Brief Reports

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In general, authors of case reports should use the Brief Report format.

Wernicke Encephalopathy after Restrictive Bariatric Surgery

A 27-year-old female with a history of morbid obesity (body mass index of 53.8) underwent an uncomplicated laparoscopic sleeve gastrectomy (LSG). Her postoperative course was uneventful, and she was discharged on postoperative day 1.

Three months later, the patient began experiencing intermittent nausea and vomiting not associated with oral intake. Before these symptoms, she had no complaints and noted a 120-pound weight loss since her operation. In the emergency department, she received an abdominal CT scan with no significant findings. The patient was discharged from the emergency department with antiemetics, hyoscyamine, and appropriate follow-up in the bariatric clinic.

Five days later, the patient was evaluated in the bariatric surgery clinic and reported negligible improvement in her symptoms. On examination, her blood pressure was normal, though she did have mild tachycardia with a heart rate of 101 beats/min. Her mucous membranes were dry. Her systolic blood pressure decreased from 115 to 100 mm Hg when transitioned from sitting to standing. On abdominal examination, she had mild epigastric tenderness on palpation. Her laparoscopic incisions were well healed. The remainder of her examination was unremarkable. Given her inability to tolerate oral fluids and evidence of dehydration, she was admitted to the hospital for rehydration and further evaluation. Initial workup consisted of the following: an esophagram demonstrated no evidence of leak or delayed gastric emptying, a right upper quadrant ultrasound was negative for cholecystitis, and an upper endoscopy was notable for grade A esophagitis without evidence of peptic ulcer disease or obstruction.

Management with maximal intravenous antiemetic and antireflux medical therapies provided minimal symptomatic relief. Due to persistent nausea and vomiting, a peripherally inserted central catheter was placed on hospital day 7 in anticipation of initiation of total parenteral nutrition. At that time, her prealbumin level was 14 mg/dL (normal 19–38 mg/dL) and her albumin was 3.6 g/dL (normal 3.4–5.4 g/dL). Given persistent abdominal pain, an additional CT scan was performed without evidence of significant pathology or mesenteric venous thrombosis.

On hospital day 7, the patient reported new symptoms of dizziness, blurred vision, and instability. On examination, the patient was noted to have horizontal nystagmus; the remainder of her neurologic examination was unremarkable. A noncontrast head CT demonstrated no acute intracranial pathology. Her thiamine level was checked; however, this result was not available immediately at that time. Given the new physical examination findings and neurologic symptoms, she was empirically started on thiamine supplementation.

The following day, the patient reported complete resolution of her symptoms. On examination, her nystagmus had resolved. She was maintained on intramuscular thiamine and total parenteral nutrition for the following four days and then discharged home tolerating oral intake. Her thiamine level was ultimately found to be 22 nmol/L (normal 70–180 nmol/L).

Discussion

Micronutrient deficiency due to malabsorption is a well-described phenomenon in postoperative bariatric surgical patients.[1] However, clinical deficiency is generally associated with malabsorbitive procedures rather than restrictive bariatric procedures.[2] Notably, the risk of developing clinically significant micronutrient deficiencies after LSG has been thought to be so low that routine monitoring is not practiced.[3]

In 2014, Stroh reported 255 cases of bariatric beriberi.[4] Of these 255 cases, 254 were diagnosed after Roux-en-Y gastric bypass.[4] In these patients, thiamine deficiency developed between one and three months

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after surgery.[4] The authors hypothesized that the cause of thiamine deficiency was due to a number of factors including (though not limited to) poor preoperative nutritional status, surgical excision of segments of the small intestine required for thiamine absorption, poor postoperative nutritional intake, and recurrent postoperative emesis.[4]

Thiamine is a water-soluble vitamin that is primarily absorbed in the duodenum and proximal jejunum *via* carrier-mediated active transport.[5] Because thiamine is an essential nutrient, is water soluble, and has a limited intracellular reserve (approximately 30 mg), constant supplementation is critical.[6] Thiamine diphosphate, the biologically active form of thiamine, acts as a cofactor for the enzymes pyruvate dehydrogenase, α-ketoglutarate dehydrogenase, transketolase, and branched-chain α-ketoacid dehydrogenase, all of which are involved in carbohydrate and amino acid metabolism.[7] Thiamine also assists in the regulation and activation of sodium and potassium ion movement in nerve and muscle cells.[4]

Thiamine deficiency impairs metabolism and function of astrocytes responsible for maintaining the integrity and functionality of surrounding neurons.[7] The result of this metabolic dysfunction leads to intra- and extracellular edema, neuronal loss, microhemorrhage, and proliferation of microglial cells involved in scar formation.[8] These changes tend to occur in the mammillary bodies, the paraventricular periaqueductal gray matter, the medial thalami, and other structures around the third ventricle.[9] Symptomatically, thiamine deficiency manifests as Wernicke encephalopathy (WE). The classic triad of WE consists of confusion, ataxia, and eye movement abnormalities.[10]

A diagnosis of WE constitutes a medical emergency, and prompt treatment is required to avoid death or the development of Korsakoff's syndrome. Symptoms of Korsakoff's syndrome include profound deficits in anterograde and retrograde memory as well as confabulation.[11] Importantly, WE is a clinical diagnosis. Although laboratory analysis is useful to help in confirmation, treatment should be prompt and empirically initiated, as a delay in diagnosis is associated with incomplete recovery.[4]

Treatment for WE involves immediate administration of thiamine. Minimal data exist to guide the optimal dosing, frequency, and duration for thiamine treatment of WE patients.[12] However, one presently accepted regimen includes 500 mg of thiamine administered parenterally three times daily for a minimum of three days.[6]

Cases of WE after restrictive procedures such as LSG are rare. In 2014, Milone et al.[2] noted only 16 documented cases of WE after restrictive bariatric surgery. The reviewers noted persistent vomiting to be the most important factor in the development of WE in these patients. However, in 2014, Saab et al.[13] reported a case of WE after LSG in the absence of intractable emesis, suggesting that intractable emesis is only one of many factors contributing to WE in postoperative LSG patients.

Our case supports the theory that persistent emesis significantly increases the incidence of thiamine deficiency in patients after LSG. Although restrictive bariatric procedures have historically been associated with a lower risk of micronutrient malabsorption, it is important to recognize that deficiencies may occur after any type of bariatric surgery. In particular, clinicians must consider thiamine deficiency as a possible etiology for altered mental status in any bariatric surgery patient with prolonged episodes of emesis, even in patients with restrictive procedures. Early recognition and treatment are critical to avoiding the dangerous and potentially permanent sequelae of WE.

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