A Case of Intentional Methanol Ingestion
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Learning Objective
To understand the complications and management of methanol toxicity.

Case Presentation
History of Present Illness
• 41 year old female with depression, seizure disorder, alcohol abuse, and history of multiple suicide attempts presented to the emergency department reporting that she had ingested anti-freeze and alcohol and inhaled gasoline in an attempt to commit suicide.

Physical Exam
• Normal vital signs
• Mild suprapubic tenderness
• Slurred speech and inappropriate affect but alert and answer questions

Labs
• ABG – pH 7.27, PaCO2 13 mmHg, PaO2 117 mmHg, Bicarbonate 6 mEq/L
• CBC - WBC 7.57 K/uL, Hgb 10.8 g/dl, Platelets 392 K/uL
• BMP – Na 143 mEq/L, K 5.8 mEq/L, CO2 < 10 mmol/L, BUN 10 mg/dl, Cr 0.71 mg/dl, Anion Gap > 32; osmolality 495 mOsm/kg (osmolar gap 188 mOsm/kg)
• Toxicology screen - Methanol >200 mg/dl but otherwise negative including ethanol and ethylene glycol
• EKG - sinus rhythm, PR interval of 108, otherwise normal

Progress and Outcome
• She was started on sodium bicarbonate and fomepizole and admitted to the MICU.
• High dose folate was started and emergent hemodialysis was performed.
• Ophthalmology was consulted and recommended IV solumedrol followed by an oral prednisone taper to prevent toxic optic neuropathy.
• Patient improved quickly and was transferred to medicine then discharged home after psychiatry clearance.

Discussion
• Any methanol ingestion of more than 1 mg/kg may be lethal.
• Methanol itself is relatively non-toxic, causing only CNS depression; the more severe manifestations of methanol toxicity are related to the breakdown product formic acid or formate.
• Metabolism of methanol: alcohol dehydrogenase oxidizes methanol to form formaldehyde which is then oxidized to formic acid. Formic acid is oxidized to non-toxic carbon dioxide and water by folate dependent reactions.
• Initial manifestations of methanol overdose can be mild confusion and the appearance of intoxication.
• Laboratory testing classically shows significant metabolic acidosis with extremely high anion gap and high osmolar gap.
• Following stabilization of airway, circulation, and breathing, initial management includes assessment of end organ damage, co-ingestions, and laboratory abnormalities.
• Initiation of sodium bicarbonate can reduce end organ damage, which can be worsened by systemic acidemia. An acidic environment promotes formic acid deposition, particularly in the eyes.
• Fomepizole is used to inhibit alcohol dehydrogenase.
• Leucovorin or high dose folate should also be used to stimulate formic acid metabolism.
• Indications for dialysis include severe metabolic acidosis and evidence of retinal damage.

Ophthalmologic manifestations can include mydriasis, retinal edema leading to retinal sheen, afferent pupillary defect, and, most commonly, toxic optic neuropathy.
• Studies have shown the optimal treatment of toxic optic neuropathy is high dose solumedrol followed by a prednisone taper.
• It is important to assess the mental health of patients treated for methanol ingestion to determine intent and risk of additional self-harm.
• Successful treatment of methanol ingestion requires early recognition and aggressive multifactorial medical management aimed at decreasing the levels of methanol and its metabolites and preventing long-term end organ damage.

References