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]

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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,

potassium 8.59 meq/l, total calcium 9.63 mg/dl; the ECG showed narrow QRS complexes; the treatment ordered was continued. Subsequent progress was good with a progressive increase in dieresis and a reduction in creatinine and potassium. After 3 weeks of hospitalisation, the creatinine was 1.72 mg/dl and the potassium was 4 meq/l.

Once haemodialysis was ruled out in our patient, we considered different conservative measures for her situation. Hypertonic glucose with insulin temporarily reduces serum potassium by facilitating cell uptake. There are different guidelines, but hypoglycaemia is not uncommon, which is why infusion of hypertonic glucose solution at a variable rate (50-75 ml/h)[1-3] is recommended after initial treatment, a method that could have exacerbated the patient's hypervolaemia. Sodium bicarbonate can also cause volume overload and its use is also controversial.[1,2] β2-antagonists can produce tachyarrhythmia and lead to myocardial ischemia at the dosages needed to reduce potassium.[1] In this patient with a significant history of ischemic heart disease, hypervolaemia and anuria, we considered to be less of a risk to use continuous infusion of calcium and wait for restoration of dieresis.

Intravenous calcium antagonises the effects of hyperkalaemia on the heart; on the one hand, it reduces threshold potential electronegativity, restores the difference between the membrane and reduces myocyte excitability; on the other hand, it increases the maximum velocity of the action potential and improves cardiac conduction.[4] It has been suggested that continuous calcium infusion will lead to more stable levels and better results than intermittent dosages.[5]

In summary, continuous calcium infusion is a therapeutic option that may offer advantages over other conservative treatment measures in extreme patients with severe hyperkalaemia.

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