

Recurrent life-threatening haemoptysis in pulmonary tuberculosis – the importance of pulmonary angiography

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Introduction

Life-threatening haemoptysis in patients with pulmonary inflammatory disease is usually due to bleeding from enlarged bronchial arteries.¹ Bronchial arteriography usually identifies the bleeding site which can be embolised at the same time.² Recurrence of haemoptysis may be due to incomplete embolisation, the presence of undetected systemic collaterals or the presence of a leaking pulmonary artery (Rasmussen) aneurysm.² We present a case of recurrent haemoptysis from a bleeding pulmonary artery aneurysm that was successfully treated by endovascular occlusion.

Case report

A 17-year-old girl from a poor

socio-economic background was referred as an emergency to the cardio-thoracic unit with a history of massive haemoptysis of at least 500 ml over 24 hours. A week earlier she had been admitted to a local hospital with a history of cough, night sweats, loss of appetite and loss of weight. She had minor haemoptysis at that time. On admission the patient was noted to be dyspnoeic and pale. The full blood count revealed a haemoglobin level of 6.6 g/dl. The chest radiograph demonstrated a miliary pattern almost certainly representing tuberculosis. A high-resolution computed tomography scan (HRCT) confirmed the miliary pattern in addition to a right upper lobe cavity and left lower lobe consolidation. Her HIV test was negative.

The patient was treated with oxygen, intravenous antibiotics and anti-TB drugs. The patient also received a blood transfusion and was booked for bronchial artery embolisation.

The bronchial and intercostal arteries were carefully studied. A common bronchial trunk gave rise to a single right bronchial artery and two

bronchial arteries on the left. The bronchial arteries and right superior intercostal artery were enlarged and demonstrated hypervascularity. Transcatheter embolisation of these vessels was performed using embospheres. Postembolisation images demonstrated successful occlusion of the distal branches.

The patient continued to have minor haemoptyses at a rate of 50 - 100 ml per 24 hours. A repeat angiogram showed occluded bronchial arterial branches. There was recanalisation of the right superior intercostal artery, which was successfully embolised once again. A left internal mammary artery was also embolised due to its hypervascularity. However the patient's haemoptysis increased to 1 000 ml over a 24-hour period. A post-contrast CT of the chest demonstrated an enhancing lesion in the left lower lobe (Fig. 1a). A selective pulmonary angiogram was performed demonstrating that this was a pseudoaneurysm arising from the left lower lobe pulmonary artery branch (Fig. 1b). The aneurysm was occluded successfully with steel coils (Fig. 1c).

The patient's haemoptysis completely resolved soon after, and the patient was discharged on anti-TB drug therapy.

Discussion

Although most patients with pulmonary inflammatory disease and haemoptysis bleed from the bronchial circulation, the pulmonary arterial circulation should be immediately evaluated if the bronchial angiogram is normal or if the patient does not show any improvement following embolisation.^{3,4} A Rasmussen aneurysm is a pseudoaneurysm of the

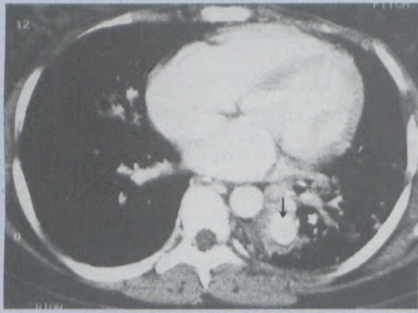


Fig. 1a. Contrast-enhanced CT of the lower chest demonstrates an aneurysm in the left lower lobe of the lung.

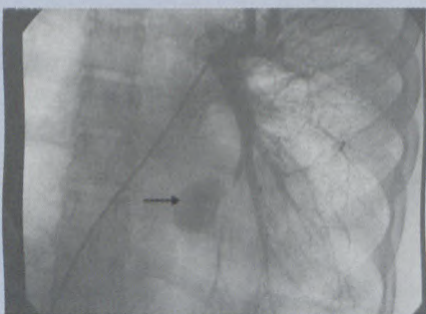


Fig. 1b. Rasmussen aneurysm of the left lower lobe pulmonary artery (arrow).

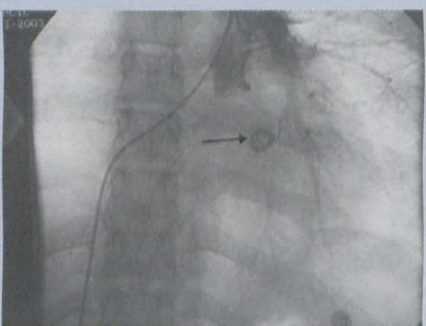


Fig. 1c. Occlusion of the aneurysm with steel coils (arrow).

terminal branches of the pulmonary artery within the wall of a tuberculous cavity caused by inflammatory necrosis or erosion of the vessel wall. In one series 4% of patients with cavitary tuberculosis had pulmonary artery

aneurysms detected at postmortem.⁵ Haemoptysis results from intimal rupture of the aneurysm. Bleeding into the cavity is expectorated or results in auto compression of the ruptured aneurysmal sac.

A Rasmussen aneurysm has to be systematically searched for in patients with haemoptysis from a destructive lung process. Bronchoscopy usually enables location of the haemorrhage to a specific lobe, which may allow directed angiographic evaluation; however fiberoptic bronchoscopy has some disadvantages in the diagnosis of massive, active haemoptysis. It is difficult to localise the bleeding site with fiberoptic bronchoscopy (FOB) in patients with massive haemoptysis because of excessive blood in the bronchi.

In a recent article, Hsiao *et al.*⁶ documented that fiberoptic bronchoscopy prior to bronchial artery embolisation is unnecessary in patients with haemoptysis of known cause if the site of bleeding can be determined on conventional imaging.

A pseudoaneurysm typically forms months to years after the initial exposure to tuberculosis or reactivation of the disease and the development of cavitary disease. A chest radiograph can demonstrate a well-defined opacity and a repeat study may show an increase in the size of the opacity. A post-contrast CT demonstrates a well-defined enhancing rounded structure, which often lies within the lung parenchyma adjacent

to the vessel, as demonstrated in our patient. A pulmonary angiogram confirms the diagnosis.

We recommend that in patients with haemoptysis, if the bronchial arteries are normal or if the embolisation does not stop the haemorrhage, pulmonary and systemic arteries (intercostals, subclavian, internal mammary arteries) should be studied. Such an approach is essential to avoid a delay in the diagnosis, as in our case. Transcatheter coil embolisation of the Rasmussen aneurysm is safe and very effective in stopping the haemoptysis.⁷ Detachable balloons and stainless steel platinum coils are generally used.

References

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