An Unusual Complication of Pulmonary Embolism

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Abstract:
Pulmonary embolism (PE) is a common and often fatal disease. The clinical presentation of PE causing infarction of lung parenchyma is variable and nonspecific, making accurate diagnosis difficult. PE is responsible for 0.4 percent of hospitalizations and untreated, is associated with an overall mortality of up to 30 percent. Course of pulmonary embolism can be varied ranging from complete clinical recovery to death. Lung Abscess is a rare complication of PE. Reports of lung abscess secondary to bland infarction have been noted relatively infrequently. In this case report, we present a case of pulmonary embolism causing pulmonary infarction which is complicated by secondary infection leading to lung abscess.

Keyword: Pulmonary embolism, Pulmonary infarction, Lung Abscess, PE Case Report

A 39 year old gentleman, who is a smoker, with a history of deep vein thrombosis 4 years back and not on any medications now, presented with complaints of acute onset dyspnoea (NYHA Class III) for 5 days, now worsening to class IV for 2 days, fever and cough with scanty white expectoration for 5 days. Patient also complained of pain in the left lower limb. Initial investigations showed total count of 23,400 cells/μL. D-Dimer was 1.7 ug FEU/ml. Procalcitonin was 3.8 ng/ml. ECG showed sinus tachycardia with a rate of 120/min with S1Q3T3. Chest X-ray showed opacity in upper zone of left lung. Local examination of the left leg showed signs of cellulitis and presence of pus in the lateral aspect of the leg. Initial working diagnosis of sepsis with left leg cellulitis and left upper zone pneumonia was made. Echocardiography showed a dilated right atrium and ventricle, RV dysfunction and PAH of 56mmHg. In view of the history of DVT and the presence of typical ECG and Echo findings, a possible diagnosis of pulmonary infarction was made. Venous doppler of left leg showed complete thrombosis of common femoral, superficial femoral, popliteal, anterior tibial and posterior tibial veins and doppler of right leg showed thrombosis extending from common femoral vein to proximal right popliteal vein.
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Initial CXR showing opacities in left lung
CTPA showing total cutoff of left main PA
CT Pulmonary Angiogram was done which showed thrombus causing complete luminal obstruction of left pulmonary artery, its segmental and subsegmental branches suggestive of pulmonary thromboembolism. Patient was thrombolysed with streptokinase for 24 hours. He was started on imipenem+cilastatin and clarithromycin based on the pus culture from leg which grew Staphylococcus aureus. Blood, urine and sputum culture were negative. Sputum AFB was negative. After 24 hours of thrombolysis, patient showed improvement in symptoms clinically and echo showed reduction in RA and RV size with normal RV function and PAH of 36mmHg. Workup for coagulopathies showed positivity for anti phospholipid antibody syndrome. Patient was started on low molecular weight heparin and oral anticoagulation and target INR was achieved. Patient continued to have fever and scanty expectoration but since patient wanted discharge due to personal reasons and so was discharged. Six days after discharge, patient came back with history of worsening breathlessness, fever and cough with purulent expectoration for 2 days. Clinical examination showed decreased air entry on the left side. Chest X-ray showed left upper lobe and lower lobe cavities. INR was 3.22. HRCT lung was done which showed large irregular cavities with surrounding consolidations and large internal air fluid levels in left upper lobe and left lower lobe replacing most of the lung parenchyma suggestive of abscess cavities.
**CXR showing air fluid level in left lung**
**HRCT lung showing large irregular air cavities**

He was started on piperacillin tazobactam and was later changed over to meropenem based on sputum culture report which grew ESBL producing Klebsiella which was resistant to piperacillin. Patient improved symptomatically with IV antibiotics for 14 days and was discharged. Discussion Lung abscess is a known but rare complication of pulmonary infarction with an incidence of 7% [2]. The source of infection has been thought to be due to secondary infection either from a distant site or by migration of pathogens from upper respiratory or dental infections [2]. Lung abscess due to an infective embolus is rare. Empyema can occur as a complication of abscesses. A case series by Levine et al showed that while the diagnosis was definitely established clinically in only three out of 26 cases who developed lung abscess, it is felt that abscess formation should be suspected in any case of infarction in which leukocytosis, unremitting fever and possibly a productive cough develop subsequently. They concluded that abscess development in a region of aseptic infarction depends upon the following factors: 1) The size of the region of infarction, 2) The state of blood supply to the region of infarction and the adequacy of collateral circulation, 3) The state of the surrounding pulmonary tissue, including such factors as coexistent congestion or atelectasis. 4) Bacteriologic factors, which include the presence or absence of dental, buccal and pharyngeal infections; the presence of bronchitis; the virulence of the organisms involved, and the massiveness of the infection and 5) Host immune status. Conclusion Lung abscess is a rare complication of pulmonary infarction. Any infection that occurs during pulmonary infarction must be treated adequately to prevent formation of lung abscess.

**References:**


