



A Case of Atypical Wernicke's Encephalopathy: Suspicion Trumps All

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INTRODUCTION

Thiamine plays a significant role in cerebral metabolism, requiring 80% of the total thiamine in nervous tissue to process glucose as the primary energy source (**Figure 1**). Risk factors for thiamine deficiency include diminished intake, increased metabolic demand, and resuscitation with intravenous glucose-containing fluids. Thiamine deficiency can manifest in multiple clinical syndromes such as dry beriberi with peripheral neuropathy, wet beriberi with cardiomyopathy, and Wernicke-Korsakoff syndrome.

Commonly associated with chronic alcoholism, textbooks diagnose Wernicke's encephalopathy as a triad of symptoms: encephalopathy, gait ataxia, and oculomotor dysfunction. However, recent meta-analyses only note one-third of patients presenting with all three clinical features.

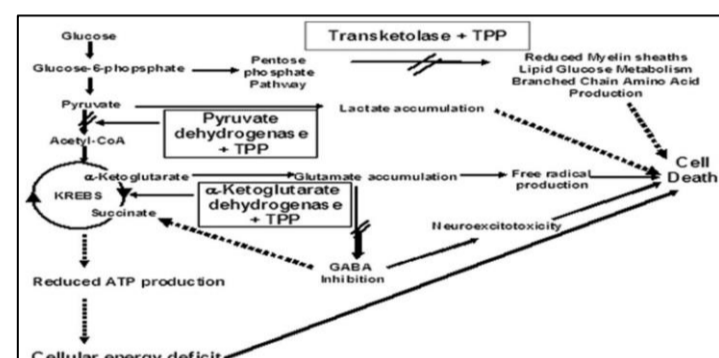


Figure 1. Thiamine pyrophosphate serving as a crucial enzyme at multiple steps for energy metabolism

CASE DESCRIPTION

HPI: 60-year-old male with medical history of transient ischemic attack (2015), hypertension, and type 2 diabetes, presented to the ED with 24-hour history of worsening disequilibrium after a seizure-like episode one day prior. At that time, patient reported 5-minute episode of sudden-onset weakness and tremors, which was attributed to hypoglycemia. The next day, patient reported worsening left-sided weakness and difficulty ambulating.

Of note, patient previously admitted in recent years for hypoglycemia and for alcohol withdrawal symptoms. At present, patient reports minimal alcohol use, with last drink of two servings of vodka two nights prior to admission.

PHYSICAL EXAM:

Vitals: Afebrile, BP 166/97, HR 79, RR 13, O2 100% on room air
Neurological - drowsy, **slowed speech**, cranial nerves II through IX intact except **pupils L reactive but R minimally constricts with light**, strength 5/5 RUE/RLE, **4/5 LUE**, +5/5 L LLE, **gait unstable, finger-to-nose test negative B/L heel-to-shin test negative B/L, rapid alternating hand movements intact**

CLINICAL COURSE

PERTINENT LABS:

Glucose: 209, CK 190, Ethyl alcohol <3, UDS negative
Urinalysis: trace protein, 3+ blood

IMAGING:

CT angiography head neck with stroke protocol: revealed no hemodynamically significant stenosis, occlusion, or aneurysm. Stable atherosclerotic plaque noted in bilateral common carotid artery.

MRI brain revealed no acute infarcts, or hyper intensities around mammillary bodies, periaqueductal region of the midbrain, or thalamus (**Figure 2**).

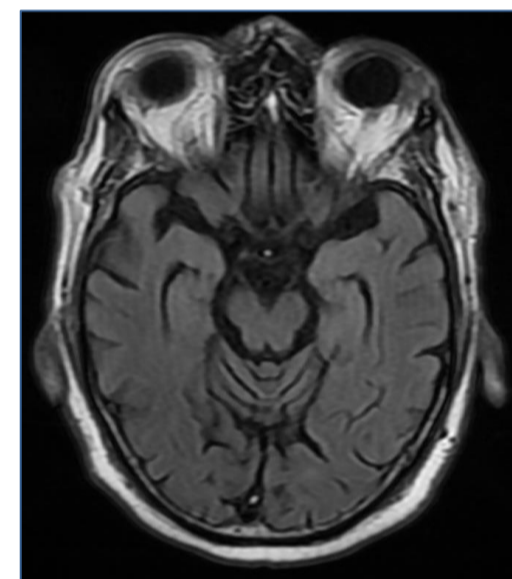


Figure 2. MRI brain without hyperintensity noted around mammillary bodies or midbrain, only noted with less than 20% of patients

DIFFERENTIALS:

- **Acute ischemic cerebellar stroke**
- **Mixed hypoglycemic episode vs seizure**
- **Vestibular neuritis**
- **Chronic alcohol use disorder**

Due to concern for potential cerebellar stroke, patient kept on permissive hypertension for the first 24 hours and started on clopidogrel and atorvastatin. Patient had no post-ictal state and normal glucose levels.

Neurology consult recommended oral prednisone due to suspicion of vestibular neuritis. Despite therapy, patient continued to have persistent disequilibrium and truncal gait difficulties. On hospital day #3, patient developed new right-beating nystagmus with subjective diplopia; differential included vestibular nystagmus due to oculo-vestibular dysfunction. Patient was started on intravenous thiamine with resolution of nystagmus and mental drowsiness.

After intense inpatient rehabilitation course, patient returned to normal gait and function. Due to persistent dizziness, patient required wheelchair on discharge with plan for continued outpatient physical therapy.

DISCUSSION

In this case, there was low suspicion for Wernicke's encephalopathy given clinical history, physical exam findings, and labs. Patient's reported history of chronic alcohol use disorder in setting of any acute neurologic changes should arouse need for thiamine supplementation. As thiamine is both concentration-dependent and transportation-dependent, intravenous thiamine replacement is preferred.

Oral thiamine is noted to have poor absorption in the setting of chronic alcohol use or other intestinal deficits. Given its low risks, all individuals suspected to have low thiamine (due to both alcoholic and non-alcoholic causes) should receive thiamine followed by glucose and magnesium supplementation.

CONCLUSION

Given the deleterious consequences of undertreating thiamine deficiency and cost versus benefit, parenteral replacement therapy needs to be prioritized for high-risk patients - alcoholics and individuals at risk for malnutrition.

High risk individuals for thiamine deficiency should be redefined as two of the following four signs:

- 1) dietary deficiencies or vomiting
- 2) eye signs
- 3) cerebellar dysfunction
- 4) altered mental status or mild memory impairment

Based on bioavailability and prior clinical-pathological studies, expert consensus for dosing recommendations are parenteral thiamine 500mg three times daily for at least two days, followed by 250mg daily for an additional five days.

- High thiamine levels are non-toxic to the body.
- Important to also co-administer magnesium.

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