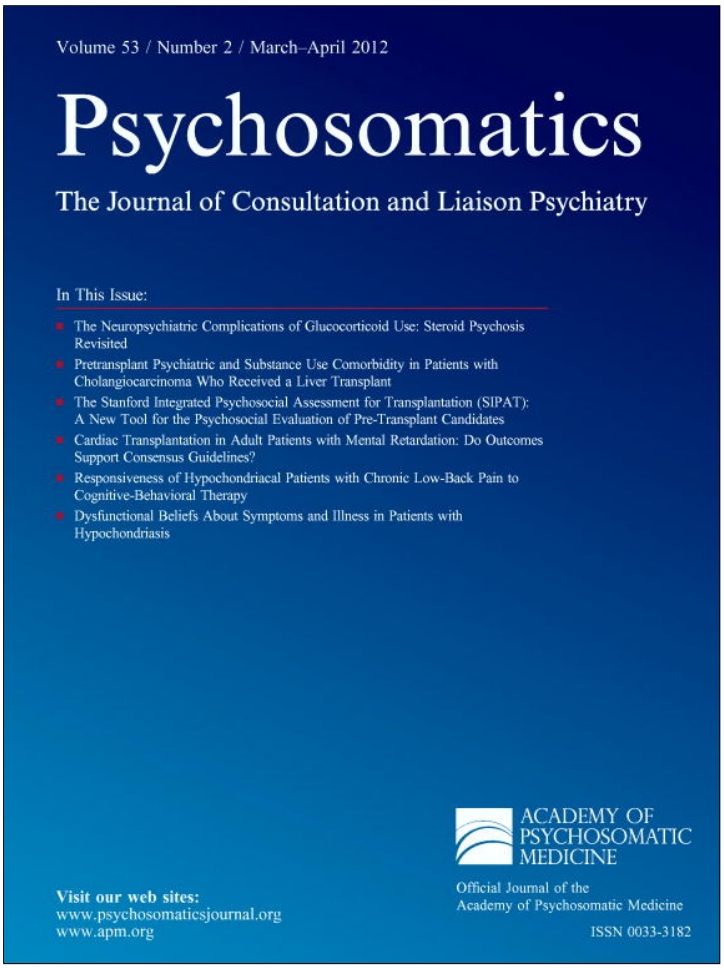

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Case Reports

Wernicke's Encephalopathy in a Patient with Hyperemesis Gravidarum

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The features of Wernicke's encephelopathy have been described for over a century, usually a combination of confusion, ocular abnormalities, and ataxia. However, over the years, the vulnerable population to this disorder has been overwhelmingly described as poorly nourished alcohol dependent patients, and this has even lead to automatic thiamine administration to such patients in emergency settings. Unfortunately, this narrowed view of the at-risk patient often leads to a missed diagnosis, as evidenced in this patient who exhibited this syndrome secondary to hyperemesis gravidarum. We hope to remind providers that the disorder is secondary to a nutrional deficiency, and therefore, today's at-risk population needs to be expanded.

Case Report

Ms. M, a 21-year old African-American (gravida 1 para 0, 14 weeks pregnant) was transferred to the labor and delivery unit of a university hospital from a small community hospital because of elevated liver enzymes, persistent vomiting for 3–4 weeks, and mild confusion. For several months prior to the transfer, Ms. M exhibited continuous severe nausea and vomiting to the point that home health was required for intravenous (IV) fluids. Then, 2 weeks before admission, Ms. M developed cholecystitis and underwent a laparoscopic cholecystectomy, without relief from the vomiting. At some point prior to her emergency admission to the local hospital, Ms. M had a syncopal episode, hit her head on a table, was taken to the local emergency department (ED), was told that "sodium left the body," and had her electrolytes replaced at an unknown rate, and then was discharged home. However, still symptomatic, Ms. M was brought back to the local ED, where given her pregnancy, her increased liver enzymes, recent surgery, and inability to tolerate PO intake, Ms. M was transferred to the university hospital ED.

On arrival, her liver enzymes were elevated (AST was 181U/L, ALT 599U/L, total bilirubin was 1.2 Mg/dL, and alkaline phosphate was normal). Ms. M's sodium was 132 mmol/L and potassium 3.0 mmol/L. Abdominal ultrasound findings were negative for any liver or gall bladder pathology. In addition, she had medical, surgical, and gastroenterology consults, who found nothing remarkable except disorientation. She was admitted to the OB-GYN service, given IV fluids, and monitored for tolerance of oral liquids. Ms. M's liver enzymes improved on the third day of admission and the obstetrics team planned for her discharge. However, on the day of discharge, she was unable to walk, and had actually fallen. This was attributed to conversion from the recent revelation that the father of Ms. M's fetus had a relationship with her friend and the current pregnancy was unplanned. Psychiatry and neurology were consulted to address conversion, ongoing confusion, and disorientation.

The neurologic examination was notable for weakness of lower limb left > right, power 3/5, and decreased deep tendon reflexes bilaterally. Ms. M had drooping eyelids with limited gaze right > left as well as up gaze palsy and horizontal nystagmus. She had dysdiadokinesia, past-pointing, and an unsteady gait. She was oriented only to self, was confused with poor attention, concentration, and recall. Her affect was anxious with depressed mood, her

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speech was slow with a low tone, and her thought process was slow. CK, EMG, and MRI were ordered. Because of a high degree of suspicion, the neurology attending ordered thiamine 100 mg IV for 5 days followed by oral thiamine 100 mg per day, due to altered mental status, ophthalmologic findings, and lower extremity weakness with a history of poor nutrition, as well as negative anti-GQ1b/GM1 antibodies to rule-out Miller Fisher syndrome.

The psychiatry consult occurred several hours after the neurology examination. Since Ms. M was an extremely poor historian, collateral information was obtained from her relatives, who revealed that the nausea and vomiting had been occurring for at least 6 months, that she had fallen at home, and had electrolyte replacement of an unknown rate, and an extended period of confusion even before admission.

Fearing central nervous system (CNS) damage from possible poor electrolyte management, the psychiatrist ordered an emergency MRI of the posterior fossa, which revealed restricted diffusion of gadolinium in both medial thalami, with hyperintensities in the FLAIR and T2 weighted images of the thalami, and hyperintense mammillary bodies; these results confirmed the diagnosis of Wernicke's encephalopathy, making conversion and CNS damage or hematomas unlikely (Figure 1).

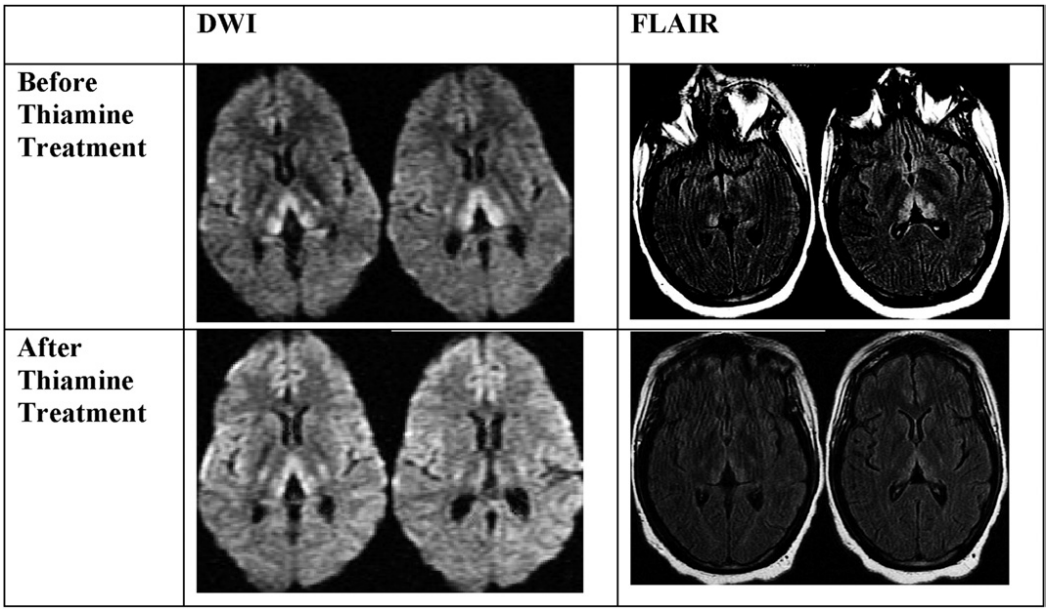
Ms. M was monitored by the obstetrics, neurology, and psychiatry service closely for the next 24 hours after thiamine administration, and her neurologic picture improved to where she was more energetic, and had less opthalmoplegia. She no longer exhibited lid lag, and her gaze palsy improved. However, she continued to have significant horizontal nystagmus in the lateral visual fields bilaterally, continued to have severe lower extremity weakness, and her thought process continued to be very slow, almost bradyphrenic. She had less dysdiadokinesia and past-pointing, but the finger-to-nose test continued to lack smoothness and appeared to be a difficult task requiring much effort for her.

Ms. M was then referred for physical therapy, where she improved neurologically with some recovery of strength in the lower extremities, but continued to exhibit poor memory, raising the question of an amnestic syndrome.

Discussion

Wernicke's encephalopathy (WE) is currently recognized as a potentially fatal but reversible condition which, if untreated, can lead to coma, Korsakoff's psychosis, and death, with mortality ranging from 10% to 20%.[1,2] Even with treatment many survivors are left with persistent neurological deficits.[2,3] It is characterized by a triad of ataxia, confusion, and ocular abnormalities, which are recognized in only 30% of patients.[3] The most frequent ocular sign

FIGURE 1. Comparison of MRI Before and After Thiamine Therapy.



findings are horizontal nystagmus and bilateral paralysis of lateral rectus muscles of the eye. Global confusion, inattention, and disorientation are most common symptoms for altered mental status.[1,2] Mostly, diagnosis is facilitated by having a high suspicion and clinical signs and symptoms. Laboratory workup for decreased blood transketolase activity is time-consuming and often unavailable in many hospitals.[4] Oftentimes, the diagnosis is confirmed with an MRI, usually after treatment with thiamine as it, and other B vitamins are deficient in these patients. Thiamine (vitamin B^1) is an essential water soluble vitamin obtained from the diet, and is involved in glucose metabolism, particularly in the brain, which needs a constant supply of glucose. Body store of this vitamin is around 25 to 30 mg, which may be depleted within 2 weeks if not replenished.[5] The need for thiamine increases during pregnancy and lactation due to the increased energy demands of the body.

Classically, the most common cause of thiamine deficiency is poor nutrition secondary to alcoholism. However, since it is a nutrient deficiency, other causes include dietary deficiency (beriberi being classic), diuretic therapy, thyrotoxicosis, and malabsorption syndromes, bariatric procedures, and as in the above case, hyperemesis gravidarum; these patients often go undiagnosed due to a focus on alcohol-dependent individuals. For instance, although HG has been clearly identified as an etiological factor for WE, it remains an under-recognized disorder[5] because of its rarity, with fewer than 60 cases reported to date. In addition, a review of the literature also suggests that a high level of caution should be practiced with eating-disordered patients, pregnant patients, as well the malnourished, psychotic, or depressed patients, especially in those exhibiting catatonia, but these individuals are rarely given thiamine supplementation. Pregnancy itself puts a patient in a deficiency state for many essential nutrients; on the top of this, poor dietary habit, a strict vegetarian diet, prolonged nausea, and vomiting can increase the risk. Treatment consist of IV thiamine 100 mg for 5 days followed by 50 mg of oral thiamine if the patient will tolerate an oral dose, but several recent studies indicated that higher doses of thiamine maybe needed.[5] In the case of Ms. M, we do not know how long she had been suffering from this condition, but it should have been suspected long before it was finally diagnosed. Thus, it is the opinion of the authors that the current strategy of identifying vulnerable patients should be based on nutritional deficiency rather than any single etiology, as the consequences of prolonged WE are fairly grave but easily preventable.

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