



Research Article

Partial Recovery of Late Renal Allograft Infarction

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Abstract

Late thrombotic occlusion is a rare complication in transplant recipients. If produced immediately posttransplantation, the treatment includes transplantectomy. However when it occurs later, as in native kidney infarction, this exceptional situation must be treated with systemic anticoagulation and avoiding the underlying condition. We present the first case of a late allograft infarction with partial recovery after systemic anticoagulation.

Keywords: anticoagulation, kidney allograft, infarction, thrombosis.

Introduction

Thrombotic occlusion is a typical complication after a kidney transplant. It occurs immediately post transplantation, and the more frequent causes are technical problems, hypercoagulable state, unidentified intimal flaps, compressing hematomas or lymphocele and stenosis as explained by Kujovich JL et al., (2004). However late allograft infarction has not been described until the date. Here we present the case of acute allograft dysfunction due to a kidney transplant infarction, 10 years after transplantation.

Case Report

A 74-year old woman was referred to our hospital due to abdominal pain. She had a history of chronic kidney disease due to interstitial nephritis, in hemodialysis

between 1982 and 1988, and 1994 to 2003. In 1988 she received her first renal allograft that progressed to chronic graft dysfunction. In 1994 she underwent her second renal transplantation with normal function before this episode (serum creatinine 0.6 mg/dL [53.04 μ mol/L]). Among its highlights history hypertension, pacemaker placement for atrioventricular block Mobitz type I, parieto-occipital hemorrhagic stroke, chronic liver disease hepatitis C and subtotal thyroidectomy for multinodular goiter. She was on treatment with everolimus, mycophenolate, levothyroxine, omeprazole, atorvastatin and prednisone.

The patient came to the emergency department with fever of 24 hours evolution of 39°C, as well as abdominal pain, predominantly in the left lower quadrant. She referred lower blood

pressures and decreased diuresis. Physical examination revealed a blood pressure of 97/53 mmHg, with a heart rate of 66 bpm and a temperature of 37.6°C. In the heart auscultation presented a mitral systolic murmur radiating to carotids and was rhythmic. Pulmonary auscultation presented bibasilar crackles. The abdominal exploration showed tenderness in the renal graft, which was enlarged. Lower limbs presented slight edema. Blood tests demonstrated: hemoglobin 11.8 g/dL (118 g/L), platelets $151 \times 10^3/L$ ($151 \times 10^9/L$), leukocytes 8100/ μ L, pH 7.40, pCO₂ 39 mmHg, bicarbonate 24 mEq/L (24 mmol/L), glucose 118 mg/dL (6.55 mmol/L), creatinine 1.63 mg/dL (144.09 μ mol/L), urea 47 mg/dL (16.77 mmol/L), sodium 132 mEq/L (132 mmol/L), potassium 4.1 mEq/L (4.1 mmol/L), lactate dehydrogenase (LDH) 2349 U/L, C-reactive protein (CRP) 2.1 mg/dL. The urinalysis objectified 10-20 leukocytes per field, with moderate squamous epithelial cells and sodium in 37 mEq/L (37 mmol/L). Given

the clinical abdominal and analytical data an abdominal CT was requested demonstrating renal graft infarction due to occlusion of the arterial branch supplying the lower pole (figure 1). Doppler ultrasonography showed a hypoperfused area in lower pole of the allograft. After that, renal function worsened until creatinine 6.2 mg/dL (548.08 μ mol/L) was introduced. She was initiated sodium heparine. Due to the renal injury she needed one hemodialysis session in the first 48 hours. The renal function improved thanks to creatinine 2.1 mg/dL (185.6 μ mol/L) and she was discharged with low molecular weight heparine. Studies for evaluating the presence of hypercoagulable state (lupus anticoagulant, anticardiolipin antibodies, levels of C and S protein, antithrombin III, homocysteine, mutation of factor V Leiden) were performed and resulted negative. Image studies showed a flap in the main renal artery as the origin of the embolus.



Discussion

Renal infarction in allografts is an uncommon event that usually occurs early after transplantation surgery. In general population, the most frequent causes of renal infarction are embolic events (due to atrial fibrillation, thromboembolism after a myocardial infarction or release of atheromatous plaque), although it may also be secondary to thrombosis or renal artery dissection as shown by Hazanov et al., (2004) and Nasser et al., (2007). Typical clinical manifestations include fever, nausea, vomiting and intractable abdominal pain, although renal infarction may also be asymptomatic, situation more frequent in kidney transplant because of denervation of the graft as mentioned by Quiroga et al., (2012) and Salehipour et al., (2011). Also, urinary sediment could demonstrate hematuria, leukocyturia (as in our case) or the patient don't show any of these and be normal. Our patient did not have any procoagulant situation (no proteinuric state, no atrial fibrillation, no coagulopathy nor malignant neoplasia). In early arterial or vein thrombosis, treatment usually requires transplantectomy as shown by Blanco et al., (2009). However, as in native kidney infarction our patient was treated with systemic anticoagulation therapy. After this, renal function improved and nephrectomy was not needed.

In conclusion, the uncommon situation of late renal allograft infarction must be treated as in native kidneys.

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