Case Report

Successful surgical revascularization of a kidney transplant after PTA-induced arterial dissection of the allograft renal artery

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Introduction

The most common vascular complication after renal transplantation is allograft renal artery stenosis (ARAS) with a reported incidence up to 5% [1]. ARAS has been shown to be associated not only with reduced long-term graft survival but also with increased morbidity of the recipient by cardiovascular complication [2]. Since 1978, when the first balloon dilatation was successfully performed by Gruentzig et al. [2], percutaneous transluminal angioplasty (PTA) became the treatment of first choice for RAS because of its technical simplicity.

Fig. 1. Before PTA. Intra-arterial angiography of the left A. iliaca showed a very severe (90%) stenosis (arrow) of the transplant renal artery located before its division into three segmental arteries.

Fig. 2. In contrast to Fig. 1 the allograft renal artery is not visible any more on the i.a. angiogram; neither is the intrarenal vasculature. A total occlusion of the renal artery (arrow) was diagnosed.

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Fig. 3. Colour duplex of the renal graft showing the occluded allograft artery with characteristic reverse components without effective flow.

Fig. 4. Colour duplex imaging of the renal graft after revascularization, exhibiting normal intrarenal Doppler profiles.

effectiveness, and acceptance by patients [3–5]. However, PTA is burdened with a considerable probability of restenosis or risk of dissection, resulting in vessel occlusion [3,4]. Therefore PTA of the allograft renal artery has not been generally favoured, considering the low success rate of surgical intervention after arterial dissection with consequent renal ischaemia. In this report the successful surgical revascularization of a renal allograft artery after PTA complicated by arterial dissection and vessel occlusion is described.
A 65-year-old woman had suffered from end-stage renal disease since 1988. The primary diagnosis is unknown, since biopsy was not taken. Chronic intermittent haemodialysis (CIHD) was started in March 1991, followed by successful cadaveric renal transplantation in November 1991. The patient had a good primary function and an eventful postoperative course under immunosuppressive treatment with cyclosporin and methylprednisolone. When she left the hospital at day 20 after transplantation, the patient’s mean serum creatinine was 153 μmol/l and her mean systolic/mean diastolic BP was 130/80 mmHg under an antihypertensive therapy with frusemide and clonidine.

In February 1992 a rejection episode was diagnosed (mild aggressive interstitial rejection, BANFF 1) and after a 6-day course with 250 mg methylprednisolone per day the serum creatinine dropped from a maximum of 279 μmol/l to 189 μmol/l. In the following 24 months the patient had a stable graft function with a serum creatinine between 153 and 171 μmol/l. Arterial hypertension was controlled at that time by frusemide, clonidine, and additionally atenolol.

In November 1993 there was a gradual and persistent increase in serum creatinine to 230 μmol/l, followed in April 1994 by severe arterial hypertension, which could not be controlled by a regimen including five antihypertensive drugs. Colour duplex ultrasound (CDS) revealed a transplant renal artery stenosis [5–7]. Intraarterial angiography showed a very tight (90%) stenosis of the transplant renal artery located before its division into three segmental arteries (Figure 1). The first attempt of PTA failed because of spastic contraction of the transplant renal artery. The following day the PTA was repeated and a total vessel occlusion occurred (Figure 2), demonstrated by CDS immediately post intervention (Figure 3). Surgical intervention was initiated without delay. A Gor-tex prothesis was placed between the left iliac artery (side to end) and the branching point of the three segmental arteries (end to end), total warm ischaemia time was about 4 h and renal perfusion was successfully re-established.

The postoperative course was characterized by acute renal failure, and haemodialysis treatment was necessary for 13 postoperative days. At that time renal allograft perfusion was monitored by CDS (Figure 4). The patient left the hospital 6 weeks later with a stable graft function (serum creatinine 450 μmol/l). Her blood pressure was controlled with an antihypertensive regimen consisting of frusemide, metoprolol, diltiazem, and doxazosin. During the following 16 months the serum creatinine was about 405 μmol/l and the patient did not require haemodialysis. Arterial hypertension was managed using the ACE inhibitor captopril. Serum creatinine was not adversely affected by this treatment.

### Comment

Although there are only rare comparative studies between surgical and endovascular treatment of ARAS so far [10], PTA has become the treatment of choice for FMD, non-ostial stenosis, and stenosis after renal transplantation in many centres [3–5,10]. It has to be pointed out that the success of PTA is limited by occurrence of restenosis and the risk of arterial dissection resulting in vessel occlusion, especially in ostial lesions [2–4,10]; elastic stenosis may be primarily resistant to balloon dilatation. Therefore a stent application in the case of ostial and elastic stenosis is favoured by some authors [8–10]. Once the decision has been made for PTA, surgical treatment should be prepared, reducing warm ischaemia time in case of renal artery dissection. In our case the surgical intervention prevented ESRD and CIHD.

### References


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