The Interesting Case

Ethylene glycol intoxication in a dipsomaniac patient

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Ethylene glycol (EG) belongs to the group of dihydric alcohols, a colourless, odourless, sweet tasting fluid. It is used for production of antifreeze and windscreen cleaner and it is also an effective solvent for many dyes. Furthermore it is used as a softener for cellophane, as replacement for glycerol and is contained in numerous cosmetics, shoe polish and lubricants. Ingestion of EG in suicidal intention or as substitute for alcohol causes intoxication with severe metabolic acidosis as the presenting sign [1].

It has been stated in literature that survival is exceptional in cases where arterial pH is below 6.8 [2]. In the following we describe a case with most massive acidosis who survived despite nondemonstrable bicarbonate. The case highlights some of the problems of diagnosis and management.

Case report

At ~11 a.m., a 47 year old female comatose patient was admitted to the hospital via emergency helicopter. She had a history of thyroidectomy, borderline hypertension and dipsomania in temporal conjunction with depressive states.

According to the patient’s relatives she had consumed a large amount of alcohol over a period of a few days. Her partner had taken the precaution of hiding the remaining supplies of alcohol. It is assumed the patient awoke at ~4.30 a.m. to go to the toilet. A few hours later she was found in a comatose state from which she could not be aroused. Breathing was fast and deep.

On admission the patient was comatose, hyperventilating and hypothermic (34.5°C) with no reaction to painful stimuli. The pupils were wide and reacting slowly, the reflexes were difficult to elicit and Babinski’s sign was negative. There was no meningism. Blood pressure was 130/70 mmHg and heart rate 110–140/min without arrhythmia.

Pertinent laboratory findings included: WBC: 27 100/μl; s-K: 6.59 mmol/l; s-GPT: 26 U/l; s-Na: 141 mmol/l; s-Cl: 106 mmol/l; s-Ca: 8.3 mg/dl; s-creatinine: 0.79 mg/dl; glucose: 188 mg/dl (during i.v. glucose 5% infusion); blood gas examination: pH 6.84, pO2 161 mmHg (under oxygen insufflation); pCO2, HCO3 and BE not measurable. The urine sediment showed excessive amounts of calcium oxalate crystals. All other laboratory tests were in normal range. Cranial CT showed no haemorrhage or oedema. Sonography of abdomen and kidneys did not reveal any abnormalities.

Hyperventilation with acidosis (pH 6.8), normochloraemia and elevated anion gap suggested ingestion of a toxic alcoholic substance. Ethanol was not detected in the blood, but abundant calcium oxalate crystals were noted in the urine sediment. This constellation lead to the suspicion of EG-poisoning which was later confirmed by documentation of high EG concentration in the urine (courtesy of the forensic department).

Immediately after admission the patient received infusions of Na-bicarbonate i.v. (cumulative amount 1500 mmol). Nevertheless acidosis could not be completely corrected. In addition ethanol was infused. The patient suffered convulsions. This prompted us to perform bicarbonate haemodialysis for 7 h. The patient became somnolent and could be aroused. Although haemodialysis treatment was continued and the patient’s condition improved, during the next day the condition of the patient worsened, since pulmonary oedema and brain swelling developed. Decreased urine output developed and the patient required a total of 17 haemodialyses for a period of 6 weeks. Thereafter she made a complete recovery. To date the patient’s condition is stable with normal levels of s-creatinine and an endogenous creatinine clearance of 140 ml/min.

Discussion

When ethyleneglycol (EG) is ingested accidentally or in a suicide attempt, the toxicological range is very narrow. Ingestion of as little as 1.5 g/kg b.w. can be

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lethal. EG is quickly absorbed from the intestine and the peak concentration is reached after 2 h [1].

The clinical manifestations include an initial state of narcosis, similar to alcohol intoxication, followed by signs pointing to problems in the central nervous system (convulsions after 30 min to 2 h), cardiopulmonary problems (after 12–24 h) and finally oliguric renal failure (after 24–72 h) [3]. Most patients die early from severe metabolic acidosis [4].

Ethyleneglycol itself exerts no toxicity, but it is metabolized via glycolaldehyde and glycolic acid to oxalate. The former accounts for much of the toxicity and for metabolic acidosis, the latter for renal damage. Although oxalate makes up no more than 2% of EG metabolites, it is important because of its role in inducing renal failure [5]. Furthermore, oxalate crystals in leptomeningeal vessels may cause 'chemical leptomeningitis' [3].

The diagnosis rests on clinical suspicion and is further supported by the findings of an elevated anion gap [6] and osmotic gap [7]. The problem of the anion gap has recently been discussed in these pages [8]. The differential diagnosis of an increased anion gap includes diabetic and alcoholic ketoacidosis, uraemia, lactic acidosis and—important in this context—intoxication with other agents such as methanol, salicylate, or ethyleneglycol [6,9]. Suspicion of the latter is further confirmed by a high osmolar gap [5]. In our case the gap reached 55 mosmol/kg.

Treatment consists of general intensive care treatment, reversal of metabolic acidosis by sodium bicarbonate infusion and bicarbonate hemodialysis (which concomitantly eliminates metabolites of ethyleneglycol) as well as alcohol infusion (to inhibit hepatic alcoholdehydrogenase and thus delay metabolism of ethyleneglycol [4,5]).

Conclusion

Although ethyleneglycol intoxication is rare, this possibility should be considered in all comatose patients presenting with severe acidosis. Prompt treatment is necessary, because prognosis is poor unless treatment is started early on. The diagnosis is suggested by a normochloremic anion gap by metabolic acidosis combined with a high osmolar gap, and by the demonstration of oxalate crystals in the urinary sediment.

References

3. Steininger H, Thierauf P. Ethylenglycolvergiftung. DMW 1988, di/DC2erential diagnosis of an increased anion gap includes diabetic and alcoholic ketoacidosis, uraemia, lactic acidosis and—important in this context—intoxication with other agents such as methanol, salicylate, or ethyleneglycol [6,9]. Suspicion of the latter is further confirmed by a high osmolar gap [5]. In our case the gap reached 55 mosmol/kg.