Measuring Blood Ethanol: Can It Lead You Astray? (Maybe)

In this case of a patient with presumed ethanol intoxication, the ED team's decision to measure his blood ethanol concentration is lifesaving. However, valid arguments exist both for and against routinely assessing blood ethanol levels in patients with presumed intoxication. Herein, these concerns are discussed, along with consideration of the value of routine toxicology screening in all patients with altered mental status.

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Case

A 25-year-old man is found unresponsive on the sidewalk and is brought to the ED. There are no suggestions at the scene of trauma or drug use. His initial vital signs include a blood pressure of 131/79 mm Hg; heart rate, 109 beats/min; respiratory rate, 18 breaths/min; and temperature, 37.6°C. His Spo_2 level is 97% on 2 L oxygen via nasal cannula. On physical examination, the patient moves all extremities to noxious stimuli, has pink emesis around his mouth, and has no signs of trauma. There are several intoxicated patients in the ED at the time, due to local festivities. Accordingly, his altered mental status is attributed to ethanol intoxication, and the plan is to observe him until sobriety is established.

Should a blood ethanol concentration be obtained to confirm the diagnosis?

The indications for obtaining a blood ethanol concentration in patients thought to be intoxicated in the ED are frequently debated in the medical literature and in the ED. One perspective (of many) reflects the tenet of laboratory testing that a test should be ordered only if its results would potentially alter clinical management.[1] As a result, many emergency physicians do not routinely perform blood ethanol analysis on a patient who has the odor of alcohol (or its congeners) on the breath, is arousable, and has a high likelihood of uncomplicated ethanol intoxication.[2] In such cases, the patient's degree of intoxication can be surmised clinically on the basis of history and physical examination, and the chief complaint often can be evaluated fully without knowledge of the blood ethanol concentration. Some are concerned that having this information compels the emergency physician to delay discharge until the concentration is in (or believed to be in) the "legal" range (below 80 mg/dL in all US states). This is associated with ED crowding and disruption and the development of ethanol withdrawal in a consequential number of patients.

Alternatively, the question of whether to assess blood ethanol concentration in a patient with deep obtundation or an unclear history is more complicated. The literature suggests that even experienced clinicians are often inaccurate in their clinical diagnosis of ethanol intoxication.[3,4] In one prospective study at an urban teaching hospital, 10% of patients noted in triage to have the "odor of ethanol" on their breath actually had un-

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detectable blood ethanol concentrations.[4] Another study found that trauma patients were "more likely to be falsely suspected of [ethanol] intoxication if they were either young, male, [or] perceived as disheveled, uninsured, or having a low income."[3]

Case Continuation

When the patient's mental status fails to improve over several hours, head CT and laboratory studies are obtained. His head CT shows no abnormalities. His blood ethanol concentration is undetectable. Venous blood gas analysis indicates a pH of 7.21; Pco_2, 31 mm Hg; and Po_2, 57 mm Hg. Other laboratory values include a lactic acid level of 7 mmol/L; acetaminophen, 447 µg/mL (therapeutic range, 10 to 15 µg/mL); aspartate aminotransferase (AST), 166 IU/L; alanine aminotransferase (ALT), 184 IU/L; and international normalized ratio (INR), 1.48.

What is the value of assessing the blood ethanol concentration in this patient?

It is often quipped that blood ethanol concentrations are clinically useful only when they are zero. In other words, knowing a patient's blood ethanol concentration, but not knowing the patient's chronic alcohol use history and degree of tolerance, does not allow the clinician to predict the degree of resulting clinical intoxication. For example, a patient with relative ethanol naiveté might present with coma resulting from an ethanol concentration of 220 mg/dL, while someone with that same concentration who chronically abuses ethanol might exhibit signs of ethanol withdrawal. Thus, a number alone is not an accurate gauge of clinical intoxication in a long-term, heavy alcohol user. In addition, the classic cognitive error of diagnostic anchoring is possible. This occurs when the clinician makes a presumptive diagnosis of ethanol intoxication based on a patient's altered mental status and, given that the blood ethanol concentration is elevated, the clinician fails to consider the broader differential diagnosis for altered mental status, including intracerebral hemorrhage, CNS infection, and any number of other potentially deadly entities that may coexist in this patient population.

FAST TRACK

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The counterpoint to the argument against the utility of obtaining a blood ethanol concentration is that this measurement, when properly interpreted, can provide clinically important information. In a nontolerant adult, inhibition of fine motor skills occurs at a concentration of about 50 mg/dL, and stupor, at around 250 mg/dL. A nontolerant individual metabolizes ethanol at a rate of approximately 15 to 20 mg/dL/h.[5] With these general parameters in mind, clinicians can use blood ethanol concentrations to guide decisions regarding further diagnostic testing if the patient's alteration in mental status or functional ability far exceeds what would be expected with the blood ethanol concentration in question, or if the patient fails to improve over an appropriate observation period.

In summary, the use of blood ethanol concentrations remains complicated and must be considered in each clinical context. This patient might have benefited if his blood ethanol concentration had been measured earlier, since the fact that it was undetectable would have prompted further evaluation. Alternatively, if this patient had had a notable blood ethanol concentration in addition to his acetaminophen toxicity, the team might have been falsely reassured and missed the acetaminophen toxicity. Thus, whether ethanol intoxication is presumed on clinical grounds to be the cause of altered mental status or whether it is "confirmed" by blood ethanol concentration measures, patients must be frequently reevaluated to ensure that they are improving over time.

Case Continuation

The standard 21-hour *N*-acetylcysteine (NAC) infusion is started and a medical toxicology consult is obtained. Features of an antimuscarinic toxidrome are noted and treatment with physostigmine 2 mg IV improves the patient's mental status. He receives the NAC infusion until his INR normalizes and transaminase levels approach normal on hospital day 7.

Should urine and serum toxicology screens be performed in all patients with altered mental status?

Ironically, medical toxicologists tend to discourage routine use of urine toxicology panels for drugs of abuse because the results are often markers of recent drug use rather than acute intoxication. For example, the immunoassay for cocaine tests for its inactive metabolite, benzoylecgonine. In a patient presenting with agitated delirium, a positive urine cocaine immunoassay points only to the fact that the patient used the drug in the previous several days; the delirium could be due to any number of pertinent medical illnesses.[6] Additionally, the tests often exhibit poor sensitivity and specificity and can therefore be misleading. For example, most urine immunoassays for benzodiazepines test for oxazepam, a common metabolite of several benzodiazepines, rather than for the parent benzodiazepine. As a result, a patient symptomatic from the commonly abused benzodiazepine clonazepam might have a clinically false-negative benzodiazepine screen because clonazepam does not generate this metabolite.[7] The phencyclidine (PCP) urine immunoassay, on the other hand, can have false-positive results in persons taking structurally similar drugs such as dextromethorphan, which is found in many nonprescription cough and cold preparations.

While history and physical examination are preferred over drug screens to diagnose most intoxications, screening for acetaminophen overdose is recommended in patients in whom suicidal overdose is suspected. This is because acetaminophen toxicity remains clinically silent in its early stage, so that even patients with a large overdose, who ultimately develop liver failure, may have no symptoms or nonspecific symptoms during the first 24 hours. Since the effective and safe antidote for acetaminophen overdose, NAC, is highly effective at preventing hepatic toxicity if administered within 8 hours of an acute overdose,[8] rapidly identifying the exposure is critical. Furthermore, approximately one in 500 patients presenting with an intentional drug overdose who do not report ingesting acetaminophen are found to have a potentially hepatotoxic serum acetaminophen concentration.[9] For these reasons, it is recommended that a serum acetaminophen concentration be obtained in every patient with intentional overdose. In the case patient, this screening was performed when the team became concerned about an intentional drug overdose because the blood ethanol concentration was undetectable. There had been no a priori suggestion of suicidality based on the history or circumstances of the presentation.

FAST TRACK

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Case Conclusion

The patient ultimately reports having ingested three "bottles" of acetaminophen and one "box" of an over-the-counter sleep medication in a suicide attempt. His AST level peaks at 10,726 IU/L, and his ALT level reaches 14,780 IU/L on hospital day 2. The INR peaks at greater than 10 on hospital day 3. His mental status and renal function remain normal throughout, and the patient has full hepatic recovery by 2 1/2 weeks after presentation. **EM**

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