

Wernicke's Encephalopathy after Bariatric Surgery with Atypical Magnetic Resonance Imaging: A Case Report

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Abstract-

Wernicke's encephalopathy (WE) is a disease caused by thiamine deficiency related to alcoholism, hyperemesis, or thiamine malabsorption. The clinical manifestations of WE are mental change, ataxia, and ophthalmoplegia. The typical magnetic resonance imaging (MRI) findings of WE are symmetrical involvement of medial thalamus, periventricular region of the third ventricle, periaqueductal area, and mammillary body. The atypical MRI findings are more common in nonalcoholic WE. Since the increasing population of obesity and the preference of weight loss surgery, the risk of developing thiamine deficiencies associated with weight loss surgery has become a considerable etiology of WE. We herein reported a case reminds clinicians that WE can be a possible diagnosis in patient who developed acute altered mental status with atypical MRI lesion involving bilateral centrum semiovale and corona radiata after receiving bariatric surgery.

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CASE

A 24-year-old non-alcoholic male patient presented to emergency department due to acute altered mental status in one day. He has received laparoscopic gastric Roux-en-Y bypass 2 months ago because of morbid obesity, and lost 18kg as a result. He had admitted to our hospital twice soon after the operation due to intractable vomiting and unsteady gait and discharged after symptomatic treatment. Neurologic examination at emergency department showed comatose consciousness, quadriplegia, skew deviation with impaired oculo-cephalic reflex. Serology exam found hypopotassemia, hyponatremia, relatively low level of albumin, normal vitamin B12 level, and there was

no hypoglycemia episode at emergency room nor after admission. Cerebrospinal fluid study was normal. Brain magnetic resonance imaging (MRI) with gadolinium enhancement was done four days after the symptoms onset, which found symmetric diffuse weighted image (DWI) hyperintensity, apparent diffusion coefficient (ADC) hypointensity and T2-weighted fluid-attenuated inversion recovery (T2 FLAIR) faint hyperintensities in bilateral centrum semiovale and corona radiata (Figure 1). His consciousness level had dramatically improved after intravenous thiamine 500mg every 8 hours for 3 days. The patient discharged without any neurological sequelae. Follow brain MRI 73 days later showed resolution of previous lesions (Figure 2).

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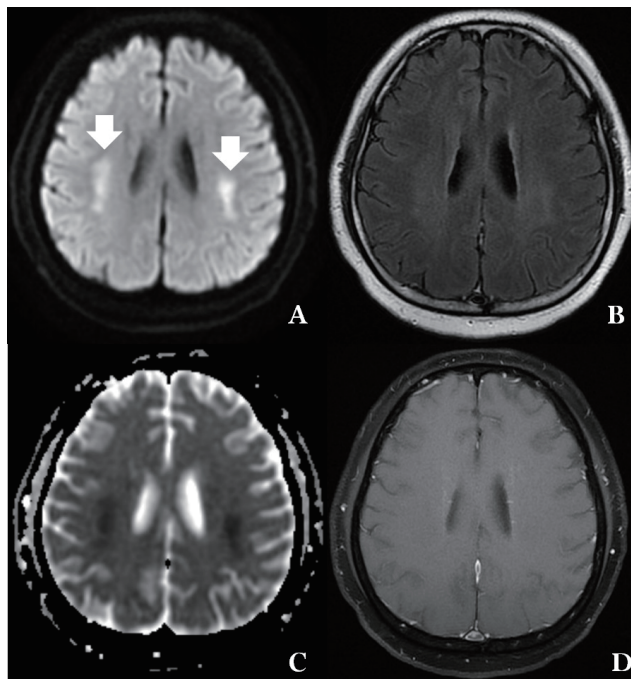


Figure 1. Relative symmetric hyperintensity at bilateral centrum semiovale and corona radiata in DWI (A), faint hyperintensities in T2 FLAIR (B), hypointensity in ADC (C) and T1-weighted contrast enhanced (D) image were normal.

DWI: diffuse weighted image

T2FLAIR: T2-weighted fluid-attenuated inversion recovery

ADC: apparent diffusion coefficient

DISCUSSION

Thiamine is a water-soluble vitamin and only obtained from exogenous sources. Human storage of thiamine can be depleted within 2 to 3 weeks after malabsorption⁽¹⁾. The risks of developed Wernicke encephalopathy (WE) include alcoholism, malignancy, gastrointestinal disease and surgery, hyperemesis gravidarum and starvation. Triad of WE are mental change, ataxia, and ophthalmoplegia, and typical MRI findings of WE are symmetrical involvement of medial thalamus, periaqueductal area and mammillary body, which is significantly related to alcohol abuse. Atypical MRI findings have been reported in the brain stem cranial nerve nuclei, the dentate nuclei of cerebellum, red nuclei, tectum of the midbrain⁽²⁻⁵⁾, which are more common in nonalcoholic WE⁽²⁾. Base

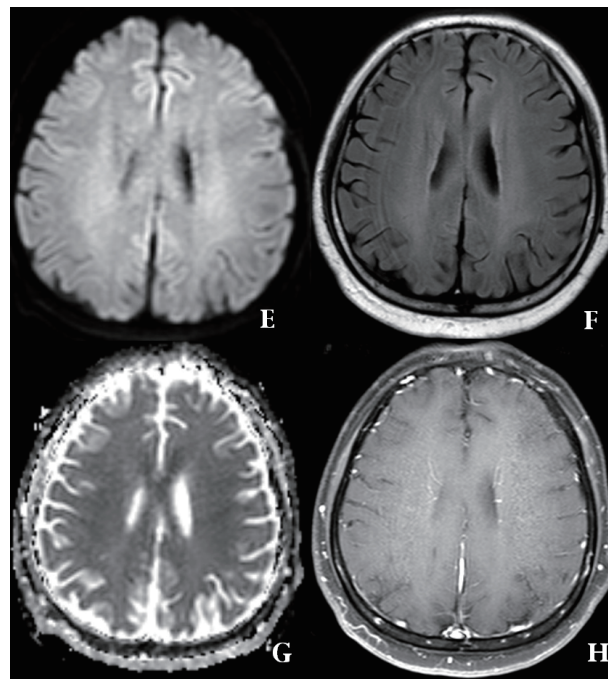


Figure 2. Follow up image 73 days later revealed resolution of previous lesions

DWI (E), T2 FLAIR (F), ADC (G), T1-weighted

contrast enhanced (H)

on 2 review articles⁽⁶⁻⁷⁾, brain MRI finding of WE can present with DWI hyperintensities with decreased ADC represent cytotoxic edema, probably due to the osmotic dysregulation, which caused by thiamine deficiency. Additionally, MRI findings of WE may be related to the timing of MRI with regard to the initiation of thiamine deficiency, which may be normal in early stage but may change over time to true restricted diffusion.

In our case, a non-alcoholic patient had the history of gastric Roux-en-Y bypass with post-operative intractable vomiting, developed acute alter mental status and ophthalmoplegia. Brain MRI showed the symmetric lesion involving bilateral centrum semiovale and corona radiata. Thiamine supplement shows a dramatic improvement of clinical symptoms and resolution of the MRI lesion, without any neurological sequelae on the follow-up. Both

clinical symptoms and radiological evolution support the diagnosis of WE.

Though weight loss surgery has become a popular treatment option for severe obesity, post-operative micronutrient deficiencies was reported in many cases. Thiamine malabsorption directly relate to resection of gastric folds⁽⁸⁾. A systemic review suggested the complication rate of central or peripheral nerve system or both among the patients who received bariatric surgery is 5%~16%⁽⁹⁾, 94% of WE occurred within 6 months after surgery⁽¹⁰⁾. The continuous vomiting is the major predisposing factor to developing WE in patients who received weight loss surgery⁽¹¹⁾, which leads to thiamine malabsorption and promote a symptomatic micronutrients deficiency⁽¹⁰⁾. A timely thiamine supplement can rapidly improve the clinical conditions and late interventions may increase the risk of long-term damage or irreversible sequelae. In another systematic review, only 36% of patients with a complete recovery after a median follow-up of 180 days, which highlights the importance of timely diagnosis and treatment⁽¹¹⁾.

According to the guideline from European Federation of Neurological Societies, the thiamine status should be followed at least 6 months after bariatric surgery. The suggestion for treatment WE is intravenous thiamine infusion with 200mg three times daily, until there is no further improvement of clinical state. All patients at emergency room with a risk condition should be given prophylactic intravenous 200mg thiamine before carbohydrates is given⁽¹²⁾. Oral supplementation may then be indicated until deficiency factors are eliminated^(10,13). In our case, for example, this patient who received bariatric surgery with developed WE needs a life-long thiamine replacement.

In conclusion, the clinicians should keep WE in mind when facing a patient with developed acute altered mental status and ophthalmoplegia, in particular with history of bariatric surgery with intractable vomiting. The atypical MRI lesion involved in bilateral centrum semiovale and corona radiata could be seen in those non-alcoholic WE patient. The prompt diagnosis and treatment can avoid irreversible sequelae.

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