Beware the Danger Lurking Under the Shroud of Alcohol

Margaret Sheehy, MD, MSc*; Patil Armenian, MD; Jessica Mason, MD; Andrew Grock, MD

*Corresponding Author. E-mail: mksheehy@mgh.harvard.edu, Twitter: @mksheehy.

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ANNALS CASE

An alcohol-dependent patient well known to your emergency department (ED) presents with…drumroll please…alcohol intoxication. A Good Samaritan called 911 after finding the patient asleep on a park bench. His chart is overflowing with ED visits for alcohol intoxication and usually conclude with an early-morning discharge. His vital signs are pulse rate 98 beats/min, respiratory rate 18 breaths/min, blood pressure 156/76 mm Hg, and oxygen saturation 97% on room air. On examination, he appears intoxicated—alcohol on his breath, opens his eyes and speaks confused words to painful stimuli—and is protecting his airway. There are no signs of acute head trauma or other injury.

So what’s the plan? Does this patient need laboratory tests, head computed tomography (CT), or withdrawal prevention?

INTRODUCTION

You have probably treated a patient like this recently. The statistics sure say you have. Rates of alcohol intoxication ED visits are increasing out of proportion to overall ED visits. Average blood alcohol concentrations (BACs) are increasing too! So patients are getting more intoxicated more frequently. Although many simply require observation until clinically sober, with an eventual turkey sandwich, badness may lurk beneath the shroud of alcohol…and it may be hard to identify on initial evaluation.

Klein et al looked for badness retrospectively in 31,364 cases of low-risk acute alcohol intoxication. One percent of their study cohort, or 325 patients, required critical care resources while in the ED. Included patients were deemed to be at low risk according to triage nurse and emergency physician assessment after a mandatory alcohol level and fingerstick glucose-level test. The rate of badness, therefore, among all comers with presumed alcohol abuse is undoubtedly higher; hence, the common refrain, “Alcoholics were put on this earth to humble emergency physicians.”

What can we learn from this study? Indicators for potential badness were abnormal vital signs, hypoglycemia, or need for chemical sedation. Although these red flags may seem obvious in hindsight, they can be easily dismissed with seemingly benign explanations. Tachycardia and hypotension may be attributed to dehydration, a coingestion, or anxiety, especially when anchored on a diagnosis of alcohol intoxication. This article reminds us to always consider other causes for the patient’s altered mental status. And make sure there is a full set of vital signs recorded. What was our example patient’s temperature? Oh, it wasn’t taken!?

COOPERATIVE PATIENTS INTOXICATED ON ALCOHOL

As usual, start with airway, breathing, and circulation assessment. Although sometimes limited to “whiskey,” taking a history should be attempted and include recent illnesses, trauma, sexual assault, medications, allergies, drug and alcohol use, social support structures, and history of withdrawal or seizures. All patients should also have a full set of vital signs recorded and receive a point-of-care test for glucose level and a physical examination with special attention for signs of trauma.

Although badness may be lurking, we know we must balance resource use with patient safety. As such, routine laboratory tests are not indicated for the typical intoxicated patient. They may help individuals with specific complaints, signs of trauma, or unexplained altered mental status. In patients previously unknown to you with presumed long-term alcohol abuse, consider laboratory tests to evaluate for anemia, thrombocytopenia, and liver function.
The utility of BACs is highly debated and often department dependent. In most patients, BACs are not necessary or helpful because specific levels do not necessarily correlate with symptoms. At any particular alcohol level, one patient may be severely intoxicated while another one is withdrawing.

Reasons for a BAC test are as follows:
1. Suspected toxic alcohol ingestion: Toxic alcohol levels such as those for methanol, ethylene glycol, and isopropyl alcohol are not available in most EDs, and diagnosis may depend on an osmolar gap. Because BAC is part of the serum osmolar gap, it can aid in diagnosing toxic alcohol ingestion.

2. Unexplained altered mental status or failure to improve at an appropriate rate: Alcohol can veil a subdural hematoma, hypoglycemia, toxic alcohol ingestion, intentional overdose, gastrointestinal bleeding, sepsis, hepatic failure, renal failure, and many, many more disease states. If you aren’t quite sure that it’s isolated alcohol intoxication, get a BAC…and start worrying if the level is low or undetectable.

Alcohol intoxication signs and symptoms, confusion, loss of consciousness, vomiting, slurred speech, and ataxia can mimic those of traumatic brain injury and intracranial hemorrhage. Yet providing CT imaging for every patient presenting with alcohol intoxication increases radiation exposure and cost. This can present a diagnostic dilemma, especially given that nearly half of patients presenting with traumatic brain injury have concomitant alcohol intoxication. One option for patients with low clinical gestalt for intracranial injury is to defer the CT for several hours and image only those who fail to improve. One retrospective review of 5,934 patients demonstrated that this delayed CT strategy was safe and that immediate CT had low clinical value.

**AGITATED PATIENTS INTOXICATED ON ALCOHOL**

Agitated patients are dangerous to themselves and ED staff, and are disruptive to the department and providers. If you are considering sedation, remember that it is for patient and staff protection, is meant to enable a complete evaluation, and requires a systematic approach. First, attempt verbal de-escalation and reduction of stimuli by placing the patient in a quiet, dark room. If needed, medications should be thoughtfully chosen after consideration of presentation, medical history, and current medications. Chemical sedation carries significant risks in intoxicated patients, including respiratory depression (benzodiazepines), pulmonary aspiration (all), dystonic reactions (haloperidol and olanzapine), laryngospasm (ketamine), prolonged QTc interval (haloperidol and ziprasidone), neuroleptic malignant syndrome (haloperidol), delirium, lowered seizure thresholds, and hypotension. Any agitated intoxicated patient requiring sedation should receive a cardiac monitor with continuous pulse oximetry.

Although to our knowledge no ED studies have specifically examined sedation of agitated alcohol-intoxicated patients, presumably a subset of agitated ED patients in general sedation studies had alcohol on board. Common sedation agents include parenteral benzodiazepines such as midazolam or lorazepam, typical and atypical antipsychotics such as haloperidol or olanzapine, and a combination of a benzodiazepine, haloperidol, and diphenhydramine. More recently, ketamine has gained favor for sedating agitated ED patients. In one prospective ED study, ketamine was found to control agitation faster than the traditional ED medications (benzodiazepines, antipsychotics, and combinations of the two).

**THE SLIPPERY SLOPE TO WITHDRAWAL**

Alcohol withdrawal symptoms of tachycardia, hypertension, tremors, diaphoresis, delirium, and seizures peak around 72 hours after alcohol discontinuation; however, symptoms may begin as early as 6 hours after the last drink. To avoid seizures, delirium tremens, and need for mechanical ventilation, withdrawal should be diagnosed and treated early and aggressively.

Most emergency physicians reach first for and are comfortable giving benzodiazepines, with escalating doses as needed. For severe withdrawal, patients may require more frequent and higher doses and must be closely monitored. Treat to symptom control rather than initially relying on a protocol, such as Clinical Incident Withdrawal Assessment—Alcohol.

When compared with benzodiazepines, phenobarbital has a longer half-life, reliable pharmacokinetics, and lack of cross-tolerance in alcohol-dependent patients. Thus, it may be equally efficacious in withdrawal. Because phenobarbital seems to be less commonly used, let’s review a few dosing regimens. For outpatients, use 260 mg intramuscularly or an intravenous push followed by 130 mg intramuscularly or intravenously every 30 minutes as needed. For inpatients, use 10 mg/kg ideal body weight during 30 minutes. After an additional 30 minutes, 130 to 260 mg intravenously can be redosed every 30 minutes as needed. There is no maximal dose and the half-life is 3 days. Once symptom control is reached, there is little more to do and the patient often remains comfortable, with almost no sign of agitation or withdrawal. Beware of...
phenobarbital and benzodiazepines’ synergistic effect! Combining them results in a high likelihood of oversedation and respiratory depression.

**CASE RESOLUTION**

Our patient’s glucose level is 83 mg/dl and his temperature is 36.9°C (98.4°F). You decided to frequently reassess and defer initial laboratory tests and imaging. Fortunately, his mental status steadily improves. After a few hours, he eats, drinks, speaks clearly, ambulates with a steady gait, and calls a friend for a ride home. Knowing that sobriety is not based on the BAC, but on clinical assessment, you complete and document a psychomotor and cognitive evaluation. Your helpful nurse agrees that the patient is clinically sober.5

Another alcohol intoxication patient successfully treated, but that critical 1% is still out there waiting for you. Keep your eyes open because treating alcohol intoxication can be a sobering experience.

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**REFERENCES**