THERAPEUTIC EMBOLIZATION OF PULMONARY ARTERIOVENOUS MALFORMATION

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ABSTRACT

A 49-year old man presented with recurrent transient ischaemic attacks and a pulmonary arteriovenous malformation (PAM) was found in his lower left lobe. Left lower lobectomy was performed, and the attacks ceased. However, 18 months later, a fresh PAM was detected in the right lower lobe. In order to avoid further lung resection, therapeutic embolization with metal-dacron coils and gelfoam was performed, resulting in complete occlusion of the PAM. The patient remained well at follow-up 6 months later. Therapeutic embolization is an effective alternative to surgery in the treatment of PAM, especially in the presence of multiple lesions when it is important to conserve functional lung tissue.

Keywords: Pulmonary arteriovenous malformation, therapeutic embolization.

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INTRODUCTION

Pulmonary arteriovenous malformation (PAM) may appear as an isolated anomaly or in association with hereditary haemorrhagic telangiectasia. Rare but serious neurological complications like brain abscess and cerebral infarction may occur due to paradoxical embolism or related polycythaemia (1, 2). Infection and rupture with massive bleeding are life threatening. Treatment is indicated for large or multiple lesions (3). Resection is an acceptable f rm of therapy when dealing with isolated lesions, while therapeutic embolization is preferrable in patients with multiple lesions in order to conserve lung tissue. In this report, we describe a patient whose PAM was successfully embolized with metallo-dacron coils and gelfoam.

CASE REPORT

A 49 year old man first presented 11 years ago with right hemiparesis and speech disturbance from which he recovered completely within 24 hours. There were two further transient ischaemic attacks (TIA) involving speech and face. He was referred to the Prince of Wales Hospital in January 1986 with drooping of the right eyelid and slurred speech. On examination, there was marked clubbing of his fingers and toes but no features of hereditary haemorrhagic telangiectasia. He had motor dysphasia, right sided VII, IX, X, XII and partial III nerve palsy, without long tract signs. Examination of the heart and lungs was normal. No bruit was heard. His chest radiograph (CXR) revealed tubular and round shadows in the left lower zone which were partly obscured by the left heart border (Fig. 1). Arterial blood gas tension (Pa02 9.52 KPa, PaCO2 4.2 kPa) did not change significantly on breathing high concentrations of oxygen. Haemoglobin concentraion was 16.3 g/dl and blood biochemistry was normal. Echocardiography did not reveal any intracardiac shunt or valvular lesion. A first pass perfusion scan using Tc 99 DTPA demonstrated a large pulmonary arteriovenous malformation (PAM) in the left lower zone. All neurological signs disappeared within 24 hours after admission.

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He was referred for surgery. At thoracotomy, a large pulsating PAM was found in the anterior segment of the left lower lobe with feeding vessels from the left lower lobe pulmonary artery. Left lower lobectomy was performed, and histological examinations showed dilated venous spaces mixed with arteries of varying size. Post-operative recovery was uneventful, and blood gas analysis showed some improvement with a PaO2 of 11.2 kPa and PaCO2 of 5.49 kPa on room air.

At follow up 18 months later, the patient was well without futher TIAs. However, persistent clubbing of digits was noted. On auscultation, a bruit which was loudest during inspiration was heard over the right lower zone. CXR showed a new tubulo-nodular shadow in the right lower lobe (RLL). He was readmitted in March 1988 for consideration of therapeutic embolization. Catheterization of the pulmonary artery was performed percutaneously from the right femoral vein and superselective angiography confirmed the presence of a PAM in the RLL (fig. 2). No other PAMs were found elsewhere. One 8 mm and two 5 mm metallo-darcon coils were introduced one by one via the selective catheter to the feeding artery of the PAM, followed by a gelfoam segment. Permanent and total occlusion of the PAM was achieved as shown by the post-embolisation angiogram (fig. 3). The whole procedure was well tolerated and the chest bruit was no longer audible afterwards. Arterial blood gas tension demonstrated an improvement with a PaO2 of 15.2 kPa and PaCO2 of 4.7 kPa on room air. The patient remained well at 6 months during follow up and a repeat CXR showed that the metallic coils were in-situ.

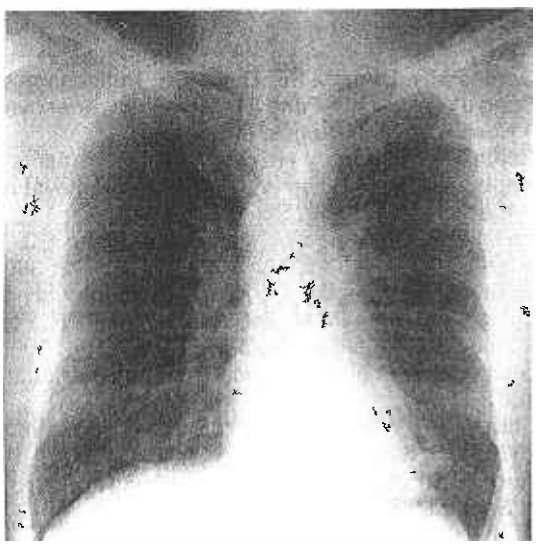

Fig. 1: CXR of the patient showing multiple tubular and round shadows in the left lower zone.

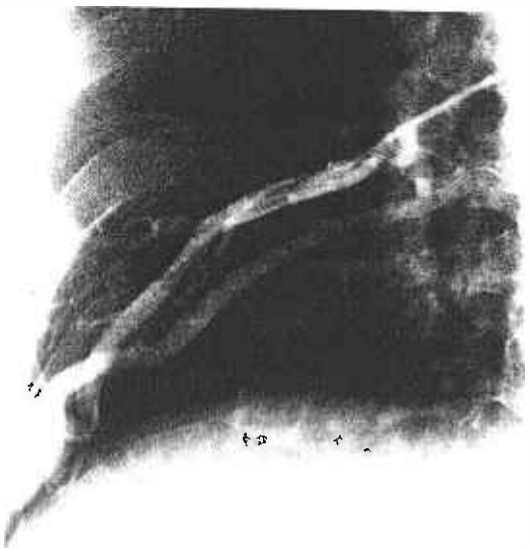

Fig. 2: Superselective pulmonary angiogram showing the right lower zone pulmonary arteriovenous malformation.

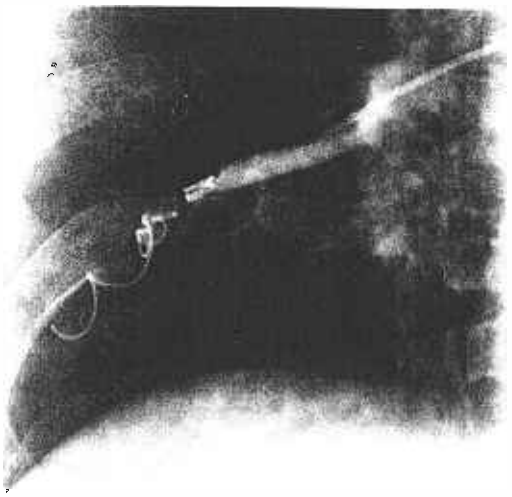

Fig. 3: Post-embolization angiogram of the right lower zone showing total occlusion of the pulmonary arteriovenous malformation and the metallo-dacron coils in-situ.

DISCUSSION

Surgery has been the mainstay of treatment for PAM since the first reported cure in 1942 by Hepburn and Dauphinee (4). It is generally believed that surgery should be conservative with resection of minimal lung tissue. In our patient, left lower lobectomy had already been performed when the RLL PAM was found. It was likely that this lesion was too small to be detected initially, but enlarged during the follow-up period after surgery. This phenomenon is well recognized (3). Another surgery on the right side would mean further loss of pulmonary function in an otherwise physically fit subject. Under these circumstances, therapeutic embolization first reported by Taylor et al (5) is an excellent alternative mode of treatment which would preserve lung function and avoid the risk and morbidity of a second operation.

It has been shown histologically that no communications exist proximal to the malformations, unless multiple feeders are present (6). Occlusion of the pulmonary artery branch leading to the PAM should result in total occlusion of the corresponding shunt. The diameters of the feeding arteries vary from 2 mm to 8 mm, and the draining veins are larger than the corresponding arteries (6). Most commonly the feeding vessel is a branch of the pulmonary circulation (96%) as opposed to systemic circulation.

Angiography provides precise information as to the size, number and location of the lesions. Embolization technique had been described in great detail by Taylor et al (5) and Zuniga et al (7). There is a potential risk of systemic embolization through the PAM if occluding devices smaller than the size of the fistula are used. The vessel occluding device as initially described by Gianturco et al (8) is useful to form a mechanical baffle. Modified coils are now available (9). In our patient, metallo-dacron coils were introduced followed by a gelfoam segment. Alternatively one can use detachable balloons for embolization (10).

While surgery remains a therapeutic option for a single large PAM, our case clearly demonstrates that therapeutic embolization is both effective and safe. This alternative form of treatment deserves strong consideration, especially in cases with multiple lesions in whom surgery is likely to be inappropriate.

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