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Case Report

Seizures in the third trimester of pregnancy- A rare case of Wernicke's encephalopathy mimicking Eclampsia ☆

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ABSTRACT

Unless otherwise demonstrated, seizures during pregnancy that happen after 20 weeks of gestation are typically caused by eclampsia. In our clinical case we report the diagnostic challenge of Wernicke's encephalopathy occurring at 34 weeks and mimicking an eclamptic attack. This is about a 30-year-old female patient who suffers from hyperemesis gravidarum during her pregnancy. she has no known history of epilepsy .The patient was brought to the emergency room by medical transport at 34 weeks of gestation due to a seizure at home. The initial diagnosis was eclampsia. Emergency high-level extraction under general anesthesia was decided for maternal rescue. However, after extubation, the patient's condition deteriorated, with the onset of a confusion syndrome characterized by agitation, temporal-spatial disorientation, attention and vigilance disorders, and challenging communication. Angio MRI was performed, revealing signs consistent with Wernicke's encephalitis.

Introduction

Wernicke's encephalopathy (WE) is a neuropsychiatric emergency caused by thiamine (vitamin B1) deficiency, secondary to various factors. It is often underdiagnosed, with a clinical prevalence of 0.04% to 0.13%, compared to 0.8% and 2.8% in pathology [1]. The symptoms may suggest other conditions such as cerebral venous thrombosis, stroke, or another metabolic disorder [2], but MRI is essential for diagnosis [2]. Brain MRI is of paramount importance in WE diagnosis [3]. The absence or delay in starting treatment influences the prognosis [4].

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Case presentation

This is about a 30-year-old female patient, followed for depressive symptoms, Gravida 2, Para 1, Abortion 0.

Her first pregnancy in 2016 was uncomplicated. The patient was brought to the emergency room by medical transport at 34 weeks of gestation due to a seizure at home. The family reported that she had been experiencing headaches and vomiting for an unspecified period. During the examination, she exhibited flexion of both upper limbs with eyeball rolling and urinary incontinence.

Clinical examination revealed blood pressure (BP) of 14/9, proteinuria on the test strip with two crosses. Laboratory results showed anemia at 7.9 g/dL, thrombocytopenia at 84.10^3/mm3, uric acid level at 834 μmol/L, and creatinine level at 174 μmol/L.

Obstetric ultrasound indicated small fetal weight for gestational age: uterine height at 29 cm and biometry corresponding to 30 weeks of gestation.

The initial diagnosis was eclampsia based on the absence of epilepsy history, no previous convulsions, a seemingly normal third-trimester pregnancy, a clinical history of headache and vomiting, admission BP of 14/9, Dipstick testing for protein was two-cross (++) positive, and ultrasound showing intrauterine growth restriction.

The decision was to carry out patient conditioning, including two peripheral intravenous lines, fluid administration, and a urinary catheter. The patient did not receive any antihypertensive treatment or magnesium sulfate due to stable blood pressure and normal osteotendinous reflexes.

Since the initially considered diagnosis was an eclamptic seizure, an emergency high-level extraction under general anesthesia was decided for maternal-fetal rescue.

Postpartum monitoring revealed normal blood pressure and negative 24-hour proteinuria. However, after extubation, the patient's condition deteriorated, with the onset of a confusion syndrome characterized by agitation, temporal-spatial disorientation, attention and vigilance disorders, and challenging communication.

Further questioning with the family revealed malnutrition and hyperemesis gravidarum throughout the pregnancy. Neurological examination did not show signs of localization. The biochemical profile indicated ionic imbalances: hypokalemia at 2.4 mmol/L, hypocalcemia at 1.8 mmol/L, and hypermagnesemia at 1.65 mmol/L, a serum sodium level of 136 mmol/l.

Cerebral angioscanner showed no anomalies. Due to the lack of improvement in neurological signs and a normal angioscanner, an Angio MRI was performed, revealing signs consistent with Wernicke's encephalitis. Figs. 1 and 2 Given the profound alteration of consciousness with a Glasgow score of 11, the patient was intubated, ventilated, and treated with thiamine 300 mg * 3/day + hydrocortisone hemisuccinate 50 mg * 4/day.

The evolution occurred towards a progressive improvement of consciousness disorders and motor deficit in the lower limbs. She also presented hydroelectric disturbances in the form of hypokalemia, corrected by supplementation. The total duration of thiamine treatment was 10 days. The patient was discharged after 10 days of hospitalization, and the attending physicians prescribed physical rehabilitation following her discharge. Upon discharge, she retained a predominant tetraparesis in the lower limbs with aphasia.

The patient was reviewed at the outpatient clinic 6 months later; we noted a complete disappearance of aphasia and tetraparesis of the lower limbs.

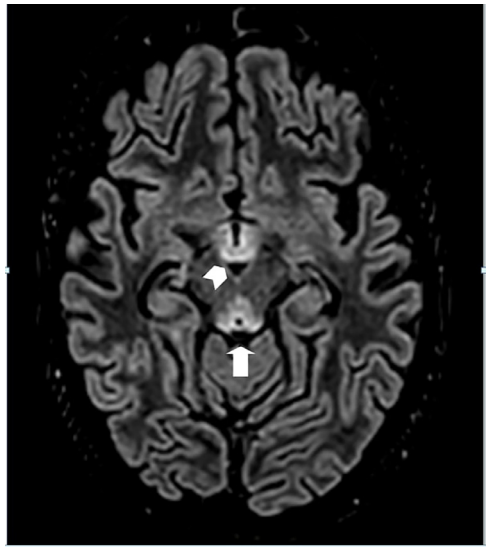

Fig. 1 – Brain MRI in axial FLAIR section showing bilateral symmetrical hyperintensities involving the mammillary bodies (arrowhead) and the periaqueductal gray matter (arrow).

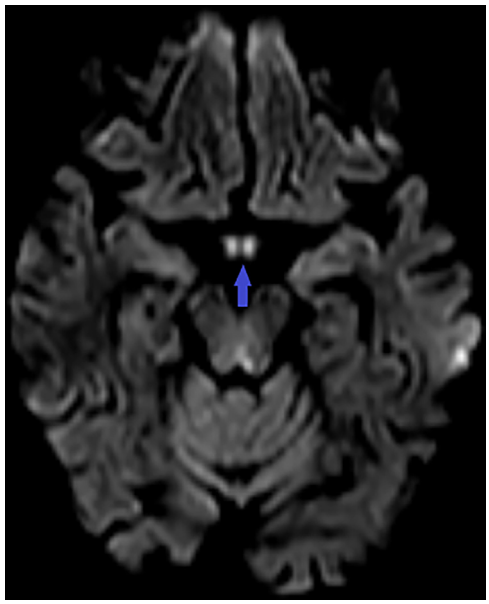

Fig. 2 – Brain MRI in axial diffusion section showing bilateral diffusion hyperintensity of the mammillary bodies (arrow).

Discussion

In our clinical case we report the diagnostic challenge of Wernicke's encephalopathy occurring at 34 weeks and mimicking an eclamptic attack.

Unless otherwise demonstrated, seizures during pregnancy that happen after 20 weeks of gestation are typically caused by eclampsia. Epilepsy and, less frequently, any central nervous system condition are additional factors. Convulsions in pregnancy may lead to other differential diagnoses when combined with unusual clinical characteristics [5].

The differential diagnoses of an eclamptic seizure are Cerebral vein sinus thrombosis, Reversible cerebral vasoconstriction syndrome, Dural puncture and metabolic disorders which are hyponatremia or hypoglycemia or hypocalcemia [5].

A thiamine (vitamin B1) deficit causes Wernicke's encephalopathy, a neurological emergency that involves the hippocampal-mammillary-thalamic network [2]. It is an uncommon but dangerous central nervous system illness [1] that has a 30% or higher death rate [2]. The syndrome was first described by Wernicke in 1881, presenting the classical triad of ataxia, ophthalmoplegia, and confusion [2]. It usually happens following a gastrectomy, in the setting of extreme malnourishment, or in the context of chronic alcoholism [1].

Prolonged hyperemesis gravidarum, can become extremely complicated. This is our patient's situation.

Hyperemesis gravidarum (HG) is a severe form of pregnancy-related nausea and vomiting, affecting up to 3% of all pregnancies. It often leads to weight loss, dehydration, and electrolyte imbalance, becoming the most common reason for hospitalization in the first half of pregnancy [6]. Nausea and vomiting are most common in the first trimester, but in HG patients, persistent vomiting extends beyond normal morning sickness and often continues into advanced stages of pregnancy and even childbirth [7]. According to a systematic review by Oudam et al., the occurrence of gravidic vomiting peaks around 10 weeks of gestation, while the peak for Wernicke's encephalopathy complicating HG occurs later, between 15 and 20 weeks of gestation (average 17 weeks). This percentage is almost negligible before 10 weeks and after 35 weeks of gestation [7].

Parenteral refeeding without vitamin supplementation may worsen the lesions. Thiamine plays a role in alcohol and glucose metabolism, acting as a coenzyme for three essential enzymes in carbohydrate metabolism, including transcetolase (involved in DNA synthesis), alpha-ketoglutarate dehydrogenase (which plays a role in neuronal excitotoxicity), and the pyruvate dehydrogenase complex. Deficiency in any of these enzymes due to thiamine deficiency can result in osmotic imbalance between extracellular and intracellular environments, leading to diffusion sequence abnormalities. Supplementation with glucose without vitamins in the context of vomiting can also worsen the situation. The periventricular regions' metabolism is particularly dependent on thiamine, explaining the prevalence of lesions in this area [8].

Thiamine is a water-soluble vitamin, with daily requirements of 1.5 mg usually obtained through diet. The body's store is approximately 25 to 30 mg, but during pregnancy, needs can reach 5 mg/day, depleting thiamine reserves rapidly in the case of hyperemesis gravidarum.

The presentation of WE with seizure episodes is rare and is generally attributed to prolonged hyperemesis gravidarum and its association with hypokalemia and hypomagnesemia. Diagnosis is clinically oriented and can be challenging. The classical triad of ophthalmoplegia, confusional state, and ataxia is observed in only 10% of cases [9].

Magnetic resonance imaging (MRI) is the reference examination for WE diagnosis [10]. It confirms the diagnosis by showing hypersignals in FLAIR, T2, and diffusion sequences in the thalamus, mammillary bodies, and periaqueductal region bilaterally and symmetrically [11]. These anomalies indicate a disruption of the blood-brain barrier. Although its sensitivity is only 53%, its specificity is 93% [12]. The term FLAIR stands for "Fluid Attenuated Inversion Recovery," a technique effective in examining cerebral tissue irregularities. It's essential to note that the typical MRI lesion pattern is observed in only 58% of patients [11]. The absence of intensity alterations in the MRI signal does not exclude the WE diagnosis. Gadolinium administration can be a useful tool to identify WE cases with a negative MRI.

The recommended thiamine dose is 500 mg intravenously three times a day for 3 to 5 days. If there's improvement after the initial treatment, the dosage can be reduced to 250 mg intravenously per day, to be continued for at least an additional 3 to 5 days [13]. The duration of treatment varies; some continue supplementation until the end of pregnancy, while others stop when vomiting ceases. Delayed initiation of treatment can lead to irreversible neurological sequelae, such as Korsakoff syndrome, characterized by cognitive disorders, anterograde amnesia, temporal-spatial disorientation, coma, or even death [7].

Conclusion

Hyperemesis gravidarum is a serious condition that can lead to neurological sequelae or even the death of the patient. Vitamin B1 supplementation is recommended as soon as vomiting occurs in pregnant women. Strict monitoring of the hydroelectrolytic balance is advised to detect associated ionic imbalances that may exacerbate symptoms.

Patient consent

The authors declare that they have obtained signed consent from the patient.

CRediT authorship contribution statement

Badra Bannour: Conceptualization, Writing – original draft. **Maroi Baazaoui:** Conceptualization, Supervision, Writing – original draft, Writing – review & editing. **Maroi Bannour:** Conceptualization, Writing – review & editing. **Imen Bannour:** Conceptualization, Supervision, Writing – review & editing.

Hiba Abdellaoui: Conceptualization, Writing – original draft. **Khaled Ben Jazia:** Conceptualization, Supervision, Writing – review & editing.

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