Port J Nephrol Hypert 2014; 28(1): 84-86
Advance Access publication 20 March 2014

Acute allograft kidney dysfunction 18 years after transplantation

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CLINICAL PRESENTATION

A 73-year-old man, with end-stage renal failure secondary to ADPKD was transplanted with a deceased allograft kidney in 1996.

Between 1996 and 2004, he was immunosuppressed with cyclosporine, azathioprine (AZA) and prednisone (PDN). In 2004, after the diagnosis of squamous-cell carcinoma of the lip, the immunosuppression was changed to sirolimus (SRL) and AZA. Since 2010, after sepsis with pulmonary origin, he was in monotherapy with SRL. Along these years, the serum creatinine increased slightly from 1.2 to 1.8 mg/dl.

A diagnosis of adenocarcinoma in the right upper pulmonary lobe was made, in September 2013, and the patient underwent a right upper lobectomy in November 2013. The serum creatinine was 1.9 mg/dl at discharge after surgery. At this time, the sirolimus was switch to tacrolimus.

An allograft kidney biopsy was done 75 days after surgery, due to creatinine increase (1.9 to 3.2 mg/dl), with a proteinuria of 50 mg/dl. The following figures demonstrate the main morphological aspects of the biopsy.

HISTOLOGICAL DIAGNOSIS

The paraffin-embedded fragment shows kidney cortical with a total of six glomeruli and several medium-sized arteries. The frozen fragment shows four glomeruli and the search of C4d in peritubular capillaries by immunofluorescence is negative.

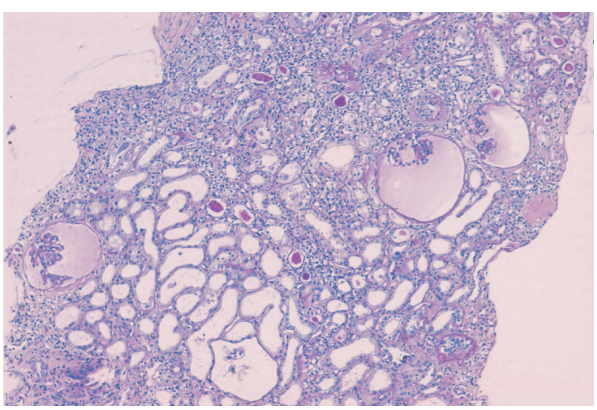

Figure 1
Periodic acid-Schiff, x100.

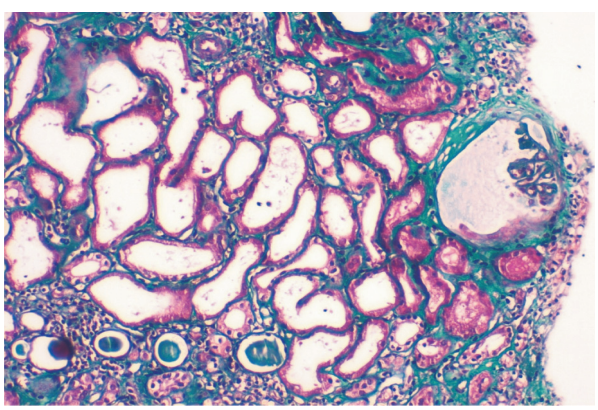

Figure 2
Masson trichrome green, x200.

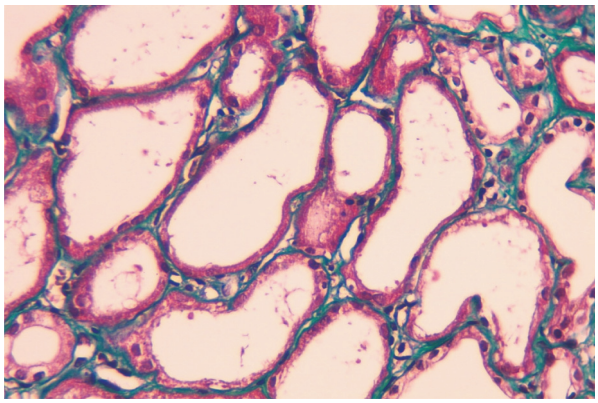

Figure 3

Masson trichrome green, x400.

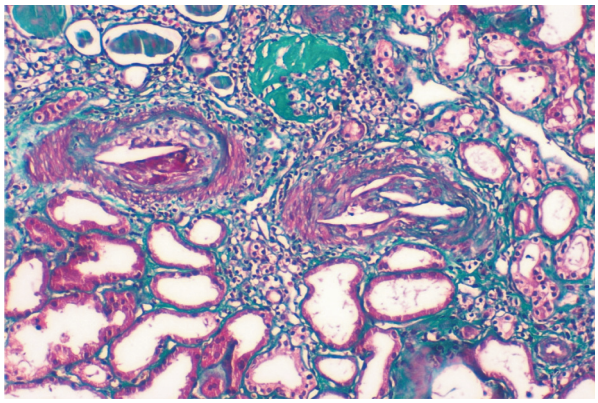

Figure 5

Masson trichrome green, x200.

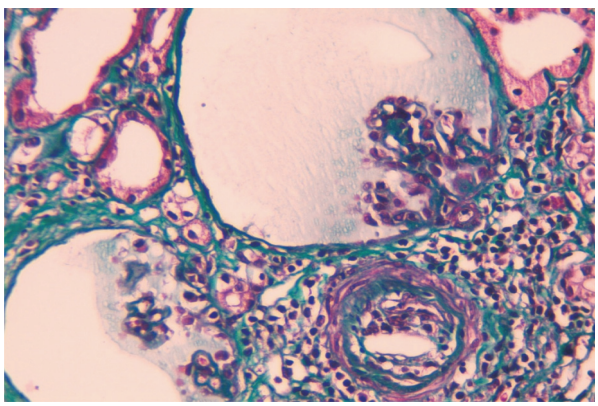

Figure 4

Masson trichrome green, x400.

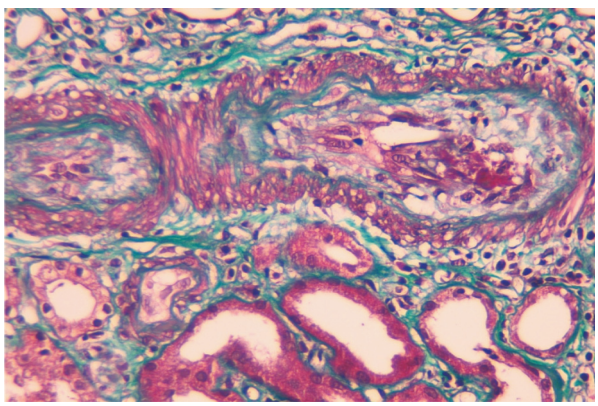

Figure 6

Masson trichrome green, x400.

Figure 1 reveals a kidney with profound alterations; an area of interstitial fibrosis with intense inflammatory infiltration and tubular atrophy stands next to a tubular necrosis area.

Figure 2 presents the acute tubular necrosis area in higher magnification; one ischemic glomerulus is visible.

In **Figure 3,** we can see that the acute tubular necrosis is very severe; almost all the tubular basal membranes are denuded of epithelial tubular cells.

Figure 4 shows two glomeruli with pseudo-cystic appearance; the capillaries tufts are severely retracted to the vascular pole. The medium-sized artery presents a reduction of vascular lumen diameter.

In **Figure 5**, we can see two medium-sized arteries with biconvex, needle-shaped clefts. The vascular lumens are occluded; the clefts are surrounded by fibrous intimal hyperplasia.

In **Figure 6,** the cleft is surrounded by eosinophilic material and fibro-oedematous intimal hyperplasia.

In summary, optical microscopy demonstrates: 3 globally sclerosed glomeruli in areas of interstitial fibrous/tubular atrophy and chronic inflammatory infiltrate; occluded medium-sized arteries surrounded

by severely ischemic, medium-sized arteries and acute tubular necrosis. The histological diagnosis was atheroembolic nephropathy.

COMPLEMENTARY EXAMINATION

Serum analysis reveals a low C3 with normal C4, without eosinophilia.

DISCUSSION

Atheroembolic disease in kidney allograft is a rarely reported disease; between 0.2% and 1% cases[1-3] of all biopsies. Until the end of 2012, we reported 0.21% cases of all biopsies[4] belonging to two patients from our department. In the last year, atheroembolic disease was diagnosis in the first 10 days after transplantation (early form) in one patient and 18 years after transplantation (late form) in this same patient.

The late form of the disease in stable allograft kidney is usually associated with the same risk factors and systemic manifestations as the general populations. The atherosclerothic plaques are disrupted by direct lesion (angiography or surgery) or by drugs (anticoagulant and thrombolytic agents). Our patient underwent a major surgical procedure and take anticoagulants for prophylaxis of pulmonary thromboembolism in the post-operative period. The pathognomonic features[5] are the biconvex, needle shaped clefts. The clefts are the "ghosts" of cholesterol crystals that are dissolved by the xylol used during routine preparation of paraffin-embedded biopsy. In this biopsy, we can see an early phase of endothelial reaction to cholesterol crystals (Fig. 6) and a later phase (Fig. 5). The fibrous intimal hyperplasia results in arteries occlusion and ischaemia of the surrounding tissue.

CLINICO-ANATOMICAL DIAGNOSIS

Atheroembolic disease in allograft kidney.

TREATMENT AND EVOLUTION

The patient was treated with 500 mg IV of methylprednisolone in 3 consecutive days; tacrolimus was switched to sirolimus and low-dose aspirin was initiated. Serum creatinine rapidly decreased to 1.9 mg/dl.

Conflict of interest statement: None declared.

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