Lung abscess: a neglected cause of life threatening haemoptysis

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Abstract

Three cases who presented with life threatening haemoptysis are reported, all of whom required surgery to control the bleeding. In all three patients chronic lung abscess was responsible for the haemoptysis. Even in the absence of typical clinical or radiographic features of an abscess this diagnosis should be considered in any patient presenting with life threatening haemoptysis.

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Lung abscess is now an uncommon condition in industrialised countries with only eight cases per year identified in one centre in the USA1 and only six per year at a British centre.2 The typical presentation is either as an acute illness with cough, sputum and chest pain, or more insidiously with weight loss and general malaise.3–5 The diagnosis should be considered in the presence of such symptoms associated with chest radiographic shadowing when a predisposing cause is identified such as a period of unconsciousness during which aspiration of oropharyngeal secretions may have occurred. Minor haemoptysis is a common symptom6 but it is often forgotten that life threatening haemoptysis can occur.7 We present three previously fit patients who presented with massive haemoptysis resulting from lung abscess.

Case 1

A 43 year old ex-smoker was admitted after two major haemoptyses of at least 500 ml on each occasion which recurred after admission. He gave a history of minor haemoptyses one month before admission; at that time chest radiographic and bronchoscopic examinations were normal. The chest radiograph on admission showed an area of consolidation 2.5 cm in diameter in the left posterior basal segment. Bronchoscopic findings were normal. A bronchial arteriogram showed abnormal vasculature with a blush appearing in the region of the opacity, but a pulmonary arteriogram was normal with no evidence of pulmonary embolism. Computed tomography showed a 3 cm well demarcated cavitating mass with patchy consolidation abutting on the pleura in the left lower zone. He was treated by left lower lobectomy. Histological examination showed an abscess cavity, considerable parenchymal scarring, chronic inflammation, and localised bronchiectasis. There was no evidence of malignancy. One year later the patient is well with no recurrence of his haemoptysis.

Case 2

A 21 year old woman presented with a five day history of haemoptysis and shortness of breath. She had a 10 year history of recurrent minor haemoptyses for which no cause had been found despite extensive investigation. Two years previously she had had an episode of pneumonia. On the day before admission she had further haemoptysis with left basal pleuritic chest pain and breathlessness. She had no fever and no calf pain. A diagnosis of recurrent pulmonary embolism was made and she was given intravenous heparin.

On examination she was breathless at rest (respiratory rate 30/min) with a tachycardia. There was no evidence of venous thrombosis of the leg veins. Her haemoglobin level was 13.6 g/dl. The chest radiograph showed left basal consolidation. Electrocardiography showed a sinus tachycardia but no right heart strain and echocardiographic findings were normal. Arterial blood gases on air were: PaO2 6.1 kPa, PaCO2 4.6 kPa, pH 7.37. There was no evidence of a systemic vasculitis or a mycetoma.

The hypoxia improved with continuous positive airways pressure but she later deteriorated suddenly after a coughing spasm; oxygen saturation fell to 35% despite inspired oxygen of 100% and continuous positive airways pressure of 20 cm H2O. She was intubated but the left hemithorax failed to expand. Urgent bronchoscopy revealed a clot occluding the left main bronchus which was evacuated and a tachycardia. Anticoagulation was reversed. Computed tomography showed a 4 × 2 cm irregular mass in the posterior segment of the left lower lobe. Left lower lobectomy was performed two weeks later and an irregular 3 cm mass was found. Histological examination of the lesion revealed an abscess cavity in communication with the bronchus which was filled with fungal hyphae thought to represent secondary infection of the abscess cavity. She made an uneventful recovery.

Case 3

A 57 year old previously fit Nigerian man gave an eight day history of haemoptyses associated with right sided pleuritic chest pain. He had been in Nigeria until four days before admission and had received unknown antibiotics there. He was a non-insulin dependent diabetic and had never smoked.

On examination he was pyrexic (39°C) with a tachycardia. In the chest there was dullness to percussion and reduced air entry at the right lower zone anteriorly and in the axilla. Chest radiography showed consolidation of the middle lobe. The haemoglobin level was
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11.8 g/dl and the white cell count was 12.4 x 10^9/l. Bronchoscopy showed inflamed bronchial mucosa only. Three weeks later he was well and the radiological changes were improving.

A month later he presented with major haemoptysis associated with the production of foul sputum. The chest radiograph showed persistent consolidation in the middle lobe (fig 1). He had developed clubbing and was febrile at night. His haemoglobin level was 7.8 g/dl and erythrocyte sedimentation rate was 80 mm in the first hour. Sputum culture (including anaerobic culture) and cytological examination gave negative results.

Computed tomography confirmed segmental middle lobe consolidation with an air bronchogram and cavitation (fig 2). He was treated with intravenous ampicillin and erythromycin, and a blood transfusion, and his symptoms improved. However the chest radiograph remained unchanged and, in spite of the radiological appearances which suggested cavitating consolidation, the changes were presumed to be caused by a squamous carcinoma. While awaiting surgery he had a major haemoptysis of about one litre of blood. He had two further large haemoptyses requiring six units of blood to resuscitate him before emergency thoracotomy and middle lobectomy. Histological examination showed a 2 cm cavity within the lung parenchyma and a few dilated bronchi. The abscess cavity was lined with granulation tissue with signs of old and recent haemorrhage and evidence of acute and chronic inflammation but no vasculitis, granuloma, or malignant cells. He made a good recovery and has had no further haemoptysis.

Discussion

All three patients presented with severe haemoptysis. The first had a history of cough and recurrent small haemoptyses but his chest radiograph during this period was normal. The second had a long history of recurrent small haemoptyses and when she presented was thought to be suffering from pulmonary embolism. She was anticoagulated which almost certainly made her condition worse. The third had a history of unresolved pneumonia, thought to be due to an underlying carcinoma in spite of the fact that he was a non-smoker. None of the patients had radiographic changes typical of lung abscess (thick walled cavity with air fluid level). Computed tomography showed cavitating consolidate in the first and third patient but did not exclude a carcinoma. In all these cases the diagnosis was not made until thoracotomy and examination of the surgical specimen.

It is difficult to estimate the number of cases of lung abscesses which present with haemoptysis. These three patients presented to a single cardiothoracic unit over a period of one year and suggest that lung abscess is probably a neglected cause of massive haemoptysis. The diagnosis of lung abscess should be considered in any patient with massive haemoptysis, particularly if there is a past history of recurrent “sentinel” haemoptysis or pneumonia and undiagnosed lung shadowing. If the haemoptysis becomes life threatening early surgical intervention is indicated.

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Figure 1 Chest radiograph of case 3 two months after presentation showing middle lobe consolidation.

Figure 2 Computed tomogram of case 3.