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CASE REPORT

Dry Beriberi Post Roux-en-Y Gastric Bypass Surgery

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Abstract

Bariatric surgery is an effective strategy for achieving substantial weight loss, prolonging survival, and improving the comorbidities associated with obesity. Nutritional deficiency is a commonly recognized post-procedural complication. Here, we present a case of a patient with paresthesia, lower extremity weakness, and altered mental status one year following Roux-en-Y gastric bypass, who was found to have multiple vitamin and micronutrient deficiencies and was diagnosed with beriberi in the setting of profound thiamine deficiency.

Keywords: Beriberi, Thiamine deficiency, Roux-en-Y bypass surgery, Malabsorption, Paresthesia, Altered mental status

1. Introduction

Obesity remains a major public health concern accounting for significant morbidity and mortality.[1] The National Health and Nutrition Examination Survey (NHANES 2021), carried out from March 2017 to March 2020, noted that the prevalence of obesity in the US approached nearly 50% of the population with a significant increase in the prevalence rate year over year.[2] This increased prevalence is directly associated with several psychosocial determinants of health as well as multiple medical comorbidities - hypertension, type 2 diabetes mellitus, dyslipidemia ischemic heart disease, cerebrovascular disease, obstructive sleep apnea, gastroesophageal reflux disease, cholelithiasis, nonalcoholic fatty liver disease (NAFLD), osteoarthritis and malignancy (breast, colon, endometrium, kidney, and esophagus).[3] Obesity is also associated with a significant economic burden resulting in substantial medical expenditures and rates of absenteeism among full-time employees.[4,5] The US healthcare system spends approximately $173 billion per year on obesity and obesity related sequelae.[2]

Bariatric surgery is an effective therapeutic option for obesity management[6]; an estimated 200,000 bariatric interventions are performed per annum in the United States.[7] A myriad of possible surgical interventions exist - gastric bypass surgery, sleeve gastrectomy, gastric banding, and biliopancreatic diversion[8] - each characterized by a variable degree of invasiveness, safety profiles, and adverse events (Table 1)[42] with the therapeutic effect (weight loss) achieved through a variety of mechanisms (Table 2).[9] Gastric banding is a restrictive procedure that limits food intake by decreasing the natural capacity of the stomach while leaving the rest of the gastrointestinal tract intact.[10] Malabsorptive procedures such as biliopancreatic diversion and sleeve gastrectomy limit the amount of nutrients absorbed by bypassing a portion of the small intestine.[11] Roux-en-Y gastric bypass (RYGB) is a combination of both restrictive and malabsorptive procedures that involves the restriction of gastric capacity due to the creation of a small gastric pouch that holds less food and malabsorption due to the bypass of small intestine segments.[12-14] RYGB was associated with better long-term weight loss when compared with other bariatric procedures.[15,16] Post-operative malabsorption remains a major complication of malabsorptive surgical techniques due to surgical bypass of regions of small intestine and diversion of biliopancreatic secretions limiting the absorption of nutrients.[17] This may result in hypoglycemia, multivitamin deficiency, caloric and protein malnutrition, trace metal (iron, copper, zinc, calcium) deficiencies, and dumping syndrome.[12] Protein and calorie malnutrition are less

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Table 1. Comparison between various approaches of bariatric surgery.[42]

Features	Approaches of bariatric surgery			
	Gastric band	RYGB	Sleeve gastrectomy	BPD ± DS
Safety	+++	++	++	+
Durability	+++	+++	?	+++
Adverse effects	+	++	+	++
Effectiveness	++	++	++	+++
Reversible	Yes	No	No	No
Adjustable	Yes	No	No	No
Minimally invasive	+++	++	++	+
Low revision rate	+	+	Questionable	+
Requirement of follow up	+++	++	+	++

RYGB: Roux-en-Y gastric bypass, BPD: Biliopancreatic diversion, BPD + DS: biliopancreatic diversion with duodenal switch.

common after RGYB surgery; however, thiamine and trace mineral deficiencies are common due to bypass of the duodenum and proximal jejunum where these vitamins and minerals are absorbed.[12,18]

Malabsorption of thiamine results in impaired glucose metabolism and ATP production, reduction in the distribution of oxygen by red blood cells, cardiac dysfunction, failure of neurotransmission, and neuronal death.[18,19] Thiamine deficiency disrupts energy supplies to neurons thus increasing oxidative stress, induces alteration in neurotransmission (mainly Glutaminergic and GABAergic system) resulting in a neuro excitatory state, and causes derangement in the pentose phosphate pathway decreasing neuronal myelination and signaling.[20,21] Eventually, this leads to impairment of the central and peripheral nervous system, manifesting as symmetrical sensorimotor neuropathy in distal extremities,[22] known as dry beriberi. Wernicke's encephalopathy is an acute form of dry beriberi; symptoms are classically described as a triad of ataxia, ophthalmoplegia/nystagmus, and confusion.[23] Identification of pre-existing nutritional deficiencies by adequate preoperative screening and post-procedure prophylactic nutrition supplementation are critical to avoid major post-procedural neurological complications.[24]

2. Case presentation

A 43-year-old woman presented to the emergency department with gradual onset, progressively worsening bilateral lower extremity weakness and paresthesia of three months duration. There were no associated exacerbating or alleviating factors, no exposure to new medications or change in baseline dosing of pre-existing medications, and no recent travel, exposures, or insect bites prior to the onset of her symptoms. Over the course of the past week, the patient's family members had also noted a new onset waxing and waning mental status, characterized by intermittent episodes of confusion and lack of clear communication (slurred speech and dysarthria). She denied bowel or bladder dysfunction, myalgias, or back pain but had noticed numerous areas of new hyperpigmentation localized to the lower back, inframammary area, and bilateral upper extremities over the course of the last month (Fig. 1). The areas were not pruritic, erythematous, or irritating but were associated with some scaling and flaking of the epidermis. She was a non-smoker and denied using any recreational drugs or alcohol.

Medical history was notable for Graves' disease status post radioactive-iodine ablation therapy, Protein S deficiency complicated by left lower extremity deep vein thrombosis, fibromyalgia, and obesity. Surgical history was remarkable for a RYGB surgery one year prior with post-operative vitamin (vitamin A, vitamin D, and niacin) deficiencies, even though her preoperative nutrition evaluation was unremarkable. Her home medications included levothyroxine, rivaroxaban, oxycodone, ferrous sulphate 325 mg every other day and multivitamins

Table 2. Cause of weight loss after bariatric surgery.[42-44,46]

1. Reduction in appetite, induction of satiety
2. Alteration in the taste of food, mainly decrease in the preference for sweet taste
3. Restricted food intake due to diminished gut's capacity
4. Diversion of nutrients from duodenum
5. Malabsorption of nutrients due to reduction of gastric secretions, and bypass of absorption surface areas
6. Increment in energy expenditure by increased brown adipose tissue activity postoperatively
7. Gastrointestinal remodeling: increased number of GLP-1 producing cell

   :enhanced glucose sensing
   :increased intestinal glucose metabolism

8. Aversion effect—dumping, steatorrhea, vomiting

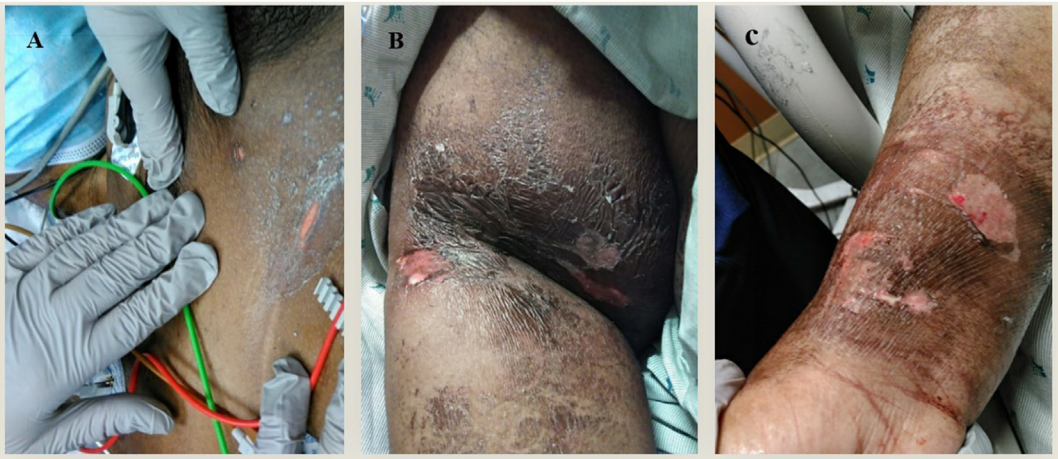

Fig. 1. Pigmented dry patches in the skin with crusting and areas of erosion in (A) left inframammary area (B) right anterior elbow, and (C) right anterior wrist.

(vitamin A 10, 000 IU daily, pyridoxine 50 mg daily, folic acid 1 mg daily, cholecalciferol 5000 IU, vitamin E 100 IU daily). She was taking multivitamins regularly for the last one year and was on six monthly follow up with bariatric surgery and nutritionist.

On presentation, the patient was noted to have sinus tachycardia (119 beats per minute), but was normotensive, with a preserved respiratory rate and oxygen saturation on ambient air. Examination revealed a well-appearing, obese woman resting comfortably in bed. Her cardiopulmonary examination was unremarkable except for the noted sinus tachycardia and 1+ pitting edema to the knees bilaterally. Angular stomatitis and patchy areas of well-circumscribed, hyperpigmented, dry, scaly, plaque-like lesion with areas of erosion localized to the inframammary area and mid-back, extending to the bilateral buttocks, medial thigh, and groin were appreciated. Neurological examination demonstrated an alert and orientated female without ophthalmoplegia, nystagmus, or focal cranial nerve deficits. Examination was notable for decreased proprioception of the bilateral lower extremities, bilateral lower extremity paresthesia, an absence of bilateral ankle and knee reflexes, and reduced strength of the bilateral lower extremities (3/5); strength and sensation were preserved in the bilateral upper extremities. Initial laboratory diagnostics (Table 3) demonstrated a macrocytic anemia (hemoglobin 6.8 gm/dl, reference range: 11.0−14.5 gm/dl; MCV104 fL, reference range: 81−100 fL), with a normal white blood cell and platelet count, with an unremarkable differential. Ferritin was elevated; iron studies failed to demonstrate iron deficiency. Metabolic panel was notable for hypoalbuminemia and a mild transaminitis. Ammonia level was elevated (76 umol/L, reference range 11−32 umol/L) and she was initiated on lactulose and rifaximin, given concern for hepatic encephalopathy contributing to her altered mentation.

Table 3. Laboratory investigations.

Parameters	Normal range	Laboratory results
Hemoglobin	11.0−14.5 gm/dL	6.8 gm/dL
MCV	81−100 fL	104 fL
Iron level	50−170 mcg/dL	58 mcg/dL
Iron saturation	54%	15−50%
TIBC	107 mcg/dL	50−170 mcg/dL
Ferritin	416 ng/ml	7.3−270 ng/ml
Blood ammonia level	11−32 Umol/L	76 Umol/L
AST	0−33 units/L	109 units/L
ALT	10−49 units/L	73 units/L
Alkaline phosphatase	46−116 units/L	176 units/L
Total bilirubin	0.3−1.2 mg/dl	2.2 mg/dl
Total protein	5.7−8.2 gm/dl	5.6 gm/dl
Albumin	3.2−4.8 gm/dl	1.8 gm/dl
Creatinine	0.50−0.80 mg/dl	0.74 mg/dl
ANA	Negative <1:40	Positive (Homogeneous pattern), 1:320
RF	0−14 IU/ml	<4 IU/ml
Free Kappa	3.3−19.4 mg/L	71.59 mg/L
Free Lamda	5.71−26.3 mg/L	60.39 mg/L
Free K/L ratio	0.26−1.65	1.19
Vitamin A (Retinol)	0.30−1.2 mg/dl	<0.06 mg/dl
Vitamin A (Retinyl palmitate)	0.00−0.1 mg/dl	<0.02 mg/dl
Vitamin B1	70−180 nmol/L	41 nmol/L
Vitamin B6	20−125 nmol/L	18.5 nmol/L
Vitamin B12	211−911 pg/ml	1421 pg/ml
Vitamin E	5.5−18 mg/L	4.1 mg/L
Copper level	80−155 mcg/dl	41 mcg/dl
Ceruloplasmin level	16−45 mg/dl	15 mg/dl
Zinc level	60−120 mcg/dl	20 mcg/dl

MCV: Mean cell corpuscular volume, TIBC: total iron binding capacity, RF: Rheumatoid factor.

A review of prior imaging and repeat imaging on presentation (Computed Tomography of the abdomen and pelvis) demonstrated hepatic steatosis without evidence of cirrhosis or stigmata of portal hypertension. In the setting of lack of clinical improvement following initiation of therapy, her intermittent episodes of encephalopathy (described as a waxing and waning mental status by family and alternating episodes of consciousness and alertness during her inpatient stay), as well as her associated weakness and paresthesia were felt to be unrelated to the noted hyperammonemia and thought to be related to her narcotic use for management of fibromyalgia.

Given this dichotomy, additional laboratory diagnostics were ordered. Serum protein electrophoresis, urine protein electrophoresis, kappa/lambda light chains, and Rheumatoid Factor were negative. Antinuclear antibody was positive in a homogenous pattern (1:320), with a negative double-stranded DNA antibody. Lumbar puncture was unremarkable. Additional diagnostic imaging including an MRI of the spine was unremarkable. Continuous EEG demonstrated no evidence of epileptiform activity but was notable for diffuse generalized slowing and intermittent generalized triphasic waves, suggestive of a diffuse metabolic encephalopathy. She also underwent skin punch biopsies for suspicious small fiber neuropathy, which was notable for benign skin with focal mild superficial dermal chronic inflammation without significant histologic abnormality. Due to her history of bariatric surgery, prior history of vitamin deficiencies (niacin, vitamin A, and vitamin D), and physical examination findings (polyneuropathy, weakness, confusion, angular stomatitis, and hyperpigmented keratosis), her presentation was felt to be consistent with a nutritional deficiency, specifically a combination of niacin, thiamine, and copper. Vitamin and micronutrient panel was ordered, and the patient was initiated on high-dose thiamine repletion with gradual improvement in her encephalopathy, paresthesia, and lower limb weakness over the course of the next 3 days. Laboratory diagnostics returned in 1 week and confirmed multiple vitamin/micronutrient deficiencies - vitamin A, thiamine, vitamin B6, vitamin E, copper, and zinc (Table 3). Serum ceruloplasmin was low. Folic acid and vitamin B12 were preserved. She was initiated on multiple vitamin supplements (A, D, E, B6, and copper). Repeat serum thiamine levels were collected and demonstrated improvement to 179 nmol/L from a nadir of 41 nmol/L (reference range 70−180 mmol/L), and she was transitioned to oral thiamine supplementation with the continuation of her adjunctive supplements on discharge. At the time of discharge, the patient was fully alert and oriented and appropriately followed commands. She noted improvement of her paresthesia and examination demonstrated resolution of proprioceptive deficits as well as improved lower extremity weakness. She was discharged to subacute rehabilitation.

3. Discussion

In adults, obesity is defined as a BMI of 30 kg/m^2 or greater[8]; its prevalence has remarkably increased in the US over the last 30 years.[25,26] The National Health and Nutrition Examination Survey (NHANES, 2021) recorded the prevalence of obesity in the US as 39.8% among adults aged 20−39 years, 44.3% among adults aged 40−59 years, and 41.5% in adults aged 60 and older.[2] Obesity is strongly associated with an elevated risk of multiple comorbid conditions − hypertension, type 2 diabetes mellitus, obstructive sleep apnea, metabolic syndrome, cardiovascular disease, malignancy (cancers of the breast, colon, endometrium, kidney, and esophagus) - as well as increased all-cause mortality.[3,19,27,28] Furthermore, a tremendous stigma is also associated with obesity in social areas such as education, employment, and health care.[3] In a group of 294 individuals pursuing consultation for bariatric surgery, half were found to have a psychiatric disorder (somatization, social phobia, hypochondriasis, and obsessive-compulsive disorder.[3] Notably, a substantial proportion of healthcare dollars is devoted to management strategies for obesity and obesity-related comorbidities,[29] totaling nearly $173 billion a year.[2] A cross-sectional analysis of retrospective data from the 2001−2016 Medical Expenditure Panel Surveys showed that adults with obesity in the United States, compared with those with normal weight, experienced higher annual medical care costs by $2505 or 100%, with costs surging remarkably with the class of obesity, from 68.4% for class 1−233.6% for class 3.[30]

Lifestyle modifications like a healthy diet, exercise, and behavioral change remain the initial management strategies for obesity, however "step-up" therapy should be considered if there is failure of conservative measures. Current pharmacologic therapies add little benefit to the management of obesity.[9] In contrast, bariatric surgery is the most efficacious and sustainable treatment option for clinically severe obesity[8]; it is associated with a 42% reduction of cardiovascular risk and a 30% reduction in all-cause mortality.[31] Current guidelines for bariatric surgery include a BMI greater than 40 kg/m^2 or greater than 35 kg/m^2 in the presence of comorbid disease conditions such as type 2 diabetes

mellitus.[6] Bariatric surgery is also indicated when a treatment goal of 10−20% weight loss in a patient with BMI 35−40 kg/m^2 or 10−30% weight loss in a patient with BMI more than 40 kg/m^2 cannot be maintained in 6−12 months of conservative therapy.[6]

There are a variety of bariatric surgery techniques - open/laparoscopic gastric bypass, sleeve gastrectomy, laparoscopic adjustable gastric banding, and biliopancreatic diversion with or without duodenal switch.[8] Vertical sleeve gastrectomy and Roux-en-Y gastric bypass (RYGB) remain the two most popular procedures and involve the removal of most of the stomach along the greater curvature or the creation of a small gastric pouch with adjunctive bypass of the intestinal tract, respectively.[32] Alterations to gastrointestinal anatomy during bariatric surgery result in neurological and physiological changes affecting hypothalamic signaling, gut hormones, bile acids, and gut microbiota, which coalesce to exert a profound influence on eating behavior.[33]

Hyperammonemia after gastric bypass surgery is underrecognized, potentially fatal but treatable complication[34]; our patient demonstrated evidence of hyperammonemia on initial labs, a feature that was suspected to be the underlying etiology of her altered mentation. Ammonia may cross the blood brain barrier leading to neurological disorder including episodic irritability, ataxia, mental retardation and ultimately alteration of consciousness and coma.[35] The pathophysiology of hyperammonemia after gastric bypass surgery is poorly understood[36] but is hypothesized to be related to increased catabolism, leading to protein breakdown in extrahepatic tissues, thus increasing the burden of nitrogenous waste in liver.[34] Likewise, bypass surgery is thought to interfere with citrulline synthesis in enterocytes, resulting in depletion of urea cycle components, further contributing to hyperammonemia.[34] The blind gastric-small bowel pouch after the procedure is also associated with alterations in gut microbiota, favoring urealytic bacterial strains and subsequently increased ammonia production.[34]

Acute nutritional disturbances remain a major complication in the post-operative period.[37] RYGB and biliopancreatic diversion result in anatomic changes to the gastrointestinal tract[24]; the prevalence of nutritional deficiency is 30−70% after bariatric surgery.[33] Hypoglycemia occurs in 64−82% of patients during the first five years of bariatric surgery and is thought to be in the context of improved B cell mass and function, reduced level of ghrelin, improved insulin sensitivity, and failure of counter-regulation.[33] Micronutrients, trace metals and electrolytes are absorbed at the specific sites of small intestine; bypass or surgical removal of these sites reduces the total effective absorptive area predisposing to multiple micronutrient and vitamin deficiencies - vitamin B1, vitamin B-12, vitamin D, thiamine, folate, iron, calcium, copper and fat-soluble vitamins.[24]

Vitamin B1 (thiamine) is a water-soluble vitamin that serves as a coenzyme in the oxidation of alpha-keto acids and 2-keto sugars; it is rapidly absorbed in the proximal jejunum by active transport and passive diffusion.[15,16,38] Thiamine has an obligatory role in the Krebs cycle for ATP production as well as in the Pentose phosphate pathway for the synthesis of neurotransmitters, nucleic acids, steroids, amino acids and glutathione.[19] Thiamine deficiency is associated with several medical conditions including alcoholism, diuretic use, diets high in carbohydrate food, and malabsorption following bariatric surgery, which can exhaust the thiamine reserve in 2−3 weeks as our body does not have an ample thiamine store.[19,38-40] Risk factors for the development of thiamine deficiency after bariatric surgery include prolonged gastrointestinal symptoms (intractable vomiting, diarrhea, dumping syndrome), rate and amount of weight loss, subclinical preoperative vitamin deficiency, postoperative loss of appetite, inadequate vitamin repletion, glucose administration without thiamine and bacterial overgrowth.[41] Its deficiency can lead to cardiovascular complications like heart failure (wet beriberi) or neurological complications like peripheral neuropathy, gait ataxia, muscle weakness, paresthesia, and Wernicke's encephalopathy (dry beriberi).[19] The classical triad of Wernicke's encephalopathy − change in mental status, ocular dysfunction, and gait apraxia is estimated to be present in only 10% of cases.[42] Korsakoff syndrome typically involves impairment of the anterior thalamic nucleus and medial mamillary nucleus of the hypothalamus.[43] If untreated, Wernicke's encephalopathy can progress to coma and death.[41]

Vitamin B1 deficiency after bariatric surgery commonly manifests as dry beriberi.[44] A study by Punchai et al. in 2017 evaluated 47 patients who underwent bariatric surgery and found 30 patients with thiamine deficiency and 12 with vitamin B12 deficiency developed a myriad of overlapping neurologic manifestations - paresthesia (66%) followed by muscle weakness (33%); however, Wernicke encephalopathy was a very rare manifestation occurring in only 6% of the patients.[44] Although paresthesia is common, symptomatic dry beriberi is a poorly recognized complication of thiamine deficiency seen in only 0.0002−0.4% of patients due to

multiple mimics of thiamine deficiency causing paresthesia and muscle weakness.[45] Given this noted symptomatic overlap, symptoms of thiamine deficiency are often indistinguishable from those occurring with other nutritional deficiencies including copper, vitamin E, and vitamin B12 deficiency.[41] Neuropathies secondary to nutritional deficiencies are length-dependent sensory axonopathies, except for vitamin B12 deficiency (a non-length dependent sensory neuropathy.[46] Vitamin B12 and copper deficient neuropathy have concordant myelopathy, however vitamin E deficient neuropathy is characteristically associated with spinocerebellar syndrome.[46] Thiamine and vitamin B12 deficiency are implicated to cause neuropathy along with encephalopathy, whereas copper and vitamin B12 are implicated causing neuropathy along with myelopathy.[41] Folate and niacin rarely cause encephalopathy and pyridoxine, folate, niacin, vitamin E rarely cause neuropathy.[41] Clinically evident neurologic complications related to vitamin B12 deficiency occurs after 2–5 years even with severe malabsorption and those related to copper deficiency may take several years (up to two decades) to develop.[41] In one case study by Kumar et al., clinical features of copper deficiency were reported in a patient 24 years after intestinal bypass surgery.[41] It is important to consider additional diagnostic clinical features of copper deficiency which are absent in vitamin B1 deficiency such as anemia, thrombocytopenia, or neutropenia when evaluating a patient with presumed nutritional deficiency.[47]

The patient described in this case report had a Roux-en-Y gastric bypass surgery and presented with lower limb motor weakness, paresthesia and encephalopathy, a potentially fatal condition with a wide range of differential diagnoses. Early diagnosis of thiamine deficiency, while suspected, remained challenging given the noted hyperammonemia as well as reported compliance with outpatient multivitamin supplementation, a feature that was further compounded by the lag time of laboratory results. Furthermore, the patient failed to demonstrate typical features of Wernicke encephalopathy. Her new skin lesions prompted rheumatologic workup, which demonstrated the presence only of a mildly positive, and non-specific ANA. Her rapid recovery following aggressive thiamine supplementation corroborated our findings and prevented more devastating consequences. Suspected thiamine deficiency should be aggressively treated with high doses of intravenous thiamine, even before lab results of thiamine levels.[41] The standard dose of thiamine is 500 mg intravenously every 8 h for one day followed by 100 mg intramuscularly for five days and then permanent oral maintenance of 50 mg/day or 100 mg/day.[48] Symptoms of dry beriberi may persist for weeks to months after replacement of thiamine.[47] Glucose infusion should be avoided before the replacement of thiamine because glucose may consume available thiamine and precipitate acute Wernicke encephalopathy.[49]

4. Conclusion

As the incidence of bariatric surgery in the US is on the rise, medical complications of nutrient deficiencies including dry beriberi are increasingly being recognized. It is important to identify thiamine deficiency as a differential diagnosis for peripheral neuropathy, lower limb Weakness and encephalopathy in the context of bariatric surgery as high-dose thiamine supplementation is a relatively inexpensive and rapidly effective treatment which prevents adverse outcomes. Adequate patient education, monitoring of the nutrient level, adequate mineral and nutrient supplementation and regular follow up with dietician and nutrition team is crucial for prevention of serious complications after bariatric surgery.

Conflict of interest

None.

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