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Severe Micronutrient Deficiencies in RYGB Patients: Rare but Potentially Devastating



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Roux-en-y gastric bypass is both a restrictive and malabsorptive weight loss procedure. The malabsorptive process is not selective to macronutrients alone, and if not carefully monitored, vitamin and mineral deficiencies can and will occur in varying degrees post-operatively. What is becoming more apparent is that some patients suffer from devastating side effects of these deficiencies. Predisposing factors may include nausea, vomiting, excessive weight loss, non-compliance with vitamin supplements and/or diet, and even preexisting vitamin and mineral deficiencies unappreciated pre-operatively. It is unclear if these severe deficiencies are actually rare, unchecked, or underreported.

Two Cases

A 39 year old was admitted to University of Virginia Health System (UVAHS) for a gastrostomy tube placement 4 months after a Roux-en-Y gastric bypass (RYGB). She had experienced persistent nausea and vomiting, poor oral intake and an excessive weight loss of 100 pounds in 5 months. Her hair was falling out, but she experienced no peripheral neuropathy, ataxia, numbness or tingling in extremities, or confusion. She was unable to consistently tolerate her multivitamin due to the nausea and vomiting.

Five days later, another RYGB patient who was 20 years post op, was admitted with dysphagia, weight loss, poor oral intake and scarring at the gastrojejunostomy

anastomosis. She had been unable to tolerate any solid food for the past 6 months and consumed only 2 cans of Ensure per day along with some gingerale. She experienced a 21% weight loss during this time. She had a revision of her previous bypass and dilation of her gastrojejunostomy along with placement of a feeding jejunostomy tube. She admitted to leg pain, numbness and tingling and was diagnosed with “neuropathy” when admitted several months prior to another hospital. She was unable to tolerate any vitamins during this time.

These are examples of two different, yet increasingly common admissions to our hospital. What vitamin and mineral deficiencies should we be looking for? What other signs and symptoms would you consider? If serum levels are low, how would you replace these deficiencies? The following article attempts to address these questions.

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INTRODUCTION

As the rate of obesity in the United States continues to climb, so do the number of gastric bypass procedures. In a 6 year period (2003-2009), the number of Roux-en-y gastric bypass procedures more than doubled to a staggering 220,000 surgeries per year (1). During a RYGB procedure, the most commonly performed, a 15-30 mL pouch is created and directly attached to the jejunum in a Roux-en-Y configuration via a narrow anastomosis. The length of the roux limb may vary from 50-150 cm depending on the patient's BMI (2). The longer the roux limb (> 75 cm), the greater will be the malabsorption.

There are many studies in the literature that discuss nutrient deficiencies after a roux-en-y gastric bypass (RYGB). Most of these studies, however, focus on the first few years post operatively and typically cover the more common deficiencies like iron, vitamin B12 and folate. What is lacking in the literature is a review of deficiencies that occur in the RYGB patient who fails to thrive and has devastating effects from profound malnutrition including vitamin and mineral deficiencies that arise months to years after surgery. Over the course of the past year, at least 12 patients have been admitted to University of Virginia Medical Center with life altering, life threatening deficiencies. This article will review what little data exists and make practical suggestions for the clinician based on our experience to date.

In a study by Toh, et al (3), 1 year post RYGB follow up laboratory results demonstrated that patients suffered from low levels of ferritin (15%), B12 (11%), RBC folate and vitamin D (12%). In one outpatient clinic, an extensive list of labs were analyzed in 121 RYGB

patients at 6, 12, and 24 months (4). Normal levels of iron, calcium, vitamin D, and vitamin B12 were found at all intervals. However, 20% were deficient in zinc, 23% in selenium, and 35% in vitamin A at 6 months postoperatively. Those numbers decreased to 6%, 12%, and 17% respectively at 24 months after taking a vitamin and mineral supplement. Actual amounts of supplementation were not disclosed. Dalcanele, et al found the following deficiencies in 75 patients at least 5 years post RYGB surgery: Magnesium (32%), zinc (40.5%), B12 (61.8%), vitamin D3 (60.5%) and B-carotene (56.8%). Interestingly, only 33% of patients reliably took a multivitamin supplement and 16% never took one (5).

Strohmayer, et al suggests routine screening for micronutrient deficiencies every 6 months for the first year and then yearly thereafter, including iron, vitamin B12, folate, vitamin D and possibly vitamin A (6). Multivitamins, in addition to the individual micronutrients that are deficient, should be supplemented with levels monitored regularly.

But what happens when the patient fails to return for follow up appointments and presents to a clinic or hospital with one (or many) signs and symptoms of life altering micronutrient deficiencies such as hair loss, muscle pain or weakness, night blindness, neuropathy, anemia, ataxia, or optic neuropathy? What vitamin and mineral levels should be considered? See Table 1.

Vitamin A

Vitamin A is a fat soluble vitamin stored in the liver. It is involved in immunological activity, cellular proliferation and plays a role in visual acuity (4).

Table 1. Deficiency-associated Symptoms

Symptoms	Potential Deficiency
Visual Impairment	Vitamin A, copper, vitamin E, thiamine
Gait disturbance	Vitamin E, B1, B12, copper, niacin
Neuropathy	Copper, vitamin E, thiamine, B6
Skin disorder/dermatitis	Niacin, vitamin A, zinc, B2, B6
Glossitis/cheilitis/stomatitis	Vitamin C, zinc, B2, B6

(continued on page 17)

(continued from page 14)

Although vitamin A deficiency occurs more often in malabsorptive procedures like the biliopancreatic diversion (61-69% of patients), Clements, et al reported a deficiency in 11% of patients based on serum levels at a one year follow up and 8.3% at a two year follow up visit post RYGB (7). The difference was attributed to improved intestinal absorption due to adaptation and increased intestinal surface area over time; it is also possible that fewer patients (n = 33 vs. 11) were available for the two year follow up visit. Similarly, Brodin found a deficiency in 10% of patients at a 4 year follow up visit (8). Common symptoms of vitamin A deficiency include poor nocturnal vision, dry skin, dry hair, pruritus, decreased visual acuity and reduced resistance to infection (9).

Despite low plasma levels, actual clinical symptoms of deficiency are rare especially in the RYGB population. One case report describes a 61 year old with a 9-month history of progressive night blindness. His past history included a gastric bypass 32 years prior to his complaint of poor night vision (10). His vitamin regimen post surgery included vitamins E, C and K, but no vitamin A. A serum vitamin A level was found to be low at 11 mcg/dl (normal 38-106 mcg/dl). The patient was treated with 50,000 IU of oral water-soluble vitamin A daily. Self-reported improvement in night vision occurred 1 month after treatment and his serum level rose to 25 mcg/dl. Two and a half months after vitamin A treatment, the serum level was normal at 44 mcg/dl and there was marked improvement in vision.

Koch et al recommends 10,000 IU oral vitamin A daily to treat a deficiency (9). The American Society for Metabolic and Bariatric Surgery (ASMBS) suggests 10,000-25,000 IU/d orally with no corneal changes for 1-2 weeks until clinical improvement and 50,000-100,000 IU intramuscularly with corneal changes for 3 days followed by 50,000 IU IM for 2 weeks (11). Toxicity can occur with daily doses of > 50,000 IU longer than 3 months. In addition, a zinc deficiency should also be considered this type of presentation, as it may alter retinol transport from the liver. See Tables 2,3.

Vitamin E and Vitamin K

Symptomatic vitamin E and vitamin K deficiencies after RYGB are rarely reported in the literature perhaps because no one is looking. The prevalence of vitamin E deficiency has been reported to be 0-22% after RYGB (6). There are no statistics reported on vitamin K deficiency.

Vitamin E is a fat soluble antioxidant essential for protecting neurons from oxidative damage (12). Symptoms of vitamin E deficiency include retinopathy, hemolytic anemia and nonspecific neurologic symptoms including ataxia, loss of vibration/sensation, and muscle weakness (9).

Ueda et al treated low vitamin E levels in gastrectomy patients with neurological symptoms (peripheral neuropathy, dizziness, or ataxia) with 300-600 mg tocopherol acetate by mouth (13). It took 4-18 months for serum levels to normalize. Serum levels dropped when doses were decreased to a maintenance amount of 50-100 mg; symptoms frequently reoccurred. Interestingly, a higher number of low vitamin E levels were found in patients with a total gastrectomy and Roux-en-Y reconstruction especially beyond 50 months postoperatively.

The ASMBS recommends 100-400 IU/d vitamin E but acknowledges that the optimal therapeutic dose is not known (11). Koch et al. recommends 800-1200 IU daily oral vitamin E to treat a deficiency (9).

Vitamin K is also a fat soluble vitamin and is essential for coagulation (12). Symptoms of vitamin K deficiency include bleeding, stomach pain, cartilage calcification, and atherosclerosis. One case report exists to date that focuses on vitamin K deficiency after RYGB (14). A 60 year old woman was readmitted to the hospital 1 year post RYGB with excess weight loss, diarrhea, weakness and edema. She became septic with *Streptococcus pneumoniae* and subsequently developed purpura fulminans. A marked decrease in vitamin K-dependent protein C and protein S as well as antithrombin were found. Once treated with parenteral nutrition, vitamin K and medical therapy the patient progressively improved and was fully ambulatory 12 months after admission. The authors concluded that poor nutrition and malabsorption of vitamin K led to depleted levels of proteins C and S and antithrombin predisposing her to purpura fulminans.

The ASMBS recommends 10 mg intravenously for repletion of vitamin K and 1-2 mg/d orally or 1-2 mg/week parenterally for chronic malabsorption (11). If elevated prothrombin time shows no improvement, the deficiency is not due to vitamin K. Koch recommends 5-20 mg orally or parenterally (9). See Tables 2, 3.

Vitamin C

Vitamin C is a water-soluble vitamin necessary in the synthesis of bone and connective tissue, has valuable

Table 2. Treatment for Deficiencies

Vitamin/Mineral	American Society for Metabolic and Bariatric Surgery (ASMBS)	Koch, et al	Others
Vitamin A	(-) corneal changes: 10-25,000 IU po x 1-2 weeks (+) corneal changes: 50-100,000 IU IM x 3 days f/b 50,000 IU IM x 2 weeks	10,000 IU po	N/A
Vitamin E	100-400 IU po (optimal doses not defined)	800-1200 IU po	*Ueda: 300-600 mg po
Vitamin K	IV: 10 mg; Chronic malabsorption: 1-2 mg/d orally OR 1-2 mg/week IV	5-20 mg po or IV	N/A
Vitamin C	N/A	N/A	Simmons: 500 mg twice daily x 5 days *Olmeda: 300 mg/d x 2 weeks f/b 100 mg/d or 500 mg/d *Wang: 1 gm x 5 days then 500 mg/d
Zinc	60 mg elemental po twice daily	220 mg zinc sulfate po daily or every other	Davies: 220 mg zinc sulfate po daily or every other
Niacin	N/A	500 mg po 3 times daily	N/A
Vitamin B2 (Riboflavin)	N/A	N/A	N/A
Vitamin B6 (Pyridoxine)	50 mg po daily or 100-200 mg if deficiency related to medication	N/A	*Gerlach: 200 mg IV until normal levels then 100 mg po daily
Copper	N/A	2 mg po copper gluconate daily or every other	O'Donnell: 2 mg po copper gluconate daily or every other Kumar, Goldberg, Shahidzadeh, Kazemi: 1.5-3 mg IV
Thiamine	Hyperemesis: 100 mg IV x 7 days then 50 mg po daily until recovery	100 mg po twice daily; if symptomatic: 100-250 mg IM	Ianelli: 120 mg IV Walker: 100 mg IV or IM *Sechi: 500 mg 3 x day for 2-3 days then 250 mg IV for 2-3 days for S&S of Wernicke's; 250 mg IM for malnourished
Selenium	N/A	N/A	*Boldery: 80 mcg daily po

*Used in population other than RYGB

antioxidant properties, and promotes iron absorption. Early symptoms of vitamin C deficiency can occur within 3-6 months of consuming less than 10 mg/d and may include fatigue, weight loss, irritability, and myalgia (15). Late symptoms manifest as perifollicular petechiae, bruising, poor wound healing, bleeding gums, and corkscrew hair. The diagnosis is usually based on clinical symptoms and diet history (16).

Clements et al (2006) report low serum levels in 35% of patients at both the one and two year follow up, whereas Strohmayer (2010) reports a range of 10-50 % (6,7). However, only one case of overt scurvy has been reported in the gastric bypass population (17). A 39 year old male presented to his bariatric surgery clinic with reports of nausea, vomiting, hair loss, numbness of extremities, weakness, fatigue, and ataxia. Upon physical examination, bleeding gums, a broken tooth and petechiae on the legs and arms were discovered. His RYGB occurred 11 months prior with a total weight loss of 276 pounds and two subsequent admissions to the hospital for dehydration, nausea and vomiting. He was not compliant with vitamin or mineral supplementation postoperatively. A vitamin C level was found to be severely low at < 0.1 mg/dL (normal range 0.6-2.0 mg/dL). The patient was instructed to consume 500 mg of vitamin C twice daily for five days. Repeat labs were not drawn but the patient reported improvement in symptoms despite taking the supplements for only three days.

Although there is limited data on how to supplement vitamin C in the gastric bypass population, Simmons reported success with 500 mg twice daily for five days (17). Olmedo et al. used 100 mg three times per day for two weeks followed by 100 mg per day in a patient with scurvy but no history of gastric bypass (16). Symptoms improved after three days of supplementation. This prompted a retrospective review by the same authors who identified six patients at their hospital with scurvy who had complete resolution of symptoms ranging from one week to one month with 500 mg daily. In another study, a malnourished patient who had no history of a RYGB was diagnosed with scurvy and treated with 1 gram/d for 5 days followed by 500 mg/d (15). Complete resolution of symptoms was seen in 1 month. See Table 2.

Zinc

Zinc is absorbed in both the duodenum and proximal jejunum and is involved in over 200 enzyme systems,

but can be summed up as having 3 basic functions: catalytic, structural and regulatory (18). It plays a role in cell division, cell growth, wound healing and immunity. Although zinc deficiency is more likely to be seen in malabsorptive procedures because of its dependency on fat for absorption (19), Strohmayer reports a 37% deficiency rate in RYGB patients (6). However in a study by Gong, only 6% of patients were deficient at a 2 year RYGB follow-up (4). Deficiency symptoms include dermatitis, alopecia, glossitis, angular cheilitis and diarrhea.

There are only a few published case reports highlighting zinc deficiency. One such case involves acrodermatitis enteropathica-like eruptions after gastric bypass (20). A 43 year old woman was readmitted to a hospital 8 months after a RYGBP with glossitis, cheilitis, alopecia, and brown plaques with desquamative scaling on her extremities, as well as massive lower extremity edema. She had lost 122 pounds since surgery and was noncompliant with her vitamins. Zinc levels were low at 287mcg/L (670-1240). She was diagnosed with a zinc deficiency and kwashiorkor. The rash, glossitis, cheilitis, and edema improved after 3-4 weeks of parenteral nutrition. Actual amounts of zinc replacement were not disclosed. What is interesting about this case is the patient had a distal RYGB which involved the formation of a 125-cm Roux limb thereby creating more malabsorption when compared to the standard RYGB with a roux limb of 50-100 cm. In addition, this patient was non-compliant with prescribed vitamins and had complaints of early satiety, abdominal discomfort and nausea contributing to poor intake, prompting an earlier admission to the hospital 6 months post-op for dehydration. Her weight loss was also excessive, losing 90% of excess body weight (expected loss is 50% during first 6-12 months).

In another case report, both a zinc and niacin deficiency were suspected in a 41 year old female who was 10 years post-op from RYGB and admitted to a hospital with failure to thrive (21). She reported no nausea, vomiting or diarrhea. On day 12 of admission, she developed scaly erythematous plaques on her groin, back and legs. A biopsy was taken and combined with clinical symptoms, zinc and niacin deficiencies were presumed. Histologically, these deficiencies are identical. Treatment was not discussed.

Supplementation for zinc deficiency is not standardized. Recommendations usually involve 220

(continued on page 22)

(continued from page 19)

mg oral zinc sulfate (50mg elemental zinc) daily or every other day (19). The ASMBS recommends 60 mg elemental zinc twice per day by mouth (11). See Tables 2, 3.

Niacin

Niacin is involved in carbohydrate, protein and lipid metabolism, neurotransmitter and hemoglobin synthesis (9). Niacin deficiency, or pellagra, is extremely rare after RYGB. Symptoms include headaches, ataxia, anxiety/depression, hallucinations, dementia, painful, scaly dermatitis and diarrhea. Currently, only 1 case report exists in which a woman developed glossitis, angular cheilitis and dermatitis presenting as Casal's necklace, the classic presentation for pellagra (22). She had a RYGB 3 months earlier with a 100 pound weight loss. Serum niacin levels were in the low range at 2.27ng/mL (0.5-15 ng/mL). In addition, low zinc and pyridoxine levels were found. The patient was supplemented with undisclosed amounts of pyridoxine, nicotinamide, zinc sulfate and riboflavin and showed marked improvement 4 weeks after therapy. Koch recommends 500 mg of oral niacin 3 times daily to treat pellagra (9). See Table 2.

Riboflavin (vitamin B2) and Pyridoxine (vitamin B6)

There are very few clinical case reports to date of riboflavin (B2) and pyridoxine (B6) deficiencies after RYGB despite low serum B2 levels of 13.6 and 7.1% at one and two year follow ups respectively, and low vitamin B6 serum levels of 17.6 and 14.2% (7).

Vitamin B2 plays a key role in the metabolism of carbohydrate, fat, and protein. Symptoms of vitamin B2 deficiency include stomatitis, anemia and scaly dermatitis (9).

Vitamin B6 plays an important role in the synthesis of amino acids and erythrocytes, hormone modulation, immune function and neurotransmitter formation (23). A deficiency may present as dermatitis, atrophic glossitis, angular cheilitis and neurological symptoms including confusion, somnolence, neuropathy and refractory seizures. A low B6 level of 0.6 ng/mL (5-30 ng/mL) was also found in the case study mentioned above by Ashourian where pellagra was diagnosed (22). There are no specific recommendations for vitamin B6 supplementation in the gastric bypass population. In one case report of adults with refractory seizures due to low vitamin B6 levels (and no history of gastric bypass), the authors used 200 mg of intravenous pyridoxine daily. As levels normalized, the patients received 100 mg oral pyridoxine daily (23). The ASMBS recommends 50 mg/d or 100-200 mg/d if the deficiency is related to medication use (such as isoniazid, cycloserine, penicillamine, and hydrocortisone) (11). See Table 2.

Selenium

The prevalence of selenium deficiency ranges from 14 to 22% after RYGB, but there have been no reports of clinical consequences (19). Selenium is a part of the enzyme glutathione peroxidase which protects cells from free radical damage (24). Cardiomyopathy is the most common symptom of deficiency.

Table 3. Select Vitamin/Mineral Toxicities

Vitamin/Mineral	Toxicity
Vitamin A	>50,000 IU for > 3 months; S&S: dry, scaly skin, hair loss, anorexia, vomiting, mouth sores, hepatomegaly, hypercalcemia, poor cognition, increased risk hip fracture
Vitamin E	Nausea, diarrhea, flatulence, can increase vitamin K requirements and lead to bleeding if taking po anticoagulants
Vitamin K	High doses may impair action of po anticoagulants
Zinc	Suppress immune function, cause hypochromic anemia due to interference with copper absorption
Copper	Severe leg tingling/cramps

Cardiomyopathy likely due to a selenium deficiency was seen in a patient who had laparoscopic banding with a 114 pound weight loss followed by a biliopancreatic bypass two years later (24). Nine months after her biliopancreatic surgery with an additional loss of 97 pounds, she was admitted to the hospital with lethargy, weakness, dyspnea and cardiomyopathy. Low selenium levels were found at <0.3 mmol/L (0.7-1.4). Parenteral nutrition with trace elements was initiated along with selenium-containing multivitamins by mouth. Total amounts of selenium were not revealed, although the author notes that in patients with heart failure, 80mcg selenium per day has been used to correct selenium deficiencies (24). However, no recommendations currently exist in the RYGB population.

Copper

Copper is a trace element important in many enzyme systems involving vascular and skeletal tissues and plays a role in the structure and function of the nervous system (25). Copper is absorbed in the proximal small bowel, placing gastric bypass patients at risk for deficiency. Excessive zinc intake has also been identified as a cause of copper deficiency due to competition within the intestinal cells (25). Although the prevalence of copper deficiency is unknown, several case studies have appeared in the literature citing symptomatic copper deficiency. Symptoms may include anemia, neutropenia, and neurological deficits including ataxia, myelopathy and polyneuropathy. Many case reports show a gradual deterioration in ambulation, resulting in the need for a cane, then walker, before finally becoming wheelchair-bound (25-28). Symptoms usually occur more than 10-20 years after surgery, however, in one case report, low copper levels along with ataxia, neuropathy, and regular falls occurred 14 months after gastric bypass along with

a near 200 pound weight loss (26).

In another case report, a woman reported bilateral loss of vision 22 years after gastric bypass surgery (27). Six years earlier, she had reported numbness in her feet which progressed to the point of needing a walker to ambulate. Serum copper levels were found to be low at 15 mcg/dL (70-155 mcg/dL). She was also found to have optic neuropathy in the setting of chronic progressive myeloneuropathy. After copper replacement therapy, vision loss persisted, but myeloneuropathy stabilized.

There are no specific guidelines for repleting copper. Many reports use 1.5-3 mg IV copper (28-30). Kazemi et al successfully treated copper deficiency in 2 gastric bypass patients with 2 mg intravenous copper chloride infused over 1 hour (31). O'Donnell used 2 mg oral copper gluconate daily, which, when combined with a multivitamin and patient's tube feeding provided a total of 4.42 mg copper (26). Severe leg tingling was reported and the copper supplement was decreased to every other day and leg cramps resolved within a few days. Griffith et al also reports severe leg pain during copper repletion using 8 mg per day in a deficient RYGB patient (25). Koch et al recommends 2 mg oral copper gluconate daily to every other day (9). The response to copper replacement is variable and although improvements in neurological symptoms are reported, symptoms do not always fully resolve. See Tables 2, 3.

Thiamine

Thiamine is a water-soluble vitamin absorbed primarily in the jejunum and proximal ileum. It plays a major role in carbohydrate metabolism and tissue storage is limited. Because of the constant demand and limited storage, thiamine is used in 18-20 days when oral intake is low (32). Risk factors for deficiency in the gastric bypass population include restricted calorie

Table 4. Symptoms of Wernicke's Encephalopathy

Altered Mental Status	Ocular Abnormalities	Gait Disturbance	Other
Confusion	Ophthalmoplegia	Ataxia	Hypothermia
Amnesia	Nystagmus	Unsteady stance	Vestibular dysfunction
Obtunded	Impaired visual acuity	Unable to bear weight	
Confabulation	Conjugate gaze palsy, Ptosis	Weakness	

intake, malabsorption, rapid and/or excessive weight loss, prolonged vomiting and noncompliance with medications. Deficiency may include Wernicke's encephalopathy (characterized by ophthalmoplegia, ataxia and neurological changes including confusion, apathy and agitation) and peripheral neuropathy primarily involving the lower extremities (33-35). See Table 4.

Not long after the 32 published cases of Wernicke's encephalopathy (WE) post-gastric bypass surgery appeared in a review in 2007, another review was published citing 84 cases (36,37). In 94% of the cases, admission to the hospital occurred within 6 months of the surgery, and 90% of the patients experienced frequent, recurrent vomiting that lasted a median of 21 days (37). Other risk factors included rapid weight loss (median rate= 0.44 kg/d), loss of appetite, or noncompliance with vitamins. Incomplete recovery, including memory deficits and gait disturbances occurred in 49% of patients. Interestingly, the classic triad of symptoms including ophthalmoplegia, altered mental status and ataxia was present in only 38% of patients. Less common features included depression, visual hallucinations, behavioral disturbances, hearing loss, blurred vision, and retinal hemorrhages. Peripheral neuropathy was present in 76% of patients.

New case reports have been cited since the above reviews. A 46 year old was diagnosed with WE 3 months after a RYGB (38). She was admitted to the hospital with a 1 week history of intractable vomiting, ataxia, and profound visual impairment. On the fourth day of admission, IV dextrose was given to provide some calories. After several hours, the patient became

somnolent. In addition, she had elevated lactic acid in serum and CSF which led to a diagnosis of WE. A brain MRI showed significant improvement after 2 weeks of IV thiamine replacement (actual amounts were not disclosed). After 6 weeks at a skilled nursing facility, the patient was able to follow simple commands. The authors concluded that a high index of suspicion for WE should be maintained when a gastric bypass patient presents with neurological symptoms. In another case report, a 27 year old woman was admitted to a hospital 10 months after RYGB with persistent vomiting for 2 weeks and severe dehydration (32). After 5 days of IV dextrose, she developed diplopia, weakness in all extremities, memory loss and nystagmus. IV thiamine was administered at 120 mg daily and symptoms improved. After 10 months in a rehab facility, the patient had complete recovery of neurologic symptoms. Finally, a 30 year old female was admitted to a psychiatric hospital 17 weeks after a gastric bypass (39). She was admitted with conversion disorder and had experienced nausea, vomiting, lethargy, confusion and ataxia several days prior to admission. Nystagmus was present and she was unable to walk due to weakness. Serum thiamine was found to be low at 0.17 mg/dL (1.1-1.6 mg/dL). Parenteral thiamine, along with total parenteral nutrition was given and after several days, all symptoms improved except the peripheral neuropathy at which point the patient was transferred to a rehabilitation facility. Actual amounts of thiamine infused were not reported.

Treatment often consists of 100 mg of daily parenteral thiamine. Sechi et al recommends 500 mg IV three times daily for 2-3 days, then 250 mg IV for

Table 5. Current UVAHS* Gastric Bypass Vitamin and Mineral Assessment Protocol in Those Admitted with the Following Signs/Symptoms

Sign/Symptom	Action
Recurrent or prolonged n/v (> 2 weeks), rapid weight loss (> 1 pound/day), and/or prolonged poor po intake	Supplement thiamine with 100-250 mg IV; may need more if symptomatic
Cheilosis, stomatitis, glossitis	Check B12, vitamin C, B2, B6, zinc
Skin disorders/dermatitis/rashes	Check niacin, vitamin A, B2, B6, zinc
Neuropathy, myelopathy, gait disturbance	Check copper, vitamin E, vitamin B1, B12, B6, niacin
Vision changes	Vitamin A, vitamin E, copper, thiamine

(continued on page 26)

(continued from page 24)

2-3 days for those with signs of WE and states that doses of 100-250 mg may not restore vitamin status or improve clinical symptoms (40). The authors also recommend prophylactic treatment of 250 mg IM daily for 3-5 days in alcoholics and malnourished patients. In the acute state, the oral route is not recommended due to decreased intestinal absorption in alcoholics. The ASMBS guidelines include 100 mg/d for 7 days followed by oral doses of 50 mg daily until recovery (11). Koch recommends 100 mg orally twice per day, unless the patient is symptomatic in which case 100-250 mg IM should be used (9). See Table 2.

CONCLUSION

It is difficult to know the true prevalence of micronutrient deficiencies in the RYGB population in that they may be unrecognized, misdiagnosed or underreported; furthermore, no guidelines exist to monitor many of them. From the above review, it appears that at-risk patients may fail to thrive soon after their surgery or suffer an adverse event (like nausea, vomiting, etc.) years later, leading to severe micronutrient deficiencies. Non-compliance clearly plays a not insignificant role also. It is incumbent upon the dietitian, physician and healthcare provider to obtain a thorough history, including medical, surgical and nutritional aspects, as well as to recognize signs and symptoms of deficiencies. Being aware of warning signs like excessive weight loss and/or persistent nausea and vomiting may prevent rare but devastating micronutrient deficiencies from occurring or progressing. Given the number of gastric bypass operations done in the U.S., a prospective, longitudinal study of both pre-op and post-op levels should be undertaken in order to prevent the dire clinical consequences in this patient population when severe deficiency occurs. See Table 5 for the current vitamin/mineral panel UVAHS will be using when gastric bypass patients are admitted with potential vitamin/mineral deficiencies until better evidence becomes available. ■

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