A 72-year-old patient presented with a history of chronic liver disease (of probable alcoholic origin) with occasional edematous decompensation and several digestive bleeding episodes, colon diverticuli, pericardiectomy due to constrictive pericarditis, and chronic renal failure (of indeterminate origin) with the start of peritoneal dialysis in 2003.

The patient was admitted in October 2007 due to sepsis of urinary origin – the causal microorganism being *E. coli*.

During admission, abdominal ultrasound showed small kidneys, with no other anomalies. Treatment was provided in the form of amoxicillin-clavulanate for three weeks, the patient remaining at home without clinical symptoms.

In December 2007 he was admitted to the Service of Nephrology due to pain in the left inguinal region and leukocytosis (35,000). The patient was without fever, hemodynamically stable and presented no other symptoms. Exploration of the affected zone revealed no alterations other than pain in response to pressure. Empirical antibiotic treatment was started with ciprofloxacin, and emergency ultrasound of the inguinal region revealed the presence of an abscess measuring 3 cm in size. After 24 hours, the patient presented abdominal pain and turbid dialysis fluid. An emergency abdominal CAT scan was thus decided, revealing (fig. 1) a water-fluid level accumulation in the retroperitoneum and left renal fossa, and abundant gas bubbles occupying the region of the psoas muscle, which appeared atrophic. The accumulation was seen to descend between the muscles of the root of the left thigh, with an abundant presence of gas.

In view of the CAT findings, emergency surgery was decided under broad-spectrum antibiotic coverage (piperacillin-tazobactam plus metronidazole), performing a left lumbotomy and drainage of the abscess. The peritoneal dialysis catheter was removed, and the patient was kept under continuous venous-venous hemodiafiltration.

The urine and peritoneal dialysis fluid cultures proved positive for *E. coli* sensitive to the previously administered antibiotic treatment.

Six days later, and in view of clinical worsening of the patient, repeat surgery was carried out to remove the left kidney and perform retroperitoneal debridement.

However, deterioration was progressive, and the patient died after 24 hours.

Emphysematous pyelonephritis is an infrequent acute disease characterized by the presence of gas within the renal parenchyma, and presents a high percentage mortality.

The condition is generally unilateral and affects patients with known risk factors, such as diabetes mellitus or urinary tract obstruction – though it has also been reported that the use of dextrose in the peritoneal dialysis fluid may be a risk factor for infections of this kind.[1]

In most cases the isolated germ corresponds to *E. coli*.[2]

Treatment is the subject of controversy. Percutaneous drainage should be considered initially, and particularly in critically ill patients. With such treatment a lesser percentage mortality has been reported compared with emergency nephrectomy. However, subsequent kidney removal may prove necessary in some cases.[3,4]

1. Anwar N, Charla LS, Lew SQ. Emphysematous pyelitis presenting as an acute abdomen in an end-stage renal disease patient treated with peritoneal diálisis. *J Urol* 2003; 169 (6): 2422-3.
2. Czaja CA, Scholes D, Hooton TM, Stamm WE. Population-based epidemiologic analysis of acute pyelonephritis. *Clin Infect Dis* 2007; 45: 273.
3. Somani BK, Nabi G, Thorpe P, Hussey J, N´dow J, ABACUS Research Group. Is Percutaneous Drainage the New Gold Standard in the Management of Emphysematous Pyelonephritis? Evidence from a Systematic Review. *J Urol* 2008 Mar 17.
4. Aswathaman K, Gopalakrishnan G, Gnanaraj L, Chacko NK, Kekre NS, Devasia A. Emphysematous Pyelonephritis: Outcome of Conservative Management. *Urology* 2008 Mar 25.

M. P. Borrajo Prol, C. Pérez Melón, J. Santos Nores and M. Camba Caride
Servicio de Nefrología. Complexo Hospitalario de Ourense.
Correspondence: *Cristina Pérez Melón. cristicpm@hotmail.com. Complexo Hospitalario de Ourense. C/ Ramón Puga, 54. 37005 Orense. España.*

Usefulness of prolonged haemodialysis in acute methanol poisoning

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To the editor: The case of a patient with severe methanol and toluene poisoning has been recently published in Nefrología.[1] We have treated a patient with severe methanol poisoning and we want to emphasize the importance of prolonged haemodialysis in its treatment.

CASE REPORT

This was a 40-year-old male patient who was brought into the Emergency Department after suffering frfom severe headache, dizziness, irritability and incoherent language. According to the family, the patient had chronic alcoholism although he had not had any alcohol in the last 3 days. In the emergency room, there was deterioration in level of consciousness with progressive coma, so the patient was admitted to the ICU where orotracheal intubation, and mechanical ventilation was used.

Laboratory analysis on admission showed an arterial blood gas with a pH of 6.98, PO_2 96, PCO_2 31, and bicarbonate 4.9 mEq/l. Plasma creatinine was 1/53 mg/dl, BUN 33 mg/dl, sodium 135 mEq/l, potassium 6.2 mEq/l, chloride 102 mEq/l, haemoglobin 16 g/dl, glucose 198 mg/dl, serum osmolarity 421 mOsm/kg and lactic acid 10.5 mmol/l. A head CT did not reveal any visible parenchymal or signs of cerebral haemorrhage.

After questioning the family again, the possibility that the patient had consumed approximately half a litre of methanol was mentioned.

Treatment was initiated with pyridoxine, thiamine, nalaxone, tiapride, flumazenil, sodium bicarbonate, inotropics and intravenous ethanol.

Haemodialysis began approximately 3 hours after admission to ICU. A high-

permeability 2.1 m^2 surface area polysulfone membrane and a bicarbonate bath for 7 hours was used with blood flow at 300 ml/min. Unfortunately, despite continuing inotropics and high-dose bicarbonate, the patient suffered from severe haemodynamic instability. Due to the persistent deep coma without response to stimuli, brain CT was repeated and demonstrated cerebral oedema and signs of transtentorial herniation that did not respond to treatment. Faced with a situation of brain death, authorisation for organ donation was requested and was given.

Later, the methanol levels from before haemodialysis (1,793 mg/l) and afterwards (173.4 mg/dl) were made available.

DISCUSSION

Acute methanol poisoning should be suspected in all patients with metabolic acidosis with an elevated anion gap, neurological deterioration or vision loss.[2-4] Although methanol does not cause significant direct intoxication, it is transformed by the liver to formaldehyde and formic acid with leads to metabolic acidosis and cases damage to all levels of the brain and the optic nerve.[2-4] Mortality from methanol poisoning is high. In a recent study, it has been established that it is much greater in patients with a blood pH below 7, coma upon admission, delay in seeking medical care and elevated plasma methanol levels.[5,6] Treatment is based on inhibition of the alcohol dehydrogenase enzyme, correction of metabolic acidosis and elimination of toxic metabolites by dialysis. Enzyme inhibition can be achieved with fomepizole or, when this is not immediately available, with ethanol.[7] Finally, haemodialysis with high-permeability membranes at a high flow rate for a prolonged period is capable of reducing methanol levels as occurred in our patient.[8]

Unfortunately, many cases lead to brain death and are potential multiorgan donors, given that many publications have demonstrated that the viability of the organs is adequate.[9]

1. Santana L, García Cantón C, Martínez Cuéllar S, Sánchez Palacios M. Intoxicación grave por metanol y tolueno: a propósito de un caso. *Nefrología* 2007; 27 (4): 517-8.
2. Hovda KE, Hunderi OH, Rudberg N, Froyshov S, Jacobsen D. Anion and osmolal gaps in the diagnosis of methanol poisoning: clinical study in 28 patients. *Intensive Care Med* 2004; 30 (9): 1842-6.
3. Hovda KE, Hunderi OH, Tafjord AB, Dunlop O, Rudberg N, Jacobsen D. Methanol outbreak in Norway 2002-2004: epidemiology, clinical features and prognostic signs. J *Intern Med* 2005; 258 (2): 181-90.
4. Sefidbakht S, Rasekhi AR, Kamali K, Borhani Haghighi A, Salooti A, Meshksar A et al. Methanol poisoning: acute MR and CT findings in nine patients. *Neuroradiology* 2007; 49 (5): 427-35.
5. Hassanian-Moghaddam H, Pajoumand A, Dadgar SM, Shadnia S. Prognostic factors in methanol poisoning. *Hum Exp Toxicol* 2007; 26 (7): 583-6.
6. Paasma R, Hovda KE, Tikkerberi A, Jacobsen D. Methanol mass poisoning in Estonia: outbreak in 154 patients. *Clin Toxicol (Phila)* 2007; 45 (2): 152-7.
7. Mycyk MB, DesLauriers C, Metz J, Wills B, Mazor SS. Compliance with poison center fomepizole recommendations is suboptimal in cases of toxic alcohol poisoning. *Am J Ther* 2006; 13 (6): 485-9.
8. Hunderi OH, Hovda KE, Jacobsen D. Use of the osmolal gap to guide the start and duration of dialysis in methanol poisoning. *Scand J Urol Nephrol* 2006; 40 (1): 70-4.
9. Mora-Ordóñez JM, Martín D, Curiel Balsera E, Muñoz Bono J. Intoxicación mortal por metanol: donante de órganos. *Med Clin* 2008; 130 (1): 37-9.

G. de Arriba, M. Torres-Guinea, J. Chevarría and M. A. Basterrechea

Servicio de Nefrología. Hospital Universitario de Guadalajara. Departamento de Medicina. Universidad de Alcalá.

Correspondence: *Gabriel de Arriba. garribad@senefro.org. Hospital Universitario de Guadalajara. Donante de Sangre, s/n. 19002 Guadalajara. España.*

Persistent severe hyperkalemia treated with a continuous infusion of calcium gluconate

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To the editor: Intravenous calcium is used in the treatment of severe hyperkalemia with cardiac impact because it antagonizes the action of potassium on the cell membrane, although it does not reduce the serum potassium level. In general, it is used in intermittent doses for 30-60 minutes and it buys time until other conservative measures take effect or until haemodialysis is available. We describe a patient with severe hyperkalemia treated with a continuous infusion of calcium gluconate.

A 79-year-old woman came in due to reduced dieresis and lower extremity weakness. Five days prior to arrival, a cardiac catheterisation has been made with placement of 2 stents. The medical history included: ischemic heart disease with 3 myocardial infarctions, dyspnea on minimum effort, diabetes mellitus, chronic renal failure (baseline creatinine 1.5-2 mg/dl), hypertension under treatment with ramipril, obesity and polyarthrosis. Physical examination: BP 130/60 mmHg, afebrile, CA: rhythmic at 60 bpm; PA: rhonchi and isolated crepitants, generalized oedema. Laboratory results: haemoglobin 8.7 g/dl, glucose 173 mg/dl, BUN 249 mg/dl, creatinine 9.31 mg/dl, normal CK and troponin-I, sodium 124 meq/l, potassium 8.89 meq/l, pH 7.3, bicarbonate 17.3 meq/l; following bladder catheterisation, a scant amount of urine was recovered, the analysis of which revealed: SG 1.005, urine protein 30-70 mg/dl, sediment: 4-6 RBC/hpf, leukocyturia. ECG: wide QRS complexes measuring 160-200 milliseconds a 60 bpm and absent P waves. Renal ultrasound: kidneys of normal size without ectasia. Chest x-ray: vascular redistribution. The patient and the family were informed of the severity of the situation and the possible need for dialysis. The family refused haemodialysis and requested conservative treatment that does not cause suffering to space out laboratory testing. The patient was initially administered seguril 250 mg bolus and 20 ml of 10% calcium gluconate over 30 minutes. Later, she was treated with 24-hour continuous infusions of seguril 250 mg, 500 ml of D10W with 10 U of rapid-acting insulin and 250 ml of D5W with 60 ml of 10% calcium gluconate plus oral Resonium®. A day later, dieresis was 500 ml,