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Rasmussen Aneurysm Revealed by Hemoptysis: About Two Cases

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Abstract **Case Report**

Hemoptysis in cases of tuberculosis sequelae most often has a bronchial arterial origin. The pulmonary arterial origin is observed in cases of false Rasmussen's aneurysm. We report two cases of patients admitted for a hemoptysis of great abundance in a context of altered general condition; fever and chills with thoracic scans that revealed false Rasmüssen aneurysms.

Keywords: Hemoptysis; Aneurysm; Embolization.

INTRODUCTION

Hemoptysis is the discharge of bright red, aerated, foamy blood from the subglottic airways through the mouth and sometimes nose during coughing effort. Severe hemoptysis is a potentially life-threatening condition with an estimated mortality rate of more than 50% in the absence of adequate treatment. It usually involves bleeding of systemic bronchial origin, or less frequently of systemic non-bronchial origin [1,2]. We report two cases of Rasmüssen Aneurysms revealed by profuse hemoptysis.

OBSERVATION: FIRST CASE

The patient was 62 years old, a gardener, with a history of chronic smoking (30 pack-years) and pulmonary tuberculosis five years ago, treated for six months on the usual regimen and declared cured. He presented two weeks before his admission the occurrence of dyspnea stage II of Sadoul, followed a few days later by profuse hemoptysis. These signs were accompanied by severe chest pain, dizziness, fever, sweating and altered general condition. Clinical examination revealed moderate paleness of the conjunctival mucosa, performance status 3, digital hippocratism and bilateral snoring. Chest X-ray revealed bilateral rail opacities with left parahilar-projecting opacity of heterogeneous dense vascular appearance, speculated with reticulated opacity at the apices (Fig.1). Thoracic angioscanner showed no pulmonary embolism, an emphysematous lung, and bronchial dilatations of sequellar appearance associated with a thrombosed false aneurysm in the left Fowler (Fig.2). The radio-clinical picture suggested a false Rasmüssen aneurysm. Further investigations were undertaken as part of an etiological work-up. The GeneXpert test in expectoration was negative. The immunological work-up was normal. The haemogram showed anemia and inflammatory markers (CRP and VS) were elevated.

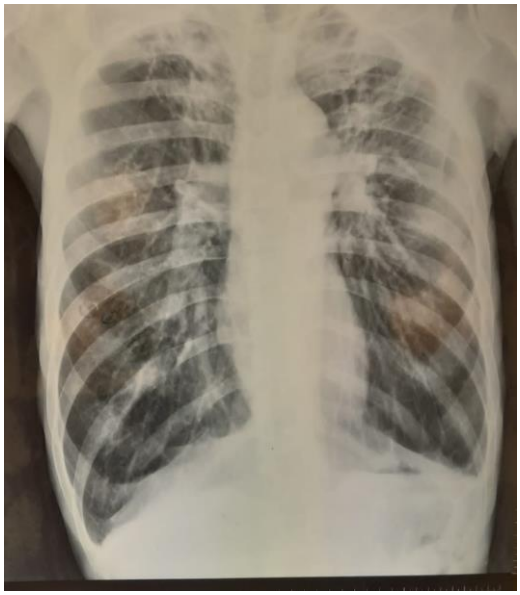

Figure 1: Bilateral rail opacities with left parahilar-projecting opacity of heterogeneous dense vascular appearance, speculated with reticulated opacity at apices, all on distended lung

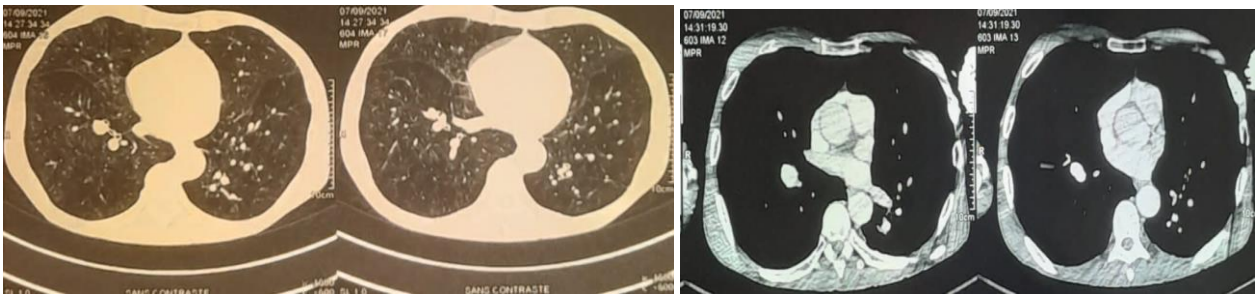

Figure 2: Absence of pulmonary embolism. Emphysematous lung with sequelae of bronchial dilatation associated with a thrombosed false aneurysm in the left Fowler

OBSERVATION: SECOND CASE

The patient was 61 years old, bricklayer, with a history of chronic smoking at rate of 04 pack-years, weaned 12 years ago, and pulmonary tuberculosis 20 years ago, treated for six months according to the regimen and declared cured. He presented for about 4 months before his admission a left laterothoracic painof moderate-intensity without particular radiation, associated with a dyspnea stage II of Sadoul, followed a few days later by a productive cough producing whitish sputum with copious hemoptysis. Clinical examination revealed mild pallor of the conjunctival mucosa, and general good health. Pleuropulmonary examination was unremarkable. Chest X-ray revealed a heterogeneous left upper lobar opacity with a blurred border and no costal lysis opposite, an emphysematous lung with a drop-heart appearance (Fig. 3). The thoracic CT scan showed destruction of the culmen with multiple cystocylindrical bronchiectasias and cavitary images with apical ovoid liquid formation with multiple bilateral bronchiectasias suggestive of a Rasmussen aneurysm (Fig.4). The radio-clinical picture suggested a Rasmüssen aneurysm. Additional investigations were undertaken as part of an etiological work-up, in particular bronchoscopy, which revealed 1st-degree inflammation with thickening of the inter-lobar spur and the presence of anthracosic spots in the left upper lobar. Aspirations were performed for Koch bacilli, GeneXpert coming negative with inflammatory cytology. Gene Xpert and Koch bacilli in expectoration test were negative, aspergillary serology negative. Blood count and inflammatory markers (CRP and VS) were normal.

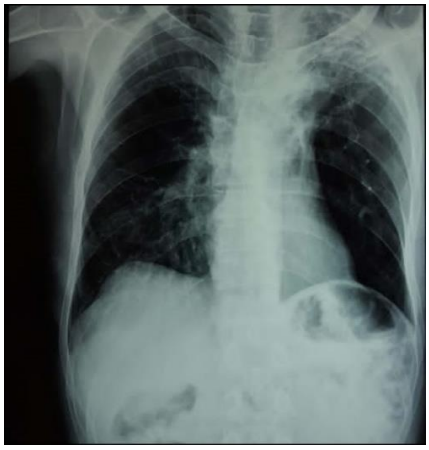

Figure 3: Thoracic distension with heterogeneous left upper lobar opacity with blurred border and no costal lysis opposite

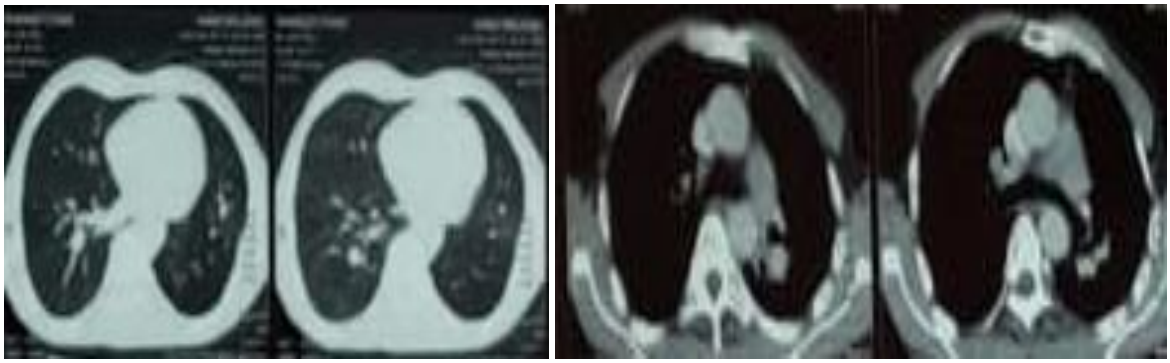

Figure 4: Destruction of the culmen with multiple cysto-cylindrical bronchiectasias and cavitary images with ovoid apical liquid formation with multiple bilateral bronchiectasias suggestive of bilateral Rasmussen's

DISCUSSION

Hemoptysis is a potentially life-threatening condition, with a high mortality rate in the absence of adequate treatment. It usually involves bleeding of systemic bronchial origin, or less frequently of systemic non-bronchial origin [1]. Hemoptysis of pulmonary arterial origin remains rare, estimated at less than 10% of hemoptysis causes, and of variable causes: post-infectious pseudoaneurysms within the framework of necrotizing lung diseases such as tuberculosis (Rasmussen aneurysm) and septic emboli [2]. Unlike true aneurysms, notably Behçet's disease [3], which result from focal dilatation of the three arterial layers. We report two cases of false Rasmussen's aneurysms occurring in the context of pulmonary tuberculosis, notably a history of pulmonary tuberculosis and tuberculosis infection with signs of tuberculosis impregnation. First described by Rasmussen in 1868 [4], false pulmonary artery aneurysms complicating tuberculosis have become increasingly rare with the widespread use of antibiotics. In 1939, Auerbach and al [5] found a prevalence of 4% in an autopsy study of 1114 subjects with chronic cavitary infection. In 1956, Plessinger and al. published a series of 56 cases of Rasmüssen aneurysm. In 47 cases (87%), massive pulmonary arterial hemorrhage was responsible for death [6]. This pseudoaneurysm results from erosion of the pulmonary artery wall by the inflammatory granuloma, which thins it and replaces the adventitia and media with fibrin. The fragility of this material leads to the formation of the pseudoaneurysm and its subsequent rupture, resulting in hemoptysis that is often abundant, devastating and potentially fatal [7].

In our series, the diagnosis was made on thoracic CT scan. Thus, the injected CT scan is the reference examination for the etiological and pathophysiological diagnosis of hemoptysis, and helps guide the clinician as to the type of angiography to be performed: either bronchial arteriography or selective pulmonary angiography. Treatment consists of embolization. The equipment used to embolize pulmonary artery aneurysms varies from team to team. Coil vaso-occlusion is the most frequently used, but some centers report the use of biological glue, detachable balloons, covered stents or sclerosing agents. Embolization may be performed on the afferent artery only. Post-embolization complications remain low, although there is a risk of aneurysm rupture, which can be fatal during the endovascular procedure, particularly if an attempt is made to occlude the aneurysm sac. After embolization of the false aneurysm, there is a risk of recurrence of bleeding in cases of mixed vascularization or aneurysmal repermeabilization.

CONCLUSION

False Rasmussen's aneurysm is a rare complication, often revealed by hemoptysis. A CT scan with contrast injection is essential for making the diagnosis. Thoracic CT scan is recommended even in the absence of hemoptysis when there are significant changes in lung parenchyma, with condensation, necrosis and cavitary lesions. In the event of hemoptysis and ineffective embolization, the procedure should be continued with pulmonary angiography. Endovascular treatment of false aneurysms, if performed rapidly, is generally effective, with a limited complication rate.

Declarations of interest

The authors declaring that they have no conflicts of interest in relation to this article.

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