Chapter

Nutritional Deficiencies Post Bariatric Surgery: A Forgotten Area Impacting Long-Term Success and Quality of Life

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Abstract

Bariatric surgery (BS) results in significant weight loss and improvement of obesity associated comorbidities. Despite the benefits achieved with these operations, deficiencies of vitamins and other micronutrients are common. Such deficiencies may become clinically significant if not discovered and treated early. Therefore, it is imperative to undertake thorough screening, and have sound preventive strategies in place in order to make BS a safer procedure. This chapter will provide the multidisciplinary bariatric team with a comprehensive review of micronutrient deficiencies before and after bariatric surgery. The focus will be on the most common micronutrient deficiencies that are encountered in various types of BS procedures, including water soluble vitamins, fat-soluble vitamins, minerals and trace elements deficiencies, as well as protein malnutrition. The chapter starts with an overview of the causes of micronutrient deficiencies in patients with obesity and before undergoing BS. It reviews the screening of patients for preexisting micronutrient deficiencies prior to their BS. Then the chapter addresses the potential causes and mechanisms leading to such deficiencies after BS. It then conducts an in depth discourse of the prevalence of deficiencies by the type of BS, the presenting symptoms, and the investigations required for the diagnoses. The chapter will also discuss the management of each deficiency according to the severity of the symptoms. The chapter also reviews the recent updated guidelines for standard nutritional care post BS. We will finally conclude with a framework of the preventive strategies for optimal care to ensure long term success post-surgery.

Keywords: obesity, bariatric surgery, micronutrients, deficiencies, water soluble vitamins, fat soluble vitamins, minerals, trace elements, protein malnutrition

1. Introduction

Bariatric surgery (BS) has proven to be an effective treatment for weight loss, reducing obesity associated comorbidities, improving quality of life, and reducing mortality rates [1, 2]. The increasing amount of evidence on the benefits of BS has contributed to its increased popularity over the last decade [1]. Despite the proven benefits of BS, it also carries the risk of short- and long-term complications.

An important complication is the nutritional and micronutrient deficiencies. Nutritional deficiencies can present with a wide range of clinical manifestations, depending on the specific nutrients/micronutrients that are involved, the severity, and the duration of the deficiency states. Additionally, these deficiencies can worsen over time, leading to severe consequences, such as anemia (iron, folate, B12), peripheral neuropathy (folate, B6, B12, copper), Wernicke encephalopathy (B1) and metabolic bone disease (vitamin D, calcium). Therefore, lifelong screening of individuals who had undergone bariatric surgery is critical to identify these complications and treat them effectively to ensure long-term success. This chapter will provide a comprehensive review of these nutritional complications. The chapter will also furnish information about the recommended micronutrient supplementations and nutritional follow-up.

2. Micronutrient deficiencies prior to bariatric surgery

Nutritional deficiencies often exist prior to BS [3–5]. Subjects with obesity typically adopt an unhealthy high calorie, low quality diet with unbalanced nutritional composition [6]. For instance, one study showed that in female BS candidates, despite consumption of high-caloric diet (2801 ± 970 kcal/day), 66% of them had at least a single micronutrient deficiency [7]. Prior to BS, low iron, ferritin, vitamin B12 and hemoglobin were observed among 12.6%, 8.7%, 10.6% and 7.7%, of patients respectively [7]; and the incidence of folate deficiency before BS was 26.8% [8]. Vitamin D deficiency is the most common deficiency in patients undergoing BS with a prevalence as high as 78.8% [7, 8].

Research found that low preoperative levels of hemoglobin, vitamin B12, and ferritin were independently associated with reduction in the levels of micronutrients postoperatively [8]. Moreover, vitamins D and B1 and albumin deficiencies before BS predicted deficiencies one year after surgery [8].

Such findings highlight the need for complete nutritional assessments and adequate correction of pre-existing deficits before BS. Therefore, all BS candidates must undergo appropriate nutritional evaluation, including micronutrient measurements at least once preoperatively. Screening should include iron studies, and vitamins D and B12 and folic acid levels. The repetition of the tests until surgery should be individualized as clinically indicated [9, 10]. In comparison with purely restrictive procedures, more extensive nutritional evaluations are required for malabsorptive procedures. For instance, thiamine and vitamins A and E levels may be assessed in patients prior to Roux en Y gastric bypass (RYGB) and biliopancreatic diversion with duodenal switch (BPD/DS) [10].

Deficiencies found on screening prior to BS should be treated accordingly to avoid worsening of the symptoms post-surgery [9]. For vitamin D, there is a lack of solid evidence regarding the cutoff value where treatment should be started. A group of experts advocated supplementation in all patients with values below 20 ng/mL, and in an individualized manner for values between 21 and 30 ng/mL [9].

3. Micronutrient deficiencies post bariatric surgery

3.1 Causes of micronutrient deficiencies post bariatric surgery

Several factors and mechanisms contribute to the development of nutritional deficiencies post BS. Below are some examples:

3.1.1 Non-compliance with nutritional supplementation

Nonadherence to the recommended nutritional supplementation is recognized as a critical factor that leads to nutritional deficiency after BS. Compliance with multivitamins tends to be good in the early post-surgery period and decreases on the long term. For instance, a study of 16,620 patients post BS showed that the pharmacy dispensing of micronutrient supplements by patients significantly decreased between the first and fifth years for iron (from 27.7 to 24.5%), calcium (from 14·4 to 7·7%), but increased for vitamin D (from 33·1 to 34·7%) [11]. Barriers to vitamin adherence post BS include forgetting to take the supplementation and difficulty in swallowing the pills [12].

3.1.2 Lack of follow up

Despite clear international guidelines, long-term follow-up after BS is poor. A study assessed the follow up with the bariatric surgeon after RYGB and demonstrated a significant increase in the time between follow ups (13.3 ± 7.8 vs. 86.9 ± 39.9 months) in the long-term [13]. The same study demonstrated that a shorter time since last surgeon visit was independently predictive of multivitamin use ($p = 0.001$) [13]. Research also reported that male sex, younger age, absence of type 2 diabetes and poor 1-year follow-up were predictors of poor 5-year follow-up [11].

3.1.3 Other causes

Other contributing factors include pre-operative deficiencies, post-surgery food intolerance, poor eating habits, vomiting, changes in taste and eating patterns [14].

3.2 Mechanisms of micronutrient deficiency after bariatric surgery

The underling mechanisms that contribute to micronutrient deficiency following BS include reduced food intake due to restrictive effect of surgery, rerouting of nutrient flow which affect absorption, and changes in gastrointestinal anatomy/physiology post-surgery. It is important to note that the anatomical changes and the mechanisms of action of the various procedures dictate the frequency and severity of nutritional deficiencies after BS. For instance, micronutrient deficiencies are less common in restrictive procedures such as gastric banding (LAGB) and laparoscopic sleeve gastrectomy (LSG), where there are no alterations of the intestinal continuity and normal digestive processes. However, micronutrient deficiencies are more common after surgical procedures that cause malabsorption such as RYGB, one-anastomosis gastric bypass (OAGB), single anastomosis duodeno–ileal bypass with sleeve gastrectomy (SADI-S) and BPD/DS [10, 15].

3.3 Water soluble vitamin deficiency post bariatric surgery

3.3.1 Vitamin B1 (thiamin)

Vitamin B1 is absorbed in the jejunum and therefore may be excluded from absorption after RYGB and BPD/DS [16]. Additionally, the storage of thiamine is low in the human body and can become rapidly devoid without regular and adequate intake [8]. These characteristics might explain why thiamin deficiency is observed subsequent to a short period of persistent vomiting after surgical complications such as band slippage post LAGB [17], stomach oedema after LSG [18],

Mico/micro deficiency	Clinical features and complications	Management
Vitamin B1 Thiamin	Wernicke encephalopathy (confabulations, ophthalmoplegia, ataxia) Korsakoff syndrome Dry Beriberi (polyneuropathy, paresthesia) Wet Beriberi (cardiomegaly, tachycardia, CHF)	Oral: 100 mg 2–3 times daily until symptoms resolve IV: 200–500 mg once or twice daily for 3–5 d, followed by 250 mg/d for 3–5 d or until symptoms resolve, then 100 mg/d orally, indefinitely, or until risk factors resolve IM: 250 mg once daily for 3–5 d or 100–250 mg monthly
Vitamin B12 Cobalamin	Macrocytic anemia, peripheral and central neuropathy, myelopathy, memory disturbance, dementia, depression, delusions	1000 μg/d to achieve normal levels and then resume dosages recommended to maintain normal levels
Folate	Macrocytic anemia, leukopenia peripheral neuropathy, myelopathy, glossitis, fetal neural defects. May aggravate B12 deficiency	Oral dose of 1 mg of folate daily to reach normal levels and then resume recommended dosage to maintain normal levels
Vitamin A	Ocular xerosis, night blindness, decreased immunity, scaling skin	Vitamin A deficiency without corneal changes: 10,000–25,000 IU/d of vitamin orally until clinical improvement Vitamin A deficiency with corneal changes: 50,000–100,000 IU of vitamin A IM for 3 d, followed by 50,000 IU/d IM for 2 weeks
Vitamin D	Osteomalacia, bone demineralization, increased risk of fractures	Vitamin D3 at least 3000 IU/d and as high as 6000 IU/d, or 50,000 IU vitamin D2 1–3 times weekly
Vitamin E	Hemolytic anemia, peripheral neuropathy, loss of deep tendon reflexes, ataxia, diminished perception of vibration and position ophthalmoplegia, myopathy, rash	Optimal therapeutic dose of Vitamin E for bariatric patients is not defined Potential antioxidant benefits can be achieved with supplements of 100–400 IU/d Additional dose may be required for replacement
Vitamin K	Coagulopathy, excessive bleeding or bruising	Parenteral dose (10 mg) for symptomatic patient acute malabsorption A dose of either 1–2 mg/d orally or 1–2 mg/week parenterally recommended for patients with chronic malabsorption
Iron	Microcytic anemia, fatigue glossitis, nail dystrophy	Oral: 150–200 mg of elemental iron daily to amounts as high as 300 mg 2–3 times daily (ferrous sulfate, fumarate, gluconate) Vitamin C supplementation may be added to increase iron absorption IV iron infusion (ferric gluconate or sucrose forms) for patients with severe intolerance to oral iron or refractory deficiency Blood transfusion for severe iron deficiency anemia
Calcium	Fatigue, arrhythmia, myopathy, bone demineralization	Repletion of calcium deficiency varies by surgical procedure BPD/DS: 1800–2400 mg/d; LAGB, LSG, RYGB: 1200–1500 mg/d
Zinc	Hair loss, pica, dermatitis, chronic diarrhea, dysgeusia, hypogonadism or erectile dysfunction (in males)	Optimal therapeutic dose is unknown. Treatment should target normal biochemical levels. For every 8–15 mg/day elemental zinc provided, 1 mg/day copper should be supplemented to avoid inducing a copper deficiency

Mico/micro deficiency	Clinical features and complications	Management
Copper	Anemia, neutropenia, myeloneuropathy sensory ataxia, impaired wound healing	Treatment varies with severity of deficiency Mild–moderate: 3–8 mg/d oral copper gluconate or sulfate until indices return to normal Severe: 2–4 mg/d intravenous copper can be initiated for 6 d or until serum levels return to normal and neurologic symptoms resolve Copper gluconate or sulfate is recommended
Selenium	Anemia, persistent diarrhea, cardiomyopathy, metabolic bone disease	Optimal therapeutic dose of selenium for bariatric patients is not defined RDA for selenium is 55 micrograms per day

IV intravenous; IM intramuscular; D: day, CHF: congestive heart failure, LAGB laparoscopic gastric band, LSG laparoscopic sleeve gastrectomy, RYGB Roux en Y gastric bypass, BPD/D biliopancreatic diversion.

Table 1.
Summary of common micro and micro nutritional deficiencies.

or stoma stenosis after RYGB [19]. Cases of thiamine deficiencies have also been reported after BPD/DS [20].

The manifestations of thiamine deficiency include peripheral neuropathy, Wernicke's encephalopathy (WE), Korsakoff's psychoses and cardiomyopathy [14, 10] (**Table 1**). These clinical conditions could be severe or even fatal if they are not recognized and treated promptly**.** Borderline deficiency may cause less severe symptoms that could be missed. Therefore, oral or parenteral thiamine supplementation should be initiated in any bariatric patient presenting with persistent vomiting severe enough to interfere with adequate nutrition, even before obtaining confirmatory laboratory data [10, 14]. In symptomatic patients, oral supplementation may be used only after 1–2 weeks of parenteral administration and continued until symptom resolution [10].

In severely malnourished patients receiving nutrition support, empiric thiamine supplementation along with fluid and electrolyte monitoring and replacement are indicated to avoid exacerbation of thiamin deficiency and refeeding syndrome [10]. Refeeding syndrome is a condition that results from fluid and electrolyte imbalances, particulalry hypophosphatemia, causing serious complications such as cardiac arrhythmias [21]. Empiric thiamine supplementation is also indicated for high-risk bariatric patients and patients with risk factors for thiamine deficiency such as females, African Americans, patients not attending the dietitian clinic, patients with gastrointestinal symptoms, heart failure, persistent vomiting, or on parenteral nutrition and those with excessive alcohol use [10]. The recommended dose for prevention and treatment of thiamin deficiency is summarized in **Table 1**.

Wernicke Encephalopathy: is a serious complication of thiamin deficiency. It is an acute neuropsychiatric syndrome characterized by ataxia, ophthalmoparesis, nystagmus, and confusion. WE most commonly occurs during the first weeks to months following BS [17]. Among patients who were diagnosed with WE, 52% had RYGB and 21% had LSG [15]. Symptoms of WE are typically preceded by malnutrition, which results from persistent prolonged vomiting, although vitamin noncompliance or increased alcoholism are also risk factors [15]. Radiologic imaging of the brain especially magnetic resonance imaging can be used to support the diagnosis of WE, but is not always sensitive to WE symptoms. Findings include hyperintensities in the thalamic region, the mammillary bodies, and the region around the third and fourth ventricle [22]. The recommended treatment is 500 mg of parenteral thiamine three times daily until symptoms of acute WE resolute [10]. The treatment

is lifesaving and has the potential to reverse this acute neuropsychiatric syndrome. Recovery typically occurs within 3–6 months of initiation of therapy if the symptoms are recognized early [23]. Studies have shown that patients who received suboptimal thiamin dose or had more than one acute symptom were more likely to progress later into a permanent neurologic deficits (Korsakoff's syndrome) [17]. Korsakoff's syndrome is neuropsychiatric disorder characterized by severe amnesia, executive problems, and confabulations, leading to lifelong impairment [17].

3.3.2 Vitamin B12 (cobalamin)

Vitamin B12 (cobalamin) binds to the intrinsic factor, a protein secreted by the stomach. The complex formed is then absorbed by the small intestine [16]. Vitamin B12 deficiency post BS can result from inadequate secretion of intrinsic factor, limited gastric acidity, and most importantly from the bypassing of the duodenum, which is the main site of vitamin B12 absorption [6, 24]. Cobalamin stores in the liver are usually high and therefore vitamin B12 deficiency is rare in the first year after BS; however the incidence tends to increase on the long term [25]. The prevalence vitamin B12 deficiency is 14.3% after LSG and 16% post RYGB [26]. In addition to anemia, vitamin B12 deficiency can cause neurological and psychiatric symptoms [6] (**Table 1**). Therefore, regular screening is required (e.g., every 3 months) in the first year after BS and at least annually after that or as clinically indicated. This is particularly important with chronic use of medications that worsen B12 deficiency such as metformin, proton-pump inhibitors, and seizure medications [10]. In some instances, serum B12 may not be adequate to identify B12 deficiency; in such cases measuring serum methylmalonic acid, with or without homocysteine, should be considered to identify metabolic deficiency of B12, especially in symptomatic or in patients with history of B12 deficiency [10]. Intramuscular or intranasal regimens is preferred over oral supplementation as only 1% of oral vitamin B12 is passively absorbed without intrinsic factor [14].

3.3.3 Folic acid

Complex dietary folates are absorbed throughout the small intestine but mainly at the brush border of the duodenum and upper jejunum [16]. Since folate is absorbed throughout the small intestine, the deficiency is primarily induced by the decrease in dietary intake and to a lesser extent due to malabsorption specially after procedures that bypass the first part small intestine (RYGB, BPD/DS) [6]. Furthermore, folate deficiency can be aggravated by vitamin B12 deficiency since the latter is necessary for the conversion of inactive methyltetrahydrofolic acid to the active tetrahydrofolic acid [6]. The reported prevalence of folate deficiency after LSG and RYGB is 3.6% and 4.2% respectively [26]. Folate deficiency has been associated with a variety of symptoms (**Table 1**) [6, 23]. Maternal folate deficiency in pregnancy can cause fetal neurological abnormalities such as growth retardation, and congenital defects (neural tube) [16, 27]. Therefore, adequate folate supplementation is particularly important after malabsorptive procedures and in women of the childbearing age [10].

3.4 Fat soluble vitamin deficiency post bariatric surgery

3.4.1 Vitamin A

The absorption of vitamin A is reduced after bariatric procedures. The incidence of vitamin A deficiency is 11.1% at one year post LSG [26]. A higher prevalence is

reported after malabsorptive procedures where deficiency was found in up to 70% of patients 4 years after RYGB and BPD/DS [28]. This is due to fat malabsorption and steatorrhoea. Therefore, routine fat-soluble vitamin supplementation is recommended in all patients post BPD/DS [10]. The clinical manifestations of vitamin A deficits are night blindness, xerophthalmia and dry hair [6].

3.4.2 Vitamin D

Vitamin D is a fat-soluble vitamin absorbed preferentially in the jejunum and ileum. Hence, a high incidence of vitamin D deficiency in seen after malabsorptive procedures despite routine supplementation [16]. The reported deficiency after LSG and RYGB is 66.7% and 65.4% respectively [26]. The prevalence of post BPD/DS vitamin D deficiency ranged from 37.1% at one year to 50.8% at 6 years [29]. The most important consequence of vitamin D deficiency is bone demineralization. Therefore, despite the absence of conclusive evidence regarding the long-term risk of fractures after BS, calcium and vitamin D routine supplementation is strongly recommended, especially after RYGB and malabsorptive procedures [10, 30]. The standard supplementation is frequently insufficient to maintain adequate vitamin D levels in patients with malabsorption, and much higher oral or parenteral doses may be required [8, 28]. For treatment, vitamin D3 is recommended as it is a more potent than vitamin D2; however, both can be utilized [10].

3.4.3 Vitamin K

Low levels of vitamin K have been observed in 1.8% post RYGB and 7.4% post SADI patients one year after surgery [31]. However, clinical symptoms such as easy bruising, and increased bleeding are rare [6]. Some cases of fetal and newborn intracranial hemorrhage related to maternal vitamin K deficiency have been described after BPD/DS [27], and have been also reported after LAGB in a pregnant woman with prolonged vomiting due to slippage of the gastric band resulting in gastric outlet obstruction [32].

3.4.4 Vitamin E

Vitamin E deficiency after BS is rare. The reported incidence is 4.8% and 0.9% after RYGB and SADI respectively [31, 33]. The most common symptoms associated with vitamin E deficiency include neuropathy, myopathy and anemia [21] (**Table 1**). Vitamin E neuropathy and myopathy can be treated with a dose of vitamin E 400 IU daily.

3.5 Minerals

3.5.1 Iron

Iron deficiency with or without anemia is frequently observed after BS [10]. The incidence after LAGB and LSG ranges between 14 to 18% [10]. The prevalence after RYGB and BPB/DS is 51.3% and 15% respectively [34, 35]. Several mechanisms lead to iron deficiency post BS. First, iron malabsorption can occur as a result of the bypassing of the duodenum and proximal jejunum post BS where most of iron absorption occurs. Second, decreased gastric acidity and accelerated gastric emptying impair the reduction of iron from the ferric (Fe 3+) to the absorbable ferrous state (Fe 2+). Third is the decreased intake of iron-rich foods (meats, vegetables) post BS. Finally, the absorption of iron may be affected by the interaction with

other nutritional supplements (e.g., calcium) [10, 14]. Menstruating women are at higher risk for iron deficiency and anemia, specially patients with polymenorrhagia [25]. Other risk factors for iron deficiency include malabsorptive procedures, young age, preoperative anemia and low baseline ferritin level [36]. The clinical features of iron deficiency are summarized in **Table 1**. The measurement of serum ferritin is the best diagnostic test for detecting iron deficiency and a better indicator of iron body capacity as it becomes abnormal prior to the decrease in serum iron concentration [6]. Prophylactic iron supplementation is recommended after all types of BS to minimize the risk of deficiency [10]. Iron is usually included in oral multivitamin and mineral preparations with the inclusion of vitamin C, which will increase iron absorption [10]. They should not be taken along together with calcium supplements as such supplements may affect the absorption of iron. Severe cases of iron deficiency anemia require intravenous iron or blood transfusion [36].

3.5.2 Calcium

Calcium absorption occurs mainly in the duodenum and proximal jejunum and is facilitated by vitamin D in an acid environment. Thus, any BS that bypass the first part of the intestine, reduces gastric acid production and lowers vitamin D levels is often associated with reduced calcium absorption [15]. The prevalence of calcium deficiency post LGG and RYGB is 3.9% and 4.3 respectively [37]. Low calcium level may affect bone mineralization, therefore, should be supplemented routinely post BS [8].

3.6 Trace elements

Although most of the literature focuses on calcium and iron, deficiencies of other essential minerals such, zinc, copper, and selenium have been reported in bariatric patients [10]. These essential minerals act as enzymatic cofactors in several biochemical pathways, and therefore, their deficiency could cause variable clinical manifestations that involve neurological, cardiac and gastrointestinal systems. Mineral deficiencies are more common after BPD and RYGB [6].

3.6.1 Zinc

Zinc is absorbed by the small intestine and hence BS such RYGB or BPD/DS which partially exclude nutrient from the small bowel, can cause zinc malabsorption [16]. The prevalence of zinc deficiency is 23.9% after LSG [38]. Moderate zinc deficiency presents with hypogeusia, hyposmia, anorexia, eczema, somnolence, and reduced dark adaptation, whereas severe forms are associated with acrodermatitis enteropathica, bullous or pustular dermatitis, diarrhea, balding, mental abnormalities including depression, and recurrent infections due to impaired immune function [16].

3.6.2 Copper

Copper functions as a cofactor in many enzymatic reactions that are vital for the hematologic, vascular, skeletal, antioxidant, and neurologic systems [39]. It is absorbed mainly in the stomach and proximal duodenum. Copper deficiency is rare and underrecognized. More recently, it has been reported after malabsorptive procedures [39]. Symptoms of copper deficiency are often similar to symptoms of vitamin B12 deficiency (hematological and neurological problems). Peripheral neuropathy, myeloneuropathy with spastic ataxic gait have been reported after BS [40]. Recently, a case of severe pancytopenia with refractory anemia secondary to

copper deficiency has been observed after BS [39]. In this case, administration of intravenous copper resulted in dramatic clinical improvement [39].

3.6.3 Selenium

Selenium is absorbed in the duodenum and proximal jejunum and it is an essential element that provides an important part of the multifunctional selenoproteins that are important for health [41]. Selenium deficiency has been associated with cardiomyopathy, immune system dysfunction and infertility in men. Since RYGB results in the bypass of the duodenum and upper jejunum, micronutrient deficiencies such as selenium are common after this procedure. The prevalence of selenium deficiency post LSG is 7.1% and post RYGB is 3.8% [26]. A case report described a 40 year-old woman that presented with symptoms of heart failure nine months after RYGB which was confirmed by echocardiography and cardiac markers [42]. The patient was diagnosed with selenium-deficient cardiomyopathy, and she had complete resolution of her symptoms after 3 months of oral selenium [42].

4. Protein malnutrition post bariatric surgery

Protein malnutrition remains the most serious macronutrient complication associated with malabsorptive surgical procedures. It can occur in up to 15% of patients after BPD/DS [43]. Studies reported that 3·0–18·5% of BPD/DS patients required reversal of their procedure because of protein malnutrition or excessive weight loss, or both [44]. Protein malnutrition can also occur after RYGB specially when the Roux limb exceeds 150 cm, where the reported prevalence is 9% at 2 years after surgery [43]; however protein malnutrition rarely necessitates reversal or conversion of a RYGB. It is also less common after LSG and LAGB, and in such cases it is likely due to maladaptive eating behaviors after surgery, especially in patients who avoid protein food sources or have protracted vomiting [6]. The clinical presentation of protein malnutrition includes edema, fatigue, skin, hair, and nail problems [6]. Because protein level often remains in the normal range until late, monitoring the serum albumin concentration is more useful for the assessment of the protein nutritional status. Patients with severe protein malnutrition should be treated with protein supplements that are rich in branch-chain amino acids and, in severe cases enteral feeding is recommended [6]. For prevention of protein malnutrition, an average daily protein intake of 60–120 g (1.1 g/kg of ideal body weight) is required and should be increased by 30% for patients post BPD/BD [16].

5. Complications of micro nutritional deficiencies post bariatric surgery

5.1 Anemia

Anemia is common after BS. The prevalence of macrocytic and microcytic anemia is 52% post LSG, 64% post RYGB and 39% after biliopancreatic diversion [45]. Patients with mild anemia post BS are likely to be asymptomatic; however, when the anemia worsens, patients could present with symptoms, such as fatigue, pallor, and dyspnea on exertion [6]. Post-bariatric anemia is in most cases due to iron deficiency, along with vitamin B12 deficiency as a secondary cause. Other causes of nutritional anemias after malabsorptive BS includes folate, protein, copper, selenium, and zinc deficiencies. Therefore, these factors should be evaluated if routine screening for iron-deficiency anemia is negative [10].

5.2 Neurological complications

Neurological complications may occur after BS. They have attracted attention because of their diversity, complexity and potentially devastating effects [46]. Different patterns of complications can be observed according to the time of presentation. For instance, at an early stage, immediate peripherical nerve injury, Wernicke's encephalopathy, and polyradiculoneuropathy are the most frequent. Late complications may appear after years, and include optic neuropathy, myelopathy, and peripherical neuropathy [47]. The prevalence of neurological events after BS is difficult to determine. A cross-sectional study reported a rate of 3% among 451 patients who underwent BS [48]. Axonal polyneuropathy was the most frequent neurological complication, but cases of Wernicke syndrome, vitamin B12 deficiency, Guillain-Barre syndrome and copper deficiency were also identified [44]. The majority of patients (93.3%) had full recovery from the neurological signs and symptoms [49]. In another retrospective study involving 592 post LSG patients, only 1.18% were found to have neurological complications [50]. In this cohort, all the patients had decrease in oral intake and rapid weight loss, with a mean weight loss of 35 kg three months after LSG suggesting that this could be the predisposing cause [50]. All patients were treated for neuropathy secondary to vitamin B1 deficiency and had significant improvement and/or resolution of their symptoms. [50]. A recent study showed that among 61 patients post RYGB and LAGB, 11.4% developed some signs of polyneuropathy, that eventually disappeared at 24 months. The most common manifestations were paresthesia and muscle weakness [51]. The majority of neurological complications post BS is attributed to vitamin and micronutrient deficiencies such as vitamins B12, B6, E, thiamine, folate and copper [23, 47, 46]. It is imperative to note that failure of diagnosis and the delay in the management of these complications can lead to irreversible neurological deficits. However, many of these complications can be prevented with regular follow-ups, routine screening of micronutrients, and nutritional supplementation where a deficiency is identified.

5.3 Metabolic bone disease

The bone mineral density rapidly decreases initially after BS, which reflects a skeletal adaptation to a lower body weight. Bone loss however, continues even after weight loss has stopped [52]. This is likely due to the lower calcium absorption and vitamin D deficiency causing secondary hyperparathyroidism [53]. The prevalence of secondary hyperparathyroidism has been shown to increase progressively with time from 35.4% at 1 year after BS to 63.3% at 5 years after surgery [54]. Patients who underwent a single anastomosis gastric bypass had the highest prevalence of secondary hyperparathyroidism (73.6%) followed by RYGB (56.6%), gastric banding (38.5%), and sleeve gastrectomy (41.7%) at 5 years after surgery [54]. The decrease in bone density may predispose patients to the risk of fractures especially with malabsorptive procedures. However, data on the incidence of fractures post BS remain controversial, with some studies suggesting an increased risk of fractures (non-vertebral fractures, especially in the upper limbs) and others showing no increased risk [55–57]. For instance, one study reported a significantly increased number of fractures only after biliopancreatic diversion (adjusted relative risk 1·60, 95% CI 1·25–2·03; $p < 0·001$, 56]. Others found that 60% of LAGB and 29% of RYGB patients had increased risk of fractures 3–4 years after surgery [55]. Future long-term studies are required to assess the effect of BS on bone health.

Evaluation of patients for metabolic bone disease after BS may include serum parathyroid hormone, total calcium, phosphorus, 25-hydroxyvitamin D, and

24-hour urine calcium levels [10]. In post-bariatric patients with established osteoporosis, pharmacologic treatment with bisphosphonates may be considered. Before starting bisphosphonate treatment, vitamin D deficiency needs to be fully corrected in order to avoid severe hypocalcaemia, hypophosphatemia, and osteomalacia. In these cases, intravenous form of bisphosphonates should be used (zoledronic acid, 5 mg once a year, or ibandronate, 3 mg every 3 months) for better absorption and to avoid potential anastomotic ulceration with orally administered bisphosphonates [10]. More research is needed to examine the effectiveness of both intravenous and oral bisphosphonates in improving bone mineralization [15].

6. Guidelines for nutritional management post bariatric surgery

Recently, updated guidelines for post-operative nutritional and metabolic support of patients post bariatric surgery were published by the American Association of Clinical Endocrinologists in collaboration with multiple societies [10].

- The follow-up should be scheduled depending on the bariatric procedure performed.
    - For LAGB, it should monthly for the first year and then annually
    - For LSG, it is recommended at 1, 3, 6, 12 months and then annually
    - For RYGB, the recommended follow up is at 1, 3, 6, 12 months and biannually or annually thereafter
    - For BPD/DS and other malabsorptive procedure, the recommended follow up is at 1, 3, 6 months and biannual thereafter.
- Routine metabolic and nutritional monitoring is recommended after all bariatric procedures. This includes:
    - Complete metabolic panel, complete blood count with each visit
    - Iron studies at baseline and after BS as needed
    - B12 annually then every 3–6 months for all type of BS (measurement of methylmalonic acid and homocysteine level are optional)
    - Folic acid level (measurement of red blood cell folic acid level is optional), 25-vitamin D and intact parathyroid hormone (PTH) post RYGB and BPD/DS
    - Vitamin A (initially and every 6–12 months thereafter) for BPD/DS and it is optional for RYGB
    - Copper/ceruloplasmin, zinc, selenium evaluation after malabsorptive bariatric surgical procedures (RYGB and BPD/DS) at least annually, or with symptoms of deficiency
    - Thiamine evaluation in symptomatic patients

- Dual-energy X-ray absorptiometry for bone density at 2 years: for RYGB and BPD/DS.

- The recommended micronutrients supplementations post bariatric surgery to prevent nutritional deficiencies include [10]:

  - Two adult multivitamins plus minerals (each containing iron, folic acid, thiamine, zinc, copper; chewable form initially then tablets).

  - Vitamin B12 (Cobalamin): 350–1000 μg dose can be administrated orally (disintegrating tablet, sublingual, or liquid), nasal spray or parenteral (1000 μg monthly intramuscular or subcutaneous).

  - Iron: 18–60 mg of elemental iron daily included in the multivitamins and additional supplements can be added if required.

  - Vitamin D: at least 2000–3000 international units of vitamin D (titrated to therapeutic 25-hydroxyvitamin D levels >30 ng/mL)

  - Elemental calcium: appropriate dose of daily calcium varies by bariatric procedure. About 200–1500 mg daily for LAGB, LSG and RYGB, and 1800–2400 mg daily for or BPD/DS. Calcium citrate is preferred than calcium carbonate because it is better absorbed in the absence of gastric acid.

Commercial products that are used for micronutrient supplementation after BS need to be discussed with a healthcare professional familiar with dietary supplements, since many products are adulterated and/or mislabeled [10].

7. Preventive strategies of nutritional deficiencies

Since increased adherence with follow-up is associated with improved outcomes, various strategies should be implemented to minimize attrition. Addressing the problem of non-adherence in BS will require the support of qualified healthcare professionals [10, 15]. Multidisciplinary teams with strong communication skills and the involvement of behavioral health experts assist in identifying and addressing compliance barriers. The following strategies may help to improve adherence in the bariatric patients and prevent nutritional deficiencies:

- Increase patient engagement in after care appointments. The bariatric team can utilize strategies such as frequent calls, reminders letters, flexible scheduling/variety of appointment times, laboratory results, newsletters to provide reinforcement for follow up [58]

- Develop innovative strategies to address barriers to follow-up, such as remotely delivered interventions, smart-phone apps, and follow up video appointments [59]

- The long-term follow-up visits should include screening for micronutrient deficiencies, bone health, and monitoring of nutrition-related diseases. Reinforcing healthy eating habits is also recommended, such as eating slowly, portion control, and meeting protein requirements

- Focus on adherence in the areas that are most critical for patient well-being. For instance, vitamin deficiency can cause serious health problems, including, in rare cases, encephalopathy

- Address barriers and causes of non-compliance with multivitamins supplementation. For example, the most frequent reasons for non-adherence to vitamins, i.e., forgetting, difficulty swallowing or not liking to take pills. These issues can be solved by using pill organizers and electronic reminders which can assist with memory issues. Offering chewable or liquid form of vitamins to will also aid bariatric patients with swallowing difficulty [59]

- The role of the family physician in bariatric post-surgery care is important to consider. However, the nature of their involvement post-surgery care is currently unclear [60]. Greater role clarity and enhanced collaboration between surgeons, general practitioners and patients following surgery is likely to enhance the experience and outcomes for patients and encourage and support the maintenance of postsurgical care [60].

- Patient education before and after surgery plays a key role in the adherence to micronutrient supplementation and improvement of BS outcomes. Patients should be encouraged to become involved in their own care. Lectures and discussions provided by healthcare experts from multiple disciplines in small groups, or individual sessions utilizing both written or web-based delivery should be done to support learning needs of the bariatric patients. Moreover, patient education methods should focus on high-quality, cost-effective, and patient-centered educational programs for bariatric surgery [61].

8. Conclusions

BS is the most effective strategy for the treatment of severe obesity and for the resolution of comorbid medical conditions. Post-surgery, patients are at increased risk for nutritional deficiencies which may result in serious complications if they are not recognized and treated promptly. Adherence to multivitamins supplementations is important to prevent such deficiencies. Multidisciplinary approach with close monitoring is the key for the long-term success after bariatric surgery.

Conflict of interest

The authors declare no conflict of interest.

Acronyms and abbreviations

BS	bariatric surgery
LAGB	gastric banding
LSG	laparoscopic sleeve gastrectomy
RYGB	Roux en Y gastric bypass
OAGB	one-anastomosis gastric bypass
SADI-S	single anastomosis duodeno–ileal bypass with sleeve gastrectomy
BPD/DS	biliopancreatic diversion with duodenal switch

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