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Percutaneous transluminal angioplasty in transplant renal arterial stenoses: a long-term follow-up

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Abstract. Transplant renal artery stenosis is a potentially treatable cause of post-transplant hypertension. Transluminal angioplasty under radiological control facilitates treatment, and in this study the results of 15 consecutive patients treated in one department have been analysed to assess the safety and success of the technique, blood pressure control and transplant function. Technical success was achieved in 14 patients (93%), with zero mortality and only 1 major complication requiring surgical intervention. Blood pressure control was significantly improved by angioplasty, mean blood pressure (diastolic + 1/3 pulse pressure) being lower 4–6 weeks later ($P < 0.05$) and a mean of 34 months later ($P < 0.01$), and transplant renal function was unaffected. In the treatment of transplant renal artery stenosis percutaneous transluminal angioplasty is effective, safe and often of lasting benefit.

Key words: Renal artery stenosis – Artery, renal, stenosis – Angioplasty, renal artery – Hypertension, renal artery stenosis

In recent years there have been major advances in the management of the renal allograft recipient, with improved patient and graft survival. However, hypertension, which is extremely common in the dialysis population, tends to recur commonly after transplantation, with an incidence between 50% and 80% in some series [7, 11]. Roughly 10% of this group are reported [1, 6] to have transplant renal artery stenosis (TRAS), when hypertension is often combined with impairment of renal function.

Percutaneous transluminal angioplasty (PTA) is the current method of choice for treatment of the hypertensive patient with TRAS [2, 12]. We describe our experience with 15 patients and report on their long-term follow-up.

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Patients and methods

All renal transplant recipients with TRAS referred to the Radiology Department at the Northern General Hospital, Sheffield, from August 1981 to November 1987 were analysed retrospectively. The study includes all patients identified with TRAS from two centres and a proportion from a third centre.

All angioplasties were performed by one of us (D. C. C.) using a 4- to 6-mm balloon dilatation catheter. Anticoagulants and antiplatelet drugs were not routinely used during or following the procedure. Technical success was assessed with an angiogram at the end of the angioplasty.

A stenosis was regarded as significant if it had a greater than 50% reduction in diameter, and the artery was judged adequately dilated if there was at least 20% improvement in the luminal diameter *and* the residual reduction in diameter was less than 50%.

Paired analysis used the Wilcoxon signed rank test so that no assumptions regarding the distribution of the data were necessary. The two-tailed probability is given.

Results

There were 15 patients, eight female and seven male, with a mean age of 40 (range 14–65) years. In all cases, preservation of the donor kidney was by simple cold flushing with kidney preservation solution. Immunosuppression was with prednisolone and azathioprine in eight patients and prednisolone and cyclosporin A in seven patients. Prior to diagnostic angiography, an arterial bruit was present over the transplant in eight patients and absent in two patients; in the other five patients, information regarding this was not available.

There were 19 renal arteries in 15 patients, with significant stenosis in 16 arteries. An end-to-side anastomosis was performed to the common or external iliac artery in 12 patients, and to the lower aorta in one patient. All except one of these included a Carrel patch, in which a patch of donor aorta bearing the renal artery/arteries facilitates anastomosis. The remaining two patients had an end-to-end anastomosis to the internal iliac artery.

The mean period of time from transplantation to detection of TRAS was 9.3 months (range 2–42 months). Ten of the 15 patients were within 6 months of transplantation and only in two was stenosis diagnosed more than

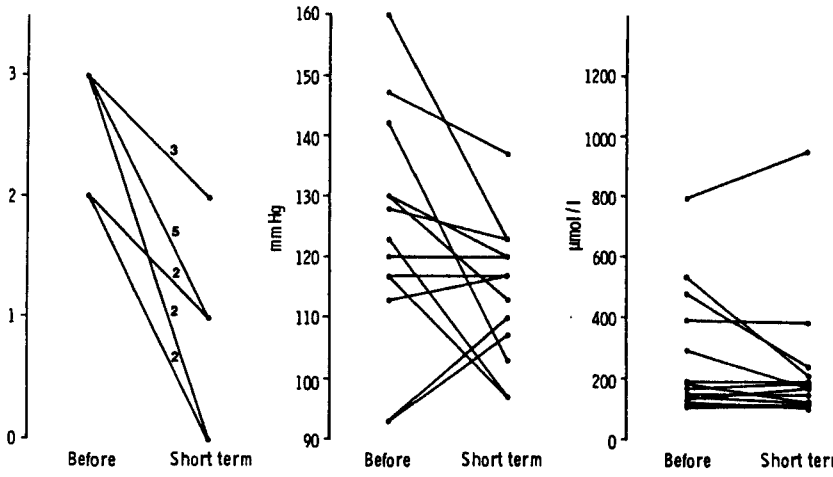

Fig. 1a–c. Short-term follow-up: **a** number of antihypertensive drugs ($P < 0.01$), **b** mean blood pressure ($P < 0.05$) and **c** serum creatinine (NS) in the 14 patients before and 4–6 weeks after percutaneous transluminal angioplasty (PTA)

12 months after transplantation. The mean duration of hypertension prior to diagnostic angiography was 2.7 months (range 2 weeks to 23 months). The indication for angiography was the presence of hypertension or unexplained impairment of renal function or both.

Diagnosis was by conventional aortography with selective renal transplant arterial catheterisation in ten patients and digital subtraction angiography through an antecubital vein in five patients. The stenosis was anastomotic in six patients, two of whom had an end-to-end anastomosis and one an end-to-side without a Carrel patch. In one other patient, who had a deficient Carrel patch, the sutures included the donor renal artery. All the remainder had post-anastomotic stenosis. One of the latter group had a proximal common iliac stenosis as well.

In 14 of the 15 patients (93%) PTA was technically successful. In one patient with two stenosed arteries the lesion was crossed with a guide wire but the catheter could not be negotiated despite attempts from the axillary and femoral route. Subsequent information refers to the 14 patients only.

Complications were few and often relatively minor. However, one patient developed a haematoma in the groin which had to be evacuated surgically with repair of the femoral artery. Another who underwent an unsuccessful attempt at a second angioplasty developed an axillary haematoma which resolved spontaneously. Two other patients developed a pyrexia treated with systemic antibiotics. In five patients there was a transient rise in serum creatinine (> 20%), recovering within a few days, and in two there was a permanent deterioration in renal function. No patient died or suffered total loss of transplant function directly as a result of the procedure.

Short-term follow-up (Fig. 1)

Four to six weeks following PTA all 14 patients were either cured ($n = 4$) or had improved blood pressure control on less medication ($n = 10$) and the reduction in medication was statistically significant ($P < 0.01$). The mean blood pressure (diastolic + 1/3 pulse pressure) after PTA was reduced in nine patients, unaltered in two patients and slightly elevated in three patients; the overall reduction was significantly different ($P < 0.05$) from pre-PTA values. The serum creatinine fell by more than 30% in four patients and was raised by 20% in one patient; nine patients had no obvious change in their serum creatinine levels and overall there was no statistical difference.

Long-term follow-up (Fig. 2)

Clinical features determined the need for reinvestigation, as routine Doppler vascular imaging was not available. Relapse of the good control of hypertension or further unexplained deterioration in the transplant function resulted in six repeat arteriograms 2–32 months after the initial PTA. Two intravenous digital subtraction angiograms revealed no evidence of restenosis and of four conventional arteriograms there was no restenosis in one, but restenosis in three.

Three patients were not available for long-term evaluation. One showed a rise in serum creatinine from 880 µmol/l to 960 µmol/l following PTA; biopsy revealed a brisk vascular rejection and the patient died 4 months later in end-stage renal failure. Another patient had recurrent stenosis 7 weeks later and, following an unsuccessful attempt at repeat angioplasty, was revascularised surgically; she developed ischaemia of the ipsilateral lower limb and haemorrhage from the abdominal suture line and died 2 months following the operation. A third patient, who had no recurrence of her stenosis on a repeat angiogram performed 4 months later, lost her transplant 19 months following her angioplasty from chronic rejection.

The remaining 11 patients were followed up for a mean of 34 months (range 12–74 months). Two patients who had recurrent stenosis and repeat PTA 9 months and 32 months later are included in the follow-up. Two patients were cured, eight were on less antihypertensive medication and the one patient who had returned to his original medication had a lower mean blood pressure and better renal function than before PTA. This level of blood pressure control was significantly better than before PTA ($P < 0.01$, Fig. 2) and no different from 4–6 weeks following the procedure. The mean blood pressure was also significantly improved ($P < 0.01$, Fig. 2). The serum creatinine was generally improved, but one patient showed a

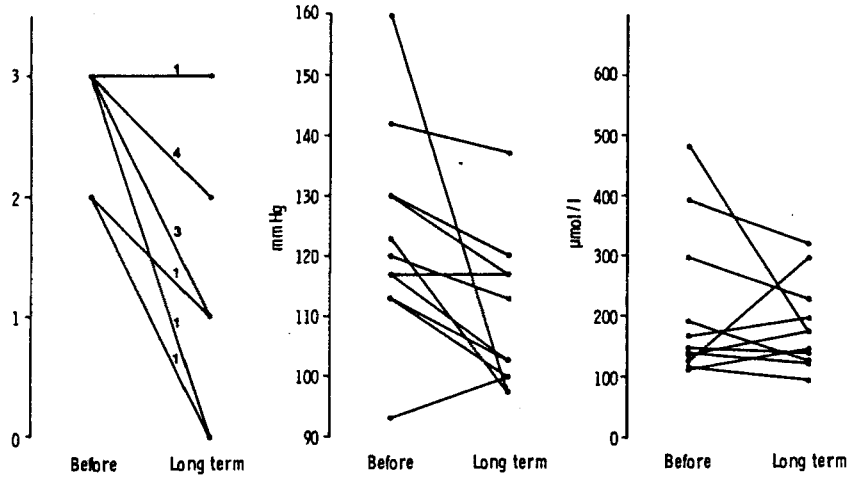

Fig. 2a–c. Long-term follow-up: **a** number of antihypertensive drugs ($P < 0.01$), **b** mean blood pressure ($P < 0.01$) and **c** serum creatinine (NS) before PTA and at last follow-up in the 11 patients followed for 12–74 months

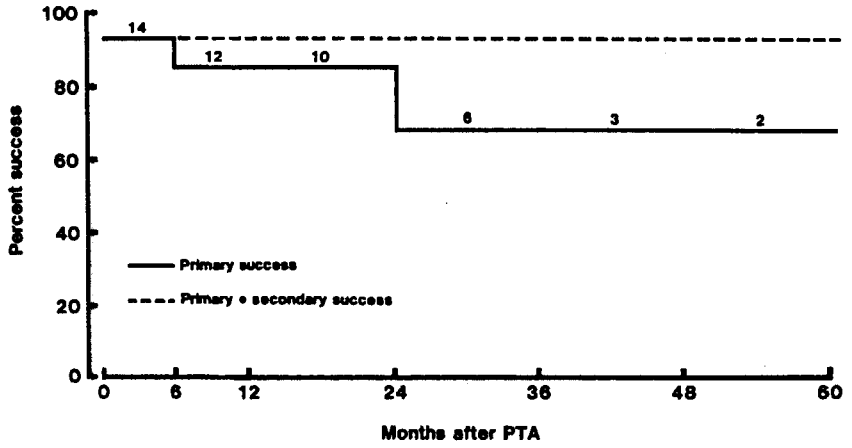

Fig. 3. Life table analysis of success after PTA for transplant renal artery stenosis. The number of patients evaluated in each time period is shown

marked deterioration and as a result of this there was no significant difference in the mean from the pre-PTA values (Fig. 2).

Restenosis occurred in three patients after successful PTA; all were in post-anastomotic stenoses associated with patched end-to-side arterial anastomoses. The restenoses were diagnosed 7 weeks, 9 months and 32 months, respectively, after PTA; failure to redilate the first of these has already been described. The restenoses after 9 and 32 months were successfully redilated with further amelioration of the respective patient's hypertension and renal functional impairment. Life table analysis of the success of PTA in the treatment of TRAS is graphically presented in Fig. 3. Primary success was 93% after 6 months, 85% after 1 year and 68% after 5 years. When the secondary successful redilatations are included, overall success after 5 years remains 93%.

Discussion

The rate of detection of transplant renal artery stenosis has varied from 1% to 14% in different studies [9, 10]. However, the true incidence is unknown and may be as high as 25%, the figure partly depending on definition. In one study [6], arteriography was routinely performed, with evidence of arterial stenosis in 23 of 100 transplant recipients, but the presence or absence of hypertension and degree of stenosis were not mentioned.

Aetiology of TRAS is multifactorial and includes progressive atheroma into the recipient artery; faulty suture technique; trauma to the renal artery during retrieval, reimplantation or by the perfusion cannula; arterial kinking and turbulent flow; and immunological factors [6]. We and others [9] have shown that stenosis occurs predominantly at the post-anastomotic site in end-to-side transplants and at the anastomosis with end-to-end transplants. Stenosis distal to an end-to-side anastomosis has been attributed, mainly, to turbulent blood flow due to the acute angle between donor and recipient arteries, whilst anastomotic stenosis is thought to result from faulty suture technique.

Until recently the method of diagnosis of TRAS has been conventional aortography with selective renal angiographic views. Where facilities exist, intravenous arteriography by digital vascular imaging using the antecubital veins and femoral veins [5] and intra-arterial digital studies [8] are now being employed with increasing success. It had been thought that the ready availability of digital imaging would allow the criteria for investigation of TRAS to be relaxed, but this has not been borne out in our series, where the mean time from onset of hypertension to detection of TRAS was 2.7 months in the centre using digital subtraction studies, compared with 3.0 months where conventional angiography was used for diagnosis.

In technically successful cases, PTA of TRAS achieved uniform primary blood pressure control. Even after prolonged follow-up, the number of antihypertensive drugs in use to maintain a satisfactory blood pressure was, on average, half the number used prior to angioplasty (1.4 vs 2.7). The rate of technical success (93 %) was better than in previous reports [2, 9, 13], and safety and efficacy were equally good. TRAS may recur, but PTA is repeatable, and in two out of three patients with restenosis a repeat procedure has maintained long-term improvement.

Drug treatment for hypertension does not cure the underlying cause, and progressive arterial stenosis, occlusion and even thrombosis may occur [13]. In addition, poorly controlled hypertension associated with multiple drug therapy may be responsible for considerable morbidity, including the acceleration of cardiovascular disease and the side effects of the drugs. Surgical revascularisation is an alternative and some centres have reported a high degree of operative success with long-term improvement [1, 6]. However, surgery is a major procedure and is associated with a 15 % rate of graft loss and a 5 % mortality [3].

In summary, we have shown that PTA is a safe and effective treatment for post-transplant renal artery stenosis in terms of blood pressure control and preservation of renal function, and often provides lasting benefit.

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