

Pulmonary atelectasis from compression of the left main bronchus by an aortic aneurysm

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ABSTRACT

Pulmonary atelectasis may be caused by endobronchial lesions or by extrinsic compression of the bronchus. However, lung collapse due to compression from a thoracic aneurysm is uncommon. We report a 76-year-old hypertensive female patient who has pulmonary atelectasis due to an extrinsic compression from a descending thoracic aortic aneurysm, and discuss possible treatment options.

Keywords: aneurysm, pulmonary atelectasis, thoracic aorta aneurysm

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INTRODUCTION

Thoracic aortic aneurysms may involve the ascending aorta, aortic arch or the descending aorta. Most patients are asymptomatic, and the aneurysms are detected on imaging studies for another indication. When symptoms are present, they are usually due to aortic dissection, rupture or the compression of adjacent structures. Although the thoracic aorta is closely related to the bronchus, pulmonary atelectasis due to an aortic aneurysm is uncommon. We report a 76-year-old hypertensive patient who has a left lung collapse due to a descending thoracic aortic aneurysm.

CASE REPORT

A 76-year-old woman, who has a longstanding history of hypertension, presented to our centre for prolonged cough of three months' duration, with occasional haemoptysis. The patient denied having any chest pain, fever, night sweat or change in appetite. She did, however, claim to have lost some weight. She was a non-smoker, and there was no significant history of occupational exposure to mineral dust or contact with patients with tuberculosis. Clinically, she was afebrile, her blood pressure was 141/72 mmHg and her pulse was 91/min. An examination of the respiratory system revealed absent breath sounds over the entire left hemithorax, with a dull percussion note from mid-zone downwards. There were



Fig. 1 Posteroanterior chest radiograph taken on admission shows a complete atelectasis of the left lung with associated pleural effusion.

no adventitious sounds heard. Examination of the other systems, i.e. cardiovascular, abdominal and neurological systems, were normal. All the peripheral pulses were palpable. The chest radiograph showed a totally opaque left hemithorax with the trachea deviated to the left (Fig. 1). The blood biochemistry showed normal urea and electrolytes with a creatinine of 112 $\mu\text{mol/L}$, and a normal liver function test. The haemoglobin, white cell and platelet counts were 11.7 g/dL, $16.7 \times 10^9/\text{L}$ and $477 \times 10^9/\text{L}$, respectively. The sputum culture grew *Klebsiella pneumoniae*, but the blood and pleural fluid cultures were sterile. Syphilis serology was negative.

She was treated for left bronchopneumonia with intravenous cefuroxime, and an urgent computer tomography (CT) of the thorax was performed. The CT showed an aneurysm of the descending thoracic aorta, just distal to the arch measuring 5.6 cm \times 9.4 cm \times 8.2 cm with a huge mural thrombus (Fig. 2). The aneurysm was seen distal to the left brachiocephalic trunk, of

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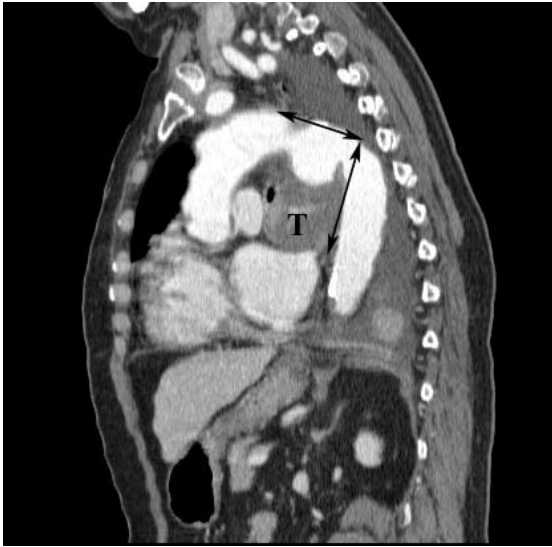


Fig. 2 Sagittal CT image shows the extent of the thoracic aneurysm (arrows) with mural thrombus (T).

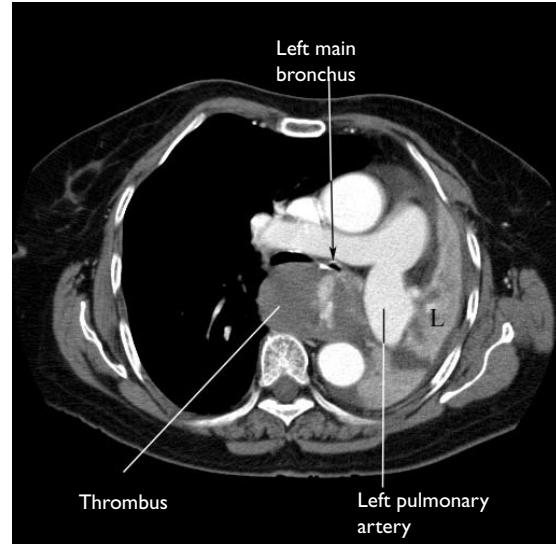


Fig. 3 Axial CT image shows a large proximal descending thoracic aneurysm compressing the left main bronchus (arrow) with left lung collapse (L).

which the left common carotid and left subclavian artery were seen arising from it. The aneurysm was compressing onto the left main bronchus, resulting in a collapse consolidation of the entire left lung (Fig. 3). A small left pleural effusion was also present. The right lung appeared hyperinflated, with no focal lesions. A cardiothoracic consultation was made, but the patient was not keen to undergo bronchoscopy or surgery.

DISCUSSION

The common causes of bronchial obstruction include endobronchial tumours, foreign bodies, mucous plugs, or external compression from tumours or infections. A left lung collapse due to an aneurysm of the descending aorta compressing onto the left main bronchus is indeed uncommon, although it has been reported before.^(1,2) In patients who have a history of blunt chest trauma, post-traumatic aortic aneurysms can occur and cause a similar picture.^(3,4) With the symptoms of weight loss, coupled with chronic cough and haemoptysis in the elderly age group, a malignant neoplasm causing atelectasis with postobstructive pneumonia needs to be considered. This important differential diagnosis was excluded due to the presence of an aneurysm and mural thrombus completely occluding the left main bronchus clearly shown on the CT thorax. Hence, the weight loss could be attributed to recurrent episodes of pneumonia.

Aortic aneurysms are divided anatomically into ascending, aortic arch and descending thoracic aneurysms. 60% of the thoracic aneurysms involve the ascending aorta with or without affecting the aortic root, while the remaining 40% involve the descending aorta.⁽⁵⁾

Cystic medial degeneration is the most common cause of ascending aortic aneurysm, and may be related to Marfan syndrome or bicuspid aortic valve. In descending aortic aneurysms, atherosclerosis appears to be the more common aetiology. Less common causes include syphilis and aortic arteritis.

The incidence of thoracic aortic aneurysm is estimated to be 5.9–10.4 per 100,000 person-years.^(6,7) Aortic diameter and the female gender are useful predictors for rupture, dissection and death.^(7,8) Treatment options include vigorous blood pressure control, surgery and minimally-invasive endovascular stent-grafting. Surgery is indicated if the diameter of the ascending aortic aneurysm exceeds 5.5 cm, or 6 cm in descending aortic aneurysms.⁽⁵⁾ Thoracic aortic aneurysm repair involves cardiopulmonary bypass with resection of the aneurysm, followed by placement of a prosthetic tube graft. In surgeries involving the descending aorta and thoracoabdominal aorta, complications that may occur include postoperative paraplegia secondary to spinal cord ischaemia, stroke, renal and respiratory failure. In untreated patients, aortic aneurysms grow at a rate of 0.1 cm/year, and patients with aneurysms that exceed 6 cm in size have a rate of rupture or dissection of 6.9% per year, and death rate of 11.8% per year.⁽⁸⁾ Unfortunately, this patient was not keen on surgery. Hence, antihypertensives e.g. beta-blockers, should be given to achieve a target systolic blood pressure of 105–120 mmHg.⁽⁵⁾ Long-term beta-blockade with propranolol has been proven to reduce the rate of aortic root dilatation and mortality in patients with Marfan syndrome.⁽⁹⁾ In the non-Marfan syndrome patients, there is limited mortality data on the role of beta-

blockers, although it is a common practice to use beta-blockers to lower the blood pressure to the desired level.

Newer approaches such as endovascular stent grafting can be offered if available, as it appears to have lower perioperative morbidity and mortality rates compared to open surgery.⁽¹⁰⁾ To address the lung collapse, endobronchial stents may be used to relieve the bronchial obstruction. Broadly, tracheobronchial stents are divided into silicone, metallic or hybrid stents. Deployment of these airway stents is usually done by a trained bronchoscopist using rigid bronchoscopy, under general anaesthesia. In a patient whose atelectasis is due to an aortic aneurysm, the possibility of stent failure due to compression from the aneurysm and delayed aortobronchial fistula need to be considered.⁽¹¹⁾ At present, there have not been any large studies examining the role of bronchial stenting in the treatment of pulmonary atelectasis from aortic aneurysm compression.

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