Seeing beyond stereotypes: Wernicke's encephalopathy in a non-alcoholic patient

Kari Brown MBBS, MSc [1], Varsha Muddasani MD[1], Zurab Azmaiparashvili MD, MS [1]
[1]Department of Medicine, Albert Einstein Medical Center, PA

Learning Objectives

- Wernicke's encephalopathy (WE) is a neurologic condition commonly linked to alcohol use, but at its core results from biochemical abnormalities caused by nutritional deficiency of vitamin B1 (thiamine)
- Absence of alcohol use should not preclude the diagnosis, and uncharacterized neurological symptoms should always prompt consideration of nutritional etiologies.

Case

A 74-year-old female was brought into the emergency room for altered mental status, double vision, imbalance, and vertigo following three weeks of reported diarrhea, nausea, and vomiting. No fevers or arthralgias. The patient's oral intake was noted to be poor over the preceding months. Past medical history was significant for depression maintained on bupropion which had been discontinued weeks prior. She denied any alcohol use.

Physical examination:
- Afebrile, hemodynamically stable
- Alert, but oriented only to person
- Neurologic examination with diffuse tremulousness and hypertonia; no hyperreflexia or nuchal rigidity
- Multi-directional nystagmus present, downbeat and worse in lateral and down gaze
- No papilledema.

Differential diagnosis:
Wernicke's encephalopathy, serotonin syndrome, hyperthyroidism, autoimmune encephalitis, Miller Fisher syndrome, Whipple's disease

Work-up:
- **Lab tests:** Significant for hypokalemia to 2.9 mmol/L and severe hypomagnesemia < 0.6 mg/dL. Blood alcohol level was negative. TSH and free T4 normal. LFTs unremarkable (See Table 1)
- **Urine drug screen:** Positive for cannabis
- **CTA:** No acute intracranial hemorrhage, patent cervical and intracranial vasculature
- **MRI brain:** No structural abnormalities of the cervico-medullary junction. No evidence of multiple sclerosis or acute CVA. Mild small vessel ischemic changes

Case Continued

Management:
- Electrolytes were repleted
- Received IV thiamine
- Rapid improvement in her mental status, with gradual resolution of other neurologic symptoms.

Serology	Value	Serology	Value
Na	145 mmol/L	Glucose	175 mg/dL
K	2.9 mmol/L	Ammonia	37.4 mcmol/L
Cl	109 mmol/L	TSH	1.32 mcIIU/mL
HCO3	16 mmol/L	Alcohol level	<10 mg/dL
BUN	31 mg/dL	RPR	Non-reactive
Cr	1.95 mg/dL	DPPX Antibody	Negative
Ca	8.5 mg/dL	B12	835 pg/mL
Mg	<0.6 mg/dL	C3/C4	Within Normal Limits

Table 1. Initial serology

Discussion

- Wernicke's encephalopathy (WE) is primarily a clinical diagnosis made in the presence of two of the four diagnostic Caine criteria: ocular signs, dietary deficiency, cerebellar dysfunction, and altered mental status[1]. Due to a low index of suspicion, its classic association with chronic alcohol use has led to underdiagnosis of the condition, particularly when frequently not all criteria are present. Our patient met all diagnostic criteria, with dietary deficiency resulting from protracted poor oral intake.
- Thiamine levels are neither necessary nor reliable in confirming the diagnosis[2], and testing should not delay empiric repletion. Additionally, magnesium is a co-factor for thiamine-dependent metabolism, and interestingly it was also severely deficient in our patient. Hypomagnesemia likely explains the patient's tremors and hypertonia, and indeed vertical nystagmus when present, essentially pins a metabolic diagnosis to either thiamine or magnesium deficiency. This makes simultaneous repletion of both important for clinical improvement in patients unresponsive to treatment with thiamine alone.
- Case highlights the importance of considering nutritional causes when evaluating patients, and of maintaining a high index of suspicion for Wernicke's encephalopathy even in the absence of alcohol use.

References

1. Caine D, Halliday GM, Kril JJ, Harper CG. Operational criteria for the classification of chronic alcoholics: identification of Wernicke's encephalopathy. J Neurol Neurosurg Psychiatry. 1997 Jan;62(1):51-60. doi: 10.1136/jnnp.62.1.51. PMID: 9010400; PMCID: PMC486695.
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