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Recognizing postoperative nutritional complications of bariatric surgery in the primary care patient: a narrative review

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Abstract: Bariatric surgery is an effective treatment for patients with morbid obesity. However, as safe and common as bariatric procedures have become, multiple complications can still result. These complications vary depending on the type of procedure performed (malabsorptive or restrictive) and are often nutritional derangements from the altered malabsorptive surface of the gastrointestinal tract and decreased capacity of the stomach. Deficiencies in vitamin D after malabsorptive procedures such as the Roux-en-Y gastric bypass can result in subsequent hypocalcemia and bone demineralization, and anemias can also present after surgery from inadequate vitamin B₁₂ and iron absorption. Because of the prevalence of these deficiencies, baseline micronutrient testing and postoperative screening are recommended in many cases. Additionally, supplemental treatment often requires higher doses than those recommended for healthy adults. The purpose of this narrative review is to outline the various nutrient deficiencies that can result from bariatric procedures and report previously-published recommendations for screening and medical treatment of patients with these deficiencies. This review is directed toward primary care practitioners because of their unique position in delivering continuity of care and the frequency with which they will encounter patients who have undergone bariatric surgery and are seeking counseling regarding weight loss modalities.

Obesity, defined as a body mass index (BMI) that is greater than or equal to 30 kg/m²,¹ is a chronic disease of increasing prevalence in the United States. According to data from the Centers for Disease Control and Prevention, 42.4% of adults 18 years of age and older were considered obese in 2017 to 2018.² Global health observatory data from the World Health Organization showed that in 2016, 39% of the world's adult population 18 years of age and older was overweight and 13% of the world's adult population was obese.³ Despite the multiple approaches available for managing weight loss in patients with obesity (including lifestyle and behavioral changes as well as medications), bariatric surgery is associated with the most successful rate of weight loss and resolution of associated comorbidities.⁴ The number of bariatric surgeries performed each year is estimated to be increasing.⁵ The increase in number of procedures performed is likely multifactorial and a result of increased public awareness of significant negative health outcomes associated with obesity as well as awareness and general acceptance of bariatric surgery as a successful modality for treatment of obesity, patient success in achieving sustained weight loss, and decreased rates of mortality from obesity-related comorbidities. The qualifying criteria for patients seeking bariatric surgery is shown in Figure 1.

Bariatric surgeries can be classified as restrictive, malabsorptive, or a combination of both. Restrictive procedures include laparoscopic adjustable gastric banding (LAGB) and laparoscopic sleeve gastrectomy (LSG).^{6,7} The Roux-en-Y gastric bypass (RYGB) is both a restrictive and malabsorptive procedure, although largely restrictive. The predominant malabsorptive procedure is the biliopancreatic diversion (BPD) and duodenal switch (DS).^{6,7} The most common bariatric procedure performed today is the LSG; the American Society for Metabolic and Bariatric Surgery (ASMBS) estimated in 2018 that 61.4% of bariatric

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- BMI of ≥ 40 kg/m² without coexisting medical problems
- BMI of ≥ 35 kg/m² and at least 1 obesity-related comorbidity^a
- BMI of 30–34.9 kg/m² with diabetes or metabolic syndrome (limited evidence)

Figure 1: Patient criteria for bariatric surgery.^{6,10}

^a Obesity-related comorbidities include hypertension, type 2 diabetes mellitus, hyperlipidemia, obstructive sleep apnea, nonalcoholic fatty liver disease, gastrointestinal reflux disease, and asthma.

Abbreviations: BMI, body mass index.

procedures performed were LSG, while 17.0% were YGB and 1.1% were LAGB.⁵

Despite the proven efficacy of bariatric surgery in achieving weight loss and reducing the incidence of associated comorbidities, there are many complications that can occur. Nutritional derangements are common, long-term complications of bariatric procedures resulting from obesity-related malnourishment, noncompliance with postoperative supplementation, and the malabsorptive nature of the procedures.^{7–9} It is thought that restrictive procedures (LAGB and LSG) are less likely to result in nutritional deficiencies than malabsorptive procedures;⁶ however, a previous prospective, randomized trial⁸ showed no difference in nutritional deficiencies after RYGB compared to LSG, although the authors noted that both groups had higher rates of deficiencies. Standard multivitamin supplementation does not often adequately prevent nutritional deficiencies after procedures like the RYGB.¹⁰ Because of the high prevalence of obesity and the increasing number of bariatric procedures, it is important that primary care physicians are familiar with postoperative management of patients who have had bariatric surgery. The purpose of this narrative review was to outline the various nutritional deficiencies that can result from bariatric procedures and review published recommendations for screening and medical management of these deficiencies.

Literature search

We performed an electronic search through PubMed/MEDLINE and the Free Medical Journals online database in November 2017 using the keywords “bariatric surgery complications” and “adverse effects.” Our search yielded 604 results. Criteria included articles in English, published in the last 10 years (2008–2018), and those for which a free full-text version was available. Additional keywords included “nutritional complications,” “nutritional

deficiencies,” and “postoperative complications,” application of which narrowed results to 108 articles. Two authors (C.C., R.T.) reviewed the titles and abstracts of those articles for topic relevance; those that specifically addressed the nonpregnant, adult population of patients who had undergone bariatric surgery and focused on nutritional complications following surgery were included. Articles that included pregnant or pediatric patients were excluded from our review. After review of abstracts, 50 articles met criteria for full text review. Further review of full text for relevance was performed and related specifically to nutritional deficiencies after surgery, including etiology, pathophysiology, diagnosis, and supplementation. After final review of full text, 29 articles were determined to be appropriate and relevant to this narrative review (Figure 2).

Fat-soluble vitamin deficiencies

Vitamin A, D, E, and K deficiencies

Malabsorptive procedures are known to cause fat-soluble vitamin deficiencies related to postoperative steatorrhea and decreased surface area for absorption. Routine screening for fat-soluble vitamin deficiencies can be performed after malabsorptive procedures, although typically this is only recommended for vitamin A deficiency.⁶ In a randomized, controlled trial¹¹ comparing 60 patients considered superobese (BMI 50–60 kg/m²) who were undergoing either DS or RYGB, the authors found that the concentration of vitamin A decreased in both the gastric bypass group ($P=0.003$) and the duodenal switch group ($P<0.001$), but to a larger degree in the DS group. Vitamin A deficiency can cause poor healing, skin changes including xerosis and acne, brittle hair, and eventually blindness from xerophthalmia.¹² Supplementation of 5,000 to 10,000 IU/day may be indicated until levels normalize.¹³ Annual screening is not recommended but can be performed after a malabsorptive procedure by measuring plasma retinol levels.^{9,11,13} Vitamin K deficiencies, although uncommon, can also occur postoperatively and result in coagulopathies, easy bruising, or increased bleeding.¹³ Signs of increased bleeding could include petechiae, purpura, or hematoma formation.¹² However, the authors of a research study¹³ in which vitamin K levels were low in 50% to 60% of patients following BPD procedure did not observe clinical signs of K deficiency.¹³ Previous guidelines agree that there is insufficient evidence to support screening for K

Figure 2: Flow chart depicting literature search results and inclusion/exclusion criteria.

^aAdditional search criteria included: free full text, available in English, published in the last 10 years. ^bArticles were excluded after full text review determined them irrelevant for this topic.

deficiencies.⁶ If a vitamin K deficiency is discovered, supplementation should be provided when the International Normalized Ratio (INR) rises above 1.4 INR units.¹³

Previous authors¹³ have recommended against screening for vitamin E deficiencies, which are uncommon but can present with rash (atopic dermatitis or acne), ataxia, muscle weakness, or loss of position or vibratory sense.^{12,14,15} For asymptomatic patients, multivitamin supplementation is sufficient; supplementation of 100 to 400 U/day is recommended when patients are symptomatic.^{14,15}

Vitamin D deficiency is often undiagnosed before surgery in patients with obesity and is a common postoperative finding.^{16,17} Because of the prevalence of deficiency in the general population, preoperative screening is recommended by measuring 25-hydroxy vitamin D levels.^{13,16,17} It is suspected that vitamin D deficiency is prevalent in patients with obesity because of decreased bioavailability of vitamin D, as it is sequestered in adipose tissue, and decreased expression of vitamin D metabolizing enzymes.¹⁷ Of patients who have undergone a malabsorptive procedure such as the RYGB, 17% to 52% develop a vitamin D deficiency by 2 years postoperatively and 50-63% develop deficiency within 4 years.¹³ Because 1,25-dihydroxy-vitamin D3 plays a role in cell growth regulation and differentiation, vitamin D deficiency can involve dermatological manifestations such as atopic dermatitis.¹²

With deficient levels of vitamin D comes reduced intestinal absorption of calcium and phosphorous. After malabsorptive procedures such as RYGB, 10% to 25% of patients develop a calcium deficiency by 2 years and 25% to 48% develop deficiency by 4 years postoperatively.¹³ In addition to vitamin D deficiency contributing to hypocalcemia, poor calcium absorption following RYGB procedures results from food bypassing the duodenum, which is the area of highest concentration of calcium transporters.^{14,16} Patients can be screened for hypocalcemia by measuring serum calcium levels; however, these levels are not entirely accurate, and use of dual-energy X-ray absorptiometry scans and Z-scores can more accurately depict calcium-related bone disease.¹⁶ Severe hypocalcemia can present as muscle stiffness, muscle cramping, perioral or acral paresthesia, or psychiatric changes such as emotional instability. Hypocalcemia can subsequently cause secondary hyperparathyroidism leading to decreased bone density, osteomalacia, or rickets.^{9,15}

A review¹⁸ comparing nutritional deficiencies in patients after RYGB and LSG procedures referenced studies that showed increases in bone mineral density and vitamin D 1 year after GS. However, there are multiple conflicting studies and reviews that show decreases in bone mineral density and vitamin D levels, validating the practice of high-dose vitamin D supplementation, even after LSG.^{10,16-19} For postoperative bariatric surgery patients,

supplementation of 400-800 U/day of vitamin D2 (ergocalciferol) or D3 (cholecalciferol) is recommended for prevention of vitamin D deficiency.¹⁹ Treatment of deficiencies requires doses up to 50,000 IU of D2 (ergocalciferol) 1 to 3 times per week.^{13,16,17,19} It is necessary to correct vitamin D deficiencies to improve calcium levels and prevent bone density loss.

Supplementation of oral calcium citrate is also essential to prevent hypocalcemia.^{6,13} Calcium citrate supplementation doses differ depending on the type of procedure undergone by the patient. Lower supplementation of 1,200 to 1,500 mg/day is typically required after restrictive procedures (i.e., LAGB, LSG) compared with the 1500 to 2000 mg/day that can be required after malabsorptive procedures (i.e., RYGB).^{6,15,16,19,20} It is important that patients take their calcium carbonate supplements with meals to enhance the absorption.

Screening recommendations for patients who have undergone RYGB, GS, or BPD procedures include measurement of vitamin D, calcium, phosphorus, PTH, and alkaline phosphatase levels every 6 months and dual-energy x-ray absorptiometry (DEXA) scans for bone density yearly until levels are stable.¹³

Hyperoxaluria

Separately from obesity causing higher prevalence of kidney stones in adults than in the non-obese population, RYGB surgery is also associated with an increased risk of nephrolithiasis secondary to hyperoxaluria.^{6,14,21} This increased incidence of hyperoxaluria has been found to be as high as 74% at 6 months postoperatively.¹⁴ Postoperative bariatric patients typically have fat malabsorption, which allows more fat to bind calcium in the gut. With calcium bound to fat, there is less calcium available to bind oxalate, and the increased free oxalate allows for oxalate nephropathy. In a cross-sectional study comparing patients who had undergone RYGB with a control group of patients who had obesity but no history of nephrolithiasis, postoperative patients had a higher prevalence of hyperoxaluria (47% vs 10.5%; $P=0.02$) and hypocitraturia (63% vs 5%; $P<0.01$) compared to obese controls.²¹ Patients with hyperoxaluria can present with acute renal failure, indicated by increasing serum creatinine, or nephrolithiasis, which can quickly progress to end-stage renal disease.¹⁴ Avoidance of dehydration, a low oxalate meal plan, oral calcium, and potassium citrate therapy can be utilized to manage hyperoxaluria and calcium oxalate stones.⁶

Protein deficiencies

Protein malnutrition, defined by hypoalbuminemia (albumin <3.5 mg/dl), is a long-term complication of BPD procedures and treatment of severe cases require hospitalization with 2 to 3 weeks of parenteral nutrition.¹³ After surgery, bariatric patients have a higher daily requirement for protein. Deficiency in protein intake by the patient is often due to intolerance to protein-rich foods and reduced total caloric intake after bariatric surgery, and it can present with weakness, decreased muscle mass, or generalized edema.¹³ Insufficient protein intake can cause increased hair loss and is a major contributor to poor wound-healing.⁹ Protein can be monitored by checking laboratory values for serum albumin, prealbumin, and serum creatinine.⁹ Guidelines recommend 60 to 120 grams of protein daily in all postoperative patients,^{13,19} which allows for maintenance of lean body mass during the weight loss process.

Anemia

Iron deficiency anemia is among the most common micronutrient deficiencies, occurring in up to 12 to 47% of patients after malabsorptive procedures and is often present before bariatric surgery in up to 44% of adults.^{9,16,22} Deficits in iron can be related to the suspected low-grade chronic inflammation present in obesity, allowing for release of hepcidin which subsequently block iron absorbing proteins.^{16,23,24} One study²⁵ measured the iron status of menstruating women after RYGB and found that 18 months after the procedure, more than 1/3 of the patients had become anemic and of these anemic patients, almost 60% were classified as having iron deficiency anemia. Consistent with another study,²⁶ these women became anemic and were deficient in iron (indicated by hemoglobin levels and iron panel studies) despite being prescribed supplemental iron during the study period.^{25,26} Iron deficiency anemia is most prevalent in women with menorrhagia who undergo bariatric surgery and therefore prophylactic iron supplementation is recommended in these patients.¹³ Conversely, a previous metaanalysis²² found that although iron levels did not significantly change after 24-month follow up, ferritin levels continued to decline in this same follow up time interval.²²

Another cause of iron deficiency anemia is bypassing the primary location of iron absorption, the duodenum and proximal jejunum, after procedures such as the RYGB.^{23,25,26} In a prospective study²⁶ of 58 menstruating women who underwent either RYGB or LSG, researchers found that heme

and nonheme-iron absorption decreased after both procedures on 12-month follow up ($P < 0.001$). The diet patients consume after these procedures is often lacking in red meat and leads to decreased heme intake.^{13,25} Calcium supplements are an additional factor that can decrease heme-iron absorption. Up to 50% to 60% of iron absorption is inhibited when it is consumed in conjunction with calcium or dairy products, so it is recommended that patients take iron and calcium at least 2 hours apart.^{13,20}

Patients with iron-deficiency anemia can be asymptomatic or they can present with generalized symptoms of fatigue and weakness, or pica in severe cases.^{13,27} Dermatological manifestations of iron deficiency could include pallor, glossitis, or alopecia.^{12,27} Anemia is typically of the microcytic, hypochromic type and diagnosis can be accurately made by measuring total iron binding capacity and serum transferrin levels (both will be increased in iron deficiency).¹⁶ Prevention of iron deficiency can be achieved with a multivitamin containing 45 to 60 mg of elemental iron taken 2 times per day.^{13,19} Standard multivitamin with iron may prevent iron deficiency but may still allow for anemia.¹³ Treatment regimens for iron deficiency, in addition to multivitamin supplementation, include oral ferrous sulfate, fumarate, or gluconate to provide up to 150 to 300 mg of iron daily.^{6,9,13,16} Vitamin C increases iron absorption and should be included with iron supplementation to maximize efficacy.²⁷ If deficiency persists despite iron supplementation, calcium and vitamin C levels should be measured to assess compounding variables.¹⁶ Continued surveillance of hemoglobin and iron studies is recommended for follow up.^{13,22}

A 2017 review¹⁸ cited a particular study that showed increasing rates of anemia in patients after LSG procedures (5% prior to procedure compared to 6.5% post-operatively) but decreasing rates of iron deficiency (38% prior to LSG compared to 18.5% postoperatively), suggesting alternative nutritional derangements leading to anemia.¹⁸ Vitamin B12 deficiency is a product of delayed or lack of mixing with parietal cell protein, intrinsic factor, causing impaired absorption (intrinsic factor protein is largely responsible for absorption of vitamin B12 in the small intestine).¹⁸ Vitamin B12 deficiency after RYGB can occur in anywhere from 33% to 40% of patients 1 year after surgery (8%-37% by postoperative year 2-4), and anemia can occur in more than 30% of patients 1 to 9 years after RYGB.¹³ Symptoms of vitamin B12 deficiency include anemia, neurological dysfunction, ataxia, paresthesias, weakness, loss of position and vibratory sense, and vision loss.^{9,15,27}

Multiple guidelines recommend baseline and post-operative evaluation for vitamin B12 deficiency in all bariatric surgery patients and annually for LSG or RYGB patients.^{6,9} To maintain normal B12 levels, oral

supplementation with crystalline vitamin B12 1000 µg/day (at least 350-400 µg/day)⁶ is indicated within 6 months of surgery to prevent the development of deficiencies.¹³ Other courses of B12 replacement include 500 to 1000 µg weekly intranasally or 1000 to 3000 µg every 6 to 12 months of parenteral B12 if oral or intranasal routes are insufficient.^{6,13}

Folate deficiencies are another cause of anemia in postoperative bariatric surgery patients, although typically seen less often because of the high amounts of folic acid fortified foods and vitamin supplementation available.^{4,9} If present, the deficiency is most likely secondary to decreased intake of folate-rich foods rather than malabsorption.⁵ Diagnosis of a megaloblastic anemia caused by vitamin B12 or folate would include an increased homocysteine level.⁹ Megaloblastic anemia caused by either vitamin B12 or folate deficiencies can be differentiated by measurement of a plasma methylmalonic acid level, which is elevated in a patient with vitamin B12 deficiency and normal when folate is deficient.⁹ Nutritional supplementation of folate should be at least 400 µg/day.^{13,15}

Thiamine deficiency

Wernicke encephalopathy is a serious complication of bariatric surgery involving a deficiency in vitamin B1 (thiamine). A systematic review²⁸ with a sample of 84 procedures, 80 (95%) of which were gastric bypass or restrictive procedures, showed that admission to the hospital for Wernicke encephalopathy occurred in 79 cases (94%). Risk factors noted in that review included bypass of the jejunum (the site of thiamine absorption), persistent emesis, and administration of glucose without thiamine.^{13,15,28} Other risks that could predispose patients to Wernicke encephalopathy include rapid weight loss, loss of appetite and failure to take prescribed vitamin supplements. Screening for thiamine deficiency following bariatric surgery is not indicated and only considered in patients with persistent emesis or signs of Wernicke encephalopathy, including eye movement abnormalities, mental status changes, peripheral neuropathy, and ataxia.^{9,15,28} Visual hallucinations, behavioral disturbances, and depression were less common symptoms according to a previous systematic review.²⁸ Mild thiamine deficiencies can be treated with 100 mg/d of thiamine for 7 to 14 days with an IV solution not containing glucose, followed by at least 10 mg/day of oral thiamine (50-100mg/day preferred) until symptoms resolve.^{6,13} Recommended treatment for severe thiamine deficiency is 500 mg/d of thiamine IV for 3 to 5 days, followed by 250 mg/d for another 3 to 5 days or until symptom resolution.^{6,13,28}

Rare deficiencies

Rare micronutrient deficiencies after bariatric surgery can include zinc, copper, and selenium. Copper plays an essential role in iron absorption and red blood cell formation, and it can be an underlying cause of persistent anemia thought to be related to iron or vitamin B12 deficiencies. Copper deficiency can present as an anemia or with neurological manifestations of unsteady gait or neuropathies.^{15,19,23} There is no recommendation for screening of copper deficiency; however, if anemia persists despite iron or vitamin B12 supplementation, copper levels should be measured.^{16,19} Copper should be part of any multivitamin supplementation for postoperative bariatric surgery patients. In patients with mild to moderate deficiencies, oral copper sulfate or gluconate 3-8 mg/d can be given until symptoms resolve.^{6,16,19}

Zinc deficiency is highly prevalent in obese patients before bariatric surgery and remains common following malabsorptive and restrictive procedures. One review¹⁹ cited zinc deficiency in 12% to 13% of patients after LSG, 21% to 33% after RYGB, and 74% to 91% after BPD, while another review⁶ found 42% of patients after RYGB and 92% after BPDDS. Zinc, like copper, is an essential cofactor for many enzymes and plays a large role in cell division and growth. A zinc deficiency can present as alopecia, poor wound healing, pica, or in men, as hypogonadism or erectile dysfunction.^{6,29} There are multiple formulations of zinc replacement. ASMBS recommends 60 mg of elemental zinc twice per day.¹⁹ Furthermore, zinc supplementation can impair the absorption of copper, and it is recommended to supplement 1 mg of copper for every 8 to 15 mg of zinc.^{6,19}

Finally, magnesium deficiency, which is common in preoperative patients with obesity, can also be found after bariatric procedures. It is an essential intracellular cation and cofactor for many enzymes. Because magnesium also contributes largely to bone formation, deficiencies can lead to osteoporosis in postoperative patients.¹⁹ Although there are no specific recommendations for screening and definitive supplementation, postoperative patients are typically provided with long-term proton pump inhibitor (PPI) therapy, further contributing to hypomagnesemia; therefore, prevention of hypomagnesemia can include supplementation with 300 mg of magnesium citrate daily.¹⁹

Recommendations for screening and management of postoperative micronutrient deficiencies as discussed in this review are summarized in the included Table 1.

Table 1: Nutritional deficiencies after bariatric procedures and guidelines for management.

Nutrient deficiency	Recommended supplementation or treatment	Screening recommendations for patients after RYGB, BPD, BPD/DS ^{6,9,13,15}
Iron	Ferrous sulfate 300mg 2-3 times/day taken with vitamin C ^{13,19} PO supplementation Take iron and calcium supplements at least 2 hours apart ²⁰	Measure preoperatively, every 6 months for up to 2 years and then annually ²⁴
Vitamin B12	Crystalline vitamin B12 at 250-350 µg/day PO ^{9,13,19,20} or: 500-1000 µg intranasal or sublingually weekly ¹³ 1000 µg every month or 3000 µg every 6 months of parenteral B12 if oral or intranasal routes are insufficient ^{6,13,19,20}	Measure preoperatively, every 6 months for up to 2 years, and then annually
Folate	Multivitamin supplement with at least 400 µg/d of folate ^{6,13}	Measure preoperatively, every 6 months for up to 2 years, and then annually
Fat-solubles (ADEK)	Vitamin A: If symptoms present, 5,000 IU/day after LAGB; up to 10,000 IU/day after RYGB, LSG, BPD/DS ¹³ Vitamin E: 100-400 U/day ¹⁴ if symptoms Vitamin K: 1 mg/day ¹⁴ if symptoms	Vitamin A screening preoperatively, then optional post-operatively Screening not recommended for vitamin E or K
Vitamin D	400-800 U/day of D2 (ergocalciferol) ^{6,9,13,19,20} 50,000 IU of D2, 1-3 times per week for severe cases ^{6,16} Titrated to maintain a 25(OH) D level of >30 ng/mL ^{6,9,13,27}	Measure preoperatively, every 6 months for up to 2 years and then annually Monitor bone mineral density with DEXA scan with Z-scores ¹⁶
Calcium	Calcium citrate dose of: 1,200-1,500 mg/day after LAGB and LSG ^{6,9,13,20,27} 1,500-2,000 mg/day after RYGB ^{9,13,19,20}	Preoperatively, every 6 months for up to 2 years and then annually
Vitamin B1 (thiamine)	100mg/d of thiamine for 7-14 days with IV solution (without glucose), followed by at least 10 mg/d PO thiamine (50-100 mg/d preferred) until symptoms resolve (or indefinitely) ^{9,13,19} 500 mg IV for 3 days when severe followed by 250 mg/d for 3 to 5 days until symptoms resolve ^{6,19,28}	Measure levels depending on clinical presentation
Protein	Mild: Protein supplementation 60-120 g/day Severe: Hospitalization	Albumin/pre-albumin measured preoperatively, every 6 months

Table 1: (continued)

Nutrient deficiency	Recommended supplementation or treatment	Screening recommendations for patients after RYGB, BPD, BPD/DS ^{6,9,13,15}
	2-3 days of parenteral nutrition ^{13,19}	for up to 2 years post-operatively and then annually
Magnesium	300 mg magnesium citrate daily Supplement with chronic PPI therapy ¹⁹	No screening recommendations
Copper	1 mg of copper for each 8-15 mg zinc ¹⁹	Measure copper levels when anemic despite iron or B12 supplementation ^{6,19}
Zinc	Oral zinc gluconate, sulphate, or acetate to provide 8-15 mg elemental zinc (up to 200% of recommended daily allowance ¹⁹	Routine screening after malabsorptive procedures ⁶

Abbreviations: BPD, biliopancreatic diversion; BPD/DS, biliopancreatic diversion/duodenal switch; IV, intravenous; LAGB, laparoscopic adjustable gastric banding; LSG, laparoscopic sleeve gastrectomy; PO, by mouth; PPI, proton pump inhibitor; RYGB, roux-en-Y gastric bypass.

Discussion

A limitation of this narrative review is that we restricted our search to 2 online databases and our search was limited to free articles available to the public. More head-to-head comparisons of the common bariatric procedures would allow for more guidance in selection of procedures.

Because of the increasing prevalence of both obesity and bariatric procedures, it is essential for the primary care physician to be knowledgeable about the various nutritional derangements commonly encountered postoperatively in these patients. Our literature review showed that the incidence of nutritional deficiencies postoperatively can be high; these micronutrient deficits also are often interrelated.¹⁶ Alterations to the anatomy of the stomach and digestive tract can lead to vitamin and mineral malabsorption that can adversely affect a patient's health. Careful attention is necessary to minimize the health risks associated with malabsorption. Regardless of the amount of weight loss achieved, osteopathic physicians understand that nutrition can play an integral role in physical and emotional well-being.

Conclusion

Bariatric surgery is an effective method for weight loss in patients with obesity who also have comorbidities. Despite the success of these procedures in achieving weight loss, a multitude of complications can arise in the postoperative period. Nutritional deficiencies are common complications of bariatric procedures that can persist for months to years after surgery. Primary care physicians are in a unique position to manage these patients because of their familiarity with chronic medical conditions and their ability to provide much-needed continuity of care.

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References

- Schroeder R, Harrison TD, McGraw SL. Treatment of Adult Obesity with Bariatric Surgery. *Am Fam Physician*. 2016;93(1): 31-37.
- Hales CM, Carroll MD, Fryar CD, et al. *Prevalence of Obesity and Severe Obesity Among Adults: United States, 2017–2018*. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, February 2020. Hyattsville, MD: National Center for Health Statistics, p.1. <https://www.cdc.gov/nchs/data/databriefs/db360-h.pdf>. Accessed August 25, 2020.
- Obesity and overweight. World Health Organization website. Updated April 1, 2020. <http://www.who.int/mediacentre/factsheets/fs311/en/>. Accessed August 25, 2020.
- Salminen P, Helmiö M, Ovaska J, et al. Effect of laparoscopic sleeve gastrectomy vs laparoscopic roux-en-y gastric bypass on weight loss at 5 years among patients with morbid obesity. *JAMA*. 2018; 319 (3):241. doi:10.1001/jama.2017.20313.

5. Estimate of Bariatric Surgery Numbers, 2011-2018. American Society for Metabolic and Bariatric Surgery. Updated June 2018. <https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers>. Accessed August 25, 2020.
6. Mechanick J, Youdim A, Jones D, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2013 update: cosponsored by american association of clinical endocrinologists, the obesity society, and american society for metabolic & bariatric surgery. *Endocrine Practice*. 2013;19(2):337-372. doi:10.4158/ep12437.gl.
7. Pories W. Bariatric surgery: risks and rewards. *J Clin Endocr Metab*. 2008;93(11 suppl 1):S89-S96. doi:10.1210/jc.2008-1641.
8. Peterli R, Wölnerhanssen BK, Vetter D, et al. Laparoscopic sleeve gastrectomy versus roux-y-gastric bypass for morbid obesity-3-year outcomes of the prospective randomized swiss multicenter bypass or sleeve study (SM-BOSS). *Ann Surg*. 2017;265(3):466-473. doi:10.1097/SLA.0000000000001929.
9. Xanthakos S. Nutritional deficiencies in obesity and after bariatric surgery. *Pediatr Clin North Am*. 2009;56(5):1105-1121. doi:10.1016/j.pcl.2009.07.002.
10. Gasteyer C, Suter M, Gaillard R, Giusti V. Nutritional deficiencies after roux-en-y gastric bypass for morbid obesity often cannot be prevented by standard multivitamin supplementation. *Am J Clin Nutr*. 2008;87(5):1128-1133. doi:10.1093/ajcn/87.5.1128.
11. Aasheim E, Björkman S, Søvik T, et al. Vitamin status after bariatric surgery: a randomized study of gastric bypass and duodenal switch. *Am J Clin Nutr*. 2009;90(1):15-22. doi:10.3945/ajcn.2009.27583.
12. Manzoni A, Weber M. Skin changes after bariatric surgery. *An Bras Dermatol*. 2015;90(2):157-166. doi:10.1590/abd1806-4841.20153139.
13. Heber D, Greenway F, Kaplan L, et al. Endocrine and nutritional management of the post-bariatric surgery patient: an endocrine society clinical practice guideline. *J Clin Endocr Metab*. 2010;95(11):4823-4843. doi:10.1210/jc.2009-2128.
14. Chauhan V, Vaid M, Gupta M, Kalanuria A, Parashar A. Metabolic, renal, and nutritional consequences of bariatric surgery: implications for the clinician. *South Med J*. 2010;103(8):775-785. doi:10.1097/smj.0b013e3181e6cc3f.
15. Becker D, Balcer L, Galetta S. The neurological complications of nutritional deficiency following bariatric surgery. *J Obes*. 2012;2012:1-8. doi:10.1155/2012/608534.
16. Gletsu-Miller N, Wright B. Mineral malnutrition following bariatric surgery. *Adv Nutr*. 2013;4(5):506-517. doi:10.3945/an.113.004341.
17. Chakhtoura M, Nakhoul N, Shawwa K, et al. Hypovitaminosis D in bariatric surgery: a systematic review of observational studies. *Metabolism*. 2016;65(4):574-585. doi:10.1016/j.metabol.2015.12.004.
18. Emile S, Hossam E. Nutritional deficiency after sleeve gastrectomy: a comprehensive literature review. *EMG Gastroenterol*. 2017;6(1): 99-105.
19. Stein J, Stier C, Raab H, Weiner R. Review article: the nutritional and pharmacological consequences of obesity surgery. *Aliment Pharmacol Ther*. 2014;40(6):582-609. doi:10.1111/apt.12872.
20. Schroeder R, Harrison TD, McGraw SL. Treatment of adult obesity with bariatric surgery. *Am Fam Physician*. 2016;93(1):31-37.
21. Maalouf NM, Tondapu P, Guth ES, et al. Hypocitraturia and hyperoxaluria after roux-en-y gastric bypass surgery. *J Urol*. 2010;183(3):1026-1030. doi:10.1016/j.juro.2009.11.022.
22. Weng TC, Chang CH, Dong YH, et al. Anaemia and related nutrient deficiencies after roux-en-y gastric bypass surgery: a systematic review and meta-analysis. *BMJ Open*. 2015;5(7):e006964. doi:10.1136/bmjopen-2014-006964.
23. Chen M, Krishnamurthy A, Mohamed AR, Green R. Hematological disorders following gastric bypass surgery: emerging concepts of the interplay between nutritional deficiency and inflammation. *Biomed Res Int*. 2013. doi:10.1155/2013/205467.
24. Muñoz M, Botella-Romero F, Gómez-Ramírez S, et al. Iron deficiency and anaemia in bariatric surgical patients: causes, diagnosis and proper management. *Nutr Hosp*. 2009;24(6):640-654. doi:10.3305/nh.2009.24.6.4547.
25. Ruz M, Carrasco F, Rojas P, et al. Iron absorption and iron status are reduced after roux-en-y gastric bypass. *Am J Clin Nutr*. 2009;90(3):527-532. doi:10.3945/ajcn.2009.27699.
26. Ruz M, Carrasco F, Rojas P, et al. Heme- and nonheme-iron absorption and iron status 12 mo after sleeve gastrectomy and roux-en-y gastric bypass in morbidly obese women. *Am J Clin Nutr*. 2012;96(4):810-817. doi:10.3945/ajcn.112.039255.
27. Lee PC, Dixon J. Bariatric-metabolic surgery: a guide for the primary care physician. *Aust Fam Physician*. 2017;46(7):465-471.
28. Aasheim ET. Wernicke encephalopathy after bariatric surgery: a systematic review. *Ann Surg*. 2008;248(5):714-720. doi:10.1097/SLA.0b013e3181884308.
29. Cunha SF, Gonçalves GA, Marchini JS, et al. Acrodermatitis due to zinc deficiency after combined vertical gastroplasty with jejunoileal bypass: case report. *Sao Paulo Med J*. 2012;130(5):330-335. doi:10.1590/s1516-31802012000500010.