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# WERNICKE'S ENCEPHALOPATHY SECONDARY TO HYPEREMESIS GRAVIDARUM PRESENTING WITH INTERNUCLEAR OPHTHALMOPLEGIA AND PONTINE DEMYELINATING LESION IN EARLY PREGNANCY: A CASE REPORT

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**ABSTRACT:**
Wernicke's encephalopathy is a rare but serious neurological emergency caused by thiamine deficiency. It is most commonly associated with chronic alcoholism but can also occur in hyperemesis gravidarum [1]. In pregnancy, hyperemesis gravidarum can lead to profound nutritional depletion, and Wernicke's encephalopathy should be suspected when neurological symptoms develop [2]. We report a case of a 24-year-old primigravida at 6 weeks and 3 days of gestation who presented with persistent vomiting, weight loss, diplopia, and gait ataxia. Clinical examination revealed right eye adduction palsy with nystagmus consistent with internuclear ophthalmoplegia, multiple cranial nerve palsies involving the third, ninth, tenth and eleventh nerves, and gait ataxia. Magnetic resonance imaging of the brain demonstrated a possible demyelinating lesion in the right paramedian posterior pons [3]. Lumbar puncture and autoimmune markers were negative. High-dose intravenous thiamine at 200 mg twice daily for five days was initiated promptly along with antiemetics, fluid resuscitation and nutritional support [4]. The patient showed rapid improvement in ocular movements and resolution of nystagmus. This case highlights that Wernicke's encephalopathy can present without the classic triad of ophthalmoplegia, ataxia and confusion and with atypical magnetic resonance imaging findings such as pontine demyelinating lesions [5]. A high index of suspicion is essential in pregnant women with hyperemesis gravidarum to prevent irreversible neurological damage [6].

**KEYWORDS:** Wernicke's encephalopathy, Hyperemesis gravidarum, Thiamine deficiency, Pregnancy, Internuclear ophthalmoplegia, Cranial nerve palsy, Gait ataxia, Magnetic resonance imaging (MRI), Pontine demyelinating lesion, Neurological emergency.

## INTRODUCTION

Hyperemesis gravidarum affects approximately 0.3 to 3 percent of pregnancies and is characterized by intractable nausea, vomiting, weight loss exceeding 5 percent of pre-pregnancy weight, dehydration and electrolyte disturbances [1]. Severe hyperemesis gravidarum can lead to nutritional deficiencies, particularly thiamine or vitamin B1 deficiency, because of reduced oral intake and increased metabolic demands [2]. Wernicke's encephalopathy is an acute neurological disorder resulting from thiamine deficiency and is classically described by the triad of ophthalmoplegia, ataxia and global confusion [3]. However, the complete triad is present in only 16 to 33 percent of cases, and atypical presentations are increasingly recognized, especially in non-alcoholic populations [4]. In pregnant women, Wernicke's encephalopathy is most frequently associated with hyperemesis gravidarum, with a reported incidence of 0.4 to 1.7 percent among women hospitalized for hyperemesis [5]. The classic neuropathological findings include symmetrical lesions in the mammillary bodies, thalamus, periaqueductal grey matter and brainstem, but atypical magnetic resonance imaging findings such as cerebellar, cortical or pontine involvement have been described [6]. Internuclear ophthalmoplegia, a disorder of horizontal gaze due to a lesion of the medial longitudinal fasciculus, is a rare manifestation of Wernicke's encephalopathy and can mimic demyelinating diseases such as multiple sclerosis [7]. We present a case of Wernicke's encephalopathy secondary to hyperemesis gravidarum in early pregnancy with atypical features including internuclear ophthalmoplegia and a pontine demyelinating-like lesion on magnetic resonance imaging. The case emphasizes the need for early recognition and prompt thiamine therapy to prevent permanent neurological sequelae [8].

### Case Presentation

A 24-year-old primigravida presented to the emergency department at 6 weeks and 3 days of gestation with a four-week history of progressively worsening nausea and vomiting. She reported vomiting two to three times daily initially, which increased to eight to ten times daily over the preceding week. She had decreased oral intake, significant weight loss of approximately 4 kilograms representing more than 7 percent of her pre-pregnancy weight, and subjective giddiness. For the past two days, she developed diplopia and a sensation that her left eye was deviating outward. She had no significant past medical history, no psychiatric illness, no alcohol use and no family history of neurological disorders. Her pregnancy was spontaneous and previously uncomplicated. She was not taking any regular medications or supplements.

On admission, the patient was conscious, oriented to time, place and person, with fair general condition. Her vital signs were blood pressure 110/70 mmHg, pulse 102 beats per minute and she was afebrile. There was no pallor, icterus or pedal edema. Neurological examination revealed that for cranial nerves three, four and six, the left eye showed full range of movements, whereas the right eye exhibited adduction palsy with inability to adduct the right eye on horizontal gaze and associated nystagmus on abduction of the right eye. Convergence was intact. These findings were consistent with right internuclear ophthalmoplegia [7]. For cranial nerves nine and ten, the uvula was deviated to the right and the gag reflex was not elicited. For cranial nerve eleven, there was weakness of the right sternocleidomastoid manifesting as weak head turn to the left. Motor system examination showed normal bulk and tone with power 5/5 in all limbs. Coordination testing revealed gait ataxia with a wide-based, unsteady gait, while finger-nose-finger and heel-shin tests were normal. Sensory system was intact to all modalities. Cognitive function was preserved with no confusion, disorientation or memory impairment; the Mini-Mental State Examination score was 30 out of 30. Cardiovascular and respiratory examinations were normal. Per abdominal examination, abdomen soft, on per vaginum examination cervix pointing downwards, and uterus anteverted. Bilateral fornices free, nontender, bulky and fetal heart rate was positive at 127 beats per minute. Ophthalmology opinion confirmed no evidence of papilledema, full visual fields and a normal fundus.

**Investigations**

Laboratory findings included a normal complete blood count. Serum electrolytes showed mild hyponatremia with sodium 132 mEq/L and mild hypokalemia with potassium 3.1 mEq/L. Blood glucose was 78 mg/dL. Liver and renal function tests were normal. Urine ketones were 3 plus. A dating scan revealed a single live intrauterine gestation of 6 weeks and 4 days with a fetal heart rate of 127 beats per minute. Lumbar puncture was performed and the cerebrospinal fluid was clear with glucose 58 mg/dL (serum glucose 82 mg/dL), protein 32 mg/dL and white cell count 2 per cubic millimetre. Cerebrospinal fluid Gram stain, culture, AFB and cryptococcal antigen were negative. Cerebrospinal fluid oligoclonal bands, NMO/MOG antibodies and serum anti-aquaporin-4 antibodies were all negative, effectively ruling out multiple sclerosis and neuromyelitis optica spectrum disorder.

Magnetic resonance imaging of the brain with whole spine showed two chronic lacunar infarcts in the left hemi pons that were incidental and likely old. More significantly, there was diffusion restriction in the right paramedian posterior pons, which was interpreted as a possible demyelinating lesion [3]. No other abnormal enhancement or mass effect was seen. The mammillary bodies and thalamus were normal. These imaging findings are incorporated as follows: MRI scan demonstrates two small chronic lacunar infarcts in the left hemi pons on an axial T2-weighted image, and shows hyperintensity with restricted diffusion in the right paramedian posterior pons on a diffusion-weighted image, suggestive of an acute demyelinating lesion.

**Diagnosis and Differential Diagnosis**

Based on the clinical triad of hyperemesis gravidarum, ophthalmoplegia in the form of internuclear ophthalmoplegia, and ataxia, and supported by a pontine lesion on magnetic resonance imaging, a diagnosis of Wernicke's encephalopathy secondary to thiamine deficiency was made [3]. Although the classical triad was incomplete because confusion was absent, the presence of internuclear ophthalmoplegia and ataxia in the context of severe nutritional deficiency was sufficient [4]. Differential diagnoses considered included multiple sclerosis, which was ruled out by negative cerebrospinal fluid oligoclonal bands and normal spine magnetic resonance imaging; neuromyelitis optica spectrum disorder, ruled out by negative aquaporin-4 antibodies; brainstem stroke, considered unlikely given the absence of vascular risk factors and the pattern of diffusion restriction not typical for lacunar infarct; and acute disseminated encephalomyelitis, which was less likely as there was no recent infection or vaccination.

**Management and Outcome**

The patient was admitted to the high-dependency unit for close neurological monitoring. Management was instituted immediately, even before all confirmatory test results were available, because Wernicke's encephalopathy is a medical emergency where delay in treatment can cause irreversible damage [8]. Pharmacological therapy consisted of intravenous thiamine at a dose of 200 mg twice daily for five days, followed by 100 mg daily orally until discharge and continued at 100 mg daily throughout pregnancy [9]. Antiemetics were given as intravenous ondansetron 4 mg three times daily and intravenous pantoprazole 40 mg once daily. Fluid and electrolyte correction was achieved with intravenous normal saline together with potassium chloride to correct hypokalemia, plus multivitamin supplementation. Nutritional support was provided by gradually reintroducing oral intake with small, frequent meals, and folic acid 5 mg daily was continued.

Daily neurological examination was performed. By day three of thiamine therapy, the patient reported significant improvement in diplopia. On day five, the right eye adduction palsy had completely subsided and nystagmus was absent. Gait ataxia also improved, although mild unsteadiness persisted at discharge. Clinical impressions before and after treatment are the pretreatment image on rightward gaze demonstrates failure of the right eye to adduct with nystagmus of

the left eye, and the post-treatment image on day five shows full adduction of the right eye with no nystagmus. The patient was discharged on day seven with oral thiamine, antiemetics as needed and a high-protein, high-calorie diet. She was referred to a high-risk obstetric clinic for ongoing prenatal care. At 12-week follow-up, the pregnancy was progressing normally, and there were no residual neurological deficits.

## DISCUSSION

Wernicke's encephalopathy in pregnancy is uncommon but underdiagnosed. The true incidence may be higher because many cases are missed due to atypical presentations [4]. Hyperemesis gravidarum is the leading cause of Wernicke's encephalopathy in pregnancy, accounting for nearly 90 percent of reported cases [5]. The pathophysiology involves depletion of thiamine stores, which are critical for glucose metabolism, neurotransmitter synthesis and membrane ion transport. Without adequate thiamine, brain regions with high metabolic demand, including the mammillary bodies, thalamus and brainstem, undergo neuronal death [3].

Our patient's presentation was atypical in two key ways. First, she had internuclear ophthalmoplegia rather than the more common horizontal or vertical gaze palsies. Internuclear ophthalmoplegia results from a lesion in the medial longitudinal fasciculus, a heavily myelinated tract connecting the abducens nucleus on one side to the oculomotor nucleus on the opposite side. In Wernicke's encephalopathy, medial longitudinal fasciculus involvement is rare, but a few case reports have described internuclear ophthalmoplegia as a presenting feature [7]. Second, the pontine demyelinating lesion on magnetic resonance imaging is atypical. Classically, Wernicke's encephalopathy shows symmetrical signal changes in the periaqueductal grey matter, thalami and mammillary bodies [6]. However, brainstem involvement including the pons is increasingly recognised, especially in non-alcoholic Wernicke's encephalopathy [10]. The diffusion restriction in our patient's pons likely reflects acute cytotoxic oedema secondary to thiamine-induced energy failure.

The negative cerebrospinal fluid and autoimmune markers were crucial to exclude multiple sclerosis, which can present with internuclear ophthalmoplegia and brainstem lesions. The rapid clinical response to thiamine within 72 to 96 hours is both diagnostic and therapeutic, a hallmark of Wernicke's encephalopathy [3]. Delayed or inadequate thiamine treatment can lead to Korsakoff syndrome with irreversible memory impairment or death [8]. The recommended regimen for Wernicke's encephalopathy is 200 to 500 mg of intravenous thiamine three times daily for three to five days, followed by oral maintenance [9]. Notably, glucose infusion without prior thiamine can precipitate or worsen Wernicke's encephalopathy, so thiamine must always be given before or with glucose. This case underscores that the absence of the classic triad should not delay treatment. Any pregnant woman with hyperemesis gravidarum and neurological symptoms, even subtle ones like dizziness or mild ataxia, should receive empirical thiamine [2].

## CONCLUSION

Wernicke's encephalopathy is a preventable and treatable neurological emergency [8]. In pregnant women with hyperemesis gravidarum, a high index of suspicion is required because classical features are often absent [4]. Internuclear ophthalmoplegia and pontine demyelinating lesions are rare but important atypical presentations that can mimic multiple sclerosis [7]. Early recognition, prompt high-dose intravenous thiamine and supportive care lead to excellent neurological recovery [9]. Clinicians should maintain a low threshold for initiating thiamine therapy in any at-risk pregnant patient [2].

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