Case Report of a Transplant Renal Artery Stenosis: Delayed Diagnosis, Correct Treatment

Transplant Renal Arter Stenozlu Olguda Gecikmiş Tanı, Doğru Tedavi

ABSTRACT

Renal artery stenosis is observed in 1-23% of the transplanted kidneys. It may result in graft dysfunction and hypertension but anuria is rare in patients with graft renal artery stenosis. We report a case of proximal transplant renal artery stenosis (TRAS) in a renal transplant recipient presenting with anuria and hemodialysis requirement in the fourth month of transplantation who completely recovered after renal artery revascularization.

KEY WORDS: Renal artery stenosis, Renal artery revascularization, Renal transplantation

ÖZ

Renal transplant hastalarında transplant renal arter darlığı %1-23 oranında gözlenmektedir. Klinikte greft disfonksiyonu ve hipertansiyon sık gözlenmekle birlikte anüri nadir olsa da görülebilmektedir. Olgumuzda renal transplantasyonunun dördüncü haftasında hastanın kliniğinde anüri ve hemodiyaliz ihtiyacı mevcuttu. Transplant böbrekte proksimal renal arter stenozu tesbit edilen hastanın renal arter revaskülarizasyonu sonrası diyaliz ihtiyacı kalmayıp renal fonksiyonları tamamen düzeldi.

ANAHTAR SÖZCÜKLER: Renal arter darlığı, Renal arter revaskülarizasyon, Renal transplantasyon

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INTRODUCTION

Renal artery stenosis is observed in 1-23% of the transplanted kidneys (1). It may result in graft dysfunction and hypertension but anuria is rare in patients with graft renal artery stenosis. We report a case of proximal transplant renal artery stenosis (TRAS) in a renal transplant recipient presenting with anuria and hemodialysis requirement in the fourth month of transplantation who completely recovered after renal artery revascularization.

CASE REPORT

A 68-year-old male patient with chronic kidney disease secondary to hypertensive nephrosclerosis had undergone cadaveric renal transplantation. He had been discharged on the postoperative 12th day with a serum creatinine of 1.1 mg/dL under treatment with prednisolone, tacrolimus and mycophenolate mofetil. He had admitted

to another hospital with decreased urine volume and a serum creatinine of 3.3 mg/ dL at the fifteenth week. His blood pressure had been measured as 130/80 mm Hg on admission. No significant abnormality had been detected on Doppler ultrasonography. He had been treated with pulse steroids, anti-thymocyte globulin, plasmapheresis and intravenous immunoglobulin with a presumed diagnosis of acute rejection. However anti-rejection treatment had been terminated when renal biopsy had revealed acute tubular necrosis without evidence of rejection. He had been started hemodialysis treatment due to increased creatinine levels and anuria at the 15th day of hospitalization. He was admitted to our clinic at the 20th week of transplantation. On admission, his blood pressure was 160/90 mmHg and he had ascites and severe peripheral edema. There was no graft tenderness or murmur on the graft. His daily urine volume was



Received: 18.01.2014 Accepted: 09.05.2014

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Phone : +90 312 596 69 26 E-mail : ahaspulat@hotmail.com 100 mL. He had severe lower urinary tract infection and was under regular hemodialysis treatment thrice weekly. Viral loads for CMV and BK were both negative and the blood level for tacrolimus was 7 ng/ml. A repeat renal Doppler examination revealed a probable stenosis in the graft renal artery that was confirmed with computerized tomographic angiography as stenosis >90% in the proximal renal artery. There was an accompanying post stenotic dilatation (Figure 1). He underwent a successful percutaneous transluminal angioplasty (PTCA) and



Figure 1: Computerized tomographic angiography imaging revealing >90% stenosis in the proximal renal artery and post stenotic dilatation.

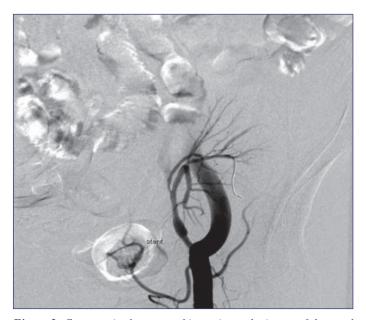


Figure 2: Computerized tomographic angiography image of the renal artery after successful angioplasty and stenting.

stenting (Figure 2) that resulted in increased urine volume in the second day after intervention. He did not require hemodialysis treatment anymore and was discharged from the hospital with a serum creatinine of 1.13 mg/dL and normal blood pressure eight days after the intervention. His discharge medications were the same immunosuppressive medications with clopidogrel as additional antiaggregant treatment.

DISCUSSION

Rejection, calcineurin toxicity, infection, post renal obstruction and renal artery stenosis are the possible causes of early graft dysfunction in renal transplant recipients. There was no evidence of rejection in graft biopsy, the blood level for tacrolimus was in the target range and there was no hydronephrosis on ultrasonography in our patient. Although urinary infection was present at the time of admission, appropriate antibiotic therapy did not result in improvement of renal functions. Despite the normal Doppler ultrasonography that had been performed in another hospital, we repeated the Doppler ultrasonography as there was no explanation for graft dysfunction and diagnosed a severe TRAS.

Renal artery stenosis is observed in 1-23% of the transplanted kidneys (1). Clinical symptoms including hypertension and graft dysfunction usually occur when greater than 50 to 80% of the renal artery is occluded (2). Chronic rejection, long renal artery, arterial kinking and atherosclerosis have been implicated in the etiology of stenosis in these patients (3). It is generally observed between post-operative 3 months and 2 years (4). Proximal TRAS, as seen in this patient, is a rare clinical entity and is usually secondary to atherosclerosis (5,6). The chronic atherosclerotic process in the elderly explains the higher prevalence of proximal TRAS in these patients (7). PTCA and stenting is the preferred treatment option in TRAS (8-10).

Although rarely observed, TRAS should be considered in renal transplant recipients presenting with hypertension and impaired graft functions. As the average age of renal transplant recipients' increase, proximal TRAS will also increase proportionally.

CONCLUSION

This case demonstrates that a single normal Doppler examination may not be enough to exclude renal artery stenosis if the clinical suspicion is high. Although diagnosis was delayed, PTCA and stenting resulted in significant improvement in graft functions.

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