Case Report

Spontaneous renal allograft rupture without acute rejection

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Abstract. Renal allograft rupture (RAR) is a rare but potentially serious complication in the transplanted recipients. The most common cause is acute rejection. We report four cases (0.5%) of RAR occurred in a series of 778 consecutive kidney transplantations due to severe acute tubular necrosis and renal vein thrombosis with no evidence of acute rejection. Transplant nephrectomy was performed in three patients, whereas graft repair was achieved in one patient. These data suggest that RAR may be associated with renal vein thrombosis or severe acute tubular necrosis in absence of acute rejection. Frequently nephrectomy is necessary, but conservative surgical treatment should be attempted to preserve the allograft in selected cases.

Key words: Allograft rupture, acute tubular necrosis, renal vein thrombosis, kidney transplantation

Introduction

Renal allograft rupture (RAR) is a rare complication in renal transplant recipients, but it potentially threatens graft and patient survival. Clinical presentation is typical, with a well definite onset of symptoms. As reported in literature, RAR is frequently associated with acute rejection, but other causes have been observed (1-2). In most of the cases the surgical treatment is nephrectomy; sometimes, graft repair could be attempted.

We report 4 cases of RAR without acute rejection occurred in 778 consecutive kidney transplants.

Case reports

Case report 1

A 37 years old woman received a cadaveric renal transplant for end-stage renal failure associated to focal glomerulosclerosis. The renal vein was anastomosed end-to-side to the external iliac vein, while renal artery end-to-end to the hypogastric artery. The ureteroneocystostomy was performed using Gregoir-Lich technique. The patient was treated with cyclosporine and azathioprine. Renal function was satisfactory and she was discharged on 10th postoperative day with serum creatinine 2 mg/dl. After 3 days the patient required urgent admission because of sudden acute pain over the graft, hypotension and anuria. Emergency surgical esploration showed the presence of a renal vein thrombosis associated with renal graft rupture. Transplant nephrectomy was performed. Histopathological examination revealed multiple renal infarctions with parenchymal rupture.

Case report 2

The patient was a 62 years old woman with chronic renal failure due to policystic kidney disease. She received a cadaveric renal allograft. The vein was anastomosed end-to-side to the external iliac vein and re-

nal artery end-to-end to the hypogastric artery. Gregoire-Lich technique was used for reconstruction of urinary tract. Immunosuppressive therapy was based on cyclosporine, steroid and azathioprine. There was a delayed renal graft function. On 4th postoperative day she developed important pain and swelling over the graft site, associated with hypotension and anuria. A renal vein thrombosis was evidenced by colour-doppler echography. Urgent laparotomy showed a large perirenal haematoma with complete renal graft rupture and renal vein thrombosis. The graft was removed; pathology examination demonstrated a massive haemorrhagic infarction of the parenchyma. No evidence of acute rejection was identified.

Case report 3

A 22 years old female was submitted to kidney transplantation for end-stage renal failure due to unknown chronic glomerulonephritis. Renal vein and artery were anastomosed end-to-side to external iliac vessels. Re-establishment of urinary continuity was carried out by Gregoire-Lich technique. Immunosuppressive regimen consisted on cyclosporine, azathioprine and steroids. In the first week there was a good renal function (serum creatinine was 2.5 mg/dl). On 8th day after surgery, pain and swelling over the incision area developed with sudden oliguria and elevation of the serum creatinine. Colour-doppler echography revealed a perinephric fluid collection and extensive renal vein thrombosis. Immediate laparotomy revealed the presence of huge hematoma with concomitant renal rupture. Nephrectomy was necessary. Graftectomy specimen confirmed a renal infarction without signs of acute rejection.

Case report 4

A 30 years old man with end stage renal failure secondary to Ig A glomerulonephritis underwent a cadaveric renal transplant. The graft was anastomosed end-to-side on external iliac vessels and the continuity of the urinary tract was established by Gregoire-Lich ureteroneocystostomy.

The patient received double drug therapy with steroids and tacrolimus. In the postoperative period he had a delayed graft function. On 5th day after transplantation, the patient had an attack of severe pain over the graft area associated with anuria and hypotension. Echography revealed the presence of a perirenal haematoma. Emergency surgical esploration showed a large linear transverse renal rupture. Bleeding was stopped by polipropilene 2/0 mattress parenchymal suture enforced with fibrin glue and absorbable collagen fleece application. The patient had an uneventful course and was discharged on 15th day after surgery with good renal function. A renal allograft biopsy obtained intraoperatively showed severe acute tubular necrosis.

Discussion

RAR was first described by Murray et al. in 1968 (3). It is a dramatic event, often associated with loss of the graft. The incidence varies from 0.3 and 9.6% with a mean of 3.4%; usually it develops within 2-3 weeks after transplantation (4).

The acute rejection is considered the main predisposing factor (60-80%) (1). RAR, in the setting of acute rejection, has been related to a significant elevation in intra-renal pressure following interstitial edema and inflammatory infiltration, as well as ischemia secondary to reduction in cortical blood flow (5-6).

Uncommon causes of RAR include renal vein thrombosis, acute tubular necrosis, ureteric obstruction, renal biopsy, heparin therapy, complete lymphatic legation, trauma, nephrostomy tubes and renal cell cancer development (1-2). Generally, no significant association among RAR, donor and recipient features, procurement, storage and cold ischemic time are showed (4).

In our experience RAR occurred in 4 cases (0.5%) and was associated with renal vein thrombosis in 3 patients (75%) and acute tubular necrosis in one patient (25%). No concomitant signs of acute rejection at allograft biopsy were observed.

Thus, renal vein thrombosis was recognized as an important etiological factor. In addition, as reported by other authors (7-9), we confirm that severe acute tubular necrosis can be itself responsible for the RAR because of interstitial edema and rise in intrarenal pressure.

RAR is usually a sudden event with clinical symptoms as pain, swelling in the graft area, hypotension, oliguria or anuria, fever, haematuria and bleeding from the incision. In our cases pain and swelling of the graft were always present, as well anuria or oliguria. Clinical diagnosis may be confirmed by ultrasound or CT scan (4).

The salvage rate varies between 40-100% (10). Mattress suture of parenchyma is the most common conservative procedure. In contrast, other authors suggest the use of fibrin glue and collagen foam to obtain haemostasis without unnecessary manipulation of the graft (11).

The additional application of a mesh (Polyglactin absorbable mesh, lyophilised dura, fascia) wrapping the graft can improve the haemostasis and prevents the expansion and fragmentation of the parenchyma (1-12). Graftectomy is also necessary in renal vein thrombosis if conservative procedures fail, in presence of irreversible acute rejection and in those patients whose hemodynamic status cannot be stabilized. Complication including death is observed in 16.6% (13).

In 1 case a careful mattress parenchymal suture enforced with application of absorbable collagen fleece was carried out successfully; 3 patients required nephrectomy due to extensive renal vein thrombosis.

In conclusion, our results emphasize the association between RAR and renal vein thrombosis. Furthermore, RAR may occur during acute tubular necrosis without acute rejection.

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