J Med Sci 2008;28(4):183-186
http://jms.ndmctsgh.edu.tw/2804183.pdf

Wernicke's Encephalopathy in a Patient after Partial Gastrectomy with Prolonged Parenteral Nutrition

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Wernicke's encephalopathy is a common complication of thiamine deficiency among alcoholics. However, there are few reports of this disorder in nonalcoholic individuals. We present a case of Wernicke's encephalopathy in a nonalcoholic 71-year-old man who received partial gastrectomy because of refractory upper gastrointestinal bleeding. Symptoms of Wernicke's encephalopathy, characterized by ocular abnormalities, ataxia and disturbances of consciousness, occurred three weeks after surgery and prolonged parenteral nutrition. The patient also showed autonomic involvement, with unstable blood pressure and tachycardia. This clinical presentation, combined with typical magnetic resonance imaging (MRI) findings of changes to the brain (thalamus, hypothalamus, mamillary bodies and periaqueductal gray matter), confirmed the diagnosis. The restriction in energy intake and the malabsorption induced by major gastrointestinal tract surgery could explain the patient's thiamine deficiency that led to Wernicke's encephalopathy. The inconsistency of MRI changes on diffusion-weighted imaging, without reduction in the apparent diffusion coefficient, indicated that damage had occurred earlier than at the onset of overt clinical symptoms, which could account for the sequelae despite thiamine administration.

Key words: parenteral nutrition, partial gastrectomy, Wernicke's encephalopathy, gastrojejunostomy

INTRODUCTION

In circumstances associated with an increased need for vitamins, such as following major surgery, the sustained nutrition shortage eventually leads to clinically severe consequences. Wernicke's encephalopathy (WE) is one of the severe forms of nutritional metabolic encephalopathy caused by thiamine deficiency. WE is clinically characterized by general confusion, extra-ocular palsies, nystagmus, ataxia, mitotic pupils and sluggish papillary reflexes, and peripheral neuropathy and reduced tendon reflexes[1]. WE is usually associated with chronic alcohol misuse, but there have been increasing reports[2] of the condition in nonalcoholic patients in clinical situations (such as following gastroenteric resection), associated with chronic wasting diseases, and in women with hyperemesis gravidarum. The presence of WE in such patients emphasizes important but often overlooked factors associated with this fatal but preventable disease. Here we describe a patient with upper gastrointestinal bleeding who developed WE three weeks after partial gastrectomy with prolonged follow-up parenteral nutrition. He had no apparent predisposing factors such as chronic alcoholism, malnutrition, starvation or gastrointestinal neoplasm. Brain magnetic resonance imaging (MRI) showed hyperintense lesions involving the circumventricular nuclear regions in the thalamus, hypothalamus and bilateral mamillary bodies. These features and the clinical symptoms were consistent with the diagnosis of WE.

Received: January 31, 2008; Revised: March 10, 2008; Accepted: April 14, 2008
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CASE REPORT

A 71-year-old man with peptic ulcer disease was admitted to our hospital because of upper gastrointestinal bleeding. Laboratory investigations provided no significant finding. He had relatively good nutritional status (serum albumin 2.9 g/dL; total cholesterol 161 mg/dL; triglyceride 170 mg/dL) and no alcohol consumption habit. Active bleeding of the gastric ulcer persisted after a few days of conservative treatment, so an exploratory laparotomy with partial gastrectomy and a Roux-en-Y gastrojejunostomy was performed. Partial parenteral nutrition (including glucose, amino acids, sodium, potassium,

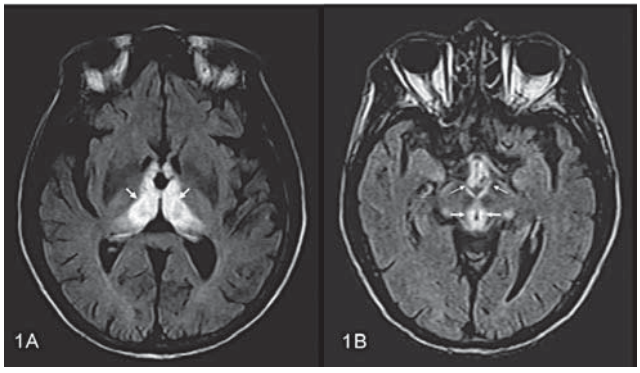

Fig. 1 Axial image of brain magnetic resonance image one week after the patient's onset of Wernicke's encephalopathy. (A) Fluid attenuated inversion recovery (FLAIR) images showed hyperintense lesions involving the circumventricular nuclear regions in the thalamus (arrows). (B) Increased signal was found in both mamillary bodies (arrows) and periaqueductal gray matter (arrowheads) on FLAIR images.

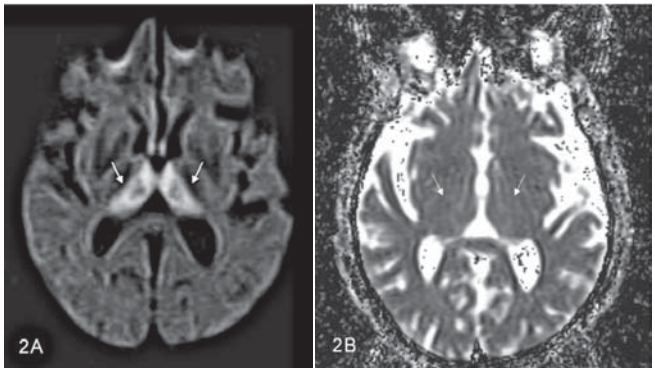

Fig. 2 (A) Increased signals in the thalamus were also apparent on diffusion-weighted imaging (arrows). (B) Changes on diffusion-weighted imaging were not accompanied by reduction in the apparent diffusion coefficient (arrows).

phosphate, trace elements and vitamins A, B, D, E, and C) was administered for more than three weeks. His poor gastrointestinal motility with general ileus limited oral intake to clear fluids. Oral intake of 5% dextrose in water was given after his gastrointestinal condition had improved.

After three weeks on this regimen, the patient showed intermittent consciousness disturbance with confused conversation. His extra-ocular movements were normal, but with nystagmus. Both pupils were normal in size, with a normal light reflex. We noted that his muscle power was grade III/V in all four limbs, with diminished bilateral deep tendon reflexes. Although able to stand, his weakness and ataxia prevented ambulation. No Brudzinski's or Kernig's signs were noted. He was judged to have a Glasgow Coma Scale score of $E_4M_5V_4$. No clinical symptoms of fever or headache were evident. Drug screening for opiates and barbiturates was negative and the patient's electrolytes were in balance. Over the next few days, his state of consciousness decreased from drowsy to coma. He developed unstable blood pressure with wide fluctuations (systolic pressure range 70-130 mmHg) and tachycardia (heart rate 130 bpm), but showed no fever. These conditions were stabilized with vasopressors and fluid resuscitation. The clinical diagnosis was coma of unknown origin.

A brain computer tomography (CT) scan was performed but showed no significant findings, whereas brain MRI showed prominent abnormalities on fluid-attenuated inversion recovery (FLAIR) imaging (Fig. 1A, B). Diffusion-weighted imaging (Fig. 2A) showed multiple hyperintense lesions involving the circumventricular nuclear regions, including the tegmentum of the brain stem from the midbrain to the medulla, thalami and hypothalamus. In particular, both mamillary bodies were involved. However, changes on diffusion-weighted imaging were not accompanied by reductions in the apparent diffusion coefficient map (Fig. 2B).

From the clinical picture and radiology findings we diagnosed WE. The serum level of thiamine determined prior to the initial treatment was 63 nmol/L (normal range, 77-199 nmol/L). Treatment with intravenous vitamin B complex containing 300 mg/day of thiamine was continued for more than two years, but there was no improvement. The Glasgow Coma Scale score at follow-up was $E_1M_2V_1$, and the patient remained comatose with disappointing sequelae of dysconjugate eyeball position with the left eye deviated laterally, and moderate to severe weakness of all four extremities distally.

DISCUSSION

Wernicke's encephalopathy is caused by a thiamine (vitamin B_1) deficiency. With decreased thiamine intake and alcohol-induced intestinal malabsorption, WE usually occurs in chronic alcoholics, but is seldom seen in nonalcoholic individuals. Recent reports indicate that most instances of WE in nonalcoholic patients involved complications that developed following gastrointestinal surgery.

In this patient, symptoms of delirium, muscle weakness of all four extremities and nystagmus occurred three weeks

after major gastrointestinal surgery and prolonged parenteral nutrition. Before surgery, he had none of the predisposing factors for WE, such as alcoholism, malnutrition, or gastrointestinal neoplasm, and he did not show persistent nausea or vomiting following surgery. However, his poor gastrointestinal motility and inability to digest limited oral food intake, so parenteral nutrition with vitamin B supplementation was prolonged for three weeks. However, a gastrojejunostomy causes the ingesta to bypass the duodenum, where the absorption of thiamine mainly occurs. The occurrence of WE in association with a variety of abnormalities of the gastrointestinal tract has been reported to develop, for example, weeks after gastrectomy[3] in association with starvation and/or prolonged parenteral feeding[4].

Unstable hemodynamics with tachycardia and a drop in blood pressure occurred subsequent to an improvement in the patient's gastrointestinal condition and commencement of oral intake of 5% dextrose in water. A carbohydrate load can precipitate WE in thiamine-deficient individuals because of the prompt consumption of the available thiamine as a cofactor in carbohydrate metabolism[5], and autonomic involvement is a core feature in patients with WE[6].

Radiological investigations are of high diagnostic value for patients with WE, and MRI is the method of choice[7]. This modality has gained favor in the diagnosis of WE, with a reported sensitivity of 53% and a specificity of 93%[8]. Typically, patients show involvement of bilateral and symmetrical lesions in the periventricular areas. These include the medial aspect of the thalamus, hypothalamus and mamillary bodies; the periaqueductal region at the level of the third cranial nerve; the reticular formations of the midbrain; and the inferior corpora quadrigemina and the floor of the fourth ventricle[9]. Doherty et al. also reported that diffusion-weighted imaging abnormalities may suggest early thiamine deficiency, and are useful in diagnosing WE[10].

In this patient, brain FLAIR MRI showed features of multiple hyperintense lesions involving the circumventricular nuclear regions from the midbrain to the medulla, thalami, hypothalamus, and both mamillary bodies. Most of these changes were also apparent on diffusion-weighted imaging, but were not accompanied by a reduction in the apparent diffusion coefficient, suggesting that the damage had occurred earlier than at the onset of clinical symptoms[10].

Thiamine hydrochloride is commonly administered prophylactically to chronic alcoholics, but unfortunately may be omitted in hospitalized nonalcoholic patients who are also liable to thiamine deficiency and WE if their nutrition is dependent solely on clear liquids or parenteral fluids. Aging, which reportedly increases the need for thiamine[11], may also contribute to the development of a relative thiamine deficiency. A decrease in thiamine supply, an increase in the need for thiamine, and ineffective absorption promote a state of thiamine deficiency. The inconsistency of brain MRI imaging changes on diffusion-weighted imaging, without reduction in the apparent diffusion coefficient, indicated that the pathological abnormalities of the brain happened earlier, in the absence of a relevant clinical presentation. This is a challenge for physicians as a delay in appropriate treatment can lead to irreversible consequences, in this case including a dysconjugate eyeball position, moderate to severe weakness of all four extremities distally, and an ongoing comatose state despite treatment with thiamine (300 mg/day) for more than two years.

Here we have reported a case of WE that occurred after partial gastrectomy with prolonged parenteral nutrition. MRI was sufficient to establish a diagnosis of WE in the presence of a typical clinical presentation and the exclusion of other possible causes of acute encephalopathy. WE remains a rare but life-threatening condition that is often overlooked in the nonalcoholic population, and can result in the further progression of an easily treatable condition. Left untreated, acute WE has a 17% mortality rate[12]. Therefore, the occurrence of WE should be considered when a patient's consciousness deteriorates for unknown reasons after major gastrointestinal surgery involving gastrectomy or gastrojejunostomy with prolonged parenteral feeding.

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