Identification of Wernicke Encephalopathy in a Patient Presenting With Altered Mental Status and Dehydration

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ABSTRACT

Introduction: Providing glucose before thiamine can cause or exacerbate Wernicke encephalopathy, a potentially life-threatening condition associated with a variety of neurological impairments.

Case Presentation: An emaciated, middle-aged woman with a longstanding history of alcohol abuse and an undifferentiated seizure disorder presented to a local emergency department with altered mental status of unknown duration. Initial labs showed signs of acute kidney injury and she could not tolerate oral intake. Overnight, dextrose-containing maintenance fluids were started. The next day, she had an acute deterioration of mental status. Empiric therapy for Wernicke encephalopathy was begun, resulting in resolution of most of her symptoms over a matter of days.

Discussion: It is generally recommended to administer thiamine treatment prior to glucose in patients with suspected thiamine deficiency. The Caine criteria can assist in the decision to start empiric treatment to prevent delays in thiamine therapy.

Conclusion: Wernicke encephalopathy is a disease with high morbidity that is usually treated with the generally benign therapy of thiamine. Given the risk of harm of untreated Wernicke encephalopathy and the benign nature of treatment, clinicians should have a low threshold to provide thiamine therapy.

INTRODUCTION

Wernicke encephalopathy results from inadequate thiamine intake. If left untreated, it can progress to coma and death and, even with treatment, can have lasting effects on patients' memory and ability to walk. Providing glucose without thiamine can precipitate or exacerbate Wernicke encephalopathy.

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We present a case of a woman who presented with altered mental status and was given dextrose-containing maintenance fluids, precipitating Wernicke encephalopathy. This case is important to share because it demonstrates a potential clinical consequence of administering dextrose-containing fluids to a patient presenting with confusion and dehydration. It serves as a reminder to clinicians to remember the clinical context and importance of thiamine therapy for patients at risk of developing Wernicke encephalopathy, particularly those who are malnourished or have a history of alcohol use.

CASE PRESENTATION

A 43-year-old woman presented to the emergency department (ED) with altered mental status. Medical history was significant for seizure disorder, pelvic inflammatory disease, alcoholic hepatitis, folic acid deficiency, and alcohol use dependence. On arrival, she was oriented to person and place but confused with time and situation. She remembered drinking last night but was unsure how she arrived at the ED. She was also uncertain of her seizure history and her antiseizure medication regimen. Vitals were stable on presentation. Physical exam revealed dry mucous membranes, pinpoint pupils, and horizontal nystagmus. Electrocardiogram showed sinus tachycardia with ST elevation in augmented vector right (aVR) and depression in inferior leads; however, both troponin and repeat troponin were negative. Chest x-ray showed no focal consolidation, and head computed tomography showed no intracranial abnormality.

Initial labs (Table 1) resulted in a urine drug screen positive for ethanol, and labs were reflective of alcoholic ketoacidosis (glucose

171, bicarbonate 10, anion gap 26, urinalysis was ketone positive) and mild transaminitis (aspartate aminotransferase [AST] 171, alanine aminotransferase [ALT] 109). Complete blood cell count was negative for leukocytosis but indicated a microcytic anemia, likely due to iron deficiency (hemoglobin 7.8, mean corpuscular volume 77, red cell distribution width 21.2). Ammonia was within normal limits. Her altered mental status was attributed to either alcohol use and subsequent withdrawal or postictal state.

Her physical exam demonstrated poor capillary refill and dry mucus membranes, concerning for dehydration. Concomitantly, she had a prerenal acute kidney injury (serum urea nitrogen [BUN] 34, creatinine 1.43, urinalysis was hyaline cast positive) and was given multiple liters of fluids. These fluids included three 1-L boluses of lactated ringers and a single 1-L bolus of 0.9% saline over the course of 4 hours, followed by 250 mL of 5% dextrose and 0.45% saline with 20 meEq of potassium chloride over the course of 3 hours. After these initial fluids, she was given 1 intravenous (IV) dose of 100 mg of thiamine, followed by 1 L of 5% dextrose and 0.9% saline over the course of 13 hours. The following morning, she was only responsive to painful stimuli and exhibited leftward gaze palsy of the right eye and bilateral myoclonic twitching. Neurology was consulted and noted her suboptimal nutritional intake and abnormal extraocular movements, consistent with the Caine criteria.[1] She was started on 500 mg of IV thiamine 3 times daily for empiric treatment of Wernicke encephalopathy.

The next day, she was awake and alert but still confused about details from the last few days. Magnetic resonance imaging was consistent with Wernicke encephalopathy showing development of symmetric hyperintense signal in the bilateral posterior medial thalami, mammillary bodies, and periaqueductal gray (see Figure). Concern for seizure activity was ruled out by bland 24-hour continuous electroencephalogram. Physical therapy noted her to have balance deficits and gait deviations, and physical therapy sessions only resulted in slow improvement of her mobility. She received 500 mg of thiamine infused over 30 minutes 3 times daily for 3 days, 250 mg of thiamine infused once daily for 2 days, a 2-day supply of 250 mg oral thiamine, and 100 mg thiamine thereafter. Horizontal nystagmus and left gaze palsy resolved after 2 days of thiamine supplementation, and orientation improved throughout her hospital stay. She was discharged home under frequent supervision of her family with a 2-wheeled walker, home physical therapy, oral thiamine supplementation, and outpatient follow-up with her primary care physician and neurology.

DISCUSSION

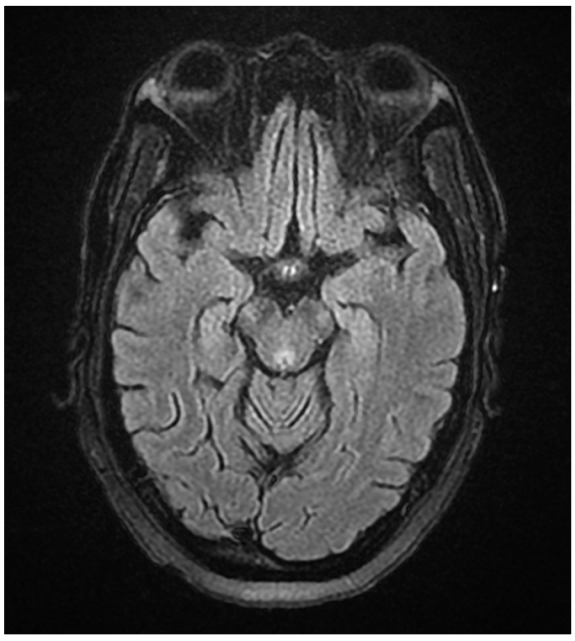
It is traditionally recommended that thiamine be given prior to glucose in patients to prevent worsening Wernicke encephalopathy; however, the amount and duration of glucose to have such an effect are still unclear. Schabelman et al conducted a literature review consisting of previously reported Wernicke encephalopathy cases and animal models to determine the effects of glucose prior

Table 1. Lab Results

Lab	ED Workup	1 Day Before Discharge	Reference Range
Sodium	138	136	136-145 mmol/L
Potassium	3.8	3.8	3.4-5.1 mmol/L
Chloride	102	105	99-105 mmol/L
Bicarbonate	10	22	22-29 mmol/L
BUN	34	5	6-23 mg/dL
Creatinine	1.43	0.61	0.5-1.10 mg/dL
Glucose	171	88	65-99 mg/dL
AST	171	--	11-33 unit/L
ALT	109	--	6-37 unit/L
Anion gap	26	9	10-18 mmol/L
Ammonia	11	--	11-60 umol/L
Urine drug screen	Ethanol only	--	Negative
Lactic acid	4.3	--	0.5-2.0 mmol/L
ABG pH	7.42	--	7.32-7.42
White blood cell	9.0	8.2	3.9-11.2 10e3/uL
Hemoglobin	7.8	7.6	11.3-15.1 g/dL
Hematocrit	27	25	34-45%
MCV	77	81	79-98 fL
Red cell distribution width	21.2	19.3	11.0-14.9%
Platelet count	490	353	165-366 10e3/uL
Urine specific gravity	1.027	--	1.005-1.030
Ketones urine	1+	--	Negative
Hyaline cast	26-50	--	0-2

Abbreviations: ED, emergency department; BUN, serum urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ABG, arterial blood gas; MCV, mean corpuscular volume.

Figure. Subtle Symmetric T2 FLAIR Signal in the Bilateral Posteromedial Thalami, Bilateral Mamillary Bodies, and in the Periaqueductal Gray Matter; Consistent With Wernicke Encephalopathy Diagnosis



Abbreviation: FLAIR, fluid-attenuated inversion recovery.

to thiamine. They concluded that prolonged or massive glucose supplementation resulted in acute deterioration of mental status.[2] This case report adds to the medical literature of a patient who had an acute deterioration of her mental status within 24 hours, even with a short infusion of 5% dextrose prior to thiamine administration. On initial presentation, she was disoriented and confused but cooperative with exam and only exhibited horizontal nystagmus.

To prevent delays in thiamine therapy, Wernicke encephalopathy should be considered a "cannot miss" diagnosis in the differential for patients presenting with altered mental status. Our patient's atypical presentation of Wernicke encephalopathy and poor history did not make diagnosis readily apparent. Wernicke encephalopathy is known to be more common in patients with chronic alcoholism since it results in suboptimal nutrition, reduced gastrointestinal absorption, and decreased liver storage.[3] However, it is a rare condition with an average prevalence of 1.3% of all autopsies.[3] Additionally, the differential for altered mental status is extremely broad and causes can include infections, metabolic disturbances, and toxins, such as alcohol. The patient's history of seizures and recent alcohol use seemed to be reasonable causes for her confusion.

Wernicke encephalopathy is traditionally taught as the triad of altered mental status, ocular dysfunction, and gait ataxia; however, reliance of this triad leads to misdiagnosis. An observational study found these 3 symptoms present in about one-third of patients.[4] Our patient exhibited the Wernicke encephalopathy triad, but these signs were not apparent initially. She was interviewed and examined laying on the hospital bed, so her unsteady gait was not noted until she started physical therapy. Horizontal nystagmus was documented during initial presentation, but nystagmus is also known to be associated with acute alcohol intoxication.[5]

According to the operational criteria proposed by Caine et al, Wernicke encephalopathy should be diagnosed if a chronic alcoholic presents with altered mental status and/or memory impairment and has at least 1 more of the following domains: dietary deficiencies, oculomotor dysfunction, or cerebellar dysfunction (Table 2).[1] There were indications in this patient's chart and exam for dietary deficiencies. She was noted to be thin with a low body mass index, and she had a documented history of folic acid deficiency. Her labs indicated a new microcytic anemia due to iron deficiency, which also could be an indication for poor nutritional intake. The Caine criteria should be used to clinically diagnose Wernicke encephalopathy rather than the classic triad.

Thiamine has been established as a safe and effective treatment of Wernicke encephalopathy.[3] Following administration of high-dose thiamine, our patient's mentation improved and ocular abnormalities resolved within a few days. Based on this experience, we recommend that clinicians have a low threshold for administering thiamine therapy immediately—especially when glucose solutions are considered.

Table 2. Caine Operational Criteria for Wernicke Encephalopathy Requires 2 of 4 Signs in Chronic Alcoholics[1]

Signs	Examples
Dietary deficiencies	Malnutrition, vitamin deficiency, low body mass index
Oculomotor abnormalities	Ophthalmoplegia, nystagmus, gaze palsy
Cerebellar dysfunction	Ataxia, balance deficits, dysmetria
Altered mental status or impaired memory	Disorientation, coma, confusion Mild-moderate memory problems, confabulation

CONCLUSION

Despite its low prevalence, Wernicke encephalopathy should be considered a "cannot miss" diagnosis for patients, since it can result in chronic neurological impairments if treatment is delayed. This case advocates for the widespread use of the Caine criteria rather than the classic triad when making a clinical diagnosis of Wernicke encephalopathy, because not all 3 signs of the triad may be readily apparent.

If Wernicke encephalopathy is viewed as a "cannot miss" diagnosis for change in mental status, clinicians can immediately start thiamine for empiric treatment. Furthermore, clinicians should be cautious in administering solutions containing glucose to patients presenting with altered mental status, since this could be a sign of thiamine deficiency and potentially precipitate or worsen Wernicke encephalopathy. Thiamine is a benign treatment, thus clinicians should have a low threshold for providing thiamine therapy to patients.

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