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Case Report

Giant Rasmussen's aneurysm in a 9-year-old boy: A case report☆

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ABSTRACT

Rasmussen's aneurysm is a pseudoaneurysm caused by tuberculosis, when cavitation occurs adjacent to a pulmonary artery, which can be lethal. It is a rare complication usually affecting adults. This is a case of an 9-year-old boy initially admitted for pneumonia that developed hemoptysis during admission. Chest X-ray done after this episode showed development of a left hilar mass not present in the previous studies. A chest CT with contrast subsequently revealed a saccular aneurysm arising from the left lower lobe pulmonary artery adjacent to a cavity, a Rasmussen's aneurysm. The patient was treated conservatively with continuation of anti-TB medication and serial monitoring due to the size of the aneurysm being less than 6 cm and spontaneous resolution of the hemoptysis. This report stresses that a Rasmussen's aneurysm should always be in the differential diagnosis of a hilar mass in a patient with hemoptysis, regardless of the patient's age.

Background

Rasmussen's aneurysm (RA) is an inflammatory pseudoaneurysm of a branch of the pulmonary arteries caused by weakening or destruction of the arterial wall by an adjacent infectious process [1]. Classically, cavitation due to pulmonary tuberculosis (PTB) occurs juxtaposed to a branch of the pulmonary or bronchial arteries. Patients with RA usually present with hemoptysis, which could be massive and fatal. Diagnosis is made with either multidetector CT (MDCT) Angiography or conventional angiography. MDCT angiography provides rapid diagnosis and is noninvasive, making it the initial modality of choice. Conventional angiography, on the other hand, is invasive and more cumbersome. However, treatment with embolization or coiling can be done concurrently [2].

This is a case of a pediatric patient that presented with hemoptysis. RA was suspected on serial chest X-ray examinations. The importance of prompt recognition of RA, investigational modalities, and common therapeutic approaches are highlighted.

☆ Competing Interests: None.

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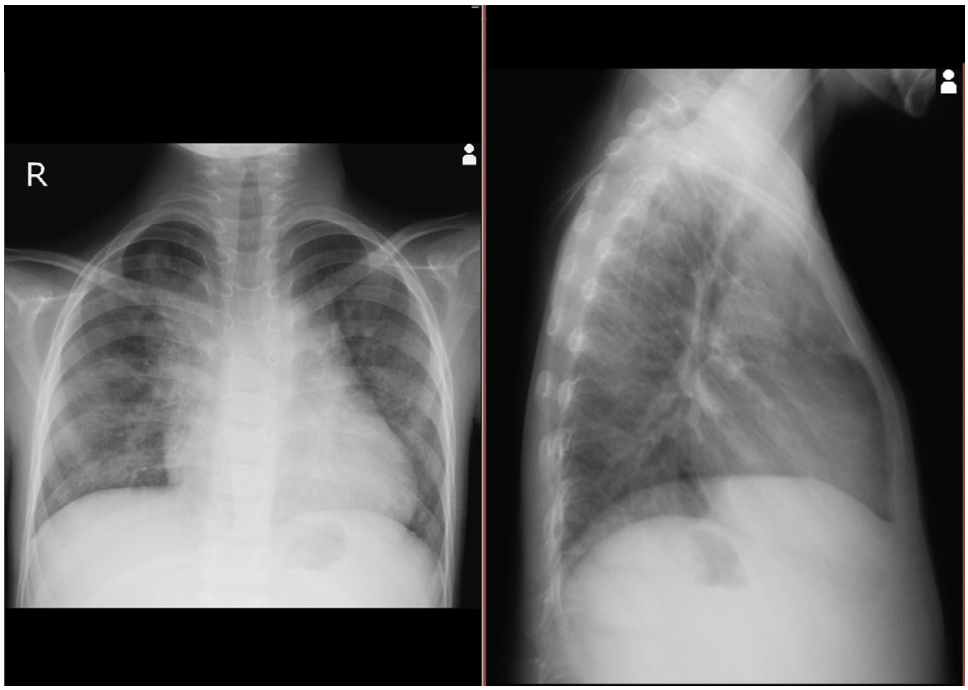

Fig. 1 – Chest X-ray at an outside institution. Perihilar haziness is noted, indicating pneumonia.

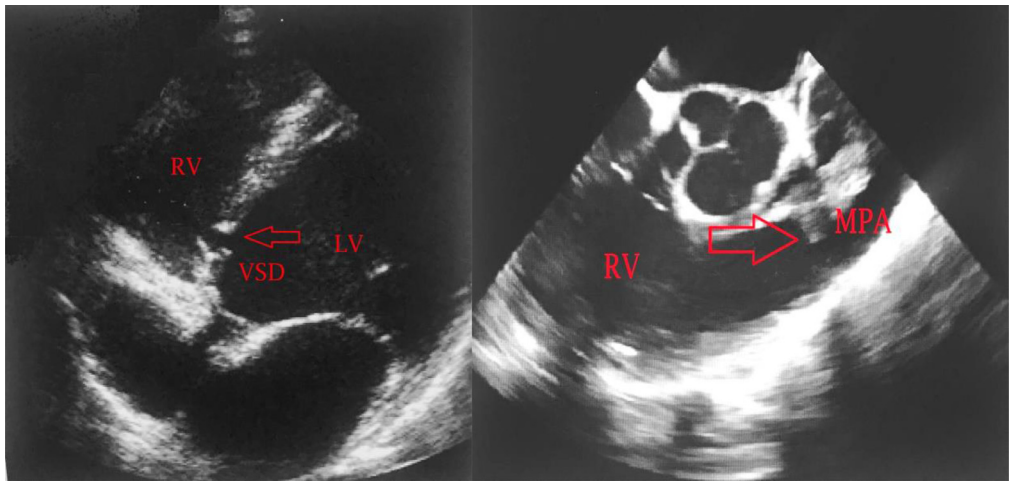

Fig. 2 – 2D echo at an outside institution. Perimembranous ventricular septal defect is shown in the left image and vegetation at the pulmonic valve is shown in the right.

Case report

An 9-year-old boy presented with a 3-day history of fever, cough, chest pain, difficulty of breathing, loss of appetite, and weight loss. Patient was brought to an outside institution hospital due to persistence of symptoms, where a chest X-ray and 2D echocardiogram were requested. Physical exam at the time revealed a holosystolic murmur. Chest X-ray (Fig. 1) showed pneumonia, biventricular cardiomegaly, and a prominent main pulmonary artery. 2D echo (Fig. 2) showed a perimembranous ventricular septal defect, moderate tricuspid regurgitation, minimal pericardial effusion, and vegetation at the pulmonic valve, a rare location occurring in only about 2% of cases [3]. The patient was admitted with a diagnosis of infective endocarditis and was started on antibiotics. After a week, the patient was transferred to our institution.

Upon admission, a chest X-ray (Fig. 3) showed hazy opacities in the right upper lobe, likely from consolidation pneumonia and cardiomegaly. Antibiotics were continued. After an unremarkable month of admission, the patient developed episodes of hemoptysis and hematemesis amounting to around 100 cc. Subsequent chest X-ray revealed development of a left hilar mass (Fig. 4). After a few days, the patient had an-

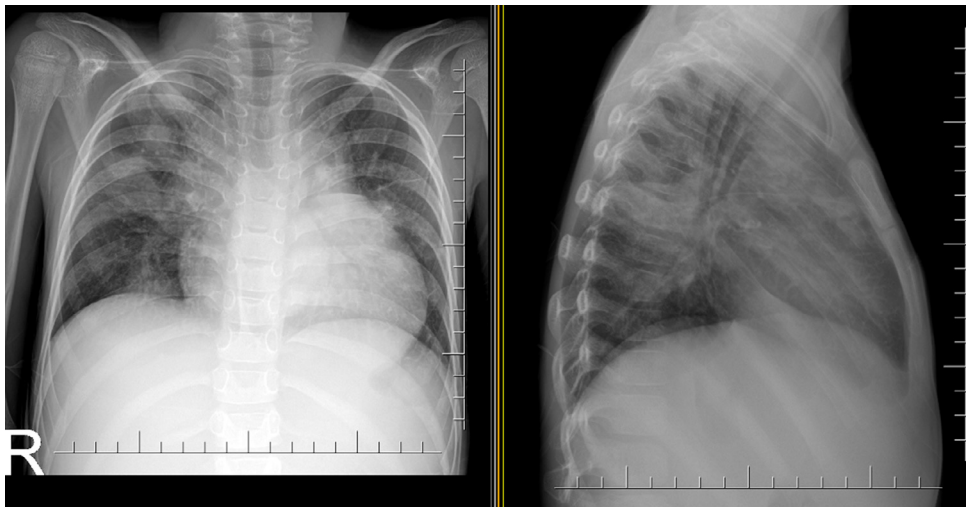

Fig. 3 – Chest X-ray upon admission into our institution. There is development of consolidation in the right upper lobe due to progression of pneumonia.

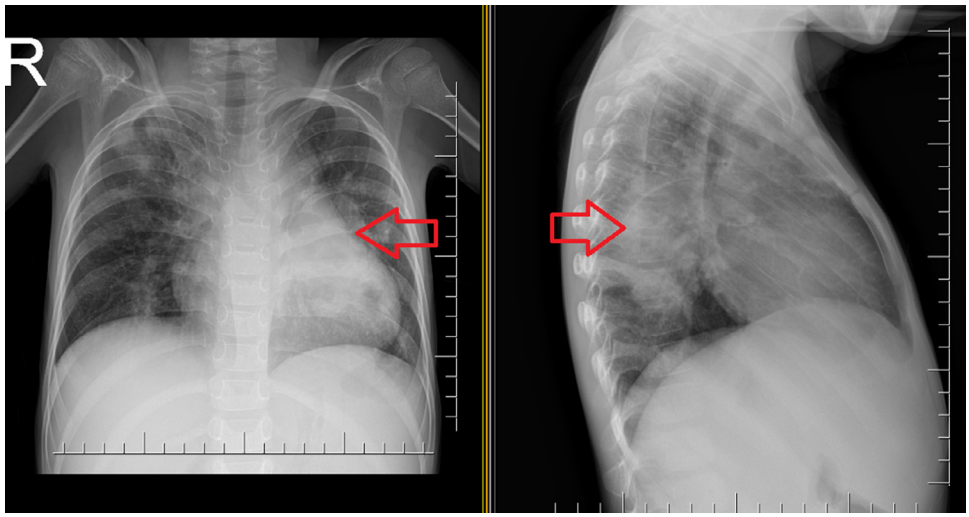

Fig. 4 – Chest X-ray around the time of hemoptysis, shows development of a well-defined left hilar mass.

other episode of hemoptysis. Chest X-ray (Fig. 5) done at this time revealed significant enlargement of the left hilar mass in 8 days. Subsequent contrast-enhanced chest CT scan and follow-up CT angiography (Figs. 6 and 7) showed a saccular aneurysm (measuring 3.8 × 5.0 × 5.7 cm [AP × T × CC]) arising from the distal left pulmonary artery adjacent to a cavitary lesion with a background of PTB (Figs. 6–10), highly suggestive of an RA. There was also an incidental note of a persistent left superior vena cava (PLSVC) (Figs. 8, 11 and 12). No thrombus, hematoma formation, or contrast extravasation seen implying stability.

A conservative approach was elected because of the stability and size of the aneurysm and because the patient was now asymptomatic with no repeat episodes of hemoptysis during the interim. Patient was discharged stable and the patient had a repeat chest X-ray after 2 months (Fig. 13), which showed slight regression in size of the left hilar mass. Again, there was no recurrence of hemoptysis during the interim. No other treatment was initiated and regular follow-up for monitoring was recommended.

Discussion

PTB affects about one-third of the global population with a prevalence of about 512 per 100,000 people in the Philippines [4]. It is caused by *Mycobacterium tuberculosis*, although other Mycobacteria species can cause the disease in rare cases. Symptoms are usually insidious and nonspecific: productive cough, minor hemoptysis, weight loss, fatigue, malaise, fevers, and night sweats [5].

For pediatric patients, diagnosis involves radiologic, laboratory, and clinical parameters. Pediatric PTB has 3 main categories: confirmed tuberculosis, probable tuberculosis, and

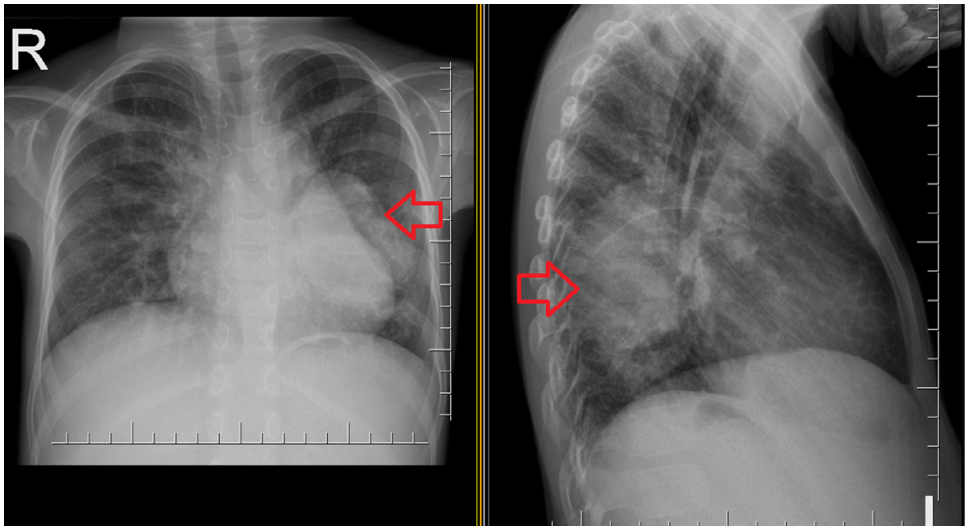

Fig. 5 – Follow-up chest X-ray done shows marked enlargement of the left hilar mass.

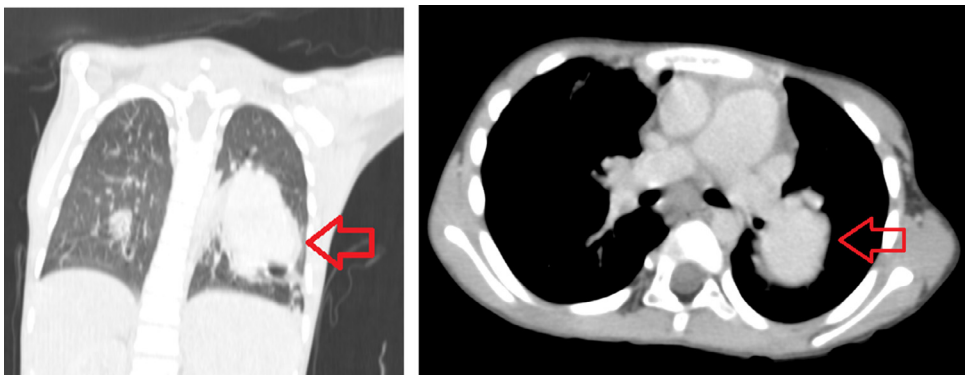

Fig. 6 – Contrast-enhanced chest CT scan done June 24, 2019, axial and coronal cuts. The left hilar mass is shown. Note the similar density (+HU 150-157) inside the mass compared to the cardiac chambers.

possible tuberculosis. Confirmed tuberculosis is the diagnosis when culture from intrathoracic disease (sputum, nasopharyngeal/gastric aspirate, pleural fluid) is positive. Xpert MTB/RIF can also be used to confirm PTB. Probable tuberculosis is when a child has a chest radiograph with findings consistent with PTB and at least one of the following: positive clinical response to antituberculosis therapy, documented exposure/close contact with a known tuberculosis patient, or positive tuberculin skin test or interferon-gamma release assay. Possible tuberculosis has 2 scenarios: chest radiograph not consistent with tuberculous disease but at least one criterion from probable PTB present, and/or radiograph consistent with PTB but without any of the previous criteria. Children without chest radiograph findings consistent with PTB, but exhibit symptoms corresponding with PTB, are classified as *unlikely PTB* [5].

Classically, PTB infection is classified as primary and post-primary, with the former more common in children. Primary tuberculosis usually involves a primary parenchymal lesion, called the Ghon focus, which then spreads to the ipsilateral central or regional lymph nodes, with the upper lobes draining to ipsilateral paratracheal nodes, while the rest of the lung drains to the perihilar nodes. When a Ghon focus is seen with an enlarged lymph node, it is now called the Ranke complex. Regional (perihilar or paratracheal) lymphadenopathy is the radiologic hallmark of childhood PTB, which requires anteroposterior and lateral views for optimal visualization [5].

Disease can progress from primary PTB, which is called progressive primary tuberculous disease. Presentation is commonly 1 year after primary infection in 90% of cases and occurs with a bimodal age distribution, most common in children younger than 5 years and in adolescents. Manifestations include consolidation, obstructive atelectasis or overinflation from compression by an adjacent enlarged node, fibrosis resulting to bronchiectasis, and formation of cavities. Enlarged lymph nodes can also erode and spread infectious materials into the airways. Pleural effusion can also occur from obstruction of lymphatic drainage or from a hypersensitivity reaction secondary to direct seeding. Pericarditis due to direct extension from lymphadenopathy can rarely occur. PTB can also cause pericardial effusion that can cause a globular enlargement of the heart shadow [5].

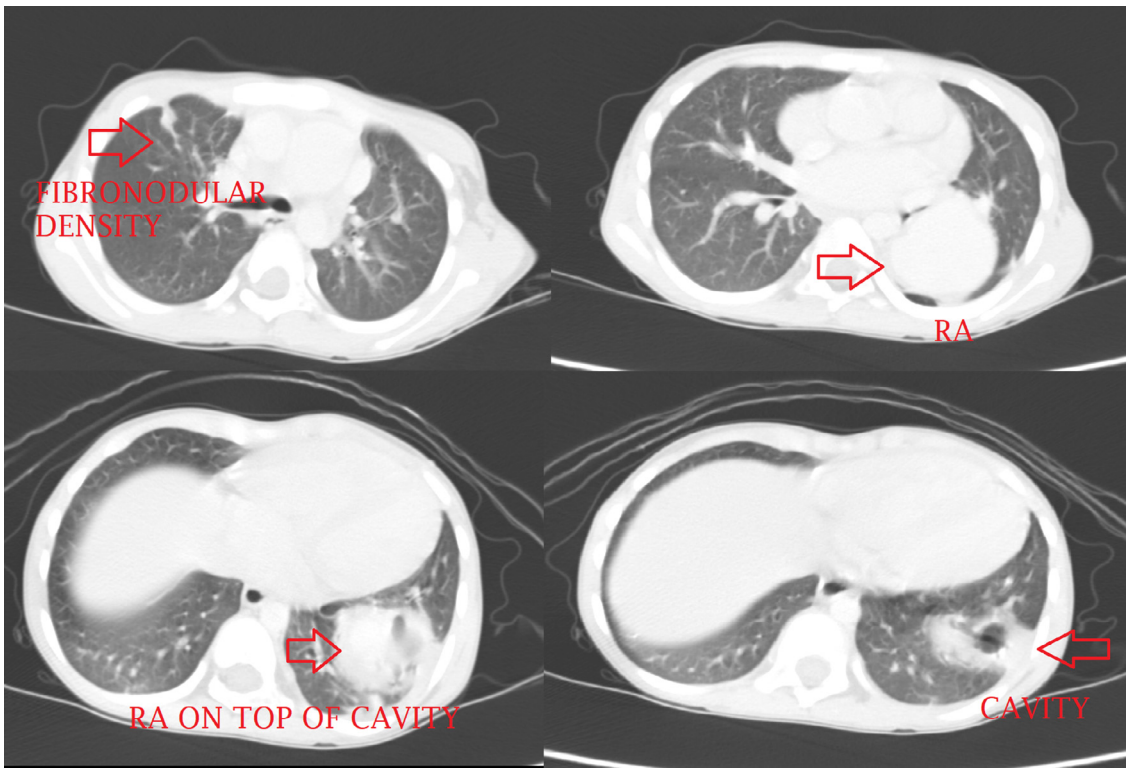

Fig. 7 – Contrast-enhanced chest CT, axial cuts. Example of a fibronodular density. Adjacency of the RA to a cavitary lesion is clearly shown.

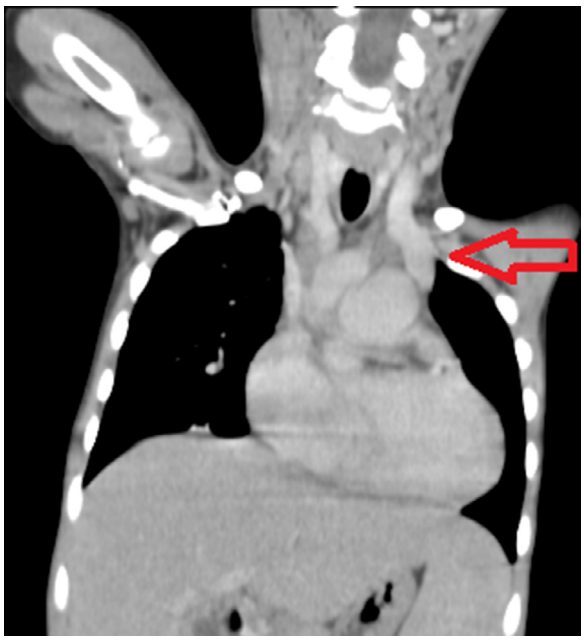

Fig. 8 – Contrast-enhanced chest CT scan, coronal cut. The PLSVC is depicted.

Late-stage PTB, called postprimary tuberculosis or adult-type, reactivation, or secondary tuberculosis, also occurs in pediatric patients, usually 8-24 months from exposure. Despite the name, it can occur in children as young as 8 years old. Post-primary tuberculosis preferentially occurs in the apical and posterior segments of the upper lobes and superior segments of the lower lobes, because of the preference of *M tuberculosis* for higher oxygen tensions. Pathology includes cavitation, bronchogenic spread with bronchopneumonic consolidation, exudative pleuritis, and cicatricial atelectasis. Unlike primary tuberculosis, lymphadenopathy is rare [5].

A common manifestation of PTB in adults is hemoptysis [6]. Unlike adults, however, hemoptysis due to PTB in children is relatively uncommon. Common causes in children include infection (pneumonia and tracheobronchitis), tracheostomy-related complications, congenital heart disease, pulmonary hemosiderosis, inflammatory bronchial mass, cystic fibrosis, and esophagitis [7].

RA, named after Fritz Valdemar Rasmussen, is a mycotic pseudoaneurysm caused by tuberculous infection. It occurs when a cavitary lesion forms adjacent to a pulmonary or bronchial artery, more commonly in the peripheral branches. It is estimated to be present in up to 5% of patients with cavitary tuberculosis, with much greater prevalence prior to modern PTB treatment. Patients with RA can present with massive hemoptysis, defined as expectoration of more than 300 ml of blood in a 24-hour period, which proves fatal up to 50% of the time [8]. Initial imaging of choice is MDCT angiography, which can rapidly localize the lesion and guide treatment strategy [2,9].

PLSVC is the most common thoracic vascular variant; however, it is still a very rare entity. It has a prevalence of 0.3%-0.5% in the general population. It is typically asymptomatic and only discovered as an incidental finding, usually when

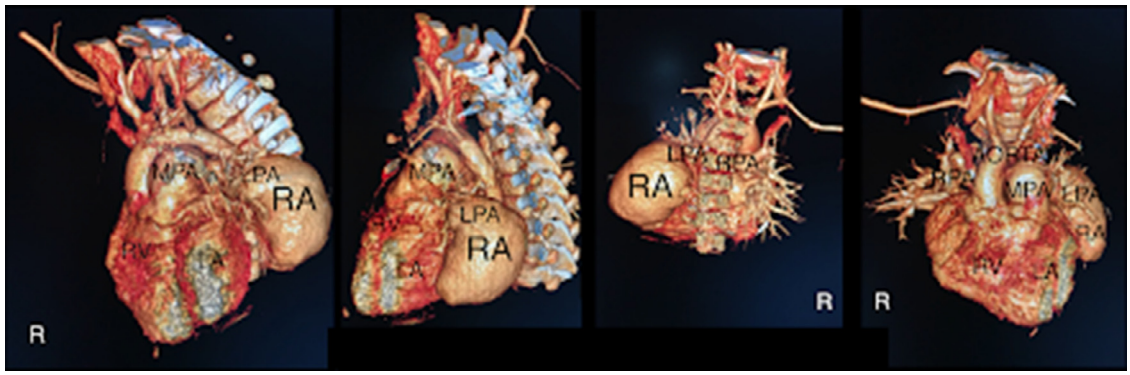

Fig. 9 – 3D reconstruction showing the RA.

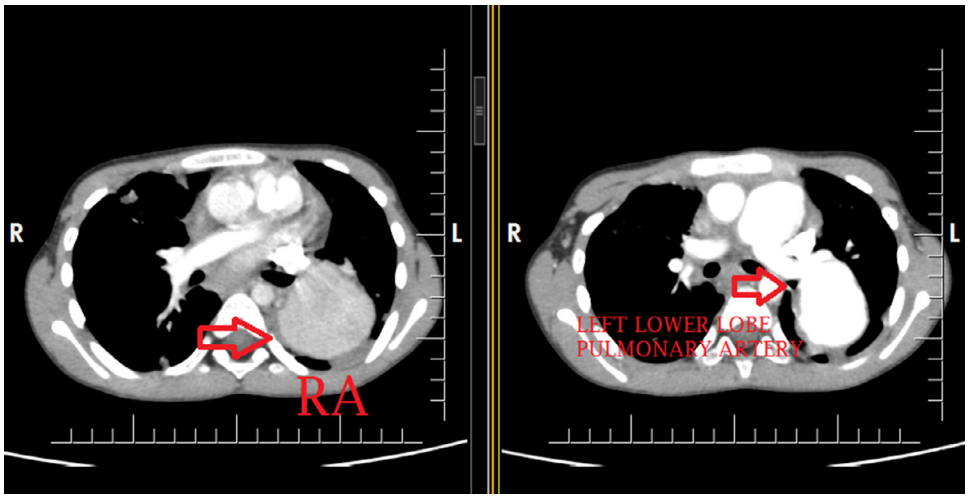

Fig. 10 – Chest CTA showing the RA to arise from the left lower lobe pulmonary artery.

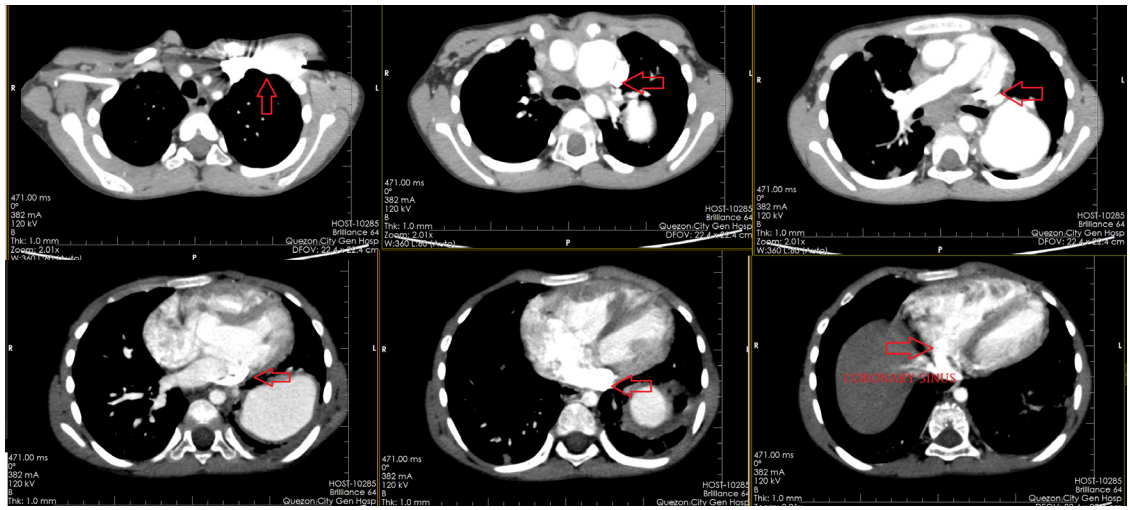

Fig. 11 – Chest CTA showing the course of the PLSVC from the level of the left common carotid artery up to the coronary sinus.

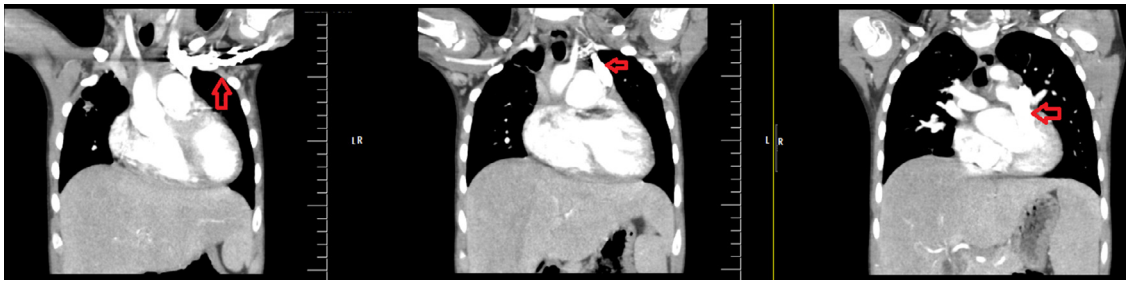

Fig. 12 – Chest CTA coronal cuts showing the descent of the PLVSC.

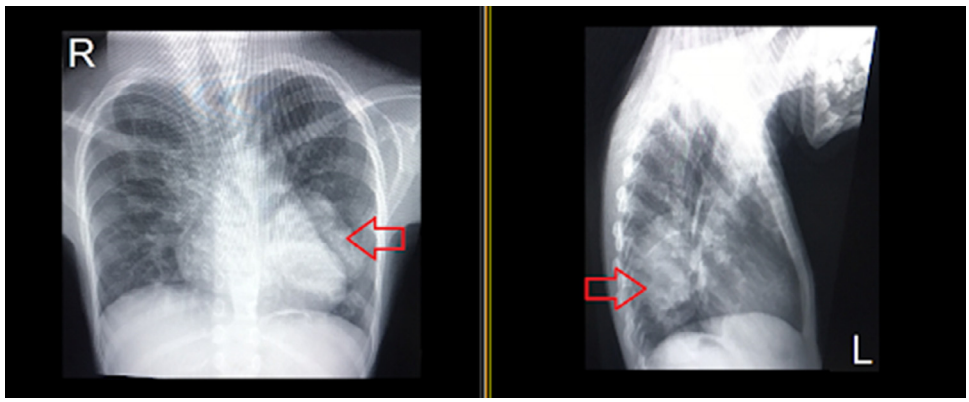

Fig. 13 – Follow-up after 3 months chest X-ray showing slight regression in size of the RA.

vascular access is needed for cardiac catheterization procedures. Forty percent of patients with PLSVC also have concomitant congenital heart disease such as atrial septal defect, ventricular septal defect (present in the patient), bicuspid aortic valve, coarctation of the aorta, coronary sinus ostial atresia, and cor triatriatum. The most common subtype is the presence of both a right and left superior vena cava, with or without a bridging innominate vein. In 80%-90% of cases, the PLSVC drains into the right atrium via the coronary sinus; this results in hemodynamic stability and an asymptomatic patient. In the rest of the cases, it may drain into the left atrium, which causes a left-to-right shunt and hemodynamic instability [10–12].

Treatment of a pulmonary artery aneurysm or pseudoaneurysm is either surgical/endovascular or medical. Endovascular techniques are preferred as first-line therapy due to significantly less morbidity and mortality. Surgical techniques include aneursymorrhapy, lobectomy, bilobectomy, aneursymectomy, and pneumonectomy [13,14].

Surgical repair is recommended in large aneurysms (>6 cm) if the patient is asymptomatic or regardless of size if the patient is symptomatic due to high risk of rupture or dissection. Conservative treatment, targeting the underlying condition, is recommended for asymptomatic patients with aneurysms less than 6 cm [15]. Several case reports have shown good outcome and regression in patients with mycotic aneurysms upon treatment of the underlying cause [1,16]. Gesuete et al. reported a case of an 8-year-old girl with an RA measuring 5.5 cm treated conservatively. The patient had severe pulmonary hypertension which precluded surgery. The patient's aneurysm decreased in size after 1 month of anti-Koch's treatment, with no further episodes of hemoptysis [1]. Gibb et al. also reported a case of a 14-year-old boy with Lemierre's syndrome, thrombophlebitis of the internal jugular vein due to pharyngitis or tonsillitis with or without peritonsillar or retropharyngeal abscess, who developed 5 large mycotic pulmonary artery pseudoaneurysms. Surgical or endovascular treatment was precluded due to the patient only having moderate hemoptysis, concurrent anticoagulant use, and the fact that multiple pseudoaneurysms were present. Antibiotic therapy in conjunction with discontinuation of anticoagulation medication and regular monitoring were done instead. The authors reported slow and spontaneous regression of the aneurysms after resolution of the causative bacterial infection [16].

Conclusion

RA is a rare cause of hemoptysis, especially in children. In the Philippines, where PTB is endemic, it should never be forgotten when a patient presents with massive hemoptysis. Diagnosis can be promptly obtained with MDCT angiography, which is also useful in treatment planning. Treatment depends on factors such as size, location, and symptoms. Conservative methods, aimed at treating the underlying cause, are used for asymptomatic patients with aneurysms less than 6

cm in size. Symptomatic patients and/or aneurysms greater than 6 cm are preferably treated surgically or endovascularly. In this patient, multiple factors (mostly PTB, but also Ventricular Septal Defect and pulmonic stenosis) contributed to the formation of the giant aneurysm. It is important to be vigilant when a patient with congenital heart disease becomes infected with PTB.

Patient consent

Complete written informed consent was obtained for the publication of this case report and accompanying images.

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