

WERNICKE'S ENCEPHALOPATHY: A CASE REPORT AND MRI FINDINGS

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Wernicke's encephalopathy (WE) is a serious, potentially fatal acute or subacute neurological disorder caused by thiamine (Vitamin B1) deficiency. Although it is most frequently observed in patients who are chronic alcoholics, WE may also be associated with hyperemesis gravidarum. We report magnetic resonance imaging (MRI) and diffusion weighted imaging (DWI) findings in this case of WE in a pregnant patient with hyperemesis gravidarum. We conclude that DWI should be included in the imaging protocols of patients suspected to suffer from Wernicke's encephalopathy.

Key-words: Pregnancy, complications – Brain, diseases.

Nausea and vomiting generally peak between the 8th and 12th weeks of pregnancy, and they are usually mild and self-limited (1). Hyperemesis gravidarum, however, is a severe condition with vomiting pernicious enough to cause weight loss, dehydration and alkalosis. It affects among 0.3-2% of all pregnant patients (2). Wernicke's encephalopathy (WE) is a disorder due to thiamine deficiency associated with alcoholism and malnutrition but can also arise during the first trimester of pregnancy, due to hyperemesis gravidarum. We present conventional magnetic resonance imaging (MRI) and diffusion weighted imaging (DWI) findings in this unusual case of WE.

Case report

A 37-year-old gravida 1 para 0 woman in her 5th week of gestation presented with the diagnosis of hyperemesis gravidarum after uncontrolled anorexia, constant nausea, vomiting and intermittent diarrhea had persisted longer than 5 days. Her physical examination was normal. Laboratory studies were remarkable only for an elevated alanine aminotransferase (ALT) level of 60 U/L [normal range 10-31 U/L] and ketonuria. Other parameters including thyroid function tests, urine and stool culture for bacterias, abdominopelvic ultrasound, serological tests for hepatitis and immunological markers for liver diseases were normal. On hospital day 6, ultrasound scan revealed a 6 week-old heart beating fetus. Routine hyperemesis gravidarum

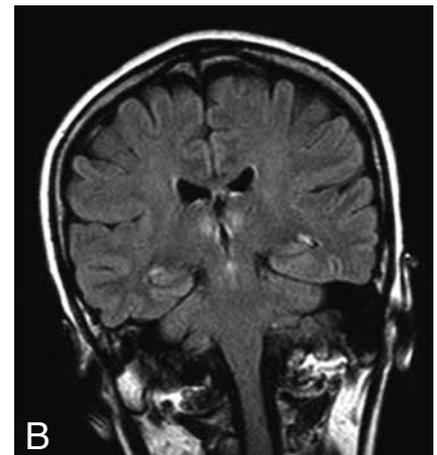
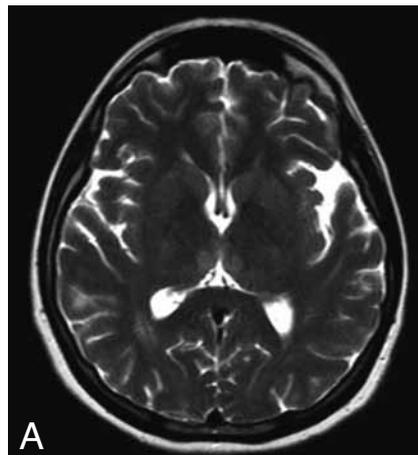


Fig. 1. — A. T2-weighted transaxial image demonstrating symmetrical bilateral hyperintense lesions in both dorsomedial thalamic regions. B. FLAIR image demonstrating symmetrical hyperintense lesions within dorsomedial thalamic regions.

therapy continued to the 10th week of gestation. At that time the patient became more depressive (had attacks of crying) and less cooperative. She had psychiatric consultation but no drugs or other medications were given. Her complete neurological examination was unremarkable, however a cranial MRI was offered to exclude any intracranial pathologies. The patient refused MRI with pretending her claustrophobia.

Her general state of nausea, vomiting and insomnia worsened over the next 2 weeks with 4 kilograms loss. During the 12th gestational week, she developed signs of confusion, depression, muscle weakness, ataxia and a tendency to fall backwards, and a sudden decrease in vision. Fundoscopic examination

revealed loss of border-lines of the discs and papiledema. Vision acuity decreased to 15 cm. The patient was not able to stand and walk because of the truncal ataxia. Total parenteral nutrition and electrolyte replacement therapy were started. With the dramatic loss in her vision, she accepted MRI. Because of the increased rates of thrombotic events in pregnancy, venous sinus thrombosis was suggested initially. Magnetic resonance venography and magnetic resonance angiography showed normal vessels. T2-weighted and fluid attenuated inversion recovery (FLAIR) images demonstrated symmetrical hyperintense lesions within dorsomedial thalamic region (Fig. 1). DWI showed symmetrical pathologic thalamic hyperintensities and the apparent diffusion coefficient (ADC) map images showed signal reductions suggesting restricted diffusion within these regions (Fig. 2).

As the neurologic signs and MRI findings pointed to a diagnosis of WE, the patient was transferred to neurology department. Laboratory

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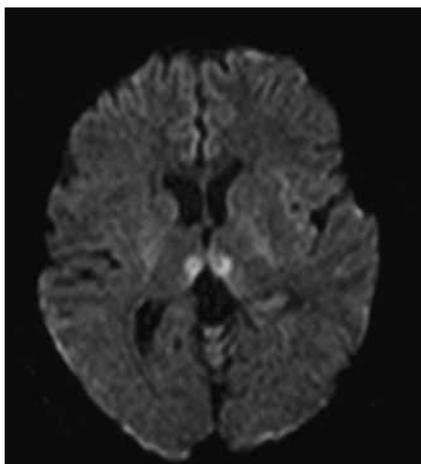


Fig. 2. — DWI image showing hyperintensities in both thalami.

estimation of serum thiamine was not available before administration of it. Her vision was back three days after the initial rapid intravenous infusion of 250 mg thiamine and the oral treatment of 250 mg/day with other vitamin B complexes. By the second week, her symptoms lost. She could stand and walk without help.

Discussion

WE is a rare but known complication of hyperemesis gravidarum resulting from the combination of poor nutritional intake, frequent vomiting, and increased metabolic demands of pregnancy (3). WE can also be precipitated by parenteral nutrition or infusion of glucose-containing solutions without prior administration of thiamine (4, 5).

The clinical features are non-specific but include the classical triad of ocular abnormalities, confusion and ataxia. It should be remembered that the clinical triad is not obligatory. Symptoms may include lethargy, fatigue, apathy, impaired awareness, equilibrium loss, disorientation, difficulty to concentrate, retrograd amnesia, anorexia, muscu-

lar weakness, peripheral numbness, paresthesia, disorientation, hallucinations, confabulation, memory loss, impaired linguistic processing, anterograde amnesia and global intellectual impairment (6). The diagnosis of WE is based on the clinical manifestations and rapid reversal of symptoms with thiamine. Early diagnosis is essential in WE in order to avoid persistent brain damage (7). Determination of blood transketolase activity and thiamine pyrophosphate reflects the thiamine status in the body. But only few centers have this diagnostic tool due to technological complexity and cost (8). Hence, MRI plays an important role in the diagnosis of WE. Common MRI findings of WE are symmetrically increased signal intensities in the mesencephalic tegmentum, mamillary body and medial thalamus on proton-density and T2-weighted images. In addition, due to the possibility that cerebrospinal fluid may mask high signal lesions on T2-weighted and proton-weighted images, fluid-attenuated inversion-recovery (FLAIR) sequences were found to be better in detecting the lesion conspicuously (9). Diffusion-weighted imaging (DWI) is based on an echo-planar MRI technique, and it is highly sensitive to intracellular edema. DWI changes with decreased signal intensity on ADC (apparent diffusion coefficient) maps are associated with restricted diffusion and cytotoxic edema, while increased ADC values represent vasogenic edema. The importance of ADC values in the diagnosis of WE is unclear, since decreased or increased ADC values have been reported and both cytotoxic and vasogenic edema patterns are present in lesions seen in WE (7, 10). Our patient showed high signal intensities within the thalami on DWI images and demonstrated decreased ADC values. We assume that the high signals seen on DWI images were, at least in part, caused by true restricted diffusion representing cytotoxic edema.

In conclusion we presented an unusual case of WE in a pregnant patient with hyperemesis gravidarum. This case emphasizes that, while conventional MRI is helpful, DWI should be considered as a valuable additional imaging sequence in patients suspected to suffer from WE.

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