CASE REPORT

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Wernicke's encephalopathy secondary to hyperemesis gravidarum

Encefalopatía de Wernicke secundaria a hiperémesis gravídica

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ABSTRACT

We report the case of a 32-year-old multigestational woman with 18 weeks of gestation hospitalized for hyperemesis gravidarum. She was admitted with signs of dehydration, weight loss (8.8%) and suffered fetal loss. There was evidence of impaired consciousness, horizontal and vertical nystagmus and ataxia. A brain tomography without contrast showed no alterations. Wernicke's encephalopathy was diagnosed and thiamine was replaced, with a slow and favorable evolution.

Key words: Wernicke encephalopathy, Hyperemesis gravidarum, Thiamine, Nystagmus, Pregnancy complications

RESUMEN

Se comunica el caso de una multigesta de 32 años con 18 semanas de gestación hospitalizada por hiperémesis gravídica, quien ingresó con signos de deshidratación, pérdida de peso (8,8%) y sufrió pérdida fetal. Se evidenció trastorno de conciencia, nistagmo horizontal y vertical y ataxia. La tomografía cerebral sin contraste no mostró alteraciones. Se diagnosticó Encefalopatía de Wernicke y se repuso tiamina, con evolución lenta favorable.

Palabras clave. Encefalopatía de Wernicke, Hiperémesis gravídica, Tiamina, Nistagmo, Complicaciones del embarazo

Introduction

Wernicke's encephalopathy (WE) is an acute neuropsychiatric syndrome associated with thiamine (vitamin B1) deficiency, characterized by the symptomatic triad of ataxia, oculomotor impairment and sensorimotor impairment. Its incidence in the general population is 0.6%[1]. In pregnancy it may occur in cases with predisposing factors, especially in hyperemesis gravidarum[2-5].

Hyperemesis gravidarum occurs in 0.5-3% of pregnant women and is defined according to the 5 diagnostic criteria of Windsor 2021, which are severe nausea and vomiting, early onset before 16 weeks, inability to drink or eat, limitations in the performance of daily activities and dehydration[6,7], which can lead to depletion of multiple nutrients including thiamine[8].

We describe this case because of its semiological richness and presentation, which is little known in our environment.

Case report

A 32-year-old multigestation with an 18-week pregnancy with no previous pathologies and the previous pregnancy without complications. Since the first trimester she presented moderate nausea and vomiting according to the PUQE scale (3-5/day), associated with abdominal pain and hyporexia which became severe on the PUQE scale (5-8/day) from 10 weeks, associated with oral intolerance, so it was decided to hospitalize her. On admission she presented poor general condition with signs of moderate dehydration. Vital signs were heart rate of 131 beats per minute, blood pressure of 90/60 mmHg, respiratory rate of 22 breaths per minute and

saturation of 95%. She had a weight loss of 4.6 kg (8.8%) in 3 weeks. Laboratory tests are shown in Table 1. Brain tomography showed no alterations (Figure 1). She was diagnosed with an 18-week pregnancy, hyperemesis gravidarum and hydroelectrolyte disorder. Since her admission, disorientation in time and space and visual and auditory hallucinations were evidenced. On the third day, horizontal and vertical nystagmus and ataxia were identified with progressive worsening. He also presented fasciculations and cramps. Pupils were isoreactive, without ophthalmoparesis and without involvement of lower cranial nerves or long pathways. Wernicke's encephalopathy was clinically diagnosed, she was hydrated and administered thiamine 500 mg IV every 8 hours for 24 hours and 100 mg every 6 hours for 2 days, repeating the same dose on the fourth day due to clinical worsening.

On the eighth day of hospitalization, the absence of fetal heartbeat was evidenced, and induction and uterine curettage were performed after fetal expulsion (fetal weight 215 g). Severe hypoalbu-

Figure 1. Multislice spiral tomography without contrast. Axial and sagittal view. Presence of calcifications. Absence of ischemic or hemorrhagic alterations.

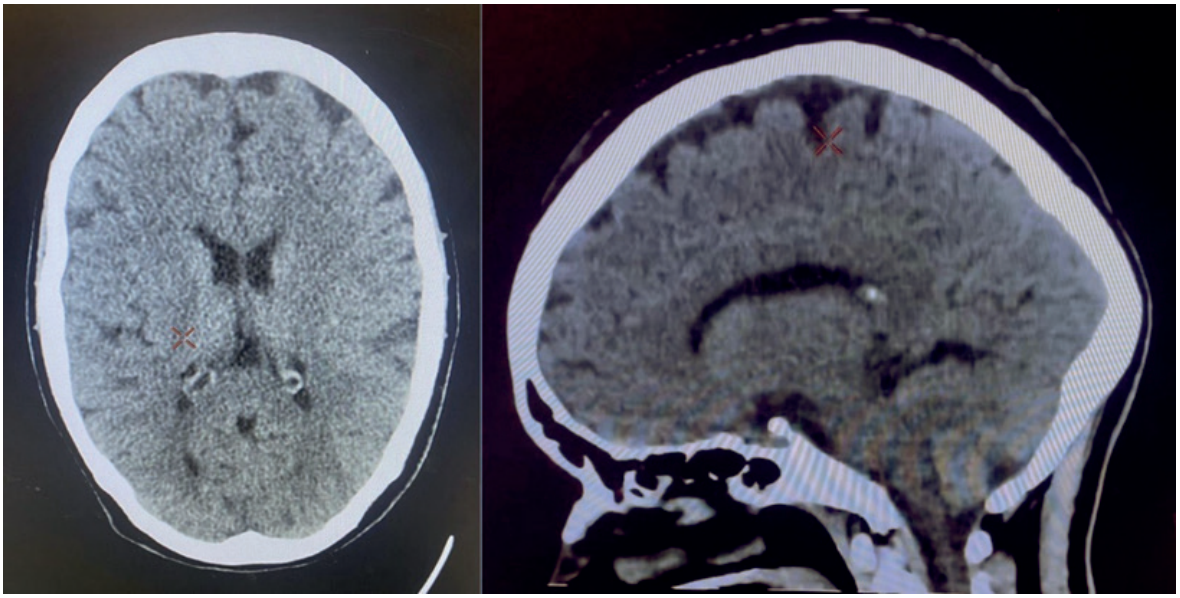

Table 1. Initial blood tests and during the fifth day of hospitalization.

Values	Admission	Fifth day of hospitalization (ICU)	Reference range
Hemoglobin (g/dL)	10	8.7	11.0-16.0
Sodium (mmol/L)	131	136	136-146
pH	7.462	7.440	7.350 -7.450
pCO2 (mmHg)	33.8	32.7	35.0-48.0
HCO3 (mmol/L)	23.9	21.8	21.0-28.0
AST (U/L)	34.5	24.4	0.0-38.0
ALT (U/L)	72.4	30.7	0.0-40.0
Alkaline phosphatase (U/L)	169.9	166.6	98.0-276.0
Direct bilirubin (mg/dL)	1.29	0.64	0.00-0.25
Total bilirubin (mg/dL)	1.79	0.87	0.00-1.00
Lactic acid (mmol/L)	0.7	1.3	0.5-1.6
Creatinine (mg/dL)	0.62	0.49	0.70-1.40
Albumin (g/dL)	2.79	2.51	3.50-5.00
PCR (mg/dL)	47.3	63.2	0.0-6.0
TSH (µUI/mL)	0.64	0.64	0.27-4.20
Free T-4 ng/dL	0.88	0.88	0.93-1.70
Urine test Ketone bodies	+++		

Note: AST, aspartate aminotransferase; ALT, alanine aminotransferase; PCR, C-reactive protein; TSH, thyroid-stimulating hormone.

minemia (1.9 g/dL) and severe anemia after the procedure (6.7 g/dL) were found and treated with globular pack transfusion and parenteral iron.

Discharge was indicated on the fourteenth day, hemodynamically stable and with partial improvement of neurological symptoms, as she maintained nystagmus and ataxia.

One month after discharge, general malaise, difficulty in walking and nystagmus persisted with partial improvement.

Discussion

We report the case of a 32-year-old pregnant woman with 18 weeks of pregnancy complicated by hyperemesis gravidarum who presented with the triad of Wernicke's encephalopathy (WE) and fetal loss, having been hospitalized for 14 days with a slow favorable evolution.

About 80% of pregnancies are affected by nausea and vomiting, mainly during the first trimester[1]. Of these, the disorders are severe in up to 3%, establishing the condition of hyperemesis gravidarum[9] according to the Windsor 2021 criteria[6,7], and may be associated with more serious conditions such as Wernicke's encephalopathy[10], peripheral neuropathy and central pontine myelinolysis[11,12]. In addition, 50% of cases of Wernicke's encephalopathy are associated with fetal loss or other fetal complications[1]. In the case presented, vomiting started early in the first trimester and progressed to oral intolerance, dehydration, hypokalemia, metabolic acidosis and fetal loss.

Wernicke's encephalopathy (WE) may be due to inadequate intake or conditions associated with increased thiamine requirement, such as pregnancy[4,8]. Depletion of thiamine reserves occurs between 4 and 6 weeks without supplementation. Diagnosis is clinical with the classic triad of consciousness disorder, oculomotor disturbances and ataxia[13]. Brain tomography is usually normal. Magnetic resonance imaging is more sensitive and specific. In T1 there is evidence of hypointensities in the periaqueductal gray matter, mammillary bodies, hypothalamus and medial thalamus. In T2 there are hyperintensities around the third ventricle, mammillary bodies, hypothalamus and periaqueductal gray matter[14]. Treatment with high doses of thiamine in a timely manner is of utmost importance, since it can reverse the syndrome. Otherwise, mortality can be 20%, and about 80% develop severe cognitive problems, such as Korsakoff's syndrome[13,15]. In the described case, the reversibility of the symptoms should be evaluated periodically, maintaining thiamine supplementation. If it persists for six months, it would be a case of Wernicke-Korsakoff with sequelae. The fetal loss in this case was probably multifactorial (due to dehydration, metabolic changes, multiple vitamin deficiency, lack of caloric intake, among others), as shown in research, with a frequency of up to 50% of cases[1]. These severe modifications and no early management would be the cause of etal death[16].

Wernicke's encephalopathy is a rare pathology, scarcely published in our environment, but it should be suspected and foreseen in cases of hyperemesis gravidarum, in order to treat it adequately, avoid long-term sequelae and maternal and fetal morbimortality.

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