thiosulphate form as it inhibits calcium precipitation and dissolves calcium deposits in tumours and calciphylaxis.[9]

We hope that by describing other cases of calciphylaxis in kidney transplant patients we are able to raise awareness about the use of treatments like cinacalcet, sodium thiosulphate and bisphosphonates, among others. Although, in this case, bone scan was uninformative, it seems that this procedure has a high sensitivity for diagnosing this disease, showing an abnormal isotope uptake on a subcutaneous level in 97% of cases.[10]

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Pneumonitis caused by sirolimus: improvement after switching to everolimus

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Dear Editor,

Sirolimus is the foremost proliferation signal inhibitor (PSI) used in medicine to prevent acute rejection in solid organ transplants. Among the main advantages of this drug is its very low nephrotoxicity and above all, its antitumour action, especially in transplanted patients that develop Kaposi's sarcoma.[1] However, one of its most serious side effects is interstitial pneumonitis, which in most cases leads to the suspension of treatment with this drug.[2]

We would like to describe the case of a 64-year-old woman who underwent a transplant for the first time in September 1992 and experienced early graft loss because of acute vascular rejection. She received a second kidney transplant in December 2000 and was treated with daclizumab, steroids, mycophenolate and tacrolimus. In the third month following the transplant, mycophenolate treatment was suspended definitively because of leukopenia. In June 2003 she developed Kaposi's sarcoma which did not respond to a reduction in the tacrolimus dose. Therefore, in November 2003 tacrolimus was substituted by sirolimus and excellent progress was made before the patient fully recovered in a short period of time. With regard to the patient's recovery from Kaposi's sarcoma, this is something that had been described previously in a study on a series of patients from different hospitals in Spain.[1]

She remained asymptomatic and with stable kidney function (CRP 1.2-1.3mg/dl) until July 2008. From that moment onwards she made several visits to the Emergency Departments complaining of dyspnoea. The chest x-ray repeatedly showed parenchymatous infiltrates in the middle and lower lobes on both sides of the chest. Although there were no significant findings in the echocardiogram (mild LVH and preserved ejection fraction), a diagnosis of heart failure was made and diuretic treatment was indicated. In September 2008, when she was checked in a transplant clinic and no clinical or radiological improvements were observed, interstitial pneumonitis was suspected and further tests were carried out: 1) full blood count and biochemistry did not indicate any relevant abnormalities; 2) sirolimus levels: 7ng/ml; 3) arterial gases at baseline: pH: 7, 44, pCO_2: 35, pO_2: 73, $SatO_2$: 95%; 4) Chest CAT scan: bilateral peripheral pulmonary infiltrates, with ground glass opacity in some areas and a reticular pattern in others, no adenopathies; 5) respiratory function tests: mild restrictive ventilatory pattern and moderately affected difussion capacity; 6) immunological study (ANA, ANCA, CRP, rheumatoid factor, complement, immunoglobulins): normal; 7) angiotensin-converting enzyme: 32U/L (normal); 8) testing for common and atypical infections, including pneumocystis, using the induced sputum technique: negative. In light of these findings, once we had ruled out infectious and autoimmune causes, we made the diagnosis of interstitial pneumonitis caused by sirolimus. Given the seriousness of the symptoms that prompted the change to sirolimus (Kaposi's sarcoma), we decided to switch to a different PSI, everolimus. The response to the new treatment was fast and very positive, the patient made a full recovery and the chest x-ray and respiratory function tests were normal within a period of a few weeks. The patient remained asymptomatic with everolimus levels at around 7ng/ml.

Interstitial pneumonitis caused by sirolimus is characterised by allergy-like symptoms and its incidence is relatively significant (4-14% according to some series).[2] This has also been observed with

the use of everolimus although it seems to be less common.[3] Until now, seven cases of recovery from pneumonitis caused by sirolimus after switching to everolimus have been reported.[4,5] The progress made in all cases has been satisfactory, except for in one case where symptoms recurred. The lower pulmonary toxicity seems to be due to the fact that everolimus is more hydrophilic because it differs from sirolimus by one hydroxyl group. Therefore, although the mechanism of action is the same, sirolimus and everolimus may have slightly different side effects. Therefore, the switch from sirolimus to everolimus may lead to recovery in cases of interstitial pneumonitis, especially for those patients who still require PSI treatment like the one described.

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Fanconi syndrome following an accident at work

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Dear Editor,
Fanconi syndrome is characterised by generally affecting the proximal tubule, impairing the reabsorption and secretion of different substances.

We would like to describe the case of a 20-year-old male patient who was admitted with first and second degree burns affecting 30% of his total body surface area (TBSA), caused by contact with a toxic substance. On admission, kidney function was normal.

One week later he began to complain of general discomfort and deteriorated kidney function (Cr 4.1mg/dl), accompanied by metabolic acidosis, proteinuria and glycosuria. Proximal tubulopathy was suspected and a 24 hour urine test was carried out which confirmed the presence of glycosuria (16g/24 h), aminoaciduria, phosphaturia, hypercalciuria, high concentrations of sodium, potassium, chloride, magnesium, and proteinuria 2.6g/day. The patient was subsequently diagnosed with Fanconi syndrome of unknown origin.

None of the treatments administered during hospital admission were considered responsible for triggering the symptoms. Another possibility that was considered was described in other cases of burned patients affected by tubulopathy, however this was associated with larger burns (TBSA greater than 50%).

After two months, we were able to obtain an analysis of the substance that triggered the symptoms which were highly toxic because of the lead and cadmium content, which both could have caused acquired Fanconi syndrome.

Three months after the symptoms appeared, the patient completely recovered from the tubulopathy.

Fanconi syndrome is characterised by dysfunction affecting the proximal tubule and impaired ability to reabsorb glucose, aminoacids, phosphate and often bicarbonate as well. This manifests as glycosuria, aminoaciduria, hyperphosphaturia and proximal tubular acidosis.

This is associated with many conditions that range from metabolic-genetic disorders to exposure to different toxic substances like heavy metals.[1,2] Some cases describe burns covering a large area (affecting over 50% of the TBSA) yet it is unclear what the underlying mechanism is.[3]

Our case is most likely to be associated with exposure to heavy metals, given the toxic concentrations of cadmium and lead in the toxic substance analysed.

Different heavy metals like cadmium, mercury, lead and platinum are toxic at low concentrations and have a long half life making exposure potentially dangerous. The kidney is the first organ targeted for toxicity by heavy metals because of its capacity to reabsorb and accumulate divalent metals. Heavy metal poisoning has contributed to nephropathies of varying severity, from tubular dysfunction to severe kidney failure.[4]

Divalent cations (zinc, iron and copper) are involved in different physiological functions and are necessary in low concentrations.[4] They are mainly transported via the proximal tubule. The same transport also reabsorbs toxic cations (cadmium, lead, cobalt, nickel and platinum).[5]

Trace elements like iron, cobalt and zinc should be used in treatment to minimise kidney damage and to saturate the transport and prevent the absorption of toxins. Our patient was treated on a casual basis with iron for mild anaemia and with topical zinc sulphate 1/1000 which is the usual treatment administered to burned patients because of its keratolytic and astringent properties.