Acute infarct of the left caudate nucleus presenting as alcohol withdrawal: A case study

Thomas J Valentine, DO[1], Jaydip N Mehta[2], and Heather PM Theibert DO[3]

1. PGY2 Psychiatry Resident, OhioHealth Riverside Methodist Adult Psychiatry Program, Columbus, Ohio
2. OMSIV, Ohio University Heritage College of Osteopathic Medicine, Dublin, Ohio
3. Assistant Program Director, OhioHealth Riverside Methodist Adult Psychiatry Program, Columbus, Ohio; Adjunct Clinical Assistant Professor, Ohio University Heritage College of Osteopathic Medicine, Dublin, Ohio

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Introduction

- Caudate strokes can present due to a variety of etiologies, with the most common being a sequela of hypertension, hypercholesterolemia, and diabetes mellitus[4].
- The symptoms of caudate stroke include restlessness, disinhibition, anxiety, confusion, and agitation[4].
- This presentation is similar to that of alcohol withdrawal.
- Alcohol withdrawal occurs after the sudden cessation of prolonged alcohol consumption and can lead to symptoms of anxiety, headache, diaphoresis, palpitations, delirium, seizure, hallucinations, agitation, and autonomic disturbance with potentially life-altering consequences[8].
- The following case describes a patient with symptoms initially concerning for prolonged alcohol withdrawal which later was confirmed as a left caudate infarction.

Patient History

- Patient is a 48-year-old male with a history of alcohol use disorder who presented to the ED from an alcohol rehabilitation facility for concern of worsening alcohol withdrawal.
- Patient's last use of alcohol was approximately six days prior to hospital admission. Detoxification of alcohol with chlordiazepoxide and PRN lorazepam was initiated four days prior to his hospital admission.
- The night prior to admission, patient's CIWA scores increased, and patient exhibited disorientation, diffuse non-focal weakness, and an episode of falling and hitting head.

Physical Exam

- Vital signs in the ED revealed the following: blood pressure 163/106, temperature 98.4, heart rate 86, respiratory rate 17, O2 stat 97%.
- Records indicate that patient was disoriented to location and circumstance.
- Neurologic exam revealed that patient exhibited +3-4/5 strength in bilateral lower extremities and +4/5 in bilateral upper extremities as well as a tremor in upper extremities bilaterally.
- HEENT, cardiovascular, pulmonary, abdominal, and musculoskeletal exam was not revealing.

Laboratory/Imaging Results

- On admission, lab work showed low magnesium at 1.3. CBC, BMP, LFT, lactic acid, troponins, TSH within normal limits. Blood alcohol level <10 (Unfortunately, a urine drug screen was not ordered at the time of admission).
- CT of head and c-spine showed no acute intracranial process.
- EKG demonstrated T-wave inversions in leads 1, 2, aVL, V4-V6.
- EEG results suggestive of mild, nonspecific encephalopathy.
- As patient's symptoms did not improve by hospital day #10, MRI of brain was ordered which revealed a lacunar infarct of the left caudate and right thalamus/internal capsule.

Differential Diagnoses

- Alcohol withdrawal with delirium
- Delirium due to unknown medical etiology
- Wernicke's encephalopathy
- Traumatic brain injury secondary to fall
- Drug intoxication

Hospital Course

- On hospital day #1, patient was started on a fixed 5-day lorazepam taper as well as daily thiamine and a multivitamin with further concerns for Wernicke's encephalopathy given additional symptom of ataxia[6]. Symptoms showed no improvement after completion of lorazepam taper. Scheduled dosing of valproic acid and as needed ziprasidone were later ordered for agitation management.
- Throughout hospital course, patient exhibited worsening neurological symptoms including the following: inability to stand without assistance, non-purposeful upper extremity movements, poor hand-eye coordination, and slowed, disorganized speech. Additionally, patient was noted to occasionally respond to internal stimuli, exhibited looseness of association, and minimal engagement with staff.
- Neurology was consulted, and an MRI ordered revealed a lacunar infarct of the left caudate and right thalamus/internal capsule. This finding provided a clear etiology of altered mental status.

Alcohol Withdrawal[7]	Shared Symptoms[3,7]	Left Caudate Stroke[3]
Hallucinations	Tremor	Speech Deficits
Seizure	Confusion	Paresis
Nausea/Vomiting	Disorientation	Gait/Motor Abnormalities
Autonomic Disturbance	Restlessness	
	Agitation/Anxiety	
	Changes in Affect	
	Disinhibition	

Table 1. Symptoms of alcohol withdrawal and left caudate stroke

Pathophysiology

- The caudate nucleus is involved in learning, memory, speech, motor control, and behavioral inhibition[3].
- Individuals predisposed to an infarction in this region have risk factors that include hypertension, hypercholesterolemia, diabetes mellitus, and previous myocardial infarct. This patient was noted to have significant hypertension throughout the admission.
- When an infarction of the left caudate occurs, patients often exhibit motor abnormalities, deficits in cognition, speech deficits, paresis, disorientation, restlessness, disinhibition, changes in affect, agitation, and impulsivity[1,2,3].

Treatment

- When the diagnosis of a lacunar infarct of the left caudate and right thalamus/internal capsule was made, patient was started on aspirin, clopidogrel, and atorvastatin as well as blood pressure control with lisinopril and clonidine.
- Patient was discharged to an inpatient rehabilitation unit.

Discussion

- This patient demonstrated signs of left caudate stroke with possible concurrent delirium which were initially mistaken as alcohol withdrawal with Wernicke's encephalopathy due to recent alcohol withdrawal and initial negative Head CT. These illnesses share several symptoms (Table 1).
- Left caudate stroke could have been further discerned if emphasis on weakness, speech deficits, and ataxia despite thiamine supplementation was prioritized.
- This clinical picture became further blurred as patient's last use of alcohol was six days before admission to the ED and had recently received a chlordiazepoxide taper while at his alcohol rehabilitation facility. In addition, CT scan of the head did not show acute stroke abnormalities. This timeline clouded the clinical picture as to whether this presentation had been due to a protracted alcohol withdrawal or an unknown medical issue given alcohol withdrawal can persist up to six days after cessation[5].
- In this case, having been cognizant of the timeline of last alcohol use and subsequent treatment with continued worsening of symptoms would have allowed for additional neurological etiologies to have been considered and investigated earlier.

Conclusion

This case demonstrates the importance of clinical timeframe, subsequent associated neurologic symptoms, and ruling in underlying neurologic pathologies. Consideration of stroke or other central nervous system damage in those with AMS should not be ruled out. Delay in diagnosis and treatment of stroke pathology could result in further neurological deterioration.

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