

Figura 2. Renal histopathology showing tubulointerstitial lymphoplasmacytic infiltrate (A) and intratubular and interstitial nephrocalcinosis with Von Kossa stain (B). Images at 40X.

In DRTA, or type 1, hypercalciuria is usually multifactorial (release of bone calcium, suppression of the calcium-sensing receptor [CaSR], increased distal sodium load and acidosis), which along with alkaline urine and hypocitraturia favours calculi and nephrocalcinosis. Initially normal GF may decrease over time due to dehydration, nephrocalcinosis, obstructive calculi and/or infection.^{4,5}

The objective of its treatment should be to normalise calciuria and citraturia to prevent nephrocalcinosis and progressive renal damage. As such, the underlying acidosis must be corrected.⁵ Nevertheless, this treatment is a palliative measure when its cause is secondary, such as in SS, where corticosteroids and cyclophosphamide have been used in accordance with the severity of renal involvement, with reports of stabilised renal function, a reduction in proteinuria and reversibility of various clinical manifestations deriving from renal tubular acidosis, therefore preventing the extension of nephrocalcinosis and reducing the risk of progression to end-stage chronic kidney disease.^{1,2} In our case, we used prednisone, which was gradually reduced, with a remission of clinical manifestations being achieved when the autoimmune tubulointerstitial nephritis deriving from SS was controlled.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

1. Ren H, Wang WM, Chen XN, Zhang W, Pan XX, Wang XL, et al. Renal involvement and followup of 130 patients with primary Sjögren's syndrome. *J Rheumatol* 2008;35(2):278-84.

2. Maripuri S, Grande JP, Osborn TG, Fervenza FC, Matteson EL, Donadio JV, et al. Renal involvement in primary Sjögren's syndrome: a clinicopathologic study. *Clin J Am Soc Nephrol* 2009;4(9):1423-31.

3. Yang J, Li X, Huang Q. Renal involvement in primary Sjögren's syndrome: a clinicopathological study of 26 cases. *Zhonghua Nei Ke Za Zhi* 1997;36(1):28-31.

4. Rodríguez Soriano J. Renal tubular acidosis: the clinical entity. *J Am Soc Nephrol* 2002;13:2160-70.

5. Ariceta G, Aguirre M. Tubulopatías en la infancia que progresan hacia la enfermedad renal crónica. *NefroPlus* 2011;4(1):11-8.

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Kaposi's sarcoma in the early post-transplant period in a kidney transplant recipient

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To the Editor:

The chronic use of immunosuppressive agents is associated with the long-term risk of a wide variety of malignancies, including Kaposi's sarcoma (KS), in renal transplant recipients compared with those of the general population. KS occurs after transplantation of 5 to 21 months and more commonly in males. The dose reduction or cessation of immunosuppressive drugs is the mainly approach for the treatment of KS in renal transplant patients, and switching calcineurin inhibitors to mammalian target of rapamycin inhibitors should be considered. Herein, we aimed to announce a 30-year-old male kidney transplant patient who had developed KS despite use of sirolimus after transplantation of 4 month and to the best of our knowledge this is the earliest onset case of KS after kidney transplantation.

CASE REPORT

A 30-year-old male patient with end stage kidney disease received a cadaveric kidney transplant and discharged with a maintenance immunosuppressive therapy consist of the combination of prednisolone, tacrolimus and mycophenolic acid. The patient was hospitalized after transplantation of 10 week because of 2-fold increase in plasma levels of creatinine and diagnosed with calcineurin inhibitor nephrotoxicity based on renal allograft biopsy findings (Presence of nodular hyaline sclerosis in the arteriolar walls of kidney in biopsy and high blood levels of tacrolimus facilitate our ability to make diagnosis of calcineurin inhibitor nephrotoxicity) (Figure 1). Tacrolimus therapy was switched to sirolimus therapy. The patient had a purple red lesion located on the pretibial area of the left leg and diagnosed to be chronic dermatitis by dermatology. In the fourth month after transplantation the patient admitted to hospital because of progression of the lesion and lymphedema on the left leg (Figure 2) and recent occurrence of bilateral lymphadenopathy. Paraaortic, parailiac and bilateral inguinal lymphadenopathies were detected by computer tomography imaging. Excisional lymph node biopsy was performed and reported as KS because of presence of spindle cells consistently stained for CD31 and CD34, and detection of HHV-8 latent antigen within those cells by immunohistochemical staining of bi-

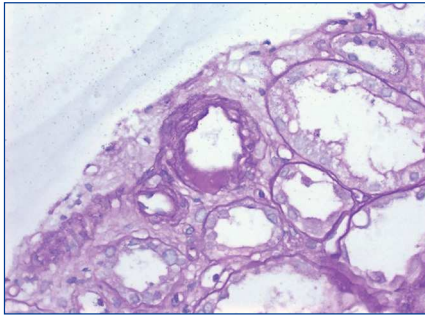


Figure 1. Arteriolar hyalinosis due to calcineurin inhibitor toxicity in a renal biopsy specimen.

opsy specimen. Because of the high risk of acute rejection we did not consider to discontinue or reduce dosage of immunosuppressive drugs and the patient was referred to oncology clinic for receiving chemotherapy due to rapidly progression of cutaneous lesions. The inguinal lymphadenopathy disappeared with chemotherapy regimen that consist of combination of vinblastine and bleomycine but skin lesions persisted.

KS is an angioproliferative neoplasm characterized by reddish-brown or purple-blue plaques or nodules on cutaneous or mucosal surfaces, including the skin, lungs, gastrointestinal tract and lymphoid tissue. Due to high incidence of HHV-8, majority of cases of posttransplant KS has been reported in patients from Mediterranean, Jewish, Arabic, Caribbean, or African descent.^{1,2} Recent advances in immunosuppressive era provide significant benefits in preventing acute rejection episodes in kidney allograft recipients (5). However, there is an increased risk of certain cancers as well as KS with use of long term immunosuppressant agents. The announced

reports has revealed a time ranging from 5 to 21 months after transplantation for time of diagnosis of KS in those patients with kidney transplantation. In our case, KS has occurred in the 4th month of the kidney transplantation.

To the best of our knowledge, this is the earliest onset case of KS that has occurred after the 4th month of the kidney transplantation.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

1. Chang Y, Cesarman E, Pessin MS, Lee F, Culpeper J, Knowles DM. Identification of herpesvirus-like DNA sequences in AIDS-associated Kaposi's sarcoma. *Science* 1994;266:1865-9.
2. Kasiske BL, Snyder JJ, Gilbertson DT, Wang C. Cancer after kidney transplantation in the United States. *Am J Transplant* 2004;4(6):905-13.

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Myeloma kidney: the importance of assessing the response by monitoring free light chains in serum

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To the Editor:

Renal failure is a common and serious complication of multiple myeloma (MM) that leads to a significant increase in morbidity and mortality,¹ with myeloma kidney being the entity most commonly found in this type of patient. The extracorporeal clearance of light chains is considered a support for chemotherapy to decrease the risk of advanced chronic renal failure and the need for chronic renal replacement therapy. It also decreases overall mortality.²

CASE STUDY

A 46-year-old patient without any relevant history, presented at the Emergency Department with pain in his left testicle irradiating to the ipsilateral flank, with no accompanying fever or urinary symptoms. The physical examination was unremarkable except for high blood pressure. The ultrasound of the kidneys and testicles-prostate was without findings. We observed rapidly progressive renal function deterioration (creatinine 9.79mg/dl, 4.85g/24 proteinuria without active sediment) and progressive anaemia (Hb 8.1g/dl, mean corpuscular volume 88fl, mean corpuscular haemoglobin 31.1, mean corpuscular haemoglobin concentration 35.4). The immunological study (antinuclear antibodies, anti-neutrophil cytoplasmic antibodies, anti-GBM, anti-streptolysin O, rheumatoid factor, C3-4), viral serology and tumour markers were normal. Protein electrophoresis-immunofixation with immunoglobulin (Ig) A-kappa monoclonal band. IgG 317, IgA 1446, IgM 15mg/dl, free light chains: (FLC, Free-Lite[®] nephelometer) kappa 4090ng/ml, lambda 1. Uric acid 10.8, LDH 269, calcium 10.2, albumin 3.3, B₂ microglobulin 23,340. Liver profile, lipids and other blood count were normal.



Figure 2. The Kaposi's sarcoma lesion located on the pretibial area of the left leg.