ECRONICON OPEN ACCESS

EC CLINICAL AND MEDICAL CASE REPORTS

Case Report

An Acute Poisoning of Ethylene Glycol

Aakash Ghai, Brian Behnke*, Kelly Schulte, Travis Jones, Deren Goker and Dmitriy Scherbak

Internal Medicine Residency, Sky Ridge Medical Center, USA

***Corresponding Author:** Brian Behnke, Internal Medicine Residency, Sky Ridge Medical Center, USA.

Received: April 27, 2022; **Published:** May 31, 2022

Abstract

This case report involves a patient who was diagnosed with a self-induced poisoning of ethylene glycol. We will discuss the patient's clinical presentation, symptoms and treatment. Due to the rarity of an acute intoxication with ethylene glycol, we will break down the essential elements to recognize the pathology of indigestion of the substance. We present this case to increase physician awareness of ethylene glycol poisoning. With timely recognition, more patients will be able to receive appropriate treatment, resulting in a decrease bodily harm and mortality.

Keywords: *Ethylene Glycol; Self-Induced Poisoning*

Introduction

Ethylene glycol is toxic alcohol present in primarily antifreeze solutions and as a raw material for the manufacture of polyester fibers. This odorless, colorless, toxic liquid is often ingested by children and animals accidentally due to its sweet-tasting quality. It is metabolized primarily in the liver into oxalic acid which is fatal if left untreated. Considered a toxic alcohol, a fatal dose of ethylene glycol is 1 - 2 ml/kg, with a high likelihood of death within 24 hours. Therefore, early recognition and rapid treatment are essential in preventing mortality. This case study will explore the signs, symptoms, and management of intentional ingestion of ethylene glycol with the goal of prompt identification of a presentation of ethylene glycol toxicity.

Case Presentation

46 year old female with past medical history of fibromuscular dysplasia, bilateral vertebral artery dissection, known patent foramen ovale, and recent motor vehicle accident presented to the hospital for concerns of slurred speech, left arm paresthesia, vision changes and quickly decompensated with altered mental status and became obtunded while in the emergency department. Arterial blood gas and labs done soon afterwards showed pH 6.90, PCO_2 24, PO_2 35, bicarbonate 5, Anion Gap 15, serum creatinine 1.13, white blood cell count 22.8, lactic acid 2.2, and patient was found to be tachypnic. Patient was immediately admitted to the intensive care unit and intubated and a key decision to measure osmolar gap was made and found to be 14. Following this discovery, patient was found to have nondetectable levels of salicylates, alcohol, and acetaminophen. The patient was also found to have calcium oxalate crystals as well as hippuric crystals. CT head was negative but MRI brain showed a possibly tiny lacunar infarct in the right centrum ovale. Toxicology was consulted through Poison Control and fomepizole and bicarbonate drip was started, while toxic alcohol labs were pending. Over the next few days, the patient was successfully extubated and the bicarb drop was discontinued as the anion gap metabolic acidosis resolved. Ultimately, ethylene glycol returned positive at 5, however this lab was drawn after fomepizole was started. The patient did admit to ingesting ethylene glycol in an attempt to commit suicide afterwards. The patient did not require emergent dialysis during her hospitalization despite her serum creatinine peaking at 7.03. She was ultimately transferred to an inpatient psychiatric facility once she was medical stable.

Discussion

Ethylene Glycol toxicity/intoxication primarily affects children and animals, through accidental ingestion, and those experiencing alcohol use disorder and/or psychiatric illnesses, through intentional ingestion. Due to the odorless, colorless and sweet taste, accidental ingestion in common with younger population whereas with those over the age of 20, intentional ingestion is much more likely [1].

In 2020, there were 3,901 cases of ingestion of ethylene glycol by individuals 20 years or older and 1609 cases of ethylene glycol ingestion by those younger than 20 years old [2]. The mortality rate can be as high as 22% depending on the timing of ingestion and initiation of early treatment [3].

Indications for antidotal therapy with fomepizole or ethanol
Documented serum methanol or ethylene glycol concentration >20 mg/dL (methanol SI equivalent 6.2 mmol/L; ethylene glycol SI equivalent 3.2 mmol/L)
OR
Documented recent history of ingesting toxic amounts of methanol or ethylene glycol and serum osmol gap >10
OR
Strong clinical suspicion of methanol or ethylene glycol poisoning and at least two of the following:
a) Arterial pH <7.3
b) Serum bicarbonate <20 meq/L (mmol/L)
c) Osmol gap >10
d) Mary oxalate crystals Present

Table 1: *Indications for antidotal therapy with fomepizole [16].*

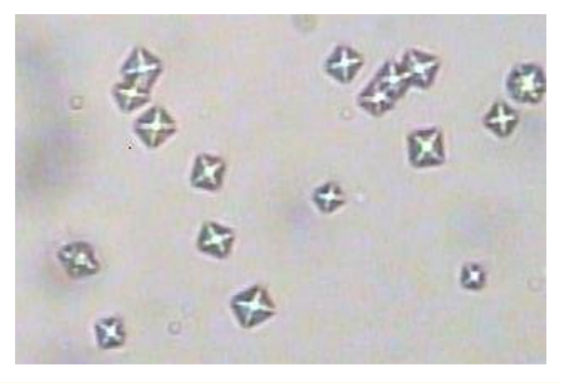

Figure 1: *Calcium oxalate crystals [17].*

A better understanding of the CNS toxic effects, metabolic acidosis, and renal impairment caused by ethylene glycol poisoning, can be ascertained from delving into the pathophysiology surrounding ethylene glycol metabolism. Ethylene glycol is easily and efficiently absorbed in the GI system with quick oxidation reactions, ultimately producing glycolic acid and lactic acid as a byproduct furthering metabolic acidosis [4-8]. Glycolic acid is ultimately converted to oxalic acid which deposits as calcium oxalate crystals. First order elimination

in concentrations < 250 mg/dL results in a $t_{1/2}$ of 4 - 6 hours, while concentrations > 250 mg/dL results in a $t_{1/2}$ of 10 - 18 hours [6,9,10]. Lethal doses of ethylene glycol without intervention are estimated to be 1 - 2 mL/kg of 95% concentrated solution [11].

Variable mental status presentation accompanied by possible stigma surrounding suicide or self-harm often makes early diagnosis challenging, however this case presentation hopes to identify key signs and symptoms that assist providers with early diagnosis and management. Within the first 12 hours of ingestion, patients will exhibit signs of confusion, ataxia, encephalopathy mimicking alcohol intoxication, nystagmus, tachycardia, and depending on dose at initial ingestion, coma and death [1]. Between 12 - 24 hours, cardiopulmonary failure usually develops, manifested with dyspnea, pulmonary edema, hyperventilation, cyanosis and/or oliguria [1,12]. From 24 - 48 hours, patients usually develop renal failure, multisystem organ failure, coma, and death [1]. Calcium oxalate crystals often result in both nephrotoxicity and neurotoxicity. As ethylene glycol oxidizes to its toxic metabolites, the osmolar gap drops and the patient develops an anion gap metabolic acidosis [1].

Alternative diagnoses are always important to consider, especially given variability of original presentation. We hope this presentation reminds the reader to consider an ABG and serum osmolality to evaluate for acidosis, as well as hypercarbia when considering a differential diagnosis, particularly if the patient presents obtunded, as in this case study. When evaluating for causes of acidosis, a first key step involves determining both the anion and osmolar gap. From there, consider salicylates, causes of lactic acidosis, including infection/sepsis, alcoholic ketoacidosis, isopropyl alcohol ingestion, methanol ingestion, diethylene glycol, toluene, diabetic ketoacidosis and uremia [1,13].

Key considerations when it comes to diagnosis and management involve measuring the osmolar gap, which can point providers in the right direction early enough to use fomepizole, which is the gold standard of treatment, as it prevents formation of toxic metabolites if used in time [8]. Ethylene glycol at low doses can be managed conservatively, however, should the patient be developing acidosis, renal dysfunction, and mental status change, strongly consider fomepizole, ethanol, or dialysis. Bicarbonate therapy will assist in correcting acidosis and prevent the formation of glycolate and oxalate [14]. Vitamin B1 and Vitamin B6 (thiamine and pyridoxine) has been shown to reduce glyoxylate into less toxic substances [14]. Ethanol is often used in more rural or county hospitals where fomepizole may not be as readily available. The last option being hemodialysis to remove toxic metabolites especially if ethylene glycol has already been oxidized, as it would be too late to use either fomepizole or ethanol [14,15].

The final method of treatment, hemodialysis, is used to remove toxic metabolites if ethylene glycol has already been oxidized, in which case it would be too late to use fomepizole or ethanol [14,15].

Conclusion

This case presentation hopes to highlight the importance of considering ethylene glycol intoxication when evaluating an adult who may present confused/obtunded, with a severe anion gap metabolic acidosis, an osmolar gap, and renal dysfunction. In addition, it recognizes the importance of starting fomepizole and bicarbonate infusion early, in order to prevent formation of toxic metabolites, with the understanding that the patient may require emergent hemodialysis as a last resort.

Bibliography

1. Iqbal A., *et al*. "Ethylene Glycol Toxicity". [Updated 2021 Sep 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing (2022).

2. David D Gummin., *et al*. "2020 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 38th Annual Report". *Clinical Toxicology* 59.12 (2021): 1282-1501.

3. Tanasescu A., *et al*. "Outcome of patients in acute poisoning with ethylene glycol--factors which may have influence on evolution". *Journal of Medicine and Life* 7.3 (2014): 81-86.

4. Heather Miller., *et al*. "American Academy of Clinical Toxicology Practice Guidelines on the Treatment of Ethylene Glycol Poisoning". *Journal of Toxicology: Clinical Toxicology* 37.5 (1999): 537-560.

5. Frommer JP and Ayus JC. "Acute ethylene glycol intoxication". *American Journal of Nephrology* 2.1 (1982): 1-5.

6. Jacobsen D., *et al*. "Ethylene glycol intoxication: evaluation of kinetics and crystalluria". *American Journal of Medicine* 84.1 (1988): 145-152.

7. Goodman LS., *et al*. "Goodman & Gilman's The pharmacological basis of therapeutics". 9th edition. New York: McGraw-Hill, Health Professions Division (1996): 1682-1683.

8. Brent J., *et al*. "Fomepizole for the treatment of ethylene glycol poisoning. Methylpyrazole for Toxic Alcohols Study Group". *New England Journal of Medicine* 340.1 (1999): 832-838.

9. Moreau CL., *et al*. "Glycolate kinetics and hemodialysis clearance in ethylene glycol poisoning. META Study Group". *Journal of Toxicology. Clinical Toxicology* 36.7 (1998): 659-666.

10. Sivilotti ML., *et al*. "Toxicokinetics of ethylene glycol during fomepizole therapy: implications for management. For the Methylpyrazole for Toxic Alcohols Study Group". *Annals of Emergency Medicine* 36.2 (2000): 114-125.

11. Jacobsen D and McMartin KE. "Methanol and ethylene glycol poisonings. Mechanism of toxicity, clinical course, diagnosis and treatment". *Medical Toxicology* 1.5 (1986): 309-334.

12. Bobbitt WH., *et al*. "Severe ethylene glycol intoxication with multisystem failure". *Western Journal of Medicine* 144.2 (1986): 225-228.

13. Ashurst JV and Nappe TM. "Methanol Toxicity". 2021 Jun 26. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing (2022).

14. Caravati EM., *et al*. "Ethylene glycol exposure: an evidence-based consensus guideline for out-of-hospital management". *Clinical Toxicology* 43.5 (2005): 327-345.

15. Mégarbane B. "Treatment of patients with ethylene glycol or methanol poisoning: focus on fomepizole. Open Access emergency medicine". *Open Access Emergency Medicine* 2 (2010): 67-75.

16. Sivilotti MLA. Methanol and ethylene glycol poisoning: Management. UpToDate (2021).

17. Polvilitis T. Topics in Conservation Biology - Researchgate. ResearchGate (2012).

Volume 5 Issue 6 June 2022