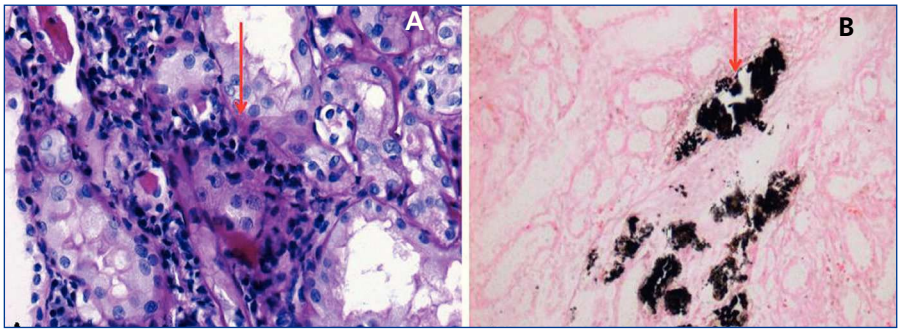

Figura 2. Histopatología renal en la que se muestra infiltrado linfoplasmocitario tubulointersticial (A) y nefrocalcinosis intratubular e intersticial con tinción Von Kossa (B). Imágenes a 40X.

supresión del receptor sensor de calcio CasR, aumento de la carga distal de sodio y acidosis), que junto con la orina alcalina e hipocitraturia favorece la litiasis y nefrocalcinosis. El FG inicialmente normal puede disminuir evolutivamente por deshidratación, nefrocalcinosis, litiasis obstructiva y/o infección[4,5].

Su tratamiento debe llevar como meta normalizar la calciuria y la citraturia, para evitar la nefrocalcinosis y el daño renal progresivo. Para ello, se debe corregir la acidosis subyacente[5]. Sin embargo, este tratamiento constituye una medida paliativa cuando su causa es secundaria, como en el SS, donde se han usado corticosteroides y ciclofosfamida, según la severidad del involucro renal, con reportes de estabilización de la función renal, reducción de proteinuria y reversibilidad de varias manifestaciones clínicas derivadas de la acidosis tubular renal, evitando la extensión de la nefrocalcinosis y reduciendo el riesgo de progresión a estadio terminal de la enfermedad renal crónica[1,2]. En nuestro caso, se usó prednisona con pauta reductiva lenta, logrando una remisión de las manifestaciones clínicas al haber controlado la nefritis tubulointersticial autoinmune derivada del SS.

Conflictos de interés

Los autores declaran que no tienen conflictos de interés potenciales relacionados con los contenidos de este artículo.

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

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Dear 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 immunosuppresive 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 immunusuppresive therapy consist of the combination of prednisolone, tacrolimus and mycophenolic acide. 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 lympedema on the left leg (Figure 2) and recent occurrence of bilateral lymphadenopathy. Paraaortic, 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-

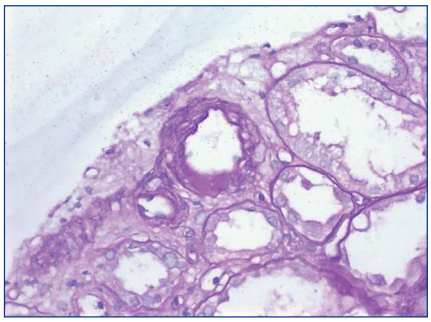

Figure 1. Arteriolar hyalinosis due to calcineurin inhibitor toxicity in a renal biopsy specimen.

opsy specimen. Because of the high risk of acute rejection we did not consider to discontinue or reduce dosage of immunusppresive drugs and the patient was refered to oncology clinic for receiving chemotherapy due to rapidly progression of cutaneuos 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, Carribean, or African descent.[1,2] Recent advances in immunosuppresive 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 immunusuppresant agents. The ennounced 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.

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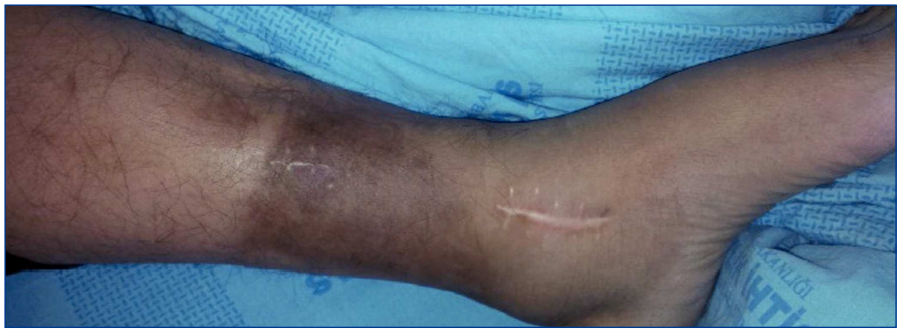

Figure 2. The Kaposi's sarcoma lesion located on the pretibial area of the left leg.

Riñón de mieloma: importancia de la valoración de respuesta mediante monitorización de cadenas ligeras libres en suero

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Sr. Director:

La insuficiencia renal es una frecuente y grave complicación del mieloma múltiple (MM), que conduce a un aumento significativo en la morbimortalidad del paciente[1], siendo el riñón de mieloma la entidad más frecuente encontrada en este tipo de pacientes. La depuración extracorpórea de cadenas ligeras se considera un tratamiento coadyuvante a la quimioterapia para disminuir el riesgo de insuficiencia renal crónica avanzada y necesidad de tratamiento renal sustitutivo crónico, disminuyendo también, a su vez, la mortalidad global[2].

CASO CLÍNICO

Paciente de 46 años sin antecedentes de interés, acude a Urgencias por dolor en teste izquierdo irradiado a flanco ipsilateral, sin fiebre ni síndrome miccional acompañante. Exploración física anodina, salvo hipertensión arterial. Eco renal y testículo-prostática sin hallazgos. Se aprecia deterioro de la función renal rápidamente progresivo (creatinina 9,79 mg/dl, 4,85 g/24 proteinuria sin sedimento activo) y anemización progresiva (Hb 8,1 g/dl, volumen corpuscular medio 88 fl, hemoglobina corpuscular media 31,1, concentración de hemoglobina corpuscular media 35,4). Estudio inmunológico (anticuerpos antinucleares, anticuerpos anticitoplasma de neutrófilo, anti-MBG, antiestreptolisina, factor reumatoide, C3-4), serología viral y marcadores tumorales, normales. Proteinograma-inmunofijación con banda monoclonal inmunoglobulina (Ig)A-kappa. IgG 317, IgA 1446, IgM 15 mg/dl, cadena ligera libre (CLL, nefelometría Free-Lite®) kappa 4090 ng/ml, lambda 1. Ácido úrico 10,8, LDH 269, calcio 10,2, albúmina 3,3, B_2 microglobulina 23 340. Perfil hepático, lipídico y resto de hemo-