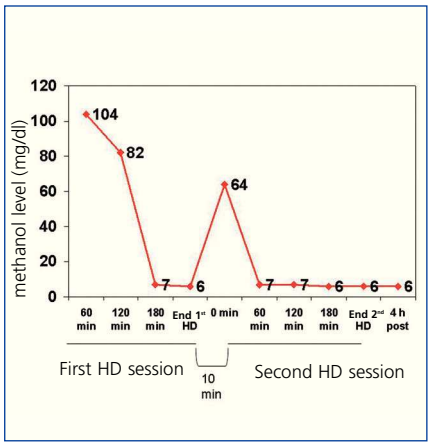

Figure 1. Evolution of blood methanol levels during the haemodialysis sessions

Discussion

HD[2-4] is used for severe methanol poisoning. Its indications are[5]: blood methanol level >50mg/dl (not immediately available in our centre), severe metabolic acidosis, visual or neurological disturbances and >30ml methanol intake.

Very prolonged HD sessions have been reported (>1 day).[6] Hirsch[7] calculates the duration of HD according to levels of methanol [t(hours)=-Vln(5/A)/0.06k, where t is time in hours, V is the volume of body water measured by Watson, A is the level of methanol in blood in mg/dl and k is 80% of urea clearance estimated by the manufacturer]. The level of methanol can be inferred using the osmolar gap, since there is a good correlation between both values,[1] minus the normal gap (10-12mOsm/kg). In our case the gap was 92mOsm/kg, which would be about 250mg/dl, with an estimated HD duration of 9 hours.

The fall of methanol in the blood was greater than in the Hirsch study,[7] with a relapse while the filter was being changed. This may be because large surface membranes with a high dialysate flow the poison is quickly removed from the vascular compartment, but time is needed for the rest of the poison to be released from the extra-vascular space into the bloodstream.

There are no studies comparing intermittent and continuous HD techniques,[1,8] but obviously intermittent HD is always faster. If these findings are confirmed, combining the two techniques should be considered (for example, HD of 3-4 hours followed by the continuous technique) or the Hirsch formula corrected.

1. Peces R, Gonzále E, Peces C, Selgas R. Tratamiento de las intoxicaciones graves por alcoholes. Nefrologia 2008;28:369-72.
2. Holubek WJ, Hoffman RS, Goldferb DS, Nelson LS. Use of hemodiaysis and hemoperfusion in poisoned patients. Kidney Int 2008;74:1327-34.
3. Peces R, Fernández R, Peces C, González E, Olivas E, Renjel F, et al. Eficacia de la hemodiálisis precoz con membranas de alto flujo en el tratamiento de las intoxicaciones graves por alcoholes. Nefrologia 2008;28:413-8.
4. De Arriba G, Torres-Guinea M, Chevarría J, Basterrechea MA. Utilidad de la hemodiálisis prolongada en el tratamiento de la intoxicación aguda por metanol. Nefrologia 2008;28:664-5.
5. Krant JA, Kurtz I. Toxic alcohol ingestions: clinical features, diagnosis and management. Clin J Am Soc Nephrol 2008;3:208-25.
6. Seyffort G. Poison Index. The treatment of acute intoxication (4.TH ed.). Lergerich: Pabst Science Publishers; 1997. p. 457-64.
7. Hirsch DJ, Jindel KK, Wong P, Fraser AD. A simple method to estimate the required dialysis time for cases of alcohol poisoning. Kidney Int 2001;60:2021-4.
8. Tyagi PK, Winchester JF, Feinfeld DA. Extracorporeal removal of toxins. Kidney Int 2008;74:1231-3

J.I. Minguela[1], M.J. Lanzagorta[2], A. Hernando[1], J. Audicana[3]

[1] Nephrology Department. Galdakao Hospital. Galdakao, Biscay, Spain

[2] Nephrology Department Haemodialysis Unit, Galdakao Hospital. Galdakao, Biscay, Spain.

[3] Intensive Care Unit. Galdakao Hospital. Galdakao. Biscay, Spain.

Correspondence:
José Ignacio Minguela Pesquera
Servicio de Nefrología. Hospital Galdakao. Barrio Labeaga, s/n. 48960 Galdakao. Bizkaia.
joseignacio.minguelapesquera@osakidetza.net

Acute renal failure due to oxalate crystal deposition and enteric hyperoxaluria

Nefrologia 2011;31(1):121-3
doi:10.3265/Nefrologia.pre2010.Oct.10644

To the Editor,

Acute renal failure associated with intratubular crystal precipitation is a common cause of renal injury which may occur in the context of a wide variety of clinical situations. The most common are those associated with uric acid nephropathy and with intravenous acyclovir, sulphonamides, methotrexate, and indinavir treatment. Most patients affected by this type of renal disease have a variety of predisposing risk factors, notable among which are effective intravascular volume depletion and previous chronic renal failure.[1]

Ingestion of ethylene glycol and, exceptionally, the administration of ascorbic acid at high doses can cause hypercalcaemia and acute renal failure due to deposition of oxalate crystals.[2] However, enteric hyperoxaluria is another potential cause of kidney stones and interstitial nephritis in patients with short bowel syndrome or other causes of fat malabsorption in patients without colectomy.[3,4]

We report the case of a 59-year old patient who underwent surgery for a Bismuth type IIIB hilar cholangiocarcinoma in September 2009. There were a number of complications after the intervention: ischaemic necrosis of the right hepatic lobe anterior segments, a biliary fistula and abscesses in the surgical site. The patient was therefore eventually

discharged wearing a drain in the surgical site. In January 2010, the patient was admitted due to acute renal failure with serum creatinine of 9.6mg/dl, vomiting and increased cardiac output due to drainage. Urine sodium was 34meq/l. Additional tests also highlighted the presence of a proteinuria of 0.6g/24h and urine sediment with 10 red cells per field without leukocyturia. The patient had a beta-2-microglobulin level of 548.72µg/g and NAG of 9U/l in the urine. The renal function continued to deteriorate despite volume replacement and a renal biopsy was performed to investigate the origin of the acute renal failure.

The renal biopsy showed varying degrees of shrinkage with fibrous thickening of the capsule in some glomeruli. Diffuse fibrosis was observed in the interstitial area, with focal tubular atrophy affecting approximately 25% of the parenchyma. Predominantly lymphoplasmacytic inflammatory infiltrate was detected in various areas, with occasional presence of eosinophils. Some tubules had necrotic cylinders in the lumen. The lumen of many tubules was occupied by birefringent crystals consistent with oxalate (Figure 1).

The oxalate in urine was 47.70mg/24h (up to 40mg/24h with normal renal function). After hydration, correction of acidosis, treatment with calcium carbonate and low oxalate diet, the patient presented a successful evolution, with a progressive decline in serum creatinine to 2mg/dl prior to discharge.

Under normal conditions, the daily load of endogenous and exogenous oxalate is completely excreted by the kidneys. When renal function is altered, renal and extrarenal deposits of oxalate begin to appear, which is known as systemic oxalosis. When there is a high oxalate load, hyperoxaluria is produced, increasing the risk of nephrolithiasis and nephrocalcinosis. In addition, acute renal failure may be triggered in patient with a precipitating factor, such as dehydration and/or metabolic acidosis.

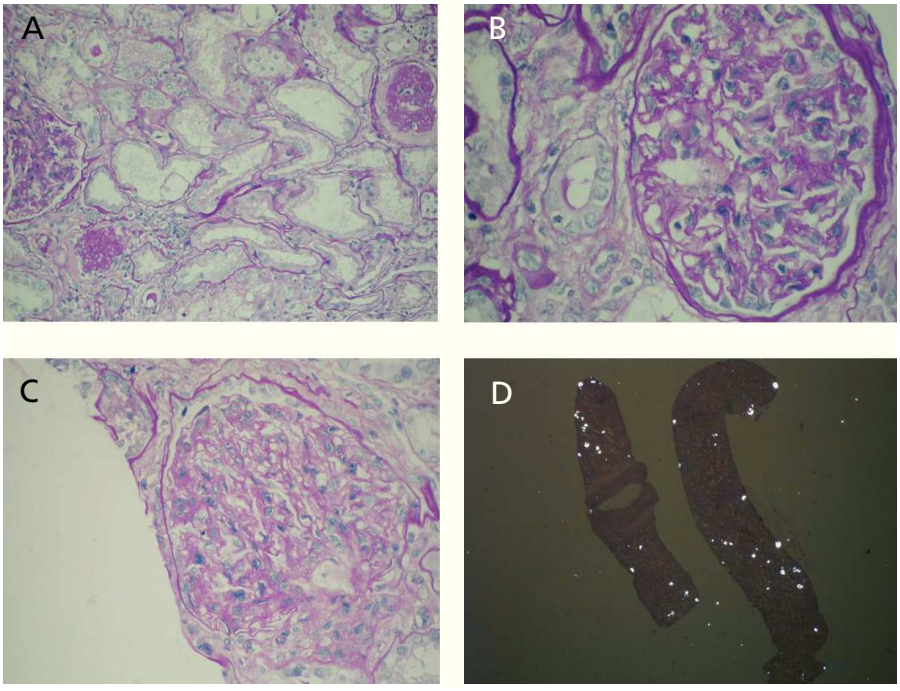

Figure 1. Renal biopsy. 1A) Diffuse interstitial fibrosis with focal tubular atrophy and interstitial foci of inflammatory infiltrate; 1B) Glomerulus with fibrous thickening of the capsule; 1C) Expansion and mesangial sclerosis; 1D) Tubules with lumen occupied by birefringent crystals consistent with oxalate

Hyperoxaluria is defined as the presence of urinary oxalate values greater than 40mg/day. It is frequently observed in patients with fat malabsorption due to digestive hyperabsorption of oxalate, unlike primary hyperoxaluria due to enzyme deficiencies associated with a hyperproduction of oxalate in the liver.[5]

The patient in question presented an enteric hyperoxaluria in relation to malabsorption of fats. The main mechanism involved is the binding of calcium in the intestine by fatty acids, which decreases the calcium oxalate in the digestive tract and increases the ionised oxalic acid absorbed in the intestine. These patients may benefit from conservative treatment based on reducing oxalate and fat in the diet, the administration of calcium carbonate as an oxalate binder, and an increased intake of fluids and administration of bases, such as sodium citrate, in order to increase urinary calcium oxalate solubility. Likewise, it has yet to be confirmed if recolonisation with *Oxalobacter formigenes* also reduces urinary oxalate excretion.[6]

In conclusion, calcium oxalate deposition associated with enteric hyperoxaluria is a rare cause of acute renal failure in the native and transplanted kidney.[5,7,8] Patients with fat malabsorption are at high risk and should be identified and treated early to prevent loss of kidney function. If parenchymal acute renal failure is seen in a patient with fat malabsorption, urinary oxalate excretion should be measured, deterioration of the acid-base balance reversed and early hydration provided.

1. Perazella MA. Crystal-induced acute renal failure. Am J Med 1999;106(4):459-65.
2. Swartz RD, Wesley JR, Somermeyer MG, Lau K. Hyperoxaluria and renal insufficiency due to ascorbic acid administration during total parenteral nutrition.Ann Intern Med 1984;100:530.

3. Wharton R, D'Agati V, Magun AM, Whitlock R, Kunis CL, Appel GB. Acute deterioration of renal function associated with enteric hyperoxaluria. Clin Nephrol 1990;34(3):116-21.
4. Mandell I, Krauss E, Millan JC. Oxalate-induced acute renal failure in Crohn's disease. Am J Med 1980;69(4):628-32.
5. Lefaucheur C, Hill GS, Amrein C, Haymann JP, Jacquot C, Glotz D, et al. Acute oxalate nephropathy: A new etiology for acute renal failure following nonrenal solid organ transplantation. Am J Transplant 2006;6(10):2516-21. Epub 2006 Aug 1.
6. Marengo SR, Romani AM. Oxalate in renal stone disease: the terminal metabolite that just won't go away. Nat Clin Pract Nephrol 2008;4(7):368-77. Epub 2008 Jun 3.
7. Rankin AC, Walsh SB, Summers SA, Owen MP, Mansell MA. Acute oxalate nephropathy causing late renal transplant dysfunction due to enteric hyperoxaluria. Am J Transplant 2008;8(8):1755-8. Epub 2008 Jun 28.
8. Cuvelier C, Goffin E, Cosyns JP, Wauthier M, De Strihou CY. Enteric hyperoxaluria: a hidden cause of early renal graft failure in two successive transplants: spontaneous late graft recovery. Am J Kidney Dis 2002;40(1):e3.

A. Sentís, L.F. Quintana, E. Massó, N.S. Peréz, A. Botey Puig, J.M. Campistol Plana
Nephrology and Renal Transplant Department. Clínic i Provincial Hospital. Barcelona, Spain.
Correspondence: Luis Quintana Porras
Servicio de Nefrología y Trasplante Renal. Hospital Clínic i Provincial. Villaroel, 170. 08036 Barcelona. Spain.
lfquinta@clinic.ub.es

Foetal hyper-echogenic colon as an early sign of cystinuria

Nefrologia 2011;31(1):123-4
doi:10.3265/Nefrologia.pre2010.Sep.10636

To the Editor,

Cystinuria is a hereditary disease caused by a defect in the renal and intestinal tubular transport affecting cystine and the dibasic amino acids (lysine, ornithine and arginine).[1] It is transmitted as an autosomal recessive disorder and has a prevalence of about 1 in 7,000 live births, with a wide geographical variation and no predominance of sex. The clinical manifestations are effectively nephrolithiasis and its consequences (colic, haematuria, etc.) which usually occur in the second or third decades of life, although they can appear as early as the first year. It is the cause of 6%-10% of paediatric urolithiasis cases.[2] The cystine stone formation is due to the excessive concentration of this amino acid in urine and its high insolubility, especially when the urine is acidic.

We had the opportunity of studying a child, currently three years old, who was referred by his paediatrician when he was five months old, after an episode of gross haematuria, which revealed the presence of a stone in the nappy. It was the first child of non-consanguineous parents, without any previous significant pathology, but with a history of renal colic on the paternal side of the family. Ultrasound foetal studies during pregnancy revealed a colon hyperechogenicity without other intestinal abnormalities (Figures 1 and 2), and a slightly increased nuchal luminescence, with no other findings of interest. As a result, a sweat test was performed at birth to rule out cystic fibrosis and the result was normal.

Subsequent ultrasound images revealed multiple bilateral stones, which grew to a diameter of 1.4cm. Persistently high cystine elimination was detected in the urine (maximum 656mg/g creatinine at 7 months old). The renal glomerular function is normal (serum creatinine 0.28mg/dl), although there was a defect in the ability to concentrate (689mOsm/kg) and elevated urinary excretion of microalbumin (microalbumin/creatinine ratio 33.9µg/µmol).

During its evolution, numerous small stones have been expelled (over 50

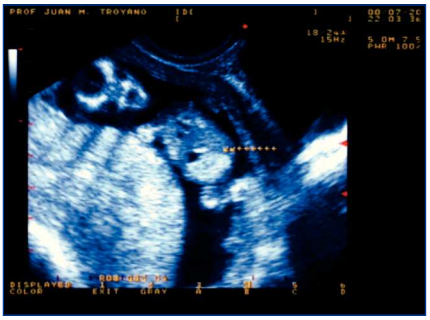

Figure 1. Hyperechogenic intestine with sound density similar to foetal bone

during the first year of life, measuring few mm in diameter), and the condition is otherwise asymptomatic. The weight-to-height ratio and psychomotor development during growth was normal. Pharmacological and dietary treatment with potassium citrate, captopril and D-penicillamine is currently being administered.

This is an early clinical presentation of cystinuria, reflecting the high lithogenic capacity of this condition. The particularity of the case is that the prenatal ultrasound found hyperechogenicity of the colon secondary to cystine crystal deposition. This form of presentation of cystinuria was described in 2006[3] and was subsequently confirmed.[4] The explanation for this finding is that the cystine crystals are formed in the foetal kidney, they enter the amniotic fluid and are then swallowed. The ultrasound finding of the foetal hyperechogenic colon has been traditionally related to cystic fibrosis,

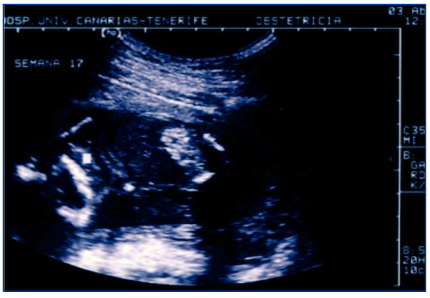

Figure 2. A similar situation early in the second trimester