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A Complicated Case of Brain Death in a 50M Patient Experiencing Alcohol Withdrawal

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Abstract:
This case study presents a 50-year-old male with a history of alcoholism who was admitted for alcohol withdrawal syndrome and subsequently experienced rapid deterioration, necessitating ICU care and endotracheal intubation. Neuroimaging revealed diffuse cerebral edema, raising concerns about anoxic brain injury and uncal herniation. The complexities of this case underscore the critical need for vigilant monitoring and comprehensive assessments in managing alcohol use disorder, particularly in patients with intricate medical histories. The findings highlight the interplay between alcohol withdrawal, cardiovascular complications, and neurological risks, emphasizing the importance of a proactive, multidisciplinary approach in Clinical decision-making.
Keywords: *Brain Death, Alcoholism*

Introduction:

Alcohol use disorder is a disease that affects nearly 100 million people worldwide and leads to close to 3 million deaths annually. Alcohol withdrawal in individuals who chronically abuse it can lead to life-threatening conditions. Delirium tremens which are characterized by seizures, tremors, hypertension, tachycardia, and diaphoresis 48 to 96 hours after withdrawal can be life-threatening for individuals. Alcohol abuse can lead to complications such as cerebral edema and increased intracranial pressure due to inappropriate secretion of vasopressin (1). If alcohol withdrawal is not managed promptly, it can adversely affect vulnerable regions of the brain such as the hippocampus, cortex, and cerebellum. Uncal herniation results from increased intracranial pressure (ICP) causing temporal lobe displacement, leading to severe neurological symptoms such as headache, altered mental status, Cushing's triad, and unilateral pupil dilation (2). Diagnosis is confirmed with CT imaging, while management
aims to rapidly reduce ICP through medical and surgical interventions.Chronic alcohol use increases the risk of head injuries, potentially leading to intracranial hemorrhage and herniation. Alcohol can also cause brain atrophy, complicating recovery and obscuring symptoms, which may delay diagnosis. Additionally, individuals with heavy alcohol use may have other health issues that worsen outcomes. Early recognition and intervention are essential. This case highlights the complex challenges in managing alcohol use disorder, emphasizing the need for healthcare providers to take a vigilant, proactive, and collaborative approach. The uncertainties in the patient's medical history stress the necessity of comprehensive assessments, including thorough substance use histories, to inform effective clinical decision-making in the complicated realm of alcohol withdrawal and its associated complications.

Case Presentation:

We present the case of a 50-year-old M, with a history of alcoholism, who was admitted to the Emergency Department (ED) on 10/28 for ETOH withdrawal syndrome. On admission, ETOH was 28 (reference < 10mg/dL), Anion gap of 18 (reference 4-12 mEq/L),

WBC 14,900 (reference 4,000 - 11,500 count/mL), Hgb 17.8 (reference 13.2 - 16.6 g/dL), Hct 51.8 (reference 41-50%), Glu 339 (reference 70-100 mg/dL). Hospital course was remarkable for overnight rapid response on 10/30 resulting in endotracheal intubation and admission to the ICU unit. The patient remained intubated and was started on Cardizem drip, and on Levophed. Labs on 10/31 revealed Glu 295, WBC 15.1. ABG pH 7.45 (reference 7.35-7.45), PCO2 41 (reference 35-45 mmHg), PO2 190 (reference 75-100 mmHg). EKG on 10/31 revealed sinus tachycardia, pulse of 125 per minute and a pattern in inferior leads possibly indicating an inferior wall myocardium infarction (MI). On 11/01, the neurology team was consulted due to loss of brain
stem reflexes and concern for brain death. A Computed Tomography (CT) of the brain was performed on 11/06, revealing unremarkable changes in comparison to previous imaging. A
Nuclear Medicine Perfusion Brain Scan (NM) was performed on 11/06 which showed "absence of intracranial uptake/cerebral perfusion. Sequential uptake seen in the common carotid arteries and external carotid arteries". An Electroencephalogram (EEG) performed on 11/07 reported signs of severe encephalopathy. CT neuroimaging revealed diffuse cerebral edema, with an
absence of cervical spinal cord injury. The patient's condition was deemed irreversible due to diffuse malignant cerebral edema secondary to hyperosmolar diabetic ketoacidosis. On 11/11/23 the patient became asystole and proxy designation stated DNR. Patient chest was auscultated and no cardiac sounds were appreciated. No pulse was appreciable in the carotid, femoral, or radial
arteries. Corneal reflex was absent bilaterally. The endotracheal tube was disconnected from the ventilator and there was no fogging of the tube. There were no spontaneous respirations. Time of death was 11/11/2023 at 14:15 hr.
Patient ranged from hypernatremic and hyponatremic with lab values ranging from 174 on 11/03 and low of 131 on 11/01.

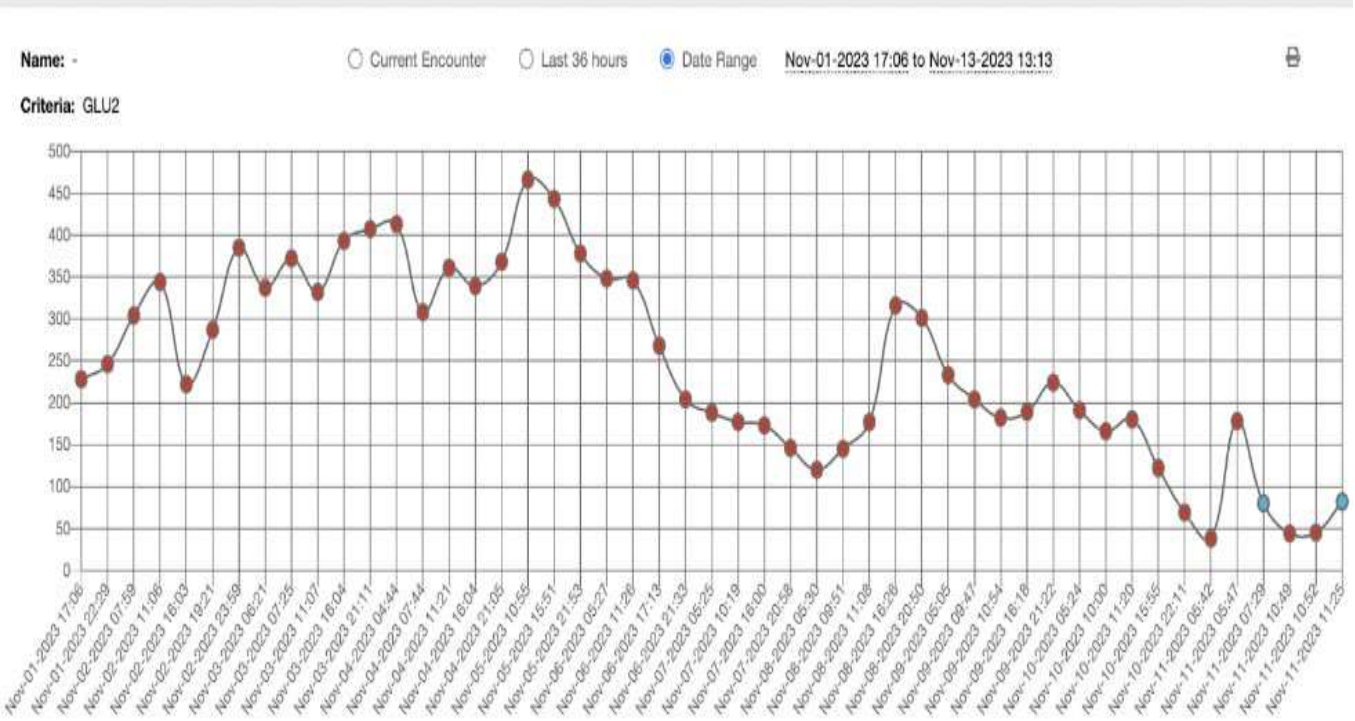

Figure 1. Glucose trends of patient during hospital stay.

Figure 2. 11/06 Brain CT IMPRESSION: 1. No distinct herniation is visualized on this CT. Note this study is suboptimal in terms of patient positioning and thus evaluation of herniation is poor
2. Imaging features suggestive of diffuse cerebral edema. Overall appearance of this edema appears similar to CT brain from 11/2/23. 3. The sulci and cisterns appear diffusely hyperdense, similar to CT brain from 11/4/23. This may be due to diffuse subarachnoid hemorrhage or may represent pseudo subarachnoid hemorrhage in the setting of diffuse cerebral edema. 4. Paranasal sinus disease. Can consider sinusitis, including acute sinusitis, in the appropriate clinical context.
5. Partial opacification of bilateral mastoid air cells. Can consider bilateral mastoid effusions. Less likely mastoiditis in the appropriate clinical context.

Discussion:

The etiology of this patient's demise remains inconclusive given the complicated interplay of diseases. One potential diagnosis includes dilated alcoholic cardiomyopathy leading to cardiac conduction abnormalities in the setting of an indeterminate timing inferior wall MI. This potentially led to a sentinel cardiac event despite going undetected with negative troponemia and insignificant D-dimer. This raises questions about the sensitivity of the diagnostic measures that were employed. Alcoholic cardiomyopathy occurs most commonly in males ages 30-55 and mortality rates are higher in males (5). The patient in this case falls into this demographic further supporting the likelihood of this diagnosis.

The cardiac echo findings further contribute to the diagnostic puzzle, revealing severe dilatation of the left ventricle, decreased left ventricular systolic function, and borderline concentric left ventricular hypertrophy with global hypokinesis. These cardiac abnormalities may have played a pivotal role in the patient's clinical course, underscoring the importance of comprehensive cardiovascular assessments in cases of alcohol withdrawal.
An additional layer of complexity is introduced with the suspicion of nosocomial arrhythmogenic polypharmacy as a potential etiology for the undetected cardiac arrest. The intricate interplay between medications administered in a hospital setting and their potential arrhythmogenic effects emphasizes the need for

cautious and vigilant pharmacological management, particularly in patients with a history of alcoholism and unknown medical backgrounds.

The unclear etiology of the anoxic brain injury highlights the challenges in attributing the patient's demise to a specific cause. The importance of close monitoring is underscored, especially in cases where the patient's medical history is unknown or complicated by substance use disorders. Of note, anoxic brain injury has been shown to be a complication after cardiac issues, which this patient showed signs of on imaging (4). This emphasizes the necessity of a proactive and vigilant approach in managing patients with complex medical backgrounds.

Imaging reveals potential uncal herniation in the patient. The discussion on uncal herniation adds another layer of understanding to the potential neurological complications in this case. Uncal herniation "occurs when rising intracranial pressure causes portions of the brain to flow from one intracranial compartment to another; this is a life-threatening neurological emergency and indicates the failure of all adaptive mechanisms for intracranial compliance" 2. The physical exam presentation for an uncal herniation will reveal Cushing's triad which consist of hypertension, bradycardia, and apnea or irregular respirations 2. This reinforces the importance of recognizing and promptly addressing such life-threatening conditions in clinical practice.

Conclusions:

In conclusion, the case underscores the importance of closely monitoring patients undergoing alcohol withdrawal, particularly in those with co-existing medical conditions. Alcohol use disorder affects approximately 100 million people globally and poses significant health risks and possible death during withdrawal. The patient, a 50-year-old male with a history of alcoholism, presented initially with alcohol withdrawal syndrome marked by elevated ETOH levels, anion gap, and abnormal blood counts. The subsequent rapid deterioration necessitated ICU admission, endotracheal intubation, and aggressive medical interventions. The complexity of the case unfolded as severe dilated alcoholic cardiomyopathy, an indeterminate timing inferior wall MI, and nosocomial arrhythmogenic polypharmacy were revealed and played a role in the patient's decline.

The diagnostic challenge lay in discerning the primary cause of death, with anoxic brain injury manifesting as diffuse cerebral edema evident in neuroimaging. The cardiac echo revealed left ventricular dilatation, hypertrophy, and global hypokinesis, suggesting cardiomyopathy as well as potential cardiac conduction abnormalities. Simultaneously, the role of nosocomial arrhythmogenic polypharmacy highlighted the intricate interplay of factors in patients with substance use disorders. The uncertainties surrounding the patient's medical history, in conjunction with the complexities of alcohol withdrawal and its potential complications, emphasize the need for comprehensive, multidisciplinary care. The limitations of diagnostic imaging, particularly in differentiating pseudo-subarachnoid hemorrhage from actual subarachnoid hemorrhage in the context of diffuse cerebral edema, add to the intricacies of thecase.

In conclusion, this case serves as an important reminder of the multifaceted challenges in managing alcohol use disorder, urging healthcare providers to adopt a vigilant, proactive, and collaborative approach. The uncertainties surrounding the patient's medical history underscore the importance of thorough assessments, including detailed substance use histories, to guide effective clinical decision-making in the intricate landscape of alcohol withdrawal and itspotential complications.

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