Hemoptysis in pulmonary tuberculosis - contrast tomography unmasking Rasmussen’s aneurysm

George Peter* , Maroli Roshan

*Department of Medicine, Yenepoya Medical College, Mangalore, S. India
bDepartment of Medicine, Fr Muller Medical College, Mangalore, S. India

Abstract. Hemoptysis is a common symptom in respiratory clinical practice, but very rarely the images of Rasmussen’s aneurysm are demonstrated in a case of hemoptysis. In this report of a 53 year old man with massive hemoptysis, we are able to demonstrate a Rasmussen’s aneurysm by contrast enhanced computer tomography (CECT) of the thorax along with features of pulmonary tuberculosis.

Key words: Rasmussen’s aneurysm, massive hemoptysis, pulmonary tuberculosis, ‘tree-in-bud’ appearance, granuloma

1. Introduction

In 1868, Danish physician, Fritz Valdemar Rasmussen, described the rupture of aneurysm of pulmonary vessel passing through the wall of tuberculous pulmonary cavity as cause for hemoptysis (1). This pulmonary arterial aneurysm on the wall of the tuberculous pulmonary cavity was later named after him as Rasmussen’s aneurysm. The usual sources of hemoptysis in cavitary pulmonary tuberculosis are bronchial arteries. Rasmussen's aneurysm refers to an aneurysm of the small to medium pulmonary artery branches, which develops in the vicinity of a tuberculous cavity. Rasmussen's aneurysms are usually located peripherally and beyond the branches of main pulmonary arteries. We report this case of a 53 year old man who presented with massive hemoptysis.

2. Case report

A 53 year old man presented with history of hemoptysis of one day duration. On examination, he was hemodynamically stable, had pallor and coarse crepitations in left lung fields. His haemoglobin was 8.5 gm/dl and packed cell volume (PCV) was 33%, erythrocyte sedimentation rate (ESR) was 91 mm/hr, with normal blood counts and peripheral blood smear. His Chest X-ray showed a cavity in left mid zone, and shadows in the left lung fields. After admission to hospital, he had a bout of massive hemoptysis, and was given blood transfusions with supportive care for stabilisation. The patient did not have recurrence of hemoptysis after the initial episode.

A CECT scan of the thorax showed a thick walled cavity in left upper lung with a contrast enhancing vessel on its wall, suggesting to a Rasmussen’s aneurysm (Image-1). The scan also showed a tuberculoma in the right mid zone, and ‘tree-in-bud’ appearance of the left lower lobe of the lungs (Image-2). These findings were consistent with pulmonary tuberculosis.

Fig. 1. The CECT showing contrast enhancement on the wall of the cavity (white arrow) suggesting a Rasmussen’s aneurysm.
Meanwhile, his tuberculin skin test PPD$_3$ (Mantoux) was +20, sputum smear for acid fast bacilli and PCR for *Mycobacterium tuberculosis* were positive. He was started on anti tubercular treatment with isoniazid, rifampicin, ethambutol and pyrazinamide.

![Reformatted CECT of the chest showing granuloma (vertical white arrow) in the right side and 'tree-in-bud' appearance (horizontal white arrow) in the lung fields.](image)

**3. Discussion**

Hemoptysis due to rupture of Rasmussen’s aneurysm is one of the fatal complications of pulmonary cavitary tuberculosis (2). Pseudo aneurysms of pulmonary arteries are occasionally encountered in patients with tuberculosis of the lung. The hemoptysis in cavitary tuberculosis could be due to the granulomatus weakening of the pulmonary artery on the wall of the cavity (3) Rasmussen’s aneurysm.

The massive hemoptysis seen in the lung fibrosis and aspergillosis is from the rupture of hypertrophied bronchial arteries. An autopsy study by Auerbach in 1939, on 1114 patients who died of chronic pulmonary tuberculosis, only 4% (45 of 1114) were found to have pulmonary artery aneurysm (4).

The lungs have a dual blood supply. The pulmonary arterial circulation which is responsible for gas exchange is a high-compliance low-pressure system that terminates in the pulmonary capillary bed. Whereas the bronchial arteries, like other systemic arterial system is a high-pressure system. In contrast to the pulmonary circulation, the bronchial circulation has an ability to proliferate (5).

According to Remy et al, the source of massive hemoptysis is commonly the bronchial circulation (90% of cases) than the pulmonary circulation (5%) and about 5% of cases with massive hemoptysis originate from the aorta (e.g. aortobronchial fistula, ruptured aortic aneurysm) or non-bronchial systemic arteries (5).

A retrospective study of 189 patients with massive hemoptysis who were treated by endovascular means showed that only 13 patients (6.9%) had hemoptysis of pulmonary arterial origin(6). In the last decade, the main therapeutic mode to control bleeding in patients with massive hemoptysis secondary to tuberculosis is bronchial artery embolisation or coil clipping of aneurysm (7). In our report the patient was not considered for intervention as his hemoptysis stopped spontaneously.

**4. Conclusion**

Hemoptysis is common in respiratory clinical practice, but very rarely the images of Rasmussen’s aneurysm are demonstrated in a case of hemoptysis. In this report of a 53 year old man with massive hemoptysis, we are able to demonstrate a Rasmussen’s aneurysm by CECT of the thorax. In the CECT other features of pulmonary tuberculosis, such as multiple granulomas and ‘tree-in-bud appearance’ are demonstrated (8). The patient improved with anti tubercular treatment and supportive care.

Even though the gold standard for diagnosis of hemoptysis remains pulmonary angiography, we recommend CECT as a non-invasive, cost effective mode of investigation to screen a patient with massive hemoptysis.

**References**

