



# Wernicke Encephalopathy Following Gastric Bypass

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## 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, including 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 two months after gastric bypass surgery, with additional findings of flat affect and concurrent polyradiculopathy. Her diagnosis was delayed due to atypical 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.
- 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%.
- Candidates for gastric bypass surgery include patients with a BMI >40.0 or BMI of 35.5-39.0 with a weight-related comorbidity.
- 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, 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.
- Due to decreased absorption, nutrient deficiencies are found in over a third of patients following gastric bypass surgery. These deficiencies go unrecognized in about half of patients and can be present for years after operation.
- 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.
- 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.
- MRI (magnetic resonance imaging) of Wernicke encephalopathy demonstrates bilateral hyperintensities in the area of the mammillary bodies, thalami, and periaqueductal area on FLAIR imaging.

## CASE PRESENTATION

- 40-year-old female with a past medical history of hypertension, hyperthyroidism, gastroesophageal reflux disease, myocardial infarction, and morbid obesity (BMI: 48.9)
- Chief complaint: worsening balance and sensory changes over several weeks
- Pertinent history: Roux-en-Y gastric bypass surgery two months prior to admission and lost about 80 pounds, recent nausea and hyperemesis in the setting of a reported GI illness
- Review of systems: numbness of inner thighs, progression to worsening balance and sensory changes, bilateral numbness from nipple line to ankles, saddle anesthesia with no bowel or bladder incontinence.
- Initial physical exam: dysesthesia from nipple line to ankles, no nystagmus
- Initial workup: negative head CT/MRI, lumbar puncture, and MRI of the cervical, thoracic, and lumbar spine; TSH and B12 levels within normal limits; negative Lyme titer
- Patient develops diplopia on hospital day 4, prompting neurology evaluation.
- Repeat exam: multidirectional horizontal and vertical nystagmus, decreased facial expression, dysesthesia from nipple line to ankles, bilateral lower extremity weakness more prominent on the left, diminished brachioradialis reflex, and absent knee and ankle reflexes
- Repeat MRI: symmetrical T2/FLAIR signal in the area of the mammillary bodies and dorsomedial thalami, consistent with Wernicke's encephalopathy (Figure 1)
- EMG: acute axonal polyneuropathy consistent with infiltrative, toxic, or nutritional neuropathies or acute motor axonal polyneuropathy, a rare form of Guillain-Barre syndrome
- Labs: significant thiamine (30 nmol/L) and vitamin A (37 mcg/dL) deficiencies; normal magnesium, copper, lead, zinc, selenium, vitamin E, and vitamin B6 levels
- Treatment: responded to high-dose intravenous thiamine supplementation, discharged to inpatient rehabilitation center with oral thiamine, vitamin A, and multivitamin supplements.

## DISCUSSION

- 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. Neurological complications after surgery due to nutritional deficiency, especially B vitamins, are relatively rare. Thiamine deficiency is becoming a more well-known complication of gastric bypass surgery, as it leads to Wernicke encephalopathy. 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. These deficits can be irreversible and potentially fatal.
- Wernicke encephalopathy is characterized by a classic triad of symptoms: gait ataxia, altered mental status, and nystagmus. This patient initially described only one of the three classic symptoms, gait ataxia, so Wernicke encephalopathy was not suspected. Diplopia and nystagmus had a later onset during hospitalization.
- Additional symptoms were present - bilateral dysesthesias, weakness of the trunk and lower body, flat affect, proptosis, and absent lower extremity reflexes. This complex presentation made Wernicke encephalopathy a less clear clinical diagnosis, which resulted in a delay in treatment.
- Initial workup revealed unremarkable imaging and labs. Repeat MRI on hospital day 5 identified symmetric hyperintensities of the mammillary bodies and dorsomedial thalami that were not present on arrival.
- EMG findings were consistent with an acute axonal polyneuropathy. Polyneuropathy resembling Guillain-Barre syndrome is a rare complication of gastric bypass surgery that has been associated with thiamine deficiency from vomiting and lack of supplementation. In contrast to Guillain-Barre syndrome, cases associated with thiamine deficiency involve normal CSF protein and axonal degeneration as opposed to demyelination.
- The initial symptoms of thiamine deficiency are often not recognized as Wernicke encephalopathy, which leads to disease progression and delayed intervention. In this case, diagnosis and treatment were delayed due to atypical patient presentation and lack of imaging findings.
- Once Wernicke encephalopathy is diagnosed or suspected, it is critical to begin immediate treatment to avoid progression. 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. Treatment includes oral supplementation with 50-100mg of thiamine 3 times daily.
- Obtaining vitamin levels as a part of the initial workup due to the patient's history of gastric bypass surgery in combination with neurologic and gastrointestinal symptoms could have led to earlier recognition.
- This 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.

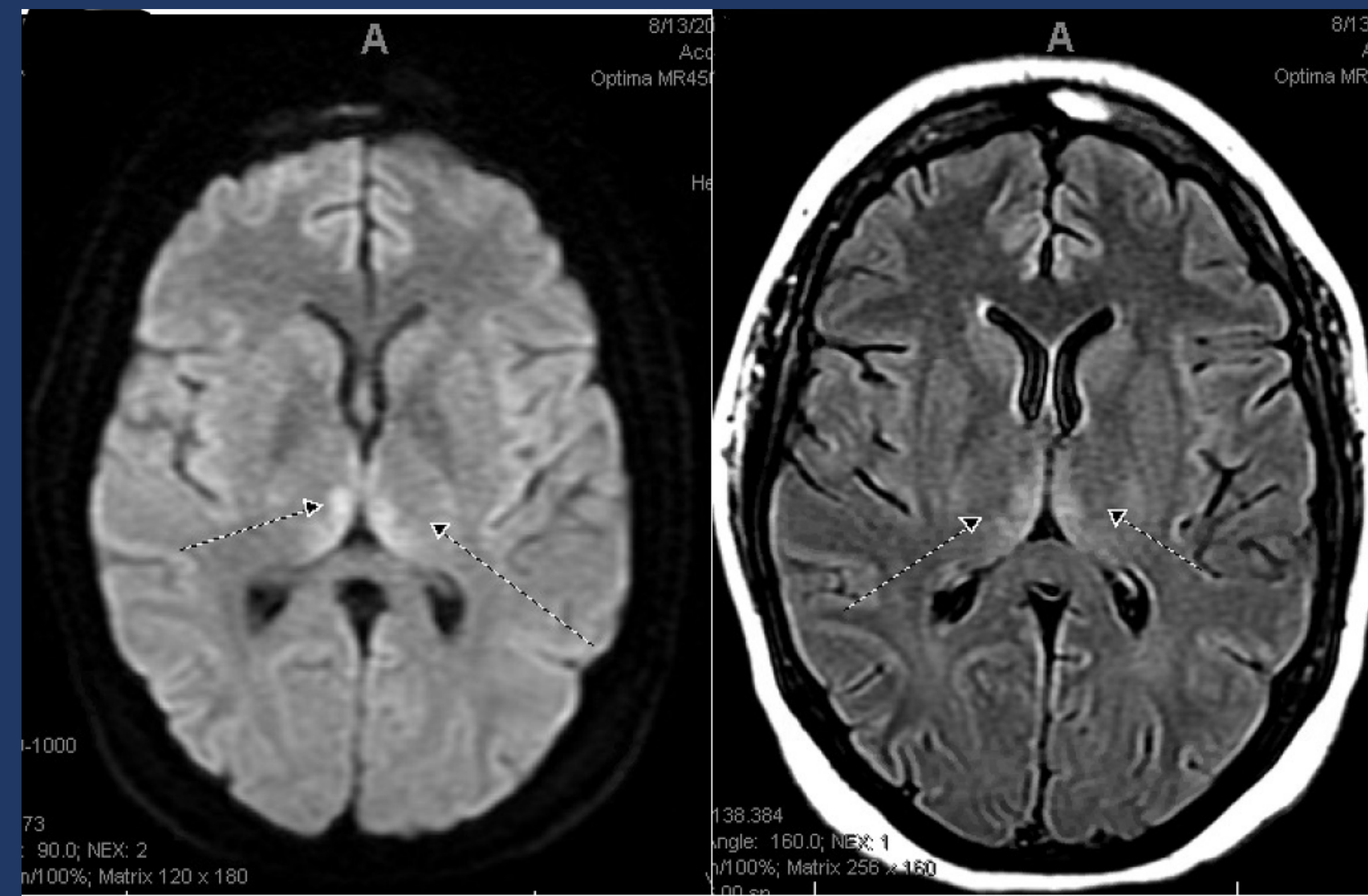


FIGURE 1.

Repeat brain MRI demonstrates symmetrical hyperintensities of the mammillary bodies and dorsomedial thalami (indicated by arrows). MRI = magnetic resonance imaging

## CONCLUSIONS

- 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.
- Healthcare providers should be able to identify the signs and symptoms readily in order to prevent irreversible complications.
- Wernicke encephalopathy should be considered in patients with any of the classic symptoms and should not be excluded with negative imaging.
- High clinical suspicion should yield a full evaluation, even if the original workup was unremarkable.

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