CASE REPORT

Pseudoaneurysm of internal mammary artery caused by pulmonary actinomycosis

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ABSTRACT. Although internal mammary artery pseudoaneurysms are a rare vascular abnormality, they are sometimes seen after sternotomy, diagnostic and therapeutic vascular access or penetrating chest trauma. To our knowledge, internal mammary artery pseudoaneurysm caused by pulmonary actinomycosis has not been reported previously. We report a case of pseudoaneurysm of the left internal mammary artery caused by pulmonary actinomycosis. A 50-year-old woman initially presented with clinical and radiological features of pneumonia, for which she was treated empirically with antibiotics. Later, she developed haemoptysis along with pain and swelling in the left upper chest wall. Multidetector CT (MDCT) with CT angiography showed a cavitating mass in the left upper lobe of the lung that infiltrated into the chest wall and a pseudoaneurysm of the left internal mammary artery. Imaging suggested that the lung mass resulted from pulmonary actinomycosis, which was confirmed by the histopathology of a fine-needle aspiration specimen.

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Internal mammary artery (IMA) pseudoaneurysm is a rare vascular abnormality that commonly results from sternotomy, insertion of a catheter or central venous line into the subclavian vessels, or penetrating chest trauma such as stab injury [1]. Very rarely, the formation of a IMA pseudoaneurysm has been described in the context of chest wall infection by staphylococcus, tuberculosis or fungi [2, 3]. Radiological manifestations of thoracic actinomycosis are varied and diverse. They include air-space consolidation, lung mass, mediastinal lymphadenopathy, empyema, osteomyelitis, chest wall mass with fistula formation, and very rarely, the formation of a pulmonary artery pseudoaneurysm [4]. To our knowledge, however, IMA pseudoaneurysm associated with thoracic actinomycosis has not been reported previously.

We report a case of thoracic actinomycosis that started as a non-specific pneumonic consolidation in a 50-year-old female. The disease progressed within a few weeks, invading the chest wall and resulting in left IMA pseudoaneurysm.

Case history

A 50-year-old woman presented to the emergency department of our hospital with a two-week history of fever, productive cough and chest pain. Her medical history was marked with type 2 diabetes mellitus,

hypertensive heart disease and glaucoma of the left eye. On admission, she was febrile with a temperature of 38°C. Physical examination of the chest showed dullness on percussion and inspiratory crackles on auscultation in the left infraclavicular area. Routine laboratory investigations revealed an elevated total leukocyte count of 17000/cumm with 85% neutrophils; blood gas analysis revealed PaO₂=65 mmHg, PaCO₂=30 mmHg and pH=7.36; and random blood sugar was 160 mg dL⁻¹. The chest radiograph on admission showed opacity over the left upper lung zone; no definite evidence of cavitation was seen even on the retrospect viewing. The patient was treated for left upper lobe pneumonia with an empirical antibiotic combination of iv third generation cephalosporin and macrolide. Her symptoms did not improve, and on the sixth day of admission she developed moderate haemoptysis and a swelling was palpable on the upper anterior chest. Multidetector CT (MDCT) was performed to search for the source of bleeding. An initial non-contrast CT study was followed by contrast-enhanced study on a 64-MDCT scanner (Somatom Sensation 64, Siemens Medical Solutions). Non-ionic contrast medium (Iohexol; 80 ml) was administered intravenously at a rate of 3.5 ml s⁻¹ via an automatic injector through an 18-gauge catheter. The imaging parameters were: 120 kV and 160 mA, pitch of 1.2 and rotation time of 0.5 s. A reconstruction thickness of 3 mm was used for the visualisation of lung parenchyma and the chest wall; for the vascular delineation, a reconstruction thickness of 0.75 mm every 0.5 mm was used. The CT study of the chest showed a soft-tissue attenuation lesion in the anterior segment of

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the left upper lobe of the lung, with areas of lower attenuation and cavitation inside it (Figure 1). An anterior chest wall mass communicated with the parenchymal lesion across the left second intercostal space with irregularity of the second rib (Figures 2 and 3). An intensely enhancing irregularly shaped vascular structure, of attenuation similar to that of the aorta, was engulfed by the cavitating mass lesion (Figures 2 and 3). Sagittal plane thick multiplanar reformation showed the vascular structure to be continuous with the left IMA.

These findings were compatible with an infective process in the left upper lobe of the lung with chest wall invasion, possibly thoracic actinomycosis with haemoptysis caused by the resulting pseudoaneurysm of the left IMA. Fine-needle aspiration from the chest wall mass was performed under CT guidance for histopathological diagnosis. CT additionally showed bilateral pleural effusion and pericardial effusion, which were considered to be signs of cardiac failure; there was no evidence of tamponade on echocardiography. Cardiac ultrasonography showed anechoic fluid around the heart, but did not rule out the possibility of pericardial involvement by the lung lesion. Surgical evacuation of the infection and pseudoaneurysm repair was planned but the patient's health continued to deteriorate and her blood pressure dropped significantly. She could not be resuscitated with iv fluids and pressor agents and died on the eighth day of admission. Her downhill course was possibly triggered by already compromised cardiac function, which was aggravated by uncontrolled infection and septicaemia. No post-mortem examination was performed. Pathological examination of the aspirated specimen revealed sulphur granules that contained Gram-positive filamentous branching organisms, consistent with actinomycosis.

Figure 1. Transverse plane mediastinal window CT image showing a cavitating lesion in the left upper lobe. The lesion extends anteriorly across the intercostal space into the chest wall and there is erosion of the second rib. An intensely enhancing vascular structure that is engulfed by the lung lesion is seen anteriorly.

Figure 2. Transverse plane lung window CT image showing a soft-tissue attenuation mass in the anterior segment of the left upper lobe, with areas of breakdown within it.

Discussion

Thoracic actinomycosis is a chronic suppurative pulmonary infection that is caused by *Actinomyces* species, most frequently *Actinomyces israeli*, a gram-positive anaerobic saprophytic organism of the oral cavity, considered to be a branching filamentous bacterium. Lung infection typically follows aspiration of endogenous organisms of the oropharynx into the lungs [4, 5]. In an early stage of infection, the disease manifests as a poorly defined peripheral pulmonary nodule or as an air-space consolidation that contains central areas of low attenuation

Figure 3. Sagittal plane multiplanar reformat image shows the irregularly shaped vascular structure to be continuous with the left IMA. The lung lesion extends across the second intercostal space into the chest wall.

with frequent cavitation. The clinical manifestations of pulmonary actinomycosis are a productive cough, low-grade fever and blood-tinged sputum. At an early stage, diagnosis can be made on the basis of histopathological findings of *Actinomyces* colonies, sulphur granules or filamentous structures within the tissue specimen that are positive for Grocott–Gomori methenamine-silver staining [4, 5]. With further progression, the classical features of chest wall invasion with multiple cutaneous discharging sinuses may form, making the diagnosis straightforward. Although aggressive infiltration and frequent haemoptysis have been described, vascular complications are rarely described in thoracic actinomycosis. The involvement of the pulmonary artery in pulmonary actinomycosis, resulting in mycotic aneurysm, has been reported [4]. IMA pseudoaneurysm caused by thoracic actinomycosis has not, however, been reported previously.

Pseudoaneurysms are a disruption in arterial wall continuity resulting from inflammation, trauma or iatrogenic causes, such as percutaneous biopsy, drainage or surgical procedures. Under the influence of sustained arterial pressure, blood dissects into the tissues around the damaged artery and forms a perfused sac that communicates with the arterial lumen [6]. Any vessel that is subjected to trauma or infection is susceptible to the development of a pseudoaneurysm and the IMA is no exception.

The formation of an IMA pseudoaneurysm is a well-known although rare complication of sternotomy, insertion of central vein catheter or pacemaker leads, and percutaneous biopsy [1, 7]. Adjacent infection is a lesser known cause of IMA pseudoaneurysm. Mycotic IMA pseudoaneurysms caused by staphylococcal, tubercular and fungal chest wall abscesses have, however, been reported in the medical literature [2, 3]. Infection adjacent to an artery can destroy all the layers of the vessel wall and induce the formation of a pseudoaneurysm. Such a post-infection pseudoaneurysm is sometimes called a mycotic aneurysm, which in essence is an aneurysm resulting from a haematogenous infection lodging in an arterial wall in bacterial endocarditis [6]. Actinomycosis is a peculiar lung infection that invades the pleura and adjacent chest wall. Adjacent systemic arteries may enlarge in response to the chronic infection. Pulmonary artery pseudoaneurysm is usually a consequence of iatrogenic and penetrating lung trauma, pulmonary neoplasms and their treatment, and bacterial and tubercular lung infections [4, 7, 8]. Such pseudoaneurysms have rarely been described as complications of pulmonary actinomycosis [4, 7, 8]. In our case, a pseudoaneurysm formed in the IMA as a complication of chest wall invasion by actinomycotic disease in the anterior segment of the upper lobe of the left lung.

IMA pseudoaneurysm may be noticed as a pulsatile swelling or present as mediastinal haematoma or haemothorax with or without haemodynamic instability [1, 9]. In our case, haemoptysis was attributable to the internal mammary pseudoaneurysm engulfed by the left upper lobe parenchymal actinomycotic lesion that extended into the chest wall.

Exact description of the size and anatomical localisation of the IMA pseudoaneurysm is crucial for planning of therapy. Of the various non-invasive imaging modalities, CT angiography with MDCT is favoured for the

diagnosis of IMA pseudoaneurysm [10]. Pseudoaneurysms appear on contrast-enhanced CT as an enhancing (contrast-filled) sac with an adjacent vessel; however, the entire aneurysm might not fill with contrast material because of variable thrombosis. The wall of a pseudoaneurysm is usually smooth and well-delineated, the exception being the mycotic aneurysm where the wall may be thickened, irregular or ill-defined. MDCT demonstrates the anatomical location of a pseudoaneurysm and directly visualises its feeder vessel and connection to the parent vessel. Catheter angiography is reserved for guiding the endovascular intervention [6, 10].

Traditionally, pseudoaneurysms have been treated with surgical repair, which is associated with complications including anaesthesia-related risks, bleeding, wound infection, prolonged recovery and death. As a result of technological advances, there is an ongoing paradigm shift towards minimally invasive management of pseudoaneurysms in most of locations within the body. Transcatheter embolisation is performed using steel coils that are placed within the pseudoaneurysm [6]. Alternatively, the feeding vessel is embolised to occlusion [6]. For the internal mammary pseudoaneurysm resulting from sternotomy, surgical repair in the form of ligation of the parent IMA and evacuation of the haematoma has been described [11]. Adjacent infective process may need to be addressed by lobectomy or chest-wall repair [3]. Endovascular embolotherapy offers an effective, efficient and safe alternative to conventional surgical management and is currently the favoured form of treatment for the IMA pseudoaneurysm [7, 12].

In conclusion, chest-wall invading pulmonary parenchymal actinomycosis can result in IMA pseudoaneurysm. MDCT is the ideal imaging technique in such a situation, demonstrating the changes in the parenchymal tissue and chest wall and delineating the pseudoaneurysm and its continuity with the parent vessel.

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