Spontaneous Regression of a Ruptured Rasmussen's Aneurysm Causing Massive Hemoptysis in a Patient with Pulmonary Tuberculosis: A Case Report

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ABSTRACT

Tuberculosis is a global disease with a high prevalence rate in the Philippines. Frank hemoptysis often occurs later in the disease and is usually not massive since the availability of anti-Koch's treatment. However, Rasmussen's aneurysm, a pulmonary vascular complication secondary to tuberculosis from the weakening of the pulmonary arterial wall adjacent or within a tuberculous cavity, can be an uncommon cause of massive and potentially fatal hemoptysis.

A 35-year-old male patient presented with episodes of hemoptysis while being treated for pulmonary tuberculosis for two weeks. An episode of massive hemoptysis of ~400ml prompted his admission. Chest tomography with contrast showed bronchiectatic changes, cavitary formation, and an aneurysmal dilatation of the anterior segmental artery of the left upper lobe. He was diagnosed with Rasmussen's aneurysm. A multidisciplinary team consisting of pulmonologists, interventional radiologists and thoracic surgeons planned for a surgical intervention as coil embolization was deemed to be difficult due to the wide neck character of the aneurysm. On re-admission after patient optimization, repeat chest tomography showed interval regression of pulmonary cavity with thrombosis of the previously identified Rasmussen's aneurysm. Patient completed his 6-month antitubercular treatment with no further episodes of hemoptysis.

In patients with tuberculosis, hemoptysis results from involvement of the parenchyma, bronchiectasis, or erosion of residual cavities. Hemoptysis from the rupture of a dilated vessel such as Rasmussen's aneurysm is a rare cause. Chest tomography with contrast is the imaging modality of choice as it demonstrates the focal pulmonary artery dilatation. Embolization or surgical lobectomy are typically utilized to control the bleeding. However, treatment with anti-tuberculous regimen may result already in regression and eventual thrombosis of the aneurysm. Watchful monitoring is imperative as massive hemoptysis may recur; radiologists and surgeons must be available at any time in case intervention is required.

Keywords: hemoptysis, tuberculosis, Rasmussen's aneurysm

INTRODUCTION

The Philippines is identified as one of the high burden countries with pulmonary tuberculosis in the world by the World Health Organization.[1] Along with 8 other countries, the Philippines accounts for almost 2/3 of all cases of tuberculosis worldwide.[1] Hence, this infection is encountered frequently in Filipino patients. Patients may present with primary and post-primary forms with adults commonly having the latter. Post-primary infection typically presents with cough, fever, and night sweats.

Hemoptysis can be a complication of pulmonary tuberculosis but it is normally self-limiting. Frank hemoptysis can occur but mostly later in the disease course and is rarely

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massive. When massive, tuberculous vascular lesions may be one of the causes with Rasmussen aneurysm identified as a rare phenomenon.[2] This vascular complication only occurs in 1 out of 14,000 patients and is a result of the erosion of the arterial wall leading to focal dilatation of a branch of the pulmonary artery secondary to chronic inflammation inside the tuberculous cavity.[3] As the hemoptysis may become life-threatening, timely work-up and definitive management are imperative. CT pulmonary angiography confirms the presence of the aneurysm seen as a well-defined, round opacity with hyperdense content.[4] Upon detection, intervention may involve transcatheter embolization or surgical lobectomy. Although surgical lobectomy is considered the definitive management of this condition, complications and mortality are significant.[5] In unstable patients, a minimally invasive technique with embolization may be considered as the standard first-line treatment option.[5] A multidisciplinary approach is crucial for the prompt identification and treatment of this tuberculosis complication.

OBJECTIVES

1. To present a case of a 35-year-old male with massive hemoptysis from Rasmussen's aneurysm with subsequent spontaneous regression.
2. To review the current literature in terms of clinical features, pathophysiology, diagnostic work up, and management of Rasmussen's aneurysm.

CASE PRESENTATION

A 35-year-old male presented with an episode of massive hemoptysis which prompted work-up at local health clinic two weeks prior to admission. Sputum work-up with direct sputum smear microscopy for *Mycobacterium tuberculosis* was positive hence the patient was started on anti-Koch's regimen. Patient was prescribed oral tranexamic acid tablets with noted resolution of the hemoptysis.

The patient was adherent to the anti-TB treatment regimen but continued to have occasional productive cough and weight loss. Two weeks after initiation of treatment, patient had massive hemoptysis, estimated at 400ml, with associated dyspnea prompting consult and admission in a tertiary hospital.

The patient was also a known asthmatic since childhood, on maintenance inhaler therapy of Salmeterol and Fluticasone. Patient was non-hypertensive and non-diabetic. He was a previous 5-pack year smoker and had quit for one year. He was a previous alcoholic beverage drinker. He had no history of use of illegal substances. The patient worked as a tricycle driver. He had no previous surgeries. He had a paternal family history of hypertension.

On physical examination on admission, patient was conscious and alert. Vital signs showed the following: blood pressure of 100/70 mmHg, heart rate of 129 beats per minute, respiratory rate of 26 cycles per minutes, temperature of 36˚C, and oxygen saturation of 97% on room air. Patient was noted to be pale with pale palpebral conjunctivae. Chest examination revealed distinct heart sounds, tachycardic, with regular rhythm. Coarse crackles were auscultated on both lung fields, mid to base, with occasional expiratory wheezing. Routine rapid point of care HIV screening for patients with tuberculosis was negative.

Chest radiograph showed findings suggestive of pulmonary tuberculosis with cavitary formation, apicopleural thickening, fibrosis, and cicatricial changes (Figure 1A). This was followed by a computed tomography of the chest with contrast enhancement which revealed extensive bronchi-

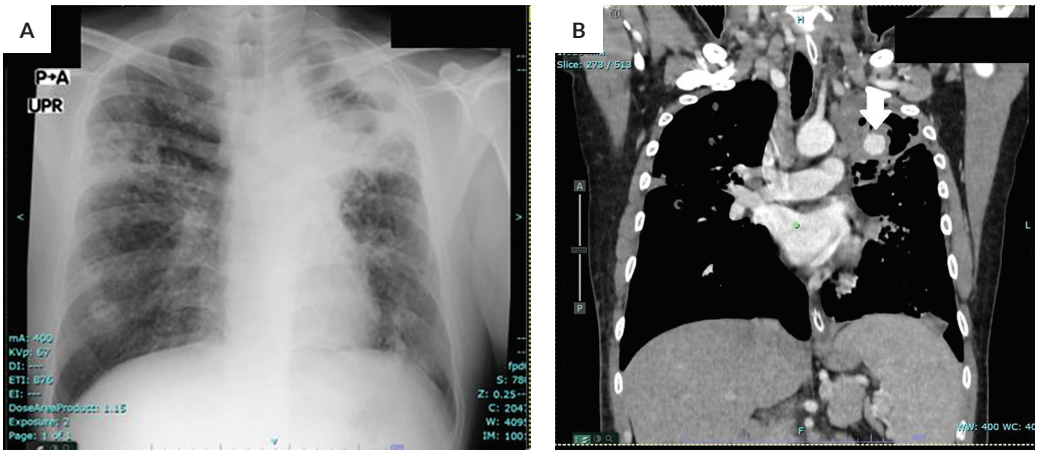

Figure 1. ***Imaging findings of the patient.*** **(A)** Initial chest radiograph showing reticulonodular opacities in both lungs with thick-wall air lucencies in the left upper lung field and right lower lung field. **(B)** Contrast-enhanced CT scan demonstrating a Rasmussen aneurysm arising from the left anterior segmental artery *(arrow)*.

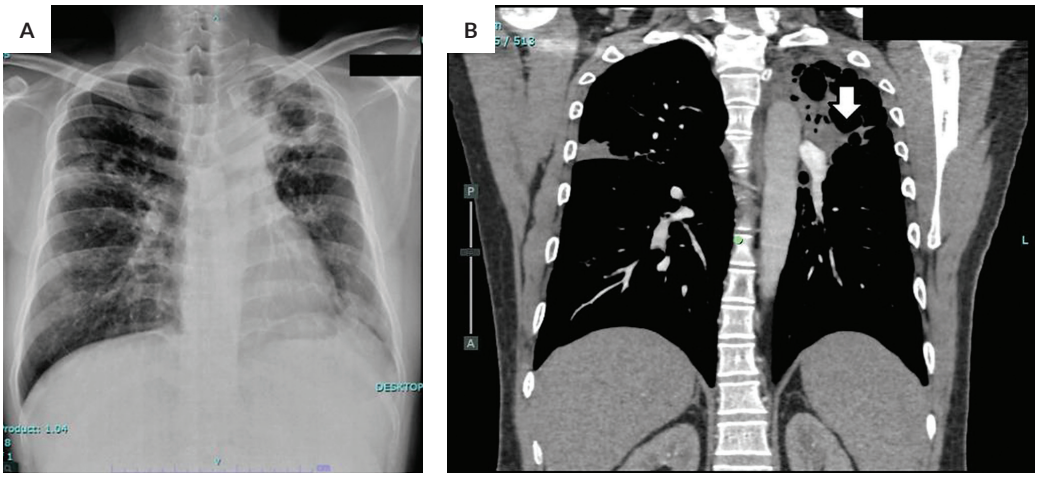

Figure 2. ***Subsequent imaging findings of the patient.*** **(A)** Chest radiograph showing interval regression of opacification of the left upper lobe. **(B)** Contrast-enhanced CT scan demonstrating a thrombosis of the previously visualized aneurysmal dilatation *(arrow)*.

ectatic changes, cavitary formation, and endobronchial spread suggestive of pulmonary tuberculosis. A Rasmussen aneurysm was also identified in the left upper lobe (Figure 1B). Hence, based on the patient's clinical findings and tests, patient was diagnosed with massive hemoptysis from active pulmonary tuberculosis, bacteriologically confirmed, with Rasmussen aneurysm.

Patient was admitted to the wards and transfused with 2 units of packed red blood cell with noted hemoglobin of 74mg/dL on complete blood count. Repeat hemoglobin post transfusion showed an increase to 93mg/dL. Hemoptysis was temporized with a combination of antitussive butamirate citrate and nebulization with alternating racemic epinephrine and tranexamic acid. Concomitant antibiotic therapy with ceftazidime and azithromycin for community-acquired pneumonia-moderate risk was also given. As localization of bleeding was identified and patient remained stable, bronchoscopy was not offered.

The patient was referred to Interventional Radiology and Thoracic Cardiovascular Surgery services for co-management. Ideally, a pulmonary angiogram should have been performed for a detailed visualization of the thoracic vasculature but was overlooked. Radiology service, however, reviewed the contrast enhanced CT scan and noted an aneurysmal dilatation with a size of 1.7cm x 1.8cmx 2.0cm with a wide neck. No appropriately sized coil was available at the time. Glue embolization was also not available. A surgical plan of possible left upper lobectomy was deemed suitable once patient was medically optimized. As the patient had no recurrence of hemoptysis during the rest of the course of his hospital stay, he was discharged and advised close follow-up.

Patient had sputum conversion after the 2nd month of intensive treatment. Patient also had monthly follow-up via teleconsultation with the medical service with no report of hemoptysis recurrence in the interim. Five months after the patient's initial admission, a repeat CT scan of the chest with contrast enhancement and pulmonary angiogram was done (Figure 2). Chest radiograph (Figure 2A) showed interval regression of the the opacification of the left upper lobe. Stents were now available, and the plan was for the patient to undergo pulmonary angiography, stenting of Rasmussen aneurysm, and a possible staged bronchial embolization. The CT scan showed lung findings compatible with pulmonary tuberculosis with extensive bronchiectatic changes, infected cavity and abscess formation, and endobronchial spread, with signs of disease regression. The previously identified Rasmussen aneurysm was noted to be thrombosed as emphasized by the arrow seen on Figure 2B. The plan for stenting was put on hold and monitoring was advised.

The patient was able to complete the 6-month anti-Koch's regimen with no recurrence of hemoptysis. On routine check-up with the pulmonary medicine service, the patient was able to return to baseline activities of daily living with no dyspnea or hemoptysis recurrence.

DISCUSSION

Pulmonary tuberculosis is the leading cause of hemoptysis especially in areas with higher tuberculosis prevalence, such as the Philippines. Caused by the *Mycobacterium tuberculosis* organism, this infection spreads through the inhalation of bacilli via droplet nuclei. The initial tissue reaction involves mobilization of the neutrophils at the site of infection where caseation necrosis then ensues.[4] The post-primary form of this disease can be progressive and depending on the interplay of host response and organism virulence, may result in a fulminant or disseminated form of infection.[2]

The hemoptysis in tuberculosis is typically seen in patients with extensive parenchymal involvement but can also occur even in inactive cases. In the latter, it may be caused by

bronchiectasis, bacterial or fungal infection in an old residual cavity, or erosion of a calcified lesion into the lumen of an airway.[6] Rarely, it is caused by the rupture of a dilated vessel in the wall of an old cavity or what is called a Rasmussen aneurysm. This occurs due to the progressive weakening of the arterial wall where the adventitia and media are replaced with granulation tissue and then by fibrin.[2] This eventually results in the segmental pseudoaneurysm formation which can rupture.[2] In a review of autopsy findings of patients with history of chronic cavitary tuberculosis, the prevalence of Rasmussen aneurysm was 5%.[4] However, case reports regarding this complication are limited.

In cases of massive hemoptysis, the primary aim is to stabilize and control the airway as it is a potentially life-threatening condition. In the work up of patients with massive hemoptysis, chest radiographs have limited value. The imaging modality of choice especially if a vascular etiology is considered is the computed tomography of the chest with contrast which can demonstrate the focal pulmonary artery dilatation.[6] Once stable, patients must undergo this radiographic imaging as this plays a crucial role in localizing the site and can identify the cause of bleeding in 70-88% of cases.[5] Treatment may involve endovascular management or surgical removal of the lesion.

Transcatheter embolization can be performed with a number of substances such as coils, glue, gel foam, balloons, stent grafts, or sclerosing agents.[4] Care must be given in choosing the appropriate intervention as for example, coils can lead to fatal rupture.[7] However, a study on the long term results of bronchial artery embolization did not show significant advantage of several techniques in comparison to each other such as stent-graft, coil packaging, or glue embolization.[8] The definitive treatment of choice is still surgery if the patient is operable. This, however, is not without complications as the mortality rate can be as high as 23% as documented in a case series of patients undergoing immediate operative management for massive, life-threatening hemoptysis.[7]

A case series of Rasmussen aneurysms demonstrated that in patients with mild hemoptysis, conservative management with antitubercular treatment may be done and result in hemoptysis resolution.[9,10] However, in patients with massive bleeding, patients typically undergo management such as bronchial artery embolization to control symptoms.[9] Aside from one other case of massive hemoptysis in a TB-COVID co-infection patient, there are limited reports detailing spontaneous regression of Rasmussen aneurysm through thrombosis of the aneurysmal dilatation.[10] The earlier case mentioned surmised that bleeding was hampered due to a local tamponade effect or thrombosis of the aneurysm possibly due to the hypercoaguable state of that patient.[11] In our patient, thrombosis of the aneurysm could have happened possibly due to the wide neck of the vascular complication. Experimental models in intracranial aneurysms have demonstrated that large orifices lead to blood stagnation, increased blood viscosity, and slow flow resulting in thrombosis.[11] As Rasmussen aneurysm are considered pseudoaneurysm, the vascular wall is not involved. Hence, leaking blood from the injured site may have been contained with products of the clotting cascade such as the fibrin and platelet crosslinks.[12] Further, this may have also been aided by the compliance of the patient to his antitubercular regimen. The control of the inflammation surrounding the vessel has been reported to aid in the regression in the size of tuberculous cavities.[2]

CONCLUSION

Massive hemoptysis from a ruptured Rasmussen aneurysm is a rare complication of pulmonary tuberculosis. The primary goal in unstable patients with this condition is stabilization and airway protection. If a vascular etiology is highly considered, the imaging of choice is a pulmonary angiogram. Once identified, intervention is based on the characteristics of the aneurysm and the condition of the patient. A multi-disciplinary team approach is important for the timely management of the patient. Spontaneous regression of the Rasmussen aneurysm with resolution of hemoptysis through conservative management may be a possible sequalae. Factors such as the intact vascular wall, characteristic wide neck of the aneurysm, and adherence to antitubercular treatment of the patient may have contributed to the thrombosis observed on repeat imaging of the Rasmussen aneurysm. However, as there is limited literature discussing spontaneous thrombosis in Rasmussen aneurysm, management with emergency angiographic embolization is still considered a first-line treatment.[13] Watchful monitoring of patients who cannot immediately undergo embolization is advised with the interventional radiologists and/or surgeons ready to intervene as appropriate.

Informed Consent

Written informed consent was obtained from the patient for the writing of this case report and any accompanying images, and publication in a journal.

Statement of Authorship

Both authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

Both authors declared no conflicts of interest.

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