Pulmonary Thromboembolism Presenting as Multiple Pulmonary Cavities

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Abstract

A 38 years old male patient presented to the emergency department with acute severe retrosternal chest pain and was found to have pulmonary thromboembolism. The patient developed new necrotic lung cavities each day due to the dissemination of emboli from the thrombus. The cause of thrombus was found to be protein C deficiency, which is an inherited thrombophilia. The interesting features in this case are multiple lung cavitations and high grade fever, which was attributed to pulmonary thromboembolism. The patient improved symptomatically with anticoagulants and antiplatelets.

Introduction

Protein-C deficiency presenting with pulmonary thromboembolism (PTE) is rare. Most often PTE is a sequelae of deep venous thrombosis of leg veins. Pulmonary embolism as a cause of multiple lung cavities is also rarely reported in literature. Pulmonary embolism with classical clinical and radiologic findings are rarely encountered because patients with major pulmonary embolism (PE) mostly die during the acute episode and minor PE goes unnoticed because of lack of symptoms. Here we report a case of PTE with interesting clinical and Radiological findings.

Case Presentation

A 38 year old male presented to the emergency department with sudden onset of retrosternal constricting type of chest pain and breathlessness of 5 hours duration. The patient was a smoker for past 15 years, with no H/O diabetes, hypertension or coronary artery disease. Patient’s father had succumbed to acute myocardial infarction at the age of 49 years.

The patient had tachycardia (HR 120/min). BP was 100/70mmHg. The ECG showed ST segment depression in inferior leads (Figure 1). The chest x ray taken at the time of admission was normal (Figure 2). Troponin T was negative. A diagnosis of acute coronary syndrome with inferior wall non-ST-elevation myocardial infarction (NSTEMI) was made and the patient was treated with inj. morphine, sublingual nitrates, unfractionated heparin and antiplatelets.

Despite treatment, the patient had persistent chest pain and he developed high grade fever on the 3rd day during his hospital stay. Lung crepitations were heard in the left interscapular region. An urgent chest radiograph showed consolidation in the left lower zone and cavitation in the left midzone close to the hilum (Figure 3). CT chest done on the 4th day showed more cavities in the anterior segment of left upper lobe, superior segment of left lower lobe and consolidation in the posterior segment of left upper lobe. It also showed partial thrombotic occlusion of both pulmonary arteries and complete thrombosis of left lower lobar pulmonary artery (Figures, 4, 5, 6, 7).
Protein-C deficiency is inherited as an autosomal dominant disease and it was described by Griffin in 1981. Deficiency is of two types. In subtype I, immunologically identifiable protein C and functional protein C are diminished proportionately. In subtype II, the deficiency is secondary to a functional defect within the protein and protein C antigen detected by immunological methods are normal.

Pulmonary thromboembolism presenting with multiple cavities is rare. Pulmonary arteries are occluded with showers of emboli which get dislodged from the thrombus in the large arteries. Cavity is formed due to pulmonary infarction and necrosis. Pulmonary embolism is the most common non-infectious cause of lung cavities. Pulmonary infarction with cavitation occurs in 10–15% of cases of PTE, according to older studies. But with the advent of computed tomography the incidence of cavitory lesion is now upto 32% in pulmonary embolism. The lesion occurs most commonly in the lung periphery, but it can occur in any location. In PTE a normal chest X-ray is also not uncommon.

Low grade fever is the usual manifestation, but a high grade fever more than 38°C can also occur. Our patient had a high grade fever of 103°F, which was an uncommon finding.

This case is reported because of the rarity of protein C deficiency presenting as pulmonary thromboembolism with multiple cavities and high grade fever. To conclude, in the absence of classical risk factors for PTE, protein C deficiency should be borne in mind while evaluating thrombotic disorders.

References