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Origins for Micronutrient Deficiencies

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1. Introduction

Dietary and activity programs result in poor weight loss or poor maintenance of weight loss, and bariatric surgery therefore remains the major treatment option for patients with medically-complicated obesity. There are now over 220,000 bariatric surgical procedures performed each year in the United States and Canada. The 'divided' or 'isolated' Roux-en-Y gastric bypass remains the most commonly performed bariatric surgical procedure in the United States and Canada for surgical treatment of patients with medically-complicated obesity.

Micronutrients in human physiology include essentials minerals such as iron, water soluble vitamins (such as the B vitamins and vitamin C), fat soluble vitamins (A, D, E, and K), and trace elements (zinc, copper, selenium, manganese, and likely chromium). Understanding the origins for micronutrient deficiencies in patient who have undergone bariatric surgery should permit better screening and maintenance techniques to prevent and discover underlying deficiencies. As a first step, the importance of preoperative evaluation for identification of micronutrient deficiencies is under evaluation. The importance of this preventative approach is supported by recent reports of major or fatal nutritional complications occurring more than 20 years after bariatric surgery.

In this chapter, dietary methods including food journals are shown to be important for examining pre-operative diets in order to predict potential micronutrient deficiencies. The relatively insufficient intake of specific micronutrients in those patients instructed post-operatively on a high protein diet is discussed.

The surgical origins for micronutrient deficiencies that are induced by bypass of physiologically relevant segments of the gastrointestinal tract are summarized. This chapter includes a discussion of the pre-operative medically-induced origins for micronutrient deficiencies including the importance of chronic gastric infection with *Helicobacter pylori* and small intestinal bacterial overgrowth related to diabetic gut autonomic neuropathy. Post-operative medical conditions that lead to micronutrient deficiencies including small intestinal bacterial overgrowth induced by achlorhydria of the gastric pouch are described.

Clinical presentations of micronutrient deficiencies after bariatric surgery are summarized. Common micronutrient deficiencies considered after bariatric surgery include iron deficiency, vitamin D deficiency and thiamine deficiency. Vitamin D deficiency with

metabolic bone disease remains common after gastric bypass and recent results suggest that the present postoperative supplements of calcium and Vitamin D are inadequate. The potential role of copper deficiency as an origin for visual disorders and neurological disorders after gastric bypass is discussed. A major goal of ongoing clinical studies is a better understanding of whether blood levels of micronutrients are sufficient to exclude underlying deficiency states or whether clinical symptoms must be identified and categorized in patients who have undergone bariatric surgery.

2. Micronutrients

Macronutrients including fat, protein, and carbohydrates are important dietary components for conversion into chemical energy and for tissue and cellular structure. By contrast, micronutrients are essential dietary factors required for biochemical and cellular processes (please see Table 1). Specific micronutrients are required in microgram or milligram quantities in a diverse group of biochemical pathways and metabolic processes. Micronutrients include trace elements (chromium, copper, manganese, selenium and zinc), essential minerals (including calcium, iodine, iron, and magnesium), water-soluble vitamins (including B vitamins and vitamin C) and fat-soluble vitamins (vitamins A, D, E, and K).

Water Soluble Vitamin	**Cofactor In**
Thiamine (Vitamin B1)	Thiamine Pyrophosphate
Riboflavin (Vitamin B2)	Flavin Adenine Dinucleotide
Niacin (Vitamin B3)	Nicotinamide Adenine Dinucleotide
Folic Acid	Tetrahydrofolate
Pyridoxine (Vitamin B6)	Pyridoxal Phosphate
Cobalamin (Vitamin B12)	5'-Deoxyadenosyl-Cobalamine
Ascorbic Acid (Vitamin C)	
Trace Elements	**Cofactor In**
Chromium	Uncertain
Copper	Cytosolic Superoxide Dismutase & Cytochrome Oxidase
Manganese	Mitochondrial Superoxide Dismutase
Selenium	Glutathione Peroxidase
Zinc	Cytosolic Superoxide Dismutase & Hundreds of Proteins
Essential Minerals	**Cofactor In**
Calcium	
Iodine	Thyroid Hormone
Iron	Catalase & Hemoglobin
Magnesium	
Fat-Soluble Vitamins	
Vitamin A	
Vitamin D	
Vitamin E	
Vitamin K	

Table 1. Micronutrients and their roles as cofactors

Many bariatric programs recommend taking a comprehensive multivitamin with minerals twice daily after a malabsorptive procedure, and a daily calcium supplement (≥1.2 g/day elemental calcium). It is not certain whether these suggestions are sufficient to prevent micronutrient deficiencies after Roux-en-Y gastric bypass. This level of supplementation is unlikely to be adequate after a biliopancreatic diversion.

3. Dietary origins for micronutrient deficiencies

3.1 Statement of the problem

Body mass index (BMI; in kg/m2) is used to classify underweight (BMI <18.5), normal weight (BMI 18.5-24.9), overweight (25-29.9), obese (BMI 30-39.9), and extreme obesity (BMI > 40). Over the last 20 years, there has been a dramatic increase in obesity in the United States. Data from two National Health and Nutrition Examination Surveys (NHANES) show that among adults aged 20-74 years, the prevalence of obesity increased from 23%, in the 1988-1994 survey, to 33.8% in the 2007-2008 survey (1). Since the prevalence of obesity is increasing rapidly, it is important to find effective weight control or weight loss strategies for these individuals. This is of international concern as it appears that other developed countries are also facing increases in the prevalence of obesity.

Lifestyle interventions that involve diet, exercise, and behavior change strategies can result in an average weight loss of 7-10% of initial body weight after 6 months of treatment (2, 3). The most effective treatment for obesity, on the other hand, is bariatric surgery. The average mean excess weight loss (%EWL) is 50% for the adjustable gastric band and 68% for Roux-en-Y gastric bypass. This dramatic weight loss not only improves quality of life, but also reduces the risk of developing health conditions and may even improve existing health problems. Although weight loss improves health, surgical treatments for severe obesity may make pre-existing nutritional deficiencies worse or produce new ones, depending on dietary intake, adherence to supplementation and the degree of malabsorption associated with the bariatric procedure performed (4). As the popularity of bariatric surgery continues to increase, it is important for clinicians working with these individuals to identify and treat preexisting nutritional deficiencies.

3.2 Proposed origins

It has been proposed that the major causes of obesity include a combination of lack of physical activity, a high-fat, high-caloric Western diet, increased portion sizes, and low socioeconomic status (5-8). Regarding diet, data from four NHANES conducted between 1971 and 2002 for trends in self-reported food intake showed that energy intake, amount of food, and carbohydrate energy have significantly increased in all race and gender groups. Findings from a study published by Neilson (7) illustrate how energy intake has increased by increasing portion sizes. They found that between 1977 and 1996, portions inside and outside the home increased for salty snacks (by 93 Kcals), soft drinks (by 49 Kcals), hamburgers (by 97 Kcals), French fries (by 68 Kcals), and Mexican food (by 133 Kcals). Clearly, this increase growth in portion sizes mirrors our growing waistlines.

In addition to diet and increasing portion sizes, socioeconomic status and the environment must also be considered as contributors to the growing problem of obesity. Fruits, vegetables, low-fat dairy, nuts and legumes, and lean proteins are considered unprocessed,

nutrient–dense foods, which contribute most of the vitamins and minerals necessary for health. While foods that are processed such as prepackaged convenience foods, sweets, and snack goods provide a high amount of calories but are nutrient-poor and lack proteins, vitamins, minerals, and fiber. These energy-dense, nutrient-poor foods are often more affordable than foods of lower energy-density or higher nutrient-density such as fruits, vegetables, and whole grains (9, 10). Poverty can lead to food insecurity and one outcome of food insecurity is obesity (11). It has been found that households characterized as food insecure also have the highest body mass index and prevalence of obesity (12). When a household runs out of food and is uncertain about the ability to obtain enough food, the quality of the diet becomes compromised and leads to an increase in the intake of energy from foods that are higher in fat and carbohydrate, but lower in nutrients (11). This may be one of the reasons why we see so many nutritional deficiencies in obese persons.

3.3 Micronutrient deficiencies and dietary intake

Studies are demonstrating that micronutrient deficiencies are common in obese people even before bariatric surgery (13, 14). As discussed above, one cause of these nutritional deficiencies in overweight and obese individuals may be due to a high intake of processed foods that are calorically dense, but nutritionally-poor. Additionally, obese individuals may have altered bioavailability of many nutrients, a condition that has been called "high calorie malnutrition" (15). The combination of low preoperative vitamin status and the malabsorption that follows bariatric surgery may leave these patients at risk for many severe vitamin deficiencies. Typical nutritional deficiencies associated with obesity include antioxidants, vitamin A, vitamin D, B-complex vitamins, calcium, iron, and zinc (16). In a retrospective study of 379 patients planning for gastric bypass, significant deficiencies were identified for thiamine (29%), iron (44%), and 25-hydroxyvitamin D (68%). A second study also examined cross-sectional data from NHANES III and found that obese subjects (n = 3831), particularly premenopausal women, were more likely to have low levels of various micronutrients than were normal-weight adults in the same sex/age category (17). Among women, low biochemical micronutrient levels were associated with increasing BMI categories for vitamin E, alpha-carotene, beta-carotene, beta-cryptoxanthin, lutein/zeaxanthin, lycopene, total carotenoids, vitamin C, selenium (premenopausal), vitamin D, and folate. And among men, low biochemical micronutrient levels were associated with increasing BMI categories for alpha-carotene, beta-carotene, beta-cryptoxanthin, lutein/zeaxanthin, total carotenoids, vitamin C, selenium, and folate. Since nutritional deficiencies are so common in the obese population, it will remain important to conduct trials involving preoperative testing with treatment of micronutrient deficiencies prior to surgery, in order to look for improved postoperative outcomes.

Behavioral weight loss programs often involve a reduced calorie diet, increased energy expenditure, and use of behavior strategies such as goal setting and self-monitoring (18). Self-monitoring has been described as the cornerstone of behavioral treatment for weight loss (19, 20). Usual techniques for self-monitoring involve keeping food, weight, and activity diaries. One recent randomized-controlled trial compared the effectiveness of self-monitoring with a paper diary versus two types of personal digital assistants (PDA) (21, 22). After 6 months, they found that, compared to the paper diary group, both PDA groups were more adherent to self-monitoring and that self-monitoring of diet in this group had a significant indirect effect on percent weight loss. The use of food diaries not only improves

ones chances for weight loss, but it also gives clinicians a tool to assess food intake and dietary patterns. In addition, it may help to identify possible nutritional deficiencies both before and after weight loss surgery.

When considering what nutritional deficiencies may be present before surgery it is important to look at what we are eating as a nation. NHANES is a continuous, cross-sectional survey designed to monitor the health and nutritional status of the civilian, noninstitutionalized U.S. population (23). Based on the most recent 2007-2008 NHANES data, adults over age 20 are not meeting the minimum recommended intakes for fruit, vegetables, and low-fat dairy and thus had diets that that did not meet the dietary reference intakes (DRIs) in fiber, vitamin A, vitamin D, vitamin E, vitamin K (men only), calcium, magnesium (women only) and potassium (24). It is interesting to note that over one-third (36.8% for men and 35.5% for women) of the total daily caloric intake for adults comes from solid fats, added sugars, and alcohol (SoFAS). This is well above the highest recommended limit and may be one answer to the growing obesity problem in America. MyPyramid recommends calories from SoFAS to range from 132-512 per day based on height, weight, gender, and activity level. According to the 2007-2008 NHANES data, the mean intake of calories from SoFAS in adult men was 923 calories/day and for women it was 624 calories/day.

Another recent study analyzed 2-day, 24-hour recall data from the 2003–2004 NHANES and found that fewer than 1 in 10 Americans met the MyPyramid recommendations for fruit and vegetable intake (25). Only 0.9% of adolescents, 2.2% of adult men, and 3.5% of adult women met the recommendations for both fruit and vegetables. A higher percentage of participants met fruit recommendations alone, 6.2% (adolescents), 8.6% (adult men), and 12.3% (adult women). Adolescents consumed less whole fruit and more juice than adults and the largest contributor to fruit intake was orange juice. Only 5.8% of adolescents, 14.7% of adult men and 18.6% of adult women met recommendations for vegetable intake. Interestingly, when fried potatoes were excluded only 2.2% of adolescents, 9% of adult men and 13.4% of adult women met recommendations for vegetable intake. The percentage meeting recommendations for vegetable subtypes for all groups was lowest for dark green vegetables, orange vegetables, and legumes but higher for starchy and other vegetables. This data makes it clear that we are not meeting the recommended intakes for low-calorie, nutrient-dense foods. And when we are choosing vegetables, they are likely to be fried starchy vegetables with added fats and thus contribute excessive calories in the diet. It is also important to consider the amount of calories we are consuming from the beverages we are drinking when looking at the obesity problem. One recent report based on NHANES data between 2005-2008, states that 50% of the population consumes sugary drinks on any given day, while 25% consumes some sugary drinks but less than 200 kcal (more than one 12-oz can of cola), and 5% consumes at least 567 kcal from sugar drinks per day (more than four 12-oz cans of cola) (26). Consumption of sugary drinks is lowest among the oldest females (42 kcal per day) and highest among males aged 12–19 (273 kcal per day). Among adults aged 20 and over, it was found that non-Hispanic white persons consume fewer sugary-drink calories as a percentage of total daily calories (5.3%) than do non-Hispanic black (8.6%) or Mexican-American persons (8.2%). Low-income individuals tend to consume more sugary beverages than higher-income individuals. Among adults living below 130% of the poverty line, they report that mean calories from sugary drinks makes up 8.8% of total calories; among those living between 130% and 350% of the poverty line, mean calories from sugary drinks is 6.2% of total calories; and among those at or above 350% of the poverty line,

mean calories from sugary beverages is 4.4% of total calories. This data is alarming since consuming sweetened beverages has been linked to poor diet quality, weight gain, obesity, and, in adults, type 2 diabetes (27, 28). Since a good portion of our calories are coming from sugary beverages and calorie-dense, nutrient-poor foods, it is understandable why we find many obese patients with micronutrient deficiencies prior to weight loss surgery. It is therefore important to screen for these and to treat their nutritional deficiencies prior to surgery to help to prevent major or fatal nutritional complications that may occur in the long-term after bariatric surgery.

4. Surgical origins for micronutrient deficiencies

4.1 Roux-en-Y gastric bypass

Roux-en-Y gastric bypass (RYGB) is the most commonly performed bariatric surgery in North America and remains the major surgical option for individuals with medically-complicated obesity. The divided RYGB combines restriction of food intake, due to the small size of the gastric pouch and constriction at the gastrojejunal anastomosis, with malabsorption induced by bypass of the duodenum and variable lengths of jejunum (see Figure 1). A small proximal stomach pouch is created with a stapler device and is connected to distally transected jejunum. The remnant stomach and proximally transected jejunum is then reattached in a Y-shaped configuration to the distal small intestine. Malabsorption of both fat and nitrogen has been identified in studies of the Roux-en-Y reconstruction (29). Malabsorption was corrected by providing oral, exogenous pancreatic enzymes. The RYGB bypasses native stomach that secretes intrinsic factor which is required for vitamin B12 absorption, as well as proximal small intestine which is required for absorption of copper, iron, and thiamine. The length of small intestine between the gastrojejunostomy and the jejunojejunostomy is not exposed to bile, thus limiting absorption of fat soluble vitamins due to absence of micelle formation. Reduced vitamin D levels due to malabsorption induce inadequate calcium absorption and utilization.

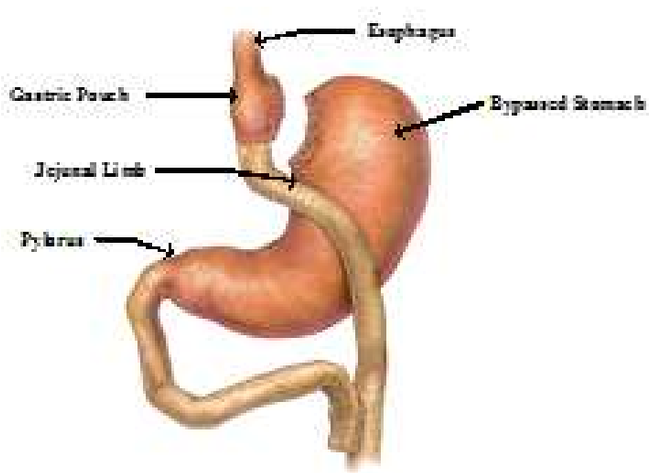


Fig. 1. Roux-en-y gastric bypass. The orientation of the gastro-jejunal anastomosis can be different depending upon whether it is formed during an open or laparoscopic surgery. The location of the jejuno-jejunal anastomosis alters the length of the common channel, which extends from the jejuno-jejunostomy to the ileocecal valve. (Adapted with permission from 31)

There are multiple studies of micronutrient deficiencies after RYGB. A major study of 493 patients after laparoscopic RYGB revealed deficiencies of vitamin A (11%), vitamin C (34.6%), vitamin D (7%), thiamine (18.3%), riboflavin (13.6%), vitamin B6 (17.6%), and vitamin B12 (3.6%) at 1 year after surgery (30). The results must be considered with regards to the body's reserves for different vitamins (18 days for thiamine and 3 to 5 years for vitamin B12 and vitamin E).

4.2 Adjustable gastric banding

Laparoscopic adjustable gastric banding (LAGB) is an effective strategy for the surgical treatment of morbidly obese patients and is the most common bariatric procedure in Europe and Australia. Restriction is created by placing a silicone band around the fundus of the stomach, approximately 4 cm distal to the gastroesophageal junction. The level of gastric constriction by the band can be adjusted by addition or removal of saline through a subcutaneous port placed above the abdominal musculature (see Figure 2). Since the introduction of the modern adjustable gastric band in 1994, this procedure has continued to gain momentum as a therapeutic alternative to RYGB. Since LAGB is a restrictive procedure, nutritional deficiencies are not expected post-operatively. However, recent reports of micronutrient deficiencies are now emerging. A study of LAGB in adolescents revealed vitamin D deficiency as the second most common micronutrient deficiency (after iron deficiency anemia) within the first two years after surgery (32).

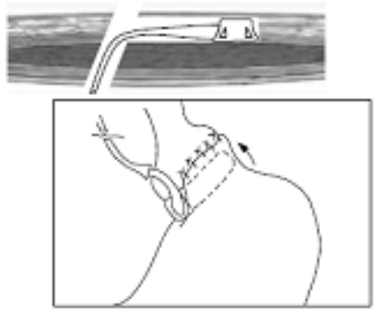


Fig. 2. Gastric Adjustable Band. As shown in the lower half of this drawing, the band is placed laparoscopically around the upper part of the stomach, approximately 4 cm below the gastroesophageal junction. As shown in the upper half of this drawing, the access port in placed subcutaneously on top of the abdominal musculature. The ring or band is connected to the access port by tubing and its volume can be adjusted by accessing the port in order to add or remove sterile saline (Adapted with permission from Allergan, Inc. from http://www.allergan.com/assets/pdf/lapband_dfu.pdf).

4.3 Vertical sleeve gastrectomy

Sleeve gastrectomy (SG), or gastric sleeve, is a surgical weight-loss procedure in which the stomach is reduced to about 15% of its original size by surgical removal of a large portion of the stomach along the greater curvature. Despite being described as a solely restrictive procedure, micronutrient deficiencies post operatively are common. A recently published study from the Netherlands showed that 21% patients were Vitamin D deficient within one year of having a SG (33). This deficiency occurred despite daily multi-vitamin

supplementation. Bone loss and bone remodeling also occurs following SG in as little as one year. Significant bone mass loss and remodeling were recorded in a study using bone densitometry and bone remodeling markers (34). Despite the presence of micronutrient deficiencies induced by SG, studies show that fewer nutrient deficiencies occur after SG as compared to RYGB (35). However, SG is often combined with the duodenal switch procedure as described in section 4.4.

4.4 Biliopancreatic diversion

Biliopancreatic diversion (BPD), or the Scopinaro procedure, is the original weight loss surgery. This procedure is now rarely performed because of problems with severe malnutrition and severe early complications, including death (36, 37). BPD is the hallmark of a malabsorptive procedure that produces substantial and sustained weight loss, but the malabsorption of fat and protein is paralleled by malabsorption of fat soluble and water soluble vitamins, minerals, and trace elements.

This operation has been replaced by malabsorptive procedures that obtain a "physiological" biliopancreatic diversion, through formation of a jejuno-enteric anastomosis ≤120 cm from the ileocecal valve (i.e. by preparation of a long Roux limb or by preparation of a long biliopancreatic limb). Malabsorption induced by a short common channel in part involves inadequate micelle formation, with subsequent development of steatorrhea.

By comparison, in the duodenal switch procedure, the duodenum is transected 5 cm distal to the pylorus; the distal duodenal segment (stump) is then oversewn. Small intestine is transected approximately 1/3 of its length to the ileocecal valve. The distal transected segment is used in the production of an anastomosis to proximal duodenum and the proximal transected segment is used in the production of an entero-enteric anastomosis (which is 75 to 100 cm proximal to the ileocecal valve).

In comparison to RYGB, low serum levels of zinc and copper are more common in individuals after biliopancreatic diversion (38). A randomized trial of RYGB compared to duodenal switch revealed increased deficiencies of thiamine, vitamin A, and vitamin D in the first year after duodenal switch (107).

5. Medical origins for micronutrient deficiencies

5.1 Preoperative deficiencies

Major micronutrients that have been examined preoperatively include vitamin D (vitamin D deficiency is reported in 25% to 96% of morbidly obese individuals) (39-42) and thiamine (15% prevalence of low preoperative thiamine levels among 437 consecutive obese patients) (43). Obesity was identified as a risk factor, in a preoperative surgical population, for finding low plasma ascorbic acid concentrations (44). In preoperative obese patients in Israel, common micronutrient deficiencies included iron (35%), folic acid (24%), and vitamin B12 (3.6%) with high levels of parathyroid hormone identified in 39% of patients (45). A high prevalence of vitamin A deficiency in obese, preoperative patients has been described (46), but daily supplementation with 5,000 IU of retinol acetate (ester derivative of Vitamin A) did not fully resolve vitamin A deficiency 6 months after RYGB.

5.2 Weight loss supplements and programs

Special dietary programs and dietary supplements, which are commonly utilized by morbidly obese individuals, have been associated with development of micronutrient deficiency (47, 48). Starvation, which has been previously suggested as a treatment for obesity, can also contribute to the development of thiamine deficiency.

5.3 Pharmacological agents

Pharmacologic agents can result in micronutrient depletion through multiple mechanisms. Excretion of urinary zinc is enhanced by diuretics, such as hydrochlorothiazide (49, 50).

A second proposed mechanism involves molecular mimicry. The prototype drug for this proposed mechanism is conversion of metronidazole to an analog of thiamine, an inhibitor of thiamine pyrophosphokinase (51).

Thiamine deficiency induced by parenteral feedings (52) may be caused by consumption induced by a high glucose load or may be induced by development of sepsis (53), especially in patients being treated for postoperative complications. More severe beriberi is associated with the development of lactic acidosis and "fulminant beriberi" may be related to thiamine depletion induced by a high glucose load in patients receiving total parenteral nutrition (54).

Alcohol consumption appears to be a toxic origin for depletion of multiple micronutrients, including B vitamins (55), folate (due to a weak antifolate effect) (56), and zinc and selenium (57).

5.4 Small intestinal bacterial overgrowth

Small intestinal bacterial overgrowth is another potential explanation for the development of micronutrient deficiencies. After Roux-en-Y gastric bypass, there is relative achlorhydria of the gastric pouch, which may permit upper gut bacterial overgrowth. As another potential origin for small bowel bacterial overgrowth, it has been suggested that small intestinal bacterial overgrowth in patients with diabetes mellitus is related to the presence of a gut motility disorder (58). In support of this mechanism, the prevalence of small intestinal bacterial overgrowth is higher in diabetic patients who have evidence for autonomic neuropathy (59). In a preliminary study, we have previously noted upper gut bacterial overgrowth among patients who have undergone an adjustable gastric band, perhaps induced by stasis in the proximal gastric pouch (60).

In our previous study, postoperative thiamine deficiency after RYGB was associated with small intestinal bacterial overgrowth (31). Consumption of micronutrients by small intestinal bacteria has been reported but is not fully understood. There are multiple studies of the effect of bacteria on vitamin B12 and thiamine. A major observation in this field has been that bacteria secrete thiaminases, which can cleave thiamine (61-63). Staphylococcus aureus produces a thiaminase type II that is regulated at both the transcriptional as well as the enzymatic level. Thiaminase type II catalyzes the cleavage (deamination) of thiamine.

6. Deficiencies of micronutrients

6.1 Water soluble vitamins

The biochemical roles of water-soluble vitamins and their associated deficiency disorders are shown in Table 2. There is only minor storage in the body of most water-soluble vitamins.

VITAMIN	DEFICIENCY STATE	SYMPTOMS
Thiamine (Vitamin B1)	Beriberi	See Table 3
Riboflavin (Vitamin B2)	Ariboflavinosis	Anemia, Stomatitis, Glossitis, Dermatitis
Niacin (Vitamin B3)	Pellagra	Diarrhea, Dermititis, Ataxia, Confusion
Folic Acid	Folate deficiency anemia	Weakness, Anorexia, Weight Loss
Pyridoxine (Vitamin B6)		Dermititis, Confusion, Neuropathy
Cobalamin (Vitamin B12)	Pernicious Anemia	Depression, Ataxia, Parasthesias
Ascorbic Acid (Vitamin C)	Scurvy	Malaise, Myalgias, Petechia, Gum Disease

Table 2. Water soluble vitamins and their deficiencies

6.1.1 Thiamine

Thiamine or vitamin B1 deficiency is a major nutritional complication following RYGB (31). Thiamine deficiency or beriberi was originally described in individuals with multi-organ involvement, including cardiac, gastrointestinal or neuropsychiatric symptoms that corrected with "beriberi factor" or thiamine (see Table 3)). We have described after RYGB the presence of thiamine deficiency that does not correct with oral thiamine. "Bariatric beriberi" is associated with small intestinal bacterial overgrowth and antibiotic therapy may be required to correct thiamine deficiency.

Among the major clinical presentations of beriberi, patients with neuropsychiatric beriberi may have auditory and visual hallucinations, or aggressive behavior. Wernicke's disease presents with confusion (impairment of memory or altered mental state), nystagmus, ataxia, and ophthalmoplegia. Patients with high output cardiovascular disease (wet beriberi) have been reported to have tachycardia, respiratory distress, or lower extremity edema, with right ventricular dilation and lactic acidosis. Patients with neurological (dry beriberi) present with numbness or muscle weakness, pain of lower greater than upper extremities, or convulsions. Gastrointestinal beriberi induces delayed emptying of the stomach. After gastric bypass surgery, common symptoms include nausea and emesis in patients who may have megajejunum and constipation in patients who may have megacolon. A recent case study supports the notion that thiamine deficiency can induce both a sensory ataxia and optic neuropathy (64), a symptom complex more commonly believed to suggest copper deficiency.

Only a small percentage of total body thiamine is present in whole blood. Measurement of erythrocyte transketolase activity is an alternative approach for determination of thiamine deficiency (65). This bioassay is based upon binding of the catalytic activity of the enzyme transketolase to thiamine pyrophosphate. Due to the seriousness of thiamine deficiency, recent European guidelines suggest that after bariatric surgery, patients should have follow-up evaluation of their thiamine status for at least 6 months and receive parenteral thiamine supplementation (66).

A standard therapy for thiamine deficiency is thiamine HCl 100 mg taken orally twice daily. In patients who do not respond to oral thiamine, the presence of small intestinal bacterial overgrowth must be considered. Acute psychosis and Wernicke's encephalopathy are medical emergencies. These conditions require hospitalization with supportive care and a minimum of 250 mg of thiamine given daily intramuscular or intravenously (infused over 3 to 4 hours to reduce the risk of an anaphylactoid reaction) for at least 3 to 5 days (67). Most patients report symptomatic improvement within several days after the first parenteral dose of thiamine HCl. An autopsy is recommending for those individuals who have died from suspected Wernicke encephalopathy (66).

BERIBERI SUBTYPE	SYMPTOMS AND FINDINGS
Neuropsychiatric	Hallucinations; Aggressive Behavior; Confusion; Nystagmus; Ataxia; Ophthalmoplegia
Neurologic (Dry beriberi)	Numbness; Muscle Weakness and Pain of Lower>Upper Extremities; Convulsions; Exaggerated Tendon Reflexes
High Output Cardiac (Wet beriberi)	Tachycardia; Bradycardia; Respiratory Distress; Leg Edema; Right Ventricular Dilation; L-Lactic Acidosis
Gastroenterologic	Nausea; Vomiting; Slow Gastric Emptying; Megajejunum; Constipation; Megacolon

Table 3. Clinical features of beriberi

6.1.2 Riboflavin

Riboflavin or vitamin B2 is present in the flavocoenzymes, flavin adenine dinucleotide and flavin mononucleotide. Flavocoenzymes are a major participant in a number of reactions important in metabolic pathways and in the proper functioning of glutathione peroxidase (required for metabolism of hydroperoxides) and glutathione reductase (generates reduced glutathione from oxidized glutathione). Biochemical but not clinical riboflavin deficiency has been reported after bariatric surgery (30). Clinical symptoms of riboflavin deficiency include sore throat, stomatitis, anemia, and a scaly dermatitis. It has been suggested that riboflavin deficiency may play a role in the development of migraine-like headaches. The standard treatment for riboflavin deficiency is 5-10 mg daily of oral riboflavin.

Since the reliability of the serum assay for vitamin B2 is uncertain, a clinical improvement in a potential symptom during supplementation with oral riboflavin may support the diagnosis of a deficiency state.

6.1.3 Niacin

Nicotonic acid or vitamin B3 is converted into both nicotinamide, a component of nicotinamide adenine dinucleotide (involved in catabolic reactions), and nicotinamide adenine dinucleotide phosphate (involved in anabolic reactions). Clinical deficiency of niacin has not been reported after bariatric surgery, although it has likely been present in patients with multiple B vitamin deficiencies. It is difficult to diagnose niacin deficiency by laboratory studies, but the diagnosis is supported by low plasma niacin. Deficiency of niacin is termed pellagra, which includes neurologic, dermatologic, and gastrointestinal involvement. Patients may present with headaches, ataxia or myoclonus, anxiety-depression, delusions or hallucinations, painful, scaly dermititis, and a malabsorptive disorder or diarrhea with colitis. The initial treatment of pellagra is initiation of oral niacin 100-500 mg, three times daily (this may induce flushing). Symptomatic improvement during niacin supplementation could support a diagnosis of niacin deficiency.

6.1.4 Folate

In studies of patients with small intestinal bacterial overgrowth, a high serum folate level is an identified and validated marker for bacterial overgrowth (68). Folate levels in patients after RYGB must therefore be considered in the context of a patient's risk for development of small intestinal bacterial overgrowth. In patients who have folate deficiency after bariatric surgery, the potential for another small intestinal malabsorptive disorder, including celiac sprue, should be considered. Patients with folate deficiency are generally detected in this patient population in individuals with a normocytic, mixed anemia with an increased red cell distribution width. Symptoms of folate deficiency include weakness, anorexia, and weight loss. Treatment of folate deficiency begins with oral folic acid, 1 to 5 mg daily.

6.1.5 Vitamin B6

The active form of vitamin B6 is pyridoxal phosphate. A clinical diagnosis of vitamin B6 deficiency has not been widely recognized after bariatric surgery. Patients with vitamin B6 deficiency can present with dermatitis, confusion, anemia and neurologic symptoms. Peripheral neuropathy has also been reported with vitamin B6 deficiency (69). Treatment can be started with oral vitamin B6 at 30 mg daily; a post-therapy increased level of pyridoxal phosphate is then consistent with repletion of vitamin B6.

6.1.6 Vitamin B12

Vitamin B12 deficiency is a well-described nutritional deficiency after bariatric surgery and is likely multifactorial in origin (30). RYGB results in the exclusion of the majority of parietal cell mass, which is a site of R factor and intrinsic factor production. Relative achlorhydria after bariatric surgery prevents oral cyanocobalamin from being deconjugated from pteryl groups, before cyanocobalamin absorption.

Because of vitamin B12 storage in the body, development of vitamin B12 deficiency may become clinically relevant 3 to 5 years after bariatric surgery. Methyl malonic acid blood levels will increase when vitamin B12 stores are depleted (70). A low normal blood level of vitamin B12 can indicate the presence of deficiency. Clinical manifestations of vitamin B12 deficiency include the multiple presentations of pernicious anemia or the development of

peripheral neuropathy. In treatment of vitamin B12 deficiency, daily oral cobalamin is considered less effective than the intra-muscular preparation. Treatments for Vitamin B12 deficiency include: i. oral vitamin B12 (cyanocobalamin) 350 to 500 mcg per day, ii. intramuscular vitamin B12 1,000 mcg every month or 3,000 mcg every 6 months, iii. nasal (500 mcg once weekly), or iv. sublingual (500 mcg, once daily) preparation.

6.1.7 Vitamin C

Biochemical evidence for vitamin C or ascorbic acid deficiency is common after bariatric surgery (44). Ascorbic acid deficiency is suggested by clinical symptoms. In our clinical practice, bariatric patients present with fatigue or complaint of arthralgias. Early reported symptoms include malaise, myalgias, and petechia, with progression to gum disease (scurvy). Standard treatment for a patient with vitamin C deficiency is oral ascorbic acid 200 mg daily.

6.2 Fat soluble vitamins

Signs and symptoms of the fat soluble vitamin (A, D, E, and K) deficiency are summarized in Table 4.

VITAMIN	SYMPTOMS
A	Nyctalopia, Blindness
D	Arthralgias, Myalgias, Fasciculations
E	Anemia, Dysarthria, Ataxia, Myopathy
K	Bleeding Disorder

Table 4. Fat soluble vitamins and their deficiencies

6.2.1 Vitamin A

Vitamin A complex includes retinols, beta-carotenes, and carotenoids, and there is approximately a 1 year supply stored in human liver. When ingested in high doses, excessive doses of vitamin A may cause headache, vomiting, diplopia, alopecia, dryness of the mucous membranes, bone abnormalities, and liver damage. Signs of toxicity usually appear with sustained daily intakes exceeding 15,000 IU. As alternative therapy, signs of toxicity have not been observed while receiving beta-carotene, a previtamin A analogue.

Vitamin A deficiency after bariatric surgery is most commonly seen in patients who have undergone a biliopancreatic diversion, duodenal switch, or extended Roux-en-Y gastric bypass. In these procedures, the mechanism of deficiency is most likely related to fat soluble vitamin malabsorption induced by bile acid deficiency. In addition, individuals with zinc deficiency have impaired protein synthesis that may alter retinol transport from the liver to other organs. Manifestations of vitamin A deficiency in a bariatric patient include nocturnal visual difficulty, dry skin, dry hair, and pruritus. Other potential manifestations include decreased visual acuity and reduced resistance to infections. Treatment of vitamin A deficiency includes supplemental vitamin A, 10,000 IU daily by mouth (with co-therapy of any existing iron deficiency since vitamin A deficiency may persist in the presence of iron deficiency).

6.2.2 Vitamin D

Vitamin D deficiency after RYGB induces metabolic bone disease (71). Vitamin D deficiency activates a metabolic cascade resulting in decreased calcium absorption and insufficient calcium availability. This cascade induces subsequently hypocalcemia, secondary hyperparathyroidism, and development of osteoporosis and osteomalacia (72-74). Patients will see their physician for symptoms of bony pain, back pain, or aching of the limbs (75).

There is a high frequency of and serious nature of metabolic bone disease after RYGB including bone biopsy-proven osteomalacia with marrow fibrosis (76). Symptoms may be present for as long as 2 to 5 years prior to evaluation.

Studies of patients after RYGB demonstrate that maintenance of normal serum calcium involves increasing bony release of calcium and decreasing urinary calcium secretion. After RYGB, a high prevalence of bone resorption and hyperparathyroidism may exist independent of intake of calcium and Vitamin D status (77, 78). This raises a concern that the present recommendations for daily calcium and vitamin D supplementation may not be protective after RYGB, especially in female patients. Commonly, in postoperative follow up after RYGB, patients will still have evidence for inadequate blood levels of vitamin D despite vitamin D oral supplements.

For monitoring, 24-hour urinary calcium determination and a serum alkaline phosphatase level every 6 to 12 months is commonly suggested. However, urine calcium secretion can be altered by concomitant use of diuretics. If urinary calcium excretion is low and the serum alkaline phosphatase activity is increased, alkaline phosphatase should be fractionated. If the alkaline phosphatase is of bone origin, then a serum parathyroid level should be measured. An increase in serum parathyroid level supports supplementation with additional vitamin D and calcium. It is important to obtain a total 25-hydroxy vitamin D level every 12 months or earlier if a patient has a low 24-hour urinary calcium excretion. Low vitamin D levels require consideration of several explanations, including bile salt deficiency, rapid weight loss phase, and small intestinal bacterial overgrowth (which may interfere with vitamin D absorption).

To try to prevent vitamin D deficiency and thus secondary metabolic bone disease, post-operative RYGB patients should receive at least 1.2 grams daily of elemental calcium and 800 international units (IU) of vitamin D daily. In those patients with low serum levels of 25-hydroxy vitamin D, 50,000 IU of vitamin D (ergocalciferol) taken orally once per week for six to eight weeks is prescribed with a recheck of the 25-hydroxy Vitamin D level after eight weeks to confirm repletion (79). The reported dose for treatment of rickets is at least 600,000 IU, which has been given as rapidly as 150,000 IU, taken four times daily during 1 day of treatment. In treatment of osteomalacia, significant improvement in patients' clinical symptoms, functional status, biochemical indices and bone mineral density has been reported after treatment with a combination of ergocalciferol (100,000 IU daily) and calcium carbonate (1 to 2.5 g daily).

6.2.3 Vitamin E

Vitamin E consists of tocopherols and tocotrienols. This fat-soluble vitamin is located in cell membranes, and it may be active in preventing lipid peroxidation. Most adults tolerate

doses of vitamin E up to 1000 mg/day (0.67 mg of vitamin E is 1 IU) without gross signs or biochemical evidence of toxicity (80).

Deficiency of vitamin E should be considered in bariatric patients who have visual symptoms (retinopathy), non-specific neurological symptoms (ataxia, dysarthria, muscle weakness due to myopathy, or ptosis), or hemolytic anemia. Treatment of vitamin E deficiency should include oral vitamin E 800 to 1200 IU daily.

6.2.4 Vitamin K

Vitamin K describes a group of compounds which contain the 2-methyl-1,4-naphthoquinone moiety. These compounds are essential for the formation of prothrombin, and five factors (factors VII, IX, and X, and proteins C and S) involved in regulation of blood clotting. Vitamin K is moderately (40 to 70%) well absorbed from the jejunum and ileum (81).

Absorption of vitamin K depends on the normal flow of bile and pancreatic secretion, and its absorption is enhanced by dietary fat. The total body pool of vitamin K is small. Most of the daily requirements for vitamin K is provided through biosynthesis by the intestinal flora. Deficiency of vitamin K leads to increase risk of bleeding disorders.

Vitamin K deficiency is rare after bariatric surgery. There is a report of 5 babies with intracranial hemorrhage whose mothers had undergone bariatric surgery (82), suggesting vitamin K deficiency. Replacement of vitamin K can be accomplished with an oral form (2.5 to 25 mg daily) or with parenteral delivery of vitamin K (5-15 mg, intramuscularly or subcutaneously).

6.3 Essential minerals

Most studies of essential minerals after bariatric surgery involve iron and calcium. It is not presently known whether long-term deficiencies occur with other minerals after bariatric surgery.

6.3.1 Iron

Development of anemia after bariatric surgery is common and the origins for anemia are complex. Many patients after bariatric surgery require iron supplementation for treatment of anemia. Several potential mechanisms may explain iron malabsorption after bariatric surgery, including relative achlorhydria (acid may improve absorption of non-heme iron from plant sources by oxidation of Fe^{2+} to the better-absorbed Fe^{3+} cation) and bypass of proximal small intestine (the major location for iron absorption). Identification of iron deficiency in a bariatric patient requires consideration of other potential gastrointestinal origins for the development of iron deficiency anemia. Routine treatment of iron deficiency includes use of an iron/vitamin C complex or 150 to 200 mg/day of oral elemental iron in any preparation (gluconate/sulfate/fumarate). Parenteral iron is occasionally needed in those patients who have a poor response to oral iron therapy, especially in premenopausal women with heavy menstrual bleeding (83).

It is not commonly known that iron supplementation has risks. Iron supplementation for whatever purpose should be monitored, since electron transfer from transition metals such as iron to oxygen-containing molecules can initiate free radical reactions. Large doses of unnecessary iron supplements could induce an acquired iron overload disorder.

6.3.2 Calcium

Vitamin D deficiency and calcium malabsorption can occur simultaneously. Vitamin D deficiency activates a metabolic cascade resulting in hypocalcemia and secondary hyperparathyroidism. Steatorrhea due to malabsorption induces calcium malabsorption through the interaction of dietary calcium with intraluminal triglycerides.

Isolated serum calcium measurement is not an adequate marker of calcium metabolism. Patients may present with complaints of bony pain, back pain, or aching of the limbs. Bariatric surgery patients can maintain normal serum calcium by decreasing urinary calcium secretion. Urine calcium secretion can however be altered by concomitant use of diuretics. Treatment of calcium deficiency requires minimally oral calcium of ≥ 1.2 grams daily and concomitant correction of vitamin D deficiency.

6.3.3 Iodine

There are no reports of iodine deficiency after bariatric surgery. Weight loss after bariatric surgery is associated with resolution of subclinical hypothyroidism (84).

6.4 Trace elements

Trace elements function as co-factors for antioxidant enzymes or proteins. Trace elements in supplements provide a relatively narrow range of safety between deficiency and toxicity. Because of their ability to donate or accept electrons, transition metals have potential antioxidant properties.

6.4.1 Zinc

Zinc is important in the bioactivity of hundreds of mammalian proteins and is a co-factor in cytosolic superoxide dismutase. Zinc may reduce the formation of the highly toxic hydroxyradical (OH^-) from H_2O_2 produced through the antagonism of redox-active transition metals, such as iron and copper (85). There are reports of biochemical zinc deficiency after bariatric surgery (86). However, clinical zinc deficiency in bariatric patients has not been well studied. Symptoms of zinc deficiency can include a dermatological eruption, alopecia, glossitis, hypoalbuminemia and nail dystrophy. Initial treatment of zinc deficiency is with oral zinc gluconate, 50 mg, taken every other day.

6.4.2 Copper

Copper is a co-factor in cytosolic superoxide dismutase as well as cytochrome oxidase. Animal studies support a linkage in the absorption of copper and zinc (most likely in the stomach and upper small intestine), but a recent study using a human cell line suggests that a different copper transport protein is active in copper absorption in humans (87). Ingestion of fructose appears to reduce the biological activity of copper, as demonstrated by decreasing the activity of superoxide dismutase in erythrocytes (88).

Copper deficiency may result from the use of liquid vitamin supplements that do not contain copper. The occurrence of decreased serum copper levels has been reported through case reports involving bariatric patients, generally >10 years after RYGB (89). Copper

deficiency in susceptible individuals can induce anemia and neutropenia, or pancytopenia (90, 91). There have also been, in the past several years, reports of RYGP patients who have developed a myelopathy-like disorder with spastic gait and sensory ataxia associated with low serum copper levels (92). The clinical and neuroimaging findings in these patients are similar to the findings identified in patients with vitamin B12 deficiency. Similar symptoms associated with low serum copper have been reported in individuals with Celiac sprue (93). This condition has been termed by Dr. Kumar "human swayback", which is an unfortunate description since research workers studying swayback in lambs have been reported to develop multiple sclerosis, raising the question of an infectious etiology (94). Unfortunately, copper supplements given to patients with low serum copper levels and symptoms of myeloneuropathy do not appear to lead to a significant improvement in their neurologic symptoms (95). Further work is needed to better understand the origin for and the treatment for this rare neurologic complication.

Optic neuropathy has been reported to occur in patients after bariatric surgery in association with low serum copper levels (96). However, copper infusion therapy had no effect on the optic neuropathy. From this report, it is therefore unclear whether the damage was irreversible or whether other micronutrient deficiencies may have been involved in the optic neuropathy.

Treatment of copper deficiency can begin with oral copper gluconate, 2 to 4 mg, taken every other day. Higher daily oral doses involving use of 6 mg copper have been reported to be required in some patients (97). Correction of copper deficiency in individual patients may necessitate intravenous infusions of copper chloride (98).

6.4.3 Selenium

Selenium is a trace element that is known to be essential for activation of glutathione peroxidase, a key enzyme in the body's defense against oxygen-derived free radicals. Selenium supplementation, alone and in combination with other micronutrients, has been extensively studied. Selenium deficiency induces a cardiomyopathy in those regions of the world in which selenium levels in the soil are low, as in China. There is a case report of a patient presenting with a severe cardiomyopathy 9 months after biliopancreatic diversion (99). Treatment of selenium deficiency begins with sodium selenite, 100 micrograms daily taken orally.

6.4.4 Chromium

It is not known whether chromium is a required cofactor in humans. Chromium deficiency has not been reported after bariatric surgery. The potential role of chromium in human nutrition is based on observations from patients receiving total parenteral nutrition. Case reports have discussed, in patients with total parenteral nutrition, development of an abnormal intravenous glucose tolerance test, weight loss, and peripheral neuropathy associated with decreased blood chromium levels (100).

6.4.5 Manganese

Manganese is an important cofactor in inducible mitochondrial superoxide dismutase. Manganese deficiency has not been reported after bariatric surgery. Deficiency of

manganese in animal models inhibits collagen deposition during wound healing and induces skeletal deformation.

7. Symptoms and findings of micronutrient deficiencies

7.1 Anemia

This is the most commonly recognized and treatable nutrient deficiency after bariatric surgery (101, 102). Patients with normal hemoglobin levels can have low ferritin levels after RYGB, supporting the addition of iron supplementation at that time. Iron deficiency anemia can be monitored by checking hemoglobin, hematocrit, and mean corpuscular volume as part of a complete blood count. Routine treatment of iron deficiency includes treatment with an iron/vitamin C complex or with 150 to 200 mg/day of oral elemental iron in any preparation (gluconate/sulfate/fumarate). If there is an incomplete response to oral iron therapy and vitamin B12 and an evaluation by a gastrointestinal specialist has not provided a specific diagnosis, one must then consider additional micronutrient deficiencies and other origins for anemia. Other nutritional origins of anemia must be excluded by examining levels of folate, zinc, copper, and vitamins A and E (see Table 5).

As mentioned above, a gastrointestinal specialist should be consulted when anemia does not correct with iron and vitamin B12 supplementation, in order to exclude blood (i.e. iron) loss from a colon source, a stomal ulcer, a duodenal ulcer, or antritis. Blood loss from the gastric remnant can at times be addressed by a double balloon enteroscopy. The lengths of the Roux limb and the pancreaticobiliary limb may preclude direct endoscopic visualization of the duodenum and bypassed stomach. If one needs to visualize the "bypassed" stomach and duodenum, this can be accomplished by intraoperative endoscopy performed through a laparoscopically-assisted gastrotomy, which will allow insertion of an endoscope directly into the bypassed stomach.

7.2 Neurologic symptoms

Neurologic complaints are reported by about 1% of post-operative patients in surveys, but are described by 5% of patients in prospective studies (103). A main determination is whether the neurologic complaint is indeed related to a post-operative disorder. Patients present most commonly with peripheral neuropathy after RYGB (104). Micronutrient deficiencies involving vitamin B12 and copper are reported in bariatric surgery patients who have been seen for neurologic symptoms (105). Reported neurologic emergencies include Wernicke's disease and Guillain-Barre syndrome.

In patients with neurologic symptoms, blood levels of vitamin B12, vitamin B2, vitamin B6, vitamin E, copper, thiamine, and niacin should be obtained (see Table 5). Physicians must remember that patients may expect to have neurological symptoms after RYGB (and therefore may not report their symptoms) and patients may believe that neurologic symptoms are related to their history of diabetes mellitus.

It is not clear whether the routine use of chewable multivitamins containing minerals after bariatric surgery prevent neurologic disorders. It is not known whether neurologic symptoms can be consistently reversed by treatment of specific micronutrient deficiencies. There are only anecdotal reports of the use of micronutrient infusions for the treatment of neurologic symptoms (a typical intravenous infusion would include, mixed in 5% dextrose

in aqueous solution, a standard injectable multivitamin formulation [several are commercially available] with a mixture of trace elements, such as Multitrace 5 concentrate, and both 100 mg thiamine hydrochloride and 1 mg folic acid). Revision of bariatric surgery in order to reduce the length of bypassed small intestine has been reported to be of clinical benefit in a patient with neurologic symptoms (105). Physicians should be cautious, should encourage strongly the use of supplemental multivitamins, and should routinely screen all patients for neurologic symptoms during their postoperative visits.

SYMPTOM	LABORATORY TESTING
Anemia	Ferritin; Vitamin B12; Folate; Zinc; Copper; Vitamins A; Vitamin E
Neurologic Symptoms	Vitamin B12; Vitamin E; Copper; Thiamine; Vitamin B2; Vitamin B6; Plasma Niacin
Visual Symptoms	Vitamin A; Vitamin E; Whole blood thiamine; Copper
Bleeding Disorder	Complete Blood Count; Prothrombin Time
Skin Disorders	Vitamin A; Vitamins B2; Vitamin B6; Zinc; Plasma Niacin
Edema	Selenium; Plasma Niacin; Whole Blood Thiamine

Table 5. Laboratory testing after bariatric surgery

7.3 Visual disorders

Manifestations of vitamin A deficiency include nocturnal visual difficulty and decreased visual acuity. Vitamin E deficiency can induce visual symptoms related to retinopathy. Patients with thiamine deficiency can present with complaints of difficulty focusing their vision or persistent blurred vision; on physical examination, nystagmus is often identified. Optic neuropathy has been reported in patients with deficiencies of copper and thiamine. In laboratory evaluation, one should consider obtaining serum levels of vitamin A, vitamin E, copper and whole blood thiamine (see Table 5).

7.4 Skin disorders

Symptoms of zinc deficiency include a dermatological eruption, but it is unclear whether this occurs after bariatric surgery. Manifestations of vitamin A deficiency include xerosis and pruritus. Essential fatty acid deficiency, niacin deficiency, and riboflavin deficiency can cause a scaly dermititis. The two essential fatty acids, linoleic acid and linolenic acid, are both present in flaxseed oil, soybean oil, and canola oil. One can consider ordering serum levels of vitamin B2, vitamin A, zinc, and plasma niacin (see Table 5).

7.5 Edema

Underlying heart failure is a major concern when a bariatric patient presents with edema. Patients with thiamine deficiency can develop high output cardiovascular disease (wet beriberi) and may present with tachycardia, bradycardia, respiratory distress, lower extremity edema, right > left ventricular dilation, and lactic acidosis. Selenium deficiency is another known cause of heart failure. Evaluation of edema could include determination of serum selenium, plasma niacin, and whole blood thiamine levels (see Table 5).

In considering other medical conditions, edema could be caused by obstructive sleep apnea. Edema can develop in patients with hypoalbuminemia. An underlying hepatic disorder, potentially the end result of steatohepatitis, should be considered. Other origins of hypoalbuminemia include an inflammatory process and small intestinal bacterial overgrowth. There is a serious syndrome of post-operative diarrhea associated with hypoalbuminenia and diffuse edema. This disorder may be induced by severe protein-calorie malnutrition due to a physiological biliopancreatic diversion, such as an extended (distal) Roux-en-Y gastric bypass or a duodenal switch. In addition, it has been reported that this syndrome improves with antibiotic therapy (106) supporting the role of small intestinal bacterial overgrowth. Finally, niacin deficiency can induce a diarrheal illness or colitis that may be responsible for development of hypoalbuminemia as an origin for peripheral edema.

8. Conclusions

There is an increasing prevalence of obesity in developed countries. Dietary intake may be both an origin for obesity as well as an origin for the development of micronutrient deficiencies. Since dietary and activity programs fail to produce sufficient weight loss in most obese individuals, bariatric surgery will continue to be the major therapeutic options for patients with medically-complicated obesity. Many patients after bariatric surgery will develop micronutrient deficiencies despite suggestions for the use of ongoing vitamin and mineral supplementation. Common micronutrient deficiencies after bariatric surgery include deficiencies of iron, vitamin B12, thiamine, and vitamin D. The risks of micronutrient deficiencies are highest in those individuals who have undergone a malabsorptive surgical procedure. Other origins for micronutrient deficiencies include the utilization of pharmacological agents or dietary supplements, and the presence of upper gut or small intestinal bacterial overgrowth. Micronutrient deficiencies must be considered when patients develop specific symptom complexes. It is not yet known whether an ongoing survey of symptoms or a regularly scheduled determination of blood levels of micronutrients will prove to be the best detection method to screen bariatric patients for micronutrient deficiencies.

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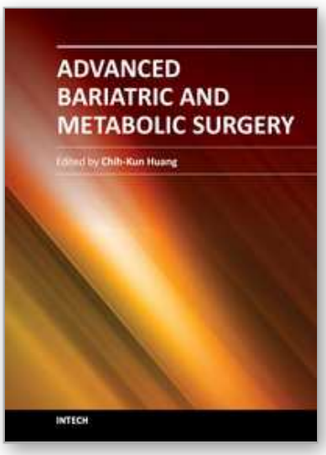
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Bariatric surgery has gained importance in the last 20 years because of the high prevalence of global obesity, and the vast understating of the physiological and pathological aspects of obesity and associated metabolic syndromes. This book has been written by a number of highly outstanding authors and pioneering bariatric surgeons from all over the world. The intended audience for this book includes all medical professionals involved in caring for bariatric patients. The chapters cover the choice of operation, preoperative preparation including psychological aspect, postoperative care and management of complication. It also extends to concept and result of metabolic surgery and scarless bariatric surgery.

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