In DRT A, 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 favors calculi and nephrocalcinosis. Initially normal GF may decrease over time due to dehydration, nephrocalcinosis, obstructive calculi and/or infection.\(^5\)

The objective of its treatment should be to normalise calciumuria and citraturia to prevent nephrocalcinosis and progressive renal damage. As such, the underlying acidosis must be corrected.\(^1\) 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.\(^2,3\) 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.


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

**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 pretribial 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. Paraortic, parailiac and bilateral inguinal lymphadenopathies were detected by computer tomography imagination. 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-

**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.

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

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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.
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, major- and bleomycin but skin lesions persisted. 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.


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 88f, mean corpuscular haemoglobin 31.1, mean corpuscular haemoglobin concentration 35.4). The immunological study (anti-nuclear 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.