- 1 Richardson JW, Greenfield LJ. Natural history and management of iliac aneurysms. *J Vasc Surg* 1988; 8: 165–71.
- 2 McCready RA, Pairolero P, Gilmore J, Kazmier F, Cherry K, Hollier L. Isolated iliac artery aneurysms. *Surgery* 1983; 93: 688–93.
- 3 Thiele B, Geddes N, Johnson N, MacLeish D, Royle J, Buxton B. Ruptured iliac aneurysms. *Aust NZ J Surg* 1975; 45: 417–21.
- 4 Sacks NPM, Huddy SPJ, Wegner T, Giddings AEB. Management of solitary iliac aneurysms. *J Cardiovasc Surg* 1992; 33: 679–83.
- 5 Byrne JL, Zaman SN, Meade JW, Aronski WP. Operative management of bilateral internal iliac artery aneurysms. *J Cardiovasc Surg* 1989; 30: 241–3.

Breathlessness in an Afro-Caribbean woman

HRS Roberts, DM Hansell, R Wilson, PJ Cole, CRK Hind

A 32-year-old Afro-Caribbean woman presented with a two-month history of weight loss and pleuritic chest pain, and a two-week history of night sweats, and cough productive of blood-stained green sputum. She had no previous respiratory history of note, was not aware of having had tuberculosis, had no haemoglobinopathy, and denied exposure to risk factors for HIV infection. Examination was unremarkable apart from signs of left upper lobe consolidation, which was confirmed by chest radiography. Sputum microscopy revealed acid-alcohol fast bacilli. Anti-tuberculous therapy (rifampicin, isoniazid, and ethambutol with pyridoxine) was started.

Despite the treatment she became increasingly breathless over the next few days, until she was dyspnoeic at rest. She refused an HIV test. Examination one week after the start of therapy revealed: no pyrexia, a pulse of 100 beats/minute and regular, blood pressure 120/80 mmHg (no postural drop), no murmurs, a respiratory rate of 28 per minute, and features of left upper lobe consolidation/collapse. Investigations showed: haemoglobin 9.6 g/dl, white cell count $14.1 \times 10^9/l$, platelets $374 \times 10^9/l$, normal serum urea and electrolytes; arterial blood gases: pH 7.43 (7.42), PaCO₂ 3.99 kPa (3.78), PaO₂ 5.95 (9.80), bicarbonate 23 mmol/l (22), oxygen saturation 85% (96%) while breathing room air (figures in brackets while breathing 60% oxygen); electrocardiogram was normal; the collapse/consolidation of the left upper lobe was unchanged on chest X-ray. Prednisolone 60 mg daily was added to the treatment.

Over the next 48 hours her breathlessness continued to increase, and her tachycardia increased to 130 beats/minute, but the findings on examination were otherwise unchanged. An arterial sample acquired while the inspired oxygen fraction was 60% showed: pH 7.52, PaCO₂ 4.02 kPa, PaO₂ 7.13 kPa, bicarbonate 24 mmol/l. A chest X-ray was performed (figure 1).

Questions

- 1 What is the likely explanation for the patient's increasing breathlessness?
- 2 Which imaging techniques might you use to reach the correct diagnosis?

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Figure 1 Chest X-ray at initial examination

Answers

QUESTION 1

The diagnosis in this case was pulmonary thrombo-embolism. The major differential, on the grounds that tuberculosis is of increased incidence in patients with HIV infection, was *Pneumocystis carinii* pneumonia (PCP)

QUESTION 2

Contrast-enhanced computed tomography (CT) of the chest was performed to identify PCP, or any other pulmonary cause for the patients severe dyspnoea. CT showed extensive upper lobe, and lingula consolidation, with cavitation (figure 2A); peripheral patches of consolidation in the lower lobes (figure 2B); filling defects bilaterally in the central pulmonary arteries (figure 2A); and a moderate sized pericardial effusion. The upper lobe and lingula changes are typical of pulmonary tuberculosis, and the pulmonary artery filling defects and peripheral patches of consolidation typical of pulmonary emboli with distal haemorrhagic infarction. No features of PCP were identified. Other investigations considered were a radionuclide ventilation-perfusion scan (but this is of relatively low specificity in the presence of infection), and bronchoscopy with lavage/biopsy for PCP (but this would not have demonstrated pulmonary emboli). The chest X-ray taken one week after starting anti-coagulation shows the pulmonary vasculature in the right lung returning to normal (figure 3). A catheter was sited in the main pulmonary artery, and infusion of tissue plasminogen activator was started. There was marked clinical improvement over 24 hours, and she was discharged after 10 days on anti-tuberculous therapy and anticoagulants.

Figure 2 Enhanced CT (A) at mediastinal settings showing emboli within the central pulmonary arteries and cavitating tuberculosis of the left upper lobe; and (B) at lung settings showing pleurally based patches of consolidation, consistent with pulmonary infarcts

Pulmonary thrombo-embolism

CLINICAL ASPECTS

The symptoms and signs depend on the proportion of the pulmonary arterial tree which is occluded, and the time-scale over which occlusion occurs. The classical triad of haemoptysis, a pleural rub, and thrombophlebitis is present in less than a third of cases. Of patients who have suffered a pulmonary embolism, approximately 80% experience dyspnoea, 70% pleuritic chest pain, 60% apprehension, and 50% cough. Only 10–33% of patients with a fatal pulmonary embolisation have symptoms of deep venous thrombosis. As a guide, the clinical features of an acute event (according to the percentage of the total pulmonary artery tree occluded) are shown in the box.

RADIOLOGICAL FEATURES

Embolism without infarction (approximately 90% of patients)

The chest X-ray shows no abnormality in one third of cases. Abnormalities which may be seen include: plate-like atelectasis, areas of

Figure 3 Chest X-ray taken one week after presentation showing normal pulmonary vasculature in the right lung and clearing of shadowing in the lingula

Clinical features of acute pulmonary embolism (depending on percentage occlusion of pulmonary artery tree)	
<20%	asymptomatic, with normal arterial blood gases
21–30%	anxiety and hyperventilation, PaO ₂ <8 kPa, PaCO ₂ <4 kPa
31–50%	dyspnoea and collapse, elevated central venous pressure, PaO ₂ <6.5 kPa, PaCO ₂ <3 kPa
>50%	shock and dyspnoea, systemic systolic blood pressure <100 mmHg, PaO ₂ <5 kPa, PaCO ₂ <3 kPa

consolidation, a pleural effusion, and vascular changes (focal oligoemia, local widening of a vessel due to impaction of a thrombus, and 'cut-off' pulmonary arteries), although the latter are subtle and often overlooked, or only identified retrospectively.

Embolism with infarction (approximately 10% of patients)

The chest X-ray shows consolidation in 50% of cases (most commonly nondescript, but classically a 'Hampton's hump', a truncated cone with a pleural base), a pleural effusion in 50%, plate atelectasis in 30%, and an elevated hemidiaphragm in 20%. CT shows pleurally based areas of consolidation. Emboli may be visualised in the central pulmonary arteries, and spiral CT is increasingly being used as an effective method of demonstrating emboli as far out as the fourth division of the pulmonary arteries.

Pneumocystis carinii pneumonia (PCP)

CLINICAL ASPECTS

PCP is the commonest cause of pneumonia in immunocompromised patients. In AIDS patients there is commonly simultaneous cytomegalovirus, *Mycobacterium avium* intracellular, or herpes simplex virus infection. Patients present with dyspnoea, which becomes severe and associated with cyanosis. Tachypnoea is the most constant sign. The white cell count is slightly elevated, and a lymphopenia is seen in 50% (a bad prognostic indicator). The PaO_2

becomes reduced as the disease progresses, commonly to less than 8 kPa when breathing room air. The arterial pH may be increased, and the PaCO_2 decreased (indicating a respiratory alkalosis).

RADIOLOGICAL FEATURES

Radiographic changes are often preceded by clinical symptoms by a day or so, and the chest X-ray on presentation may therefore be normal. Typically, early during the course of the infection, the chest X-ray shows bilateral basal reticulonodular opacity, or perivascular cuffing. This progresses peripherally, and over three to five days assumes a ground glass pattern, which characteristically shows a bilateral, diffuse perihilar and basal predominance. Lymphadenopathy is rare. Atypical patterns occur (cavitating, nodular, honeycomb, lobar distribution), but their presence should raise the suspicion of a mixed infection.

CT features are: ground glass appearance, which may be uniform (50%), or geographical (25%); an interstitial pattern of bilateral linear/reticular opacities (25%); and, additionally, bullae and thin-walled cysts in 40%, pleural effusion in 20%, and pneumothorax in 10%.

Final diagnosis

Bilateral pulmonary thrombo-embolism complicating pulmonary tuberculosis.

Keywords: pulmonary embolism, tuberculosis

1 Teigen CL, Maus TP, Sheedy PF II *et al.* Pulmonary embolism: diagnosis with contrast enhanced electron beam CT and comparison with pulmonary angiography. *Radiology* 1995; 194: 313–9.

2 Primack SL, Müller NL. High-resolution computed tomography in acute diffuse lung disease in the immunocompromised patient. (review). *Radiol Clin N Am* 1994; 32: 731–44.