## Title of Case

**Delirium Tremens: A Serious Complication of Alcohol Withdrawal**

## Authors of Case

*Please indicate corresponding author by *

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## Summary

**Up to 150 words summarising the case presentation and outcome**

Patient is a 51 year-old Caucasian male recently hospitalized for an acute episode of alcohol withdrawal and alcoholic ketosis with an elevated anion gap, delirium tremens and melena. He also was noted to have blood loss anemia. The hemoglobin dropped to as low as 8.5g/dL. He received a total of 2 units of packed red cells. He was started on proton pump inhibitors and Carafate. He underwent esophagogastroduodenoscopy and the results showed long-segment Barrett esophagus, beginning at approximately 22 cm from the incisors, irregular ulceration in distal esophagus in the setting of Barrett’s, concerning for adenocarcinoma. Mild diffuse gastropathy and proximal duodenopathy. He was placed on alcohol withdrawal precautions and started on Librium. Patient's hemoglobin stabilized at around 10.3 to 11.5. Four days later he was transferred to a transitional care facility in stable condition to further monitor his lab values and withdrawal symptoms.

## Background

*Why you think this case is important – why you decided to write it up*

Delirium tremens is recognized as a potentially fatal and debilitating complication of ethanol withdrawal. Identifying at risk patients who are admitted to the hospital can prevent them from progressing to DTs by placing them on alcohol withdrawal precautions. This is an area that cannot be overlooked when admitting an alcoholic patient.

## Case Presentation

**Presenting features, medical/social/family history**

**HPI:**

- RD is a 51 y/o Caucasian male with past medical history significant for anxiety, hypertension and alcoholism. He presented to the ED with a chief complaint of hallucinations and passage of dark stool.
- The Pt has been drinking heavily lately. He drinks 1 pint of vodka and a 6 pack of beer every day. He stopped drinking 3 days prior and has been going through a period of tremulousness and agitation. The morning of his arrival he had experienced a “vivid dream” while he was awake. He said he saw a vehicle and a man running behind his barn. At some point, he realized that he was awake and this was not actually occurring. The Pt also described that over the past few weeks, he has been experiencing frequent falls both while intoxicated and while sober.

**PMH/PSH:**

- Alcoholism
- Anxiety Disorder
- Hypertension

**Meds:**

- Tenormin 25mg PO QD
- Gabapentin 200mg PO TID
- Hydrochlorothiazide DOSE???
- Lisinopril
- Folic Acid
- Thiamine

**Allergies:** Poison Ivy

**Family Hx:**

- Noncontributory
Social Hx:
- Smokes cigars occasionally
- Drinks 1 pint of vodka and 6 pack of beer daily

ROS:
- Gen:
  - Has been experiencing frequent falls over the past few weeks: both while intoxicated and while sober.
  - (-) Sensory disturbances in the extremities
  - (-) Fatigue/weakness.
- Cardiac:
  - (-) CP/palpitations
- Resp:
  - (-) SOB/cough/hemoptysis
- GI:
  - Has been experiencing dark diarrhea over the past few weeks
  - Denies any bleeding per rectum
  - Denies hematemesis
- Neurological
  - Denies seizures
- GU
  - Denies urinary incontinence.
- Otherwise, unremarkable.

PE:
- Vitals on admission: T: 99.2 F   P: 105   R: 20    BP: 123/60    O2: 99% room air
- Gen: Pt appears anxious but is A&Ox3
- HEENT: PERRLA, oropharynx is unremarkable.
- Neck: No JVD, no carotid bruit, no palpable goiter.
- Heart: RRR, S1 and S2 are heard, no MRG
- Lungs: CTA B/L
- Abd: Soft, non-tender. No organomegaly, masses or acites. + BSx4
- Extremities: No edema or calf tenderness

INVESTIGATIONS If relevant

Laboratory Studies:
- Alcohol <20
- Cocaine level is negative
- UA unremarkable
- TSH: 1.52
- WBC: 11, Hgb: 10.4, HCT: 29%, PLT: 99
- Na: 129, K: 3.3, Cl: 82, CO2: 29, BUN: 47, Cr: 1.4
- Total bilirubin 2.1
- AST: 48
- ALP: 81
- Anion gap: 18
- INR: 1.2

Radiological Studies:
- CT of the head: unremarkable
- CXR: normal

Consults
- GI

DIFFERENTIAL DIAGNOSIS If relevant
N/A, the negative CT and a history of strong ETOH abuse with abrupt withdrawal clearly points to delirium tremens. Possible upper GI bleed would be investigated during his admission because of his recent passage of dark stool.

TREATMENT If relevant
- Pt was admitted to the medical telemetry floor.
- Hgb and Hct were monitored q6 hours
- Pt was placed on alcohol withdrawal precautions
OUTCOME AND FOLLOW-UP

- EGD displayed long-segment Barrett esophagus, beginning at approximately 22 cm from the incisors, irregular ulceration in distal esophagus in the setting of Barrett's, concerning for adenocarcinoma. Mild diffuse gastropathy and proximal duodenopathy.
- The patient spent four days in the critical care unit, where he was monitored and treated for his alcohol withdrawal symptoms. He was transferred in stable condition to a transitional care unit to further monitor his progress.

DISCUSSION including very brief review of similar published cases (how many similar cases have been published?)

1. What screening tools should be used when evaluating a patient in the outpatient setting, to stratify their risk for AWS?
2. What are the current guidelines for diagnosis & management of alcohol withdrawal syndrome?
3. What are the comorbidities associated with the alcoholic patient?
4. What is Delirium Tremens?

1. Clinicians should constantly be practicing preventative medicine in the outpatient setting. Being proactive rather than reactive is key to reducing hospital admissions and increasing the overall well being of all patients. The key aspect of every patient encounter is the history. The clinician will make the majority of their decisions based on what is discovered from the history. When evaluating alcohol history it is important to not only ask about personal alcohol consumption but also family history of alcohol abuse. Alcoholism is a heterogeneous disorder with an estimated heritability of 40%-60% (8). The alcoholic history includes: estimating consumption (types of alcoholic drinks consumed, volume, frequency and drinking pattern); establishing if the patient is alcohol dependent (daily drinking, drinking early in the day, rating the priority of alcohol in a patient's life, previous medical interventions required in relation to drinking); and establishing whether problems have arisen in relation to drinking (social, domestic, emotional, occupational, financial and legal) (6). Many times the patient will either not reveal the extent of their personal alcoholic history or diminish their alcohol dependence. In these instances, biochemical markers of heavy alcohol consumption are of some help in clarifying the diagnosis. Gamma glutamyl transferase (GGT) and carbohydrate-deficient transferrin are both sensitive markers for alcohol overuse, particularly when tested as a combination. Other lab values which may be of some use would be macrocytosis indicating folate and/or vitamin B12 deficiency as a result of poor nutrition. Elevated alanine aminotransaminase and aspartate aminotransferase are seen in alcohol-dependent individuals who have acute or chronic hepatitis secondary to excessive alcohol consumption. Homocysteine levels are elevated in non-abstinent alcoholics and levels are associated with alcohol withdrawal seizures (6).

2. According to the DSM-IV, to diagnose alcohol withdrawal syndrome, the patient must have two or more of the following after cessation or reduction of alcohol use that has been heavy or prolonged: autonomic hyperactivity (sweating, tachycardia); increased hand tremor; insomnia; nausea or vomiting; transient visual, tactile, or auditory hallucinations or illusions; psychomotor agitation; anxiety and tonic-clonic seizures (6). During an acute episode of alcohol withdrawal syndrome, there are other laboratory tests which need to be drawn. This is especially important in the Emergency Department, where other presenting symptoms of withdrawal exist. Laboratory tests which are generally ordered for patients who are experiencing an AWS seizure include: barbituate screen, creatinine level, CPK, electrolytes, BUN, glucose, ethanol, hemoglobin and hematocrit, magnesium, serum osmolality, methanol and ethylene glycol, toxicology screen, UA and white blood cell count (5). Once the diagnosis has been established, the severity of baseline withdrawal symptoms should be assessed in order to guide the need for treatment. The Clinical Institute Withdrawal Assessment Scale for Alcohol (CIWA-Ar) was developed to aid in monitoring the progress of treatment. This scale consists of ten items which scores the patient based on severity of nausea, sweating, agitation, headache, anxiety,
tremor, sensory disturbances and orientation (6).

The mainstay of treatment is to alleviate symptoms of withdrawal, prevent further progression, treat underlying co-morbidities, and plan for long-term rehabilitation. Benzodiazepines (BZD) have been established as the medications of choice for the prevention and treatment of AWS and DTs. There are no studies showing one BZD to be more efficacious than another. Lorazepam may be favorable in some clinical situations because it offers IM administration, and does not have active metabolites making it an attractive choice for patients with decreased hepatic or renal functions (3). Other medications used in treating the psychiatric symptoms associated with DT such as anxiousness, hallucinations, and combativeness are those such as haloperidol. Caution needs to be taken, because this drug can cause prolonged QT and torsades. Phenobarbital is another choice in managing DT because of its similar action to BZD. There is increased risk of respiratory depression and coma, has a longer half-life which makes it harder to titrate and harder to gauge the patient’s progress (3).

In 1997, studies were performed on rats to test the effect that dexmedetomidine had on alleviating ethanol withdrawal symptoms. Dexmedetomidine is a selective alpha 2-adrenoceptor agonist which was injected into rats after a 4-day ethanol intoxication period. The severity of the ethanol withdrawal symptoms (rigidity, tremor, irritability, hypoactivity) was monitored up to 58 hours after withdrawal of ethanol. It was found that the dexmedetomidine at a dose of 10mcg, reduced the withdrawal symptoms when compared to the control group (7). It was suggested that this was sufficient evidence that this drug should be further studied for the treatment of AWS.

In 2008, dexmedetomidine was studied as an adjunctive therapy to BZD for AWS. In this case study, a 30-year-old man with history of alcohol abuse was admitted to the general medical unit for altered mental status and agitation. He was treated with BZD and his condition deteriorated and he was then transferred to the ICU. Due to the poor response to BZD, he was treated with dexmedetomidine, and his BZD were tapered to discontinuation. The patient’s symptoms remained controlled. After 40 hours in the ICU, the dexmedetomidine was tapered to discontinuation. He was discharged 5 days later (2). It is being suggested that dexmedetomidine be used as primary treatment in AWS and DT. BZD are limited in their mechanism of action, only stimulating the GABA receptors, whereas the dexmedetomidine being a central alpha 2-adrenoceptor agonist, induces a state of cooperative sedation and does not suppress respiratory drive. As mentioned above, the CIWA-Ar is used throughout the management of patients with AWS and DT to monitor their progress.

3. The comorbidities associated with the alcoholic patient are vast. The complexities that are seen with alcoholics exist in many organ systems. Respiratory complications include a higher incidence of bacteremia with *Klebsiella pneumoniae* and a mortality rate of up to 66%. There is a higher incidence of tuberculosis, pleurisy, bronchitis and empyema. There is a depression of normal defense mechanisms, depression of normal mucociliary function as well as the impaired function of neutrophils and macrophages in alcoholics. Upper GI bleeding is the biggest gastrointestinal complication among alcoholics. Varices develop in 12-77% of patients and are the most common cause of UGI bleeding. Other GI complications include acute necrotizing pancreatitis and occurs in 20-30% of cases of pancreatitis. Ethanol causes dysfunction of the sphincter of Oddi and subsequent biliary, duodenal and pancreatic secretion reflux. There are many metabolic and renal complications, the most common include hypokalemia, hypomagnesemia, hypophosphatemia, hypoglycemia, ketoacidosis and lactic acidosis. Cardiovascular complications include cardiomyopathy (due to myocyte loss, ganelle dysfunction and a decrease in contractile proteins); dysrhythmias (due to myofibrillar necrosis, interstitial fibrosis and dysfunction of myocyte sarcolemma and mitochondria); and variant angina (due to elevation in catecholamine) (1).
4. Neurologic complications are the hallmark of alcohol withdrawal. There are four stages of withdrawal: autonomic hyperactivity, hallucinations, neuronal excitation and lastly delirium tremens (1). Delirium tremens is a serious complication of ethanol withdrawal, affecting 5-10% of patients admitted to the hospital. The mortality rate ranges from 5-15% and is attributable to the complications associated with the manifestations. Patients with hallucinations may become destructive and hurt themselves, or an otherwise cardiac-healthy patient can die from an MI secondary to coronary spasms associated with the intense autonomic hyperactivity (3). Ethanol disrupts the balance between GABA and NMDA in the brain. Prolonged ETOH exposure causes NMDA to be upregulated and GABA to be downregulated, leading to tolerance. During ETOH abstinence this is reversed. There is enhanced NMDA function and reduced GABA transmission (3). There have been studies showing that in alcoholic-delirious patients, thiamine absorption is less than 40% which is thought to lead to the vivid optical hallucinations (4). DTs poses many challenges to the clinician, but knowing the pathophysiology and treatment can limit the severe complications associated with withdrawal.

LEARNING POINTS/TAKE HOME MESSAGES 3 to 5 bullet points

- Alcohol abuse is a very common problem facing clinicians. Risk stratifying patients and obtaining laboratory tests can give valuable information to the provider about the severity of alcohol abuse and potential of progressing to withdrawal.

- For any patient presenting to the ED with altered mental status, other etiologies need to be ruled out. Treatment for alcohol withdrawal should not be withheld in order to prevent progression to DT.

- It is important to remember that the alcoholic patient has many other comorbidites that cannot be overlooked. Attention needs to be paid to cardiac, gastrointestinal, renal and other organ systems which are also effected by alcohol and alcohol withdrawal.

REFERENCES


**Date:**
July 23, 2009

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Author’s last name and date of submission, eg,

Smith_June_2008.doc