Case Report

Pulmonary Artery Pseudoaneurysm: Mimicking a Lung Mass

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Abstract

Pulmonary artery pseudoaneurysms are an uncommon pulmonary pathology; but associated with high mortality. Untreated lesions can enlarge, rupture or lead to exsanguination and death. Pseudoaneurysms are either idiopathic or may be caused by tuberculosis, fungal infection, penetrating trauma, blunt trauma, bacterial endocarditis, catheterisation of either pulmonary artery or right heart. [Indian J Chest Dis Allied Sci 2020;62:65-68]

Key words: Inflammatory myofibroblastic tumours, Benign, Pedunculated, Coblation.

Introduction

Pulmonary artery pseudoaneurysms is a rare pulmonary entity. Pseudoaneurysm do not have a covering of all three layers of the arterial wall and are effectively contained arterial leaks considered to be at high risk of rupture. The common clinical manifestations of pulmonary artery pseudoaneurysms include cough and haemoptysis. Pulmonary artery pseudoaneurysms can also present as life-threatening haemorrhage or incidental lesions that enlarge for days, months or years. Vascular complications in the chest, involving the pulmonary as well as bronchial vasculature are known to occur due to tubercular infection.¹ We present a case of a female who was diagnosed as a case of pulmonary artery pseudoaneurysm with tuberculosis as an aetiology.

Case Report

28-year-old female presented with cough, А haemoptysis, fever and dyspnoea since two months. She had no history of loss of appetite, loss of weight, and chest pain. Initial clinical assessment revealed normal vital signs without any obvious respiratory distress with an oxygen saturation of 98% by pulse oximetry on room air. Laboratory investigations revealed: haemoglobin 11.6g/gL, total leucocyte count 4060/mm³, platelet count 2.73 lakhs/mm³ and erythrocyte sedimentation rate of 50mm. Renal function tests, urine analysis, serum electrolytes and liver function tests were within normal limits. Human immunodeficiency virus (HIV) was non-reactive. Sputum for Ziehl-Neelsen staining and cartridge-based nucleic acid amplification test (CBNAAT) was negative. No fungus grown on culture. Two-dimensional (2D) transthoracic echocardiography (2D-TTE) was normal. Chest radiograph (posterior-anterior view) showed right lower lobe homogeneous opacity without air-bronchogram (Figure 1). Contrast enhanced computed tomography (CECT) of the chest revealed a well-defined, enhancing lobulated mass with irregular margins in the right lower lobe with positive feeding vessel sign (Figure 2). Feeding vessel sign indicates either the lesion has a haematogenous origin or the disease process occurs near the small pulmonary vessels.

Dual phase CT pulmonary angiography revealed a lesion in the superior segment of the right lower lobe (54mm×50mm) with an enhancement and a filling defect in the right lower lobar branch of the pulmonary artery reflecting peripheral thrombus (Figure 3). These



Figure 1. Frontal chest radiograph showing homogeneous opcity in the right lower lobe without airbronchogram.

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Figure 2. Contrast enhanced computed tomography of the chest showing a well-defined enhancing lobulated mass with irregular margin.



Figure 3. Dual phase computed tomography pulmonary angiography showing a lesion in the superior segment of the right lower lobe (54mmX50mm) showing enhancement on contrast and peripheral thrombus during angiography phase.

findings established the diagnosis of pulmonary artery pseudoaneurysm. The patient underwent digital subtraction angiography and endovascular coil embolisation of the bronchial artery and pulmonary artery with resolution of haemoptysis after the procedure (Figure 4). The patient also underwent bronchoscopy to establish the aetiology. The bronchoalveolar lavage showed growth of *Mycobacterium tuberculosis* on liquid culture at the end of two weeks. She was put on antitubercular therapy. After six months of follow-up, she has no haemoptysis.

Discussion

Although pulmonary artery pseudoaneurysms are rare, the mortality is as high as 50% in the diagnosed cases. Pulmonary artery pseudoaneurysms may be congenital or acquired. The most common cause of acquired pulmonary artery pseudoaneurysms are infections, like *Mycobacterium tuberculosis*, pyogenic bacteria, lung abscesses, septicemboli, bronchiectasis and fungi.^{1,2}Lung neoplasms (including primary squamous cell carcinoma, primary sarcoma, and metastatic sarcoma) and other rare causes of pulmonary artery pseudoaneurysms including chest wall trauma and iatrogenic trauma from pulmonary artery catheters are also reported.³⁻⁵

The development of pulmonary artery pseudoaneurysms in patients with pulmonary tuberculosis has been well described.⁷ In an autopsy series of three patients, Auerbach concluded that the aneurysm develops as a result of the progressive destruction and replacement of the elastic fibers of the pulmonary artery from without inward of outer wall of a tuberculous cavity by the granulation tissue. As the cavity undergoes progressive healing, the granulation tissue within its wall continuously increases and erodesthe elastic fibers of the artery in a circumscribed area, resulting in aneurysm formation.⁷

Patients with pulmonary artery pseudoaneurysms commonly present with haemoptysis and hypoxaemia and occasionally experience chest pain. Chest radiograph may show non-specific focal lung consolidation, a solitary pulmonary nodule, or early consolidation evolving to a nodule or mass near the central or the peripheral pulmonary vasculature.⁸ Computed tomography is more diagnostic when there is central enhancement within a haematoma or lung consolidation. Other findings include an enhancing mass next to a pulmonary artery, thrombus within a dilated pulmonary artery, and an enhancing nodule with a low attenuation halo. The definitive diagnosis



Figure 4. Post procedure dual phase computed tomography pulmonary angiography showing cavitatory lesion in the superior segment of the right lower lobe with no enhancement and stasis of contrast.

is made with computed tomography pulmonary angiography (CTPA). This modality not only establishes the diagnosis, but also helps in the management plan with endovascular treatment modalities. On CTPA, pulmonary artery pseudoaneurysms appear as focal outpouchings of contrast adjacent to a pulmonary artery branch following the same contrast density as the pulmonary artery in all phases of the study.⁹ Pulmonary angiography demonstrates delayed emptying of contrast material from the sac.9 As the pulmonary artery lacks an adventitial wall, pulmonary artery pseudoaneurysms are more likely to rupture than true arterial aneurysms. Therefore, haemoptysis due to ruptured pulmonary artery pseudoaneurysms is often fatal and must be promptly recognised and treated.

Urgent endovascular treatment is considered the treatment of choice in managing haemoptysis resulting from pulmonary artery pseudoaneurysms. Endovascular treatment by direct coil embolisation, stent placement, or embolisation of the feeding vessel has been reported to be effective in occluding pseudoaneurysms.⁹⁻¹³ In a small pseudoaneurysm, occluding the feeding vessel might be adequate; however, coil embolisation is preferred for larger aneurysms. Coil embolisation of pulmonary pseudoaneurysms offer the potential for selective embolisation of the sac; while preserving flow through the affected pulmonary artery segment. However, there is an associated risk of distal coil migration in case of aneurysms with a wide neck. In wide-necked pulmonary artery pseudoaneurysms, endovascular stenting has been described as successful treatment modality.¹³ In patietns with coagualiton abnormanlities, balloon embolisation can be considered.¹⁴

Amplatzer vascular plug (St Jude Medical, Inc, St Paul, MN, USA) has advantages of single device occluding the feeding vessel, increased precision, and control while deployment and firm anchorage to vessel wall by outward radial force due to elasticity of nitinol. These are difficult to be used in case of peripheral pulmonary arteries in view of requirement of a large area of access and deploying devices.¹⁵ Complications of endovascular embolisation are less frequent and contrast-induced nephropathy, include arterial dissection, and pulmonary infarct. Operative repair for pulmonary artery pseudoaneurysms involves open thoracotomy and aneurysm resection, with lobectomy for the involved lobes. Surgical treatment, is associated with increased mortality and morbidity compared with endovascular treatment. Surgical approaches should be reserved for the patients with pleural haemorrhage, uncontrolled haemoptysis, or infections, such as mucormycosis that may not respond to medical therapy alone. Surgery is also preferred in patients who have a high chance of endovascular graft or coil infection, such as patients with sepsis.

In conclusion, pulmonary artery pseudoaneurysms is a rare entity and may present as mass like lesion. Timely recognition of pulmonary artery pseudoaneurysms improves the outcome.

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