Wernicke's encephalopathy associated with hyperemesis gravidarum

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INTRODUCTION

Wernicke's encephalopathy is an uncommon neurological disorder caused by thiamine deficiency, the triad of which includes ocular abnormalities, ataxia and a global confusional state (Reuler et al, 1985). Wernicke's encephalopathy can develop in women with hyperemesis gravidarum, especially when vomiting lasts for more than 4 weeks (Togay-Isikay et al, 2001). However, timely thiamine treatment is often delayed because this disease remains underdiagnosed. This article reports an unusual case of Wernicke's encephalopathy associated with hyperemesis gravidarum.

DISCUSSION

Wernicke's encephalopathy adversely affects maternal and fetal morbidity and mortality. This patient had residual neurological deficits, although she delivered a healthy baby. Togay-Isikay et al (2001) reviewed 29 cases of Wernicke's encephalopathy associated with hyperemesis gravidarum. They reported that all patients developed this disorder after at least 4 weeks of persistent vomiting. Two patients remained with severe neurological deficits and three patients died because of a delay in diagnosis. Three patients underwent planned abortion and 12 patients experienced fetal death. Only 10 patients delivered a healthy baby.

However, awareness of Wernicke's encephalopathy makes a correct diagnosis possible. Magnetic resonance imaging (MRI) also has diagnostic value during the acute phase of Wernicke's encephalopathy. Characteristic MRI findings in Wernicke's encephalopathy include high signal intensities in bilateral caudate nuclei, periaqueductal gray matter, thalami and hypothalami (Ohkoshi et al, 1994), and central pontine myelinolysis (Bergin and Harvey, 1992). However, brain lesions on MRI can resolve after thiamine replacement (Omer et al, 1995), probably because MRI abnormalities in Wernicke's encephalopathy may be the result of reversible cytotoxic oedema caused by thiamine deficiency (Chu et al, 2002).

Delay in diagnosis leads to permanent neurological deficits, coma and death, but prompt thiamine treatment can prevent development of Wernicke's encephalopathy. Tan and Ho (2001) describe that parenteral thiamine (50–100 mg) should be given when Wernicke's encephalopathy is suspected and that oral thiamine following parenteral thiamine is necessary as long as the patient remains at risk of thiamine deficiency. Early recognition of Wernicke's encephalopathy as a complication of hyperemesis gravidarum is important to prevent maternal morbidity and mortality. HM

CASE REPORT

A 30-year-old woman, who had had one previous pregnancy and successful delivery, developed hyperemesis gravidarum at 11 weeks of gestation and experienced a sudden onset of vertigo at 12 weeks. She lost 20 kg in weight between 11 and 15 weeks. At 15 weeks, she was admitted to a local hospital because of persistent vomiting, vertigo and confusional mental state. Vomiting and altered mental state recovered with intravenous hyperalimentation, but vertigo persisted. At 18 weeks, she was hospitalized to a regional otolaryngology unit for evaluation of vertigo and ataxia. She had bilateral vertical nystagmus and truncal ataxia. She showed an abnormal response in vestibulocollic reflex and galvanic body sway test despite a normal response in caloric test. These results indicated that vertigo was mainly caused by the dysfunction of the semicircular system, otolithic system and brainstem. Magnetic resonance imaging (MRI) of the brain at 22 weeks was normal.

On suspicion of Wernicke's encephalopathy, administration of oral thiamine (75 mg/day) and vitamin B_{12} (1500 μg/day) was started with rehabilitation for ataxic gait. Truncal ataxia made an incomplete recovery, but nystagmus did not improve. At 27 weeks, she was referred to the authors for antenatal care. She could not stand and walk unaided because her nystagmus worsened when turning the head. She had short-term memory disturbance during the initial hospitalization. Neurological examinations revealed obtund sensations and hyporeflexia of the lower limbs. There were no ophthalmoplegia, papilloedema or ptosis. Fluid-attenuated inversion recovery MRI of the brain displayed no abnormal signals. A diagnosis of Wernicke's encephalopathy was established based on the preceding history, persistent amnesia, nystagmus and cerebellar dysfunction. Oral thiamine and vitamin B_{12} had been continued until term. The pregnancy was otherwise normal and she delivered a baby weighing 3352 g spontaneously at 39 weeks. She still had vertigo and gait ataxia at 1 month postpartum.

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