Single Case – General Neurology

Wernicke Encephalopathy following Gastric Bypass: A Case Report

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Keywords
Gastric bypass · Wernicke encephalopathy

Abstract
Bariatric surgery is used as a treatment for morbid obesity and often results in rapid weight loss. This procedure has been associated with postoperative nutritional deficiencies. Neurological complications due to nutritional deficiencies include Wernicke encephalopathy, a disorder that affects the central and peripheral nervous system due to thiamine (vitamin B1) deficiency. Wernicke encephalopathy can lead to irreversible consequences if not treated early. Here, we present a case of a 40-year-old woman that developed Wernicke encephalopathy 2 months after gastric bypass surgery, with additional findings of flat affect and concurrent polyradiculopathy. Her diagnosis was delayed due to unique symptoms and an initial workup with negative imaging findings, making the identification of this disorder more complex.

Introduction
Morbid obesity is associated with a decreased life expectancy of about 5–10 years and significantly increased mortality due to cardiovascular causes and cancers [1]. Bariatric surgery has been known to be the most effective treatment for morbid obesity, leading to rapid weight loss, reduced macrovascular complications, and decreased all-cause mortality by up to 40% [2]. Candidates for gastric bypass surgery include patients with a BMI > 40.0 or BMI of 35.5–39.0 with a weight-related comorbidity, such as hypertension, type 2 diabetes, or severe sleep apnea [3]. This procedure is frequently done...
after changes in diet and exercise have been ineffective weight loss solutions. The most common technique is Roux-en-Y gastric bypass, in which the surgeon uses staples to separate the stomach into upper and lower sections, sealing off the bottom, and then connects the small intestine to each portion. Food enters the upper pouch, which leads to quicker satiety due to its smaller size. The food passes from the pouch directly into the jejunum, avoiding calorie absorption [4].

Due to decreased absorption, nutrient deficiencies are found in over a third of patients following gastric bypass surgery [5]. These deficiencies go unrecognized in about half of patients and can be present for years after operation [6]. A nutritional cause for neurologic symptoms following gastric bypass surgery is thiamine (vitamin B1) deficiency, which causes Wernicke encephalopathy [7]. Thiamine is absorbed in the duodenum, which is bypassed in the new route of the GI tract, and lack of absorption leads to neurologic deficits [5]. Wernicke encephalopathy is classically characterized by a triad of ophthalmoplegia, ataxia, and encephalopathy. If symptoms are not treated promptly, it can lead to Wernicke-Korsakoff syndrome, which can involve irreversible short-term memory loss and confabulation [8]. Magnetic resonance imaging (MRI) of Wernicke encephalopathy demonstrates bilateral hyperintensities in the area of the mammillary bodies, thalami, and periaqueductal area on FLAIR imaging [9]. Most reports about thiamine deficiency from gastric bypass surgery focus on the classic triad of Wernicke encephalopathy symptoms. Our case exhibits a rare presentation of Wernicke encephalopathy with concurrent polyneuropathy, vitamin A deficiency, and flat affect following gastric bypass surgery. In addition, symptoms were present and continued to progress before evidence of Wernicke encephalopathy appeared on imaging.

**Case Report/Case Presentation**

A 40-year-old female with a past medical history of hypertension, hyperthyroidism, gastroesophageal reflux disease, myocardial infarction, and morbid obesity (BMI: 48.9) was transferred from an outside hospital due worsening balance and sensory changes. The patient underwent Roux-en-Y gastric bypass surgery 2 months prior to admission and lost about 80 pounds. Weeks prior to admission to an outside hospital, the patient complained of nausea and hyperemesis in the setting of a reported GI illness. She then began to experience numbness in her inner thighs that continued to progress to worsening balance and sensory changes, described as bilateral numbness from her nipple line to her ankles. She had saddle anesthesia but experienced no bowel or bladder incontinence. Upon arrival, physical examination revealed dysesthesia from the nipple line to ankles and no nystagmus. Initial workup at the outside hospital included head CT and MRI; lumbar puncture; and MRI of the cervical, thoracic, and lumbar spine. Cervical spine MRI showed mild disc bulging at C5–6 with no demyelination, thoracic spine MRI showed disc protrusion and cord impingement at T6–7, and lumbar spine MRI showed degenerative changes with central canal stenosis at L4–5. These findings did not correlate with her current symptoms, likely making them incidental findings. The remainder of the workup was unremarkable. TSH and B12 levels were within normal limits. Lyme titer was negative.

On her fourth day in the hospital, the patient developed diplopia in addition to her symptoms, for which she was reevaluated by neurology. Physical examination was notable for multidirectional horizontal and vertical nystagmus, decreased facial expression, dysesthesia from the nipple line to ankles, bilateral lower extremity weakness more prominent on the left, diminished brachioradialis reflex, and absent knee and ankle reflexes. These findings prompted further evaluation. Repeat MRI the following day demonstrated symmetrical T2/FLAIR
signal in the area of the mammillary bodies and dorsomedial thalami, consistent with Wernicke’s encephalopathy (Fig. 1). EMG showed acute axonal polyneuropathy consistent with infiltrative, toxic, or nutritional neuropathies or acute motor axonal polyneuropathy, a rare form of Guillain-Barre syndrome. Repeat lumbar puncture showed normal white blood cells and protein, with low glucose levels in a setting of hypoglycemia prior to the procedure.

The patient was found to have significant thiamine (30 nmol/L) and vitamin A (37 μg/dL) deficiencies. Magnesium, copper, lead, zinc, selenium, vitamin E, and vitamin B6 levels were within normal limits. She was given high-dose intravenous thiamine supplementation, which improved her neurologic symptoms over the next few days. It was determined that the Wernicke encephalopathy was likely correlated to the recent gastric bypass surgery, worsened by gastric illness. She was discharged to an inpatient rehabilitation center with oral thiamine, vitamin A, and multivitamin supplements.

**Discussion/Conclusion**

Roux-en-Y is the most common gastric bypass surgery performed and has been associated with nutritional deficiencies due to smaller stomach size and bypass of absorptive areas of the small intestine [3]. Neurological complications after surgery due to nutritional deficiency, especially B vitamins, are relatively rare [10]. Thiamine (vitamin B1) deficiency is becoming a more well-known complication of gastric bypass surgery as it leads to Wernicke encephalopathy [7]. Early recognition of this disorder is important because it can progress to Wernicke-Korsakoff syndrome, a disorder of short-term memory loss, psychosis, confabulation, and gait disturbances [11]. These deficits can be irreversible and potentially fatal, so it is critical to avoid progression [12].

Wernicke encephalopathy is characterized by a classic triad of symptoms: gait ataxia, altered mental status, and nystagmus. In this case, the patient initially described only one of the three classic symptoms, gait ataxia, so Wernicke encephalopathy was not originally
suspected. Rather than altered mental status, this patient expressed flat affect and proptosis.
Visual symptoms are the least likely of the three classic symptoms to be present and can
indicate a full thiamine deficiency [13]. This patient developed diplopia and multidirectional
nystagmus while in the hospital, indicating worsening disease. In this case, additional
symptoms were present, including bilateral dysesthesias and weakness of the trunk and
lower body. This complex presentation made Wernicke encephalopathy a less clear clinical
diagnosis, which lead to a delay in treatment. In addition, the patient reported a recent history
of GI illness involving nausea and vomiting. The presence of vomiting could have been an
indicator of vitamin deficiency and should have prompted vitamin level checks, especially
considering her recent bypass surgery.

MRI of Wernicke encephalopathy typically shows bilateral hyperintensities of the
mammillary bodies, thalami, and periaqueductal area on FLAIR imaging [9]. For the patient
in this case, initial lumbar puncture and imaging of the brain and spine were unremarkable
for findings that explained her symptoms. It was around the fifth day of her hospital
admission that repeat MRI identified symmetric hyperintensities of the mammillary bodies
dorsomedial thalami, confirming the diagnosis of Wernicke encephalopathy. In addition,
this patient’s EMG findings were consistent with an acute axonal polyneuropathy. Polyra-
diculoneuropathy resembling Guillain-Barre syndrome is a rare complication of gastric
bypass surgery that has been associated with vitamin B1 deficiency from vomiting and lack
of supplementation [14]. In contrast to Guillain-Barre syndrome, cases associated with
thiamine deficiency involve normal CSF protein levels and axonal degeneration as opposed
to demyelination [15].

The initial symptoms of thiamine deficiency are often not recognized as Wernicke enceph-
alopathy, which leads to disease progression and delayed intervention [13]. In this case, diag-
nosis and treatment were delayed due to atypical patient presentation and lack of imaging
findings. Symptoms continued to progress during her hospital stay. Obtaining vitamin levels
as a part of the initial workup due to the patient’s history of gastric bypass surgery in combi-
nation with neurologic and gastrointestinal symptoms could have led to earlier recognition
of Wernicke encephalopathy. Our case highlights the fact that Wernicke encephalopathy
should not be excluded from the differential diagnosis in a patient that develops any of the
classic symptoms or has a progression of their initial symptoms, even with an unremarkable
initial workup. These patients should undergo repeat imaging if clinical suspicion is high
because workup may be initially unrevealing.

Once Wernicke encephalopathy is diagnosed or suspected, it is critical to begin imme-
diate treatment to avoid progression [12]. Prompt treatment can reverse the symptoms of
nutritional deficiencies in up to 85% of patients, but patients that develop Wernicke-
Korsakoff syndrome are less likely to fully recover [16]. Treatment includes oral supple-
mentation with 50–100 mg of thiamine 3 times daily. Patients with emesis may require
intramuscular or parenteral administration [17]. The patient in this case had thiamine and
vitamin A deficiencies. She was treated with intravenous thiamine until her neurological
symptoms improved and then was discharged to an inpatient rehabilitation unit with oral
thiamine, vitamin A, and multivitamin supplementations. For this patient, the postoper-
ative risk of Wernicke encephalopathy could have been reduced with vitamin supplemen-
tation [13].

Neurologic complications of bariatric surgery can have irreversible and possibly fatal
consequences [12]. Wernicke encephalopathy is becoming a more recognized nutritional and
neurological complication of bariatric surgery that can have devastating effects if it is not
recognized quickly [8]. Healthcare providers should be able to identify the signs and symptoms
readily in order to prevent irreversible complications. High clinical suspicion should yield a
full evaluation, even if the original workup was negative.
Statement of Ethics

Ethical approval was not required for this case report in accordance with local and national guidelines. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

This research was not funded.

Author Contributions

Sarah Glisan and Nazim Khan, MD, were part of the team that cared for the patient. They compiled relevant information and contributed to writing and editing the case report.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Lab values were extracted from the patient's chart. Further inquiries can be directed to the corresponding author.

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