



Steal Syndrome in a Patient with Heart and Kidney Transplantation Caused by Arterio-Venous Fistula

Lugo-Baruqui Jose A^{1,2}, Burke George W^{1,2}, Guerra Giselle^{1,3}, Salsamendi Jason⁴ and Ciancio Gaetano^{1,2*}

¹The Lillian Jean Kaplan Renal Transplant Center of the Division of Transplantation, Miami, USA

²Department of Surgery, University of Miami Miller School of Medicine and Miami Transplant Institute at the Jackson Memorial Hospital, Miami, USA

³Department of Medicine, Division of Nephrology, University of Miami Miller School of Medicine and Miami Transplant Institute at the Jackson Memorial Hospital, Miami, USA

⁴Department of Radiology, Division of Interventional Radiology, University of Miami Miller School of Medicine and Miami Transplant Institute at the Jackson Memorial Hospital, Miami, USA

***Corresponding author:** Gaetano Ciancio, M.D., Department of Surgery, Division of Transplantation, University of Miami Miller School of Medicine, USA, P.O. Box 012440 (R-440), Miami, FL 33101, USA, Tel: 305-355-5111, Fax: 305-355-5134, E-mail: gciuncio@med.miami.edu.

Abstract

Steal syndrome is an infrequent complication of arteriovenous fistulas (AV) graft for patients on hemodialysis. We report a case of a patient with history of orthotopic heart transplant with end-stage renal disease that subsequently underwent renal transplant from a living donor that developed delayed graft function due to steal syndrome of a thigh AV fistula. We discuss the presentation, diagnosis and treatment.

Keywords

Kidney transplant, Steal syndrome, Arterio-venous fistula

Abbreviations

AV: Arterio-venous, ESRD: End stage renal disease, GFR: Glomerular-filtration rate

Introduction

Steal phenomena or steal syndrome is an infrequent complication of arteriovenous fistulas (AV) for patients on hemodialysis. This represents a vascular insufficiency distal to the site of AV fistula creation. We present a case of a patient who presented with delayed graft function after kidney transplant and steal syndrome was diagnosed after extensive work up and high suspicion of diagnosis.

Case Report

This is a 23 year old Hispanic female patient with history of orthotopic heart transplant in 2003 secondary to right ventricular dysplasia. She presented acute rejection of the heart graft in 2010 requiring transient use of extra-corporeal membrane oxygenation

(ECMO). The rejection was appropriately treated and heart graft recovered appropriate function. Nevertheless, as a consequence of the ischemic insult, the patient developed end-stage renal disease (ESRD) requiring renal replacement therapy with intermittent hemodialysis.

The patient presented at our Institution for evaluation of kidney transplant from a related living donor (sister). During pre-transplant workup, recipient was cleared from cardiologist point of view with a normal left ventricular size and function. She had history of multiple vascular accesses in upper extremities and previous endovascular procedures in the left femoral vessels for cardiac catheterizations. At time of evaluation, the patient had a right polytetrafluoroethylene (PTFE) femoral arterio-venous (AV) graft for vascular access. The graft consisted on a 7-Artegraft® (Artegraft Inc. North Brunswick, NJ) loop AV graft using the right common femoral artery to right common femoral vein. The graft was placed in 2012 and since the fistula creation the patient denied any history leg pain, intermittent claudication or other ipsilateral vascular symptoms. There was no history of flow problems during dialysis sessions. We performed a hand-assisted laparoscopic left donor nephrectomy with normal anatomy of the renal graft. The kidney was prepared in the back table and transplanted in the right iliac fossa of the recipient. Standard technique was used anastomosing renal vessels to iliac artery and vein and the ureter reconstructed with an extravesicular ureterocystostomy. After reperfusion, the renal had good color and turgor, no evidence of outflow obstruction. Immunosuppression as per Institution protocol with induction using thymoglobulin (3 mg/kg divided in 3 doses), and basiliximab (two doses of 20 mg day 0 and 4) with steroids. For maintenance immunosuppression she was started on tacrolimus (1 mg/kg, trough level 6-8 ng/dL), mycophenolate sodium (720 mg twice a day) and low dose steroids

Citation: Lugo-Baruqui JA, Burke GW, Giselle G, Jason S, Gaetano C (2015) Steal Syndrome in a Patient with Heart and Kidney Transplantation Caused by Arterio-Venous Fistula. Clin Med Rev Case Rep 2:065. doi.org/10.23937/2378-3656/1410065

Received: August 29, 2015: **Accepted:** October 26, 2015: **Published:** October 28, 2015

Copyright: ©2015 Lugo-Baruqui JA. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.



Figure 1: Renal Angiogram

Right transplant renal artery and right external iliac CO_2 angiogram demonstrated a normal expected take off for a transplant renal artery anastomosis without evidence of tortuosity, kinking, or stenosis.

Table 1: Arteriography with arterial pressure measurements (mmHg). Marked increase in BP in all measurements after external temporal occlusion of the AV-fistula.

	Without Graft Occlusion (mmHg)	Graft Occlusion (mmHg)
Aorta	100/50 (69)	108/60 (79)
Common Iliac	97/49 (65)	107/59 (77)
Distal Common Iliac	99/47 (64)	108/59 (77)
Perianastomotic	94/44 (62)	105/58 (76)
Mid External Iliac	75/40 (53)	108/60 (76)
Distal External Iliac	83/42 (56)	110/50 (77)
Right Femoral Artery	78/39 (54)	103/58 (74)
Graft	68/37 (48)	108/50 (76)

(5 mg daily). Patient was transferred to intensive care unit for monitoring cardiac and renal function more closely.

After the first 24 hours the patient remained oliguric without a significant change in serum creatinine from pre transplant value. Renal graft ultrasound reported patent vessels with normal resistive indexes. Technetium 99 m-diethylenetriamine penta acetic acid nuclear renogram showed signs of acute tubular necrosis and good flow. Repeated echocardiogram showed preserved cardiac ejection fraction. The patient was managed cautiously with intravenous fluid for volume expansion with special consideration for prevention of lung edema. She was transferred to the service ward three days later with diagnosis of slow graft function.

Over the course of the hospitalization, the oliguric state did not improve even with use of intravenous diuretics. Her serum creatinine persisted elevated with discrete decrease from 7.7 mg/dL to 4.5 mg/dL after one week. Ejection fraction remained stable in serial cardiac echocardiograms without signs of ventricular dilatation. Repeated nuclear renal scan reported persistent acute tubular necrosis. Two weeks after transplant, patient presented dyspnea requiring ultrafiltration. She was discharged with intermittent hemodialysis with diagnosis of delayed graft function.

Three weeks after discharge the patient presented to the emergency department with signs of urinary tract infection. Cultures were ordered and patient was admitted for empiric antibiotic treatment. We performed a kidney biopsy at this point, which reported no evidence of acute T cell mediated rejection, moderate glomerulosclerosis

(10/15), moderate interstitial fibrosis and tubular atrophy (26 – 50% of cortical area), and chronic allograft arteriolopathy and arteriopathy. Repeated renal transplant Doppler ultrasound showed patent vessels, with normal resistive indexes with suspicion for renal artery stenosis. A CO_2 renal arteriogram was performed, which raised concern for a steal syndrome from the ipsilateral thigh AV fistula. There was no evidence of stenosis in arterial anastomosis or tortuosity of renal artery (Figure 1). Pressure arteriogram with sensor probe of the renal artery demonstrated an increase in perfusion pressure of the renal graft after compression of the arterio-venous fistula in the ipsilateral femoral artery (Table 1). Decision was made to thrombose the AV graft via endovascular approach to preserve graft perfusion pressure by interventional radiology. The urinary volumes and serum creatinine improved after the AV fistula thrombosis. The patient was discharged three days later free of hemodialysis with a serum creatinine of 1.4 mg/dL. After 18 months post-transplant patient remains with stable renal and cardiac function.

Discussion

Patients with end-stage kidney disease on hemodialysis have different vascular access alternatives from tunneled catheters to creation of arterio-venous (AV) fistulas. Prosthetic AV grafts are indicated in patients with failed AV fistulas (AVF) or difficult vascular access due to previous line insertion and thrombosis [1]. Steal phenomena or steal syndrome is essentially a vascular insufficiency that has been described in the vascular literature since 1969 [2]. This condition presents symptomatic extremity ischemia caused by the diversion of arterial flow through the access site. Literature reports this presentation in approximately 5 - 10% of the cases. The hemodynamic changes that are believed to cause symptoms in these patients consist in arterial stenosis, high flow in the fistula, and lack of compensation perfusion by collateral flow [3]. Diagnosis can be made by clinical suspicion, and confirmed with doppler ultrasound or arteriogram [4]. Treatment options vary from observation if the symptoms are mild, to surgical fistula ligation, percutaneous angioplasty, and distal revascularization with interval ligation of the fistula. Surgical treatment for ischemic steal syndrome is successful for ischemia resolution in up to 95% of patients [5].

Use of grafts for AVF creates a high flow state with high velocities and turbulent flow in distal vessels. Venous stenosis and proximal hypertension in the site of anastomosis can present in up to 38% of cases after graft placement [6]. This condition can compromise the distal flow and perfusion. In the condition of a femoral AVF, proximal venous hypertension of kidney grafts connected to iliac vessels, can potentiate an artificial vascular insufficiency [7]. Steal syndrome has been previously described in patients with kidney transplant. The classic report of Kuwertz-Bröking suggested that steal syndrome can present after kidney transplantation and affect the function on the graft, independently of the location of the AV graft in respect to kidney transplant [8]. Ligation of the thigh AV fistula usually results in rapid improvement of renal function after the period of chronic hypoperfusion of the graft. Gourlay reported the experience of three renal transplant patients with ipsilateral femoral loop vascular grafts with normal renal function without ligation of the graft. This results suggest that ligation of AV grafts is not an absolute requirement for ipsilateral kidney transplant [9].

In the case presented, the decision for selection of the site for revascularization of the kidney graft was based on history of multiple catheterizations of left iliac vessels for cardiac procedures. In the setting of oliguria and slow creatinine clearance after kidney transplant, with history of heart transplant, our main concern was hypoperfusion of renal graft secondary to heart failure. Cardiac studies showed preserved cardiac function which turned our attention to alternative causes of acute tubular necrosis such as ischemia-reperfusion injury, early acute rejection, or calcineurin induced nephrotoxicity (CNI). Immunological cross-match for living donor was ideal with 5/6 HLA matches. Panel reactive antibody (PRA) was 0% for class I and class II antibodies and no donor specific antibodies (DSA) were detected. Patient was initiated on tacrolimus on day 2 after transplant because of history of previous heart transplant.

This experience shows that renal graft revascularization can be done disregarding the site of previous thigh fistula. There is no correlation of the site of the transplant and fistula in function of renal clearance. Interestingly, the location of the thigh AV fistula and renal function does not correlate with the site of the transplanted kidney [6]. Therefore, it may not be possible to determine which renal grafts will present compromised function due to steal syndrome from a femoral fistula before implantation.

Conclusions

The best strategy for avoidance of graft damage secondary to hypoperfusion by a steal syndrome consist in clinical suspicion and close follow up by diagnostic image techniques such as Doppler ultrasound. In case of any hypoperfusion suspicion, the fistula needs to be revised and possibly occluded by surgical means to improve graft perfusion.

References

1. Akoh JA (2009) Prosthetic arteriovenous grafts for hemodialysis. *J Vasc Access* 10:137-147.
2. Storey BG, George CR, Stewart JH, Tiller DJ, May J, et al. (1969) Embolic and ischemic complications after anastomosis of radial artery to cephalic vein. *Surgery* 66: 325-327.
3. Malik J, Tuka V, Kasalova Z, Chytlova E, Slavikova M, et al. (2008) Understanding the dialysis access steal syndrome. A review of the etiologies, diagnosis, prevention and treatment strategies. *J Vasc Access* 9: 155-66.
4. Chaudhri Y, Horrow M, Zaki R (2006) Vascular Steal of Renal Transplant Sonographic Diagnosis, *J Ultrasound Med* 25: 939-941.
5. Suding PN, Wilson SE (2007) Strategies for management of ischemic steal syndrome. *Semin Vasc Surg* 20:184-188.
6. Choudhury D, Lee J, Elivera HS, Ball D, Roberts AB, et al. (1995) Correlation of venography, venous pressure and hemoaccess function. *Am J Kidney Dis* 25: 269.
7. Shukla PA, Contractor S, Huang JT, Curi MA (2012) Coil embolization as a treatment alternative for dialysis-associated steal syndrome. *Vasc Endovascular Surg* 46: 546-549.
8. Kuwertz-Bröking E, Bulla M, Langer K, Frosch M, Lison AE, et al. (1988-1989) Steal syndrome after kidney transplantation caused by A-V fistula at the thigh. *Child Nephrol Urol* 9:163-168.
9. Gourlay WA, Legiehn G, Manson DC, Landsberg DN (1999) Successful renal transplantation with ipsilateral femoral arteriovenous grafts. *Transplantation* 67: 493-495.