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

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

Table 1. Evolution of laboratory parameters

	14/3/11	15/3/11	16/3/11	17/3/11	18/3/11	19/3/11	21/3/11	22/3/11
Ph	7.10	7.1	7.11	7.26	7.39	7.4	7.45	
Bicarbonate (mmol/l)	6.1	7.9	7.6	17.5	15.5	20.4	22	
PCO$_2$ (mm Hg)	18	25	24	39	25	33	36	
Crp (mg/dl)	8.1	10	11	9.7	7.1	4.5	1.4	1.2
Urea p (mEq/l)	194	231	261	241	228	203	85	45
Potassium p (mEq/l)	2.9	3.1	2.8	2.3	2.8	3	4.2	4.3
Chlorine p (mEq/l)	120		115			112		110
Sodium p (mEq/l)	138	142	140	136	139	143	142	144
Sodium o (mEq/l)		47						
Potassium o (mEq/l)		35						
pHurine		5.5						

6mEq/l. The acidaemia caused increased ventilation that induced a hypocapnic response that tends to normalise pH. The level of compensation expected can be deduced from the following formula: $pCO_2 = 1.5\ [HCO_3] + 8$ (± 2). In our patient, the level of compensation was adequate (pCO$_2$: 17mm Hg), and so this was a case of pure metabolic acidosis, with correct respiratory compensation.[3,4]

The patient's anion gap upon hospitalisation was normal, near 12. The cases of metabolic acidosis with a normal anion *gap* (hyperchloremic) suggest that bicarbonate has been replaced by chlorine as it is lost. The most common causes of this type of acidosis are gastrointestinal loss through diarrhoea and renal loss which, in our case, were both caused by acetazolamide.

The patient had low potassium levels, lost through the faeces, and volume depletion that increase the synthesis of renin and aldosterone, which in turn raises the renal secretion rate of potassium, in this case, upon admission, to 2.9mEq/l despite oral supplements. Instead of acidic urine we can observe urine pH of six or higher because the metabolic acidosis and hypokalaemia increase the renal excretion and synthesis of NH$_4^+$ and in turn buffers the H$^+$ and increases urine pH. The fractional excretion of sodium tends to be low (<1%-2%) in patients with gastrointestinal losses of HCO$_3$, although this value was high in our patient, without the classic inversion of sodium/potassium in urine samples typical of prerenal conditions due to the combined effects of diarrhoea and diuretics.[5,6]

In the case that proximal reabsorption of bicarbonate is ineffective due to the use of acetazolamide, which inhibits carbonic anhydrase, bicarbonate losses are produced, such that this molecule escapes from the proximal tubule and reaches the cortical collecting tubule or the distal convoluted tubule, producing reabsorption of sodium and excretion of potassium and hydrogen ions. The low permeability of bicarbonate impedes its reabsorption, which creates a negative potential in the tubular lumen that increases the excretion of potassium and hydrogen ions.

With regard to treatment, the correct approach is to treat the diarrhoea, as well as hydrosaline correction in order to halt the perpetuating stimulus of the renin-angiotensin-aldosterone pathway, with the use of bicarbonate in a continuous perfusion and potassium chloride in order to replace the lost potassium, along with the evident removal of the diuretics. In this manner, our patient responded satisfactorily, with an excellent clinical evolution.

Conflicts of interest
The authors have no conflicts of interest to declare.

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