

Wernicke's Encephalopathy Associated With Hyperemesis Gravidarum: A Case Report

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ARTICLE INFO	ABSTRACT
Article type: Case report	Background & aim: Wernicke's encephalopathy (WE) is a rare but serious neurologic complication of hyperemesis gravidarum. The most commonly known etiology for WE is chronic alcohol misuse. Hyperemesis gravidarum is one of the many etiologies of WE. In this paper, we report a case of HG who presented with neurological symptoms and was subsequently diagnosed with WE.
Article History: Received: 09-Jan-2024 Accepted: 14-Apr-2025	Case report: A case of WE associated with HG in a 22 years old primigravid woman, with a gestational age of 15 weeks and 4 days, is reported. The patient presented with ataxia, confusion, bilateral blurred vision, and lower extremity weakness. She had normal vital signs, and neurological examinations showed confusion and spatial, temporal, and personal disorientation. Laboratory studies showed no abnormal findings. Due to the presence of hyperthyroidism, thyroid storm was another suspected differential diagnosis; however, it was ruled out after further thyroid functioning test and brain MRI.
Key words: Wernicke's Encephalopathy Hyperemesis Gravidarum Pregnancy Thiamine Deficiency	Conclusion: The MRI findings were indicative of WE. The patient received full recovery after high dose supplementation of thiamine.

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Introduction

Wernicke's encephalopathy (WE) is a common but frequently overlooked condition resulting from vitamin B1 (thiamine) deficiency (1). Since Sheehan's first report in 1939, which identified a link between HG and WE, a systematic review by Oudman E (2019) documented 177 cases of WE associated with HG up to recent years (2, 3). This association underscores the critical need for awareness and timely diagnosis of WE in pregnant women experiencing severe nausea and vomiting to prevent significant morbidity and mortality. This case report highlights the critical importance of recognizing and promptly

treating WE in patients with HG to prevent severe maternal and fetal outcomes. Maternal complications described in the literature include persistent neurological deficits, peripheral neuropathy, Korsakoff syndrome, coma, and maternal death in severe untreated cases. Permanent cognitive impairment and incomplete neurological recovery have also been reported, particularly in delayed diagnoses (4). Fetal complications include spontaneous abortion, intrauterine growth restriction, preterm delivery, low birth weight, fetal neurological injury, and intrauterine fetal

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demise, mainly secondary to prolonged maternal malnutrition and thiamine deficiency. Chiossi et al. reported an overall pregnancy loss rate of 47.9% attributable to Wernicke's encephalopathy complicating hyperemesis gravidarum (5).

Case presentation

On December 20, a 22-year-old primigravida woman, at 15 weeks and 4 days of gestation based on first trimester sonography, presented with complaints of true vertigo, imbalance, bilateral blurred vision, and lower extremity weakness. The patient had a history of severe nausea and vomiting since the beginning of her pregnancy, managed with outpatient symptomatic therapy. Additionally, she was diagnosed with hyperthyroidism during her pregnancy and was being treated with methimazole 5 mg three times a day, along with folic acid, calcium, and vitamin D supplements. Her social and family histories were unremarkable. On physical examination, the patient's vital signs were within normal limits: blood pressure 120/80 mmHg, pulse rate 96 beats per minute, respiratory rate 18 breaths per minute, and temperature 37°C. Neurologically, she was confused and disoriented to time, place, and person. Her pupils were mid-sized and reactive, with normal eye movement and no nystagmus. Visual acuity was intact, but lower extremity strength was reduced to 3/5. Initial laboratory tests revealed the results as presented in Table 1.

Given her symptoms and history, Wernicke's encephalopathy was suspected. However, considering the patient's history of hyperthyroidism, a thyroid storm was also considered as a differential diagnosis. An endocrinology consultation concluded that symptoms were unlikely to be due to a thyroid storm in the absence of a significant goiter and other typical symptoms of Graves' disease. On December 22, the patient's free T4 was 1.5 ng/dL, total T4 was 18.8 µg/dL, free T3 was 2.7 pg/dL, and T3 was 2.9 nmol/L. Table 2 shows the trend of her thyroid-stimulating hormone (TSH) levels (Table 2).

Table 1. Initial laboratory test results

Test	Result
White Blood Cell Count	8100/µL
Neutrophils	68.7%
Hemoglobin	11.2 g/dL
Platelet Count	222,000/µL
Creatinine	0.4 mg/dL
Aspartate Aminotransferase (AST)	59 U/L
Alanine Aminotransferase (ALT)	169 U/L
Alkaline Phosphatase	152 U/L
Blood Glucose	62 mg/dL
Prothrombin Time	14 seconds
Partial Thromboplastin Time	29 seconds
INR	1.19
C-Reactive Protein (CRP)	9 mg/L
Sodium	139 mEq/L
Potassium	3.6 mEq/L

Table 2. Thyroid-stimulating hormone levels

Date	TSH Level (U/L)
July 3	1.5
August 13	0.72
November 5	0.04
December 8	<0.01
December 22	0.01

MRI of the brain revealed bilateral hyperintense lesions in the prethalamic region and midbrain, consistent with WE. Cardiology consultation and echocardiography indicated normal ventricular size and function, with an ejection fraction of 65% and no evidence of pericardial effusion. Portable ultrasound confirmed a live 15-week fetus with normal activity, a right anterolateral placenta, and normal amniotic fluid volume.

The patient was treated with 500 mg of intravenous thiamine daily for three days, leading to significant improvement in her symptoms. She was subsequently discharged with oral antiemetic medications and nutritional recommendations, with no maternal or fetal complications, and advised to attend routine follow-up visits for ongoing assessment.

Discussion

Wernicke's encephalopathy secondary to hyperemesis gravidarum is an uncommon but severe condition that poses significant diagnostic and therapeutic challenges. Despite its rarity, the potential for high morbidity and mortality rates necessitates heightened clinical

awareness. This case underscores the critical importance of early recognition and intervention to prevent severe maternal and fetal outcomes.

The patient's symptoms began early in her pregnancy and progressively worsened, leading to her hospital admission at 15 weeks and 4 days of gestation. The clinical presentation included confusion, blurred vision, and ataxia, without nystagmus or ophthalmoplegia. The clinical presentation of WE typically includes a triad of confusion, ataxia, and ophthalmoplegia. However, variability in presentation can complicate diagnosis. Studies indicate that 83.6% of WE cases present with cognitive impairment, 83.1% with ataxia, and 86.4% with ophthalmic problems, with nystagmus being the most frequent ophthalmic sign (3, 6, 7). In our patient, the absence of classic signs such as nystagmus and ophthalmoplegia did not preclude the diagnosis, emphasizing the importance of considering WE in patients with severe HG and atypical presentations.

These findings, seen in T2, FLAIR, and DWI sequences, are indicative of cytotoxic edema reversible with prompt thiamine administration (8). In our patient, MRI findings were consistent with WE, facilitating timely diagnosis and treatment.

The patient received 500 mg of intravenous thiamine daily for three days, leading to significant improvement in her symptoms. This treatment regimen aligns with current guidelines recommending high-dose thiamine until symptoms resolve (1, 9).

This case adds to the limited number of documented WE cases associated with HG, emphasizing the importance of clinical vigilance. However, the lack of long-term follow-up data limits the assessment of chronic cognitive outcomes and pregnancy success rates.

Preventive measures are paramount in managing HG to avert complications such as WE. Routine thiamine supplementation for pregnant women experiencing severe and persistent vomiting is recommended. Intravenous or intramuscular thiamine administration, particularly in cases refractory to oral supplementation, is crucial. The European Federation of Neurological Societies and the Royal College of Physicians recommend 100 mg

of thiamine for such patients, aligning with our case management (10).

Conclusion

In conclusion, this case report highlights the need for heightened clinical awareness of WE in the context of HG. Early recognition and intervention with high-dose thiamine can significantly reduce morbidity and mortality. Prophylactic thiamine supplementation should be a standard component of prenatal care for women with severe HG, as timely diagnosis and treatment of WE is critical to prevent adverse maternal and fetal outcomes.

Declarations

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Conflicts of interest

The authors declared no conflicts of interest.

Ethical considerations

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from the patient for publication of the case details.

Code of ethics

The present study was approved by the Ethics Committee of Mashhad University of Medical Sciences under the ethical code: IR.MUMS.REC.1402.045.

Use of Artificial Intelligence (AI)

Artificial intelligence tools were used only for language editing and grammatical refinement of the manuscript. The authors reviewed and approved all final content and take full responsibility for the scientific accuracy and integrity of the article.

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Authors' contribution

MSP contributed to patient management, data collection, literature review, and manuscript drafting. EH and ShA contributed to patient management and clinical interpretation. JJ supervised the study, contributed to clinical interpretation, critically revised the manuscript, and approved the final version for publication. All authors read and approved the final manuscript.

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