Diplopia after Sleeve Gastrectomy: The Canary in the Coal Mine

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ABSTRACT

Wernicke's encephalopathy (WE) is a neurologic emergency requiring timely intravenous thiamine supplementation to prevent permanent neurologic deficits. Historically, the WE diagnosis was limited to individuals with alcohol use disorder. However, it is now widely recognized to occur in patients who are chronically malnourished, post-bariatric surgery, pregnant with hyperemesis gravidarum, and with severe anorexia nervosa. Here we present a young woman who developed WE after undergoing a recent sleeve gastrectomy followed by protracted emesis for several days. This case underscores the importance of performing a thorough neurological review of systems and physical exam in high-risk patients and having a low clinical threshold to initiate appropriate thiamine treatment.

KEYWORDS: Thiamine Deficiency; Wernicke's Encephalopathy; Obesity; Bariatric Surgery; Medical Tourism

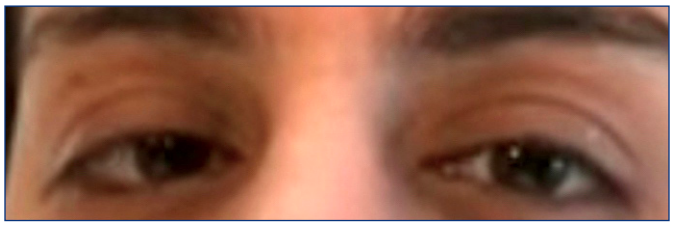
INTRODUCTION

Sleeve gastrectomy (SG) has become the most common bariatric surgery performed worldwide.[1] Its popularity arises from it being a technically simpler surgery with similar weight loss results and presumed lower postoperative risk of nutritional deficiencies compared to other bariatric surgeries.[2] However, significant nutritional deficiencies frequently occur, even with restrictive operations such as SG.[1] Here we present a cautionary tale of diplopia with progressive neurological deficits in a patient admitted with protracted emesis shortly after undergoing sleeve gastrectomy.

CASE

A 26-year-old female with class III obesity (BMI = 40) and rapid 60lb weight loss following sleeve gastrectomy in Turkey two months prior, presented with severe fatigue and three-day history of non-bloody, nonbilious emesis with inability to tolerate oral intake. Starting one month after surgery, this patient developed progressive weakness and nausea with occasional postprandial vomiting. She also described more recently two days of dizziness and altered balance without any falls and one day history of double vision, all of which she self-attributed to her decreased oral intake. She reported adherence with the postoperative bariatric diet and vitamins prescribed by her medical provider in Turkey, except for the four days prior to presentation due to emesis. She did not have any fevers or chills.

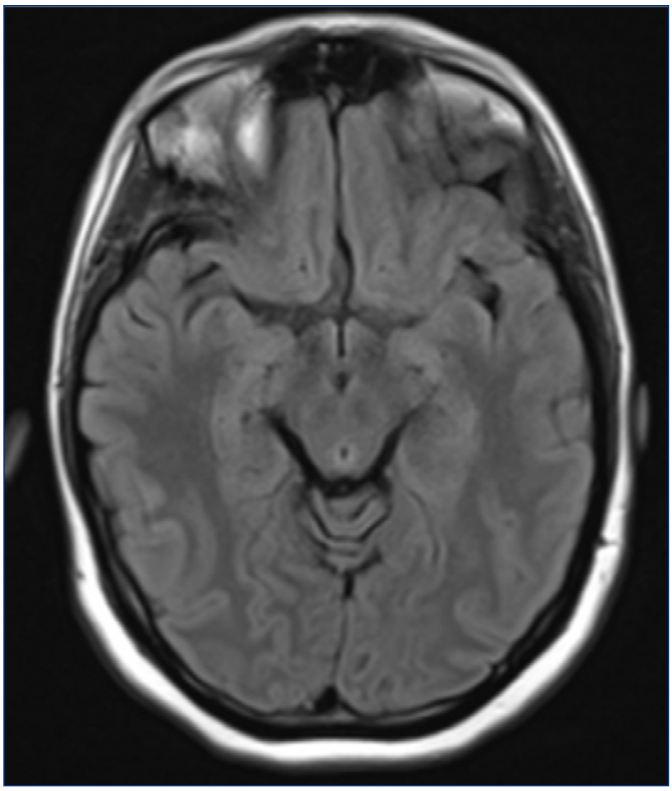
Figure 1. Inability to abduct left eye on extraocular movement exam.



On presentation, the patient was in no acute distress, well nourished, and alert and oriented to self, date, and place. New right esotropia at rest was identified prompting a more thorough neurological exam. Cranial nerve exam revealed inability to abduct her left eye (**Figure 1**) and evoked bidirectional horizontal nystagmus. Binocular diplopia was present on primary gaze and worse on leftward gaze. Visual fields were intact to frontal confrontation in all four quadrants. Cranial nerves were otherwise intact. The patient did not show signs of appendicular ataxia and gait testing was deferred. Her lab studies, Complete Blood Count (CBC), Basic Metabolic Panel (BMP) and Magnesium (Mg) were all within normal limits, except for a mildly elevated anion gap of 14 mEq/L. Computerized tomography (CT) of the abdomen/pelvis did not reveal any surgical complications. She was admitted to the general internal medicine service after receiving 2 L of IV normal saline and 200mg of thiamine IV.

A repeat neurological exam, approximately 12 hours after the first exam, was significant for progression of her ophthalmoplegia with now complete inability to abduct her eyes bilaterally, as well as a mild truncal ataxia with standing and wide-based gait. Her exam otherwise showed a negative Romberg sign and normal finger-to-nose testing. Thiamine supplementation was increased to 500mg IV every 8 hours. Magnetic resonance imaging (MRI) with and without contrast did not show areas of increased or decreased intensity, including no cranial nerve or mamillary body enhancement (**Figure 2**). CT angiogram did not show signs of anterior or posterior circulation aneurysm or stenosis.

Figure 2. MRI Axial Flair imaging showing lack of enhancement at mamillary bodies and periaqueductal gray.



After her third administration of 500mg IV thiamine, her ophthalmoplegia and ataxia had resolved, but she continued to have diplopia on left gaze. After her sixth dose of thiamine, her diplopia had completely resolved. Esophagogastroduodenoscopy (EGD) showed sharp angulation of the stomach but no functional gastric stenosis. Her ability to tolerate oral intake improved throughout the hospital stay. She was discharged with thiamine 100mg daily in addition to her bariatric vitamins. After discharge, the patient's thiamine level, drawn after receiving three doses of IV thiamine, resulted at 48 nmol/L (normal 70–180 nmol/L). Her vitamin B12 and folate were within normal limits and myasthenia gravis antibodies were negative.

DISCUSSION

This case of diplopia shortly after sleeve gastrectomy is an unusual presentation of severe thiamine deficiency with neurological involvement consistent with Wernicke's encephalopathy (WE). WE is a clinical diagnosis made when a patient has two of the following four updated Caine criteria: thiamine deficiency, oculomotor abnormalities, cerebellar dysfunction and either altered mental status or mild memory impairment. The classic Wernicke's triad of ophthalmoplegia, ataxia, and altered mental status presents in a minority of patients, raising concerns for under-diagnosis.[3] Historically, in developed countries, this diagnosis was limited to individuals with alcohol use disorder, but it is now widely recognized to occur in patients who are chronically malnourished, post-bariatric surgery, pregnant with hyperemesis gravidarum, and with severe anorexia nervosa.[1]

Thiamine pyrophosphate (TPP) is integral to the anabolic metabolism of neuronal and glial cells. Without TPP, astrocyte-related functions are impacted, leading to increased blood brain barrier permeability. Edema and accumulation of lactic acid lead to eventual neuronal necrosis I.[4] The presence of mammillary body or other diencephalic or periventricular lesions on MRI can support the diagnosis of WE, but MRI is only 53% sensitive.[5] The postulated blood brain barrier alterations from thiamine deficiency take weeks to develop on imaging, so it is not uncommon for MRI findings to lag behind the progression of the clinical picture.

Up to 25% of post-sleeve gastrectomy patients experience thiamine deficiency. However, progression to Wernicke's encephalopathy is rare, occurring in less than 1% of patients.[6,7] The majority of WE cases develop within the first 6 months after surgery but cases of post-SG WE have been reported up to six years after surgery.[8] Thiamine deficiency in non-malabsorptive restrictive weight loss surgery (gastric banding, sleeve gastrectomy, vertical gastroplasty, gastric balloon) has been attributed to preexisting nutritional deficiency, impaired thiamine absorption from gastric wall edema, and dietary non-compliance post-operatively.[7,9] In a systematic review, 84% of post-SG WE patients were women and 38% were aged 21 to 30 years old.[10] Other risk factors for development of post-SG WE are functional gastric stenosis, excessive or rapid weight loss, prolonged vomiting and those not attending an outpatient nutrition clinic.[9,11] Our patient was young, female, and had rapid postoperative weight loss. Her 60 lbs. of weight loss at 2 months was at least double the expected weight loss. She may also have been sub-clinically deficient in thiamine prior to the procedure, as a high-calorie malnutrition picture is demonstrated in 15.5–29% of patients with obesity.[9]

Consensus clinical guidelines cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery advise daily thiamine supplementation of 50 to 100mg as part of the bariatric vitamin regimen. Updated guidelines in 2019 now also recommend routine thiamine screening after bariatric surgery for all patients.[12] Due to our patient's participation in medical tourism, any preoperative nutritional screening results are not available for review, and she did not have the typical longitudinal bariatric surgical nutrition counseling.[10] The contents of her prescribed vitamins could not be confirmed. The specifics of the prescribed supplementation are vitally important as general multivitamins have been shown to be inadequate in preventing deficiencies in patients after sleeve gastrectomy.[11]

Patients with suspected thiamine deficiency should be

treated before or in the absence of imaging confirmation and monitored for signs of neurologic recovery following thiamine supplementation.[10] Strength and duration of treatment for Wernicke's is less clear, but it is widely accepted that oral supplementation is inadequate to prevent permanent disability.[14] The majority of studies have been in patients with alcohol use disorder, which may not be generalizable in the bariatric patient population. The 2019 consensus guidelines recommend changing from 200 mg IV 3 times daily to 500 mg IV 1–2 times daily for 3–5 days, followed by 250 mg IV daily for 3–5 days or until symptoms resolve. Following symptom resolution, patients should be maintained on oral thiamine supplementation of 100mg daily indefinitely or until risk factors have been resolved. In patients with recalcitrant or recurrent thiamine deficiency with one of the above risks, the addition of antibiotics for small intestine bacterial overgrowth should be considered.

In conclusion, WE is a neurologic emergency requiring timely intravenous thiamine supplementation to prevent permanent neurologic deficits. This case underscores the importance of performing a thorough neurological review of systems and physical exam in high-risk patients, including those with a history of bariatric surgery patients and protracted vomiting. As IV thiamine is a well-tolerated treatment without known independent toxicity risks, there should be a low threshold to treat patients presenting with acute neurological deficits with a history of any gastric surgery. If their condition fails to improve, supplementation should be stopped after three days. With a low threshold for clinical suspicion, this low-risk therapy can prevent long-standing neurological deficits in our progressively younger bariatric surgery patients.

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Disclosures

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