Alcohol-Related Emergencies

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Cover Illustration by Andrew Grivas, MA, CMI

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INTRODUCTION

Toxic conditions from ethanol, methanol, isopropanol (isopropyl alcohol), or ethylene glycol ingestion are routinely encountered by emergency physicians. Although these alcohols are related in terms of chemical structure, they produce different clinical effects when ingested. Emergency physicians should be familiar with the pathophysiology and clinical features of alcohol toxicity and know how to manage toxic ingestions of each alcohol. In addition, they should be able to recognize and treat conditions associated with chronic ethanol use.

ETHANOL INGESTION

Ethanol is the most commonly abused substance in the United States. It is reportedly involved in 30% of all motor vehicle crashes and 47% of all fatal crashes. Ethanol is associated with 50% of all accidental deaths, and it has been calculated that ethanol use reduces an individual’s life expectancy by 12 years when all factors are considered. One study showed that 33% of all evening emergency department patients were intoxicated. It has been estimated that there are at least 10 million alcohol abusers or dependent drinkers in the United States. Although it may have limited use in the management of alcohol-related emergencies, the CAGE questionnaire (Appendix) may be used to screen for alcohol problems.

ACUTE ETHANOL INTOXICATION

Metabolism

Ethanol is absorbed primarily from the stomach and small intestines. The rate of absorption depends on the concentration of ethanol in the beverage ingested and the presence or absence of food in the gastrointestinal tract.
tract. Peak blood levels of ethanol occur 30 to 90 minutes after ingestion. Ethanol is metabolized in the liver via 3 enzyme pathways, although only 2 are important: alcohol dehydrogenase and a microsomal ethanol oxidizing system (MEOS). Alcohol dehydrogenase metabolizes the vast majority of ethanol by converting it to acetaldehyde. The acetaldehyde is converted to acetic acid by aldehyde dehydrogenase. The acetic acid is converted to acetyl coenzyme A (CoA), then to carbon dioxide and water. Interestingly, the MEOS is responsible for up to 25% of metabolism in alcohol abusers but only 5% in other persons. Ethanol follows zero-order kinetics, although the MEOS can change this to first-order when high levels are present. In most persons, ethanol is metabolized at a rate of approximately 15 to 20 mg/dL per hour, while in alcohol-dependent persons the rate of metabolism is approximately 30 mg/dL per hour.

Clinical Features

The main clinical effect of ethanol is depression of the central nervous system (CNS). Initially, there are changes in mood, judgment, and fine motor coordination. As the blood level of ethanol rises, slurred speech, nystagmus, ataxia, lethargy, and coma can occur. Tachycardia, hypothermia, and hypotension also may be observed. Chronic alcohol users develop tolerance to the CNS effects of ethanol but not to its respiratory effects; death from respiratory depression may occur at any ethanol level.

Evaluation and Treatment

The evaluation of a patient with alcohol ingestion consists of a thorough history and physical examination with attention to signs of infection or trauma. If the patient has an altered level of consciousness, blood glucose level should be checked or dextrose administered empirically. Thiamine 100 mg and naloxone should be administered intravenously (IV) as well. A complete blood count (CBC) should be obtained, and electrolyte and calcium levels should be measured. Assessment of liver enzymes and prothrombin time should be considered, particularly in chronic alcohol abusers. Obtaining magnesium levels is controversial since the serum level is an inaccurate measure of the total body stores. However, magnesium should be administered to alcohol abusers. A computed tomography (CT) scan of the head should be obtained for individuals presenting with first-time seizures, focal seizures, or mental status that does not improve. CT scanning may identify spontaneous or chronic subdural hematomas, which may result from coagulopathies or acute or remote head trauma from injury or falls.

Obtaining a serum ethanol level is indicated when the cause of the patient’s altered level of consciousness is in doubt. Measuring the ethanol level in an awake patient who admits to ethanol use or is a chronic ethanol abuser can sometimes be problematic because individuals with an increased tolerance to ethanol can appear sober and can function safely despite substantial levels. For example, some chronic abusers with an ethanol serum level of 200 mg/dL appear sober, while novice or binge drinkers are intoxicated at this level. Thus, checking serum ethanol level raises the issue of determining what level must be reached before the patient can be safely discharged. In addition, if one checks a level in a chronic abuser then waits for the level to reach zero, the issue of impending withdrawal syndromes develops. The approach of most emergency physicians is not to check ethanol serum levels. Thus, in an awake patient who admits to alcohol ingestion, management begins with frequent reexaminations and documentation of improving mental status. If possible, a meal should be provided. When the patient is awake and alert and can ambulate safely, preparations for discharge should be made.

Disposition with this subset of patients can be difficult. Patients who have a safe home and a responsible person to observe them can be discharged. More often, however, chronic abusers have more tenuous living environments, and social service resources will need to be accessed. Referral to detoxification programs should be offered.

CONDITIONS ASSOCIATED WITH CHRONIC ETHANOL USE

Alcoholic Ketoacidosis (AKA)

AKA is an anion gap acidosis that occurs secondary to high levels of ketoads in the body tissues and fluids. AKA occurs most commonly in binge drinkers who have not eaten in several days, but it also occurs in chronic alcohol abusers. A number of mechanisms that lead to ketosis are believed to contribute to AKA, including mobilization of free fatty acids from adipose tissues, the conversion of acetic acid to ketones, vomiting and starvation, and a reduction in available nicotinamide adenine dinucleotide (NAD) in liver mitochondria. These conditions lead to increased levels of β-hydroxybutyrate and acetoacetate, with the former in much higher concentrations.

Patients with AKA present with a history of recent heavy ethanol use with little or no food intake over several days. They will complain of diffuse abdominal pain, nausea, and vomiting. Both alcohol and any food intake are discontinued after the development of nausea and vomiting. Patients may have tachycardia and dehydration.
and be mildly febrile. Laboratory abnormalities consist of a metabolic acidosis, often with a compensatory respiratory alkalosis and/or metabolic alkalosis secondary to emesis. Both serum and urine ketone assays may be low or normal because the nitroprusside reaction (the standard assay for ketones) is unable to detect β-hydroxybutyrate. As the patient’s condition improves, increasing levels of ketones may be measured by nitroprusside reaction as the β-hydroxybutyrate is converted to acetoacetate.

Treatment of AKA consists of administration of dextrose-containing IV fluids, thiamine, and a meal once the patient is able to eat; antiemetics may be necessary. Hospital admission is necessary for patients with persistent vomiting, acidosis, or abdominal pain. AKA usually resolves within hours after treatment and in 1 to 2 days without treatment.

**Alcohol Withdrawal**

Alcohol withdrawal is a potentially fatal syndrome that can occur in alcohol-dependent persons following the cessation of ethanol use. The spectrum of clinical findings in alcohol withdrawal is divided into 4 overlapping stages (Table 1). Stage 1 includes intention tremors, anxiety, gastrointestinal upset, mild tachycardia, hyperthermia, and hypertension. These effects last up to 3 days, and often patients will treat themselves with alcohol (the “eye opener”). Most patients do not progress past this stage.

Stage 2 is alcohol hallucinosis. Hallucinations usually are visual or auditory but can be tactile or olfactory. A second phase of this stage begins 3 to 4 days after cessation of alcohol. In this phase, the autonomic changes are absent, the hallucinations are primarily auditory, and the patient’s behavior may mimic that of a paranoid schizophrenic. Stage 3 is manifest by seizures. Up to 10% of untreated patients will have a withdrawal seizure, and one third of these progress to delirium tremens. Seizures can be multiple, are most commonly generalized tonic-clonic, and rarely recur after 6 hours. Focal seizures, status epilepticus, or seizures occurring outside the 6-hour window should prompt clinicians to search for another etiology (eg, structural, metabolic, or infectious causes). Stage 4 is delirium tremens. This stage is rare, occurring in only 4% to 6% of untreated patients. Patients in this stage present with severe confusion, agitation, delusions, tremors, and autonomic hyperactivity. This stage is almost always preceded by a withdrawal seizure. Its onset is usually 3 to 5 days after cessation of alcohol but can occur as late as 14 days. The mortality rate of delirium tremens is 5% to 15%, making this a true medical emergency.

Treatment of all stages of alcohol withdrawal includes reversing metabolic abnormalities (especially hypoglycemia) and administering glucose-containing IV fluids, thiamine, magnesium, and benzodiazepines. The goal of treatment with benzodiazepines is symptom control. Benzodiazepines cross-react with ethanol, controlling tremors and autonomic hyperactivity, and they may prevent progression to seizures and delirium tremens. Diazepam and lorazepam are commonly recommended. One must balance the effects of the benzodiazepines (CNS depression) with the resolution of autonomic abnormalities in the patient. There is no role for phenytoin in the treatment of alcohol withdrawal seizures.

Patients with delirium tremens, alcohol withdrawal unresponsive to medical therapy, or a decreasing level of consciousness should be admitted. Patients with mild withdrawal can be discharged with a family member or responsible adult following therapy and referral for detoxification.

**Wernicke’s Encephalopathy**

Wernicke’s encephalopathy occurs as a result of thiamine deficiency. Frequent ingestion of ethanol, which reduces absorption of thiamine from the gastrointestinal tract, along with an alcohol abuser’s poor nutrition

<table>
<thead>
<tr>
<th>Table 1. Stages of Alcohol Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage 1:</strong> tremors, anxiety, mild hypertension, and mild tachycardia</td>
</tr>
<tr>
<td>Onset: 6–8 hours</td>
</tr>
<tr>
<td>Peak: 24–36 hours</td>
</tr>
<tr>
<td>Duration: 48–72 hours</td>
</tr>
<tr>
<td><strong>Stage 2:</strong> most commonly, auditory and visual hallucinations occur; tactile or olfactory hallucinations occur less often</td>
</tr>
<tr>
<td>Onset: 6–24 hours</td>
</tr>
<tr>
<td>Peak: 24–48 hours</td>
</tr>
<tr>
<td>Duration: 48–72 hours</td>
</tr>
<tr>
<td><strong>Stage 3:</strong> seizures</td>
</tr>
<tr>
<td>Onset: 7–48 hours</td>
</tr>
<tr>
<td>Peak: 12–24 hours</td>
</tr>
<tr>
<td>Duration: 6–12 hours</td>
</tr>
<tr>
<td><strong>Stage 4:</strong> delirium tremens</td>
</tr>
<tr>
<td>Onset: 3–5 days</td>
</tr>
<tr>
<td>Peak: variable</td>
</tr>
<tr>
<td>Duration: 2–5 days</td>
</tr>
</tbody>
</table>

can lead to the development of Wernicke’s. The classic triad of ataxia, ophthalmoplegia, and altered mental status actually occurs only 16% of the time. Thiamine deficiency can also lead to wet beriberi and/or a peripheral neuropathy. Treatment is thiamine 100 mg IV, which can be given safely before glucose. Ophthalmoplegia is the first symptom to reverse, usually within hours.

**Gastrointestinal Disorders**

Alcohol is a risk factor for the development of gastrointestinal disorders. Alcohol abusers are at higher risk for esophagitis and gastric and esophageal cancers compared with those who do not abuse alcohol. Persistent emesis, common in alcohol abusers, can lead to Mallory-Weiss tears or Boerhaave’s syndrome (esophageal perforation). Alcoholic gastritis is the most frequent cause of gastrointestinal bleeding in alcohol abusers, and esophageal varices may cause significant gastrointestinal bleeding. Up to 30% of cirrhotic patients develop varices, which can lead to life-threatening bleeding. Careful nasogastric intubation is a safe and necessary part of the evaluation of gastrointestinal bleeding. Alcohol abusers may have conditions that make hemorrhage control difficult, including coagulopathies due to liver disease or thrombocytopenia. Physical examination may reveal dark stools, melena, or gross blood, depending on the severity and duration of the bleeding. Evaluation includes a CBC, assessment of electrolyte and calcium levels, prothrombin time, and thromboplastin time, and type and screen/crossmatch of blood. Management includes establishing IV access, replenishing volume with either isotonic saline and/or blood products, and nasogastric intubation to evaluate ongoing blood loss. Alcohol abusers with upper gastrointestinal bleeding should be admitted to the hospital. Those with ongoing blood loss and/or hemodynamic instability should be admitted to the intensive care unit.

**Pancreatitis**

Ethanol use is the most common cause of chronic pancreatitis and is one of the most common causes of acute pancreatitis. Patients with pancreatitis will present with nausea, vomiting, and epigastric pain often radiating to the back and will frequently have tachycardia due to dehydration. Examination will reveal epigastric tenderness, usually without rebound because of the retroperitoneal location of the pancreas. Evaluation includes a CBC and measurement of electrolyte, calcium, lactate dehydrogenase (LDH), aspartate transaminase (AST), amylase, and lipase levels. Of note, the lipase level is a more accurate marker than amylase, which is nonspecific. A nasogastric tube should be used in all but the mildest cases of pancreatitis. Management includes administration of IV fluids, analgesia, and antibiotics. Complications of pancreatitis include pleural effusions, hypocalcemia, hyperglycemia, hemorrhagic pancreatitis, and, in the most severe cases, disseminated intravascular coagulation (DIC), acute respiratory failure, and myocardial depression.

Ranson’s criteria are used to predict outcome in acute pancreatitis (Table 2). The presence of fewer than 5 criteria is associated with a mortality rate of less than 1%; 3 or 4 criteria, 15%; 5 or 6 criteria, 40%; and 7 or more criteria, 100%. All patients with acute pancreatitis should be admitted to the hospital for IV hydration and pain control. Patients with chronic pancreatitis who are stable, able to tolerate oral fluids, have adequate pain control, and have good follow-up may be discharged.

**Infection**

Chronic ethanol use results in depression of cell-mediated immunity and damage to polymorphonuclear (PMN) leukocytes, leading to immunosuppression. The most common infectious disease in alcohol abusers is pneumonia, but these individuals are also susceptible to spontaneous bacterial peritonitis (SBP), tuberculosis, and endocarditis. *Streptococcus pneumoniae* is the most common cause of pneumonia in alcohol abusers, but alcohol abuse is a risk factor for *Klebsiella*,

### Table 2. Criteria for Predicting Outcome in Acute Pancreatitis

<table>
<thead>
<tr>
<th>Admission</th>
<th>Initial 48 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 55 years</td>
<td>Hematocrit decrease &gt; 10%</td>
</tr>
<tr>
<td>White blood cell count &gt; 16,000/mm³</td>
<td>Blood urea nitrogen increase &gt; 5 mg/dL</td>
</tr>
<tr>
<td>Glucose &gt; 200 mg/dL</td>
<td>Ca²⁺ &lt; 8 mg/dL</td>
</tr>
<tr>
<td>Lactate dehydrogenase &gt; 350 U/L</td>
<td>PaO₂ &lt; 60 mm Hg</td>
</tr>
<tr>
<td>Aspartate transaminase &gt; 250 U/L</td>
<td>Base deficit &gt; 4 mEq/L</td>
</tr>
<tr>
<td></td>
<td>Fluid sequestration &gt; 6 L</td>
</tr>
</tbody>
</table>

Methanol is made by distilling wood and is most commonly found in antifreeze, canned fuel, paint solvent, and windshield cleaner. It is rapidly absorbed from the gastrointestinal tract, and 90% to 95% is metabolized in the liver. The potentially lethal dose is 0.4 mL/kg of a 40% solution of methanol (approximately 30 mL for an adult). There have been reports of toxicity from inhalation.

**METABOLISM**

Methanol is eliminated primarily via hepatic metabolism. It is oxidized by alcohol dehydrogenase in the liver to formaldehyde, then to formic acid by aldehyde dehydrogenase. These 2 compounds, particularly formic acid, are responsible for methanol’s toxicity. Formic acid inhibits cytochrome-c oxidase and mitochondrial metabolism, leading to cellular hypoxia, and causes the profound anion gap metabolic acidosis and ocular toxicity that occur with methanol ingestion. Lactate, acetate, and butyrate also contribute to the acidosis.

**CLINICAL FEATURES**

A history of ingestion is often absent or unreliable. The 4 major signs and symptoms of methanol toxicity are visual changes, abdominal pain with nausea and vomiting, CNS depression, and metabolic acidosis. The latent period between ingestion and symptom onset is usually 12 to 24 hours, but latent periods as brief as 40 minutes and as long as 72 hours have been reported. Co-ingestion of ethanol may prolong the latent period.

Visual complaints such as cloudy, misty, or blurred vision, photophobia, and descriptions of “looking into a snowstorm” occur frequently in symptomatic cases. On examination, the pupils are often fixed and dilated, and the optic discs will be hyperemic; however, the fundus can appear normal, even in patients with visual symptoms. Papilledema may occur, sometimes within hours of ingestion. These visual signs and symptoms correlate with the onset of the acidosis. Gastrointestinal symptoms in methanol toxicity include a triad of nausea, vomiting, and abdominal pain, and patients often develop a hemorrhagic gastritis and pancreatitis. However, it is important to note that gastrointestinal signs and symptoms can be absent in a significant ingestion. CNS complaints range from asymptomatic to obtundation. Headache and vertigo are common complaints. Seizures may occur, but the classic cerebral edema is found only about 10% of the time. Methanol is less intoxicating than ethanol, so patients may have toxic levels without seeming intoxicated. The metabolic acidosis with a high anion gap occurs when methanol is converted to formic acid. Methanol toxicity is one of the rare instances when the serum bicarbonate level can be 0, but because this occurs several hours after ingestion, an elevated osmolar gap is often the only early abnormality. However, the osmolar gap can be normal soon after ingestion; therefore, measurement of serum methanol level is the most reliable laboratory diagnostic tool.

Cardiovascular effects are rare, but shock, bradycardia, and myocardial depression have been reported.

**LABORATORY TESTING**

The workup of suspected methanol ingestion includes a CBC and measurement of ABG, BUN, serum
osmolality, and serum levels of electrolytes, glucose, calcium, magnesium, creatinine, and methanol. From this information, the anion gap (AG), serum osmolality, and the osmolar gap (OG) should be calculated and compared to normal values (AG = 8–12 mEq/L, OG = 10 mOsm/kg). The OG is the difference between the measured osmolality and the calculated osmolality:

\[
AG = Na^+ - (Cl^- + HCO_3^-)
\]

Osmolality (calculated) = \(2(Na^+)+ \frac{glucose}{18} + \frac{BUN}{2.8}\)

An elevated osmolar gap signifies that osmotically active substances (eg, ethanol, methanol, ethylene glycol, isopropanol, mannitol, and/or glycerol) are present in the patient's serum. An elevated anion gap should prompt a search for the cause. The mnemonic MUDPILES (methanol, uremia, diabetic ketoacidosis [DKA], paraldehyde, isoniazid/iron, lactic acidosis, ethylene glycol/ethanol, salicylates) is helpful for remembering the potential causes of an elevated anion gap.

**TREATMENT**

As in all emergencies, airway, breathing, and circulation should be the priorities in any patient with methanol ingestion. If the patient has an altered level of consciousness, glucose, thiamine, and naloxone should be administered. Most authorities suggest that gastric lavage be performed (with the airway protected) if the ingestion occurred within 2 hours of presentation. Charcoal is of little use in methanol ingestion because it binds poorly to alcohols; however, it should be administered if a co-ingestion is suspected.

The treatment goals for methanol ingestion include correction of acidosis, inhibition of further metabolism, and removal of the substance and metabolites via dialysis. The metabolites of methanol and ethylene glycol cause a significant metabolic acidosis that is difficult to correct with bicarbonate, unlike a lactic acidosis. Treatment with bicarbonate should begin when the serum pH is below 7.2, which in some cases may reverse the visual impairment.

Inhibition of further metabolism of methanol can be accomplished in 2 ways. Because ethanol's affinity for alcohol dehydrogenase is 10 times greater than that of methanol, an ethanol infusion will prevent further metabolism of methanol to the toxic metabolites. A loading dose of 0.8 g/kg and maintenance dose of 0.15 g/kg/hr of 100% ethanol should be administered intravenously or orally with a goal of obtaining serum ethanol levels between 100 and 150 mg/dL. Individuals tolerant of ethanol will need higher rates to obtain the same levels. 4-Methylpyrazole (4-MP), or fomepizole, was recently approved by the U.S. Food and Drug Administration (FDA) for treatment of ethylene glycol ingestion. Fomepizole inhibits alcohol dehydrogenase, making ethanol administration unnecessary. Although fomepizole is not yet FDA-approved for methanol ingestions, cases of successful treatment of methanol ingestions using fomepizole have been reported. In addition, at least 2 studies have suggested that fomepizole eliminates the need for dialysis. The loading dose of fomepizole is 10 mg/kg for 48 hours then 15 mg/kg every 12 hours.

Folate should be administered at a dose of 50 mg IV every 4 hours. Folate is the cofactor in the final step of degradation, breaking formic acid into water and carbon dioxide. Subclinical deficiencies of folate will slow this step.

Hemodialysis is the definitive treatment for methanol ingestion. Although administration of ethanol or fomepizole will slow metabolism of the parent compounds to delay the production of more toxic metabolites, only dialysis will remove them. Dialysis should be performed for methanol ingestion if the patient has acidosis, acute renal failure, visual symptoms, or serum levels of methanol greater than 25 mg/dL.

All patients with a history of methanol ingestion should be admitted because of the delayed onset of symptoms and the frequent unreliability of the history. Any patient with hemodynamic or airway compromise or any signs or symptoms of toxicity should be admitted as well.

**ETHYLENE GLYCOL INGESTION**

Ethylene glycol is a sweet-tasting, colorless, odorless alcohol most commonly found in antifreeze. The potentially lethal dose is 1.0 to 1.5 mL/kg. Ethylene glycol has a plasma half-life of 3 to 6 hours, which is increased to 17 hours by ethanol.

**METABOLISM**

Ethylene glycol is readily absorbed in the gastrointestinal tract, with peak blood levels occurring 1 to 4 hours after ingestion. It is metabolized in the liver and renally excreted. Like methanol, only its metabolites are toxic; these metabolites affect the brain, lungs, kidneys, and heart.

Ethylene glycol is metabolized by alcohol dehydrogenase to glycolaldehyde, which is then metabolized to glycolic acid and glyoxylic acid. Glyoxylic acid is then
changed into 6 different compounds, the most important being oxalic acid. Oxalic acid is responsible for end-organ damage, while glycolic acid is responsible for the metabolic acidosis.

**CLINICAL FEATURES**

The clinical presentation of ethylene glycol ingestion can be divided into 3 stages: CNS, cardiopulmonary, and renal. The CNS stage is usually the first and begins 30 minutes to 12 hours after ingestion. The patient acts as if intoxicated on ethanol and may have slurred speech, ataxia, altered mental status, and/or hallucinations but will not have an odor of ethanol on his or her breath. Ocular complaints are very rare and should alert the physician to a possible co-ingestion. The cardiopulmonary stage usually occurs 12 to 24 hours after ingestion and is characterized by hypertension, tachycardia, congestive heart failure, and/or tachypnea. The renal stage occurs 1 to 3 days after ingestion. Patients may complain of flank pain and may have either oliguric or anuric renal failure. Renal failure is secondary to the deposition of oxalate crystals in renal tubules and direct toxicity of various metabolites. Urinalysis will show hematuria and proteinuria, and approximately 50% of patients will have the classic calcium oxalate crystals. Renal failure may be partially reversible with supportive care.

**LABORATORY TESTING**

The workup of a suspected ethylene glycol ingestion should include a CBC, urinalysis, assessment of ABG, and measurement of serum levels of electrolytes, BUN, creatinine, glucose, calcium, magnesium, ethanol, and ethylene glycol. Since fluorescein is added to commercial antifreeze, the urine of patients who have ingested this substance will glow when illuminated by a Wood’s lamp. The white blood cell count is frequently elevated, but this finding is nonspecific. Urinalysis will often reveal the calcium oxalate crystals. Approximately one third of these patients will have hypocalcemia secondary to the calcium precipitating with oxalate. As with methanol poisoning, a severe anion gap metabolic acidosis is often present secondary to glycolic and glyoxylic acid buildup.

**TREATMENT**

The treatment goals for ethylene glycol ingestion are identical to those for methanol ingestion (ie, correction of acidosis, inhibition of further metabolism, and removal of the substance and metabolites via dialysis). Fomepizole is FDA-approved for this ingestion; it should be administered at a loading dose of 10 mg/kg for 48 hours, then 15 mg/kg should be given every 12 hours. Indications for hemodialysis in ethylene glycol ingestion are the same as in methanol ingestion. Finally, when the patient has adequate concentrations of thiamine and pyridoxine, glyoxylic acid is degraded into 2 nontoxic compounds rather than oxalic acid. Thus, thiamine 100 mg and pyridoxine 100 mg should be administered intravenously to drive the reaction towards the nontoxic metabolites. All patients with ethylene glycol ingestion should be admitted.

**ISOPROPANOL (ISOPROPYL ALCOHOL) INGESTION**

Isopropanol is a clear, colorless alcohol found in rubbing alcohol and various skin and hair products. It is the second most commonly ingested alcohol. It is less toxic than methanol and ethylene glycol but more toxic than ethanol. The potentially lethal dose is 2 to 4 mL/kg. Isopropanol is a strong CNS depressant, as is its metabolite, acetone. Deaths from this ingestion are rare.

**METABOLISM**

Isopropanol is absorbed from the gastrointestinal tract; skin absorption occurs only after prolonged contact. It is metabolized in the liver to acetone by alcohol dehydrogenase. Acetone is primarily excreted by the kidneys, but it is also partially excreted via the lungs and may be smelled on the patient’s breath. The half-life of acetone is 29 hours. Isopropanol is 2 to 3 times more potent as a CNS depressant than ethanol, partially because of the depressant affects of acetone. Other major effects include gastrointestinal irritation, vasodilation, and cardiac depression.

**CLINICAL FEATURES**

Patients with isopropanol intoxication present primarily with CNS and gastrointestinal signs and symptoms, including CNS depression, nausea and vomiting, and abdominal pain. Severe ingestions may cause respiratory depression and hypotension (secondary to myocardial depression). The patient will appear intoxicated but will not smell of ethanol; he or she may smell of acetone. Gastric irritation is an early finding, and hemorrhagic gastritis may occur. Onset of effects occurs 30 to 60 minutes after ingestion, and effects may last up to 24 hours. Rhabdomyolysis and acute tubular necrosis are also well-documented complications of this ingestion; acute hepatitis and hemolytic anemia are rare.
LABORATORY TESTING

The laboratory workup consists of a CBC, urinalysis, assessment of ABG and serum osmolality, and measurement of serum levels of electrolytes, BUN, creatinine, glucose, calcium, magnesium, and ketones. Because acetone is not a ketoacid, an acidosis is not prominent. Acetone can be found as a ketone in the serum within 30 minutes of ingestion and in the urine within 3 hours. Isopropanol causes an osmolar gap, but if an elevated anion gap is present, a co-ingestion should be suspected. Hypoglycemia and a falsely elevated creatinine level (due to acetone’s interference with the assay) are common.

TREATMENT

Gastric lavage can be performed if the ingestion occurred within 2 hours of presentation. As with all alcohols, activated charcoal is of little use. Overall, treatment is supportive with attention to hemodynamic status. Hypotension should be corrected with fluids and vasopressors. Dialysis is useful, but most experts recommend dialysis only in the setting of refractory hypotension or an isopropanol level greater than 400 mg/dL.

All patients with isopropanol ingestions should be observed for 6 hours. If the patient remains hemodynamically stable and shows no signs of CNS depression, he or she can be discharged with appropriate follow-up.

APPENDIX

CAGE Questionnaire

C Have you ever felt that you need to Cut down on your drinking?
A Have people Annoyed you by criticizing your drinking?
G Have you ever felt bad or Guilty about your drinking?
E Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (ie, an Eye-opener)?

Adapted with permission from Ewing JA. Detecting alcoholism: the CAGE questionnaire. JAMA 1984;252:1905–1907.

REFERENCES


SUGGESTED READINGS


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