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Case Report

Case Report - Early Recognition of Methanol Poisoning

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Abstract

This case illustrates a classic presentation of methanol poisoning with its associated radiologic and clinical manifestations. It also provides a comparison with another toxic alcohol, ethylene glycol, highlighting the similarities and the importance of rapid diagnosis to prevent devastating long-term consequences. The patient presented to Kaiser-Permanente South Sacramento with signs and symptoms of methanol poisoning. Clinical and imaging findings included severe metabolic acidosis and altered mental status that progressed to coma and bilateral lesions of the putamen on MRI, characteristic of methanol poisoning [1]. Upon awakening from the coma the patient was noted to have abulia, psychomotor slowing, and decreased executive functioning. Neurologic consultation was obtained on hospital day 11. Patient was followed and demonstrated gradual improvement in neurologic function that continued until the time of discharge. This clinical picture is very similar to that associated with ethylene glycol poisoning that can also present with altered mental status, severe metabolic acidosis and bilateral lesions of the putamen. This report provides a direct comparison of methanol and ethylene glycol toxicity including clinical and imaging findings. The case shows that methanol poisoning can cause short-term and long-term morbidity, but prompt recognition and treatment can lead to meaningful recovery.

Keywords: Case Report; Methanol Poisoning; Ethylene Glycol

Abbreviations

MRI: Magnetic Resonance Imaging; CT: Computed Tomography

Introduction

Methanol poisoning is an uncommon but significant cause of morbidity and mortality worldwide. Methanol can be easily accessed in household and industrial products such as windshield wiper fluid, automobile antifreeze, perfumes, and other types of fuels [2]. It is much cheaper than ethanol, so it is often used to dilute illegally produced alcoholic beverages or used instead of ethanol in victims unaware of the consequences [2]. It is also sometimes intentionally ingested in suicide attempts, or unintentionally ingested by unattended young children [2]. Methanol is one of three toxic alcohols with the other two being ethylene glycol and isopropyl alcohol. They get this name for their propensity to cause severe consequences if ingested [2]. These alcohols are highly toxic not only with ingestion but also with absorption through the dermis and respiratory inhalation. Patients with methanol poisoning will often present with characteristic clinical and imaging findings.

Case narrative

This report is evaluating a case of a male with a longstanding history of alcohol abuse who was brought to the emergency department by his family due to a stuporous state which advanced to coma requiring intubation. He had a severe anion gap metabolic acidosis with high suspicion of methanol poisoning. He was given fomepizole, thiamine, and hemodialyzed and was stabilized for extubation. The neurologist was consulted to establish a prognosis for this patient. He was noted to have abulia from brain injury, partial orientation, and psychomotor slowing with poor short term memory and executive functioning. He spoke in 2-3-word phrases however did not have significant weakness, ataxia, or long tract signs. MRI images demonstrate significant bilateral signal alteration of the basal ganglia including the putamen and caudate nuclei with some involvement of the subcortical frontal region as seen in (Figure 1). Of note, the patient visited psychiatry 3 weeks after this event and was markedly improved, speaking in full sentences and admitted to ingestion of windshield wiper fluid.

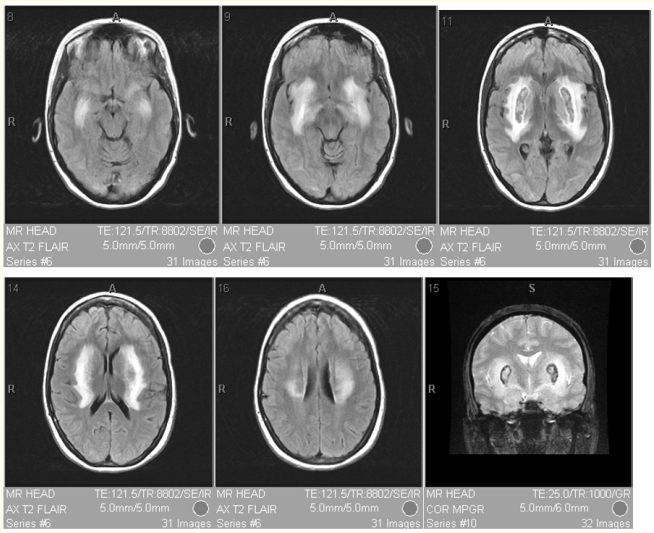

Figure 1: MRI images from the patient's case demonstrating significant bilateral signal alteration of areas including the basal ganglia, putamen, and caudate nuclei consistent with toxic methanol ingestion.

Discussion

Both methanol and ethylene glycol poisonings can lead to a clinical picture including anion gap metabolic acidosis with end organ damage. Other differentials to be considered include salicylate toxicity, ethylene glycol or isopropyl alcohol toxicity, and polysubstance abuse. Acquiring a history may be difficult in patients with intentional ingestion or self-harm tendencies due to hesitancy or inability to provide accurate information. If ingestion is not witnessed, clinicians must maintain a high index of suspicions for toxic alcohol ingestion - especially in patients with metabolic acidosis. Clinical severity may also be initially mild and develop to a severe presentation, requiring adequate observation and initiation of treatment.

Methanol

Methanol itself is not toxic, however, when metabolized through the liver into formaldehyde and formic acid by alcohol dehydrogenase, it can have devastating effects on the body in a short amount of time. These effects can present as blindness, profound anion gap metabolic acidosis, gastrointestinal symptoms, headache, vertigo, altered mental status and can progress to coma [2]. Some of the neurologic deficits including blindness can be permanent [2,3]. There is often a latent period of 6 hours so patients may not present for many hours after ingestion further exacerbating the toxicity [2]. Physical examination may range from decreased visual acuity, papilledema on fundoscopic exam, and mild discomfort to respiratory and circulatory failure, coma, and death [2]. The toxic effects of methanol on the brain often present on MRI with characteristic imaging findings. The most frequently observed finding on MRI is bilateral putaminal lesions [1,4-7]. A case report involving a cohort of 46 patients with documented methanol poisoning revealed that 21 of those patients had imaging findings consistent with methanol poisoning, and of those 21, 13 had putaminal lesions [1]. Other findings reported in the study included lesions in the globus pallidus, subcortical white matter, cerebellum, brainstem and optic nerve, with globus pallidus being the second most commonly afflicted area [1]. Some studies report parkinsonian-like symptoms in patients with lesions in the basal ganglia and globus pallidus [2]. Hyperintensities in the frontal lobe and occipital lobes [6] as well as in optic tracts and radiations [3] on T2 imaging have been

reported as well. The diagnosis may be confirmed with elevated serum concentrations and is notable for elevated anion gap metabolic acidosis with an osmolality gap that is decreasing longitudinally [2].

Ethylene glycol

Ethylene glycol is also commonly used in antifreeze solution and has similarly devastating consequences if ingested, even in small quantities. This sweet-tasting liquid may contribute to accidental ingestions, while ingestions can also be intentional with the purpose of inebriation or suicidal attempt [8]. Toxic levels are attained usually by ingestion, as ethylene glycol has limited dermal absorption. Ethylene glycol is metabolized by the liver via oxidation by alcohol dehydrogenase and glycolic acid, which are further metabolized into toxic byproducts [8]. Glycolic acid causes a severe anion gap metabolic acidosis while oxalic acid is associated with end organ injury, specifically nephrotoxicity as it forms deposits causing proximal tubular necrosis. This compound also has high affinity to calcium, forming calcium oxalate crystals in the urine and leading to systemic hypocalcemia with QT prolongation, arrhythmia, tetany, and even seizures [8,9]. These metabolic pathways predominantly cause symptoms in the central nervous system, cardiopulmonary, and renal system, however they can cause multiorgan damage due to inhibition of cellular mechanisms such as cellular respiration and metabolism [10]. CT imaging in acute toxicity commonly shows diffuse edema with localization of the basal ganglia, thalami, midbrain, and pons [9,11]. Few case reports mention MR imaging, however Morgan., *et al*. report necrosis of the bilateral putamen [11]. Multiple case reports also mention significant bilateral facial nerve palsy with involvement of other cranial nerves including IX and X, which persisted months after treatment [12,13].

Treatment for toxic alcohol ingestion of both methanol and ethylene glycol are aimed at preventing the metabolism of these substances into their toxic metabolites. This includes competitive alcohol dehydrogenase inhibitors fomepizole or ethanol. Patients should be initially evaluated to ensure secure airway and oxygenation, correction of metabolic abnormalities and hemodialysis if necessary.

Conclusion

In summary, intoxication with dangerous chemicals can quickly become deadly, and early recognition is essential to prevent mortality. While history is often limited in situations of toxic alcohol ingestion, we have discussed similarities and differences aiding in further establishing a diagnosis. Red flag symptoms such as metabolic acidosis with visual disturbances should guide health care professionals to seek appropriate consultations with toxicology or poison control to ensure early intervention. Health care professionals should also ensure proactive education for safe storage and use of these chemicals, and what to do if ingestion has occurred.

Author Contributions

Krista Tenerelli and Alexander Nagourney: planning, preparation, and drafting of the manuscript.

Jason Chang: provision, management, and discussions about the patient and review of the manuscript

Forshing Lui: Advice, review, and edit if the manuscript. Corresponding author.

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Conflict of Interest

All authors have NO conflict of interest exist.

Disclosure Statement

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