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Pulmonary thromboembolism presenting as a cavitating lesion on chest X-ray

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ABSTRACT Pulmonary embolism presenting as a cavitating lesion on a chest X-ray is described in a patient with a six-week history of worsening dyspnoea. A chest X-ray performed 27 days earlier did not show the cavitating lesion. On the day of presentation, the patient had developed streak haemoptysis. Due to the progressive nature of dyspnoea, hypoxia and the rapid development of the cavitating lesion on the X-ray, pulmonary infarction leading to cavitation secondary to pulmonary thromboembolism was considered to be among the list of differential diagnoses. The modified Wells criteria for this patient were 2·5 (Heart rate > 100, haemoptysis). This diagnosis was confirmed on CTPA, which showed the presence of large pulmonary emboli in the pulmonary vasculature supplying the affected lobe. Subsequent Doppler ultrasound revealed the presence of a below right knee thrombosis. Although rare, cavitation has previously been observed in patients with pulmonary thromboembolism.[1–3]

KEYWORDS Cavitating lesion, chest X-ray, deep vein thrombosis, pulmonary embolism

LIST OF ABBREVIATIONS Computerised tomography (CT), Accident and Emergency (A&E), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), General Practitioner (GP), chest X-ray (CXR), pulmonary embolism (PE), computerised tomography pulmonary angiogram (CTPA), international normalised ratio (INR), deep vein thrombosis (DVT), echocardiogram (ECG), arterial blood gases (ABG), ventilation-perfusion (V/Q), Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED)

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A 68-year-old Caucasian man presented to A&E with a one-day history of streak haemoptysis, a two-week history of dull right-sided chest pain, which increased on deep inspiration, and six weeks' history of worsening shortness of breath on exertion. Two months earlier, he had had an unlimited exercise tolerance but it was now limited to around 200 yards on the level. There was no history of coughing or wheezing. There was no significant past medical history of note. He had been working as a long-distance truck driver with weekly trips to continental Europe and had continued working until around one week prior to presentation. He had given up smoking around 20 years previously, and had in total around 20-pack years' history of smoking.

On examination in A&E, he had a respiratory rate of 34. Saturations were 94% on air. He was afebrile and blood pressure was found to be 113/75. Examination of chest, cardiovascular and abdomen were normal. There were no signs of DVT clinically. Echocardiogram showed sinus tachycardia with rate of 106/min and no other abnormalities. Arterial blood gases on air are noted in Table 1:

pH = 7·471	pCO_2 = 35·0 mmHg (4·66 kPa)
pO_2 = 65·1 mmHg (8·68 kPa)	HCO_3= 25·0
BE = 1·5	FiO_2 = 0·21

TABLE 1 Patient's arterial blood gases.

Blood tests showed neutrophil leucocytosis with white cell count of 16·7 and neutrophil count of 14·1. Haemoglobin was 10·0 g/dL (MCV and MCH were normal) and platelet count 589.

He had initially gone to his GP four weeks previously, when his sole complaint was the increasing shortness of breath on exertion. A chest X-ray was requested by his GP (see Figure 1A). It was thought to have a normal appearance and the patient was treated with antibiotics for a presumed pleurisy secondary to lower respiratory tract infection. His chest X-ray was repeated on admission to A&E and this showed presence of a large cavitating lesion in the right upper zone. (see Figure 1B)

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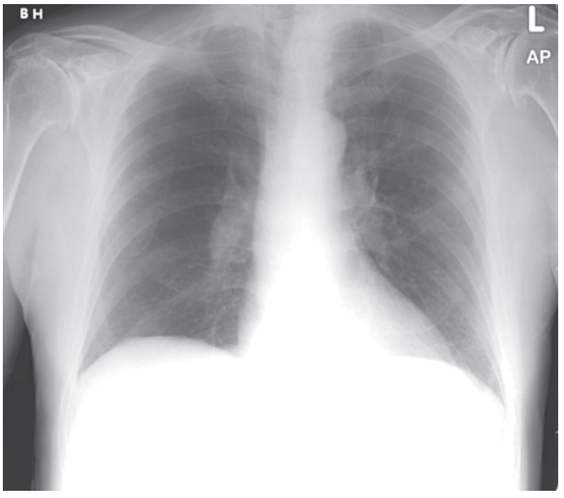

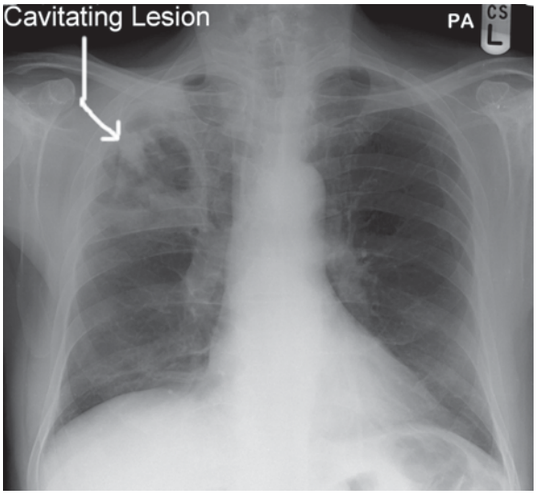

FIGURES 1A AND 1B Chest X-rays taken 27 days apart. Although on first look, the X-ray in Figure 1A (on the left) appears to be normal, a more detailed and systematic examination shows there to be lack of vascular markings in the right upper lobe area. This area of oligemia is sometimes referred to as Westermark's sign, and although not seen very commonly, when present, is suggestive of PE. Figure 2B (on the right) shows the X-ray at presentation and shows presence of a large cavitating lesion in the right upper zone.

The history of worsening shortness of breath over six weeks, the presence of haemoptysis, and the presence of oligemic area on the first chest X-ray which later developed into cavitation, were highly suggestive of a diagnosis of PE. The patient's occupation as a long distance truck driver with a higher risk of lower limb thrombosis was consistent with this diagnosis.

As the patient was haemodynamically stable, it was decided to treat him with low molecular weight heparin. A CTPA confirmed the presence of pulmonary emboli in the right pulmonary artery and its branches, as well as the presence of the cavitating lesion. (see Figures 2A–2D)

Although the patient did not have clinical signs of DVT in his lower limbs, a Doppler ultrasound examination of both legs showed the presence of a right-sided below knee thrombus. The only risk factor for thromboembolism was the patient's frequent trips as a truck driver to continental Europe. The patient was started on warfarin, the low molecular weight heparin stopped when the INR reached the therapeutic range and he was then discharged home. The patient's total in-hospital stay was six days.

The final diagnosis was made of DVT of the right femoral vein resulting in PE leading to cavitation in the infarcted lung. The patient subsequently had a transthoracic echocardiogram performed as an outpatient and this showed normal right side of heart and normal pulmonary artery pressure. He was reviewed in the outpatient clinic six weeks after this, and continues to remain in good health.

DISCUSSION

Pulmonary embolism is a difficult diagnosis to make. The clinical presentation is variable and can be non-specific. Symptoms and signs are not helpful diagnostically because their frequency is similar in patients with and without PE. In a large prospective study,[4] the following frequencies of symptoms and signs were noted among patients without pre-existing cardiopulmonary disease:

- The most common symptoms were dyspnoea (73%), pleuritic pain (66%), cough (37%) and hemoptysis (13%). Hemoptysis was blood tinged, blood streaked, or pu re blood; it was rarely massive.
- The most common signs were tachypnea (70%), crepitations (51%), tachycardia (30%), a fourth heart sound (24%), and an accentuated pulmonic component of the second heart sound (23%). Circulatory collapse was uncommon (8%).
- Fever, usually with a temperature <102·0°F (38·9°C), occurred in 14 percent of patients with no other apparent source for an elevated temperature. Fevers >103·0°F (39·4°C) were rare.[5]

In patients with massive PE, these physical findings may be accompanied by acute right ventricular failure, manifested by increased jugular venous pressure, a right-sided third heart sound, and a parasternal lift.

Most patients with PE have no leg symptoms at the time of diagnosis. For example, in one study <30% of patients with PE had symptoms or signs of lower extremity venous thrombosis.[6] Reciprocally, patients with symptomatic DVT may have asymptomatic PE.[7–9] In a study of 350 patients with phlebographically proven DVT, PE was shown to be present in 56% by ventilation-

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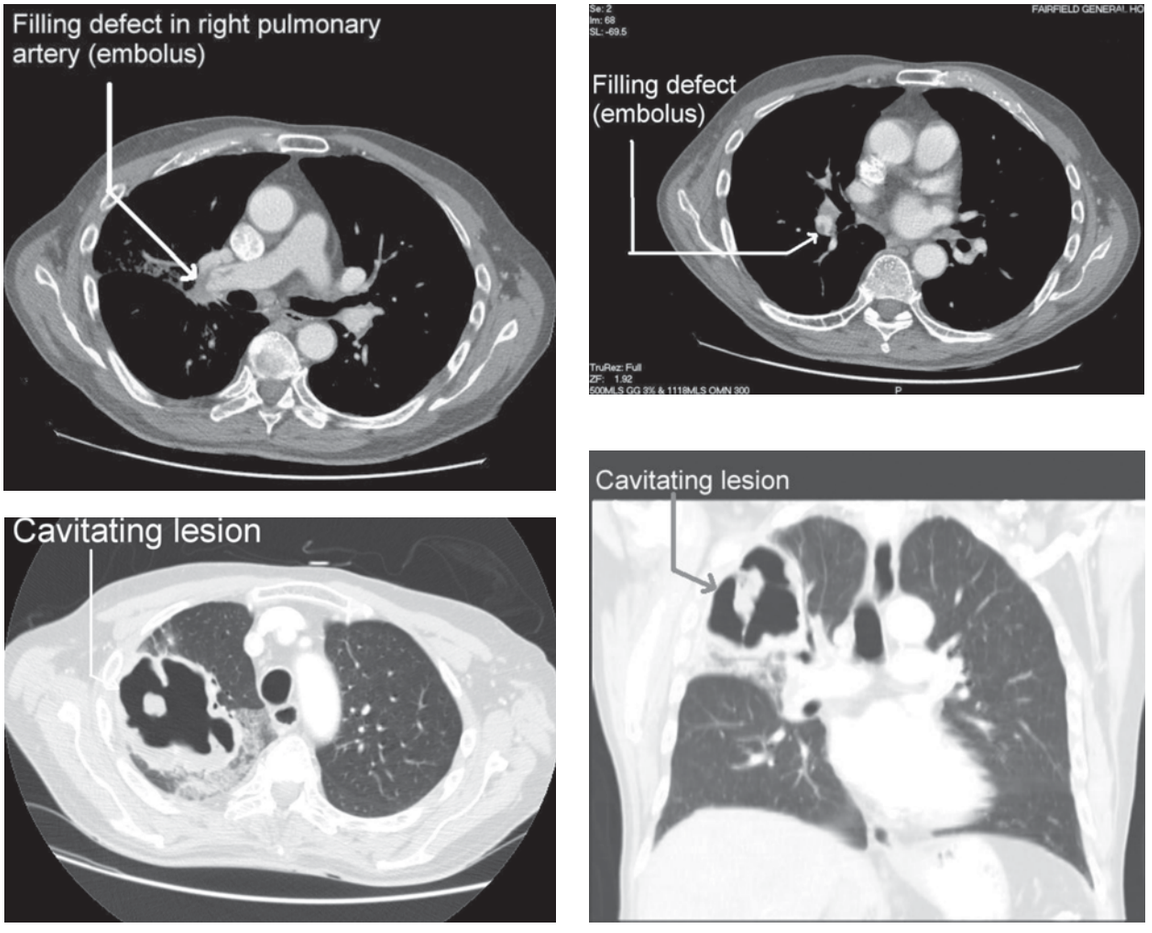

FIGURES 1A–1D CTPA images showing a filling defect in pulmonary vasculature and a cavitating lesion in the right upper lobe.

perfusion lung scan or angiography. Among patients with PE, 26% were asymptomatic.

Radiologically, PE can result in a variety of chest X-ray changes, although it is frequently normal. In a previous study[10] looking at the presence of different chest X-ray abnormalities in patients with proven PE on angiography, the chest X-ray was normal only in 12% of patients with PE. However, the abnormalities were non-specific. Pulmonary oligaemia (Westermark's sign) was noted in around 8% of patients who had PE in the left hemi-thorax and 14% who had PE in the right hemi-thorax. However, pulmonary oligaemia was also present in 4% of patients with no angiographically demonstrable PE. The most common changes were found to be atelectasis and parenchymal areas of increased opacity. In a later study,[11] the presence of oligemia, amputation of the pulmonary artery and consolidation compatible with pulmonary infarction, were seen in 45%, 36% and 15% of patients with PE respectively. Although the individual sensitivity of each of these findings is low, at least one of them is present in 75% of patients. By contrast, these signs were seen in only 1% of patients without PE. This suggested that the chest X-ray is useful in not only excluding PE but also, due to the high specificity of some findings, can strengthen the suspicion of the disease in conjunction with other clinical information. Interestingly neither of these studies mentioned the incidence of cavitation in PE.

The most extensive evaluation of the accuracy of the V/Q scan in PE was the PIOPED.[12] In PIOPED, the accuracy of the V/Q scan was determined by comparison to the gold standard, the pulmonary angiogram. Diagnostic accuracy was greatest when the V/Q scan was combined with clinical probability. Patients with high clinical probability and a high-probability V/Q scan had a 95% likelihood of having PE. Patients with low clinical probability and a low-probability V/Q scan had only a 4% likelihood of having PE. A normal V/Q scan virtually excluded PE. Unfortunately, the combination of clinical and lung scan probability found in most patients (up to 72%) had a diagnostic accuracy of only 15–86%, insufficient to either confirm or exclude the diagnosis of PE. Thus, in these circumstances, additional testing is required. This is usually in the form of CTPA.

In the largest study to date[13] (824 patients), the accuracy of CTPA with and without prior clinical probability assessment (using the Wells criteria) was determined by

comparison against a composite reference standard. Key findings included: 83% of patients with PE had a positive CTPA (i.e. sensitivity). Conversely, 96% of patients without PE had a negative CTPA (i.e. specificity). Addition of venous-phase imaging improved the sensitivity to 90% with a specificity of 95%. The CTPA is therefore a fairly sensitive and specific investigation and also has the added benefit of being able to provide an alternative diagnosis. The PIOPED study found that, by using isotope scanning, PE could only be diagnosed or excluded reliably in a minority of cases. Patients with intermediate probability V/Q scans and those with discordant clinical and V/Q scan probability require further imaging, which is usually in the form of CTPA. Based on these observations, the British Thoracic Society recommends CTPA as the initial imaging modality in patients with non-massive PE. In case of massive PE, treatment with thrombolysis should be instituted on clinical grounds alone if cardiac arrest is imminent. In massive PE, again the CTPA can reliably diagnose PE. Although it is now widely accepted that CTPA is the preferred imaging modality in patients suspected to have PE, clinical resources may make it impracticable to be performed in every case. The selective use of isotope imaging can alleviate pressure on CTPA examinations and V/Q scan can be considered if the facilities are available on site, the chest radiograph is normal and there is no significant concurrent cardiopulmonary disease.

Although cavitation secondary to PE and infarction is a rare phenomenon and the exact incidence is not known, previous studies[14, 15] have shown that this is more commonly seen in patients with other co-morbid conditions such as left ventricular failure and interestingly it is more common in the upper lobes rather than the lower. This is thought to be due to a comparatively lesser amount of vascularity in the upper lobe putting this area at a higher risk of infarction and cavitation. It is interesting to note that this patient's cavitation was in the right upper lobe. In other texts,[16] aseptic cavitation in pulmonary infarction is said to occur more commonly if the area of infarction is greater than 4 cm in diameter and typically occurs two weeks after appearance of focal consolidation.

This case serves to illustrate how PE can be missed if the index of suspicion is low and should always be considered when a patient presents with dyspnoea, which does not have an obvious explanation. It also illustrates PE as a cause of cavitatory lesion in the lung.

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