Pulmonary artery pseudoaneurysm arising secondary to cavitary pneumonia

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Pulmonary artery pseudoaneurysms have classically been associated with the cavitary lesions of reactivation tuberculosis and termed Rasmussen’s aneurysm. There have been relatively few case reports of pulmonary artery pseudoaneurysms arising secondary to cavitary pneumonia. We present a case of pulmonary artery pseudoaneurysm occurring secondary to cavitating aspiration pneumonia that was treated successfully with coil embolization.

Introduction

Aneurysms of the pulmonary arteries are rare. They may occur in association with congenital cardiovascular anomalies, infection, trauma, neoplasm, vasculitis, or pulmonary hypertension. Early recognition and treatment are important for reducing morbidity and preventing fatal complications such as massive hemoptysis.

Case report

A 60-year-old African-American male with a past medical history of hypertension, COPD, and rheumatoid arthritis presented to an outside hospital with a mild amount of hemoptysis. Bronchoscopy demonstrated no endobronchial lesion. Cytology and bronchial washings were negative. The patient did well after bronchoscopy; however, the night before planned discharge, the patient experienced an episode of massive hemoptysis, and emergent bronchoscopy with fiberoptic intubation was performed. Approximately 2-3 units of blood (900-1350 cc) was seen in the airway. The patient received 6 units of pRBC, 2 units of FFP, and subcutaneous vitamin K. The next morning, bleeding decreased; however, the patient required an additional 2 units of pRBCs before transfer to our facility. Acid-fast bacilli smears were negative, and PPD skin test was negative. Bronchial washing cytology, fungal stain, and cultures were negative as well.

Upon arrival at our facility, the patient underwent a CT angiogram of the chest that demonstrated airspace consolidation in the posterior segment of the right upper lobe with associated cavitary lesion. There was adjacent contrast blush consistent with active extravasation, which appeared to communicate with a pulmonary arterial branch (Figs. 1 and 2). Interventional radiology was consulted, and pulmonary arteriogram with possible intervention was planned.

The right pulmonary artery was catheterized with a pigtail catheter, and angiography demonstrated a small pseudoaneurysm in the right upper lobe. The pseudoaneurysm appeared late in the parenchymal phase and lingered past the venous phase (Fig. 3). Subsequently, the posterior segmental branch of the right upper lobe of the pulmonary artery branch was catheterized, and an angiogram demonstrated the pseudoaneurysm arising from a small branch (Fig. 4). This branch was subselected with a microcatheter, and multiple coils were deployed into the pseudoaneurysm (Fig. 5). Subsequent angiograms showed no residual filling of the pseudoaneurysm (Fig. 6).

The patient did not experience any additional episodes of hemoptysis, and his hemoglobin and hematocrit levels remained stable throughout the remainder of the hospitalization. He remained afebrile without leukocytosis. He was discharged and treated for his presumed aspiration pneumonia with Clindamycin, although a specific causative bacterium was not identified.

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Massive hemoptysis has been defined as the expectoration of a large amount of blood ranging from 100 mL to greater than 1,000 mL over 24 hours. However, even a smaller amount of hemorrhage may compromise the patient’s ability to maintain a patent airway, and a more functional definition of “massive” is an amount sufficient to cause a life-threatening condition. A wide variety of pathologic conditions can cause massive hemoptysis by invasion or injury to vascular structures within the chest. Massive hemoptysis usually arises from a bronchial arterial source (90% of cases) rather than a pulmonary circulation source (5% of cases) (1).

An aneurysm is defined as focal dilation of a blood vessel that involves all three layers of the vessel wall. A pseudoaneurysm does not involve all three layers and is therefore at greater risk of rupture. The term Rasmussen’s aneurysm is classically defined as a pseudoaneurysm resulting from erosion of a pulmonary arterial branch by a tuberculous cavity (2), and it usually involves the upper lobes in the setting of reactivation tuberculosis. Rasmussen aneurysms have been reported in up to 5% of patients with cavitary tuberculosis (TB) in autopsy studies (3). However, pulmonary arterial pseudoaneurysms have also been reported in association with both bacterial (4) and viral (5) pneumonias.

Infection and inflammation have been associated with pseudoaneurysms in the systemic circulation, but are seen less commonly in the pulmonary circulation. Pyogenic infection is a potential cause of pulmonary artery pseudoaneurysm formation secondary to the active inflammatory process that erodes the feeding vessel into the bronchi (4, 5). Causative microorganisms include S. Aureus (22%), Salmonella species (17%), Streptococcus species (11%), and Enterococcus species (11%) (6). Pseudoaneurysms can often be seen in IV drug users with associated infective endo-
carditis and septic emboli (2). In the setting of septic emboli, these are more commonly multiple or bilateral.

Additionally, there are many noninfectious causes of pulmonary artery aneurysm and pseudoaneurysm formation. Congenital pulmonary artery aneurysms can be seen in cases with increased hemodynamic shear stresses caused by left-to-right shunt such as patent ductus arteriosus, ventricular septal defect, or atrial septal defect. These aneurysms can grow to be quite large, and the risk of rupture or dissection is highest in patients with severe pulmonary hypertension (2). Iatrogenic cases are usually caused by malpositioned Swan-Ganz catheters, in which the catheter tip has been positioned too far into a pulmonary arterial branch. Penetrating trauma, neoplasm, connective-tissue disorders, and vasculitis (especially Bechet’s syndrome and Hughes-Stovin syndrome) are also known causes.

In cases of massive hemoptysis, imaging studies should include thoracic multidetector CT angiography and digital subtraction angiography to differentiate pulmonary from bronchial origin of bleeding. The mortality rate for emergency surgery in unstable hemodynamic patients is high, so
endovascular intervention is recommended as a first-line therapy in these patients (3).

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