Infection of modified Blalock shunts

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SUMMARY A left-sided interposition graft (modified Blalock-Taussig anastomosis) was constructed with polytetrafluoroethylene in a three month old child with tetralogy of Fallot. This shunt thrombosed and a replacement shunt became the site of chronic *Pseudomonas* infection. The second anastomosis was excised and a third interposition graft was inserted on the right side because the anatomy was unsuitable for a classical Blalock-Taussig shunt. The patient died when he was 12 months old, after signs of infection and shunt occlusion had developed. At necropsy the acutely thrombosed right sided shunt was found to be the site of *Candida albicans* infection. Gallium and labelled white cell scans, computed tomography, and ultrasound scans had all failed to identify the sites of infection, which were only confirmed at operation or necropsy.

Subclavian to pulmonary artery shunts constructed from interposed grafts of polytetrafluoroethylene have been widely used since the introduction of this material to vascular surgery.¹ Such shunts are easy to construct and take down, they are less likely to distort the pulmonary arteries, they preserve flow to the subclavian artery (avoiding ischaemia of the arms), and they can provide adequate blood flow to both lungs.^{2,3} Early thrombosis, particularly in neonates, is less common after this shunt procedure than after a classic Blalock-Taussig shunt.³ Complications reported after the modified Blalock shunt include excessive pulmonary blood flow,⁴ seroma formation,⁵ and false aneurysm.⁶

We describe an infant, who probably had an immunological disorder, in whom successive shunts became infected with different organisms. These infections eventually led to his death.

Case report

A left-sided modified Blalock shunt (5 mm Impra graft) was constructed soon after the onset of hypercyanotic spells in a three month old boy with tetralogy of Fallot. His colour did not improve and a shunt murmur was not audible. In addition, there was a hypercyanotic spell on the first postoperative

day. Angiography showed poor filling of the shunt because of a proximal stenosis. At reoperation the shunt was replaced by a second 5 mm Impra graft. His colour and arterial PO₂ improved considerably and a loud continuous murmur was audible. Artificial ventilation was required for four days because he had left phrenic nerve palsy. Two and six weeks after operation there were episodes of acute infection and blood cultures were positive for *Pseudomonas aeruginosa*. An intensive search for a focus of infection with ultrasound, computed tomography, and white cell scan incriminated only the left lower lobe of the lung. A sweat test for cystic fibrosis was negative. After a prolonged antibiotic course with azlocillin, amikacin, and benzylpenicillin he recovered and was transferred to another hospital for management of feeding difficulties. While he was there he had several episodes of *Pseudomonas aeruginosa* septicaemia and left-sided pneumonia despite appropriate antibiotic treatment.

At the age of nine months he weighed 5.6 kg and was mildly cyanosed with clubbed fingers; the liver was palpable 3 cm below the right costal margin. The respiratory rate was 40 per minute with subcostal recession and bilateral crepitations. The shunt murmur was easily audible. Infection of the Impra graft was strongly suspected. A right thoracotomy was performed with the intention of constructing a right classic Blalock-Taussig shunt before excision of the left shunt. The anatomy was unfavourable, however, and a 5 mm Impra graft was inserted on the right. A left thoracotomy was performed. Dense adhesions were found. The subclavian artery was

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ligated and divided, the left Impra graft was completely excised, and the left pulmonary artery was repaired with a patch of autologous pericardium. After operation he was treated with intravenous azlocillin and amikacin for 21 and 14 days respectively. *Pseudomonas aeruginosa* sensitive to these antibiotics was isolated from this shunt. Six days after operation, while he was still on the ventilator, he became acutely unwell and *Candida* was cultured from the tip of an indwelling urinary catheter. He was treated with flucytosine and he improved gradually with no further evidence of *Candida* infection. When antibiotic treatment was stopped, however, episodes of fever and leucocytosis occurred. Screening for sepsis was negative on all these occasions. A chest radiograph showed bilateral chronic patchy opacities and there was a widening of the mediastinum on the right. Tests of immunological function were generally abnormal, with reduced complement components, reduced *Saccharomyces* opsonins and a serum-dependent neutrophil killing defect for *Candida albicans*. Concentrations of IgG, IgA, IgM, and IgE were all raised, as was the C-reactive protein (20 mg/l), which indicates active infection at the time of the study. The patient did not react to *Candida* skin tests. Finally, repeated blood cultures grew *Staphylococcus epidermidis*. A gallium scan showed no evidence of focal infection.

He was treated with flucloxacillin, fucidic acid, and several infusions of fresh frozen plasma, and his general condition and the appearance of the chest radiograph improved dramatically. The right mediastinal shadow persisted and was thought to represent organised haematoma around the right shunt. In the mean time, plication of the left hemidiaphragm was performed because of continued ventilator dependence. He was eventually extubated three weeks after this procedure. He was again transferred to another hospital for convalescence.

Two weeks later he became acutely ill and cyanosed. The shunt murmur was inaudible. He had a fever and signs of disseminated intravascular coagulation, and despite treatment with antibiotics and pressor agents he died. At necropsy no pus was found either near the shunt or elsewhere. There was no sign of endocarditis. The right Impra graft contained recent thrombus that occluded the lumen. The shunt was surrounded by a greenish-brown mass 1.8 cm in diameter which compressed but did not invade the right upper lobe (fig 1). Microscopy of this mass showed organising Surgicel and fibrin traversed by scores of *Candida* hyphae. Microscopy of the thrombus showed collections of neutrophils and the presence of *Candida* yeasts and pseudo-hyphae (fig 2). Large numbers of alveolar macrophages with a light lymphocytic infiltrate were seen

Fig 1 Photographs of the heart and lungs. (a) Anterior view of the whole specimen. (b) Close up view of the right hilum dissected to show abscess in front of the shunt (arrows).

Fig 2 Photomicrograph of the thrombus showing numerous *Candida pseudohyphae* growing within organising fibrin.

in lung sections. There was no evidence of bronchopneumonia and in the lungs stains for *Candida* and other micro-organisms were negative.

Discussion

The modified graft constructed from polytetrafluoroethylene effectively palliates children with congenital heart disease who have reduced pulmonary blood flow and, although it offers advantages over the classic Blalock-Taussig anastomosis, complications occasionally occur. Formation of false aneurysms in patients with polytetrafluoroethylene grafts was reported and ascribed to infection. Monarrez *et al* attempted to excise a shunt that was presumed to be infected and in which detachment of the proximal end had resulted in pseudoaneurysm formation.⁶ The patient died of uncontrollable blood loss. The shunt was occluded by a sterile thrombus. No mention was made of the results of bacterial culture of the graft. Opie *et al* described five patients with polytetrafluoroethylene grafts in whom ipsilateral pneumonia developed within six months of operation.⁴ In two patients with positive blood

cultures, aneurysms developed that were associated with the shunt. In neither case, however, were results of bacterial culture of the shunt given.

Confirmation of an infection affecting a prosthetic shunt may be difficult. In the present case none of the investigations designed to find the site of chronic infection pointed to either of the last two shunts. Eventually only surgery to resect the shunt and subsequent direct bacteriological culture proved that the left Impra graft was indeed infected. Proof of sepsis in the right Impra graft was only obtained at necropsy. In view of this experience, we agree with others^{4,6} that the occurrence of repeated episodes of septicaemia, late shunt thrombosis, and the development of aneurysms related to the shunt are all strong indicators of graft sepsis. Definitive treatment should not be delayed because diagnostic investigations are negative. Mediastinal enlargement on the chest radiograph is more likely to be caused by haematoma or seroma⁵ than pseudoaneurysm secondary to shunt infection. In the presence of other suspicious features, however, angiography should be performed to identify the cause.⁶

We do not know whether the repeated infections

and graft sepsis were a result of a primary immune deficiency affecting neutrophil and complement dependent mechanisms, or whether the abnormal results of immunological function were secondary to chronic infection caused by deep-seated graft sepsis (with continual seeding of the blood stream) and recurrent pneumonia. We were not able to perform immunological tests when he was clinically well.

The management of shunt infection may be difficult. When a pseudoaneurysm is present there will be torrential bleeding at the time of excision.⁶ Because prosthetic material used either to create another shunt or to correct the intracardiac anomaly may be contaminated the use of the classic Blalock-Taussig or even Waterston shunt may be preferred. Bovine pericardium sterilised with antibiotic may be better than synthetic cloth for the closure of ventricular or atrial septal defects or both. A complete course of the appropriate intravenous antibiotics should be given when any infection is diagnosed soon after construction of a prosthetic shunt. If the same organism is repeatedly isolated after the end of antibiotic treatment, a shunt infection must be presumed to exist and the septic graft should be removed without delay. It can be disastrous to wait for the results of expensive and complicated tests whose sensitivity and specificity for diagnosing shunt infection are unknown.

We thank Dr A J Pinching, Department of Immunology, St Mary's Hospital Medical School,

for performing the tests of immunological function; Dr E A Sheffield, Department of Histopathology, Brompton Hospital; Dr M L Rigby for permission to report this case; and Dr E A Shinebourne for his helpful advice. J S C is supported by CNPq-Brazilian Research Council.

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