

# Nonalcoholic Wernicke Encephalopathy: An Entity Not to Be Missed!

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## CLINICAL BACKGROUND

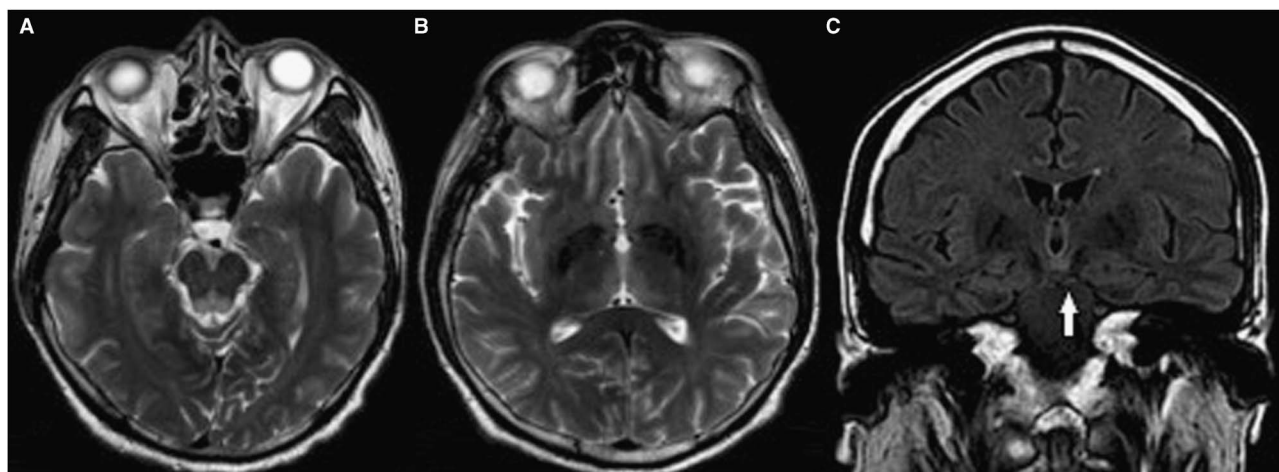
A 52-year-old man was admitted for acute abdominal pain and vomiting. Laparotomy revealed dense adhesions, likely secondary to a ruptured appendix 30 years previously; adhesiolysis was performed and a segment of ileum resected. His postoperative course was complicated by recurrent bowel obstruction and persistent vomiting. Five weeks after his initial presentation, total parenteral nutrition was commenced. One day after this, he developed diplopia and bilateral horizontal nystagmus. There was no other medical history. He did not smoke or consume alcohol. Plain computed tomography of the brain was normal. Over the next several days, he developed an unsteady gait and gradual worsening of his conscious state (Glasgow Coma Scale score, E2V2M5 = 9). Tone was normal in all limbs and tendon reflexes were symmetrically brisk with flexor plantar responses. Limb power and sensory examination could not be tested reliably because of his poor conscious state. Eight days after the onset of neurological symptoms, he was intubated for airway protection. Serum electrolytes were normal. Brain magnetic resonance imaging (MRI) (Figure 1) showed T2 hyperintensities in

the midbrain tectal plate/periaqueductal region, medial thalami, and mammillary bodies, findings that were highly suggestive of Wernicke encephalopathy (WE).<sup>1</sup>

## DISCUSSION

WE is an acute neurologic complication resulting from thiamine (vitamin B1) deficiency. Thiamine replacement was started promptly, with clinical improvement. The patient was extubated on day 2 after treatment and was able to walk with a frame on day 7. Repeat brain MRI a week later showed near-complete resolution of the signal changes. After a month of treatment, he could walk independently, with minimal difficulty on tandem gait. The nystagmus has persisted after 1 year of treatment, but with no visual impairment.

A high index of suspicion for WE is needed in the setting of poor nutrition (e.g. caused by gastrointestinal disturbances, bariatric surgery, hyperemesis gravidarum, or malignancy), regardless of whether there is a history of alcoholism.<sup>1-4</sup> WE can also be triggered by administration of total parenteral nutrition.<sup>4</sup>

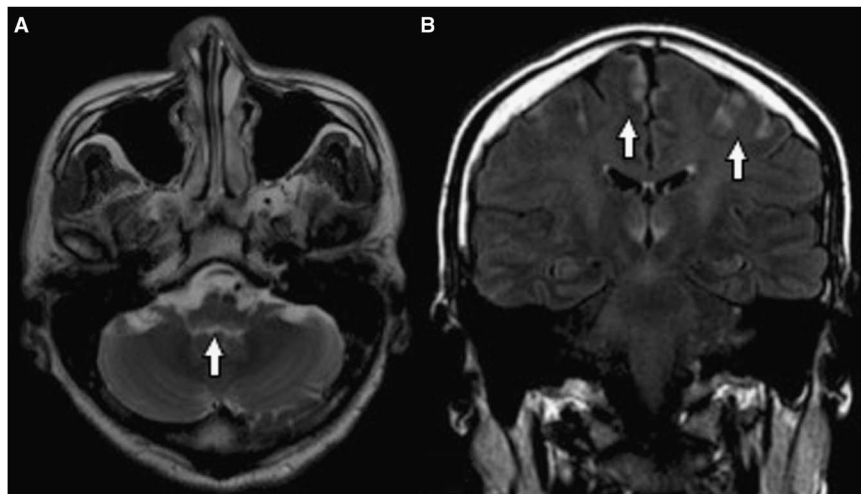


**Figure 1:** Typical MRI findings in Wernicke encephalopathy – Axial T2-weighted images showing hyperintensities in the periaqueductal area of the midbrain (A) and medial thalami (B); Coronal FLAIR image showing hyperintensities of the mammillary bodies (C), as indicated by the arrow.

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**Figure 2:** Atypical MRI findings in Wernicke encephalopathy – Axial T2-weighted image showing hyperintensities in dorsal medulla (A); Coronal FLAIR image showing cortical involvement (B).

In this case, it is likely that both the gastrointestinal disturbances and total parenteral nutrition were culprits. Often, not all features of the classic triad of ataxia, nystagmus/ophthalmoplegia, and confusion are seen, especially in nonalcoholic cases (the full triad occurring in only 33.6% of such cases versus 53.9% in alcoholic patients).<sup>2</sup> Therefore, WE is often clinically underdiagnosed.<sup>1-4</sup>

Reversible cytotoxic edema in WE causes distinctive lesions and brain MRI is very useful in aiding the clinical diagnosis.<sup>1</sup> The typical and common MRI findings of WE are symmetrical lesions in the medial thalami/periventricular region of the third ventricle (seen in 85% of cases), periaqueductal region (65% of cases), and mamillary bodies (58% of cases). The maintenance of cellular osmotic gradients in these brain regions has been postulated to be strictly related to thiamine concentration, thus rendering them vulnerable to thiamine deficiency.<sup>1</sup> Interestingly, our patient's MRI also showed involvement of the dorsal medulla and cerebral cortex (Figure 2). In cases of nonalcoholic WE, atypical MRI findings can be observed in the cerebellum, cranial nerve nuclei, red nuclei, dorsal medulla, caudate nuclei, splenium, and cerebral cortex.<sup>1,3</sup> Measurements of blood thiamine concentration or red blood cell transketolase activity are limited by a lack of specificity and technical difficulty,<sup>4</sup> and were not done in this case.

The treatment of WE is immediate administration of parenteral thiamine. One recommended initial regime, instituted in our patient, is to give 500 mg intravenously three times daily for 2 to 3 days; if a response is observed, this is followed by thiamine 250 mg intravenously or intramuscularly daily for 3 to 5 days,

or until clinical improvement ceases.<sup>4</sup> Persistent neurological deficits such as impaired memory or learning disability, gait ataxia, and nystagmus are common. Early recognition and treatment of WE is critical to reduce the risk of permanent neurological sequelae or a fatal outcome.<sup>4</sup>

#### STATEMENT OF AUTHORSHIP

The study concept was developed by AHT and SYL. Data were acquired by CCL, KR, JSYM, and AHT. The manuscript was drafted by CCL, AHT, SYL, KR, and JSYM.

#### DISCLOSURES

None of the authors have anything to disclose.

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