

Commentary

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Generalised pulmonary artery disease is relatively common. It occurs particularly in association with prolonged residence at high altitude and as a consequence of prolonged left heart dysfunction or chronic obstructive pulmonary disease. It may also occur as a rare and fatal disease affecting young women – primary pulmonary hypertension. The symptoms of this generalised pulmonary vascular disease are those of pulmonary hypertension – that is, increasing breathlessness, syncope and, occasionally, right ventricular angina. In the case of patients with heart disease and chronic obstructive pulmonary disease the symptoms of the pulmonary hypertension may, in fact, be overshadowed by the symptoms of the primary cardiac or lung disorder, but undoubtedly the pulmonary circulatory abnormality contributes to the overall morbidity of these diseases. In contrast to this common and generalised defect of pulmonary arteries which can be associated with marked pulmonary vascular remodelling is a localised disease affecting the pulmonary circulation. This can take the form of arteriovenous anastomoses, aneurysms to the pulmonary arteries, pulmonary artery stenosis and, of course, thromboembolic pulmonary vascular disease. Pulmonary embolism is common, but even so can be difficult to diagnose with presently available imaging techniques. In approximately 1% of cases thromboembolism will progress to chronic thromboembolic pulmonary hypertension where the symptoms are of progressive breathlessness associated with the signs of marked pulmonary hypertension. In this issue of the journal are presented three cases of unilateral pulmonary artery disease.

The case described by Ferretti *et al* from Grenoble was a 44 year old man suffering from progressive interstitial lung disease who underwent successful unilateral lung transplantation. Following the transplant, however, he remained persistently breathless and hypoxaemic. The perfusion scan showed very poor perfusion of the transplanted lung so he was catheterised and pulmonary angiography was performed. Angiography demonstrated kinking of the donor pulmonary artery and there was a 44 mm Hg gradient across the resulting stenosis. Attempts to dilate the stenosis by balloon angioplasty were unsuccessful because, although the artery was easy to dilate, it returned to its kinked stenotic state on deflation of the balloon. The team therefore proceeded to place an endovascular stent into the pulmonary artery and successfully reversed the kink, resulting in improved perfusion to the lung. This improved perfusion allowed the patient to be weaned off the ventilator and he has remained well since.

In the second case Gibbs and Hami from UCLA describe the sad history of a 25 year

old man who presented to the medical centre with haemoptysis. Chest radiography demonstrated increased left lower zone shadowing and fiberoptic bronchosopic examination revealed an endobronchial mass. A biopsy sample was taken which caused profuse bleeding, resulting eventually in the death of the patient. Necroscopic examination showed a saccular aneurysm of the pulmonary artery which had ruptured into the bronchial tree. There was no obvious cause for this aneurysm and the patient did not have any of the systemic diseases associated with aneurysm.

In the third report by a group from Geneva (pp 1014–1015) a 37 year old man presented with cough and chest pain. Chest radiography showed decreased vascularity in the left lung and a ventilation perfusion scan showed an absence of perfusion of the same area. A CT scan showed obstruction to the left pulmonary artery, a mass in the truncus arteriosus, and three densities in the left lung, later found to be pulmonary emboli. The authors went on to perform CT guided pulmonary artery biopsy and a diagnosis of malignant sarcoma was made. The patient was treated by left pneumonectomy and replacement of the pulmonary valve by a homograft. This was followed by radiotherapy and the patient was well post-operatively but there are no details of subsequent follow up.

These three cases demonstrate the variation in clinical symptoms that can occur in association with unilateral pulmonary artery disease. The first case was more straightforward because the authors knew that the stenosis had followed a surgical procedure, namely lung transplantation. The interest here is that they successfully treated the kinked pulmonary artery with an endovascular stent and there is every reason to believe that this patient will remain well.

The other two cases show the difficulty of making accurate diagnoses of unilateral pulmonary artery masses. In the second case the mass presented as an endobronchial lesion. The authors should perhaps have been alerted by the presence of a “tumour” in such a young man and proceeded with further imaging before taking a biopsy sample.

In the third case the authors used multiple imaging techniques and did find obstruction of the pulmonary artery by a mass of unknown type. Despite the fact that the mass was in the pulmonary artery, they felt it safe to perform CT guided biopsy and rationalised this on the basis that the obstructed pulmonary artery would not bleed. This showed considerable confidence in the diagnostic techniques.

It is likely that we are all going to see more cases of masses on chest radiography which

may be due to abnormalities of a single or many pulmonary arteries. Clearly it is vitally important to make an adequate diagnosis of the problem before taking a biopsy sample which can have fatal results as demonstrated in the second case. Most hospitals will have available adequate imaging technology. If there is any doubt at all the patient should be referred for spiral CT scanning which can successfully demonstrate clot or other abnormalities in the

pulmonary artery and then, if the doubt persists, proceed to angiography. Only when the diagnosis is really clear should a biopsy sample be taken. Many would argue that this should only be done where adequate surgical control of any subsequent bleeding is possible. In most cases local pulmonary artery disease occurs in young, otherwise fit, people who have an excellent prognosis provided a firm diagnosis can be reached before any biopsy procedure.

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Chronic lung abscess due to *Pasteurella multocida*

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Abstract

A case of chronic lung abscess due to *Pasteurella multocida* presenting as a solitary pulmonary mass with a computed tomographic appearance suggestive of malignancy is described.

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Keywords: lung abscess, *Pasteurella*.

Solitary pulmonary masses frequently remain a difficult diagnostic challenge even though computed tomographic (CT) scanning and percutaneous needle biopsy have greatly improved the distinction between benign and malignant lesions. We present a case of a chronic lung abscess due to *Pasteurella multocida* which simulated a malignant process.

Case report

A 70 year old ex-smoker presented with purulent sputum and haemoptysis 10 years after left lower lobectomy for a T2N0M0 adenocarcinoma. He experienced mild chronic bronchitis but continued to work as a farmer. The chest radiograph showed a small ill defined opacity in the right retroclavicular area. Sputum culture grew *Pasteurella multocida* and *Moraxella catarrhalis*. The patient was treated with doxycycline 100 mg daily for two weeks. Although the symptoms subsided, the chest radiograph showed persistence of the opacity. Physical examination was non-contributory. Erythrocyte sedimentation rate was 37 mm in the first hour and the white blood cell count was normal. Spirometric measurements showed a forced expiratory volume in one second (FEV₁) of 2.05 l (67% predicted) and a forced vital capacity (FVC) of 4.75 l (120% predicted). The FEV₁ improved by 22% after inhaled bronchodilator. A chest CT scan revealed a 2 cm lesion with a central area of low attenuation and spiculated borders in the apical segment of the right upper lobe. Bronchoscopic examination showed patent bronchi but brush cytological specimens from the right upper lobe yielded atypical cells. Bacteriological studies of bronchial lavage fluid, including cultures for mycobacteria and fungi, remained negative. Percutaneous needle biopsy under CT guidance resulted in parenchymal haemorrhage and haemoptysis which resolved spontaneously after 24 hours. Cytological examination showed non-specific inflammatory cells. No material could be collected for bacteriological studies.

The lesion remained unchanged on a CT scan performed three months later, but appeared to have enlarged by about 50% by six months (figure). Another bronchoscopic examination with cytological and bacteriological studies of bronchial lavage fluid and brushings remained negative. Because of the increasing suspicion of malignancy, the patient underwent resection of the right upper lobe. Histological examination showed an accumulation of neutrophils surrounded by fibrosis without evidence of malignancy. Culture of the surgical specimen grew *P multocida* sensitive to penicillins and tetracyclines. On questioning, the patient denied any animal bite, but had been exposed to dogs and cattle. He was treated with amoxicillin/clavulanic acid 4 g



Computed tomographic scan of the chest showing a heterogeneous lesion with spiculated borders in the apical segment of the right upper lobe.