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Wernicke's encephalopathy rapidly progressed to Korsakoff syndrome after laparoscopic sleeve gastrectomy

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ABSTRACT

We report the case of a 26-year-old woman who developed Wernicke's encephalopathy following a sleeve gastrectomy for obesity. Despite receiving supplemental thiamine therapy, her condition deteriorated into severe Korsakoff syndrome, characterized by significant memory impairment and confabulation. This case highlights the critical need for vigilant postoperative dietary management and early detection of nutritional deficiencies in bariatric surgery patients. Clinicians should prioritize comprehensive nutritional education and monitoring to prevent life-altering complications associated with thiamine deficiency.

Introduction

Wernicke's encephalopathy (WE) is a severe and life-threatening condition caused by vitamin B1 (thiamine) deficiency, with an estimated prevalence of 0.4% to 2.8% in the general population (1). Patients who undergo bariatric surgery are at increased risk for nutritional deficiencies and metabolic complications. Approximately 16% to 18% of cases develop neurological complications due to thiamine, folate, and cobalamin (vitamin B12) deficiencies, which typically appear within weeks to months after bariatric surgery (2,3). Since thiamine absorption primarily occurs in the proximal jejunum, Roux-en-Y gastric bypass presents the highest risk for WE, with an overall incidence of 4.29 cases per 100,000 surgeries, compared to 1.06 for sleeve gastrectomy (3). When WE is suspected, prompt Thiamine administration is crucial to prevent further neurological damage.

Case Presentation

A 26-year-old woman diagnosed with severe obesity [body mass index (BMI) of 40.75 kg/m^2] presented without a history of hypertension, diabetes, or heart disease. She underwent laparoscopic sleeve gastrectomy on February 28, 2023. One month post-surgery, she resumed a regular diet, although her eating patterns were erratic. On May 12, 2023, she developed a fever and vomiting of gastric contents. Anti-infective treatment was only partially effective. Her condition gradually worsened, marked by fatigue and drowsiness, and on May 18, she experienced syncope and was hospitalized.

The patient was unconscious and uncooperative during the examination, but a withdrawal response was observed upon stimulation. The left pupil had a diameter of 2.5 mm with a sluggish light reflex, while the right pupil had a diameter of 3.0 mm with no light reflex. Muscle strength testing in all four limbs was inconclusive. Muscle tone was not increased, the neck was supple without resistance, physiological reflexes were present, and no pathological reflexes were elicited. Blood tests revealed elevated white blood cell count (13.69×10^9/L), neutrophil percentage (73.4%), and absolute neutrophil count (10.04×10^9/L), alongside a low blood phosphorus (0.59 mmol/L), potassium (2.5 mmol/L), and serum folate (2.4 ng/mL), though vitamin B12 was normal. Bacterial cultures, cerebrospinal fluid analysis, and autoimmune tests were negative; however, lumbar puncture showed elevated protein concentration and pressure (260 mmH_2O).

A cranial computed tomography scan was negative, but an electrocardiogram indicated sinus tachycardia and significant ST-segment depression. Sputum culture was positive for *Staphylococcus aureus*. Treatment included levofloxacin, electrolyte correction, and supportive care, but the patient's condition deteriorated further into a continuous coma with complications such as severe pneumonia, respiratory failure, sepsis, and multi-organ dysfunction. On May 30, brain magnetic resonance imaging (MRI) revealed symmetric T2-weighted imaging (T2WI) and fluid-attenuated inversion recovery (FLAIR) hyperintensities around the cerebral aqueduct and bilateral thalami (Figure 1). Considering the history of sleeve gastrectomy, persistent vomiting, and inadequate nutrition, the patient was diagnosed with WE. Intravenous thiamine (500 mg) was administered three times daily for five days, and the dosage was gradually reduced to 100 mg (Figure 2).

Following treatment, inflammatory markers and microbial cultures were negative, and respiratory and hemodynamic stability improved; however the patient remained in a coma.

Discussion

Obesity presents a global challenge, and for individuals with a BMI exceeding 40, traditional dietary and exercise interventions often prove ineffective, prompting the recommendation of bariatric surgery. Common procedures include sleeve gastrectomy, Roux-en-Y gastric bypass surgery, or duodenal switch (4,5). Thiamine deficiency can disrupt energy metabolism in the nervous system, provoke oxidative stress, and trigger inflammatory responses, ultimately leading to nerve damage, particularly in specific brain regions such as the thalamus, mammillary bodies, and the area surrounding the cerebral aqueduct, culminating in WE (6,7). In patients undergoing weight loss surgery, only a minority (0.0002% to 0.4%) develop vitamin B1 deficiency progressing to WE, typically manifesting within six months postoperatively (7).

WE is a neurological syndrome associated with a mortality of 20%. Due to the gradual onset and nonspecific neurological symptoms of WE, misdiagnosis is common. The classic clinical triad comprises ataxia, confusion, and ophthalmoplegia (1,7). WE is frequent in individuals with a history of alcohol abuse, and other risk factors include weight loss surgery, hemodialysis, severe vomiting during pregnancy, and malabsorption syndrome. Its diagnosis requires at least two of five features: dietary deficiency, ocular signs, cerebellar ataxia, memory impairment, or altered mental status (1,8). Moreover, some patients exhibit atypical manifestations, making diagnosis challenging. In cases where patients are at risk of nutritional malabsorption or imbalance, WE should be suspected, even if only part of the triad is observed.

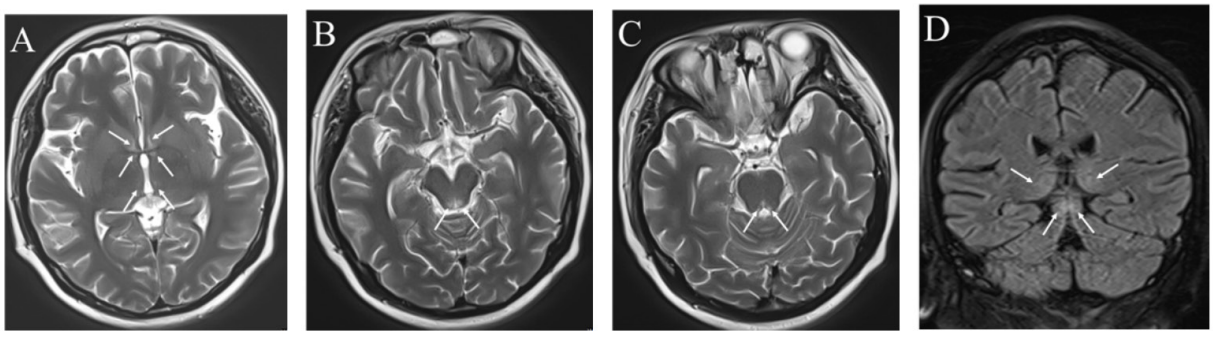

Figure 1. Brain MRI showed bilateral high-signal intensity lesions surrounding the cerebral aqueduct and thalamic regions, a typical finding of WE
MRI: Magnetic resonance imaging, WE: Wernicke's encephalopathy

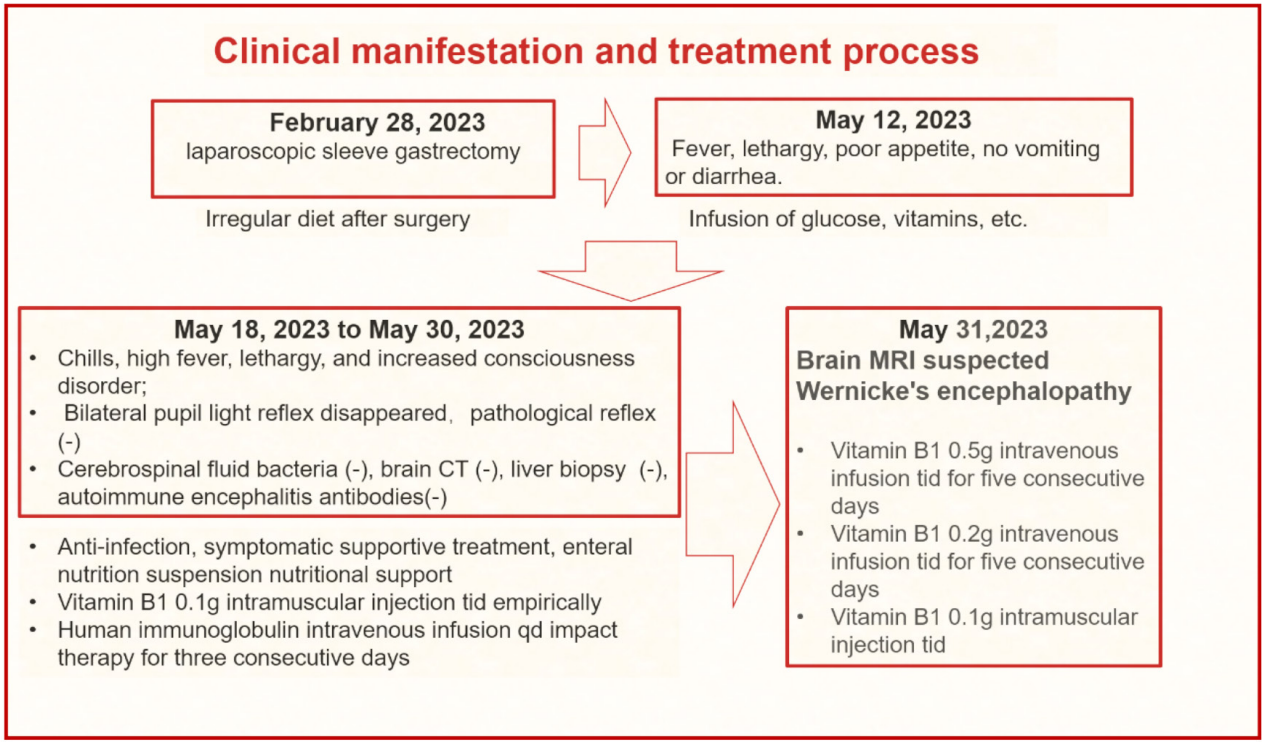

Figure 2. A flow chart of the patient's symptoms and treatment
CT: Computed tomography, MRI: Magnetic resonance imaging

In cases where WE is strongly suspected, immediate intravenous thiamine injection is crucial. The European Federation of the Neurological Societies guidelines (9) recommend thiamine 200 mg thrice daily for suspected or confirmed WE, preferably via intravenous line, because oral supplements are poorly absorbed in large doses. For bariatric surgery patients, the risk of developing WE persists in the long term, with follow-up on thiamine status recommended for at least six months: 94% of WE cases occur within this timeframe post-operatively. On the other hand, alcohol-dependent patients may require higher daily doses, with the suggested regimen of 500 mg three times daily. This intervention not only confirms the diagnosis but also prevents progression to severe Korsakoff sydrome, characterized by anterograde and retrograde amnesia, confabulation, apathy, and potentially coma or death. Moreover, serum thiamine levels and erythrocyte transketolase activity are commonly used as diagnostic measures. WE diagnosis can be confirmed through MRI, which typically reveals high signals on T2WI and FLAIR sequences in the mammillary bodies, around the cerebral aqueduct, thalamus, and hippocampus. In MRI studies, the sensitivity for diagnosing WE was found to be 53%, with a specificity of 93% (1).

Following weight loss surgery, the patient displayed erratic dietary patterns and insufficient nutritional intake, coupled with symptoms of fever and vomiting. Subsequently, the patient rapidly deteriorated, experiencing syncope and entering a coma, meeting at least two diagnostic criteria for WE. Moreover, MRI findings were consistent with the typical presentation. Due to the patient's unstable vital signs upon admission, the MRI examination and diagnosis were delayed until 18 days after symptom onset. Despite intravenous thiamine injections, the patient developed severe Korsakoff sydrome and remained comatose.

Conclusion

This case underscores the critical importance of comprehensive nutritional management and proactive monitoring of patients undergoing bariatric surgery. The progression of WE to irreversible Korsakoff syndrome, despite thiamine supplementation, illustrates the severe consequences of delayed diagnosis and inadequate dietary oversight. Early recognition of clinical symptoms and prompt intervention are paramount for preventing permanent neurological damage. This case serves as a reminder for clinicians to prioritize patient education on post-operative nutrition, reinforce adherence to dietary guidelines, and maintain vigilant follow-up to ensure early detection of nutritional deficiencies.

Ethics

Informed Consent: Informed consent was obtained from the patient's parents for the anonymous use and publication of clinical and imaging data.

Footnotes

Authorship Contributions

Concept: C.H., W.Y., Data Collection or Processing: W.Y., Literature Search: C.H., W.Y., Writing: C.H., W.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

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