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Acute care for alcohol intoxication

Be prepared to consider clinical dilemmas

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CME learning objectives

- To define conditions that may mimic the mental status changes of alcohol intoxication
- To recognize medications that may cause an acute disulfiram-type reaction
- To identify key physiologic implications of alcohol intoxication

The author discloses no financial interest in this article.

This is the first of two articles on alcoholism.

This page is best viewed with a browser that supports tables.

Preview: The clinical assessment of an acutely intoxicated patient should be performed with meticulous care and include repetitive examinations to properly determine the patient's condition. Multiple factors, such as trauma and concomitant use of other drugs, can confuse the diagnostic picture and affect the choice of therapy. In this article, Dr

Yost reviews the diagnostic considerations, appropriate treatment, and clinic discharge for the intoxicated patient. *Yost DA. Acute care for alcohol intoxication. Postgrad Med 2002;112(6):14-26*

With surprising frequency, intoxicated persons present diagnostic and therapeutic challenges in a wide variety of clinical settings. The Centers for Disease Control and Prevention's National Center for Health Statistics estimates that of people in the United States aged 12 years and older, 52% drank alcohol in the past month (1). Of these, 16% are binge drinkers, consuming five or more drinks on the same occasion at least once in the past month. Although data from the National Hospital Discharge Survey showed a decrease in the rate of alcohol-related diagnoses from 1994 to 1997, nondependent abuse of alcohol increased in both sexes during the same period (2).

Physicians working in an emergency department, hospital, or office setting are likely to encounter acutely intoxicated patients. Because an estimated 20% to 25% of emergency department patients have been drinking, physicians should be prepared to consider the clinical dilemmas of intoxication (3,4). The temptation to minimize issues in pleasant drunken patients or rapidly discharge unruly ones must be avoided.

Recognizing, defining intoxication

Recognition of intoxication is a physician's initial challenge on seeing a patient. The extent of the patient's intoxication is a function of body weight and tolerance of alcohol, the volume of alcohol consumed, the percentage of alcohol in the drinks consumed, and the period over which the alcohol was ingested. Although estimation of the intoxication level may be possible from findings on history taking and physical examination (5) (table 1), a conclusive determination usually should rest with measurement of alcohol in breath or blood. Various conditions can cause symptoms that mimic the mental status changes of alcohol consumption (table 2), and thus diagnosis of intoxication should never be made on the basis of behavior alone.

Table 1. Alcohol consumption, approximate BAC, and clinical manifestations in nontolerant persons

Alcohol consumption (drinks)*		Approximate BAC, mg/dL (mmol/L)**	Probable clinical manifestations
55-kg person	90-kg person		
1-3	2-5	50-100 (10.9-21.7)	Impaired sensation, incoordination

3-5	5-8	100-150 (21.7-32.6)	Behavioral changes, ataxia, cognitive and memory difficulties
5-7	8-11	150-200 (32.6-43.4)	Marked incoordination, worsening ataxia, cognitive impairment
7-9	11-14	200-300 (43.4-65.1)	Nausea, vomiting, diplopia, lethargy, aspiration risks (impairment of protective reflexes)
≥10	≥15	300-400 (65.1-86.8)	Decreased respiratory drive, hypoventilation, amnesia, hypothermia, cardiac arrhythmias
Extreme		>400 (>86.8)	Coma, respiratory arrest, death

BAC, blood alcohol concentration.

*One drink equals 1 oz (one shot) of 80-proof liquor, 12 oz of beer, or 4-5 oz of wine.

**1 oz of 80-proof liquor in a 70-kg person raises BAC by 25 mg/dL (5.4 mmol/L).

Adapted from Marco and Kelen (5).

Table 2. Factors in differential diagnosis of altered mental status in an acutely intoxicated patient

Toxic
Ethanol intoxication
Intoxication with other alcohols (eg, methanol, isopropyl alcohol)
Other psychoactive drugs (eg, tetrahydrocannabinol, cocaine, opiates)
Disulfiram (Antabuse) reactions, disulfiramlike reactions

Metabolic
Hepatic encephalopathy
Hypoglycemia (alcohol-induced)
Electrolyte abnormalities (hypernatremia, hyponatremia)
Hypoxia secondary to aspiration or depression of respiratory drive

Infectious disease
Sepsis
Meningitis
Encephalitis

Neurologic
Alcohol withdrawal or alcohol hallucinosis
Postictal states
Wernicke-Korsakoff syndrome
Cerebrovascular accident

Miscellaneous
Hypotension (due to dehydration, vomiting, hemorrhage)
Hypothermia

Trauma
Closed head injuries (eg, intracranial bleeding, concussion syndromes)

Blood alcohol concentration (BAC) remains the definitive standard for assessing intoxication and is expressed in milligrams per deciliter (mg/dL). The legal intoxication level varies by state, but in general it is 80 to 100 mg/dL (17.4 to 21.7 mmol/L). Serial BAC determinations may be useful in assessing whether a patient's BAC has peaked. The average clearance rate in both adults and children is 18 to 20 mg/dL (3.9 to 4.3 mmol/L) per hour (6,7). Consideration should also be given to the fact that elderly drinkers have a higher BAC than younger abusers of the same weight for any given amount of alcohol consumed (8).

Physiologic factors

Alcohol has the potential to adversely affect almost any organ system. However, cardiovascular, gastrointestinal, and neurologic problems are of particular concern to a patient's health. Alcohol is absorbed primarily through the small intestine; only minimal absorption occurs in the stomach. High concentrations of alcohol can irritate the stomach mucosa, and conditions that slow stomach emptying can exacerbate this irritation. The ingestion of large volumes of alcohol in high concentrations (>40%, or 80 proof) or use of alcohol concomitantly with the consumption of fatty foods or use of drugs that impede gastrointestinal motility can result in acute nausea, vomiting, or other signs of gastric inflammation.

Alcohol also may affect morbidity and mortality through the genesis of cardiac arrhythmias. Acute alcohol consumption has been linked with induction of tachyarrhythmias, particularly idiopathic atrial fibrillation. Ventricular tachyarrhythmias also may be provoked, and heavy drinking may increase the risk of sudden cardiac death from fatal arrhythmias (9).

Diagnostic implications

Laboratory testing should be approached with caution in an acutely intoxicated patient. Ethanol inhibits gluconeogenesis, which can result in hypoglycemia. Although elevated lipid levels, ethylene glycol, and solutes such as other alcohols may contribute to an increased osmolar gap, ethanol is the most common cause of hyperosmolality. When BAC testing is not available, an estimation of BAC can be obtained by calculating an increase of 22 mOsm/L in serum osmolality for every 100 mg/dL (21.7 mmol/L) rise in serum alcohol level (5).

Thrombocytopenia has been associated with intoxication, although this condition may normalize after alcohol withdrawal (8). Habitual drinkers may show evidence of leukopenia and an associated susceptibility to bacterial infections. However, the physiologic stresses of vomiting, pain, and other comorbidities of sudden severe intoxication may produce a transient leukocytosis.

Treatment of intoxication

Determination of proper treatment for a patient with acute intoxication requires that a physician assess the patient's mental status changes, evaluate drug interactions, and appraise and manage the patient's physical status and vital signs.

Mental status changes

Alcohol consumption can induce or mimic any mental status level and psychiatric symptom. Although alcohol is a primary depressant of the central nervous system (CNS), alcohol-induced psychopathologic conditions can range from lethargic depression to violent delirium. Because as many as 90% of alcoholic persons younger than 30 years use another drug in addition to alcohol, the confounding psychoactive effects of other drugs of abuse always must be considered (10).

For patients with a history of previous intoxication episodes, mental status changes tend to be similar with each drinking binge. Additionally, detoxification and symptom resolution typically follow a predictable pattern in repetitive users. Mental status changes noticed to be markedly uncharacteristic of a patient's previous intoxication pattern are often a warning sign that more aggressive assessment is needed for head injuries, electrolyte abnormalities, adverse reactions to illicit or prescription drugs, or other causes of mental deterioration.

Drug interactions

Alcohol may have a modest effect on the absorption of some drugs. However, the potential for disulfiram-type drug interactions poses the most significant pharmacologic concern. Disulfiram (Antabuse) and alcohol interact to produce increased levels of acetaldehyde, the accumulation of which leads to flushing, nausea, vomiting, vertigo, headache, abdominal pain, and diaphoresis. Although

disulfiram is prescribed to promote abstinence from alcohol, other drugs interact with alcohol in the same fashion. Metronidazole (Flagyl), cephalosporins, chlorpropamide (Diabinese), sulfonamides, and chloral hydrate (Aquachloral Supprettes) can each produce disulfiramlike reactions. Management of these syndromes is supportive therapy, including adequate hydration and antiemetics (5).

Many drugs compete metabolically with alcohol for the microsomal oxidizing pathways of the liver. Metabolism of antihistamines, barbiturates, benzodiazepines, narcotics, and phenothiazines can be slowed significantly in acutely intoxicated patients. Alternatively, alcohol clearance can be decreased in the presence of verapamil hydrochloride or high-dose cimetidine (Tagamet). The increased activity of the microsomal oxidizing systems that results from repetitive heavy alcohol use can result in a reduction of up to 50% in the half-life of drugs such as warfarin sodium (Coumadin), phenytoin (Dilantin), propranolol hydrochloride (Inderal), isoniazid (Nydrazid), and phenobarbital (Bellatal, Solfoton) (5,11). Thus, assessment of the hepatic function of an acutely intoxicated patient should be a priority before drugs are administered.

Physical assessment and management

Immediate attention must be given to the vital signs of an intoxicated patient, particularly signs that indicate hydration status. The natural diuretic effects of ethanol may commonly lead to dehydration in intoxicated patients. Tachycardia or hypotension, or both, may indicate dehydration but also may be the only clues to unappreciated pain, early alcohol withdrawal, internal bleeding, or hypoglycemia. Airway assessment and protection also are crucial because of the suppressed protective reflexes that can result from intoxication and the increased potential for vomiting secondary to gastric irritation.

In contrast to opiate and benzodiazepine toxic syndromes, there is no specific antidote or reversal agent for alcohol toxicity. Therapeutic intervention priorities include hydration with intravenous fluids, symptomatic control of nausea and vomiting, and correction of electrolyte imbalances, such as hypomagnesemia. Glucose administration may be critical for patients with confounding hypoglycemia, while thiamine supplementation should be considered for chronic drinkers who exhibit mental status changes consistent with Wernicke-Korsakoff syndrome. A premixed intravenous solution of 1 liter of 5% dextrose and 0.45% sodium chloride, 2 g of magnesium sulfate, 1 mg of folate, and 100 mg of thiamine has been used routinely by many institutions (5).

Specific attention should be given to the neurologic examination of an intoxicated patient. Although moderate drinking ($\leq$2 drinks per day) may protect against ischemic stroke (12), recent heavy alcohol use may increase the risk of all major types of stroke (13). The blood pressure-elevating effects of heavy drinking pose additional risk to intoxicated patients with preexisting hypertension. Because ataxia, slurred speech, and many other physical signs of

acute intoxication can mimic a cerebrovascular event, clear definition of a patient's neurologic deficits is often achieved only through a period of prolonged observation and serial neurologic assessments.

Concerns in special settings

Persons who are intoxicated may present with complicating conditions, such as trauma, pregnancy, and severe alcohol poisoning.

The intoxicated trauma patient

Acute alcohol ingestion is particularly likely to complicate the management of a trauma patient. The masking effects of alcohol may make the impaired patient oblivious to the pain of severe hepatic, splenic, or other intra-abdominal injury. Discomfort from significant orthopedic injury, such as an occult fracture or dislocation, may also be minimized during the initial trauma survey. Trauma-related hypovolemia may be compounded by the hypotensive effects of alcohol intoxication and may lead to circulatory collapse. Additionally, for a trauma patient who survives with significant wounds, alcohol's immunosuppressive effect increases the likelihood of posttraumatic infection (4).

The sensory-altering influence of alcohol often makes the immediate evaluation of head injuries in an intoxicated patient extremely difficult. Research data have indicated that alcohol intoxication has the potential to exacerbate brain injury through a variety of mechanisms. However, repeated studies have failed to consistently demonstrate a measurable adverse relationship between head injury outcome and acute alcohol use (14).

In a trauma setting, physicians and field emergency workers often underestimate the presence and degree of alcohol consumption, which makes rapid BAC determination a critical component of trauma management (4). Comparison of serial quantitative BAC values with the progression of physical and mental status examination findings can be useful in delineating injuries that are not appreciated initially. The temptation to let an intoxicated trauma patient "sleep it off" for an extended period without regular reassessments must be avoided.

Intoxicated obstetric patients

The rapid absorption of alcohol from the gastrointestinal tract into maternal circulation is followed by an equally rapid distribution into fetal circulation. Alcohol readily crosses the placenta, and its depressive effects may manifest in flat or minimally reactive external fetal monitoring tracings. Particular attention should be given to any findings during history taking or physical examination that suggest bleeding. Heavy alcohol use during pregnancy is associated with an increased risk of both spontaneous abortion and abruptio placentae (15).

Each episode of acute intoxication presents an opportunity for risk counseling, and intervention for an alcohol-abusing

pregnant patient may prevent the devastating consequences of fetal alcohol syndrome (FAS). This constellation of growth retardation, CNS dysfunction, and congenital abnormalities is generally believed to occur when consumption approaches six or seven alcoholic drinks per day. (One drink is 12 oz of beer, 4 to 5 oz of wine, or 1 oz of 80-proof liquor.) The resulting incidence of FAS in children of alcoholic women is estimated at 10% (15).

Severe alcohol poisoning

Rapid ingestion of large quantities of alcohol constitutes a medical emergency that can quickly lead to lethal respiratory depression. Initial mucosal absorption begins within 10 minutes, and serum BAC peaks between 30 and 90 minutes after ingestion (5). Effects of severe ingestion can be especially profound in young, nontolerant binge drinkers. Expeditious respiratory support through airway management and mechanical ventilation is essential.

Although gastric lavage in a patient seen within minutes of ingestion may lower the maximum BAC, the rapid absorption of alcohol through intestinal mucosa makes this procedure of limited value in a patient with delayed presentation of intoxication.

Discharge and medicolegal issues

As a rule, hospital admission is not necessary for uncomplicated intoxication, and with careful discharge planning the vast majority of intoxication patients can be managed as outpatients. Nevertheless, the clinical condition of a patient who abuses alcohol may decline significantly and rapidly due to repetitive drinking, worsening metabolic problems, infection, or unappreciated trauma. Many alcohol-abusing patients do not have a reliable source of primary medical care, which makes follow-up care arrangements particularly important.

Agitated patients must be protected from themselves and from similarly intoxicated persons. However, intoxication by itself is usually not sufficient cause to implement physical or chemical restraints. Careful documentation of specific physical and emotional findings should exist before use of restraints is initiated.

Physicians need to be prepared to contact local law enforcement authorities when a belligerent patient with unresolved mental status changes insists on leaving the hospital, emergency department, or office setting and driving away. The concept of extended liability may hold physicians responsible for premature discharge of intoxicated patients who subsequently cause harm to themselves or others (5). With the passage in October 2000 of a federal legal intoxication standard of 80 mg/dL (17.4 mmol/L), legislative reassessments of intoxication limits are anticipated in the 15 states still using a limit of 100 mg/dL (21.7 mmol/L).

Physicians also need to resist frustration and complacency

when deciding to intervene with a patient's alcohol abuse. Referral options for intervention, such as Alcoholics Anonymous, family counseling, detoxification units, and treatment centers, should be available in every setting. Physicians can take encouragement from data showing that in previously untreated patients who presented with intoxication to urgent ambulatory settings, almost 50% of those referred for alcohol intervention kept their treatment appointment (3).

The final decision to discharge an intoxicated patient should be based on repeated clinical assessments rather than BAC alone. Every intoxicated patient should be evaluated for suicide risk. The presence of aggravating factors, such as domestic violence and homelessness, also should be explored. Social service options, including shelter referrals, need to be readily available. For urgent procedures, consent of a capable and sober guardian should be obtained; this person also should receive written discharge instructions, including warning signs of deterioration, comorbid conditions, and alcohol withdrawal.

Conclusion

Evaluation of an acutely intoxicated patient should be approached with meticulous care, repetitive examinations, and a quantitative assessment of intoxication wherever possible. The patient should be carefully screened for the comorbidities of chronic drinking, such as liver disease and infection. Interventions and referrals to promote sobriety should be readily available, and discharge planning should include the assurance of a safe and monitored home setting.

By replacing complacency with clinical suspicion and anticipation, the majority of acutely intoxicated patients can be handled with supportive care at the presenting facility.

References

1. **Centers for Disease Control and Prevention.** National Center for Health Statistics. Fast stats: alcohol use. Available at: http://www.cdc.gov/nchs/fastats/alcohol.htm. Accessed Apr 24, 2002
2. **Whitmore CC, Stinson FS, Dufour M.** Alcohol epidemiologic data surveillance: surveillance report 50. Trends in alcohol-related morbidity among short-stay community hospital discharges, United States, 1979-97. Rockville, MD: National Institute on Alcohol Abuse and Alcoholism, 1999
3. **Green M, Setchell J, Hames P, et al.** Management of alcohol abusing patients in accident and emergency departments. J R Soc Med 1993;86(7):393-5
4. **Waller JA.** Management issues for trauma patients with alcohol. J Trauma 1990;30(12):1548-53
5. **Marco CA, Kelen GD.** Acute intoxication. Emerg Med Clin North Am 1990;8(4):731-48
6. **Simon HK, Cox JM, Sucov A, et al.** Serum ethanol clearance in intoxicated children and adolescents presenting to the ED. Acad Emerg Med 1994;1

(6):520-4
7. **Brennan DF, Betzelos S, Reed R, et al.** Ethanol elimination rates in an ED population. Am J Emerg Med 1995;13(3):276-80
8. **Ballard HS.** Hematological complications of alcoholism. Alcohol Clin Exp Res 1989;13(5):706-20
9. **Kupari M, Koskinen P.** Alcohol, cardiac arrhythmias and sudden death. Novartis Found Symp 1998;216:68-85
10. **Miller NS.** The pharmacology of drug testing: indications and methodology. In: Miller NS. The pharmacology of alcohol and drugs of abuse and addiction. New York: Springer-Verlag, 1991:291-305
11. **Sands BF, Knapp CM, Ciraulo DA.** Interaction of alcohol with therapeutic drugs and drugs of abuse. In: Kranzler HR, ed. The pharmacology of alcohol abuse. New York: Springer-Verlag, 1995:475-505
12. **Sacco RL, Elkind M, Boden-Albala B, et al.** The protective effect of moderate alcohol consumption on ischemic stroke. JAMA 1999;281(1):53-60
13. **Hillbom M, Numminen H.** Alcohol and stroke: pathophysiologic mechanisms. Neuroepidemiology 1998;17(6):281-7
14. **Kelly DF.** Alcohol and head injury: an issue revisited. J Neurotrauma 1995;12(5):883-90
15. **Levy M, Koren G.** Obstetric and neonatal effects of drugs of abuse. Emerg Med Clin North Am 1990;8 (3):633-52

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