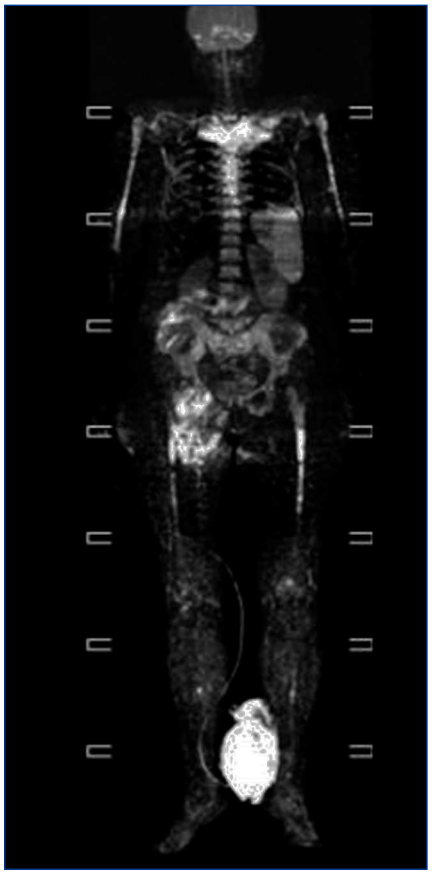

Figure 2. Full-body magnetic resonance.

oligosecretory MM with a minimal monoclonal component and very extensive spread that led to patient death one week after diagnosis.

Conflicts of interest

The authors have no conflicts of interest to declare.

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M. Heras[1], J. Izquierdo[2], A. Saiz[3], J. Hernández[4], J.A. Queizan[4], J. González[5], M.J. Fernández-Reyes[1], R. Sánchez[1]

[1] Nephrology Department.
General Hospital of Segovia, Spain
[2] Department of Internal Medicine
General Hospital of Segovia, Spain
[3] Anatomical Pathology Department.
Ramón y Cajal Hospital. Madrid, Spain
[4] Haematology Department.
General Hospital of Segovia, Spain5 Radiology Department.
General Hospital of Segovia, Spain
Correspondence: M. Heras
Servicio de Nefrología.
Hospital General de Segovia. 40002 Segovia. Spain.
manuhebe@hotmail.com

Unusual epidemiological pattern in kidney transplant patient with HIV and Kaposi's sarcoma. Resolution after sirolimus therapy

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To the Editor,

Kaposi's sarcoma (KS) is associated with human herpes virus 8 (HHV-8), although, alone, HHV-8 is not a major risk factor. Situations that affect immunity, such as infection from human immunodeficiency virus (HIV) or immunosuppressant treatment in solid organ transplant (SOT) patients, markedly increase risk.[1,2] KS in SOT patients tends to occur within the first few months post-transplant.[1,3] A high CD4 lymphocyte count and high-activity anti-retroviral therapy (HAART) both significantly reduce the risk in patients with HIV.[2,3]

Proliferation signal inhibitors (PSI) inhibit tumour angiogenesis by reducing the production of vascular endothelial growth factor (VEGF) and its Flk-1/KDR receptor. The VEGF system plays a central role in the development of KS, and so the effect of PSI is particularly relevant. The results observed in SOT with KS after converting to PSI demonstrate this relationship, and these drugs are currently the primary therapeutic option.[1] In KS associated with HIV, the first line of treatment is controlling the HIV with HAART, and the usefulness of PSI is still being researched.[2] Here we present the case of an HIV patient with a kidney transplant (KT) that developed KS.

A 59-year old male infected with HIV (by sexual transmission) that was well controlled with HAART received a KT in May 2001. He then continued treatment with HAART, always maintaining a negative viral load and CD4>200 cells/μl. The immunosuppressant treatment consisted of steroids, mycophenolate, and tacrolimus. The mycophenolate was suspended in January 2003 due to haematological intolerance. In august 2003, we performed a biopsy due to progressive deterioration of renal function and the patient was diagnosed with chronic nephropathy.

In February 2010, purple nodular lesions appeared on the patient's left arm, leading to the histopathological diagnosis of KS with intense immunohistochemical expression for CD31, CD34, and HHV-8. An analysis of the extent of the disease ruled out visceral involvement. Blood PCR analysis was negative for cytomegalovirus (CMV), Epstein-Barr virus HHV-6, HHV-7, and HHV-8, with positive HHV-8 serology (IgG-IFI). At this moment the patient already had creatinine levels nearing 4.5mg/dl due to the chronic nephropathy. We decided to significantly reduce the tacrolimus prescription (to 3-4ng/ml) and start treatment with sirolimus (at 4-6ng/ml. We did not completely remove the tacrolimus treatment for fear of poor tolerance to PSI due to renal failure. Renal function continued to deteriorate, and we recommenced dialysis in November 2010, suspending tacrolimus. We continued sirolimus treatment at low doses until the KS was resolved in Match 2011. The patient currently continues on dialysis with a complete remission of all lesions.

Our patient developed KS very late (9 years post-KT) and in spite of a good control of the HIV infection. He had several different risk factors (HHV-8, HIV, immunosuppressant treatment) that were able to act synergistically, favouring the development of the tumour with an atypical epidemiological pattern. Given the experience published regarding HIV-negative SOT, we believe that it was the conversion to sirolimus rather than the decreased tacrolimus levels that caused the favourable evolution of the KS. Only three other cases of KS in HIV-positive SOT (two KT, one live transplant) have been communicated with a positive response following conversion to PSI.[4,5] However, these cases were not described in detail since they made up part of a general series on SOT in patients with HIV.

Although the most extensive treatment in HIV-positive SOT is the combination of MMF and anti-calcineurinics, good results have also been obtained with protocols based on PSI.[5,6] Additionally, PSI appear to have beneficial effects on the replication of HIV.[5] We believe that a treatment regimen based on PSI could be the therapy of choice as the primary immunosuppressant following KT in transplant recipients with HIV infection and risk factors for KS (male, sexual practices, ethnicity, HHV-8 positive), as long as contraindications do not exist.

Conflicts of interest

The authors have no conflicts of interest to declare.

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L. Calle[1], A. Mazuecos[1], T. García Álvarez[1], F. Guerrero[2], A. Moreno[1], R. Collantes[1], M. Rivero[1]

[1] Nephrology Department. Puerta del Mar Hospital. Cádiz, Spain

[2] Department of Internal Medicine. Puerta del Mar Hospital. Cádiz, Spain

Correspondence: A. Mazuecos
Servicio de Nefrología.
Hospital Puerta del Mar. Ana de Viya, 21.
11007 Cádiz. Spain.
mauxiliadora.mazuecos.sspa@juntadeandalucia.es
auxiliadoramazuecos@ono.com

Acute renal failure secondary to sodium and water depletion due to diarrhoea plus acetazolamide

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To the Editor,

The use of acetazolamide for the treatment of Meniere's syndrome[1,2] is uncommon, since other drugs constitute the first line used in the treatment of this pathology.

Here we present the case of a 70-year old woman whose most relevant background involved Meniere's syndrome, currently treated with acetazolamide at 250mg/12h with oral potassium supplements.

The patient was referred to us due to severe renal failure (urea: 194mg/dl; C-reactive protein: 8.1mg/dl), with extremely severe metabolic acidosis (venous gas: pH: 7.1, bicarbonate: 6.5mmol/l, PCO_2: 18mm Hg, PO_2: 69mm Hg) and anuria. Laboratory analyses from a few months prior indicated C-reactive protein of 1mg/dl. The patient had been admitted to the nearest reference hospital 24 hours before as a result of severe diarrhoea, and continued taking the normal treatment. Hydration treatment had been started without diuresis, and the patient was transferred for monitoring. Upon hospitalisation, dehydration signs were present, with a blood pressure of 80/50mm Hg, anuria, and no fever. At this point the patient did not have diarrhoea.

Laboratory analysis revealed acidosis, hypokalaemia (K: 2.9mEq/l) and renal failure similar to that described. We continued to provide intense rehydration therapy, despite which the patient continued with anuria for 24 hours more, with C-reactive protein levels reaching 11mg/dl. Once the volume level was normalised, diuresis started to improve, with clinical and laboratory improvements until reaching a C-reactive protein of 1.2mg/dl upon discharge, with a complete acid-base correction. The evolution of the laboratory values are shown in Table 1.

The patient was discharged with the diagnosis of prerenal acute renal failure due to severe hydrosaline depletion. Metabolic acidosis with a normal anion-gap, in the context of acute renal failure due to the ingestion of acetazolamide and diarrhoea.

Metabolic acidosis is defined as a process in which the blood pH decreases, with the first effect of decreased bicarbonate concentrations followed by the secondary effect of reduced PCO_2. Our patient was admitted with acidosis: a pH of 7.1 and a bicarbonate level of