Clinical Case Report

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Wernicke encephalopathy concurrent with polyradiculoneuropathy in a young man after bariatric surgery

A case report

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Abstract

Rationale: Bariatric surgery is the recommended treatment for morbid obesity because of its rapid and sustained body weight loss effect. Nutrient deficiency-related neurological complications after bariatric surgery are often disabling. Thus, early recognition of these complications is important. Neurological complications involving the central and peripheral nerve system after bariatric surgery were reported. However, the report on the clinical course of the concurrent involvement of central and peripheral nervous system is limited. We present a rare case of a patient who developed Wernicke encephalopathy concurrent with polyradiculoneuropathy after receiving bariatric surgery.

Patient concerns: A 22-year-old man with a history of morbid obesity presented progressive bilateral lower limbs weakness, blurred vision, and gait disturbance 2 months after receiving laparoscopic sleeve gastrectomy. Bilateral lower limb numbness and cognition impairment were also noted.

Diagnosis: Brain magnetic resonance imaging and electrophysiologic studies confirmed the diagnosis of Wernicke encephalopathy concurrent with acute polyradiculoneuropathy.

Interventions: Vitamin B and folic acid were given since admission. He also received regular intensive rehabilitation program.

Outcomes: The subject's cognitive impairment and diplopia improved 1 week after admission under medical treatments, yet lower limb weakness and gait disturbance were still noted. After a month of intensive inpatient rehabilitation, he was able to ambulate with a walker for 30 munder supervision.

Lessons: Nutrient deficiency-related neurological complications after bariatric surgery are often disabling and even fatal. Prevention of neurological complications can be improved through close postsurgical follow-up of the nutritional status. Recognizing the signs and symptoms and evaluating the medical history are critical to the early diagnosis and treatment of this potentially serious yet treatable condition.

Abbreviations: LSG = laparoscopic sleeve gastrectomy, MR = magnetic resonance.

Keywords: bariatric surgery, obesity, polyradiculoneuropathy, sleeve gastrectomy, wernicke encephalopathy

1. Introduction

Obesity, along with overweight, affects over a third of the world's population. Obesity-attributable medical expenditure is a major public health challenge worldwide.[1] Nonsurgical weight loss treatments, such as lifestyle management or medications, are often ineffective for the adults with extreme obesity and with body index mass (BMI) at 95th percentile.[2] Bariatric surgery is the recommended treatment for morbid obesity due to its rapid and sustained body weight loss effect.[2,3] With numerous bariatric procedures being performed on an increasing obese population, evidence on the long-term benefits of the procedure is being discovered.[2]

Bariatric surgical procedures are associated with malabsorption and micronutrient deficiency, but their prevalence and nature depend on the type of surgery.[3] Sleeve gastrectomy, Rouen-Y gastric bypass, and adjustable gastric banding are the most commonly performed procedure.[3] Nutrient deficiency is a common complication in postoperative patients and is observed in more than one-third of this population.[4] Nutrition deficiency-related neurological complications after bariatric surgery are often disabling and even fatal.[5,6] Thus, early recognition of neurological complications after bariatric surgery is important. Thiamine deficiency occurs as a result of either the bypass of the jejunum, where thiamine is primarily absorbed, or of an impaired nutritional intake from recurrent emesis, which is a

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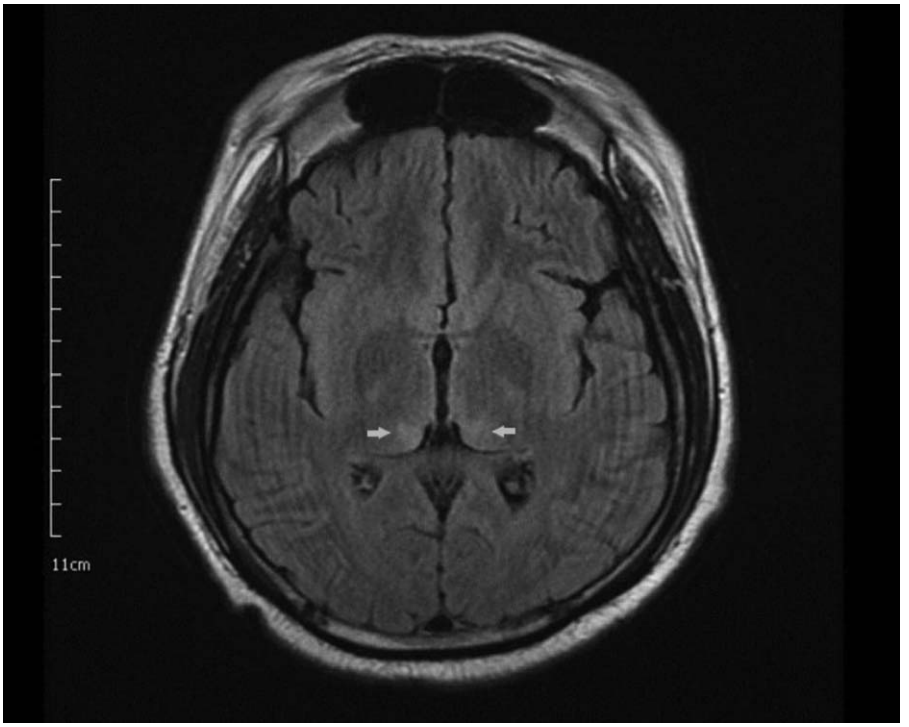

Figure 1. Brain MR imaging shows hyperintense lesions in the bilateral medial thalami (arrow), in fluid-attenuated inversion recovery (FLAIR) images. MR = magnetic resonance.

well-documented postbariatric surgery complication.[6] Stemming from thiamine deficiency, Wernicke encephalopathy is a triad of ataxia, altered mental status, and ophthalmoplegia and is a potentially fatal condition that may be underdiagnosed in this population. Neurologic deficit may still persist in cases where Wernicke encephalopathy is identified and treated.[7,8] Neurological complications after bariatric surgery involving the central and peripheral nervous system after bariatric surgery were reported.[5,6,8] However, most of these studies focused on encephalopathy. Reports on clinical manifestation and recovery course in such complications with both central and peripheral nervous system involvement concurrently are limited. Here, we report a rare case of Wernicke encephalopathy concurrent with acute polyradiculoneuropathy in a 22-year-old man after receiving laparoscopic sleeve gastrectomy (LSG). The neurological recovery clinical course was followed up for 3 years. Furthermore, we compared his clinical course with the clinical course of encephalopathy alone reported in the literature. The clinical manifestation and recovery course are fully discussed.

2. Case presentation

A 22-year-old man with a history of morbid obesity (BMI 45.45, 142 kg) underwent LSG. He lost approximately 20 kg 2 months after receiving LSG. During follow-up visits, he occasionally complained about nausea, vomiting, and constipation. Eight weeks after surgery, the patient began experiencing intermittent nausea and vomiting associated with poor oral intake. An upper gastrointestinal endoscopy was arranged, which revealed grade A gastroesophageal reflux disease without evidence of obstruction. Nine weeks after receiving LSG, he was brought to the emergency department for progressive bilateral lower limbs weakness, blurred vision, and gait disturbance. Insidious bilateral lower limb numbness and confabulation of past memory and current events were also noted. At the emergency department, physical examination revealed disorientation to time and place, dull response, diplopia, bilateral lower limbs weakness [muscle power: 3/5 in bilateral lower limbs, 5/5 in upper limbs on Medical Research council scale], and decreased deep tendon reflex in lower limbs. He could not walk because of ataxia and bilateral lower limb weakness. Initial laboratory data showed leukocytosis, elevated erythrocyte sedimentation rate, and normal thyroid function. Folic acid level was low (3.7 ng/mL), whereas thiamin level was within normal range. No recent fever episodes or infection was noted.

Additional examinations were performed after admission. Nerve conduction velocity study showed polyradiculoneuropathy, sensorimotor type. Cerebrospinal fluid study via lumbar puncture was within normal limits. Brain magnetic resonance (MR) imaging showed hyperintensity in lesions within bilateral medial thalamus in fluid-attenuated inversion recovery image (Fig. 1). Wernicke encephalopathy concurrent with acute polyradiculoneuropathy was impressed. In addition, vitamin B and folic acid were administered since admission. His cognitive impairment and diplopia both improved a week after admission. However, bilateral lower limb weakness and gait disturbance were still noted. Residual bilateral quadriceps weakness (muscle power: 3/5) was noted and moderate assistance was still needed in sit-to-stand and static standing balance 4 weeks after the onset of neurological deficits. After continuous rehabilitation program, he continued to make progress in the area of muscle power, balance, ambulation, and daily life activity. He was able to walk with a walker for 30 m under supervision after 1 month. Three years after the surgery, the patient can walk without device, but unsteady gait was still noted. He needs minimal assistance in climbing up and down the stairs.

The patient gave informed consent for the description of this case and the publication of this report.

3. Discussion

Bariatric surgical procedures are associated with nutritional deficiencies, but their prevalence and nature depend on the types of surgeries.[3] Sleeve gastrectomy, Rou-en-Y gastric bypass, and adjustable gastric banding are the commonly performed procedures.[3] Laparoscopic gastric banding, which is conceptually a restrictive rather than a malabsorptive intervention, is less frequently associated with deficiencies. One exception is the occurrence of early postoperative thiamine deficiency. Sleeve gastrectomy was also conceived as a restrictive intervention. Similar to laparoscopic gastric banding, sleeve gastrectomy is typically less associated with nutritional deficiencies, especially when the postoperative patients adhere to the of prescribed supplements regimen. The operation procedure in our case is LSG. Although the complication rate is low in LSG, the patient still exhibited the neurological symptoms. Severe vomiting and poor and insufficient oral intake before the onset of neurological symptom may be the predisposing factors.

The incidence of neurological complication after bariatric surgery ranges from 0.7% to 5%.[6] The reported complications include encephalopathy, optic neuropathy, acute polyradiculoneuropathy, polyneuropathy, posterolateral myelopathy, and myopathy.[5,6,8] Encephalopathy and acute polyneuropathy are often the early complications that usually appear less than a year after bariatric surgery.[8] In our case, the onset of the symptom is 9 weeks after surgery, which is the same with those in previous studies. Some investigations on the factors affecting the outcome of neurological complications were conducted. Punchai found that 85% of the neurological symptom resolved after nutritional intervention and pharmacotherapy.[6] The MR imaging findings of Wernicke encephalopathy in fluid-attenuated inversion recovery (FLAIR) images commonly show symmetric hyperintense lesions in the bilateral thalami, mammillary bodies, tectal plate, and periaqueductal area. Cases with pathological findings in the bilateral cortex may lead to a poor outcome.[9] The MR imaging of our case showed hyperintense lesions in the bilateral medial thalami. The classical triad of Wernicke encephalopathy includes ophthalmoplegia, gait ataxia, and altered mental status. In our case, the patient has recovered fully from the symptoms of diplopia and cognitive impairment after receiving adequate nutritional intervention and pharmacotherapy after diagnosis. However, persistent gait disturbance was still noted after the completion of inpatient rehabilitation course. Abnormal gait was still noted in follow-up interview 3 years after the onset. The slow recovery course may be due to the concurrent involvement of the central and peripheral nervous system. In Koffman's study, the neurological recovery of the peripheral nerve system is usually delayed compared with that of the central nerve system.[10] Our case echoes the previous conclusions, which stated that the recovery course can be longer with worse outcomes in cases with concurrent Wernicke encephalopathy and peripheral nerve involvement.

The clinical presentation of acute polyradiculoneuropathy after bariatric surgery often resembles that of Guillain Barré syndrome. The occurrence of polyradiculoneuropathy after bariatric surgery is rare. The incidence in Koffman's report is 2%.[11] The concurrence of Wernicke encephalopathy with polyradiculoneuropathy, such as our case, is rarer. The symptoms begin with pain in the feet, followed by ascending paralysis, hyporeflexia, and vibratory sensory loss.[8,10] Electrophysiological study confirms the diagnosis and shows axonal and sensorimotor polyradiculoneuropathy. Vitamin B1 deficiency is associated with the cause of polyradiculoneuropathy. The role of inflammatory and immune mechanism in the peripheral involvement after bariatric surgery is also suggested.[11,12] Neurological symptoms improved after thiamin supplementation therapy.[10,12]

Dietary restrictions to achieve weight loss prior to surgery may also affect the nutritional status. Postsurgical diet and supplementation recommendations and recommendations for prevention of neurological complications have been published. Malabsorption should be considered when the patient presents with abdominal (diarrhea, abdominal distension, flatulence, abdominal pain, and ascites) or general symptoms (persisting or excessive weight loss, anemia, amenorrhea, impotence, infertility, night blindness, xeropthalmia, peripheral neuropathy, tiredness, fatigue, and weakness) after receiving bariatric surgery. Thiamine (vitamin B1) deficiency reported in up to 49% of patients presenting for bariatric surgery.[13] Clinical alertness and prompt adequate treatment in case of deficiency are required because this condition may lead to potentially irreversible neurological manifestations. Vomiting after bariatric intervention together with poor food intake and lack of supplement intake is the principal risk factor for developing thiamine deficiency. The half-life of thiamine is around 2 to 3 weeks.[14] Thus, in cases of recurrent vomiting, inadequate nutrition, or malabsorption, body's thiamine reserve is quickly depleted. In our patient's case, vomiting with constipation and other gastrointestinal symptoms were noted during the outpatient follow-up before the onset of neurological symptoms.

Wernicke encephalopathy occurs in less than 1% of all patients after receiving bariatric surgery, although the condition is likely underdiagnosed.[7,15] Between 10% and 20% of patients present the complete triad of gait ataxia, ophthalmoplegia, and altered mental status.[16] The most frequent ocular abnormalities include nystagmus, bilateral sixth cranial nerve palsies, and conjugate gaze palsy.[17] Thus, the level of suspicion must be high to make a proper diagnosis. Frequent vomiting and medication noncompliance are present in the majority of these patients. Although symptom onset occurs often between 4 and 12 weeks after surgery, most cases of Wernicke encephalopathy develop postoperatively within 6 months.[8] Wernicke encephalopathy is a medical emergency. Several reviews showed that its mortality rate is up to 20%.[7] Once the diagnosis is suspected, treatment should be rapidly initiated with parenteral thiamine. Current guidelines recommend 500 mg of intravenous (IV) thiamine 3 times a day for 2 days to 3 days followed by 250 to 500 mg IV once daily for 5 days.[18] In addition, daily 50 mg to 100 mg oral thiamine should be initiated and continued postoperatively for at least 6 months.

This report presents the clinical course and neurological recovery in a rare case of concurrent Wernicke encephalopathy and acute polyradiculoneuropathy after receiving bariatric surgery. We observed that after nutritional supplement, the symptoms including blurred vision and change in mental status had improved in our case. However, unsteady gait was still noted 3 years after the operation. The limitation of our study is the lack of follow-up brain MR imaging and electrophysiological study which could have provided objective information on the recovery course.

4. Conclusions

Nutrient deficiency-related neurological complications after bariatric surgery are often disabling and even fatal.[8] Prevention

of neurological complications can be improved through close postsurgical follow-up of the nutritional status, especially in patients with significant or persistent vomiting. Recognition of the signs and symptoms is critical to early diagnosis and treatment of this potentially fatal but easily treatable condition. The clinical course and neurological recovery of complications involving the central and peripheral nervous system should be further studied. Recognizing the signs and symptoms and medical history is critical to the early diagnosis and treatment of this potentially serious yet treatable condition.

Author contributions

Pei-Yu Yang and Nai-Hsin Meng acted as corresponding authors. Both of them have equal contribution to this manuscript.
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