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Ethanol Toxicity

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Continuing Education Activity

Ethanol toxicity results from the ingestion of large amounts of ethanol, usually in the form of alcohol. It affects multiple organ systems in both the acute and chronic phases. This activity outlines the evaluation and management of ethanol toxicity and reviews the interprofessional team's role in managing patients with this condition.

Objectives:

- Identify the etiology of ethanol toxicity.
- Outline the appropriate evaluation of ethanol toxicity.
- Review the management options available for ethanol toxicity.
- Describe interprofessional team strategies for improving care coordination and communication of ethanol toxicity and improving outcomes.

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Introduction

Ethanol toxicity results from the ingestion of ethanol, usually in large quantities. This can occur from the ingestion of beverage ethanol, commonly known as alcohol, and non-beverage ethanol, present in substances such as mouthwash, cologne, and cough medicine. Alcohol is the most common form of ethanol and is a widely used and abused substance, mostly in Western culture, representing the oldest and most widely abused substance. The demographic most likely to present for acute alcohol intoxication are adolescents and young adults. Emergency departments see disproportionately more of these patients than in other settings.[1][2]

Etiology

Ethanol toxicity can occur in both acute and chronic settings, representing two different spectrums of disease. Acute ethanol intoxication usually follows the ingestion of a large amount of alcohol and is a clinically harmful condition.[1]

Epidemiology

No demographic group is unaffected by alcohol, but adolescents and young adults are most likely to present for intoxication and toxicity. They are also most likely to present for traumatic injuries sustained while drinking alcohol. Approximately 3.3 million deaths can be attributed to alcohol use; it is the fourth leading preventable cause of death in the United States. One in 12 adults has alcohol use disorder, which is defined as more than 3 drinks a day in men and more than 2 drinks a day in females or binge drinking.[2][3]

Pathophysiology

Alcohol is absorbed through the proximal GI tract. It is primarily metabolized in the liver by alcohol dehydrogenase to acetaldehyde. The primary site of action in acute toxicity is the central nervous system, where it increases central nervous system (CNS) inhibition and decreases excitation. Gamma-aminobutyric acid (GABA) is the primary CNS inhibitory neurotransmitter. GABA binds to receptors allowing chloride to enter the cell, which decreases cellular excitability. Alcohol binds strongly to GABA receptors, activating the inhibitory cascade, which results in sedation, cognitive dysfunction, and decreased coordination.

With chronic alcohol use, the number of GABA receptors is increased, requiring more and more alcohol to create the same level of inhibition. This is a phenomenon known as tolerance. This tolerance partly explains the alertness of chronic alcohol users at blood alcohol levels that, in others, would cause coma or death. Benzodiazepines also bind to the GABA receptor, making them useful in alcohol withdrawal. Alcohol also inhibits the primary excitatory neurotransmitter in the CNS, glutamate. Patients with alcohol use disorder have increased numbers of NMDA receptors and increased sensitivity of these receptors to glutamate. Due to the increased sensitivity of these receptors, patients with alcohol use disorder are at risk for seizures and hallucinations when alcohol is withdrawn.[2][4][5]

History and Physical

Criteria for diagnosing alcohol intoxication include known or admitted ingestion of alcohol, behavior change, clinical signs including slurred speech, incoordination, nystagmus, memory loss, and lack of another condition to account for the symptoms. The signs and symptoms associated with alcohol toxicity depend on the blood alcohol concentration (BAC). As the BAC increases, so does the severity of the symptoms. At a BAC of 0 to 0.05%, one would expect to see relaxation, increased talkativeness, and decreased fine motor control. At a BAC of 0.05% to 0.1%, patients develop impaired judgment and coordination. From 0.1% to 0.2%, one sees gait instability, slurred speech, and mood and behavior changes. At a BAC of 0.2% to 0.4%, patients develop nausea and vomiting, hypothermia, dysarthria, amnesia, diplopia, and nystagmus. With a BAC of greater than 0.4%, patients can develop respiratory depression followed by coma and even death. The extent and severity of these symptoms vary depending on how quickly the alcohol is ingested and the rapidity of the rise and fall of the BAC.

The speed of absorption can be affected by co-ingested food, female sex, cigarette use, and concentration of alcohol in the beverage. It is also affected by tolerance to alcohol; a significant history of alcohol use can allow a patient to be conscious, cohesive, and free of motor deficits at BACs that would cause severe symptoms in patients without tolerance. It is important to ascertain the quantity and type of alcohol consumed and over which period of time it was consumed. Patients may complain of nausea, vomiting, and diarrhea. A full physical exam is necessary, with special attention paid to the vital signs, nutritional status of the patient, and skin findings such as capillary prominence, spider naevi, telangiectasia, palmar erythema, and muscular atrophy. As with all physical exams, airway, breathing, and circulation should be the first focus. Acute alcohol intoxication can cause respiratory depression; establishing that the patient is protecting their airway is of primary importance. The physical exam should be repeated often as some patients will become aware of injuries as their intoxicated state improves.[1][2]

Evaluation

Acute alcohol intoxication causes several metabolic abnormalities, including lactic acidosis, hypoglycemia, hypokalemia, hypomagnesemia, hypocalcemia, and hypophosphatemia. Laboratory analysis should include a full electrolyte panel as well as liver function tests. Alcohol can cause acute effects on the cardiovascular system, such as atrial and ventricular tachydysrhythmias. An EKG should be obtained. One particular syndrome known as “holiday

heart syndrome" can develop, which is characterized by new-onset arrhythmias following acute ingestion of alcohol and can include new-onset atrial fibrillation. Serial EKGs should be done if an arrhythmia is found, as the majority will resolve with the elimination of alcohol. If the EKG changes persist, an alternate cause should be considered. In the case of altered mental status, when a full history cannot be elucidated, a CT scan of the brain should be obtained to rule out any intracranial pathology contributing to the patient's mental status. Many intoxicated patients may state suicidal thoughts or make such gestures. A psychiatric evaluation should be performed and may have to be repeated as the patient becomes more lucid.[1][6][7]

Treatment / Management

Treatment for acute ethanol toxicity is mostly supportive. The first priority, as always, is airway protection. The main life-threatening complication of alcohol intoxication is respiratory depression. Although most patients who present for alcohol intoxication receive intravenous fluids, there is no solid evidence to support this. Alcohol does act as a diuretic; thus, most patients who receive intravenous fluids are in an attempt to treat dehydration. As mentioned above, checking a point of care glucose is important, as many patients with alcohol use disorder will have depleted glycogen stores, and treating hypoglycemia is important, especially before replenishing vitamins such as thiamine. Few studies have shown vitamin deficiencies in intoxicated patients; thus, the routine use of IV multivitamins should be considered on a case-to-case basis. In contrast, routine use of thiamine is recommended for patients with alcohol use disorder, especially in the setting of altered mental status. Detecting occult thiamine deficiency and Wernicke encephalopathy is difficult, and this condition has a high mortality. Thus, the cost/benefit analysis falls in favor of administering thiamine. Patients with alcohol use disorder may not benefit from IV fluids, and consideration must be made for alcoholic cardiomyopathy in this patient population before administering fluids. Some patients may become agitated or violent. In these situations, sedative substances may be required, including droperidol or haloperidol, keeping in mind the potential interaction between the drug and alcohol. Depending on the severity of the intoxication and complications such as Wernicke encephalopathy, alcoholic hepatitis, or dysrhythmias, patients may have to be admitted to the hospital for further treatment.[1][2][5]

Differential Diagnosis

The differential diagnosis for alcohol toxicity is very broad and includes anything that can cause an altered mental status. Considerations include trauma, sepsis, CNS infections, seizure, nonalcoholic toxicologic ingestion, hypo- or hyperthermia, hypo- or hyperthyroidism, hypoxia, and metabolic derangements. Many of these can coexist with alcohol toxicity, so it is important to have a low threshold to obtain laboratory workup and CNS imaging. Complicating the diagnosis of alcohol toxicity is the potential for the patient to ingest non-beverage alcohol such as cologne, cough syrup, and isopropyl alcohol. This can be accidental, such as in pediatrics, or intentional, such as in patients with alcohol use disorder who do not have access to alcohol. Ethylene glycol, methanol, and isopropyl toxicity are discussed in separate articles, and please see these articles for further details. Once alcohol use has been confirmed, further diagnoses have to be considered, such as Wernicke encephalopathy and hepatic encephalopathy.[1][2]

Prognosis

The prognosis for ethanol toxicity depends on multiple factors, including chronicity of use, degree of intoxication, associated traumatic injuries, and end-organ damage. Patients who have uncomplicated ethanol toxicity have a good prognosis and need to be counseled on abstinence. Most of the chronic complications that can develop from ethanol toxicity can be helped and sometimes reversed with alcohol abstinence.[3][4][5]

Complications

Alcohol affects multiple organ systems and can cause complications with both acute and chronic use. Patients under the influence of alcohol are more likely to be involved in trauma-related injuries. Trauma patients under the influence of alcohol have a longer length of hospital stay, higher mortality, and are more likely to have traumatic injuries in the future. Alcoholic liver disease is one of the primary causes of chronic liver disease. Acute alcohol intoxication can cause alcoholic hepatitis and acute on chronic liver failure. This is usually in patients who are chronic alcohol abusers or patients already affected by alcoholic cirrhosis. Active excessive alcohol consumption is the second most frequent precipitating event for acute on chronic liver failure, with bacterial infection being the first. The most effective therapy for alcoholic liver disease is prolonged abstinence from alcohol. Alcohol can cause both acute and chronic effects on the cardiovascular system. Acutely, it can precipitate dysrhythmias such as atrial fibrillation, supraventricular tachycardia, and ventricular tachycardia and can lead to lethal arrhythmias in patients with myocardial infarction. Also, it can cause contractile dysfunction leading to heart failure, stroke, and increased risk of cardiac death.

Heavy drinkers have a much higher risk of heart failure when compared to non-drinkers. Wernicke syndrome, also known as Wernicke encephalopathy, is due to thiamine deficiency and is characterized by the triad of ataxia, oculomotor abnormalities, and global confusion. It develops over days to weeks. While it is most commonly seen in conjunction with patients with alcohol use disorder, it can occur in any disorder leading to a thiamine deficiency. Neurobehavioral findings with Wernicke syndrome include decreased attention, impaired memory, and disorientation. In its severe form, it can lead to coma. Untreated, Wernicke encephalopathy can progress to Korsakoff syndrome, characterized by anterograde and retrograde amnesia without impaired alertness and attention or extraocular movement findings. Chronic alcohol use can lead to dementia, cerebellar degeneration, and peripheral neuropathy.[1][3][4][5][7][8][9][10]

Deterrence and Patient Education

All patients who present to the emergency department for acute alcohol intoxication should be screened for alcohol use disorder. If a patient is found to have alcohol use or dependence, they should be referred for alcohol treatment. In patients who consume alcohol at harmful levels, it is important to intervene early. Presentation to the emergency department for drunkenness should be considered an indicator of pathological use.[1][11]

Enhancing Healthcare Team Outcomes

Alcohol intoxication and use is and will likely remain prevalent in our culture. It is important to fully assess the intoxicated patient for acute organ dysfunction or injuries from their intoxication and educate them on the importance of moderation to prevent or slow the development of chronic diseases. In cases of suspected or known alcohol toxicity, the entire interprofessional healthcare team must work to achieve improved patient outcomes. This team includes clinicians, mid-level practitioners, nurses, pharmacists, and mental health professionals, who can play a crucial role in recovery following alcohol toxicity in those patients with alcohol use disorder. The interprofessional approach with shared information increases the chances of successful recovery. [Level 5]

Review Questions

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