

Management and Prevention of Surgical and Nutritional Complications After Bariatric Surgery



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KEYWORDS

• Bariatric surgery • Complications • Nutrition

KEY POINTS

- Bariatric surgery procedures are safe and carry a low risk of complications, especially in the setting of specialized centers.
- Intraoperative complications should be recognized and their management is usually straightforward.
- Surgeons should be aware of postoperative complications and have a high index of suspicion when caring for bariatric patients.
- All patients after bariatric surgery should be on nutritional (proteins, vitamins, and minerals) supplements, for life.

Bariatric and metabolic surgery has proven health benefits, such as weight loss (and most importantly long-term weight loss maintenance) and resolution of comorbidities associated with obesity. The field has been revolutionized in the last decades with the advent and diffuse adaptation of laparoscopy as a standard of care and the development of centers of excellence.¹ Many large cohort studies demonstrated a decrease in the complication rates (most notably mortality, surgical site infections [SSI], and cardiopulmonary events) of a laparoscopic approach over an open approach.^{2,3} This is reflected by the increase of bariatric procedures in the last decade.⁴ This article reviews the surgical and nutritional complications of the commonly performed bariatric procedures, including Roux-en-Y gastric bypass (RYGB), laparoscopic adjustable gastric band (LAGB), laparoscopic sleeve gastrectomy (LSG), and biliopancreatic

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diversion with duodenal switch (BPD-DS). An emphasis is made on ways to prevent specific complications.

An important element in prevention of complications is patient selection, preoperative investigations, and optimization. It is important to assess the patient for medical conditions that would increase the risk of complication, such as a coagulation disorder, obstructive sleep apnea, or uncontrolled diabetes (see Fouse T, Brethauer S: [Resolution of Comorbidities and Impact on Longevity Following Bariatric and Metabolic Surgery](#); Cruz ADG, Portenier DD: [Patient selection and Surgical Management of High Risk Patients with Morbid Obesity](#); and Genser L, Mariolo JRC, Castagneto-Gissey L, et al: [Obesity, Type2 Diabetes and the Metabolic Syndrome – Pathophysiologic relationships and Guidelines for Surgical Intervention](#), in this issue).

PERIOPERATIVE COMPLICATIONS

The Bariatric Outcomes Longitudinal Database is a large cohort study from Bariatric Centers of Excellence across the United States.⁵ Their analysis of 36,254 patients who underwent an RYGB (92% laparoscopic) demonstrated a 1.38% rate of adverse events at 30 days post-RYGB, the most common complications being anastomotic leak (0.42%), renal failure (0.31%), respiratory failure (0.27%), and death (0.12%). The Scandinavian Obesity Surgery Registry reported the 30-day complications (8.7%) of 25,038 laparoscopic RYGB in 44 accredited centers in Sweden.⁶ The most common complications were bleeding (2.1%), leaks or abscesses (1.8%), and small bowel obstruction (1%). They also reported their intraoperative adverse events, such as unintentional small bowel injury (1.5%), bleeding (0.6%), and instrument failure (0.2%). The Michigan Bariatric Surgery Collaborative evaluated the 30-day operative complications that presented in 7.3% of 15,275 patients undergoing surgery, more specifically 3.1% for RYGB, 2.2% for LSG, and 0.78% for LAGB.⁷ Most were minor complications (SSI, 5.9%) and the overall mortality was reported at 0.1%. The Longitudinal Assessment of Bariatric Surgery reported the outcomes of 5882 patients (73% RYGB [88% laparoscopic] and 27% LAGB).⁸ Thirty-day major surgical complication rate was 4.1% and mortality is reported at 0.3%.³ Adverse intraoperative events were associated with worse outcome and consisted of anesthesia events (0.9%), bowel injury (0.9%), instrument/equipment failure (0.8%), liver injury (0.4%), anastomosis revision (0.3%), and splenic injury (0.2%) with all events being more frequent for RYGB.

INTRAOPERATIVE SURGICAL COMPLICATIONS

Laparoscopic Entry Technique

A recent Cochrane review based on 46 randomized controlled trials that included 7389 participants demonstrated no statistical difference in the rates of major complications (vascular or visceral injury) when comparing different entry techniques.⁹ All three major techniques (open-entry, Veress needle entry, and direct vision entry) have been described safely for morbidly obese patients.¹⁰ It is important, however, to always rule out an injury after gaining access and be familiar with different techniques.

Small Bowel/Hollow Organ Injury

Although not specific to bariatric procedures, the morbidly obese patient presents with specific characteristics that might lead to a bowel injury. Torque on the trocars (caused by a thick abdominal wall) might lead to lack of control of the instrument and a thermal injury/laceration. Blind instrument insertion (especially if the trocar is very lateral) might also lead to unrecognized injury. For a higher risk trocar location

or a nonexperienced assistant, it is therefore recommended to always insert instruments under direct visualization. It is recommended to use sharp dissection and not surgical energy when lysing adhesions. If an injury arises and is recognized, it can usually be repaired primarily (in a transverse fashion to prevent stricture) either laparoscopically or open, depending on the surgeon's comfort level. An unrecognized bowel injury can lead to deep SSI and peritonitis and should be suspected if a patient becomes septic in the postoperative period.

Solid Organ Injury

The left segment of the liver rests on top of the stomach and is particularly prominent in the setting of obesity and steatosis, which makes it very fragile. When using the liver retractor to lift it up, great attention should be paid to not lacerate the parenchyma. Another risk for injury is blind entry of instruments through a port in the right upper quadrant (especially if placed too cephalad). It is important to keep in mind the position of susceptible organs and entering instruments under direct visualization is recommended to prevent this avoidable complication. The spleen lays in close proximity to the gastric fundus. It is particularly at risk while dissection of the greater curve during LSG and while performing retrogastric dissection at the angle of His during LAGB and RYGB. Management of bleeding varies with the degree of laceration but common basic principles are as follows:

1. Packing compression with an opened surgical sponge passed through a 10+ mm port
2. Use of surgical energy: either electrocautery (bipolar or monopolar) or advanced energy, such as vessel-sealing bipolar devices or ultrasonic energy devices
3. Use of hemostatic agents, such as oxidized regenerated cellulose, topical thrombin, and fibrin sealants^{11,12}
4. Use of argon beam coagulation
5. Resection (partial hepatectomy or splenectomy) for severe refractory cases¹³
6. Conversion to laparotomy based on surgeon's experience and judgment

Anastomosis Revision

During RYGB and BPD-DS, it is important to assess the anastomosis for two important criteria: adequate perfusion and integrity. Ischemia can be diagnosed by discoloration of either the serosa by laparoscopic view or the mucosa by endoscopic view. It can be caused by the following factors:

1. Tension (commonly gastrojejunal [GJ] anastomosis of RYGB) on the mesentery, which can be reduced by using a retrocolic-retrogastric technique, achieving a complete omental split and, for a patient with a short mesentery, creating a slightly longer gastric pouch.
2. Twisting of the mesentery, always important to rule out. A standardized technique can avoid this problem.
3. Undercutting the mesentery of the biliopancreatic limb of the Roux limb, which is important to rule out before performing the anastomosis, at which point the compromised portion of bowel (tip of the limb) can easily be resected as needed.

Integrity of the anastomosis is evaluated through a leak test, by insufflating at the anastomosis (either with the use of an endoscope or an orogastric tube connected to air flow), clamping the distal bowel, and immersing the anastomosis under water to look for bubbles. It can also be done by injecting methylene blue dye and looking for extravasation. In the case of a positive leak test, it is important to examine all sides

of the anastomosis and look for a defect (the site of leak is guided by the origin of extravasation of either air or dye). The defect can be repaired primarily and an omental patch reinforces the repair. It is afterward important to repeat the leak test to confirm adequate repair of the leak.

Staple Line Bleeding

During LSG, the long staple line is often a source of bleeding. It is often caused by superior staple height of tissue (selected for thick gastric tissue) that does not compress vessels, especially when the staple line is close to the larger vessels of the lesser curve. Management of bleeding is compression with opened surgical sponge or application of clips or hemostatic agents. For diffuse bleeding, the staple line can be oversewn, but great care should be taken to prevent obstruction, especially in critical areas, such as the angle of His and the incisura angularis. Using staple line reinforcement can prevent bleeding.^{14–16}

Bleeding from Trocar Site

Trocars compress injured abdominal wall vessels (most importantly the inferior epigastric vessels) during the case and tamponade off the bleeding. It is important to take trocars out under direct visualization. If hemorrhage is noted, a figure-of-eight stitch using a suture passer can be placed and tied after the trocar is removed. If not recognized during the case, it leads to a hematoma of the abdominal wall. If the hematoma is expanding or large, anticoagulants (thromboprophylaxis) should be suspended. Surgical intervention is rarely required but is warranted in cases of hemodynamic instability, hemoperitoneum, and superinfection of the hematoma.¹⁷

POSTOPERATIVE SURGICAL COMPLICATIONS

Bleeding

Although rare, a postoperative hemorrhage is life-threatening. For RYGB or BPD-DS, the incidence in the early postoperative period (less than 30 days) has been reported to be less than 5% and can arise from many sources and vary in clinical presentation.^{18–20} Intraluminal bleeding (GJ or duodenoileal anastomosis and jejunojejunal [JJ] or ileoileal [II] anastomosis) presents with either hematemesis (GJ/duodenoileal) or melena/hematochezia (JJ/II). It can also present as a small bowel obstruction caused by a clot (discussed later). Late (more than 30 days postoperative) intraluminal bleed is classically caused by a marginal ulcer.^{21,22} Extraluminal bleeding can appear through an incision site or a drain or be diagnosed by findings of hemoperitoneum on imaging or a drop in hemoglobin. It can arise from a staple line, a trocar site, mesentery, omentum, or solid organ injury, unrecognized during surgery.²³ Management of patients depends on hemodynamic stability and failure of self-resolution after suspending anticoagulants and transfusions as needed (more than 2 units of packed red blood cells).¹⁸ Endoscopic management of intraluminal bleeding by adrenaline injection, electrocoagulation, or hemostatic endoclips is successful and safe, with low failure rates. This therapy can be repeated.^{24,25} Management of extraluminal bleeding refractory to conservative management is return to the operating room for exploratory laparoscopy and hemostasis, as previously described.

Postoperative bleeding after LSG has been reported as below 5%.^{26,27} The main source of bleeding is the staple line and can present as intraluminal or extraluminal hemorrhage. Management is as previously described.

Postoperative bleeding after LAGB is even rarer. Early bleeding is usually caused by solid organ injury and is managed as previously described. Delayed hemorrhage can be secondary to erosion and is life-threatening.²⁸

Obstruction

A common complication of gastrointestinal (GI) surgery, small bowel obstruction has been reported in 1% to 5% of RYGB and BPD-DS patients who underwent an open procedure.²⁹ Although less frequent with the laparoscopic approach, it has been described in up to 3.6% of patients.^{29–31} Adhesive disease represents a common cause when performing open surgery but should not be considered as the default cause for patients who have undergone laparoscopy. Internal hernias are common causes of small bowel obstruction and can occur through well-recognized spaces: Petersen space (between the mesentery of the Roux limb and the transverse colon), the JJ, or II mesenteric defect for both RYGB and BPD-DS and the transverse mesocolon defect created to perform a retrocolic approach for the GJ anastomosis of RYGB.³⁰ Internal hernias are prevented by closing mesenteric defects.^{30,32,33} For RYGB, an antecolic configuration of the Roux limb for the GJ anastomosis was demonstrated to lead to fewer internal hernias and small bowel obstruction than a retrocolic approach.³⁴ Another common cause of small bowel obstruction postbariatric surgery is abdominal wall hernia, including trocar site hernias.³¹ Great care should be applied when closing the trocar site fascial defect to prevent those from occurring.

The patient presenting with small bowel obstruction should be evaluated and managed while keeping that differential diagnosis in mind. Routine investigations usually consist of abdominal series, upper GI studies $\pm$ computed tomography scan (with oral and intravenous contrast). Diagnostic laparoscopy is often warranted to rule out closed loop obstruction or bowel ischemia. The cure of obstruction can also be performed laparoscopically or open, depending on the surgeon's abilities and comfort. Bowel distention is too important to perform the laparoscopic approach safely and conversion to laparotomy should be perceived as a good judgement for patient safety instead of a technical failure.

For patients with a history of LAGB who present with pain and obstructive symptoms (nausea, vomiting, dysphagia), it is important to rule out a gastric slip, which has been reported to occur in up to 24% of the patients after undergoing LAGB.³⁵ Suspicion of slippage arises when the band lies in a horizontal orientation and is confirmed by upper GI series showing the obstructed passage of contrast through the band with dilation proximal to the band.³⁶ A band that is too tight appears in a correct position but shows the same pattern on the upper GI series. Immediate management should be to access the system and remove all fluid.³⁷

For patients that underwent LSG, stenosis of the tabularized stomach has been reported to occur in up to 4% of cases.³⁸ The gastroesophageal junction and the incisura angularis are well-recognized critical zones to protect from narrowing when performing the sleeve gastrectomy. It is important to always use a Bougie as a guide.³⁹ When a stenosis is suspected, it should be investigated by upper GI series and upper GI endoscopy. The initial management is endoscopic and consists of balloon dilation and may require stent placement.^{38,40} Refractory cases are managed by either laparoscopic surgical correction (seromyotomy) or conversion to RYGB.⁴¹

Infections

SSI are minor complications but can lead to pain and undesired discomfort from wound care and consequences on the health care system, such as increased visits in the emergency department and increased readmission rates.⁴² Bariatric surgery

patients have multiple risk factors for the development of SSI, such as obesity and diabetes. Great efforts should be conducted to limit the prevalence of SSI. Apart from prophylactic antibiotics, many changes in operative techniques have been linked to a decrease in SSI rates.^{43,44} A laparoscopic approach over open leads to a decrease in the incidence of SSI, for both RYGB² and BPD-DS.²⁹

Leaks are life-threatening complications of bariatric surgery. Although they remain rare, at a reported frequency of usually less than 5%, they can happen after RYGB, BPD-DS, and LSG.^{2,18,27,45} Leaks can happen at staple lines (sleeve, gastric pouch, or duodenal stump) or anastomoses.²⁹ It is important to perform a leak test on proximal anastomoses, as previously reported. Leaks for LSG happen most commonly proximal, close to the gastroesophageal junction and great care should be applied to prevent the superior staple line to be on esophagus, because the tissue is thinner and more fragile.⁴⁶ It is also believed that a tighter sleeve leads to increased pressure and would lead to increased risk of leak. Leaks should be suspected in all postbariatric surgery patients presenting with sepsis (tachycardia, pain, fever, hypotension). A leak can be confirmed radiographically with an upper GI series or computed tomography scan with water-soluble contrast, but even if the test is negative, it is important to explore laparoscopically when clinically suspected. Imaging should never delay intervention in an unstable patient. Goals of surgical management of a leak are peritoneal washout and wide drainage, identification (leak test) and repair of the defect as previously described, enteral access for postoperative nutritional support (feeding jejunostomy for LSG and BPD-DS; gastrostomy in the bypassed gastric remnant for RYGB), and cultures and wide-spectrum antibiotics.

NUTRITIONAL COMPLICATIONS

Although the goals of bariatric surgery are weight-loss and resolution of comorbidities, it is important to realize that those operations affect either intake or absorption of nutritional elements, or both at the same time.⁴⁷ The benefits of reduced intake of fat and carbohydrate are obvious, but great care needs to be taken to make sure patients after their surgeries do not suffer malnutrition or micronutrient deficiencies (Table 1).

Postoperative Diet

Before discharge, the patients are placed on a clear liquid diet to ensure tolerance.⁴⁷ While in the hospital, intravenous fluids are given to compensate for a limited oral intake. Nausea is frequent and can impede oral intake, therefore it should be

Table 1 Vitamin supplement recommendations				
Vitamins	Procedure			
	LAGB	LSG	RYGB	BPD-DS
Multivitamin with minerals	✓	✓	✓	✓
Vitamin B ₁₂	—	✓	✓	—
Calcium with vitamin D	✓	✓	✓	✓
Iron	—	—	✓	✓
Vitamin C	—	—	✓	—
Vitamin A	—	—	—	✓
Vitamin K	—	—	—	✓

controlled. We routinely use a scopolamine patch to prevent and antinausea medications to treat this complication as needed. Once the patients demonstrate adequate oral intake, they are discharged home.

The diet progression is staged, with the goal of tolerating a solid diet 1 month after surgery.⁴⁸ Although the duration of each of the phases varies from different programs, the concept is progression in consistencies (to minimize early satiety and reflux) and selection of foods to maximize weight loss and preservation of lean body mass. It goes from clear liquid diet (inpatient), to full liquid diet (10–14 days), to puree (10–14 days), to mechanical soft (14 days), to regular. Emphasis is put on maintaining proper hydration status by drinking at least 60 oz of fluids daily. Protein intake should be 60 to 80 g per day after LAGB, LSG, and RYGB and 80 to 120 g per day after BPD-DS.⁴⁹

Nutrient Metabolism

It is important to understand basic concepts of macronutrient and micronutrient metabolism to understand the need for supplementation for these patients, and the potential for deficiencies. The American Society for Metabolic and Bariatric Surgery nutritional guidelines review all of them in detail.⁴⁷

Vitamin B₁₂ needs the intrinsic factor as a cofactor to be absorbed in the distal ileum. Intrinsic factor is produced by parietal cells of the stomach. LSG and RYGB limit the production of intrinsic factor, therefore leading to deficiency, although the level of deficiency is more than 3 times more for RYGB than for LSG.^{50–52}

Iron is primarily absorbed in the duodenum and proximal jejunum. The bypassing of these parts of the GI tract after RYGB and BPD-DS therefore puts patients at an increased risk for iron deficiency, which leads to anemia.⁵³ Iron deficiency has also been reported after LSG, frequencies of which were similar to RYGB.⁵² This is believed to be caused by the reduced levels of gastric acid, an important element to cleave dietary iron. Vitamin C deficiency is rare after bariatric surgery and leads to scurvy. Consuming fruits, especially citrus, can prevent it. However, vitamin C has been demonstrated to increase the absorption of iron after RYGB and is therefore recommended as a supplement.⁵⁴

Calcium is absorbed preferentially in the duodenum and proximal jejunum. Its absorption is facilitated by vitamin D, which is itself absorbed in the jejunum and ileum. It is therefore limited after RYGB and BPD-DS. Because calcium is better absorbed in an acidic environment, LSG patients are also at an increased risk for deficiency.⁵⁵ A vitamin D deficiency would not lead to hypocalcemia right away because of the parathyroid hormone (PTH) pathway. As calcium stores deplete, PTH levels rise, leading to bone resorption and preservation of calcium by the kidneys. Therefore, hypocalcemia occurs only in the setting of osteoporosis, which is to be avoided.

An important mechanism of action of BPD-DS is the reduction in absorption of fat caused by the delay to mix pancreatic and gastric enzymes with nutrients until the last 50 to 100 cm of ileum (common channel).⁵⁶ Fat-soluble vitamins A, D, E, and K are mainly absorbed in the distal jejunum and ileum and are therefore at a very high risk to become deficient after BPD-DS.⁴⁹ Deficiencies have, however, also been described after RYGB, LAGB, and LSG, albeit at a lesser extent as for BPD-DS.^{57,58}

Thiamine (vitamin B₁) has many important roles that regulate the heart, the GI tract, and the peripheral and central nervous system. Thiamine deficiency can lead to devastating consequences, such as Wernicke encephalopathy (WE) and Korsakoff psychoses. WE presents with peripheral neuropathies and the classic triad of confusion, ataxia with unsteadiness of gait, and nystagmus with diplopia.⁵⁹ Because thiamine is primarily absorbed in the duodenum and proximal jejunum, RYGB and BPD-DS are at an increased risk for deficiency but thiamine deficiency has been

associated with all bariatric procedures. The main cause is limited intake and the classical presentation of WE is following a period of nausea and vomiting while not taking vitamin supplements, or a patient maintained on a prolonged clear liquid diet without vitamin supplementation, typically in the first 4 to 12 weeks after bariatric surgery.⁶⁰ Thiamine deficiency is treated by supplements and needs to be recognized as soon as possible to increase the chances of recovery. Infusing bariatric surgery patients with a solution of dextrose without giving vitamins and thiamine first can deplete thiamine stores and can precipitate WE and aggravate neurologic damage.⁶¹

Deficiencies (Macronutrients and Micronutrients)

Protein deficiency usually arises from intolerance to protein-rich foods and failure to take protein supplements.^{47,62} Common symptoms of protein deficiency are edema, weakness, decreased muscle mass, and thinning hair. Dosage of serum albumin, prealbumin, and creatinine can confirm the deficiency. The first line of treatment is increasing oral protein supplements. Refractory cases may need enteral or parenteral nutritional support and might require reversal of the procedure in extreme cases.

Vitamin B₁₂ deficiency can present with fatigue (pernicious anemia) and neurologic symptoms, such as tingling or numbness in fingers and toes, ataxia, mood changes, memory loss, and dementia. It is confirmed by drawing a complete blood count (CBC) and serum B₁₂ levels. Repletion with oral or sublingual B₁₂ (500 µg/d) for 1 month is recommended as the first phase. Refractory cases should be treated with intramuscular injections of B₁₂ (1000–2000 µg/month) for 2 to 3 months.

Iron deficiency also leads to anemia (fatigue, pale skin, palpitations, cold hands and feet). It is confirmed with CBC, serum iron, iron-binding capacity, and ferritin. It is treated with ferrous sulfate, 300 mg two to three times per day with vitamin C. Refractory cases should get intravenous iron infusions.

Hypocalcemia rarely gets severe enough to be symptomatic but can present with tetany and cramping. Dosage of total and ionized serum calcium levels and PTH are necessary to pose the diagnosis. Treatment is with oral supplements of calcium citrate, 1200 to 200 mg/d.

Vitamin D deficiency can lead to hypocalcemia. A low 25-hydroxy vitamin D level confirms the diagnosis. First-line repletion therapy is oral vitamin D, 50,000 IU once weekly for 3 months, which is increased to 50,000 IU twice a week for another 3 months if still deficient. Refractory cases should get oral calcitriol vitamin D, 1000 IU/d.

Folate deficiency leads to the development of macrocytic anemia and neural tube defects in pregnant postbariatric surgery patients. Measuring CBC, serum folate levels, and homocysteine levels leads to the diagnosis. Oral repletion with 400 µg/d (included in multivitamin) for 1 month is recommended. Refractory cases should have the dosage increased to 1000 µg/d.

Zinc deficiency leads to poor wound healing, hair loss, diarrhea, and alteration in taste. It is diagnosed when low levels of serum zinc are found. Repletion is with oral zinc, 220 mg/d for 1 month. Refractory cases can have the dosage increased to 220 mg two or three times a day for another month.

Copper deficiency leads to weakness, fatigue, skin sores, and hair and skin discoloration. These patients should be repleted with oral copper, 2 to 4 mg/d for 1 month.

Vitamin A deficiency leads to night blindness, dry eyes, dry skin, and dry hair. It is diagnosed with a low serum vitamin A level. Treatment is initially with oral vitamin A, 5000 to 10,000 IU/d for 1 month. Refractory cases should then have the dosage increased to 50,000 IU/d for 1 month.

Box 1**Nutritional follow-up laboratory studies***LAGB: 6 months, 12 months, and annual*

CBC

Comprehensive Metabolic Panel

Folate

PTH

Iron and transferrin

Vitamin B₁₂

Vitamin D, 25-OH

LSG: 6 months, 12 months, and annual

CBC

CMP

Folate

PTH

Iron and transferrin

Vitamin B₁₂

Vitamin D, 25-OH

RYGB: 6 months

CBC

CMP

Folate

PTH

Iron and transferrin

Magnesium

Phosphorus

Zinc

Copper

Vitamin B₁₂

Vitamin D, 25-OH

RYGB: 12 months and annual

Bone density: dual photon study

CBC

CMP

Folate

PTH

Iron and transferrin

Magnesium

Phosphorus

Zinc

Copper
Vitamin B ₁₂
Vitamin D, 25-OH
<i>BPD-DS: 6 months</i>
CBC
CMP
Folate
PTH
Iron and transferrin
Magnesium
Phosphorus
Zinc
Copper
Vitamin A
Vitamin B ₁
Vitamin B ₁₂
Vitamin D, 25-OH
Vitamin E
<i>BPD-DS: 12 months and annual</i>
Bone density: dual photon study
CBC
CMP
Folate
PTH
Iron and transferrin
Magnesium
Phosphorus
Zinc
Copper
Vitamin A
Vitamin B ₁
Vitamin B ₁₂
Vitamin D, 25-OH
Vitamin E

Vitamin E deficiency leads to hair loss, leg cramps, and pain/tingling in the extremities. It is diagnosed with finding of low serum vitamin E levels and is repleted with oral vitamin E (initially 400 IU/d for 1 month, then 800 IU/d for another month for refractory cases).

Vitamin K deficiency leads to coagulopathy and can present with bruising, hematuria, or menorrhagia. It is diagnosed with low serum vitamin K levels (more commonly

used for research purposes) but can also be suspected with an international normalized ratio greater than 1.4. Repletion is with oral vitamin K, 5 to 10 mg/d, or it can also be treated with a single dose of 10 mg intramuscular.

Follow-Up of Laboratory Studies for Monitoring

It is important to always emphasize at every encounter with the patient the importance of observance in taking supplements after bariatric surgery. It is also very important to monitor nutritional status of the patient to catch a deficiency or more importantly prevent a deficiency from arising. In our centers, laboratory studies are performed routinely at 6 months and 12 months postoperative and more frequently should a suspicion arise (either by symptoms suspicious of deficiency or by behaviors that would lead to a deficiency, such as poor observance, food aversion, or nausea/vomiting).

Box 1 lists the laboratory studies drawn per our actual protocol, dependent on procedure and timing postoperatively.

SUMMARY

Although major complications after bariatric surgeries remain rare, it is important to recognize their presentation and understand their management. Prevention is key and a few of these complications are avoided by simple actions in the operating room. Observance of nutritional supplements after bariatric surgery is a crucial determinant of whether or not complications will occur. Counseling on supplements and nutritional monitoring is important. “Are you taking your vitamins and supplements?” should be part of every encounter with patients after a bariatric procedure, for the rest of their life.

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