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AN INTERESTING CASE OF WERNICKE'S ENCEPHALOPATHY SECONDARY TO HYPEREMESIS GRAVIDARUM

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Abstract

Background

Wernicke's encephalopathy (WE) is a rare but serious neurological disorder caused by thiamine deficiency. It is often underdiagnosed in pregnant women due to vague and overlapping symptoms. Delayed recognition can lead to severe neurological impairment and progression to Korsakoff's syndrome.

Case Presentation

A 26-year-old primigravida at four months of gestation presented with a two-day history of progressively worsening altered sensorium. She had persistent vomiting throughout the first trimester, which worsened in the second trimester (5–7 episodes per day for one week) and low-grade fever for two days. Additionally, she had small maculopapular lesions with vesicles on her back and anterior chest wall for two months. On examination, she was hemodynamically stable with a Glasgow Coma Scale (GCS) score of E2 V2 M4. Fundus examination showed bilateral papilledema with peri-papillary haemorrhages, which led to the suspicion of a space-occupying brain lesion. Meningitis was also considered as a provisional diagnosis.

Laboratory investigations, including complete blood count, renal function, and arterial blood gas analysis, were normal, but liver function tests showed elevated liver enzymes. Viral markers were negative. MRI brain revealed bilateral T2 and DWI hyperintensities in the dorsomedial and pulvinar nuclei of the thalami. Cerebrospinal fluid analysis was unremarkable. A clinical diagnosis of Wernicke's encephalopathy was made, and the patient received intravenous thiamine, leading to significant improvement in GCS within two days, confirming the diagnosis.

Discussion

Hyperemesis gravidarum affects 0.5–2% of pregnancies and can cause severe thiamine deficiency. The classic WE triad—confusion, ataxia, and ophthalmoplegia—may be absent, making diagnosis difficult. A high index of suspicion and early thiamine replacement are crucial to prevent permanent neurological damage.

Conclusion

Early recognition of WE in hyperemesis gravidarum is vital. Prompt intravenous thiamine administration leads to significant recovery, preventing irreversible complications.

INTRODUCTION

Wernicke's encephalopathy (WE) is a frequently overlooked condition resulting from a deficiency of thiamine. Wernicke's encephalopathy that results as a consequence of hyperemesis in pregnant women often poses a challenge and diagnostic dilemma to health care providers, given the vague and wide range of symptoms exhibited by the patient. Many of the early neurological symptoms such as fatigue, confusion, and mood changes, can be mistaken for normal pregnancy-related symptoms, leading to delayed diagnosis

Case Report

A 26-year-old primigravida at 14 weeks + 5 days of gestation presented with a two-day history of progressively worsening altered sensorium. She had persistent vomiting throughout the first trimester, which worsened in the second trimester (5–7 episodes per day for one week). Low grade fever was present for two days preceding admission. A rash, described as small maculopapular lesions with vesicles, had been present on her back and anterior chest wall for two months, shown in figure 1.

On examination, she was afebrile and her vitals were stable. Neurological assessment showed a depressed sensorium (GCS E2 V2 M4), normal deep tendon reflexes, bilateral flexor plantar responses, and negative meningeal signs. Notably, horizontal nystagmus was observed.

Fundus examination indicated bilateral blurring of disc margins, creating an impression of raised intracranial pressure, which initially led to suspicion of a space-occupying lesion, seen in Figure 2. Meanwhile, the low-grade fever combined with vesicular lesions on the skin suggested a possible viral aetiology, prompting an initial provisional diagnosis of viral meningitis or encephalitis.

Investigations

Routine laboratory tests—including complete blood count, renal function, and arterial blood gas analysis—were within normal limits. Liver function tests showed elevated liver enzymes, though viral markers for hepatitis were negative. Magnetic resonance imaging (MRI) of the brain demonstrated bilateral symmetrical T2 and diffusion-weighted imaging (DWI) hyperintensities in the dorsomedial and pulvinar nuclei of the thalami, with no significant contrast enhancement, shown in figure 3.

Treatment

Given the possibility of central nervous system infection, the patient was started on empirical treatment with acyclovir, ceftriaxone, dexamethasone. Cerebrospinal fluid analysis subsequently ruled out meningitis or encephalitis. Under clinical suspicion of Wernicke's encephalopathy, parenteral thiamine was initiated. The patient's neurological status improved dramatically within 48 hours, with her GCS rising to E4 V5 M6. This rapid response to thiamine supported the final diagnosis of Wernicke's encephalopathy secondary to hyperemesis gravidarum.

DISCUSSION

Thiamine (vitamin B1) is a water-soluble vitamin with a recommended daily allowance (RDA) of 0.4 mg per 1000 kcal. In a non-pregnant adult, the average diet generally meets this requirement. However, during pregnancy, thiamine needs increase to approximately 1.5 mg per day due to higher metabolic demands. Failure to meet these requirements can result in Wernicke's encephalopathy (WE), a neurological emergency that arises from acute thiamine deficiency.

Pathogenesis

Thiamine pyrophosphate is an essential cofactor for several enzymes in the pentose phosphate pathway, predominantly in the brain. Inadequate thiamine disrupts energy metabolism and leads to necrosis or apoptosis of neuronal cells. The classical presentation of WE includes the triad of encephalopathy, ataxia, and oculomotor dysfunction (nystagmus and gaze palsies). Many patients can also develop antegrade and retrograde amnesia, often referred to as Korsakoff's psychosis.

Clinical Diagnostic Aids

A combination of clinical features, rapid reversal of neurological symptoms with thiamine supplementation, supportive MRI findings, and transketolase activity assays help confirm the diagnosis of WE. Transketolase enzyme activity, measured in red blood cells, serves as a functional test for thiamine deficiency. Normal values range from 0% to 15% stimulation; 15–25% indicates thiamine deficiency, and values above 25% imply severe deficiency. Elevated blood pyruvate and lactate can further suggest thiamine deficiency. On MRI, T2-hyperintensities typically involve areas such as the medial thalami, peri-aqueductal region, and near the third and fourth ventricles; less commonly, lesions are found in the caudate nucleus, hypothalamus, or isolated mammillary bodies. These regions rely heavily on oxidative phosphorylation, rendering them particularly vulnerable to thiamine lack. Follow-up imaging after adequate thiamine treatment often shows resolution or significant reduction of these lesions.

Similar case reports

In our case, bilateral papilledema initially suggested raised intracranial pressure or a space-occupying lesion, mirroring a scenario reported by Mumford [1] and later by Mun-Wei et al. [2], where papilledema complicated or delayed the diagnosis of WE. Furthermore, the presence of fever and vesicular rash in our patient led us to suspect meningitis or encephalitis, underscoring the diagnostic complexity. Similar diagnostic pitfalls have been documented in other reports of WE secondary to hyperemesis gravidarum [3-5].

Management of Wernicke's encephalopathy in pregnancy

Early diagnosis and prompt administration of parenteral thiamine remain paramount. Recommended doses in pregnancy range from 50 mg/day to 200–1200 mg/day intravenously for at least two weeks, followed by oral supplementation (60 mg/day) until delivery [6]. Moreover, concurrent intravenous magnesium sulfate (1–2 mL of 50% solution) can optimize transketolase function, as magnesium is also a cofactor in this enzymatic pathway [7-8].

CONCLUSION

This case highlights the varied and sometimes deceptive presentation of Wernicke's encephalopathy in pregnancy. Bilateral papilledema and a confounding rash can easily obscure the correct diagnosis. However, a high index of suspicion, coupled with early thiamine supplementation, can result in rapid improvement and prevent long-term neurological sequelae.

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Author contributions

Each author has contributed significantly to this manuscript's conception, design, data acquisition, analysis, interpretation, drafting, and revising, in line with accepted authorship standards.

Conflict of Interest: NIL

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Figures – Captions and Legends

Figure 1: Maculo-papular rash with vesicles noted over trunk.

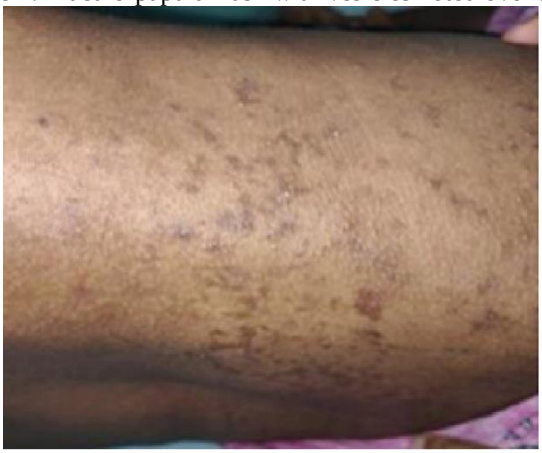


Figure 2: Papilloedema noted in bilateral eyes

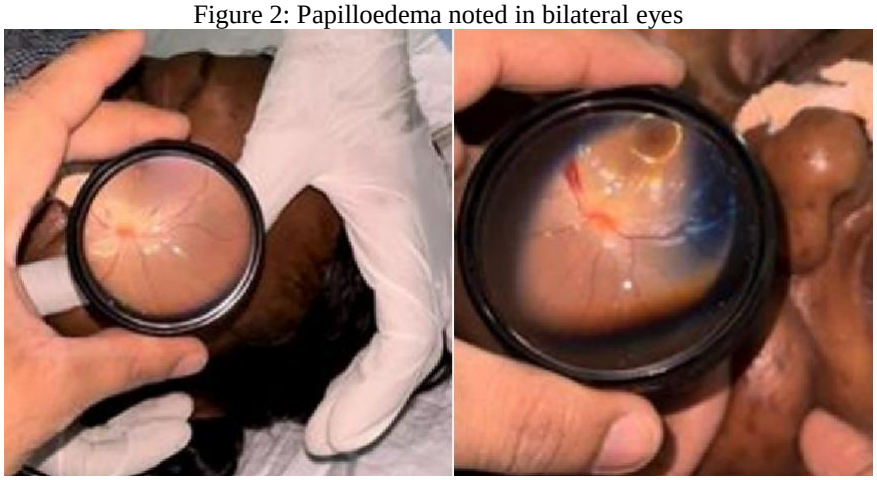
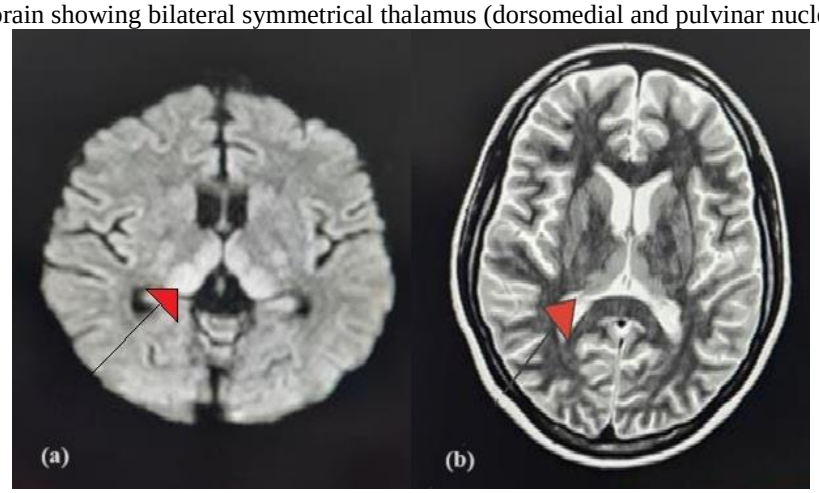


Figure 3: MRI brain showing bilateral symmetrical thalamus (dorsomedial and pulvinar nuclei) involvement.



(a) Diffusion weighted imaging
(b) T2-weighted imaging

The lesion is marked by red arrows. This appearance, termed as “hockey-stick” appearance, is not really specific for Wernicke’s encephalopathy and is also seen in Cruetzfeld-jacob disease.