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.

1. Gutiérrez-Dalmau A, Sánchez-Fructuoso A, Sanz-Guajardo A, Mazuecos A, Franco A, Rial MC, et al. Efficacy of conversion to sirolimus in posttransplantation Kaposi's sarcoma. Transplant Proc 2005;37:3836-8.
2. Weiner SM, Sellin L, Vonend O, Schenker P, Buchner NJ, Flecken M, et al. Pneumonitis associated with sirolimus: clinical characteristics, risk factors and outcome – a single center experience and review of the literature. Nephrol Dial Transplant 2007;22:3631-7.
3. Alexandru S, Ortiz A, Baldovi S, Milicua JM, Ruíz-Escribano E, Egido J, et al. Severe everolimus-associated pneumonitis in a renal transplant recipient. Nephrol Dial Transplante 2008;23:3353-5.
4. Rehm B, Keller F, Mayer J, Stracke S. Resolution af sirolimus-induced pneumonitis after conversion to everolimus. Transplant Proc 2006;38:711-3.
5. De Simone P, Petruccelli S, Precisi A, Carrai P, Doria R, Menichetti F, et al. Switch to evrolimus for sirolimus-induced pneumonitis in a liver transplant recipient – Not all proliferation signal inhibitors are the same: a case report. Transplant Proc 2007;39:3500-1.

L. Calle, C. Tejada, C. Lancho, A. Mazuecos
Department of Nephrology. Puerta del Mar Hospital. Cádiz, Spain
Correspondence:
Auxiliadora Mazuecos Blanca
Servicio de Nefrología.
Hospital Puerta del Mar. Cádiz.
27541jcg@comb.es

Fanconi syndrome following an accident at work

Nefrología 2009;29**(5)**:491-492.

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.

It is possible that these measures may have partially contributed to the patient's recovery.

1. Roth KS, Foreman JW, Segal S: The Fanconi syndrome and mechanism of tubular transport dysfuntion. Kidney Int 1981;20:705-16.
2. Izzedine H, Launay-Vacher V, Isnard-Bagnis C, Deray G: Drug-induced Fanconi's Syndrome. Am J Kidney Dis 2003;41(2):292-309.
3. Lindquist J, Drueck C, Simon NM, Elson B, Hurwich D, Roxe D. Proximal renal tubular dysfunction in severe burns. Am J Kidney Dis 1984;4(1):44-7.
4. Barbier O, Jacquillet G, Tauc M, Cougnon M, Poujeol P. Effect of heavy metals on, and handling by the kidney. Nephron Physiol 2005;99:105-10.
5. Ferguson CJ, Wareing M, Ward DT, Green R, Smith CP, Riccardi D. Cellular localization of divalent metal transporter DMT-1 in rat kidney. Am J Physiol Renal Physiol 2001;280:F803-14.
6. Sabolic I. Common mechanisms in nephropathy induced by toxic metals. Nephron Physiol 2006;104:107-14.

A. Vello Román, M. Samprón Rodríguez, B. Pazos Arias
Povisa Hospital. Vigo, Spain.
Correspondence:
Arantxa Vello Román
Hospital Povisa. Vigo.
arantxavr6@hotmail.com

Pregnant patient with acute pyelonephritis and renal corticomedullary abcess: ultrasound and MRI imaging

Nefrología 2009;29**(5)**:492-494.

Dear Editor,

Urinary infections are common during pregnancy, affecting 10-15% of women. In 1-2.5% of pregnancies there are complications due to acute pyelonephritis[1] and the infection is recurrent in up to 10% of women. The development of a kidney abscess secondary to acute pyelonephritis is uncommon during pregnancy. This can affect patients with urinary tract alterations and diagnosis of the condition requires a high index of suspicion and confirmation using ultrasound.[2-5] The most common causal agents are enterobacteria. The infection most commonly affects the right kidney (90%), it is usually unilateral and associated with high morbidity, which is why early diagnosis and treatment are necessary.[1,2-4]

This is the case of a 23-year-old woman with a history of repeated urinary tract infections since the age of 16. In 2005 she miscarried at 12 weeks and this coincided with a urinary infection caused by *Escherichia coli*. In November 2006 when she was 12 weeks pregnant, the following results were obtained from a urine test: pH 7.5, nitrites (+), leukocytes (++). Sediment: leukocytes 20-40 per field and abundant bacteria. No treatment was prescribed. On 4 January 2007 when she was 20 weeks pregnant, she had a fever of 39º C and pain in the right renal fossa. The biochemistry showed: leukocytes 27,800/mm^3 (85% neutrophils), Hb 8.7g/dl, platelets 288,000/mm^3 and GSV 16-39mm/h. Other data: Na 138mmol/l, K 4.8mmol/l, Cl 96mmol/l, glucose 66mg/dl, urea 48mg/dl, creatinine 0.7mg/dl, uric acid 3.4mg/dl and CRP 31mg/dl. Urine: blood 25/ml, nitrites (+), leukocytes 100/ml. Sediment: leukocytes 31-50 per field, isolated red blood cells and abundant bacteria. Urine culture: *Escherichia coli*. She received 1g/12 hours of intravenous cefotaxim treatment for four days. The fever disappeared after 48 hours and there were improvements in her clinical condition and test results. On 18 January 2007, in the 22nd week of pregnancy, she was admitted into hospital again with the same symptoms. The biochemistry showed: leukocytes 36,800/mm^3 (91% neutrophils), Hb 8.4g/dl, platelets 357,000/mm^3. Urine: leukocytes (+++), proteinuria (++), urea 55mg/dl, creatinine 1.2mg/dl, total proteins 5.1g/dl and CRP 69mg/dl. The ultrasound showed: the right kidney increased in size (14cm larger in diameter), with a reduction in the corticomedullary differentiation and a hypoecogenic image of 8mm at cortical level in the upper pole, with echogenic content that was suggestive of a corticomedullary abscess, moderate dilatation of the calyx, pelvis and proximal ureter (figure 1). The major axis of the left kidney measured 12.7cm, echogenicity was normal and there was mild pyelocalceal dilatation. The patient was first administered treatment with amoxicillin clavulanate 500mg/8 hours, and then 850/125mg/8 hours; after 48 hours her fever subsided and her symptoms improved. Nine days after the first ultrasound, a second one was carried out which continued to show the image of the abscess in the right kidney. On 8 February 2007, when she was 24 weeks pregnant a MRI scan showed bilateral pyeloureteral dilatation that was greater on the right side, with blunting of the calyceal fornices and a renal pelvis measuring 2.8cm. The right kidney was enlarged with deteriorated corticomedullary differentiation. There was a hyperintense image 8-10mm in diameter in the upper pole around the corticomedullary area, which seemed to be a parenchymatous abscess (figure 2). Treatment using amoxillin clavulanate 875/125mg/8 hours was administered for three weeks. The clinical progress made by the patient was favourable and leukocyte levels were normalised (figure 3). After two weeks, a new MRI scan showed a microabscess which was 15mm in diameter and a renal pelvis of 3cm. In a new ultrasound scan carried out three weeks later, the lesion had decreased in size. Antibiotic therapy continued (oral

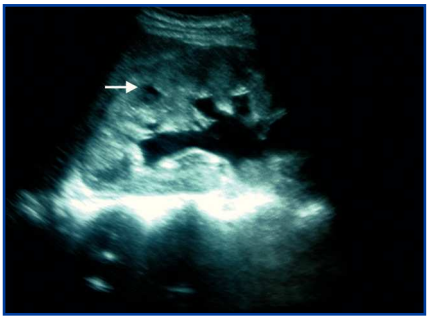

Figure 1.