Urine ‘picket fence’ crystals in ethylene glycol poisoning

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ABSTRACT

A 46-year-old woman intentionally ingested ethylene glycol and overdosed on paracetamol. She had clinical and laboratory features suggestive of ethylene glycol poisoning, and examination of the urine revealed calcium oxalate monohydrate, or ‘picket fence’, crystals. She responded well to therapy that included haemodialysis. Clinicians should be aware that these crystals appear late during the evolution of ethylene glycol poisoning and, along with other clinical and laboratory findings, should prompt the initiation of haemodialysis.

Keywords: ethylene glycol; calcium oxalate monohydrate; crystals; poisoning.

CASE PRESENTATION

A 46-year-old woman with a history of depression presented to the emergency department (ED) 12 hours following an overdose of 25 g of paracetamol tablets and 500 mL of anti-freeze. On examination, her blood pressure was 110/75 mmHg, pulse rate was 80 beats per minute, body weight was 85 kg and she was afebrile. Other than Kussmaul breathing, the rest of the clinical examination was unremarkable. Her random blood glucose was 13.2 mmol/L and a urine dipstick was negative for ketones. Arterial blood gas revealed metabolic acidosis with a pH of 7.13, bicarbonate of 10.4 mmol/L and a lactate concentration of 10.6 mmol/L. The serum potassium concentration was 4 mmol/L and creatinine was 38 µmol/L. The calculated anion gap (AG) was high at 22 mmol/L. Urine microscopy showed calcium oxalate monohydrate crystals, or ‘picket fence’ shaped crystals, exclusively (Figure 1). A diagnosis of ethylene glycol poisoning (EGP) and paracetamol overdose was made. The patient was started on 70% ethanol in 5% dextrose water via nasogastric tube over a 10-hour period, as well as N-acetylcysteine by intravenous infusion. As a result of the severe metabolic acidosis, haemodialysis was initiated. She responded well to treatment and made a complete recovery. She was subsequently transferred to our psychiatric services for further evaluation and care.

EGP is frequently encountered in the ED, usually following intentional or accidental ingestion. It is found in brake fluid or anti-freeze [1]. A lethal volume of ethylene glycol is 1.4–1.5 mL/kg of body weight [1]. Following ingestion, it is converted into glycolic acid by hepatic alcohol and aldehyde dehydrogenases [2]. In the ED, clinical clues include a history of toxin ingestion, and examination may reveal inebriation or coma and Kussmaul breathing [2]. Laboratory clues include severe hyperkalaemia in the presence of mild kidney failure, a high osmolar gap (OG) with a normal AC during the early stages, followed later by a mildly elevated/absent OG and a high AG as the ethylene glycol is converted into glycolic acid. The latter results in a falsely elevated serum lactate concentration. Calcium oxalate crystals may be found on urine examination. Crystalluria may persist for up to 40 hours, depending on the presence or absence of kidney failure [1]. The type of calcium oxalate crystals changes over time. During the initial 4–5 hours, calcium oxalate dihydrate, or ‘envelope-shaped’, crystals may appear; at 5–7 hours a combination of monohydrate and dihydrate crystals may...
be found, and after more than 7 hours only calcium oxalate monohydrate, or ‘picket fence’, crystals will be present [1]. Therefore, the latter is a late finding during the evolution of EGP.

General therapeutic principles include the use of ethanol or fomepizole and extracorporeal therapy such as intermittent haemodialysis. Ethanol and fomepizole inhibit the enzymes alcohol dehydrogenase and aldehyde dehydrogenase, thus preventing the conversion of toxic alcohols into their toxic metabolites. Since toxic alcohols have a small volume of distribution, low protein binding and molecular weight, they are easily removed during haemodialysis [1]. The American Association of Clinical Toxicology recommends haemodialysis when there is clinical deterioration despite intensive supportive care, severe metabolic acidosis with pH < 7.25–7.30, and kidney failure or electrolyte disorders not responding to medical management [3]. Equations for predicting the duration of the haemodialysis session have been formulated [4]. In our patient, the decision to initiate dialysis was based on the large volume of ethylene glycol ingested, the severity of the patient’s metabolic acidosis and the pre-sence of ‘picket fence’ crystals on urine microscopy.

Clinicians should be aware that the exclusive appearance of calcium oxalate monohydrate, or ‘picket fence’, crystals is a late finding during the evolution of ethylene glycol poisoning. Along with other clinical and laboratory findings, they should prompt the initiation of haemodialysis.

**Ethical considerations**

Consent to publish this case report was granted by the Health Research Ethics Committee of Stellenbosch University (reference number C21/06/015, project ID 22087).

**REFERENCES**


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**Figure 1.** Urine microscopy after spinning at 2000 rpm for 1 minute, showing calcium monohydrate ‘picket fence’ crystals. 10x and 40x high-power field magnification.