PATHOLOGICAL PULMONARY SYSTEMIC HYPERVASCULARISATION — A CASE REPORT

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SYNOPSIS

A 44 year old woman with past history of tuberculosis presented initially with recurrent haemoptysis associated with a continuous murmur over the right scapular area. Angiography of the pulmonary systemic circulation revealed pathological hypervascularisation of the bronchial arteries which were embolised resulting in immediate arrest of the haemoptysis.

INTRODUCTION

Pathological hypervascularisation of the pulmonary systemic circulation occurs frequently and is a common cause for haemoptysis (1). Angiography of the pulmonary systemic circulation is well-documented and can be employed to treat haemoptysis by embolisation. We report here a patient with recurrent haemoptysis from tuberculous bronchiectasis: trated successfully by therapeutic embolisation.

CASE REPORT

A 44 year old Chinese woman was first seen in this medical unit in 1976 for two month history of cough productive of whitish sputum. Clinical examination then showed bronchial breath sounds in the right upper zone posteriorly. Chest x-ray revealed an opacity in the right apical region (Fig. 1). Her direct smears were positive for acid-fast bacillus and she was treated with short course chemotherapy (Streptomycin, pyrazinamide, rifampicin and isoniazid). When she completed therapy in April 1977, her progress chest x-ray showed good resolution of the right upper zone opacity (Fig. 2)

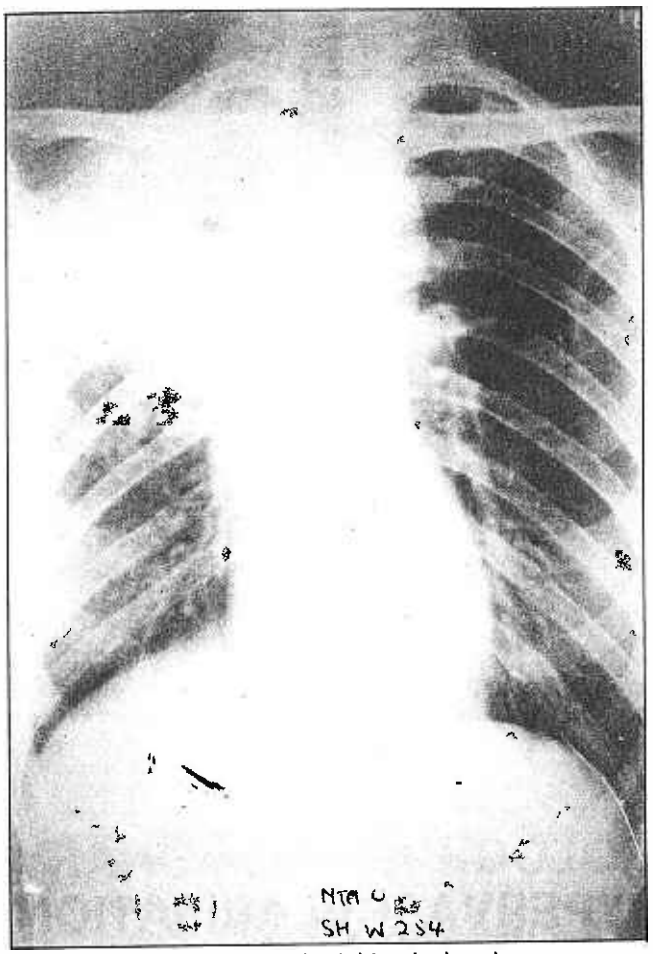

Fig. 1 Opacity in right apical region

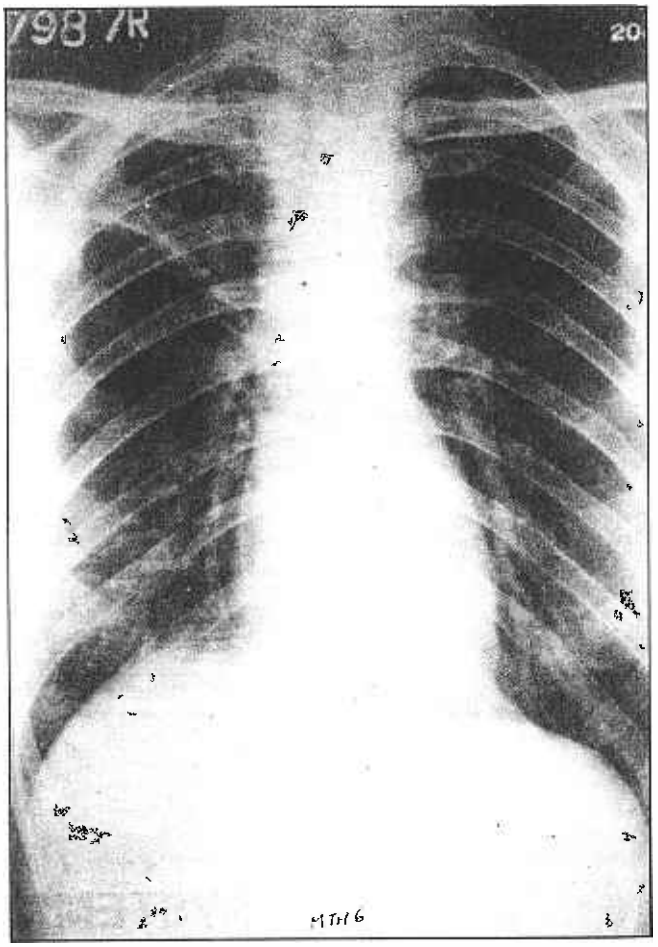

Fig. 2 Resolution of opacity

She was seen again in June 1982 for her first episode of haemoptysis. Her chest x-ray then showed residual fibrotic changes in the right apical region (Fig 3). On examination she was noted to have a grade 3/6 continuous murmur in the right apical area posteriorly. In view of the haemoptysis and the continuous murmur, an urgent pulmonary and bronchial angiogram was performed. This showed the presence of a shunt between the bronchial and pulmonary arteries. The bronchial arteries were catheterised via the descending aorta. These bronchial arteries were noted to be dilated and tortuous with connections by an arborised network of vessels to the pulmonary artery (Fig. 4, 5). All the three bronchial arteries were identified and were embolised. The right subclavian vessel was subsequently catheterised and numerous branches from the subclavian artery were noted to traverse the thickened pleura to supply the same arteriovenous malformation (Fig. 6). As these branches were small and numerous they were not embolised. Following the embolisation of the three bronchial vessels the haemoptysis was arrested.

A right upper lobectomy was subsequently performed and the histology showed the following:-

(1) segmental bronchiectasis of the lung,

(2) balls of fungus tissue in the bronchial lumen. Septate hyphae and vesicular dilatations were shown by GMS stain (Fig. 7)

(3) caseating granulomatous inflammation compatible with tuberculosis. However, no acid-fast bacillus was seen,

(4) dilated vessels which formed part of the arteriovenous malformation (Fig. 8).

Following discharge, this patient had no further episodes of haemoptysis and was started on second line tuberculous chemotherapy in view of the pathological findings.

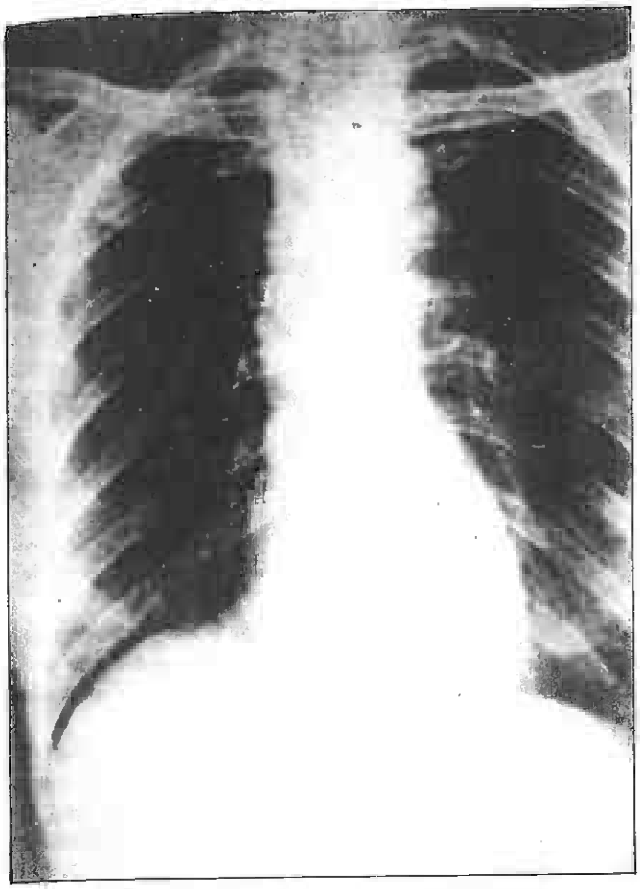

Fig. 3 Fibrotic change right apical region

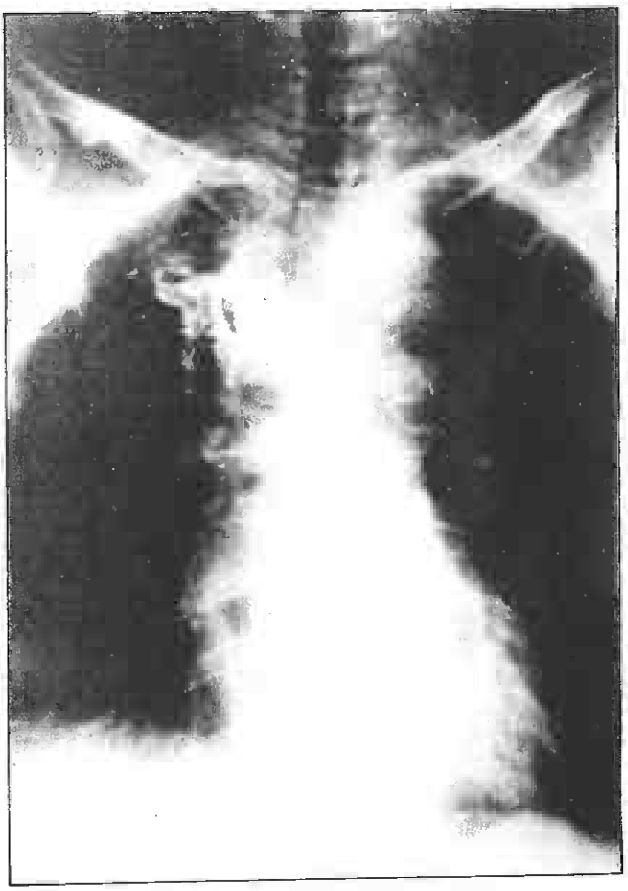

Fig. 5 Network of blood vessels

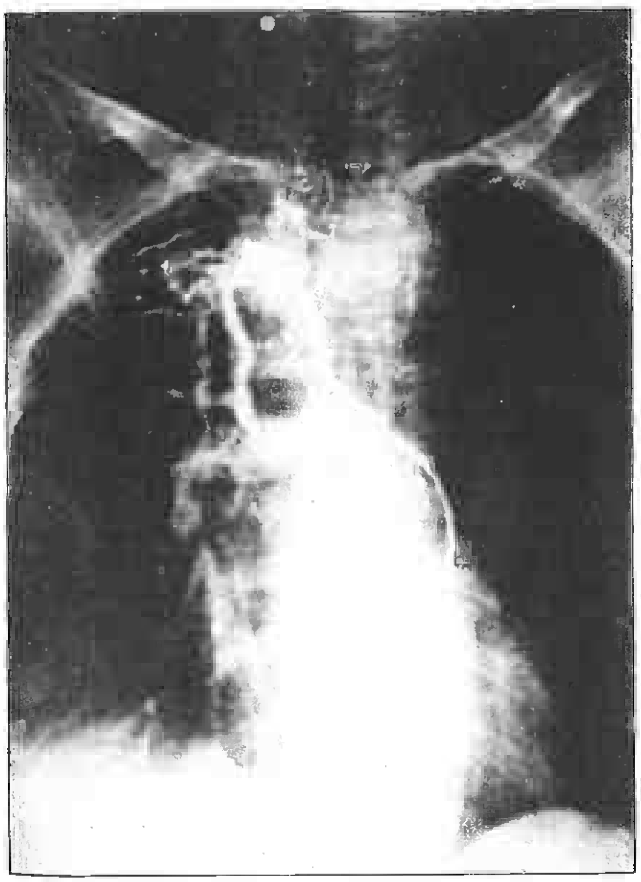

Fig. 4 Network of blood vessels

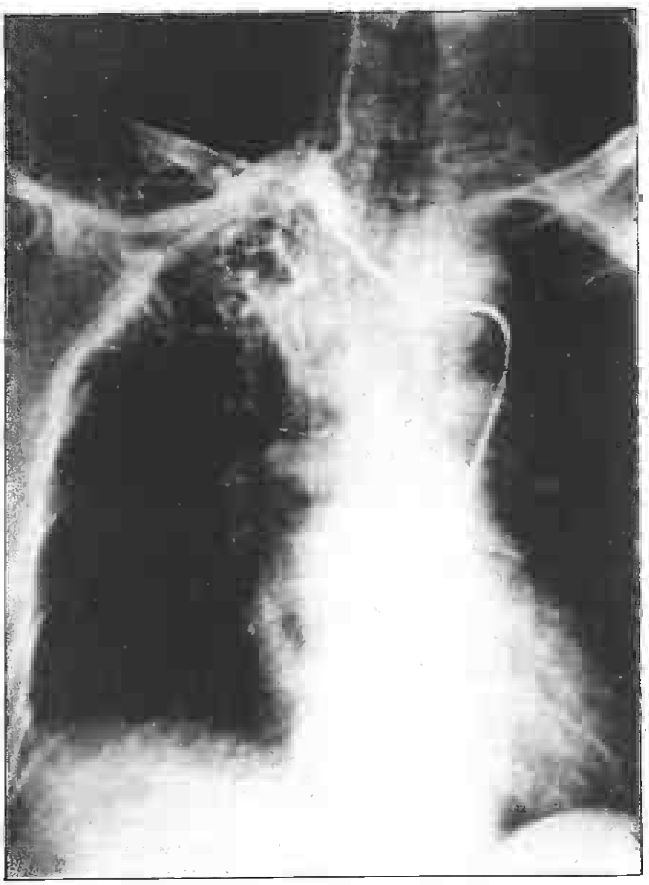

Fig. 6 Embolisation of vessels

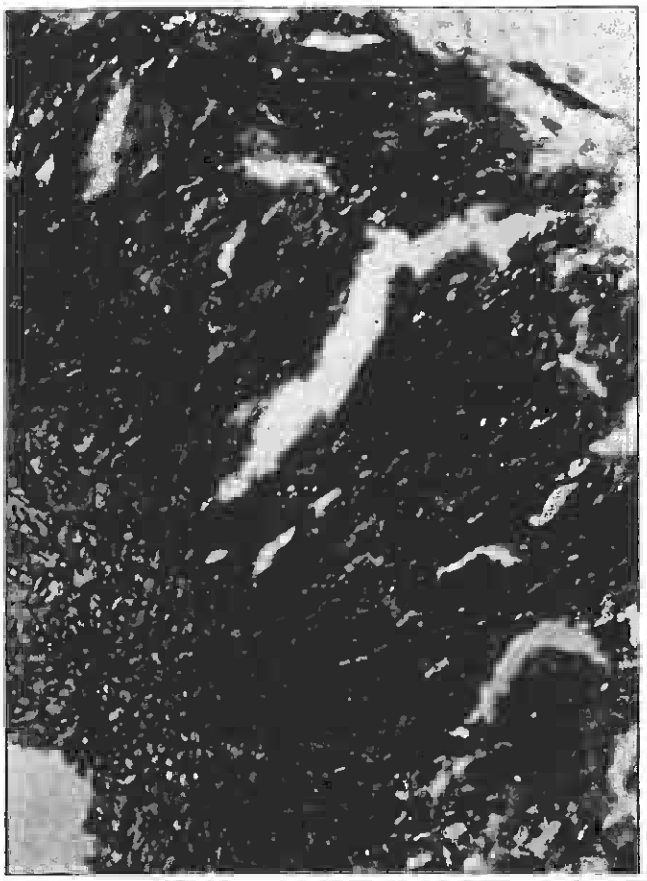

Fig. 7 Fungus tissue in lumen

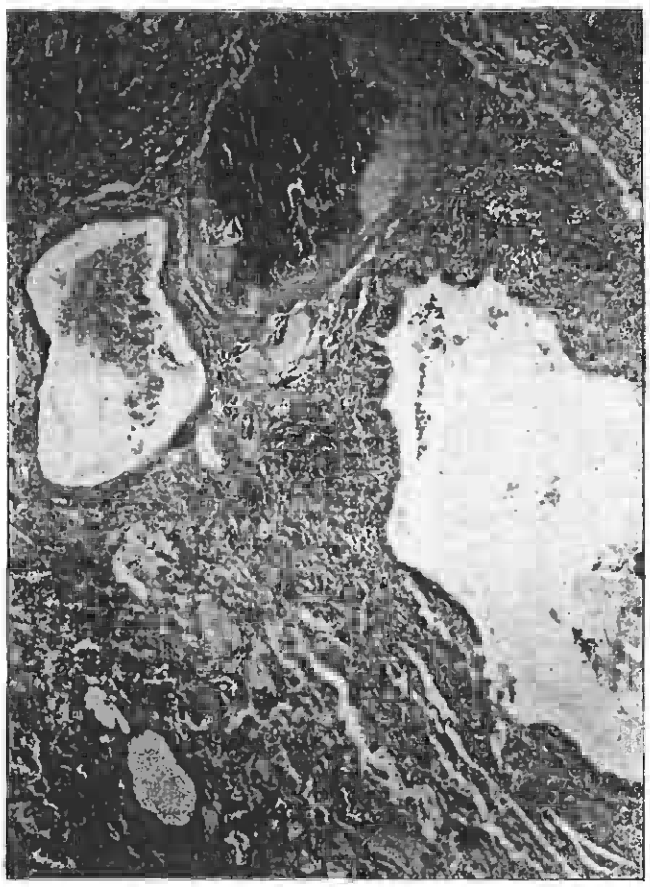

Fig. 8 Dilated vessels — part of A-V malformation

DISCUSSION

Pathological hypervascularisation of the pulmonary systemic circulation involves both the bronchial arteries and the non-bronchial systemic arteries such as the intercostal arteries, diaphragmatic arteries and branches of the subclavian arteries. The bronchial arteries are derived from the thoracic aorta and supply the bronchus and pulmonary tissue up to the level of the respiratory bronchiole while the pulmonary artery supplies the lung lobules. Normally, the pulmonary artery and the bronchial artery system do not communicate but in certain acquired pulmonary pathology, systemic pulmonary shunts arise (2).

Three types of distal shunts have been described.

(i) Systemic arteries to the pulmonary arteries, when the pulmonary vascular bed is destroyed,

(ii) Systemic arteries to the pulmonary veins, indicating preservation of the pulmonary vascular bed,

(iii) Systemic arteries to the pulmonary arteries and then to the pulmonary veins. This occurs with partial destruction of the pulmonary vascular bed.

Pathological hypervascularisation may lead to increased tortuosity and caliber of the vessels. Such changes may be unilateral or bilateral, diffuse or localised and commonly produce a cap-shaped image in the apex of the lung as in the patient described here. The apical arborisation seen in this patient was the result of the pulmonary-systemic shunting, increased tortuosity of the bronchial arteries and the numerous branches from the subclavian artery which traversed the pleural adhesions to the network of vessels.

The cause of the pathological hypervascularisation in this patient might be due to the destruction of lung tissue from tuberculosis, bronchiectasis and aspergillosis. These inflammatory processes produced hypoxemia of the lung parenchyma leading to neovascularisation. Systemic hypervascularisation may also be a sequelae of other conditions such as chronic bronchitis, bronchogenic carcinoma and pleural adhesions from surgical intervention. Chronic thromboembolism may also lead to the development of new collaterals from reduced pulmonary circulation.

Therapeutic embolisation of the systemic pulmonary arteries is indicated in life-threatening or recurrent haemoptysis which has been resistant to medical therapy and when surgical intervention is contraindicated. The presence of an anterior spinal artery arising from the cervico-intercostal trunk or the intercostal artery is a strong contraindication to the procedure as accidental medullary embolisation may occur (3).

Remy reported the arrest of haemoptysis in 41 out of the 49 patients treated (4). Of the 41 patients, 35 had remission lasting between 2 and 30 months. The main complication which occurred in his study was that of dysphagia associated with burning retrosternal sensation. Epigastric pain also occurred from necrosis of the small bowel. There was no complication in the patient described here.

Therapeutic embolisation is only a palliative procedure for the arrest of haemoptysis as revascularisa-

tion or recanalisation of the vessels may occur (5, 6). The result of the procedure depends on the underlying pathological lung condition. In tuberculosis or bronchiectasis, the success rate is high as pathological vascularisation involves mainly the bronchial arteries which can be readily identified and embolised (4). However, in aspergillosis arising in post-tuberculous cavities the procedure is less successful as the blood supply is derived from the numerous branches supplied by the axillary and subclavian branches and embolisation of all these branches may be difficult.

In summary, any patient with recurrent or massive haemoptysis from acquired pulmonary pathology such as tuberculosis may benefit from angiographic exploration of the pulmonary systemic circulation which enables one to identify the bleeding points and embolise them. However, as the procedure is palliative definitive surgery is indicated at a later stage when the patient is more clinically stable.

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