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Transplant renal artery stenosis without hypertension: A case report

Fereshteh Saddadi^{1*}, Seyyed Mehdi Hoseini¹, Faezeh Rasolian¹,
Mohammad Hassan Fallahkohan²

¹Iran University of Medical Sciences, Tehran, Iran

²Shahid Beheshti University of Medical Sciences, Tehran, Iran

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ABSTRACT

In kidney transplant recipients, the renal artery stenosis is cause of graft dysfunction and graft loss. Here we describe a case that leads to graft loss. The nephrologist should know about its etiologies, symptoms and diagnostic tools and also its treatment after renal transplantation.

Keywords: Transplant renal arterial stenosis, Kidney transplant, Renal artery stenosis, Graft loss, Acute renal artery thrombosis, End-stage kidney failure

Implication for health policy/practice/research/medical education:

Acute renal artery thrombosis is a complication of renal transplantation can result in graft loss.

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Introduction

In kidney transplant recipients, stenosis of the renal artery is cause of graft dysfunction and graft loss. The prevalence is, ranging from 1% to 23%. The cause of transplant renal artery stenosis (TRAS) is multifactorial and includes surgical technique, type of allograft, immunologic factors, and CMV infection. TRAS is more prevalent in cadaveric allografts with long-cold ischemia time. Here we described a case that led to graft loss.

Case Presentation

A 55-year-old man underwent renal transplantation of deceased donor for end-stage kidney failure due to autosomal dominant polycystic kidney disease. The patient had zero panel reactive antibodies. He received a kidney from a 24-year-old deceased donor who died due to a car accident 30 days earlier. During transplantation, renal reperfusion was normal and delayed graft function. He had uncomplicated postoperative course. The immunosuppression regimen included anti-thymocyte immunoglobulin as induction therapy, was followed by tacrolimus, mycophenolate mofetil and corticosteroids

as maintenance therapy. He presented to the emergency department with hematuria, graft pain and increased serum creatinine levels. Physical examination revealed normal blood pressure, no pulmonary rales and had abdominal tenderness on graft side. Laboratory tests were considerable for a plasma creatinine, which elevated from a post-transplant baseline of 1.1 to 5.2 mg/dL over 2 days. The tacrolimus trough level was normal at 10 ng/mL (target 8–15 ng/mL). Urine and blood cultures were negative. Chest X-ray depicted normal. The patient was admitted to the transplantation ward and Duplex sonography was performed and was suggestive of renal artery thrombosis with abnormal heterogeneous mass near to vascular renal anastomosis with a pressure effect on main renal artery. On Doppler no waveform pattern was detected in intrarenal artery, and the main renal artery was narrow without significance focal stenosis (Figures 1A and 1B).

Angiography was not conducted since the patient underwent emergency surgical intervention after 2 hours of diagnosis. The graft was necrotic and the renal artery was thrombosed. Unfortunately, nephrectomy was done

*Corresponding author: Fereshteh Saddadi,
Email: iumssaddadi@gmail.com

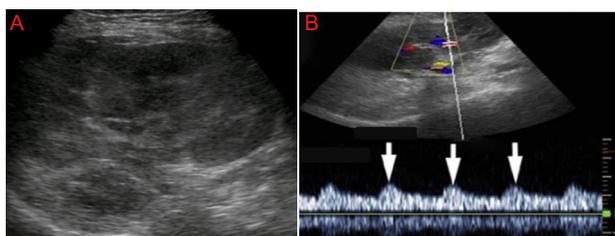


Figure 1. (A) Ultrasound of transplanted kidney- enlarged hypochoic renal transplant heterogenous mass near to vascular renal anastomosis. (B) Segmental renal artery shows delay in the systolic upstroke and rounding of the systolic peak, consistent with a tardus-parvus waveform.

and the patient returned to hemodialysis. Morphologic lesions of transplanted kidney were extensive parenchymal infarction with transplanted kidney rejection (Figure 2).

Discussion

TRAS can occur after renal transplantation (1), and it should be considered at any time after transplantation but usually occurs between 3 months and 2 years after renal transplantation (2). Individuals with TRAS typically may present with fluid retention, graft dysfunction without evidence of rejection or hypertension (3). In our case, 30 days post-transplantation the patient presented with hematuria, graft pain and renal dysfunction without hypertension. The most important risk factors for early TRAS are classified as surgically related factors, recipient and donor related causes. Surgical-related complications (such as anastomotic stenosis) are the main etiology (4) and in this presented case the process of surgery was without complication. Extrinsic mechanical compression due to patient's enlarged native polycystic kidney may occur (5). Cold and warm ischemic time is one of the significant causes of TRAS and it depends on the post-procedures of harvesting the kidney most commonly in deceased donor graft transplantations. Prolonged cold ischemia is a reason for vascular damage (6). Our patient's graft warm and cold ischemic time was taken longer than 4 hours. Among the post-surgical complications, ischemic reperfusion injuries, early acute rejection and delayed graft functions can cause TRAS. Our patient did not have these complications although we could not clinically rule out ischemic reperfusion injuries. The character of immune injuries or calcineurin-induced angiopathy in the development of TRAS is uncertain (7). In addition, one of the etiologies of late graft stenosis is immune injuries and inflammations after acute cellular rejections (8). Recipient related etiologies are old age and peripheral vascular disease, such as atherosclerosis are common findings (4), and the other causes are endothelial damage in end-stage kidney failure patients due to chronic inflammations and homocysteinemia. Cytomegalovirus virus (CMV)

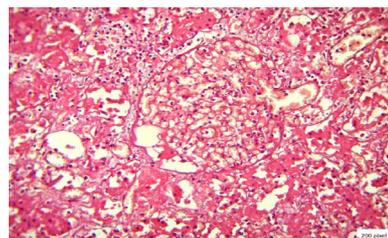


Figure 2. Extensive parenchymal infarction with transplanted kidney rejection and reactive acute inflammation (H&E×200).

infection is a predisposing factor for TRAS due to its mitogenic effects of viral cytomegalic gene products (9). Our patient's viral markers were negative since the donor had negative viral marker too. Donor related causes are deceased donor, old age, hypo-perfusion of brain death donor. Old age of recipient and donor are a risk factor for TRAS. In this case, the recipient was middle age and the donor had 24 years old; therefore, the donor age could not be the major risk factor.

Conclusion

Acute renal artery thrombosis is a complication of renal transplantation, which can result in graft loss. The nephrologist should know about its etiologies, symptoms and diagnostic tools and also its treatment after renal transplantation.

Authors' contribution

FS, SMH and FR designed the study. MHF wrote the manuscript. All authors approved the final version of the manuscript.

Conflicts of interest

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors. The patients gave his consent to publish as a case report.

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References

1. Patel NH, Jindal RM, Wilkin T, Rose S, Johnson MS, Shah H, et al. Renal arterial stenosis in renal allografts: retrospective study of predisposing factors and outcome after percutaneous transluminal angioplasty. *Radiology*. 2001;219:663-7. doi: 10.1148/radiology.219.3.r01jn30663.
2. Chen W, Kayler LK, Zand MS, Muttana R, Chernyak V, DeBoccardo GO. Transplant renal artery stenosis: clinical manifestations, diagnosis and therapy. *Clin Kidney J*.

- 2015;8:71-8. doi: 10.1093/ckj/sfu132.
3. Fervenza FC, Lafayette RA, Alfrey EJ, Petersen J. Renal artery stenosis in kidney transplants. *Am J Kidney Dis.* 1998;31:142-8. doi: 10.1053/ajkd.1998.v31.pm9428466.
 4. Bruno S, Remuzzi G, Ruggenti P. Transplant renal artery stenosis. *J Am Soc Nephrol.* 2004;15:134-41. doi: 10.1097/01.asn.0000099379.61001.f8.
 5. Lee L, Gunaratnam L, Sener A. Transplant renal artery stenosis secondary to mechanical compression from polycystic kidney disease: a case report. *Can Urol Assoc J.* 2013;7:E251-3. doi: 10.5489/cuaj.548.
 6. Rengel M, Gomes-Da-Silva G, Incháustegui L, Lampreave JL, Robledo R, Echenagusia A, et al. Renal artery stenosis after kidney transplantation: diagnostic and therapeutic approach. *Kidney Int Suppl.* 1998;68:S99-106. doi: 10.1038/sj.ki.4490573.
 7. Agroyannis B, Mourikis D, Tzanatos H, Chatzioannou A, Vlahakos DV, Vlachos L. Transplant renal artery stenosis: hypertension and graft function before and after angioplasty. *J Hum Hypertens.* 2001;15:741-3. doi: 10.1038/sj.jhh.1001256.
 8. Fauchald P, Vatne K, Paulsen D, Brodahl U, Södal G, Holdaas H, et al. Long-term clinical results of percutaneous transluminal angioplasty in transplant renal artery stenosis. *Nephrol Dial Transplant.* 1992;7:256-9. doi: 10.1093/oxfordjournals.ndt.a092116.
 9. Humar A, Uknis M, Papalois V, Gillingham K, Matas A. Is there an association between cytomegalovirus and renal artery stenosis in kidney transplant recipients? Abstract#1052. *Transplantation.* 2000;69:S386

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