MASSIVE HAEMOPTYSIS IN A PULMONARY TUBERCULOSIS PATIENT: A CASE REPORT OF RASMUSSEN'S ANEURYSM

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ABSTRACT: Haemoptysis can occur as a complication or sequelae in pulmonary tuberculosis. A 52 year old patient presented with progressive cough, breathlessness, fever and haemoptysis. On evaluation, he was diagnosed with multidrug resistant tuberculosis (TB) with Rasmussen's aneurysm being the cause of his haemoptysis.

KEYWORDS: Tuberculosis, Rasmussen's aneurysm.

INTRODUCTION: Tuberculosis usually affects the lungs, although other organs are involved in upto one-third of cases.[1] Massive haemoptysis is a serious complication of pulmonary tuberculosis. We present a case report of a patient of massive haemoptysis which was later proven to be a case of Rasmussen's aneurysm.

CASE REPORT: A 52 years old male patient, manual labourer by occupation, presented with complaints of persistent cough and progressive breathlessness for seven months, fever for one month and massive haemoptysis since two days. Patient was a chronic smoker and diabetic on irregular treatment. Patient had completed RNTCP Category I anti-tubercular treatment (Under Revised national Tuberculosis Control Programme) one month ago for pulmonary Koch’s. On general physical examination, patient was anaemic. On chest examination, chest wall retraction was present in right infraclavicular area. Rhonchi were present over all lung fields along with crepitations in right infraclavicular and mammary areas. Chest X ray showed bilateral diseased lung with heterogenous opacity in right upper zone (Fig. 1). Lab investigations showed a haemoglobin of 9.5gm%, TLC 10,600/mm3, platelet count 2.2 lakh/mm3. Renal, liver function tests as well as coagulation profile were in the normal range.

![Fig. 1: Chest X-ray showing a heterogenous opacity in Right upper lobe](image-url)
Patient came sputum positive for Acid Fast Bacilli (AFB) and Line Probe assay (LPA) showed resistance to Rifampicin and Isoniazid. He was treated with DOTS PLUS regimen for MDR TB and symptomatically for haemoptysis. But haemoptysis was persistent. Contrast enhanced computerised tomography (CECT) of the chest showed bilateral emphysematous changes with consolidation and cavity formation in right upper lobe which had a focal dilatation not changed by positional variation (Fig 2). Multidetector computerised tomography angiography (Fig. 3a and 3b) revealed a pseudoaneurysm in one of the segmental branches of pulmonary artery in right upper lobe cavity, suggestive of Rasmussen's aneurysm.

DISCUSSION: Haemoptysis usually arises from bronchial arteries, but in case of tubercular lesions, new and collateral vessels develop from systemic circulation.\(^2\) Rasmussen's aneurysm is an uncommon complication of pulmonary tuberculosis and represents an a pulmonary artery aneurysm adjacent or within a tuberculous cavity.
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It is caused by weakening of the pulmonary artery wall from adjacent cavitary tuberculosis. There is progressive weakening of the arterial wall as granulation tissue replaces both the adventitia and the media. This is then gradually replaced by fibrin, resulting in thinning of the arterial wall, dilatation (Pseudoaneurysm formation) and subsequent rupture with haemorrhage. It can present as repeated episodes of mild or moderate haemoptysis or massive haemoptysis as in our case. In CECT Chest, it’s seen as a focal dilatation of one of pulmonary segmental arteries adjacent to tuberculous parenchymal change or chronic tuberculous cavity. Embolisation[3] and surgery are very effective measures to control bleeding. Embolisation of the aneurysm is a temporary measure and bleeding can recur. Surgical excision is recommended in patients with severe destructive process as in our case.

CONCLUSION: Massive haemoptysis in a pulmonary tuberculosis patient should always be investigated to rule out Rasmussen’s aneurysm which needs aggressive management.

REFERENCES:

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