Case Report

Rasmussen's Aneurysm Associate With Pulmonary Artery Vascular Malformation Presented With Haemoptysis

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Abstract
Rasmussen's aneurysm is an inflammatory pseudo-aneurysmal dilatation of a branch of pulmonary artery associated with a cavitary lung lesion. Like any aneurysm, a Rasmussen's aneurysm is at increased risk of rupture and bleeding into the lungs. A 52 years old male presented with low-grade fever and haemoptysis, chest x-ray revealed a well margined nodular shadow with calcifications in mid zone of left lung associated with ipsilateral upper zone fibrosis and bronchiectasis. The patient had past history of pulmonary tuberculosis eight years back. For characterization of left pulmonary nodule the patient was referred to radiology and Imaging department and contrast CT scan of chest was done. Contrast CT scan of chest with reformat MIP pulmonary angiogram revealed a small pulmonary artery vascular malformation in lateral basal segment of lower lobe of left lung associated with left upper lobar cavitating lesion with fibrosis, traction bronchiectasis and Rasmussen's aneurysm arising from upper lobar apical segmental pulmonary artery. Early surgical or angiographic interventions with endovascular embolization are recommended once it be clearly diagnosed.

Keywords: Hemoptysis, Pulmonary tuberculosis, Rasmussen's aneurysm.

Introduction
Rasmussen's aneurysm, named after Fritz Valdemar Rasmussen, is an aneurysm arising from the pulmonary artery adjacent to or within a tuberculous cavity. The term is now used for the anatomic aneurysm associated with other destructive lung lesions. Although it is reported in 5% of autopsies of patients with tuberculous cavities, only a few clinical case reports exist in literature. In patients of tuberculosis after an active infective etiology of hemoptysis has been ruled out, the cause of hemoptysis is generally looked for in the bronchial arteries. Bronchial arteries in such a patient gets hypertrophied and enlarged and show rich broncho-pulmonary communications, which are the usual source of bleed. Rasmussen's aneurysm, if present, can be another treatable cause of hemoptysis in such patients. We report one such patient who had recurrent hemoptysis, on evaluation by CT scan of chest with reformat pulmonary angiogram was found to have Rasmussen's aneurysm arising from the pulmonary artery. Incidentally a small pulmonary artery vascular malformation also detected in lateral basal segment of lower lobe of left lung which have feeding vessel calcification causing 80% luminal obstruction.

Case report
Present case, 52 years old man, was presented with one month history of cough, purulent yellowis sputum and haemoptysis. His medical history was marked by pulmonary tuberculosis eight years back. On admission, he had a body temperature of 38.80°C. Physical examination was remarkable only for inspiratory crackle over the left lung. The routine laboratory data revealed an elevated white blood cell count of 16000 cells/mm3 and hemoglobin 10 g/dl. Chest x-ray revealed a well margined nodular shadow with calcifications in mid zone of left lung associated with ipsilateral upper zone fibrosis and bronchiectasis (Illustration 1).

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For characterization of left pulmonary nodule the patient was referred to radiology and Imaging department and contrast CT scan of chest was done. With reformat pulmonary artery angiogram revealed a small pulmonary artery vascular malformation which had feeding vessel calcification causing 80% luminal obstruction in lateral basal segment of lower lobe of left lung associated with left upper lobar cavitating lesion with fibrosis, traction bronchiectasis and Rasmussen's aneurysm arising from upper lobar apical segmental pulmonary artery (Illustration 2). The coronal reformatted CT scan image demonstrated the existence of aneurysm (25mm in diameter) originating from upper lobar pulmonary artery indicating a Rasmussen's aneurysm and a small pulmonary artery vascular malformation with marked intralesional calcifications which have feeding vessel calcification causing 80% luminal obstruction in lateral basal segment of lower lobe of left lung (Illustration 3). The presumptive diagnosis of tuberculosis was confirmed by sputum smear examination for acid-fast bacilli and PCR for Mycobacterium tuberculosis. Conservative management was proposed, because the patient's hemoptysis had ceased and there was no sign of contrast extravasation to indicate active bleeding.

Illustration 1: Chest x-ray A/P view showing, well margined nodular shadow with calcifications in left mid zone associated with ipsilateral upper zone fibrosis and bronchiectasis which was also revealed in HRCT scan.

Illustration 2: Contrast enhanced CT of chest in axial plane showing, left upper lobar cavitating lesion with fibrosis, traction bronchiectasis and aneurysm arising from upper lobar apical segmental pulmonary artery

The patient started standard antituberculous treatment. In our case, the aneurysm was small, hemoptysis was self-limiting and there was no sign of active bleeding. In such instances, conservative management with antibiotics and a close follow-up documenting resolution. Otherwise, endovascular aneurysm occlusion might be indicated.

Illustration 3: Contrast enhanced CT in coronal plane showing pulmonary artery vascular malformation.

Discussion

Hemoptysis is a known complication of active or old burnt-out pulmonary tuberculosis. Mild hemoptysis in a patient with history of old treated tuberculosis of lungs is usually secondary to tubercular reactivation or secondary bacterial or Aspergillus infection of a tuberculous cavity. Chronic bronchitis or bronchiectasis in such patients could also be responsible for hemoptysis. Massive hemoptysis in them is often secondary to a vascular complication. Such massive hemoptysis usually originates from the bronchial circulation (95%) rather than pulmonary circulation. Bronchial arteries in such patients with chronic inflammation get hypertrophied and lead to development of broncho-pulmonary and arterio-venous communications, which are the common sources of bleed. Rasmussen's aneurysm in patients with cavitary tuberculosis is caused due to gradual weakening of adjacent pulmonary arterial wall. The granulation tissue replaces the adventitia and media of the pulmonary arterial wall, which is in turn replaced by fibrin, leading to thinning and pseudoaneurysm formation. Being rare, Rasmussen's aneurysms are not looked for and so are often missed. CT pulmonary angiography is the investigation of choice to diagnose them and should be done in patients with recurrent hemoptysis, especially if there is recurrence after prior bronchial artery embolization. Our case emphasizes this fact, as CT pulmonary angiography in this case was crucial in diagnosing Rasmussen's aneurysm, which would have been otherwise missed. Embolization procedure technique for treating Rasmussen's aneurysm needs special care, as being pseudoaneurysm, they are very prone to rupture and bleeding during the procedure.
Conclusions
Rasmussen's aneurysm is a rare and often missed cause of hemoptysis in patients with tubercular lung cavities. CT pulmonary angiography is the best investigation to confirm their existence and should be done in patients with recurrent hemoptysis, especially if it happens after bronchial arteries have been successfully embolized.

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