Ethylene glycol poisoning

A 22-year-old male presented to the emergency centre after drinking 300 ml of antifreeze. Clinical examination was unremarkable except for a respiratory rate of 28 bpm, GCS of 9 and slight nystagmus. Arterial blood gas revealed: pH 7.167, pCO_2 3.01 kPa, pO_2 13.0 kPa (on room air), HCO_3^- 15.2 mmol/l and lactate of 13.8 mmol/l. The calculated anion gap was elevated at 20.3. Ethylene glycol levels were not done as this test is not available to us. Blood chemistry and full blood count were normal.

Empiric treatment for acute ethylene glycol (EG) intoxication was begun with a 10% ethanol solution (loading dose and IV maintenance infusion), pyridoxine (100 mg) and thiamine (100 mg). Gradually the patient's mental status improved and wide anion gap metabolic acidosis resolved. He developed epigastric tenderness but gastroscopy was normal. The Psychiatry Department was consulted and admitted the patient because of ongoing suicidal ideation.

EG is water soluble and readily absorbed when ingested orally. EG is metabolised by alcohol dehydrogenase. The organic acid metabolites and calcium oxalate crystal precipitation produce the clinical syndrome of EG intoxication and end-organ damage (particularly renal) (Table I). Ethanol is a competitive inhibitor of alcohol dehydrogenase and this is the mainstay of treatment in our setting.

When a patient presents with signs of alcohol intoxication without the typical ethanol smell and a wide anion gap metabolic acidosis, ingestion of a toxic alcohol should be suspected and treatment instituted.

Further reading available at www.cmej.org.za

Table I. The stages of ethylene glycol poisoning

Stage	Features
1: CNS depression stage (0 - 4 hours)	* Gastric irritation: nausea, vomiting, haematemesis * Acute intoxication (without ethanol odour), euphoria, nystagmus, ophthalmoplegia, CNS depression
2: Cardiopulmonary complications (4 - 12 hours)	* Profound metabolic acidosis (accumulation organic acids): cardiac failure, acute respiratory distress syndrome, cerebral oedema * Hypocalcaemia (due to calcium oxalate precipitation): dysrythmias (prolonged QT), hyper-reflexia, muscle spasms, convulsions
3: Renal complications (>24 hours)	* Renal failure: Acute tubular necrosis, oliguria (due to direct glycolic acid damage to the tubules, obstruction of the tubules by calcium oxalate crystals)

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SINGLE SUTURE

Supermice run far and dodge diabetes

Faster, further, fatter? Knocking out a particular gene in muscle lets mice run twice as far as normal; knocking out the same gene in fat cells allows the animals to put on weight without developing type 2 diabetes.

The discoveries could lead to new treatments for diabetes or for people with wasting diseases, say Johan Auwerx and colleagues of the Federal Polytechnic School of Lausanne, Switzerland.

The team knocked out the gene for a protein called nuclear receptor corepressor 1 (NcoR1) in the muscles of mice. Without this gene, mitochondria keep working at full speed. 'The treated mice ran 1 600 metres in 2 hours, compared with 800 metres for untreated mice.'

Athletes shouldn't be tempted to try this, though, as knocking out the gene could have unpredictable side-effects. Another experiment reported in *Cell* gives an example: Auwerx found that knocking out NcoR1 in fat cells alone made the mice get fatter, but they didn't develop diabetes.

New Scientist 19 November 2011, p. 22.