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A Case Report of Wernicke's Encephalopathy During a Pregnancy Complicated by Preeclampsia: A Diagnosis Not to Be Overlooked

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Abstract

Wernicke's encephalopathy (WE) is a potentially life-threatening neurological complication of thiamine deficiency. Although classically associated with chronic alcohol use, it may occur during pregnancy, particularly in the setting of prolonged vomiting and hyperemesis gravidarum. Its clinical presentation may overlap with neurological complications of preeclampsia, making diagnosis challenging.

We report the case of a 19-year-old primigravida at 24 weeks of gestation who was admitted to the intensive care unit with prolonged vomiting, altered consciousness, cerebellar ataxia, and oculomotor abnormalities. She presented with severe hypertension, proteinuria, tachycardia, and significant metabolic disturbances, in the context of severe preeclampsia. Initial brain computed tomography was unremarkable. Due to persistent neurological manifestations, brain magnetic resonance imaging was performed and revealed symmetrical lesions involving the mammillary bodies, periaqueductal region, thalami, and periventricular areas, consistent with WE. Thiamine was promptly administered, along with correction of metabolic abnormalities and nutritional support. Neurological status improved progressively, with complete clinical recovery and normalization of laboratory abnormalities.

WE should be considered in pregnant women presenting with neurological symptoms and prolonged vomiting, even when preeclampsia is present. The overlap with neurological complications of preeclampsia may delay diagnosis. Early recognition and prompt administration of thiamine are essential to prevent irreversible neurological sequelae.

Categories: Neurology, Radiology, Emergency Medicine
Keywords: hyperemesis gravidarum, magnetic resonance imaging, preeclampsia, pregnancy, thiamine deficiency, wernicke's encephalopathy

Introduction

Wernicke's encephalopathy (WE) is a potentially serious neurological emergency resulting from a deficiency of thiamine (vitamin B1), which is essential for cerebral energy metabolism [1]. Although traditionally associated with chronic alcoholism, it can also occur in cases of malnutrition, particularly during prolonged vomiting or hyperemesis gravidarum, which lead to rapid depletion of thiamine reserves [2,3].

The classic presentation combines impaired consciousness, oculomotor abnormalities, and cerebellar ataxia, but this triad is not always present. A systematic review of 82 cases of WE associated with hyperemesis gravidarum reported oculomotor signs in 93.9% of cases, ataxia in 74.4%, and impaired consciousness in 76.8% [3]. Brain magnetic resonance imaging (MRI) can aid in diagnosis by identifying typical lesions in the medial thalamus, mammillary bodies, and periaqueductal region, although normal imaging findings do not rule out the diagnosis [1,4].

In pregnant women, the combination of hypertension and neurological symptoms poses a significant diagnostic challenge, particularly due to the overlap with the neurological complications of preeclampsia and eclampsia. However, the presence of prolonged vomiting and malnutrition should prompt investigation for thiamine deficiency and lead to early treatment.

We report a case of WE in a pregnant woman at 24 weeks' gestation, complicated by preeclampsia, highlighting the diagnostic challenges and the importance of early, multidisciplinary management.

Case Presentation

A 19-year-old patient, G1P0, 24 weeks' gestation, with no reported medical history, was admitted to the intensive care unit due to altered neurological status in the context of prolonged vomiting accompanied by

epigastric pain that had been present for several weeks. Upon admission, the patient was confused and agitated, with a Glasgow Coma Scale score of 12/15, along with impaired motor coordination and cerebellar ataxia on neurological examination. Hemodynamically, the patient presented with hypertension at 161/95 mmHg, tachycardia at 126 beats per minute, and a respiratory rate of 32 breaths per minute. Urinalysis revealed 3+ proteinuria.

Laboratory investigations revealed several abnormalities, including electrolyte and renal disturbances, hepatic cytolysis, and abnormalities in coagulation and protein excretion (Table *1*).

Parameter	Patient value	Reference range
Hemoglobin	11.3 g/dL	11-14 g/dL
Platelets	182000/µL	150000-400000/µL
PT	55%	70-100%
AST	322 U/L	10-40 U/L
ALT	287 U/L	7-56 U/L
Potassium	3 mmol/L	3.5-5 mmol/L
Sodium	132 mmol/L	135-145 mmol/L
Calcium	87 mg/L	85-105 mg/L
Serum creatinine	11.31 mg/L	0.6-1.2 mg/L
P/C ratio	120 mg/mmol	<30 mg/mmol
Urinary protein	900 mg/24 h	<150 mg/24 h

TABLE 1: Initial hematologic and biochemical findings with their normal reference ranges

AST: aspartate aminotransferase; ALT: alanine aminotransferase; PT: prothrombin time; P/C ratio: protein-to-creatinine ratio

The unfavorable evolution of the neurological condition, despite initial therapeutic measures, led to an emergency termination of the pregnancy by cesarean section. The initial neurological assessment, including a brain computed tomography (CT) scan with contrast, revealed no abnormalities that could explain the symptoms. However, the persistence of neurological symptoms prompted further investigation. A brain MRI then showed periventricular signal abnormalities in the third ventricle (V3), periaqueductal region, pulvinar, mammillary bodies, and two spinal cords, symmetrical on T1-weighted imaging and hyperintense on 12/FLAIR, and a slight diffusion-weighted hyperintense signal that did not change after contrast injection, initially suggesting WE (Figure *1*).

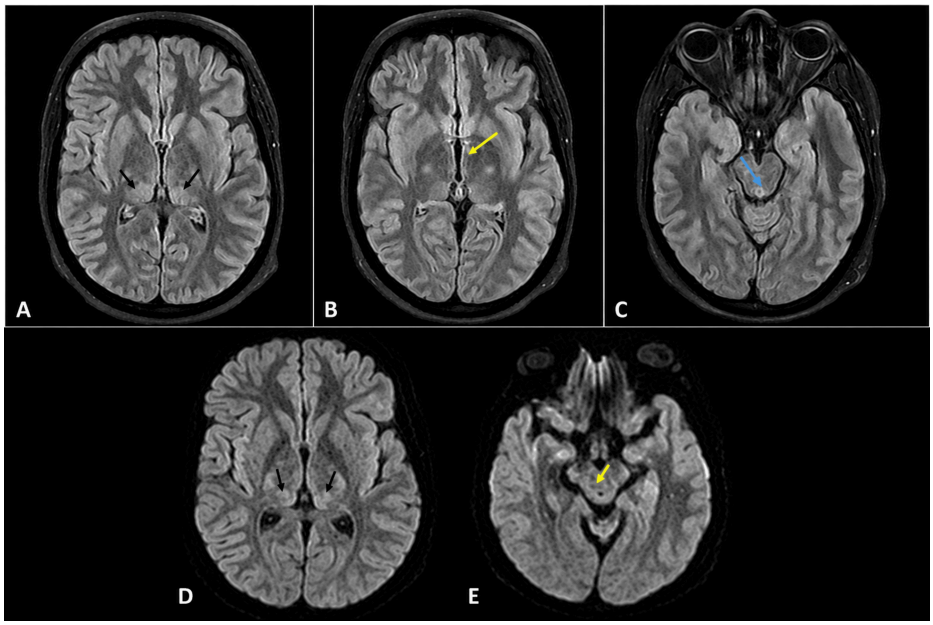


FIGURE 1: MR images in axial FLAIR (A-C) showing bilateral hyperintensity in the thalamic pulvinar (black arrows), around the third ventricle (yellow arrow), and in the periaqueductal region (blue arrow) and axial diffusion-weighted (D-E) showing subtle diffusion hyperintensity in the thalamic pulvinar (black arrows) and in the periaqueductal region (yellow arrow)

MR: magnetic resonance; FLAIR: fluid-attenuated inversion recovery

Urgent thiamine supplementation was initiated at a dose of 200-500 mg three times daily for 3-5 days, followed by 250-1000 mg daily orally. The treatment also included correction of hydro-electrolytic and metabolic disorders, nutritional support, and close neurological monitoring.

Neurological progress was favorable after the administration of thiamine, with a gradual improvement in the patient's level of consciousness and neurological symptoms, including normalization of laboratory test results. The patient was discharged five days later.

A notable feature of this case is the onset of WE secondary to hyperemesis gravidarum, associated with severe preeclampsia.

Discussion

WE is a neurological emergency secondary to thiamine (vitamin B1) deficiency. Although classically associated with chronic alcoholism, with a prevalence of approximately 12.5% [5], it can occur in numerous non-alcoholic situations that cause thiamine depletion, including prolonged vomiting, malnutrition, and various other conditions [1,3].

In pregnant women, it is classically associated with the persistent vomiting of hyperemesis gravidarum. The link between hyperemesis gravidarum and WE was reported as early as 1914 [6]. Hyperemesis gravidarum is a preventable risk factor for non-alcoholic WE. Due to the increased need for thiamine during pregnancy, symptoms can appear as early as the sixth week of gestation and progress rapidly [3]. The clinical presentation is variable, and the classic triad of altered consciousness, oculomotor abnormalities, and ataxia is not always present; other manifestations may occur, including hypothermia, hypotension, and coma [7]. Thiamine deficiency can become clinically apparent in any situation of malnutrition that depletes the body's reserves, which are generally sufficient for about 2-3 weeks. Alcoholism, bariatric surgery, and hyperemesis gravidarum are among the conditions that can lead to this deficiency [1].

The coexistence of preeclampsia presents an additional diagnostic challenge. In a hypertensive pregnant woman with neurological manifestations, eclampsia or posterior reversible encephalopathy syndrome (PRES) should be investigated first. However, PRES and WE can share common clinical features. Therefore, persistent nystagmus and ataxia, particularly in the context of vomiting and malnutrition, should prompt a search for an associated nutritional cause [8,9].

Brain MRI plays an important role in the early diagnosis of WE, with a sensitivity of approximately 92-93% and a specificity close to 100% [10]. It can show characteristic abnormalities in the mammillary bodies, thalamus, periaqueductal region, and periventricular regions [11]. Conversely, brain CT scans may remain normal, as in our case. However, the absence of radiological abnormalities should not delay treatment when the clinical context is highly suggestive.

Management is based on the immediate intravenous administration of thiamine as soon as the diagnosis is suspected, without waiting for laboratory confirmation. The European Federation of Neurological Societies (EFNS) guidelines recommend 200 mg of thiamine IV three times a day in cases of suspected or confirmed WE, administered before carbohydrate intake [12]. Early treatment largely determines the prognosis and helps limit the risk of irreversible neurological sequelae.

The outcome can range from complete recovery to motor sequelae, Korsakoff's syndrome, coma, or death. The mortality rate reported in the article is 20-30% [13].

This observation thus underscores the importance of considering WE in the differential diagnosis of any neurological manifestation in a pregnant woman presenting with prolonged vomiting, even in the presence of preeclampsia. Early awareness of this complication would allow for the rapid initiation of thiamine supplementation and prevent potentially serious sequelae.

Conclusions

WE is a rare but potentially serious complication of pregnancy, particularly in cases of prolonged vomiting and malnutrition. In a pregnant woman presenting with neurological signs, the coexistence of preeclampsia can be a diagnostic pitfall and should not lead to overlooking a thiamine deficiency. The diagnosis is primarily clinical, supported by MRI, and thiamine treatment should be initiated early, without waiting for laboratory confirmation. Prompt recognition helps prevent potentially irreversible neurological sequelae.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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