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CLINICAL VIGNETTE

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Introduction

Methanol intoxication can cause serious illness with potentially fatal consequences. Early identification and appropriate treatment is essential for preventing mortality and long-term sequelae. We present a case of methanol toxicity and review current literature concerning pathophysiology, acute management and prognostic indicators in order to accurately and expeditiously treat this life-threatening condition.

Case Presentation

A 52-year-old male was brought into the Emergency Department by Emergency Medical Services (EMS) after being found intoxicated in a public park. On arrival, the patient was somnolent yet aroused to vigorous verbal and tactile stimuli. He was not able to provide a reliable history but per EMS report, was noted to have several empty vodka bottles strewn near his person in the park. The patient was not able to provide any further information with respect to his past medical history nor the circumstances precipitating his visit to the ED. Field glucometry revealed a value of 119 mg/dL.

On initial examination, the patient appeared to be modestly disheveled with a strong odor of alcohol and ammonia emanating from his breath. His temperature was 37.1C, pulse of 98, blood pressure of 112/68, respiratory rate of 24 and a pulse oximetry reading of 98% on room air. There were no external signs of head trauma. The neurological exam was significant for the presence of marked truncal ataxia, impaired heel-shin testing and bilateral cogwheel rigidity. There were no other significant findings on the remainder of the exam.

Given the patient's limited history and poor cooperation with the physical exam, ancillary testing including serum chemistries, osmolality, a complete blood count, blood gas analysis, serum ethanol and ammonia levels as well as urine analysis and toxicology were ordered. In addition, a computed tomography (CT) of the brain was ordered.

Interestingly, the serum analysis revealed the presence of an anion gap metabolic acidosis with a calculated osmolal gap of 24. The arterial pH was 7.21 and serum ethanol resulted as 109 mg/dL, which was much lower than expected for the patient's clinical level of intoxication. Furthermore, CT of the brain revealed small bilateral basal ganglia hemorrhages along with hypodensities in the putamen bilaterally. Given the above findings, a presumptive diagnosis of toxic alcohol ingestion was made and treatment with fomepizole, thiamine, folate and pyridoxine was initiated. Nephrology was consulted for emergent hemodialysis. Additional blood was sent for serum methanol and ethylene glycol levels but as these are not processed in-house, the results were not immediately available.

In the interim, the patient's sensorium cleared to the point that he was able to relate a history of ingesting several bottles of self-produced liquor (colloquially known as "moonshine") along with several cans of beer. At this point, he endorsed having new onset bilateral "blurry" vision.

The patient was admitted to the ICU where he underwent dialysis and completed several days of fomepizole therapy. On hospital day five, the patient had returned to his normal baseline function with resolution of his visual complaints and no longer exhibited any abnormal neurological findings. He was provided with education regarding the dangers of consuming home distilled alcohol and was discharged to a shelter in good condition.

Discussion

Methanol poisoning is associated with a high rate of mortality that rapidly increases with delays in recognition and treatment. Classically, toxicity manifests as a wide anion gap metabolic acidosis in the presence of visual symptoms. Basal ganglia hemorrhage and putaminal necrosis with associated neurologic symptoms are well-documented complications[1]. The clinical syndrome of methanol poisoning is a result of toxic metabolites, principally formic acid, while the parent molecule is mostly harmless. Following ingestion, methanol is metabolized to formaldehyde via alcohol dehydrogenase and subsequently converted to formic

acid[2]. Accordingly, the most important therapeutic intervention is the timely administration of an alcohol dehydrogenase competitive inhibitor, either fomepizole or ethanol[2]. Competitive inhibition decreases formic acid production allowing time for methanol excretion via renal and pulmonary mechanisms[3]. Importantly, methanol is a dialyzable substance and according to the American Academy of Clinical Toxicology, hemodialysis should be considered in patients with any of the following: metabolic acidosis, visual abnormalities, renal failure, or electrolyte imbalance unresponsive to conventional therapy and/or serum methanol concentration of 50 mg/dl[4]. It has been hypothesized that folate improves the metabolism of formic acid and can be considered as an adjunct to fomepizole therapy[3]. Furthermore, treatment with base, such as bicarbonate, has been shown to enhance the renal excretion of formic acid and should be considered in all patients presenting with acidosis[3].

Poor prognosis is associated with coma on admission, severe acidosis (pH <7.00), and delay in treatment greater than 24 hours following ingestion. Intuitively, the presence of ethanol co-ingestion improves outcomes[5]. Although rare, approximately 1000 to 2000 cases are reported per year. Therefore, physicians must maintain a high index of suspicion for methanol toxicity in patients presenting with a wide anion gap acidosis in the setting of an altered level of consciousness and visual symptoms.

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