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Wernicke's Encephalopathy in the Context of Hyperemesis Gravidarum: A Case Report and Literature Review

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Abstract

Introduction

Wernicke's encephalopathy (WE) is a rare but potentially fatal neurological emergency caused by thiamine deficiency. It can occur during pregnancy, especially in cases of prolonged hyperemesis gravidarum.

Case Report

We report the case of a 28-year-old primigravida admitted at 16 weeks of gestation with muscle weakness and suspected polyradiculoneuropathy. Electromyography revealed a sensory-motor axonal polyneuropathy. Secondary worsening with confusion and nystagmus led to the diagnosis of Wernicke's encephalopathy confirmed by brain MRI. The patient improved following thiamine supplementation.

Discussion

This case illustrates an atypical form of WE with predominant peripheral involvement, which delayed diagnosis. Early treatment with intravenous thiamine enabled partial neurological recovery and prevented severe sequelae.

Conclusion

Any unexplained neurological symptoms in a pregnant woman with prolonged vomiting should raise suspicion for WE. Empirical thiamine supplementation is justified in the presence of clinical doubt.

Keywords: Wernicke's Encephalopathy, Pregnancy, Thiamine, Hyperemesis Gravidarum, MRI, Vitamin Deficiency

Introduction

Wernicke's encephalopathy (WE) is an acute neurological disorder caused by a deficiency in thiamine (vitamin B1), an essential cofactor in cerebral energy metabolism [1]. First described by Carl Wernicke in 1881, it is classically characterized by a triad of symptoms: Confusion, ataxia, and oculomotor abnormalities [2]. However, this complete triad is observed in only one-third of cases [3]. Pregnant women—particularly those suffering from hyperemesis gravidarum—are considered at risk due to increased metabolic demands and prolonged gastrointestinal losses [4]. We report an atypical case of WE occurring in the second trimester of pregnancy, initially misdiagnosed as acute polyradiculoneuropathy.

CaseReport

A 28-year-old primigravida with no past medical history was admitted to the emergency department at 16 weeks of gestation for progressive muscle weakness. On examination, Glasgow Coma Scale was 15, temperature 36.8 °C, and blood pressure 110/70 mmHg. History revealed persistent vomiting for three weeks, anorexia, and a 2 kg weight loss.

Neurological examination showed symmetric motor weakness (MRC score: 3/5 proximal and 2/5 distal in the lower limbs; 4/5 and 3/5 in the upper limbs), global areflexia, and muscle cramps predominantly in the calves.

Laboratory tests revealed inflammatory anemia (Hb 10.2 g/dL, CRP 42 mg/L), cytolysis (AST 352 U/L, ALT 198 U/L), hyponatremia (128 mEq/L), hypokalemia (2.63 mEq/L), and suppressed TSH with normal T3/T4 levels. Abdominal ultrasound showed isolated biliary sludge. Dorsolumbar spine MRI, fundoscopy, lumbar puncture, and viral serologies (HIV, HBV, HCV) were unremarkable. Electromyography confirmed a sensory-motor axonal polyneuropathy.

On day 3, the patient developed sudden confusion (Glasgow 13) and bilateral multidirectional nystagmus. Brain MRI revealed symmetrical T2/FLAIR hyperintensities in the thalami, mammillary bodies, and periaqueductal areas, consistent with Wernicke's encephalopathy.

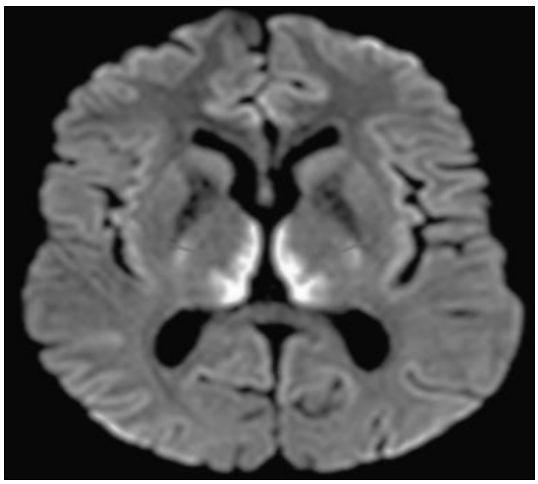
Intravenous thiamine treatment was initiated at 500 mg three times daily for 3 days, followed by 250 mg daily. Rapid improvement in consciousness and resolution of nystagmus were observed within 48 hours. By day 5, partial motor recovery was noted (MRC 4/5). At 6 months postpartum, follow-up brain MRI had normalized, although distal motor deficits persisted.

Discussion

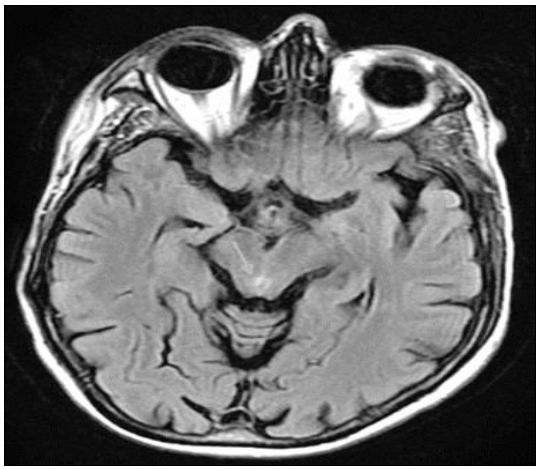
This case highlights an atypical presentation of Wernicke's encephalopathy (WE), initially misleading due to its peripheral neurological symptoms. In the context of hyperemesis gravidarum, thiamine depletion can develop rapidly as a result of prolonged gastrointestinal losses combined with increased metabolic demands during pregnancy [1, 4].

Peripheral-dominant clinical forms mimicking Guillain-Barré syndrome are well documented in the literature but remain underdiagnosed [5, 6]. Approximately 25–30% of non-alcoholic WE cases initially present with isolated neuromuscular symptoms. This atypical presentation significantly contributes to diagnostic delays, particularly in non-alcoholic and pregnant patients [3, 5, 7, 8].

Magnetic resonance imaging (MRI) is the gold standard diagnostic tool. It typically reveals bilateral symmetrical hyperintensities in the thalami, mammillary bodies, periaqueductal regions, and the floor of the third ventricle,areas rich in mitochondria and particularly sensitive to thiamine deficiency [9, 10].



Bilateral thalamic hyperintensities



Periacqueductal Hyperintensity

Plasma thiamine measurement, although useful, is not widely available in routine clinical practice, especially in resource-limited settings. Clinical judgment, supported by patient history, imaging findings, and response to treatment, remains the most reliable practical approach.

Treatment requires urgent intravenous thiamine supplementation. It must precede any glucose administration to avoid paradoxical worsening of cerebral lesions [11]. Current recommendations suggest administering 500 mg IV three times daily for 3–5 days, followed by 250 mg daily until clinical improvement is achieved [4, 11, 12].

Neurological prognosis depends primarily on the promptness of treatment. Delayed intervention increases the risk of severe cognitive sequelae or death [3, 8, 13]. It is crucial to raise awareness among clinicians, particularly in obstetrics, neurology, and emergency medicine, about this condition.

Any unexplained neurological symptoms in a pregnant woman experiencing severe vomiting for more than 7 to 10 days should raise suspicion for WE, even in the absence of the full clinical triad. Early thiamine supplementation is a simple, safe, and potentially life-saving intervention.

Conclusion

This case underscores the importance of recognizing atypical presentations of Wernicke's encephalopathy, particularly in obstetric contexts. Prolonged hyperemesis gravidarum should prompt early suspicion of severe thiamine deficiency and its neurological complications. Management must be rapid, empirically initiated if necessary, and integrated within a rigorous clinical approach to prevent potentially irreversible sequelae.

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