Renal allograft failure related to a lower extremity vascular access—a case report

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Introduction

Early renal transplant dysfunction can be caused by acute rejection, acute tubular necrosis (ATN), infection, ciclosporin toxicity, bleeding, urethral obstruction, urinary leak, lymphocele and thrombosis [1]. Prompt treatment of early allograft dysfunction is essential and therefore accurate diagnosis mandatory. We describe a patient with an unusual cause of allograft dysfunction, which was resolved by a simple surgical intervention.

Case report

A 32-year-old man with congenital blindness, hypertension and end-stage renal disease underwent renal transplantation. He had been haemodialysis-dependent since the age of 24 years. Dialysis was performed through an arteriovenous fistula (AVF) between the greater saphenous vein and the superficial femoral artery of his right leg. Access operations on the upper extremity were avoided, because of his congenital blindness and dependency on his hands. The patient had received two renal transplants at the ages of 25 and 27 years, of both which failed due to chronic rejection and urinary tract infection. After the first day, urinary output decreased and serum creatinine levels increased from 480 μmol/l (the lowest value after transplantation) to 770 μmol/l. Ultrasound examination showed haematoma around the kidney. At exploration, a normal perfused kidney with a patent artery and vein with haematoma around the kidney was found. A biopsy was taken, which showed slight arteriosclerosis without any sign of thrombosis or necrosis. The renal function deteriorated further and Mag-3 scintigraphy showed decreasing perfusion. Ultrasound of the kidney showed slight dilatation of the pyelum, for which a percutaneous nephrostomy catheter was inserted, that was without urinary production. A second biopsy of the kidney, one week after transplantation, showed no rejection but interstitial bleeding with acute tubular necrosis, indicating renal vein obstruction. Venography showed a patent venous anastomosis and a pressure of 15 mmHg in both the renal and iliac vein.

Doppler ultrasonography of the AVF estimated a blood flow, through the fistula, of 3.5 l/min. During fistula compression, the flow velocity in the renal vein increased from 41 to 75 cm/sec, indicating improved transplant perfusion. Subsequently, the AVF was surgically closed. Hereafter, Mag-3 scintigraphy showed a marked improvement in perfusion. One week after the fistula closure, urinary output and creatinine clearance started to increase and haemodialysis was discontinued.

Discussion

Deterioration of renal transplant function is usually due to ATN, acute rejection, ciclosporin toxicity and local vascular or ureter complications [2]. Many cadaveric renal grafts suffer various degrees of ATN in the living donor kidney; however, ATN is uncommon because of the reduced ischaemic injury. Ciclosporin toxicity is relatively common; 50% of patients may experience ciclosporin toxicity in the first post-transplant year. Ultrasound and radionuclide
studies are the most commonly used diagnostic studies to evaluate transplant function. Ultrasound findings such as renal enlargement or increased cortical thickness are subjective, and the negative predictive values vary between 17 and 50%. Doppler ultrasonography can provide an overall qualitative impression of renal perfusion. The use of an elevated resistance index (RI) > 0.8 is a non-specific parameter of renal transplant dysfunction. The RI in conjunction with clinical parameters and biochemical findings is a useful guide, but cannot differentiate between ATN and acute rejection [2]. Radionuclide studies can evaluate kidney function because not only perfusion but also excretion rates are measured. The sensitivity for acute rejection is shown to be 87% [3] and for output efficiency, 92% [4]. Nonetheless, it is impossible to differentiate between ATN and acute rejection. Histological specimens remain mandatory for this differentiation.

AVF in the leg is usually not the first choice for vascular access and is used only if all upper extremity sites are exhausted. In this patient, blindness and dependency on the hands forced us to create an AVF in the upper leg. Haemodialysis through this fistula was performed without complications over a period of almost 2 years. One may dispute the decision to implant the renal transplant into the ipsilateral iliac fossa. However, extensive scar tissue after previous transplant operations may seriously affect the dissection of vessels, and this was the major reason for transplantation at the side of the thigh access. In addition, hypoperfusion of a renal transplant in combination with a high-flow elbow AVF has been described [5], thus unrelated to the location of the AVF. If the heart is regarded as a permissive pump that provides the flow the organs ‘ask’ for, then the heart may be unable to meet the demands in patients with excessive access shunting, when a maximum in the cardiac output is reached. The side of transplantation at the ipsi- or contralateral side of the AVF is therefore probably not important.

The decision to close a well-functioning access in a patient with poor allograft function is arbitrary. This particular patient depended on his legs for vascular access, which limits the possible sites for new AVF when necessary. The high venous pressure induced by the high flow through the AVF and the substantially improved renal flow during compression of the fistula made us to decide to close the AVF in an attempt to rescue allograft kidney function.

Conflict of interest statement. None declared.

References

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