Alcoholic Metabolic Emergencies

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KEYWORDS

• Ethanol intoxication • Beer potomania • Alcoholic ketoacidosis
• Alcohol encephalopathy • Wernicke encephalopathy • Korsakoff syndrome

KEY POINTS

- Hypoglycemia in the alcohol intoxicated adult is no more common than the rest of the population; children often have hypoglycemia with alcohol overdose.
- Beer potomania can be treated with simple fluid restriction; isotonic resuscitation should be approached with caution.
- Alcoholic ketoacidosis may not present with ketonemia.
- Alcoholic encephalopathy syndromes, such as Wernicke encephalopathy and Korsakoff syndrome, should be treated with 500 mg intravenous thiamine every 8 hours.

INTRODUCTION

As all emergency providers are aware, habitual users of alcohol frequently find themselves seeking treatment in the Emergency Department (ED). Patients who abuse alcohol, a term that is synonymous with ethanol for the purpose of this article, can present with a myriad of complaints and may exhibit a range of clinical illnesses. Acute alcohol intoxication and withdrawal make up the largest subsets of illnesses stemming from alcohol abuse, and physicians may get lulled into a familiar management pattern for patients with acute alcohol intoxication. However, because of the high risk of significant morbidity if not recognized and treated appropriately, emergency physicians must pay careful attention to the metabolic derangements that plague alcohol abusers. This article discusses how to diagnose efficiently and provide appropriate medical therapy for some key underrecognized alcohol emergencies.

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ALCOHOL INTOXICATION

Acute alcohol intoxication, herein referred to as "intoxication," is defined as the pathologic state produced by the ingestion of alcohol. Blood alcohol levels (BAL) are sometimes used to supplement clinical decision-making in the ED, and a working understanding of the pharmacokinetics of ethanol is helpful in these circumstances. The degree and duration of symptoms of intoxication are governed by the body's absorption, metabolism, and elimination of alcohol over time. Ethanol is absorbed through the gastrointestinal tract and achieves its concentration in the circulation within minutes to a few hours. A standard drink is classified as 14 g of alcohol. Usually this equals about 1.5 ounces of liquor, 5 ounces of wine, or 12 ounces of beer. One serving will increase the BAL by approximately 25 mg/dL. The character of symptoms associated with intoxication varies with every drinker, so reliably predicting blood alcohol level on clinical features alone is difficult.

Ethanol is metabolized via hepatic oxidation by zero-order kinetics, which means that a set amount is metabolized per unit time. Prolonged ethanol exposure induces hepatic enzymatic activity, resulting in increased alcohol degradation in chronic drinkers.[1] Drinkers who do not chronically abuse ethanol eliminate it at a rate of 15 mg/dL/h, whereas chronic abusers eliminate it around 25 mg/dL/h.[1] Observational data from ED patients have found clearance rates of 18 to 20 mg/dL/h with only minor variability among habitual drinkers.[2,3] Although these typically quoted studies provide reasonable estimates of ethanol metabolism, their wide confidence intervals suggest substantial clinical variability among patients. Therefore, acute alcohol intoxication can be diagnosed clinically or by BAL.

To reduce the amount of unnecessary time intoxicated patients spend in the ED for supportive measures, studies have attempted to identify the utility of various therapies to enhance ethanol elimination. Unfortunately, these studies, which have included naloxone, flumazenil, and intravenous fluids, have failed to demonstrate improvement in ethanol clearance with anything other than time.[4–6] Despite a lack of increasing ethanol elimination, intoxicated patients should receive good supportive care until they are lucid and competent and can be safely discharged.

INTOXICATION AND THE ENDOCRINE SYSTEM

Alcohol can cause clinical abnormalities of endocrine function. Its effects on gonadal function, bone and mineral metabolism derangements, and glucocorticoid secretion rarely result in acute illness requiring ED management.[7] However, acute intoxication may result in alterations in glucose metabolism requiring emergency treatment.

Habitual drinkers who consume alcohol in the absence of other nutrition have a theoretical risk of developing hypoglycemia. Fasting states deplete existing glycogen stores, forcing the body to rely on gluconeogenesis to maintain normoglycemia. The metabolism of alcohol by alcohol dehydrogenase creates a molecular milieu that may prevent the normal conversion of amino acids and pyruvate into glucose, allowing the development of hypoglycemia.[7] However, clinical studies of acutely intoxicated patients do not support this theory. Two different cohort studies, using prospective convenience sampling and retrospective laboratory analysis of intoxicated ED patients, found a low incidence of hypoglycemia (4% and 1%, respectively).[8,9] Although intoxicated adults do not seem to have a higher risk of developing hypoglycemia, children who present following acute alcohol ingestion are at risk for hypoglycemia.[10] Finger-stick glucose evaluation should be used in any intoxicated patient, similar to the evaluation in other ED patients with altered mental status.