Salvage of renal allograft using venous thrombectomy in the setting of iliofemoral venous thrombosis

Samual P. Sterrett, David Mercer, Jason Johanning and Jean F. Botha

Introduction

Acute renal allograft dysfunction in the setting of iliofemoral thrombosis following renal transplant is extremely rare and highly morbid, resulting in graft rupture or even death. We report a case of successful iliofemoral venous thrombectomy for renal allograft salvage in the setting of severe acute renal dysfunction.

Case

A 49-year-old Caucasian female underwent a second kidney–pancreas transplant in May 2002 for end-stage diabetic nephropathy. Her postoperative course was complicated by intra-abdominal sepsis and the development of bilateral deep venous thrombosis (DVT). The patient was treated conservatively, with a 6-month course of oral anticoagulation.

One month after discontinuation of oral anticoagulation, the patient noted a 7-day history of a painful and swollen left lower extremity. Three days prior to admission she noted paresthesia, pain and pallor of her left lower extremity. The next day she noticed left lower quadrant abdominal pain over her renal allograft associated with anuria. On the day of admission, initial creatinine at her referring physician's office was 1.7 mg/dl. Seven hours later, her creatinine had climbed to 4.7 mg/dl with persistent anuria.

On examination, her left leg was oedematous and swollen. Her renal allograft was tender to palpation in the left lower quadrant. Duplex examination revealed venous thrombosis arising in the post-tibial veins and extending to the iliac veins, an enlarged renal allograft (>1 cm in increase in length compared with previous ultrasound) and a patent renal artery with absence of diastolic flow consistent with venous outflow obstruction. The patient was systemically anticoagulated with unfractionated heparin. A decision was made to attempt graft salvage by performing a venous thrombectomy.

Via local cut down to the femoral vein, venography of the vena cava and the left common iliac veins was completed using <30 cc of half strength Visipaque contrast. This confirmed the extension of thrombus distal to the renal allograft venovenostomy and the absence of flow within the left iliac vein. A standard venous thrombectomy was performed via a common femoral vein venotomy. A Fogarty thromboembolectomy catheter (Edwards Lifesciences, Irvine, CA) and Esmarch compression were used proximally and distally, respectively, for thrombus removal. Repeat venography (30 cc of half strength Visipaque) demonstrated a widely patent left iliac vein with dye washout at the level of the renal allograft venostomy. Repeat duplex of the renal allograft on postoperative day 1 demonstrated normal arterial and venous flow to the transplanted kidney with appropriate diastolic flow. Her creatinine steadily declined to baseline (1.0 mg/dl) postoperatively and she was discharged on postoperative day 5 with lifelong anticoagulation. At 6 months postoperative, her creatinine is normal (0.8 mg/dl) with venous duplex examination demonstrating absence of residual thrombus.

Discussion

Venous thrombosis contributing to renal allograft dysfunction complicates 0.3–6% of renal transplants. Thrombosis peaks at 2 weeks postoperatively and aetiologies include paediatric donors, multiple renal veins, prolonged ischaemic time, delayed graft function, extrinsic compression, torsion and hypotension/hypoperfusion [1]. Late thrombosis, as in our patient, generally occurs secondary to acute rejection, glomerulonephritis, immunosuppressive therapy, increased haematocrit or extension of DVT due to a hypercoagulable state [2–4]. It is no longer felt that calcineurin inhibitors represent a risk factor for renal vein thrombosis. It is postulated that our patient has a hypercoagulable state due to extension of her previous DVT.
after appropriate anticoagulant therapy. Hypercoagulable states in the setting of renal transplantation are common and include well known conditions such as deficiencies in antithrombin III, protein C and S, factor V Leiden mutation, prothrombin G20210 mutation, elevations in antiphospholipid antibodies and hyperhomocystinaemia. While inherited coagulation defects are quite common, acquired defects such as elevated antiphospholipid antibodies are common in end-stage renal disease [5,6].

Regardless of the aetiology, early diagnosis is key to reducing the morbidity of associated renal allograft dysfunction. Clinical features include allograft tenderness and swelling, anuria or oliguria and symptoms of DVT, all within the setting of an elevated creatinine [7]. Duplex ultrasonography is the initial test of choice. Duplex examination can evaluate the iliofemoral veins for DVT and preserved proximal venous flow at the renal allograft venovenostomy. In addition, the renal allograft should be assessed for graft size, arterial flow characteristics especially diastolic flow, venous outflow and presence of urine within the bladder. However, duplex findings are not specific for venous thrombosis and acute rejection or acute tubular necrosis may present similarly [8]. Venography is considered the gold standard for diagnosis of DVT but must be used judiciously in the setting of acute renal allograft dysfunction. Venography was utilized in our case to confirm patency of the vena cava, thrombosis of the renal allograft outflow and post-thrombectomy to assess venous patency. Utilizing minimal contrast, we were able to limit our dye load to 30 ml of total contrast for the procedure.

Until the initial publication of renal graft salvage in the setting of iliofemoral DVT [9], treatment of all episodes of acute graft dysfunction were treated by renal allograft explantation. Removal of the allograft was recommended due to the risk of allograft rupture and mortality. Currently, the only documented method of renal allograft salvage in the setting of iliofemoral DVT with associated graft dysfunction is thrombolytic therapy. Thrombolytic therapy of an iliofemoral DVT resulting in renal allograft salvage has been reported in a patient 5 months post-transplant [9]. Surgical thromboembolectomy has been documented in early renal allograft vein thrombosis but not in late presentations of renal allograft dysfunction in the setting of iliofemoral DVT [10]. Thrombolytic therapy was considered in our patient, but due to her acutely deteriorating graft function and 24-h history of anuria,
we proceeded to perform a venous thrombectomy with prompt restoration of venous outflow compared with lengthy infusion of thrombolytic agents and avoid the administration of thrombolytics in the setting of potential graft rupture.

Venous thrombectomy should be attempted in the setting of renal dysfunction due to its rapid nature and ability to create immediate reversal of the dysfunction. Venous thrombectomy in this setting is not without risks. These include pulmonary embolism and a high recurrence rate of DVT. Recently, however, venous thrombectomy for iliofemoral DVT has been employed with good results utilizing adjunctive measures such as arteriovenous fistula, the use of full dose perioperative anticoagulation and intermittent pneumatic compression. The patient’s follow up venous duplex at 1 month demonstrates partially occluding thrombus at the level of the posterior tibial vein with an otherwise patent deep venous system.

We believe that iliofemoral venous thrombectomy in the setting of acute renal allograft dysfunction and anuria is a viable option for allograft salvage and treatment of iliofemoral DVT. Ilio f emoral DVT resulting in graft dysfunction must be considered in the patient with signs/symptoms of iliofemoral DVT and oliguria/anuria, graft tenderness and elevated creatinine. Venous thrombectomy under local anaesthesia and sedation allows for prompt diagnosis and evacuation of thrombus with restoration of venous outflow for the compromised allograft and treatment of the patients DVT.

Conflict of interest statement. None declared.

References


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