CASE REPORT

Treatment of Arterial Steal Syndrome Secondary to Haemodialysis Access

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Introduction

An uncommon complication of arteriovenous fistulae constructed for haemodialysis is vascular steal syndrome.1 In most cases this occurs because large volumes of blood are shunted through the fistula leading to distal limb ischaemia. Consequently, treatment of vascular steal has concentrated on decreasing or abolishing the blood flow through the arteriovenous shunt.2–4 Vascular steal syndrome may also occur if a fistula is proximal to occlusive arterial disease in the limb. The restriction of flow to the limb is then compounded by the shunting of blood through the proximal fistula. Unfortunately, this is not commonly recognised and it is scarcely mentioned in vascular access texts.5 We describe two patients in whom symptomatic arterial steal was successfully treated by angioplasty or thrombolysis of a distal arterial lesion, thereby obviating the need for operative correction of the blood flow through the shunt.

Case Reports

JP is a 78-year-old male who developed end-stage renal failure due to an unknown cause in 1997. A right-sided radiocephalic fistula was formed, but immediately found to have insufficient flow. The fistula was taken down and an ipsilateral brachio-cephalic fistula was formed at the same anaesthetic. Following surgery the patient developed ischaemic rest pain in his right hand. A duplex scan showed a 60–80% stenosis (peak systolic velocity ratio 3.0) in the brachial artery, just distal to the arteriovenous anastomosis. At angiography the peripheral arteries could only be visualised with the fistula occluded by external compression, but confirmed the presence of a diaphragm-like stenosis of the brachial artery, distal to the anastomosis. The venous limb of the fistula was punctured and after pressure measurements were taken across the stenosis, sequential angioplasty to 3 mm and then 5 mm was performed through a 5 French sheath. The run-off improved substantially and clinically the patient experienced resolution of the ischaemic symptoms.

DT is a 59-year-old male who developed end-stage renal failure due to glomerulonephritis in 1979. Previous radiocephalic wrist fistulae and a right brachio-basilic PTFE arteriovenous shunt had all failed. In 1997 a left-sided 6 mm PTFE brachio-basilic arteriovenous shunt was formed. One month after the procedure he started complaining of pain in the left hand at rest and developed patches of necrosis on the index and middle finger. An angiogram demonstrated shunting through the fistula with no demonstrable distal arterial flow. On fistula compression an occlusion of the brachial artery above the elbow was revealed. A guidewire was readily passed through the occluding thrombus and rtPA thrombolysis successfully recanalised the vessel. Distal arterial flow was improved and subsequently the rest pain and necrosis in the left hand resolved without any further intervention.

Discussion

Arterial steal may develop in 3–7% of patients after formation of an arteriovenous fistula.2,3 It may occur
in both the upper and lower limbs and is more common in proximal fistulae using large inflow vessels, although it may rarely occur in wrist fistulae. Arterial steal may also be more common in diabetic patients.

The two patients in this report developed severe upper limb ischaemia secondary to the combination of arterial disease in the upper limb and the presence of an arteriovenous fistula. These patients are important to identify as it may be possible to correct their ischaemia without surgical intervention to the fistula. The underlying distal arterial lesions will be identified by colour flow duplex or angiography, but in case of the latter it should be performed with external compression of the fistula. Without compression, as in our cases, it may be impossible to demonstrate the distal arterial tree and treatable occlusive arterial lesions may be missed. Once identified, endovascular treatment may be successful in dealing with distal arterial disease.

Previously described treatments such as banding may lead to loss of the fistula, as will fistula ligation. This may result in significant morbidity, and may not always be successful in the presence of occlusive distal arterial disease.

In patients with arteriovenous fistulae and arterial steal it is important to remember that a distal arterial lesion may be responsible. Appropriate investigation and treatment will maximise the chances of treating the ischaemia and preserving the arteriovenous fistula.

References