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Ischemic Distal Ureteric Obstruction Resulting From Transplant Renal Artery Stenosis: A Case Report

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ABSTRACT

Ureteric stenosis is a known urological complication of kidney transplantation. We report a 40-year-old female patient who was treated for transplant renal artery stenosis (TRAS) with angioplasty and primary stenting. Five months later, she presented with renal dysfunction and moderate hydronephrosis on ultrasound. A tight stenosis of the distal ureter and the ureterovesical anastomosis was documented on an antegrade nephrostogram. It was balloon-dilated and stented, leading to improvement in renal function. This is the first known report that focuses on TRAS as a possible cause of distal ureteric ischemia resulting in stenosis.

KEYWORDS: Distal ureter; Ureteric stenosis; Transplant renal artery stenosis; Ischemia; Pathogenesis

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Abbreviations and Acronyms
ACR, acute cellular rejection
DGF, delayed graft function
TRAS, transplant renal artery stenosis

INTRODUCTION

Ureteric stenosis is the most common urological complication of renal transplantation, with a prevalence ranging from 2% to 10%. The stenosis affects the distal ureter and ureterovesical anastomosis in 80% of cases [1]. Ischemia is the most common cause of distal ureteral stricture formation. It can result from any pathology that reduces blood supply to the allograft ureter, which is dependent on the transplant renal artery as its sole source of blood [2,3].

The distal ureter may be rendered ischemic by: (1) traction, stripping, or diathermy during surgery, (2) edema associated with acute rejection, or (3) reduced inflow in kidneys with diseased arteries that are recovered from elderly donors [2,4,5]. Renal dysfunction that is observed in cases of transplant renal artery stenosis (TRAS) is the direct result of significant allograft hypoperfusion that is also likely to compromise the blood supply to the distal ureter [6]. We present a case of distal ureteric stenosis that developed 5 months after the diagnosis and treatment of symptomatic TRAS. We also discuss the possible role of TRAS in the etiology of ureteric stenosis.

CASE REPORT

The patient is a 40-year-old female who was on regular hemodialysis for 2 years because of end-stage renal disease secondary to systemic lupus erythematosis and hypertension. She received a standard-criteria deceased-donor allograft. The ureter was recovered with substantial surrounding tissue, which is typical from a deceased-donor procedure. The single donor artery with its aortic patch was anastomosed end-to-side to the external iliac artery using a running 6/0 nylon suture. The tunneled extravesical ureteroneocystostomy was stented. She received depleting antibody induction. Immediate graft function was noted and she was discharged home following an uneventful postoperative period. Her serum creatinine was 112µmol/L. Maintenance immunosuppression was comprised of tacrolimus, mycophenolate mofetil, and prednisolone.

Five weeks later, the patient was treated for an episode of acute cellular rejection (ACR) (Banff grade IA) that responded to pulse steroids. Serum creatinine settled at 125µmol/L. A month later, she presented with worsening hypertension and an increase in serum creatinine to 205µmol/L. TRAS was

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case report

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Figure 1. Angiogram demonstrating a tight stenosis in the transplant renal artery (arrow).

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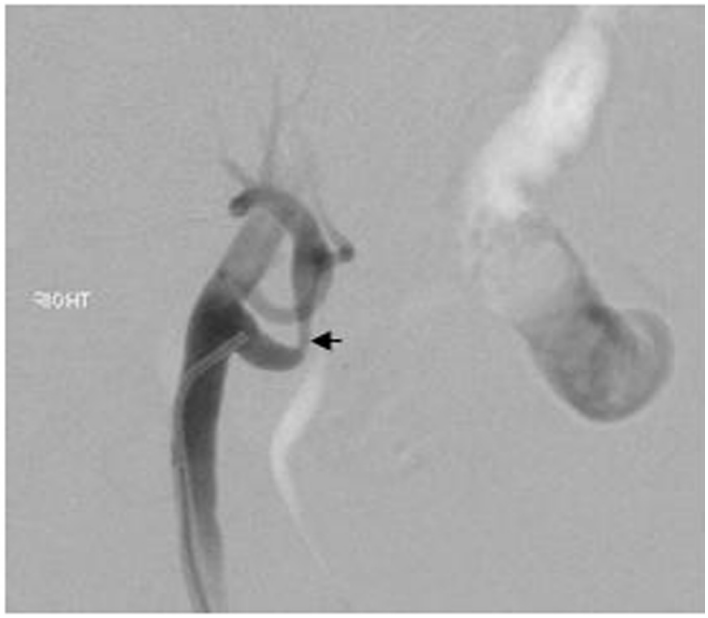

Although 1 of its branches may give the impression of 2 arteries, this is a single artery.

Figure 3. Antegrade nephrostogram confirming a tight stenosis of the distal ureter (arrow).

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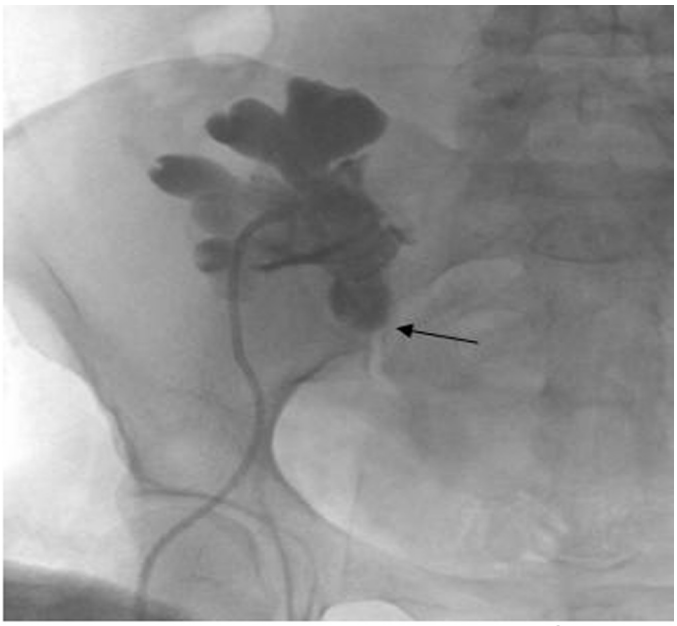

It appears that the stricture is proximal because of distention; however, after a week of decompression, this is a single distal stricture that required dilatation over the long segment.

suggested by ultrasound and confirmed on angiography (Figure 1). The site of renal artery stenosis was 1 cm away from the site of anastomosis. The stenosis was treated with endovascular stenting, which led to improvement in hypertension and a return to baseline serum creatinine.

Figure 2. Ultrasound of transplant kidney showing moderate hydronephrosis (white arrows).

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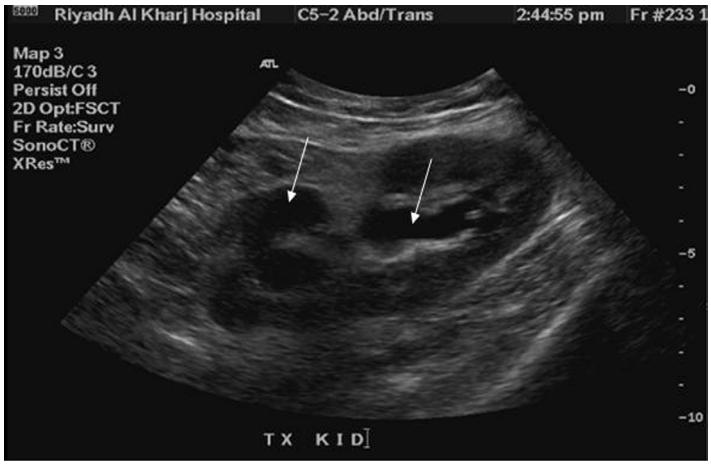

The patient remained stable until 5 months later, when the serum creatinine increased to 340µmol/L and ultrasound confirmed moderate hydronephrosis (Figure 2). She underwent percutaneous nephrostomy with improvement in serum creatinine. A tight stenosis was documented on the antegrade nephrostogram (Figure 3) and screening for BK virus was negative. The stenosis was dilated with a 6 mm x 2 cm balloon dilator, and a 16 cm x 6 mm double-J stent was placed across the stenosis (Figure 4a; Figure 4b). The serum creatinine continued to improve, with a gradual return to baseline renal parameters. The stent was removed after 8 weeks. Serum creatinine remained stable and was 120µmol/L at 12 months.

DISCUSSION

Despite improvements in kidney recovery and ureteric implantation, ureteric stenosis continues to be the most common urological complication after kidney transplantation [1]. The etiology of this complication is variable, but the common underlying pathophysiology is ischemia and hypoperfusion. Possible causes are: (1) damage to the ureteric branch of the renal artery during surgery [4]; (2) diseased arteries in elderly donors that reduce inflow [2]; (3) edema from acute cellular rejection [5]; or (4) delayed graft function (DGF) [2]. These possible causes are also indirectly supported by the fact that

Figure 4a. Balloon dilatation of the distal ureteric stenosis (arrow).
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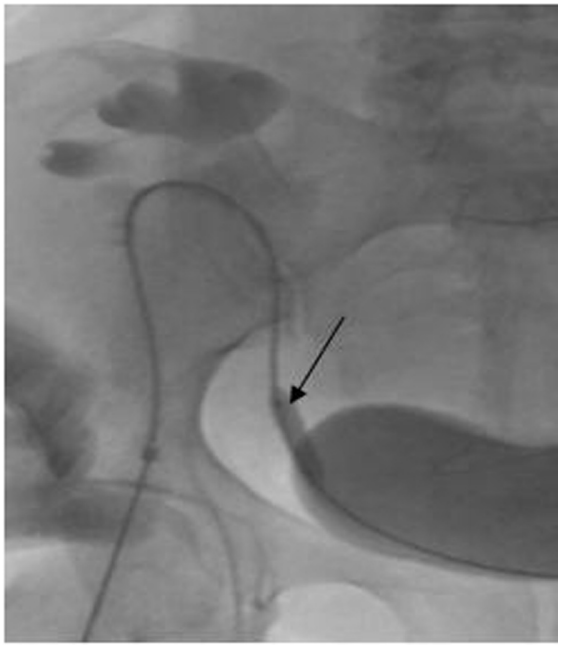

Figure 4b. Double-J stent in place after balloon dilatation (arrow).
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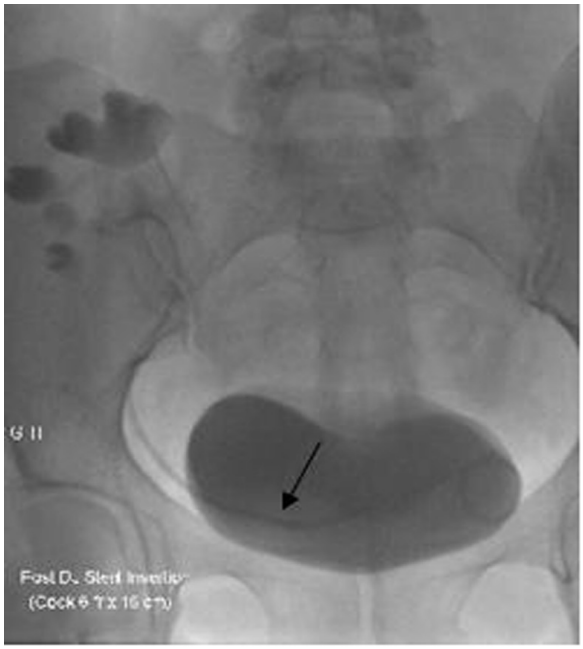

donor age, DGF, and more than 2 allograft arteries all increase the risk of stenosis [2]. A deceased donor kidney is less likely to have injury to the uretral blood supply than live donors because the dissection in deceased donor recovery is performed well away from the ureter and is further protected by the accompanying fat and gonadal vessels. However, deceased donor allografts are subject to longer cold ischemia and DGF, both of which have also been shown to cause TRAS [7].

Our recipient developed TRAS after an episode of ACR that responded to steroid pulse therapy. It would be reasonable to assume that TRAS was responsible for a longer period of ischemia and hypoperfusion in our recipient than was the episode of ACR because the rejection was diagnosed promptly and treated. TRAS, on the other hand, is known to become symptomatic only after at least 50% of the artery is occluded [8]. However, throughout its asymptomatic period, the ureter receives reduced blood supply that is proportional to the degree of arterial stenosis. Because of timely treatment of ACR in our recipient, the ischemia that may have been caused by edema was short-lived; indeed, it was much shorter than the ischemia of TRAS. On the other hand, neither is it unequivocally established that ACR causes ureteric stenosis [2,5]. Even though there is consensus that ischemia is the underlying pathophysiology, it is puzzling that a condition that is well known to reduce inflow to the kidney and, in turn, the ureter has not been considered in its etiology.

We present a case of ureteric stenosis that we believe developed 5 months after our patient was diagnosed with TRAS that required endovascular stenting. Surprisingly, ischemia and hypoperfusion of TRAS that is significant enough to cause graft dysfunction and even graft loss has not yet been considered a possible cause of ureteric ischemia resulting in stenosis. We hope that this report will provide reason for researchers to consider ischemia resulting from TRAS in the etiology of ureteric stenosis.

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