P-06
Acute 1,4-butanediol ingestion can mimic toxic alcohol poisoning

Maurizio Stefani[1,2], Darren Roberts[1,2]
(1) Department of Clinical Pharmacology and Toxicology, St Vincent's Hospital, Sydney, Australia
(2) St Vincent's Clinical School, University of New South Wales, Sydney, Australia

Objective: Decreased level of consciousness with a raised osmol gap and high anion gap metabolic acidosis is highly suggestive of toxic alcohol exposure. We report a case of a patient with this clinical syndrome secondary to 1,4-butanediol (1,4-BD) intoxication.

Case Report: A 44 year-old male was found GCS 7/15 with a collateral history of ethanol plus gamma-hydroxybutyrate (GHB) ingestion within the hour. The patient had pulse 100/min, normotensive, afebrile, and respiratory rate 14/min saturating 88% (room air). An initial VBG showed pH 7.22, pCO_2 51 mmHg, bicarbonate 20 mmol/L, lactate 3.3 mmol/L. His GCS deteriorated to 3 within an hour of first contact, pupils 3 mm bilaterally, nil reaction to light, nil rigidity or clonus, areflexic globally, with occasional myoclonic jerks. He was intubated for airway protection. ECG was normal. A limited urine drug screen was positive for ethanol and amphetamine, but blood alcohol concentration was <0.03 % at 4 hours post presentation. At 4 hours, ABG showed pH 7.15, pCO_2 45 mmHg, bicarbonate 16 mmol/L, lactate 2.2 mmol/L. Anion gap 23 mmol/L on formal blood tests prompted further investigations, noting serum osmolality 322 mmol/L at 2 hours with osmol gap 30.4 mmol/L. The lack of rapid recovery which is characteristic of GHB, and the pattern of blood test results suspicious for toxic alcohol poisoning in an unconscious patient with an unknown exposure prompted a change in treatment. Intravenous ethanol, folic acid and thiamine, plus continuous venovenous haemodiafiltration were commenced. However, at 12 hours post-exposure, salicylic acid, methanol and ethylene glycol were reported as negative on the admission bloods. The patient improved, was following commands at 13 hours post first contact, and admitted to accidental ingestion of a mouthful of purported GHB that tasted unusual, 'like plastic'. Later specific drug testing was positive for 1,4-BD and GHB at 2 hours and negative for both at 27 hours post presentation. 1,4-BD is reported to taste different to other analogues, and its physicochemical properties and metabolic disposition readily explain the clinical manifestations and progression noted in this patient.

Conclusion: This is an interesting case of recreational 1,4-BD ingestion mimicking toxic alcohol exposure. Concomitant ethanol intake, and then later use of intravenous ethanol may have further prolonged the duration of decreased level of consciousness due to interference with the action of alcohol dehydrogenase and conversion of 1,4-BD to GHB. Access to laboratory assays can positively contribute to clinical management.