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Case report

Ethylene glycol poisoning: A diagnostic challenge in a patient with persistent seizures and a severe metabolic acidosis

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ABSTRACT

Introduction: Due to the nationwide lockdown to mitigate the spread of COVID-19 and subsequent alcohol ban in South Africa, several cases of toxic alcohol ingestion presented to our emergency unit. Many of these patients admitted to making home brews of alcohol while others simply use industrial toxic alcohols. The diagnosis of these poisonings is challenging as direct assays are not available in our setting.

Case report: We present a case of presumed ethylene glycol poisoning that presented with persistent seizures and a high anion gap metabolic acidosis (HAGMA).

Discussion: A high index of suspicion for toxic alcohol poisoning should be maintained in patients presenting with an altered mental status, seizures and a HAGMA. Indirect markers such as clinical features and laboratory results can lead to the diagnosis when direct assays are unavailable.

African relevance

- Toxic alcohol ingestion is a common and lethal type of poisoning in Africa with many documented cases of mass poisoning.
- During the COVID-19 a spike in similar cases can be expected in countries where alcohol bans were instituted.
- The diagnosis of toxic alcohol poisoning is challenging in our context as most laboratories do not do diagnostic assays.

Introduction

On 15 April 2020 a nationwide hard lockdown was instituted in South Africa to curb the transmission of COVID-19. Alcohol sales were banned which led to an increase in the consumption of toxic alcohols. Indirect evidence for this was anecdotal observations in emergency units as well as media reports. In one such report, 14 deaths and 10 hospitalisations due to toxic alcohol poisoning was reported [1].

This case demonstrates the diagnostic challenges that were faced in a patient presenting with persistent seizures of unknown cause. Ultimately, the combination of a high index of suspicion combined with blood gas analysis, urinalysis and low serum calcium levels pointed to the diagnosis of toxic alcohol poisoning. This diagnosis was supported by the dramatic improvement the patient had to emergency haemodialysis.

Case report

A 29-year-old male was brought in with seizures for 20 min prior to arrival. The family reported that he had no comorbid conditions. No history of toxic alcohol ingestion, home brews or illicit drug use was obtained.

Upon arrival the patient was taken to the resuscitation area. His glucose was 11.4 mmol/L. His seizures were terminated with 4 mg lorazepam intravenously. His Glasgow Coma Scale (GCS) was 5/15 and he could not maintain his airway without a jaw-thrust manoeuvre. As the team prepared for intubation, an arterial blood gas was obtained. It showed a severe mixed metabolic and respiratory acidosis with a pH of 6.33, pCO2 of 78.7 mmol/L, HCO3 of 2.8 mmol/L, calcium of 1.30 mmol/L, an unrecordable high lactate (measuring range 0.0–30), glucose of 11.4 mmol/L and a base excess of −31.0. The anion gap was 40.8.

The patient was hypotensive with a blood pressure (BP) of 88/42 and pulse rate of 56 prior to intubation. A bolus of 1000 mL balanced crystalloid solution was infused and a single push-dose of 20 μg adrenaline was administered prior to intubation to prevent peri-intubation cardiac arrest. It spite of these measures, the patient deteriorated into cardiac-arrest directly post-intubation. Fortunately, return of spontaneous circulation (ROSC) was achieved after one cycle of CPR. The post arrest blood gas showed an unrecordable low PH (measuring range 6.30–8.00)

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and unrecordable high lactate. An adrenaline infusion was initiated. The patient stabilized with a BP of 115/64, pulse rate of 127 and saturation of 99%. The Blood gas after stabilization showed a pH of 6.69, pCO2 of 54.2 mmol/L, HCO3 of 4.5 mmol/L, a base excess of −26.2 and an anion gap of 35.5.

A non-contrasted CT brain was normal. The ECG showed global ST depression which was interpreted as myocardial depression due to severe acidosis. The impressive HAGMA of 40.8 suggested a toxicological cause for the seizures. The patient had no history of prior seizures or access to isoniazid. Sodium channel blockade was unlikely based on history and ECG findings. There were no other features of a sympathomimetic toxidrome to suggest cocaine or amphetamine use. At this stage, toxic alcohol poisoning was considered a likely diagnosis based on index of suspicion and HAGMA.

Fomepizole is not available in South-Africa. Ethanol was omitted (see Discussion). The patient received emergency haemodialysis within 2 h of arrival.

Initial blood results showed a normal urea and creatinine with a high creatinine kinase and myoglobin, likely due to the seizures. The serum and urine tox-screen was only positive for benzodiazepines which were administered to abort the seizures. Amphetamines, cocaine and tricyclic antidepressants tested negative. Direct assays for toxic alcohols are not available at our laboratory nor would they be timeous. Subsequent blood results showed persistently low calcium at 1.93 mmol/L and an acute kidney injury with urea of 11.5 mmol/L, creatinine of 207 mmol/L and eGFR of 33 mL/min/1.73 m^2. A significant amount of Calcium oxalate crystals were later found in the urine.

After dialysis the patient improved markedly. The blood gas post dialysis showed a PH of 7.36, pCO2 of 41.7 mmol/L, HCO3 of 22.7 mmol/L, lactate of 1.6 mmol/L and a base excess of −2.1. The patient was extubated within 24 h and he made a complete neurological recovery post extubation. No further dialysis was required.

Discussion

Ethylene glycol poisoning can present a diagnostic challenge in the emergency unit. It is estimated that 6% of new-onset seizures are due to drug toxicity [2]. The major factor prompting us to consider a toxicological cause for seizures in this patient, was the severe HAGMA. A number of toxicological agents could be considered in addition to toxic alcohols, including tricyclic antidepressants, Isoniazid and sympathomimetic agents. These were ruled out in this patient.

Early after ethylene glycol ingestion, patients present with a high osmolar gap [3]. As the osmolar gap decreases (ethylene glycol being metabolized by alcohol dehydrogenase (ADH)), the anion gap increases (glycolic acid is produced). Eventually, the osmolar gap may be normal while the HAGMA is predominant [4,5]. An osmolality was unfortunately not requested. Even though a high value would have been useful, a normal value would not rule out the diagnosis.

Oxalic acid leads to the formation of calcium oxalate crystals [4]. These crystals deposit in organs such as the brain, kidneys and heart causing multi-organ failure [5]. Hypocalcaemia with or without resultant tetany or seizures could develop, as in this patient [4]. Calcium oxalate crystals may be found in the urine, however, this finding is neither highly sensitive nor specific for ethylene glycol toxicity [4].

Ethylene glycol poisoning is primarily treated with ADH inhibitors, fomepizole or oral ethanol [4,5].

Early haemodialysis is indicated for patients who exhibit multi-organ dysfunction and persistent metabolic acidosis despite ADH inhibition. Ethanol alone is seldom effective in significant ingestions, particularly in late presenters [6]. Patients presenting to smaller hospitals should be up-referred early to centres where haemodialysis is available.

Due to the seizures and severity of the acidosis, ethanol was omitted and emergency dialysis was our treatment of choice.

Conclusion

In patients presenting with persistent seizures and a HAGMA, toxic alcohol poisoning must be considered. Indirect markers that can aid in the diagnosis of ethylene glycol poisoning are an increased osmolar gap, hypocalcemia and calcium oxalate crystals in the urine. While fomepizole and ethanol may be effective antidotes in milder cases, early haemodialysis is life-saving when severe.

Dissemination of results

This case was discussed in an academic ward round with the SBAH emergency medicine team led by one of the co-authors of the case report, Dr Vidya Lalloo. We also plan to share the link to the article on various platforms such as our hospital Whatsapp groups as well as on the Emergency medicine Pretoria social media platforms (Twitter, Facebook).

CRediT authorship contribution statement

Authors contributed as follow to the conception or design of the work; the acquisition, analysis, or interpretation of data for the work; and drafting the work or revising it critically for important intellectual content:

AS – 50%
VL - 25%
AE – 25%

All authors approved the version to be published and agreed to be accountable for all aspects of the work.

Declaration of competing interest

The authors declare no conflict of interest.

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