a retrospective study in The University Clinical Center, Katowice, the incidence of PE with normal D dimer is 12.7–27%.[3] He initially responded to intravenous antibiotics, but due to the persistence of symptoms, he was further evaluated and found to have concealed acute PTE with PI. This kind of case specifies the importance of considering PE in patients with pneumonia when there would be an initial therapeutic response followed by a worsening of the condition during the treatment of pneumonia.[4]

When the distal pulmonary artery gets obstructed, it causes PI. As per various reports, the incidence of PI in PE is 30%.[5] PE has an incidence of 39–115 per 1 lakh[5] with an estimated annual mortality rate of 8.3 per 1 lakh population.

The prevalence of PI in association with PE is the most common etiology for the incidence of PI. Other disease states that can lead to PI include infection, malignancy, surgery, amyloidosis, sickle cell disease, and vasculitis.

Smoking itself acts as a risk factor for the incidence of PI as well as PE.[6] Younger age of 40 years and taller stature have a high risk of developing PI from a PE.[6,7]

Previously, cardiac patients were at the highest risk for PI, as per the hypothesis that it could be due to the development of poor collateral circulation in the lungs.[8]

However, recent literature suggests particularly younger patients without any cardiac ailments are more likely to suffer a PI secondary to a PE. This is secondary to the formation of prominent bronchial vascular collaterals due to longstanding local tissue hypoxia from chronic cardiopulmonary disease. These provide blood supply to the lung parenchyma, thus preventing infarction.[8]

As per one study by Miniatti et al., in patients presenting with coexisting PE and PI, common symptoms at presentation were breathlessness (69–78%), chest pain (49–70%), swelling or pain in unilateral lower limbs (27–31%), fever (5–11%), and hemoptysis (4–19%).[6]

In another study by Zhang et al., symptoms like chest pain, breathlessness, hemoptysis, and fever were identified as individual risk factors of PE in patients who were initially diagnosed with pneumonia.[9]

Pulmonary infarction (PI) sets in when the compromised blood supply is not reversed in a few days. Around 77–87% of PIs occur unilaterally in the right lower lobe. As per many studies, there is a higher predominance of PI in the lower lobes than in the upper lobes.[4]

Chest X-ray as an initial mode of investigation for pneumonia. It may show PI with various signs. A wedge-shaped consolidation at the lung periphery known as "Hampton's hump," "Wester mark's sign" with focal oligemia or increased lucency of lung fields, and a "Fleischer sign" prominent central pulmonary artery are specific findings for PI due to PE.

Computed tomography (CT) with pulmonary angiography is the most commonly used imaging technique for diagnosing PI as well as PE. If a feeding vessel sign with a central lucency without any air bronchogram is present on CT, the specificity for detecting PI is 99%.[10]

Elevated levels of D-dimer will direct the diagnosis better than other inflammatory markers, such as CRP and total leukocyte count, for the diagnosis of underlying PE in patients with pneumonia.[1]

In our case, in view of strong suspicion of PI despite intravenous antibiotics and normal D-dimer, CT pulmonary angiography was done that confirmed the presence of acute pulmonary arterial thromboembolism and PI after initiation of the therapeutic dose of anticoagulation patient improved well.

The average time of resolution of PI has not been well studied. As per one retrospective review, out of 32 patients evaluated by CT scanning after PI with intervals varying from 1 to 69 weeks after initial diagnosis, 10 were found to have continued evidence of PI at an average interval of 10 weeks from the time of initial diagnosis.[11]

Conclusion

Hereby, we conclude that in our case, slow flow vascular malformation, smoking, and increased homocysteine levels contributed to the development of acute PTE, overwhelming the possibility of PI even though his D-dimer was normal.

When evaluating any young person presenting with unexplained pleuritic chest pain or hemoptysis to the emergency, a PI complicating an acute PE should be considered a possible diagnosis, among other conditions like pneumonia, TB, and malignancy.

Various differential diagnoses like TB, pneumonia, and malignancy should be kept in mind, and early diagnosis using CT

pulmonary angiography in suspected cases and early initiation of anticoagulation reduces mortality.

Supplementary Material

The supplementary video 1 is available online on the website of https://www.ijrc.in/journalDetails/IJRC

Video 1: Computed Tomography Pulmonary Angiography (CTPA) video shows filling defects in left lower lobar basal segmental pulmonary arteries.

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