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Unrecognized Wernicke Encephalopathy in Pregnancy: Clinical Worsening after Surgery Highlights Diagnostic Oversight: A case report

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Abstract

Background: Wernicke encephalopathy (WE) is an acute neuropsychiatric syndrome caused by thiamine deficiency. Though often linked to alcoholism, it can result from prolonged vomiting during pregnancy (hyperemesis gravidarum).

Case Presentation: We describe the case of a 21-year-old primigravida at 16 weeks' gestation, presenting with incoercible vomiting. Initial investigations revealed hepatic cytolysis and biliary sludge. Post-surgical deterioration led to neuropsychiatric symptoms. Brain MRI revealed periaqueductal hyperintensities suggestive of WE. High-dose intravenous thiamine resulted in marked clinical improvement.

Conclusion: WE is a medical emergency that should be considered in pregnant women with persistent vomiting. Early recognition and treatment are crucial to prevent irreversible complications.

Keywords: Wernicke encephalopathy, hyperemesis gravidarum, thiamine deficiency, pregnancy, neurological emergency, Postoperative Neurological Deterioration

Introduction

Wernicke encephalopathy (WE) is a severe neurological condition resulting from a deficiency in thiamine (vitamin B1). While classically associated with chronic alcoholism, non-alcoholic causes such as hyperemesis gravidarum (HG) during pregnancy are increasingly recognized [1,2]. WE remains underdiagnosed, with many patients failing to present the complete clinical triad of confusion, ophthalmoplegia, and ataxia [3]. Delayed diagnosis can lead to permanent neurological damage or death. This case illustrates the importance of early recognition and intervention in the context of HG.

Case Presentation

A 21-year-old primigravida at 16 weeks of gestation was admitted for persistent vomiting, epigastric pain, and general deterioration. On examination, she was conscious but exhibited significant psychomotor slowing. Vital signs showed a blood pressure of 110/70 mmHg, heart rate of 115 bpm, and normal

oxygen saturation. Laboratory tests revealed hyponatremia, hypokalemia, hepatic cytolysis (ASAT 245 UI/l, ALAT 150 UI/l), suppressed TSH, and mildly elevated lipase. Abdominal ultrasound revealed biliary sludge without cholelithiasis. She was transferred for presumed biliary pancreatitis and underwent cholecystectomy.

Postoperatively, the patient's condition worsened rapidly. She developed vertigo, bilateral tinnitus, sudden vision loss, confusion, visual and auditory hallucinations, and delusional thoughts.Given her altered mental status and clinical deterioration, she was transferred to the intensive care unit (ICU) for closer monitoring. Initial suspicion was sepsis; however, infectious workup was negative, and broad-spectrum antibiotics were discontinued. By day six, brain MRI (Figure 1) demonstrated symmetrical periaqueductal T2/FLAIR hyperintensities consistent with Wernicke encephalopathy [4]. High-dose intravenous thiamine (1,500 mg/day for three days) was administered along with electrolyte repletion and antiemetics. Within a week, her visual and cognitive symptoms significantly improved. However, mild attentional deficits persisted.

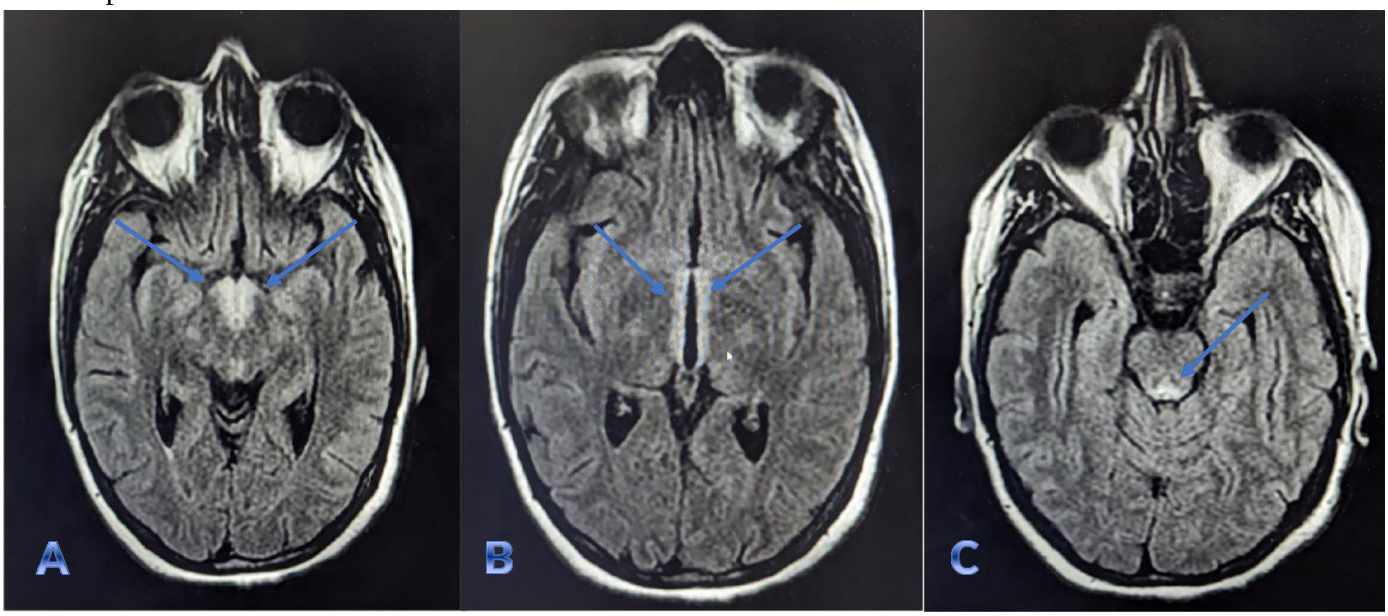

Figure 1 :Axial MRI T2 FLAIR sequences showing hyperintensities of the medial thalami (A and B) and the periaqueductal region (C).

Location: University Hospital HASSAN 2 , FEZ, Morocco

Discussion

This case underscores a rare but critical complication of hyperemesis gravidarum. Thiamine is essential for cerebral metabolism, and deficiency may lead to cytotoxic and vasogenic edema. The classic triad is seen in only 16–38% of patients, making diagnosis challenging [1,5]. In pregnant women, differential diagnoses often include psychiatric conditions, electrolyte imbalances, or obstetric complications, which can delay specific treatment.

While Wernicke encephalopathy is commonly associated with chronic alcohol consumption, it can also occur in individuals suffering from severe malnutrition. Despite its classification as a rare disease, its autopsy prevalence is estimated at 0.8% to 2.8%, which is significantly higher than the frequency of clinical diagnosis (0.04%–0.13%). In fact, 75% to 80% of cases remain undiagnosed [2].

Thiamine is found in our diet, with a daily requirement estimated at about 1.4 mg/day, which may increase with hypercaloric or carbohydrate-rich diets. Absorption occurs in the duodenum, and transport across the blood-brain barrier is active, depending on the concentration gradient. In its biologically active form—thiamine pyrophosphate—it acts as an essential coenzyme in several biochemical reactions in the brain [3]. A deficiency in thiamine causes brain lesions of varying severity, from hemorrhagic suffusions to neuronal destruction.

Importantly, hyperemesis gravidarum is a leading cause of non-alcoholic Wernicke encephalopathy, particularly in developing countries where vitamin supplementation may not be routine. The exact prevalence of WE in pregnancy is unknown due to underdiagnosis, but reported cases highlight significant maternal and fetal morbidity [2]. Thiamine stores can be depleted in just 2–3 weeks of insufficient intake, particularly during increased metabolic demand in pregnancy.
In our patient, several factors contributed to the development of Wernicke encephalopathy: intractable vomiting over two months and a surgical procedure performed without vitamin supplementation. Thiamine deficiency causes brain lesions within 2 to 3 weeks, as the body's B1 stores are depleted after approximately 18 days. The average onset of clinical signs is around four weeks [4].

In this case, 72 hours passed between surgery and symptom onset, but her vomiting had started long before.

The classical description of Wernicke encephalopathy includes a triad of confusion, oculomotor disturbances, and cerebellar ataxia. However, only 16.5% of patients present the full triad. In 37% of cases, only one sign is present, and 27.8% present two. Surprisingly, 18.6% show none of the triad signs.

Thiamine deficiency can manifest with a range of symptoms including hypothermia, hypotension, tachycardia, hallucinations, headache, fatigue, and abdominal discomfort. Other features may include dysarthria, dysphagia, hypotonia of the lower limbs, hearing loss, myoclonus, dyskinesia, dystonia, epilepsy, and psychiatric disorders such as psychosis with auditory hallucinations, paranoid delusions, or bulimia. Additionally, peripheral neuropathy is often associated, though rarely investigated [5]. In our patient, the clinical picture was marked by persistent confusion, a sudden bilateral loss of visual acuity, and deterioration of higher mental functions.

MRI remains a cornerstone in diagnosing WE when available. Periaqueductal, thalamic, and mammillary body involvement are characteristic radiologic features, though its sensitivity is 53% and specificity 93% [6].However, imaging findings may lag behind clinical suspicion, and normal MRI does not exclude the diagnosis.

In our patient, the deterioration of her neurological state prompted neuroimaging, which was considered safe for the fetus. Brain MRI confirmed the diagnosis of Wernicke encephalopathy. A normal MRI does not exclude the diagnosis. Definitive confirmation can be achieved through measurement of serum thiamine levels [7], but in our case, this was not done due to the urgency of treatment and the unavailability of the test in our facility. Additional abnormalities such as hepatic cytolysis, mild hyperlipasemia, and

suppressed TSH were observed. These are common in the context of hyperemesis gravidarum and generally do not require specific treatment [8,9].

Wernicke encephalopathy is a medical emergency. Treatment should be initiated promptly upon clinical suspicion and should not be delayed for vitamin level assays. However, therapeutic protocols remain non-standardized. A 2016 literature review covering 15 years of data proposed algorithms for both the treatment and prevention of WE. The recommended curative regimen includes intravenous thiamine 500 mg three times daily for 3 to 5 days, followed by 250 mg per day IV for an additional 3 to 5 days if improvement is noted [10].

Our patient was treated with isotonic saline rehydration, intravenous potassium supplementation, antiemetic therapy, and injectable vitamin B1 at a dose of 1,500 mg/day for three days, leading to a favorable outcome.
Prevention is equally important. Guidelines recommend prophylactic thiamine supplementation in women with persistent vomiting beyond the first trimester or with signs of malnutrition [2]. Routine assessment and early nutritional support should be part of antenatal care in patients with HG.

Conclusion
Wernicke encephalopathy, though rare, is a medical emergency in the context of hyperemesis gravidarum. Its early recognition and management with high-dose thiamine are essential for favorable outcomes. This case reinforces the importance of routine thiamine supplementation in pregnant women with prolonged vomiting to prevent severe neurological sequelae.

Conflict of Interest
The authors declare that there is no conflict of interest regarding the publication of this case report.

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References
1. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy.
2. Galvin R1, Bråthen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA; EFNS. Eur J Neurol. 2010 Dec; 17(12): 1408-18 Wernicke's Encephalopathy G. B. Young Encyclopedia of the Neurological Sciences, 2014, 753-754
3. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. Sechi G, Serra A. Lancet Neurol. 2007 May; 6(5): 442-55.
4. Schenker S, Henderson GI, Hoyumpa AM jr, Mc Candless DW. Hepatic and Wernicke's encephalopathies: current concepts of pathogenesis. Am J Clin Nutr. 1980;33(12):2719-26.
5. Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. J Neurol Neurosurg Psychiatry 1986;49(4):341–5
6. Cerase A, Rubenni E, Rufa A, Vallone I et al. CT and MRI of Wernicke's encephalopathy. Radiol med. 2011;116(2):319-33.

7. Wernicke's Encephalopathy: Role of Thiamine E. Jane Marshall Practical gastroenterology, JUNE 2009, 21-30
8. Transient hyperthyroidism and hyperemesis gravidarum: clinical aspects. Goodwin TM, Montoro M, Mestman JH. Am J Obstet Gynecol 1992; 167: 648–52.
9. Wernicke's encephalopathy in hyperemesis gravidarum: association with abnormal liver function. Rotman P, Hassin D, Mouallem M, Barkai G, Farfel Z.Isr J Med Sci 1994; 30: 225–8.
10. Boulanger AS, Paquette I, Létourneau G, Richard-Devantoy S. Thiamine et encéphalopathie de Gay-et-Wernicke: quelles règles de prescription? 2016;43(3):259–267.