A 59-year-old man with diabetes mellitus Type II, arterial hypertension and depressive disorder was hospitalized due to unexplained severe inebriation followed by sedation. Regular medications consisted of metformin 1500 mg, enalapril 20 mg and paroxetine 20 mg.

Blood tests showed a creatinine of 0.9 mg/dL (79 µmol/L), glucose 110 mg/dL (6.1 mmol/L) and potassium 6.4 mmol/L. Blood gas test showed a pronounced metabolic acidosis, with pH 7.04, bicarbonate 3 mmol/L, PCO2 10 mmHg, lactic acid 4.8 mmol/L, Na 145 mmol/L, chloride 105.4 mmol/L and anion gap 43 mmol/L.

Cerebrospinal fluid analysis and cerebral computerized tomography scan were normal. The patient's condition, including cognitive status, was rapidly deteriorating, requiring orotracheal intubation. Anuria developed in the following few hours, requiring the referral to our hospital.

Laboratory data confirmed the high anion gap metabolic acidosis and showed a serum creatinine of 3.8 mg/dL (336 µmol/L). Renal replacement therapy was soon initiated with continuous venovenous haemodiafiltration; after 21 h acid–base status was normalized, but anuria persisted requiring intermittent haemodialysis.

Examination of urine sediment (Figure 1) showed both needle-shaped and dumbbell-shaped calcium oxalate monohydrate crystals; these crystals are considered a clue to ethylene glycol poisoning since oxalate derives from the metabolism of ethylene glycol [1, 2]. Furthermore, renal damage most likely results from a crystal-induced injury to proximal tubule cells [3]. The coma lasted 2 weeks; upon regaining consciousness the patient admitted to having ingested automobile anti-freeze (containing ethylene glycol) in a suicide attempt.

Progressive recovery of renal function occurred 4 weeks after admission; renal replacement therapy was interrupted 2 weeks later.

A prompt examination of urine sediment, often forgotten in the current ‘high-tech’ medicine, would have suggested the correct diagnosis and allowed administration of fomivazol to prevent brain and renal damage.

Conflict of interest statement. None declared.

References


Received for publication: 14.11.11; Accepted in revised form: 16.11.11