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POISONING FROM ALCOHOL AND ITS SUBSTITUTES

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Abstract: This atricle discusses the range of clinical presentations seen with poisonings by the major toxic alcohols-methanol, ethylene glycol, and isopropyl alcohol. It outlines a straightforward diagnostic strategy and discusses in detail the current treatment recommendations.

Keywords: Alcohols, Ethylene Glycol, Isopropanol, Methanol, Patient Care Management, Poisoning.

INTRODUCTION

Poisonings with methanol, ethylene glycol, and isopropanol—commonly referred to as the toxic alcohols—often present the emergency physician with a major diagnostic challenge. The identity of the ingested substance is frequently a mystery on presentation. Patients with an intentional ingestion, either for recreation or with suicidal intent, may be less than forthcoming. Young patients may not be able to identify the substance. Patients may be in significant distress or comatose and unable to give any useful history. In these cases, the clinician must rely upon the nature of the presentation and the presence of metabolic derangements—and must always keep a high index of suspicion for toxic alcohol poisoning.

MATERIALS AND METHODS

Because toxic alcohol poisoning may cause potentially irreversible damage in a time-dependent fashion, prompt diagnosis and treatment are crucial (2). Though we lack immediate testing for the toxic alcohols, there are useful laboratory clues that can help clinicians quickly zero in on the most likely toxic agent. Those clues include the osmol gap, the anion gap, and the patient's acid-base status. This article will review the pathophysiology of these intoxications, the clinical presentations, the laboratory workup, and the treatment of toxic alcohol ingestions. This paper will also discuss the limitations of the emergency department workup and how the absence of particular laboratory findings does not necessarily rule out the diagnosis.

RESULTS AND DISCUSSION

Toxic alcohols are found in many readily available household and industrial products. Methanol ("wood alcohol") is a major component of windshield washer fluid, many industrial solvents, and may also be ingested as a recreational intoxicant sometimes mislabeled "moonshine". Ethylene glycol is a chief component of antifreeze (3). Isopropanol, widely known as rubbing alcohol, is a common antiseptic (4). Although fatalities from toxic alcohol ingestions are relatively rare in the United States (< 30 per year), delayed diagnosis and treatment are the main reasons for poor outcomes (5). It cannot be over-stressed that early identification and treatment can significantly reduce morbidity and mortality.

The toxicity of methanol and ethylene glycol arises primarily from highly toxic intermediate metabolites generated by the action of alcohol dehydrogenase (ADH), the key enzyme in their breakdown. The inhibition of ADH, therefore, becomes a crucial step in treatment (5). ADH catalyzes the first oxidation of methanol and ethylene glycol to formaldehyde and glycoaldehyde respectively. These compounds undergo further oxidation by aldehyde dehydrogenase (ALDH)

to form carboxylic acid metabolites. Methanol is ultimately metabolized to formic acid, while ethylene glycol is metabolized to glycolic acid and oxalic acid (Figures 1, **,2).**2). These metabolic byproducts are potent organic acids that generate a high anion gap metabolic acidosis. They are also responsible, as further discussed below, for other significant toxic effects (1).

Figure 1: The metabolic pathway of Methanol

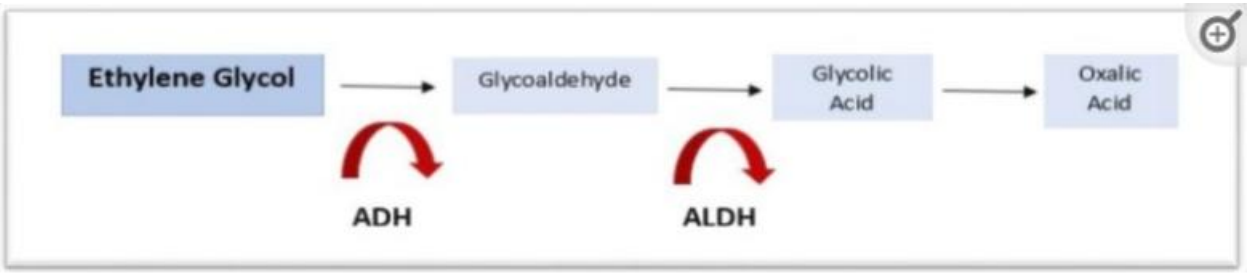

Figure 2: The metabolic pathway of Ethylene Glycol

Although methanol and ethylene glycol both produce CNS depression, isopropanol generates the most profound degree (1). Isopropanol is rapidly absorbed and inebriates much like ethanol. It is directly converted to acetone by alcohol dehydrogenase, and acetone itself is a CNS depressant that leads to further sedation.

Methanol is a colorless, volatile liquid at room temperature, possessing only a faint, sweet odor. Intoxications are rare (1000–2000 cases per year, about 1% of all significant poisonings) and occur primarily through the intentional or unintentional ingestion of methanol containing liquids including windshield washer fluid or antifreeze (2). Methanol has been used as an ethanol replacement and can be found in some products called “moonshine”. (Classic “moonshine” is a high-concentration ethanol solution distilled from fermented corn). Methanol is rapidly absorbed following ingestion and is metabolized by ADH. Toxicity has been reported with as little as 15 ml of 40% methanol solution, which is its approximate concentration in -30-degree F windshield washer fluid (2). Peak levels generally occur within 60–90 minutes (1).

CONCLUSION

Poisoning with methanol, ethylene glycol, and isopropanol present diagnostic and therapeutic challenges to emergency physicians. Toxic alcohol poisonings lead to an elevated osmolar gap and, with the exception of Isopropanol, a metabolic acidosis. In order for the timely initiation of life-saving treatment, emergency physicians need a solid understanding of the pathophysiology, clinical presentation, laboratory workup, and treatment.

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