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WERNICKE'S ENCEPHALOPATHY IN A PREGNANT FEMALE WITH HYPEREMESIS GRAVIDARUM

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ABSTRACT Wernicke's encephalopathy (WE) is an acute neurological condition caused by a deficiency of thiamine (vitamin B1). Although it is commonly associated with chronic alcoholism, WE can also occur in non-alcoholic situations, such as hyperemesis gravidarum (HG), which is characterized by severe nausea and vomiting during pregnancy. A 24-year-old first-time pregnant woman, at 12 weeks of gestation, experienced continuous vomiting for 15 days, resulting in dehydration and considerable weight loss. She later exhibited neurological symptoms, including confusion, ataxia, and visual impairments. A neurological assessment revealed dysarthria, bilateral lateral rectus palsy, and truncal ataxia. Brain magnetic resonance imaging (MRI) displayed symmetrical hyperintensities in the medial thalami, periaqueductal gray matter, and mammillary bodies, indicative of WE. The immediate administration of intravenous thiamine led to marked clinical improvement. This case highlights the necessity of considering WE in pregnant individuals who present with prolonged vomiting and neurological symptoms. Timely identification and intervention are essential to avert irreversible neurological harm. WE are a rare yet significant complication of HG. Healthcare providers should maintain a high level of suspicion to facilitate prompt diagnosis and treatment, ultimately enhancing outcomes for both mother and fetus.

KEYWORDS :

INTRODUCTION

Wernicke's encephalopathy (WE) is a serious and potentially fatal neurological condition caused by a deficiency in thiamine (vitamin B1). This condition was first recognized by Carl Wernicke in 1881 and is typically characterized by a triad of symptoms: mental confusion, ophthalmoplegia (paralysis of the eye muscles), and ataxia (loss of coordination). However, this classic triad is observed in only a minority of patients, making the diagnostic process challenging in many cases (1).

Although it is commonly linked to chronic alcoholism, WE can also occur in various non-alcoholic situations that result in malnutrition or hinder thiamine absorption. These situations include gastrointestinal surgeries, extended periods of parenteral nutrition without sufficient supplementation, and particularly hyperemesis gravidarum (HG) (2).

Hyperemesis gravidarum affects around 0.5% to 3% of pregnancies and is marked by severe and persistent nausea and vomiting, which can lead to considerable weight loss, dehydration, and imbalances in electrolytes. This condition can quickly deplete the mother's thiamine reserves due to the heightened nutritional requirements during pregnancy, thereby elevating the risk of developing WE (3).

The classic symptoms of WE consist of mental confusion, ophthalmoplegia, and ataxia; however, these signs do not always manifest, complicating early diagnosis. In pregnant women, the similarity of WE symptoms to common pregnancy-related issues can further hinder timely identification. A delay in diagnosing and treating WE in pregnant individuals has been linked to serious complications for both the mother and fetus, including lasting neurological impairments, fetal loss, and maternal death (4).

This case report emphasizes the vital need for healthcare providers to maintain a high level of suspicion for WE in pregnant patients who exhibit prolonged vomiting and neurological symptoms, even if there is no history of alcohol use. It also stresses the importance of immediate thiamine supplementation to avert irreversible consequences.

CASE PRESENTATION

A 24-year-old primigravida, at 12 weeks of gestation, was admitted due to ongoing, severe vomiting that had persisted multiple times a day for the last 15 days. This condition resulted in inadequate oral intake, dehydration, and a weight loss of approximately 5 kilograms. Two days before her admission, she began experiencing slurred speech, blurred vision, an unsteady gait, and confusion. There was no reported history of fever, seizures, head injury, or substance use.

Upon examination, the patient exhibited disorientation regarding time and place, with a Glasgow Coma Scale (GCS) score of 13 out of 15. Neurological evaluation indicated dysarthria, bilateral lateral rectus palsy, multidirectional nystagmus, truncal ataxia, and generalized muscle weakness. Deep tendon reflexes were reduced, and bilateral plantar responses were absent. The sensory examination yielded normal results Figure 1.

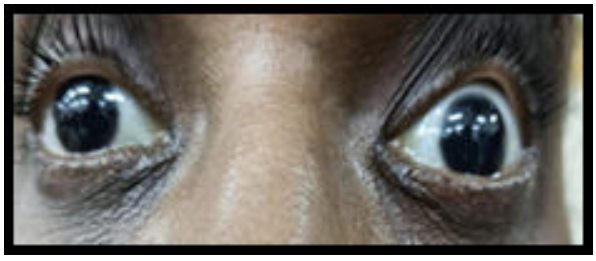

Fig1: Bilateral lateral rectus palsy in a patient with Wernicke's encephalopathy secondary to hyperemesis gravidarum, showing restricted outward eye movement.

Laboratory tests revealed normocytic normochromic anemia with a hemoglobin level of 10.2 g/dL, leukocytosis with a total leukocyte count of 14,670/mm³, and an elevated erythrocyte sedimentation rate (ESR) of 90 mm/hr. Both renal and liver function tests were normal. An obstetric ultrasound confirmed the presence of a single live intrauterine fetus, consistent with the gestational age.

Magnetic resonance imaging (MRI) of the brain showed symmetrical hyperintense signals on T2-weighted and FLAIR images in the bilateral medial thalami, periaqueductal gray matter, and mammillary bodies, which are indicative of Wernicke's encephalopathy (WE) Figure 2.

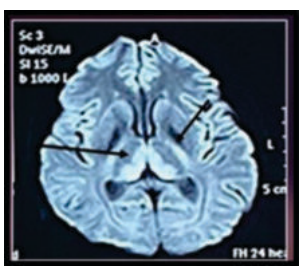

Fig 2: MRI brain (DWI sequence) showing symmetrical

hyperintensities (arrows) in the bilateral medial thalami and mammillary bodies-characteristic findings of Wernicke's encephalopathy.

The diagnosis of Wernicke's encephalopathy secondary to hyperemesis gravidarum was established. The patient was immediately started on high-dose intravenous thiamine at 500 mg three times daily, along with supportive care that included antiemetics, intravenous fluids, and electrolyte management. Over the following days, her neurological symptoms showed significant improvement. By the 18th day of hospitalization, she was alert, oriented, and able to walk independently. She was discharged with a prescription for oral thiamine supplementation and advised to maintain regular antenatal follow-up.

DISCUSSION

Wernicke's encephalopathy (WE) is a rare yet severe neurological condition resulting from a deficiency of thiamine (vitamin B1). While it has traditionally been linked to chronic alcoholism, recent studies indicate that non-alcoholic factors, particularly hyperemesis gravidarum (HG) during pregnancy, are increasingly recognized as significant contributors to the risk of developing WE. HG is characterized by persistent and severe vomiting in pregnant individuals, which can result in nutritional deficiencies, electrolyte imbalances, and a depletion of thiamine reserves, potentially leading to WE if not addressed (3).

In our case, the patient exhibited classic symptoms of WE, including altered mental status, ophthalmoplegia, ataxia, and nystagmus, aligning with the classical triad described by Harper et al. (5). However, it is important to note that this triad is only observed in a minority of cases in clinical settings, as highlighted by Sechi and Serra, who reported that fewer than one-third of patients display all three classical symptoms concurrently (2).

A similar case was reported by Chiossi et al., involving a pregnant woman with WE induced by HG, who presented with ophthalmoplegia, gait disturbances, and confusion. The authors underscored the critical need for early identification and timely thiamine supplementation to avert lasting neurological damage. This aligns with our findings, where the prompt administration of intravenous thiamine resulted in marked clinical improvement (3).

Radiologically, MRI is regarded as the definitive imaging technique for diagnosing Wernicke's Encephalopathy (WE). In our patient, MRI results demonstrated symmetrical hyperintensities in the medial thalamus, periaqueductal area, and mammillary bodies characteristic findings that have also been noted in prior research by Zuccoli et al., which highlighted MRI's high specificity in diagnosing WE, particularly in non-alcoholic individuals (6).

Additionally, Oudman et al. performed a systematic review on WE in hyperemesis gravidarum (HG) patients, noting that the heightened metabolic demands during pregnancy can lead to a more rapid onset of thiamine deficiency in pregnant women compared to alcoholics, sometimes occurring within just 2-3 weeks of ongoing vomiting. This finding corroborates the swift emergence of symptoms observed in our patient after only a few weeks of hyperemesis (4).

Another critical point raised in earlier studies is the potential for progression to Korsakoff's syndrome if WE are not diagnosed and treated promptly. Thomson et al. underscored that delayed intervention could result in lasting cognitive deficits, amnesia, and considerable morbidity. Fortunately, our patient experienced a complete recovery following early treatment, with no lasting neurological impairments (7).

CONCLUSION

Wernicke's encephalopathy, while uncommon, represents a serious complication of hyperemesis gravidarum that can result in considerable morbidity if not swiftly diagnosed and managed. This case highlights the critical need for early detection and the administration of thiamine in pregnant individuals suffering from severe and extended vomiting. Increased clinical vigilance can avert negative consequences and promote improved health outcomes for both mother and fetus.

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