A tale of two embolisations

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Massive haemoptysis, defined as the expectoration of 300 - 600 ml of blood per day, is a frequent complication of acute and chronic pulmonary tuberculosis (PTB) (caused by Mycobacterium tuberculosis). Owing to epidemic HIV/AIDS in the South African population, PTB has flourished. Invariably, owing to their limited pulmonary reserve, such patients are poor surgical candidates.[1] Systemic arterial embolisation by particles or metallic coils is therefore the core management in many of these patients.[2] The haemoptysis usually originates from the bronchial arteries but rupture of an underlying Rasmussen aneurysm might cause recurrent haemoptysis, sometimes with fatal consequences.[2]

Case report

A 49-year-old man presented with a week-long history of haemoptysis, haematemesis and melena stools. He complained about loss of weight, a chronic cough and night sweats. The patient was known, with sputum-formation and eventual rupture.[5] In a study by Sanyika et al., 38% of patients who re-bled after successful bronchial artery embolisations for haemoptysis had Rasmussen aneurysms.[1] Haemoptysis is usually the presenting symptom and may be fatal when substantial.[5] Since 1973, the use of bronchial artery embolisation (BAE) has become customary in management of massive haemoptysis. Published mortality rates for elective surgery performed for massive haemoptysis are less than 20%. The mortality rate for emergency surgical control, however, escalates to about 40%.[4]

Pulmonary tuberculosis can affect the pulmonary arteries and veins, leading to vasculitis and thrombosis.[5] The bronchial arteries may enlarge and hypertrophy.[1] Rasmussen aneurysm is a rare complication (in 5% of autopsied patients with cavitatory tuberculosis[6]) caused by pulmonary artery wall weakening owing to the adjacent cavitatory tuberculosis.[6] Granulation tissue replaces the adventitia and media, leading to gradual weakening of the arterial wall.[5] The granulation tissue is replaced by fibrin, leading to thinning, pseudoaneurysm formation and eventual rupture.[5] In a study by Sanyika et al., 38% of patients who re-bled after successful bronchial artery embolisations for haemoptysis had Rasmussen aneurysms.[1]

The present case serves to emphasise the fact that, in cases of recurrent haemoptysis, complicating pulmonary TB, a CT angiogram of the pulmonary vessels should be considered, to exclude aneurysms that might lead to catastrophic haemoptysis. It furthermore illustrates the complementary role that particle and coil embolisation has to play in management of massive haemoptysis.

Chronic pulmonary tuberculosis may present as massive haemoptysis. Haemoptysis usually originates from the bronchial artery but the pulmonary artery might be the culprit vessel in recurrent haemoptysis. We present a case where bronchial artery embolisation had to be augmented by pulmonary artery coil embolisation for a Rasmussen’s aneurysm after recurrent haemoptysis. In cases where recurrent haemorrhage occurs, sources other than the bronchial artery should be considered.


**PICTORIAL INTERLUDE**

Fig. 1. Chest radiograph demonstrating cavitary pulmonary tuberculosis with marked destruction of the right lung.

Fig. 2. (a) Selective right bronchial angiogram (arrow) demonstrates a large, hypervascular lesion in the right upper lobe (arrow heads). (b) Post-particle embolisation angiogram reveals complete obliteration of the hypervascular region.

Fig. 3. (a) Axial contrast enhanced CT demonstrates filling of the Rasmussen aneurysm (arrow). The hyperdense ovoid structure in the oesophagus is a medicine tablet; (b) coronal reformation illustration of the aneurysm (arrow); (c) pulmonary angiogram demonstrates a Rasmussen aneurysm (arrow) in the right upper lobe posterior segment pulmonary artery; (d) post-embolisation pulmonary angiogram with occlusion of the vascular pedicle and exclusion of the Rasmussen aneurysm. The coils are indicated by the arrowheads.