Reminder of important clinical lesson

Ethylene glycol ingestion masked by concomitant ethanol intoxication

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Correspondence to Mr Justin M Head, OMS-III, justin.head@lmunet.edu**Summary**

An obtunded male with a history of alcohol abuse presented to the emergency department with metabolic acidosis, an osmolar gap and lactic acidosis. The patient was initially treated for alcohol intoxication due to an extremely high blood alcohol level. Following respiratory failure and intubation, a large volume of dark green liquid was removed via nasogastric suction; bedside fluorescence for ethylene glycol was negative. Twenty-four hours later, the patient's glomerular filtration rate decreased significantly, serum osmolality was 807, the osmolar gap was 407, complete metabolic panel showed pH of 6.8, sodium of 156 mmol/l, potassium of 7.3 mmol/l, chloride of 116, CO₂ of 3.9 and anion gap of 30.7. Blood lactic acid was >56 mmol/l. The patient received emergency haemodialysis. Four days after presentation, the patient began to respond to voice commands and was extubated. Currently, the patient still receives haemodialysis due to ongoing renal failure, but no long-term neurologic complications are evident.

BACKGROUND

Ethylene glycol is a common industrial solvent responsible for accidental, suicidal and epidemic poisonings or may be ingested as a substitute to ethanol. Ethylene glycol metabolites result in acute renal failure via reversible oliguric or anuric kidney injury, resulting in slowed elimination of ethylene glycol.¹ Intoxication is associated with a profound anion gap metabolic acidosis that directly correlates with the accumulation of toxic metabolites.²

Intoxication of ethylene glycol typically follows three, often concurrent, phases: neurological phase, cardiopulmonary phase and renal phase.³ In the first 12 h after ingestion, inebriation and euphoria are followed by profound central nervous system depression and possible coma, hypotonia and seizures.^{4 5} By 24 h, a compensatory hyperventilation can develop due to the severe metabolic acidosis and physical findings are consistent with congestive heart failure.^{5 6} During the second and third day following ingestion, oliguria and flank pain indicative of acute renal failure are present. Calcium oxalate crystals are formed from metabolites, resulting in hydronephrosis and hypocalcaemia.^{3 5}

Treatment of ethylene glycol intoxication includes correction of metabolic acidosis, alcohol dehydrogenase (ADH) blockade and removal of the alcohol and metabolites. These goals are accomplished by sodium bicarbonate administration, ethanol or fomepizole treatment, and haemodialysis.^{3 7} Prognosis typically depends on the extent of metabolic acidosis at presentation, with pH<7.00 being associated with a high mortality.⁸

The patient described in this case co-ingested ethanol and ethylene glycol, which provided a competitive inhibitor for alcohol dehydrogenase, thus serving to limit acute toxic sequelae while confounding the clinical picture of suspected ethanol intoxication.

CASE PRESENTATION

A 55-year-old male found obtunded and unresponsive in a motel room was brought to the emergency department for suspected acute alcohol intoxication. The patient had a documented history of alcohol abuse and numerous empty liquor bottles were found at the scene.

INVESTIGATIONS

Laboratory tests at presentation revealed a blood alcohol level of 371 mg/dl; arterial blood gas results showed pH of 7.27, pCO₂ of 34, pO₂ of 68 and pCO₂ of 15; lactic acid was 11.9 mmol/l, white blood cell count was within normal limits, and urinalysis ruled out infectious aetiology. An anion gap of 26.5 and serum osmotic gap of 370 was present. Serum calcium was low, at 7.9 mg/dl.

DIFFERENTIAL DIAGNOSIS

- ▶ Acute ethanol intoxication
- ▶ Acute ethylene glycol intoxication
- ▶ Acute methanol intoxication.

TREATMENT

Following intubation to maintain airway and secretions, the patient received judicious fluid replacement with normal saline. Upon nasogastric suction of 200 ml of a dark green liquid, a bedside fluorescence test for ethylene glycol was performed and showed a negative result. The patient was admitted to the intensive care unit to monitor renal function and continue treatment. Over the subsequent 24 h the patient's glomerular filtration rate (GFR) decreased from 117 to 43.8 ml/min, and creatinine increased from 0.70 to 1.64 mg/dl, and serum osmolality was 807 with a 407 osmolar gap. A complete metabolic panel showed a pH of 6.8, sodium of 156 mmol/l, potassium of 7.3 mmol/l,

chloride of 116, CO₂ of 3.9 and an anion gap of 30.7. Blood lactic acid was >56 mmol/l. A 2 meq/kg bolus of sodium bicarbonate followed by sodium bicarbonate in 1 L D5W ran at 200 cc/h to address the metabolic acidosis. Upon obtaining further history from the motel manager and housekeeper, it was discovered that the patient had been on a drinking binge for the previous 3 days and a bottle of antifreeze, with half of its contents missing, was found in the room. The patient received emergent haemodialysis but was given a poor prognosis due to the overall clinical picture. A 16-channel EEG showed severe diffuse cerebral dysfunction commonly observed in toxic metabolic or hypoxic abnormalities. Following daily haemodialysis and aggressive intravenous fluids, the patient began to respond to voice commands and was extubated 4 days after presentation.

OUTCOME AND FOLLOW-UP

At present, the patient is awake, oriented ×3 and openly communicates with little difficulty. He initially showed focal neurological signs, including past-pointing and mild ataxia, but these symptoms have since resolved. His pH has normalised, and his anion gap is 12.8. The long-term renal status of this patient remains questionable, as his calculated GFR is 8.5 ml/min/ssa, creatinine is 6.8 mg/dl and blood urea nitrogen is 34 mg/dl. He remains on haemodialysis and fluid management. Upon questioning, he denies suicidal ideation and does not recall ingesting ethylene glycol. Confirmation of ethylene glycol toxicity was provided by reference laboratory a week following presentation; the patient's serum ethylene glycol was 1067 mg/dl.

DISCUSSION

Ethylene glycol is a common industrial solvent responsible for accidental, suicidal and epidemic poisonings. The non-specific clinical signs and symptoms of ethylene glycol poisoning and the limited measurement capabilities of many hospitals make diagnosis difficult; in such situations, a combined metabolic acidosis and an osmolar gap is considered the hallmark of toxic alcohol intoxication.⁷ It has been known for some time that blood lactate levels increase in ethylene glycol intoxication⁹; recently Verelst¹⁰ described a case where glycolate, a metabolite of ethylene glycol, caused a falsely elevated lactate level in an ethylene glycol poisoning. The current case showed a lactic acid level elevating to >56 mmol/l with no other clinical explanations other than falsely elevated readings due to glycolate interference. Many facilities regularly use blood gas analysers to measure lactate levels, and these findings remind clinicians not to exclude ethylene glycol intoxication in patients with an elevated lactate.

Before the use of fomepizole, ethanol was widely used to competitively inhibit ADH, and it appears that this patient's co-ingestion of antifreeze with liquor may have actually been protective. His blood alcohol level of 371 mg/dl was well above the absolute ethanol level of 100 mg/dl to treat ethylene glycol intoxication and remained above 100 mg/dl for 36 h following admission. The

concomitant ingestion of ethanol provided an unintended competitive inhibitor of ADH and allowed the patient to survive an ethylene glycol level that could have been fatal. As long as the ethanol was on board, the ethylene glycol was renally eliminated unchanged,¹¹ but, once his blood ethanol level decreased, the toxic effects of the ethylene glycol were manifested. This highlights the importance of identifying clinical presentations and obtaining a thorough history as well as the confounding factors at play in cases of toxicity.

Given the patient presentation, the rapid clinical decompensation following admission, and the high mortality of metabolic acidosis below pH 7.0 secondary to ethylene glycol intoxication,⁸ this patient's recovery is quite remarkable. While he still receives haemodialysis, renal recovery is typically complete several months following toxic insult and no long-term neurological sequelae are expected.⁷

Learning points

- ▶ A combined metabolic acidosis and an osmolar gap is considered the hallmark of toxic alcohol intoxication.
- ▶ Ethylene glycol intoxication should be included in the differential diagnosis of cases with elevated lactic acid with no obvious aetiology.
- ▶ Co-ingestion of ethanol with ethylene glycol will mask initial toxicity and may be protective while blood ethanol level remains above 100 mg/dl.

Competing interests None.

Patient consent Obtained.

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