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Cavitary Pulmonary Infarction Mimicking Koch's Disease

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Abstract

Pulmonary infarction (PI), defined as localized destruction (necrosis) of lung tissue due to obstruction of the arterial blood supply by an embolus, is a rare condition because of the dual blood supply of the lungs. It occurs more in elderly patients who typically have associated co-morbidities, such as chronic heart and lung disease, which affect blood circulation. Pulmonary infarction may present with chest pain, fast breathing, blood tinged cough and fever, resembling more common conditions such as bacterial pneumonia and tuberculosis. High index of suspicion is required for early diagnosis of this condition as mortality from this condition can be as high as 70%. We present a case of cavitary pulmonary infarction in a middle-aged female with no major risk factors for pulmonary embolism who was initially managed as a case of pulmonary tuberculosis. The clinical presentation, pathogenesis, imaging findings and management of PI have also been discussed. The purpose of this report is to increase the awareness of this less common condition among clinicians and highlight the radiologic differences between PI and the more common inflammatory diseases of the lung.

Keywords

Pulmonary Embolism, Infarction, Haemoptysis, Computed Tomography, Tuberculosis

1. Introduction

Pulmonary infarction (PI) is defined as focal destruction (necrosis) of the lung parenchyma due to cut-off of the arterial blood flow by an embolus [1].

It is a rare complication of pulmonary embolism (PE) because the lung rece-

ives dual arterial supply from the pulmonary and bronchial arteries). Pulmonary infarction occurs in about 10% - 15% while PI with cavitation occurs in 4% - 7% of patients with pulmonary embolism [2].

PI occurs more commonly in elderly patients due to higher prevalence of co-morbidities [1]. The predisposing factors for pulmonary infarction include congestive cardiac failure, pleural effusion, pulmonary infection, atelectasis, hypotension, chronic lung disease, central venous line placement, and an immuno-compromised state [1].

PI may be mild or rapidly fatal, with mortality rates being up to 73% for secondarily infected cavitary PI [3]. It commonly presents with pleurisy due to mild pleural effusion, increased pulse rate, difficulty in breathing, low-grade fever, and productive cough which may be blood-tinged. When mild, its clinical presentation closely mimics that of other much commoner inflammatory conditions such as bacterial pneumonia and tuberculosis.

Treatment of PI is mainly supportive with antibiotics as well as anticoagulation given to patients at low risk of active bleeding. Thrombolysis may also be considered in patients with large emboli [4].

Following is a case presentation of cavitary pulmonary infarction in a middle-aged woman mimicking pulmonary infection. It highlights the features which help differentiate this condition from common inflammatory conditions in our environment, and aims to increase the index of suspicion for cavitary PI as a differential diagnosis of pulmonary cavities among radiologists.

2. Case Presentation

Mrs. B.S was a 42-year-old hospital attendant at Braithwaite Memorial Specialist Hospital (BMSH) who presented to the Accident and Emergency unit with a 5-day history of haemoptysis, chest pain, fever and left lower limb swelling. There was neither history of significant weight loss or excessive night sweats. She was not overweight, or on oral contraceptive medication. There was no previous history of chronic cough, smoking, previous deep venous thrombosis or known malignancy.

On examination, she was apprehensive and dyspnoeic with a respiratory rate 30 breath/min and blood oxygen saturation of 92%. Chest expansion was symmetric with normal breath sounds. Her heart rate was 116 bpm and temperature was 38.5˚C. She had pitting pedal oedema that was present up to mid-leg on the left with no calf tenderness or differential warmth. There was no cyanosis or jaundice and her blood pressure was normal. Her Wells' score for pulmonary embolism was 2.5.

A clinical diagnosis of severe bronchopneumonia was made and she was commenced on intravenous fluids, antibiotics and referred for an urgent Chest radiograph on account of the hemoptysis. CXR (**Figure 1**) showed peripherally located, mottled opacities in both lower zones with obliteration of the right hemidiaphragm and costophrenic angle. There was mild loss in lung volume on

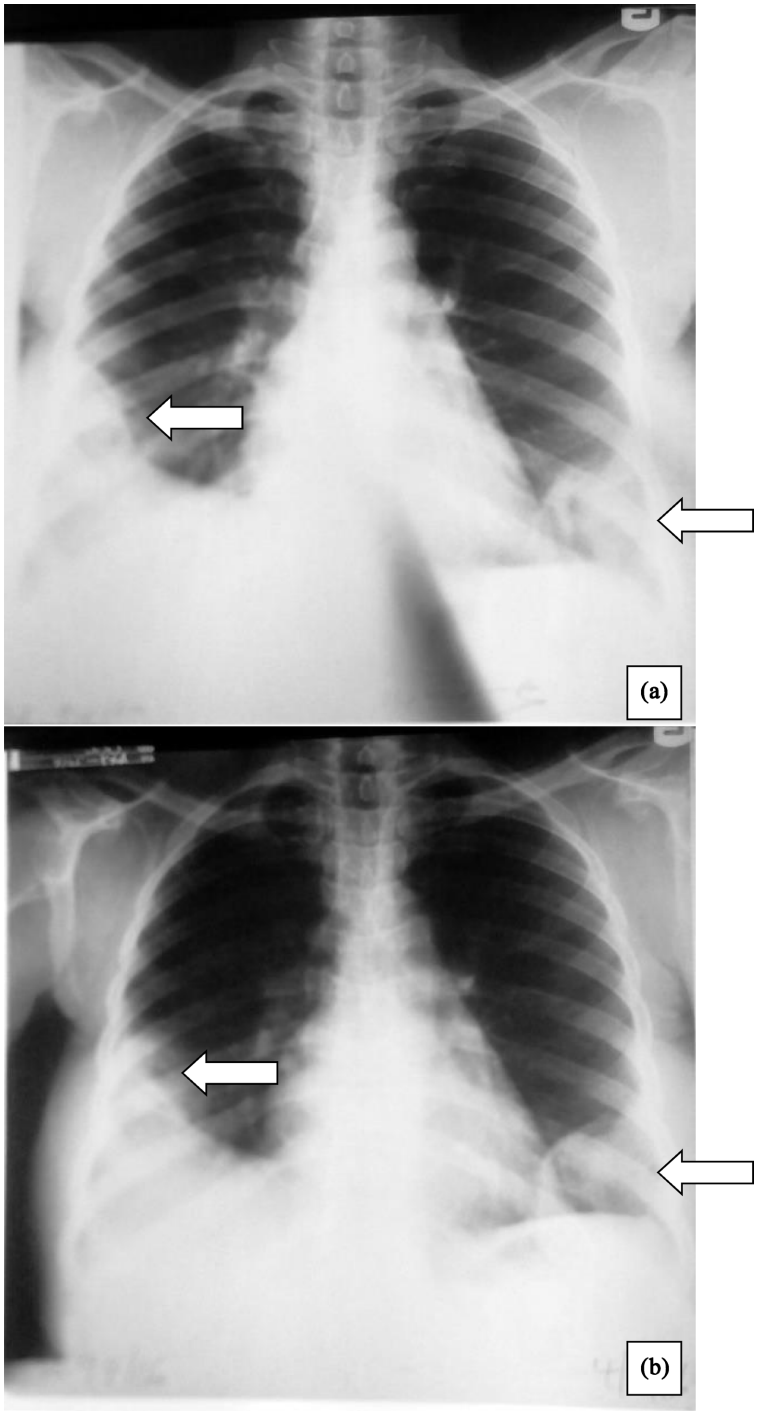

Figure 1. (a) PA Chest X-ray done at presentation showing pleural based opacities with mottled lucencies (arrows) in both lower lung zones with associated silhouetting of the right hemidiaphragm. Repeat radiograph a week later (b) shows minimal resolution with evidence of cavitation of the lesions.

the right with rib crowding. The bony ribcage was within normal limits. An impression of pulmonary tuberculosis (PTB) was made and sputum Acid-Fast bacilli (AFB) test for confirmation of the diagnosis was recommended. The result of the AFB showed no tubercle bacilli and Gene X-pert sputum test was re-

quested. This was also negative for PTB. Other blood tests done showed normal full blood count and clotting profile. The retroviral screen was negative. Follow-up CXR done a week later (Figure 1(b)) showed no new lesions, but there was cavitation of the previously identified opacities.

Based on the non-diagnostic sputum results and the CXR findings, a contrast-enhanced chest CT was requested on the 10th day of admission which showed non-enhancing, wedge-like, pleural based opacities with irregular central cavitation in the right middle lobe and left lung base (Figure 2, Figure 3). The cardiothoracic ratio was normal with no mediastinal or hilar lymphadenopathy. Major intra-thoracic vessels were normal in course and caliber. There was no pleural effusion. However, a rounded filling defect in the distal aspect of the left lower segmental pulmonary artery was noted suggestive of a pulmonary embolus. Pulmonary infarction was diagnosed based on the chest CT findings of pleural based opacities with irregular central cavitation, her clinical presentation, as well as the negative Acid fast bacilli and Genexpert tests.

The patient was commenced on Warfarin 5 mg daily and intravenous broad-spectrum antibiotics were continued. Patient's clinical condition improved and she was discharged home a few days later on oral anticoagulants to be follow-up in the Medical outpatient department. She made remarkable clinical recovery.

3. Discussion

Pulmonary infarction (PI), defined as focal destruction (necrosis) of the lung

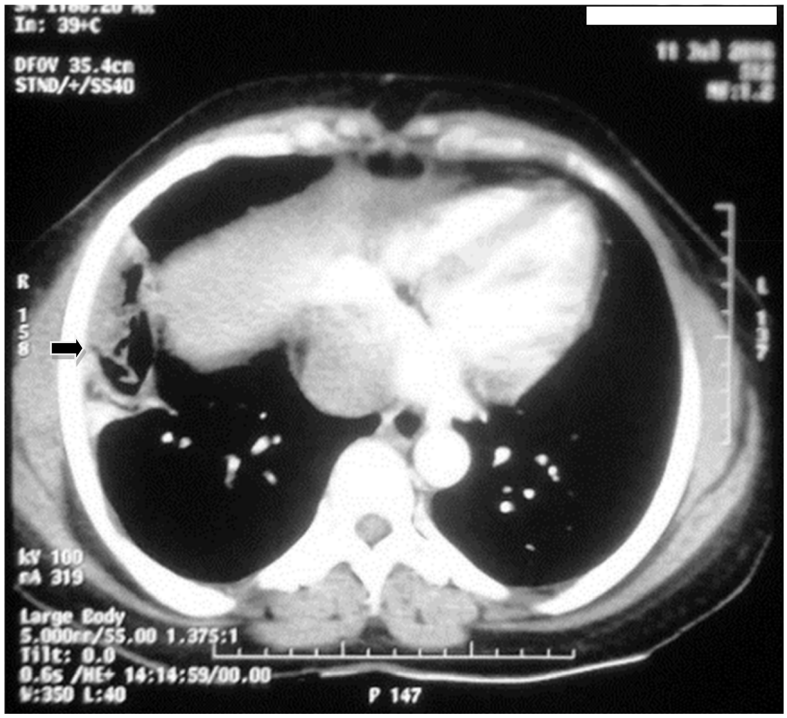

Figure 2. Contrast enhanced axial HRCT image showing a wedge-shaped peripheral opacity (arrow) with irregular internal lucencies in the right middle lobe. There was no pleural effusion.

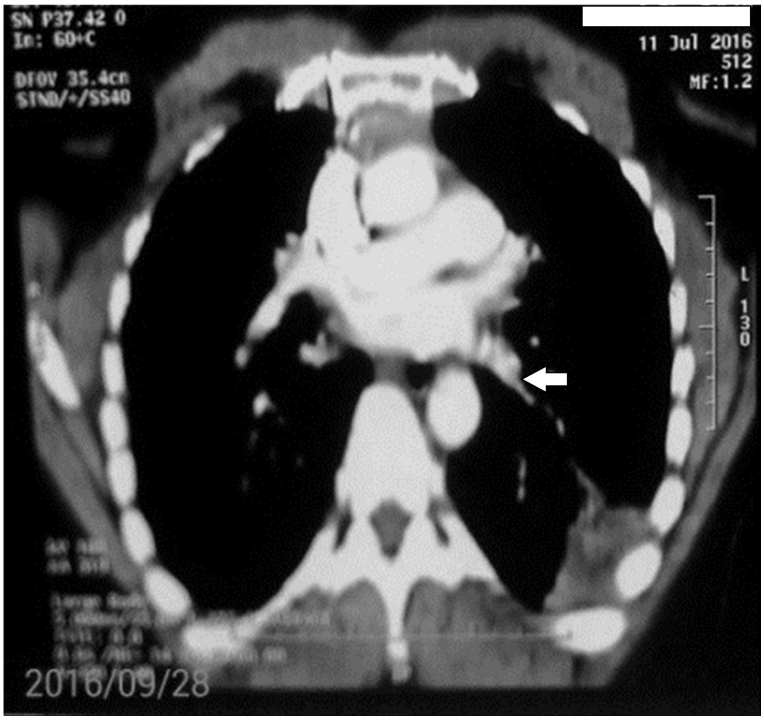

Figure 3. Contrast enhanced coronal HRCT image showing pleural based wedge-shaped opacity with a rounded filling defect (arrow) the distal aspect of the left descending pulmonary artery.

parenchyma due to cut-off of the arterial blood supply by an embolus is an uncommon condition [1]. This is due to the double arterial blood supply of the lungs, as well as direct oxygenation of the lung parenchyma via diffusion of oxygen in the alveoli [2]. Cavitary PI is seen in about 4% - 7% of patients with acute PE. Cavitation after pulmonary infarcts may result from either aseptic necrosis of the infarcted lung (bland infarct) or from superimposed bacterial infection with formation of an abscess cavity [4]. However, several case reports indicate the later is more common, and typically present with present with a triad fever, elevated white cell count, and positive sputum [5]. The index patient presented with fever, but the white cell count was within normal limits so sputum analysis was done for only acid-fast bacilli.

Clinically, differentiating between either of these two as the origin of PI remains a diagnostic challenge, but in situations where the superimposed infection develops as a complication of aseptic PI, there is worsening of the clinical state as well as leukocytosis [6]. The differential diagnosis includes bacterial pneumonia, tuberculosis, aspergillosis, nocardiosis, actinomycosis, and pulmonary granulomatous vasculitides. Others are primary or metastatic sarcomas and bronchogenic carcinoma with invasion of the pulmonary arteries [7] [8].

When compared to those with bacterial pneumonia, PI patients are usually more dyspnoeic and tachypnoeic with mild physical and radiographic findings. They tend to present more with hypotension, and commonly have findings suggestive of pulmonary hypertension on auscultation (pronounced pulmonic

component of the second heart sound, tricuspid regurgitation murmur) and elevated jugular venous pressure [9]. The index patient was dyspnoeic with chest pain and pedal oedema. A pleural rub is suggestive of pulmonary embolism when the chest radiograph does not suggest parenchymal abnormalities [5].

Complications of PI include pneumonia from secondary infection, empyema, pneumothorax, lung abscess, bronchopleural fistulae and rarely massive haemorrhage [4]. None of these complications were present in the index patient one month after discharge.

On chest roentgenograms, PTB, bacterial pneumonia and PI produce overlapping parenchymal changes and that are difficult to distinguish radiographically. Each may or may not present with pleural effusion, atelectasis and cavity formation. In contradistinction to PTB and bacterial pneumonia which may be located in any part of the lung, pulmonary infarcts always adjoin a pleural lining (usually in lower lobes) producing the "Hampton's hump", as was seen in our patient (**Figure 1(b)**). There may also be associated dilatation of one or both main pulmonary arteries and attenuated vascular markings in the periphery of affected portion of lung due to oligemia known as Westermark's sign [10]. Further radiographic clues to pulmonary infarction are lung opacities appearing first in one lung and then the other or "pneumonia" unresponsive to chemotherapy.

Contrast enhanced helical computed tomography is extremely helpful in establishing the diagnosis of pulmonary infarct and should be done if the chest radiograph is abnormal [11]. Also, it could help track the resolution of the thrombo-emboli, but it is relatively expensive [12]. CT Pulmonary angiography (CTPA) is diagnostic test of choice for distinguishing pulmonary infarction from other inflammatory conditions and shows filling defect(s) in the pulmonary arteries and branches down to the segmental level [1]. Although a CTPA could not be done due a faulty automatic injector, the reformatted image showed filling defect suggestive of an embolus (**Figure 3**). Central lucencies in peripherally located consolidations abutting the pleura (**Figure 2**) were highly suggestive of pulmonary infarction on HRCT [13]. This was the diagnostic feature for cavitary PI in the index patient. Larger infarcts are more likely to have central hypoxia with consequent necrosis, and infarcts greater than 4 × 4 cm in size have been found to have a higher likelihood for cavitation [14]. The average time from the initial detection of consolidation to cavitation is about two weeks (range: 6 - 40 days) for infected emboli and about four weeks for bland emboli [15]. Another sign which may be present on HRCT is the "reverse halo sign" which appears as central area of ground-glass opacity surrounded by a denser complete or near-complete ring of consolidation. This sign is however non-specific and may be present in tuberculosis, sarcoidosis, and pneumocystis pneumonia [16]. The reverse halo sign was absent in the index case.

Sputum examination is one most reliable methods of differentiating PTB from infarction. In pulmonary infarction, sputum, when expectorated, usually is

frankly blood with few bacteria or inflammatory cells while staining for Acid Fast Bacilli is diagnostic for pulmonary tuberculosis. However, in cases of secondary infection of a pulmonary infarct, as was likely in the index case, sputum findings become similar to that in bacterial pneumonia.

Treatment of pulmonary infarction depends on whether it is sterile or not and a combination of anticoagulants, antibiotics and cardiopulmonary support are the mainstay of therapy. In cases of infected pulmonary infarction, anticoagulation and antibiotic therapy should be selected and initiated based on local resistance patterns and patient characteristics [17]. Surgical interventions have also been used to manage complications of infected pulmonary infarction [5].

4. Conclusion

Clinicians should always have a high index of suspicion for pulmonary embolism in patients presenting with haemoptysis and cavitary pulmonary infarction should be on the radiologists' list of differentials for patients with cavitary lung lesions and haemoptysis. Where available, radiologic imaging can be very useful in differentiating this condition from more common inflammatory conditions in our environment.

Informed Consent

Informed consent for publication of this case report was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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