POISON PEARLS

Toxicology Topics for the Healthcare Team of a Poisoned Patient

For ALL patient recommendations call the Drug and Poison Information Center at 1-800-222-1222

Methanol Toxicity

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Background

Methanol, also referred to as methyl alcohol or "wood alcohol," is a toxic low–molecular weight alcohol encountered in a range of industrial and household products. Common sources include fuels, solvents, windshield washer fluid, certain antifreeze formulations, improperly distilled ethanol (e.g., illicit "moonshine"), and some foreign-manufactured hand sanitizers.

According to 2024 data from U.S. poison centers, 705 single-substance methanol exposures were reported, along with an additional 2,649 cases involving methanol as part of a multi-substance exposure (Beuhler et al., 2025). Toxicity most commonly results from oral ingestion; however, clinically significant exposure may also occur via inhalational or dermal routes, particularly in settings of prolonged or high-concentration contact.

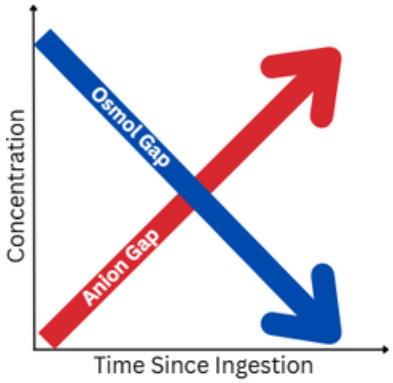
Pathophysiology

The clinical toxicity of methanol is primarily mediated by its major metabolite, formic acid which exerts its toxicity through inhibition of mitochondrial cytochrome c oxidase, leading to disruption of oxidative phosphorylation, impaired cellular respiration, and resultant cellular hypoxia. This process culminates in the development of a high anion gap metabolic acidosis (AGMA). In severe cases, accumulation of formic acid is associated with selective toxicity to the optic nerve, which may result in optic neuropathy and permanent visual impairment.

Clinical toxicity and evaluation

Early or mild methanol toxicity typically manifests with nonspecific symptoms, including abdominal pain, nausea, vomiting and central nervous system depression. As toxicity progresses, patients may develop high AGMA, tachycardia, hypotension, seizures, and coma. Visual disturbances ranging from blurred vision to complete and potentially irreversible blindness are a hallmark feature and may be delayed (Barceloux et al., 2002).

Methanol toxicity may present with a broad and evolving clinical spectrum, rendering diagnosis challenging in the absence of a reliable exposure history. The onset of clinical manifestations may be delayed up to 24 hours, particularly in cases of co-ingestion with alcohol dehydrogenase inhibitors such as ethanol or certain antiretroviral agents (e.g., abacavir), which can prolong the metabolism of methanol to its toxic metabolites (Kraut, 2016).



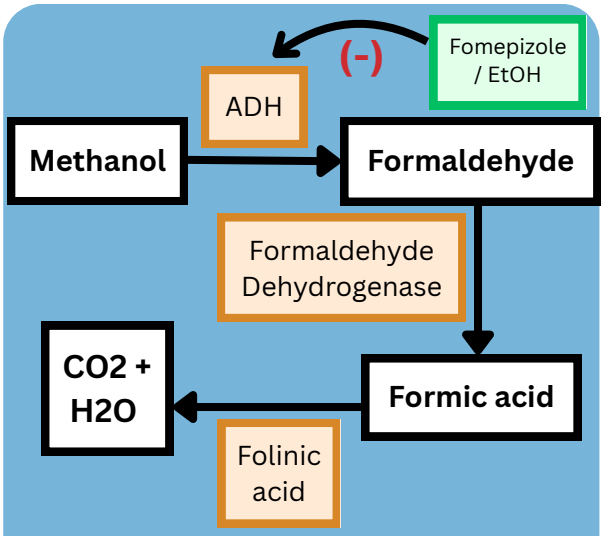
The diagnostic gold standard for methanol poisoning is direct measurement of the serum or plasma methanol concentration. However, this test is not always rapidly available and does not reliably correlate with clinical severity. In Ohio, several laboratories can perform methanol testing, and clinicians should promptly contact their regional poison control center for assistance to locate them. Every effort should be made to obtain a level as quickly as possible. In situations where assay availability is limited or delayed, the serum methanol concentration may be estimated using the osmolar gap, applying a correction factor of approximately 3.2. As hepatic metabolism proceeds, the osmolar gap declines while the anion gap increases, reflecting the accumulation of formic acid. Interpretation of the osmolar gap requires adjustment in the presence of co-ingested ethanol, as elevated ethanol concentrations contribute to serum osmolality and may confound estimation.

Treatment

In methanol poisoning, inhibition of alcohol dehydrogenase should be initiated when serum methanol concentrations exceed 20 mg/dL and continued until levels decline below this threshold. Both fomepizole and ethanol are effective antidotal agents that prevent the formation of the toxic metabolite, formic acid.

Fomepizole is the preferred agent due to its predictable pharmacokinetics and lack of additional central nervous system depression. Ethanol may be used as an alternative; however, its administration requires close monitoring.

Hemodialysis provides rapid and efficient clearance of both methanol and formic acid and should be strongly considered in cases with markedly elevated methanol concentration (eg. > 70 mg/dL), or severe toxicity, including those with significant metabolic acidosis, visual disturbances, or end-organ dysfunction. Intermittent hemodialysis is generally favored over continuous renal replacement modalities (Roberts et al., 2015). Adjunctive therapy with leucovorin (folic or folinic acid) is recommended, as it may enhance the metabolism of formic acid to carbon dioxide and water (Kraut, 2016).



Key Points:

- Methanol is a life-threatening toxic alcohol found in common industrial and household products.
- Visual disturbance is a hallmark feature of methanol toxicity.
- Obtaining a methanol level is the gold standard for diagnosis.
- Early treatment is critical and focuses on supportive care and ADH blockade with fomepizole or ethanol.
- Folate can be used as adjunct therapy.
- Consider hemodialysis in severe cases.

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