Module 23.4.

Bariatric Surgery - Nutritional and Metabolic Complications

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Introduction and bariatric procedures based on:
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Learning Objectives

- To understand the principles of various bariatric surgical techniques;
- To know the various nutritional and metabolic complications of bariatric surgery;
- To be familiar with appropriate post bariatric surgery follow-up;
- To be aware of the importance of protein intake after bariatric surgery;
- To be familiar with routine supplementation of micronutrients after bariatric surgery;
- To be aware of the importance of continuing follow-up after bariatric surgery.

Contents

Key Messages

- Bariatric surgery is associated with long-lasting effects on obesity as well as on obesity-associated morbidity;
- Bariatric surgery may cause major nutritional deficiencies;
- Various metabolic phenomena may complicate bariatric surgery;
- Prevention of nutritional deficiencies depends upon attendance to follow-up programmes;
- Adherence to adequate dietary protein and micronutrient supplementation after surgery is of utmost importance;
- Early identification and treatment of nutritional deficiencies could prevent irreversible complications.

1. Introduction

Obesity is a multifactorial disease with an increasing incidence, particularly in the western world. Its co-morbidities such as type 2 diabetes, hypertension, dyslipidaemia, joint disorders and significant reduction in health related quality of life constitute a health problem with major medical and socio-psychological as well as economic consequences. In order to acknowledge the fact that the epidemic of obesity affects developing as well as developed countries, the term "globesity" has been introduced. According to WHO, in 2008 1.4 billion adults were overweight and approximately 500 million obese (200 million men and 300 million women).

Primary treatment for obesity should include lifestyle changes such as exercise and diet, behavioural modification and/or pharmacological treatment. Unfortunately, many patients experience unsatisfactory results with such conservative treatment modalities. Increasing amounts of data demonstrate that bariatric surgery is the only treatment for obesity with documented long-lasting effects not only on obesity itself, but also on associated disease. Most reports after bariatric surgery record losses of 20-30% of preoperative total body weight, corresponding to loss of 50-80% of excess body weight (EBWL) (1). Moreover, in a large matched-control study comprising more than 4 000 subjects (2) bariatric surgery was demonstrated to be associated with a significant reduction (approximately 30%) in mortality compared to controls who were traditionally treated in primary health care. These positive effects are especially true for patients with obesity related diseases. In a recent report a lower incidence of all-cause mortality (49%), and mortality from cardiovascular disease (34%), and positive effects on severe kidney disease were demonstrated in patients with type 2 diabetes who underwent bariatric surgery compared with a control group (3).

In parallel with improved results after bariatric surgery, and lower surgery-related morbidity and mortality, the number of surgical procedures for obesity has increased during the past decade: in 2011, 340 000 procedures were performed worldwide (4), while in 2017 228,000 procedures were performed in the U.S alone (1).

The health-related gains from bariatric surgery should be weighed against the risks associated with the procedure including the short- and long-term complications. The immediate complications are mainly directly related to the surgical procedure itself, with ~0.1% mortality, anastomotic leaks, infectious complications, cardiovascular events, and thromboembolic complications (3). About 20-30% of patients experience suboptimal weight loss or significant weight regain within the first few postoperative years (5). This module will focus on the nutritional and metabolic complications of bariatric surgery, namely macro- and micro-nutrient deficiencies and their consequences (e.g. liver failure), cachexia, fractures and bone density decrease, hypoglycaemia, dumping syndrome, pancreatic insufficiency, and adverse pregnancy related outcomes. Prevention of such complications has prompted specific peri-operative evaluation and follow-up. Metabolic and nutritional complications occur after bariatric surgery, sometimes as early as a few days or weeks post-surgery, and sometimes even years after the surgery.

2. Bariatric Surgical Procedures

Surgical bariatric procedures are traditionally classified as restrictive, malabsorptive, or combined. In restrictive procedures, weight loss is achieved solely by reduced capacity for nutritional intake, whereas in malabsorptive procedures, the effect is induced through bypass of absorptive and secretory areas of the stomach and small intestine. Vertical

banded gastroplasty (VBG, **Fig. 1**)**,** adjustable gastric banding (AGB, **Fig. 2**), and sleeve gastrectomy (SG, **Fig. 3**)**,** are purely restrictive procedures whereas biliopancreatic diversion (BPD, **Fig. 4**), and biliopancreatic diversion with duodenal switch (BPD-DS**, Fig. 5**), are malabsorptive. In roux-en-Y gastric bypass (RYGB, **Fig. 6**), a small gastric pouch is connected to the small intestine. The "biliopancreatic limb" (remaining stomach, duodenum and proximal small intestine) is connected via an enteroanastomosis to the alimentary limb 120-150 cm below the gastric pouch. By this means, RYGB combines restriction of food intake with reduction of gastrointestinal absorptive area. An operation which is gaining popularity is the one-anastomosis gastric bypass (OAGB), also known as mini-gastric bypass (MGB) or omega-loop gastric bypass **(Fig. 7**). It seems that 'you get what you pay for' applies here - the malabsorptive procedures are more effective while holding a greater potential for complications (6).

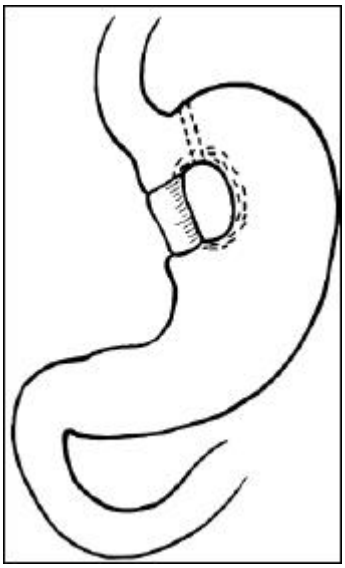

Fig. 1. Vertical banded

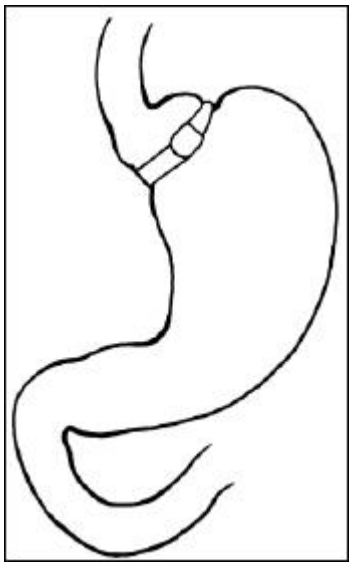

Fig. 2. Adjustable gastric banding

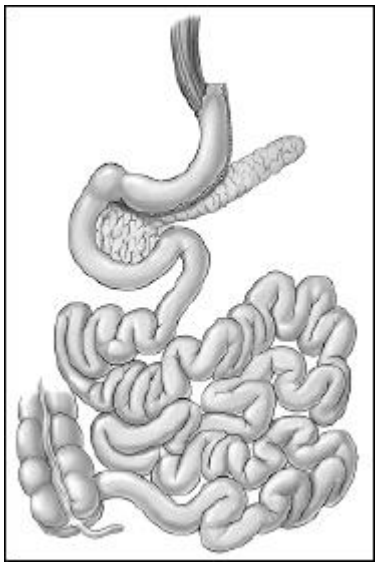

Fig. 3. Sleeve gastrectomy

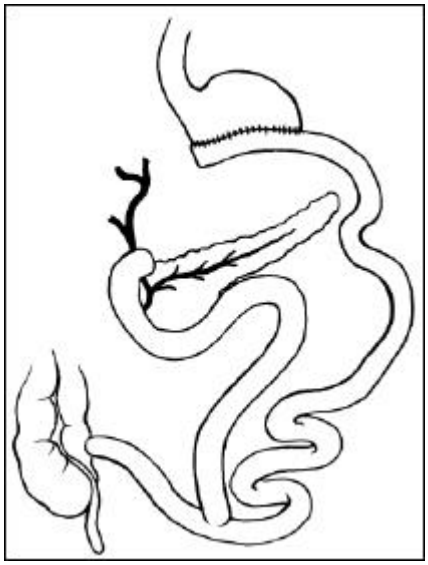

Fig. 4. Biliopancreatic diversion

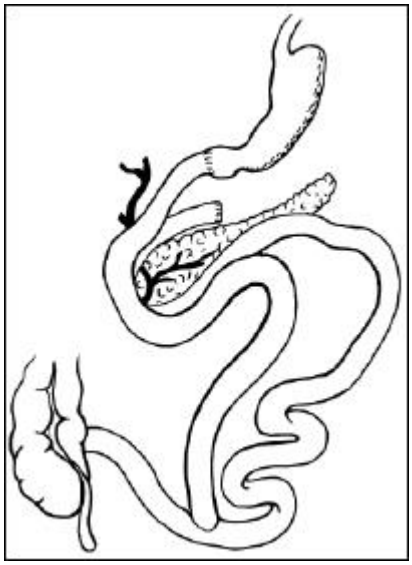

Fig. 5. Biliopancreatic diversion with duodenal switch

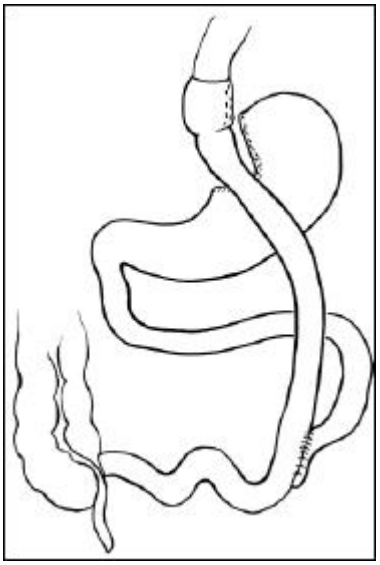

Fig. 6. Roux-en-Ygastric bypass

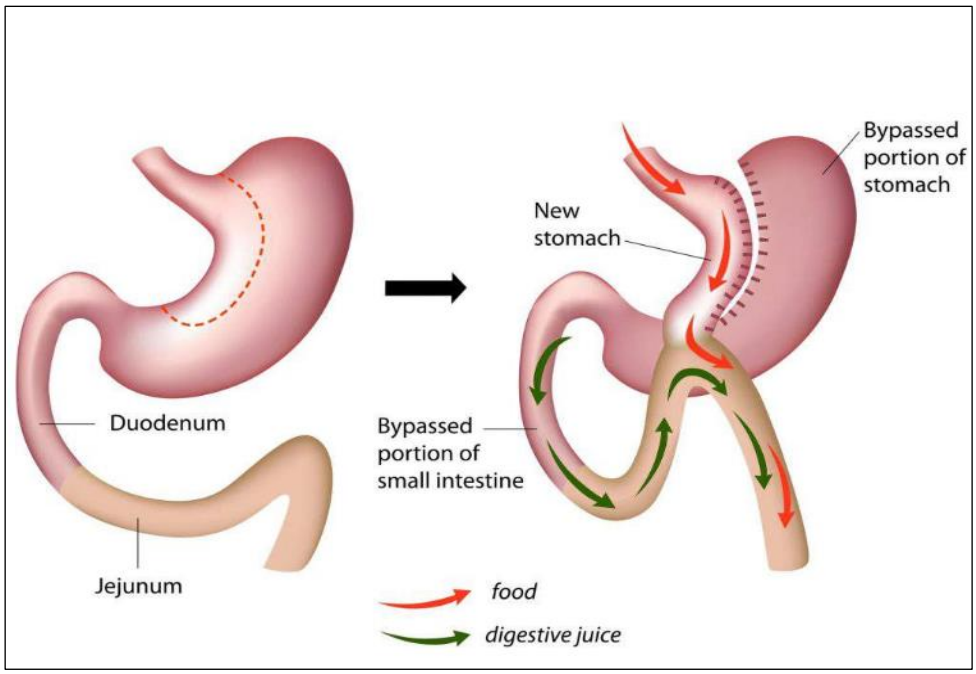

Fig. 7 One-anastomosis gastric bypass (OAGB)

3. Nutritional Deficiencies

Macro and micro-nutritional deficiencies are common after bariatric surgery and can occur at different time points, even many years post-operatively. The mechanism by which nutritional deficiencies occur is different for restrictive procedures and for malabsorptive procedures. The pathophysiology for nutritional deficiencies after restrictive procedures is low intake and/or vomiting, whereas in malabsorptive procedures the aetiology is mostly malabsorption. In both instances unbalanced diet, poor food preferences along with non-adherence to recommendations regarding nutritional supplements play a major role in the nutritional deficiencies. On the whole nutritional deficiencies are more prone to occur after malabsorptive than with restrictive procedures**.** Prevention of nutritional deficiencies relies on adequate pre-operative assessment and post-operative follow-up. Fieber et al. found that six percent of the patients were malnourished prior to bariatric surgery (7). These patients had a higher propensity for peri-operative complications. **Table 1** presents results of two studies which assessed nutritional status/intake prior to bariatric surgery (8, 9). Vitamin D, Vitamin A, Vitamin E, Vitamin B12, and iron deficiencies are common in this group of patients. Full laboratory evaluation of micronutrient status is not considered mandatory as part of the pre-operative evaluation. Normalization of nutritional deficiencies are recommended by various groups, but are not yet part of the routine management of all patients.

Table 1
Pre-surgical assessment of micronutrients (8, 9)

	% Deficient/Low	% Excess
Vitamin B1	0.3	7
Vitamin B2	0.7	16.7
Vitamin B6	0	2.9 - 24
Folate	0	3.3
Vitamin B12	15.7 - 16.4	84.3 - 0.4
25 (OH) Vitamin D	75.6-100	0-0.2
Ferritin	28.2	0
Transferrin saturation	59	2
Vitamin A	44.1	55.9
Vitamin E	34.1	65.9

3.1 Macronutrient Deficiency

Severe malnutrition might ensue after bariatric surgery. This can lead to morbidity and mortality. Short bowel after bariatric surgery and malnutrition may lead to end-organ failure and death. Pancytopenia and liver failure leading to liver transplantation or death have been described (10, 11).

Protein deficiency after bariatric surgery

Protein deficiency may be encountered after bariatric surgery, more commonly following malabsorptive procedures. Clinical presentation includes oedema, and sometimes fulminant anasarca with ascites, pericardial effusion and pleural effusion. Hypoalbuminaemia is usually present, but is not universal. It should be noted that where hypoalbuminaemia in patients who haven't undergone bariatric surgery reflects inflammation, in post-bariatric surgery patients this phenomenon could be isolated, without inflammation, as a result of pure malnutrition (12). Protein malnutrition may be severe leading to recurrent hospitalizations, parenteral nutrition, and at times revisional surgery (10, 13, 14). Annual hospitalization rates due to hypoalbuminaemia are 1% per year (15) Hypoalbuminaemia was found in 2-3.7% of patients one year after bariatric surgery (10, 13). The highest rate was after single anastomosis gastric bypass, followed by RYGB. About 50% of patients who developed hypoalbuminaemia needed revisional surgery. The most important risk factors for developing protein deficiency with hypoalbuminaemia are a short common (alimentary) limb and low protein intake. A minimal protein intake of 60 g/d and up to 1.5 g/kg ideal body weight per day should be targeted (16).

3.2 Micronutrient Deficiency

Micronutrient deficiency is common after bariatric surgery. Surgical anatomy can provide an explanation to deficiencies evolving post-operatively. Vomiting, mainly due to restrictive procedures, can cause multi-nutrient deficiencies due to deficiency of effective intake (i.e. even if the patient consumes food, recurrent/continuous vomiting turns this into inefficient intake). Different nutritional deficiencies evolving from surgical manipulation of the proximal to the distal part of the intestinal may ensue. Gastrectomy is bound to cause lack of acidity. This could lead to lack of intrinsic factor or its activation. Lack of active intrinsic factor is apt to cause vitamin B12 deficiency. Bypass of the proximal small bowel predisposes to iron and folic acid malabsorption. Bypass further down may cause

malabsorption of fat soluble vitamins. Other micronutrient deficiencies have been described with corresponding laboratory and clinical manifestations. **Table 2** provides detailed information regarding the different micronutrients.
Prevention of micronutrient deficiencies relies on supplementation and adequate clinical and laboratory follow-up which is adjusted to the type of surgery and the time elapsed since surgery (see below).

Vitamin B1 (thiamine) deficiency

Described especially in the period of a few weeks to a few months post-operatively. This is due to vomiting accompanying the post-surgical period. There are no significant stores of thiamine, therefore inadequate supply of this vitamin is likely to cause clinically significant deficiency sometimes even within days. Clinical manifestations could include- peripheral and/or central nerve involvement and/or cardiac failure (17, 18). Treatment consists of high dose thiamine given parenterally. According to the European Federation of Neurological Societies and the Royal College of Physicians, 500 mg of parenteral thiamine should be given three times daily until symptoms of acute Wernicke Encephalopathy resolve. Most patients improve with parenteral thiamine treatment, but neurologic sequelae are common; moreover a progressive clinical course was visible in about a third of the patients. Prevention is the key action. Any patient with vomiting, especially if protracted, should be considered for parenteral thiamine treatment.

Anaemia

Anaemia is of multifactorial cause after bariatric surgery. Iron, B12, and folic acid deficiency are the most common aetiologies for anaemia, but other deficiencies such as copper deficiency might play a role as well. In one longitudinal study with follow-up of five years after RYGB, the prevalence of anaemia increased from 4% preoperatively to 24% in females, and from 0% to 7% in males (19). In an additional study, the percentages of 2116 patients developing mild, moderate, and severe anaemia were 27%, 9%, and 2% at 1 year after RYGB and increased to 68%, 33%, and 11% at 5 years (20). Risk factors for developing anaemia include female gender, pre-operative low ferritin level, and rapid weight loss.

Vitamin B12 deficiency

Vitamin B12 is water soluble vitamin absorbed in the distal ileum in combination with intrinsic factor released from the parietal cells of the stomach. Any surgery affecting the parietal cells, and/or the distal ileum, and/or bile acid function is apt to cause B12 deficiency. Since usually there is a reserve of B12 it could take years until overt deficiency is evident. Deficiency will occur earlier in patients who have low or borderline levels of B12 before surgery. Clinical presentation can include neurological manifestations and symptoms of anaemia. Laboratory results reveal macrocytic anaemia and sometimes pancytopenia. Neurological abnormalities might not always be reversible with treatment. Treatment is by the sub-lingual route, parenteral route or by nasal vaporising.

Iron deficiency

Bariatric surgery and specifically operations which affect the integrity of the stomach and/or absorption in the small bowel are likely to cause iron deficiency unless this is specifically addressed. In a longitudinal study over five years, levels decreased gradually in both genders. Iron deficiency increased from 6% preoperatively to 42% at 5 years in females, and from 0% to 9% in males (19). In a study of type 2 diabetic patients the

frequency of iron deficiency anaemia after RYGB reached ~25% two years after surgery (21).

Folate deficiency

Folate deficiency causes macrocytic anaemia. There is a differential diagnosis for macrocytosis. It is of great importance to rule out other causes of macrocytic anaemia (e.g. B12 deficiency) before treatment since treatment with folate can unmask B12 deficiency resulting in irreversible consequences if one is treated without the other (see above).

Vitamin A deficiency

Vitamin A is a lipid soluble vitamin involved in vision and embryonal development. Vitamin A deficiency has been described in up to 90% of pregnant women after RYGB; 75% of them developed frank night blindness (22, 23).

Vitamin D

Vitamin D deficiency is frequent in patients before surgery. After surgery the frequency of deficiency differs according to the operation type, the highest being after BPD-DS where it reaches 100% of patients in some series (see below). Vitamin D deficiency is related to poor bone density and fractures. Since it is involved in numerous additional functions it might affect additional processes which are at this time still ill defined. Supplementation is the rule with serum level follow-up.

Additional deficiencies

Copper deficiency can occur with pancytopenia, neurologic manifestations, fatigue, myeloneuropathy-like syndrome, spastic gait, sensory ataxia, and sudden bilateral blindness. Intake of Zinc supplements competes with copper absorption and might cause worsening of copper deficiency.
Zinc deficiency presents with dermatitis, B6 deficiency with diarrhoea and dermatitis. Vitamin E and vitamin K deficiency are also encountered.

Miscellaneous metabolic derangements

Reduced bone density, fractures, and sarcopenia

There are numerous studies assessing these issues. Most of the longitudinal studies describe a decline in bone density with fractures. When looking into studies with a comparative group the results are less clear. When the comparative group consists of patients with BMI similar to the pre-operative BMI of the patients, even when adjustment is made for other risk factors, after bariatric surgery patients have lower bone density and a higher frequency of fractures. It is uncertain however whether the comparative group should consist of people with similar pre-operative BMI or similar post-operative BMI. Ablett et al conclude in a recent review and meta-analysis: '…that bariatric surgery, predominantly malabsorptive in nature was associated with an increased risk of fracture compared to people of similar starting weight who did not undergo surgery. However, it is unclear whether the risk of fracture for adults, post-bariatric surgery at their lower weight exceeds people of similar weight in the general population' (14, 20).
De novo sarcopenia or worsening of sarcopenia may occur after bariatric surgery. About a third of patients are found to be sarcopenic after surgery. The most predictive factor for

sarcopenia after bariatric surgery is the presence of sarcopenia before the surgery (24, 25).
It seems that lifestyle modifications including exercise - aerobic as well as endurance exercise - can reduce or even prevent decline in bone density and occurrence of sarcopenia after surgery. This needs to be proven in additional studies. It should be stressed that when providing recommendations for physical activity to an obese patient care should be taken to avoid activities which might cause damage due to the obesity itself or the co-morbidities. Weight bearing activities should be postponed to prevent skeletal damage.

Post-bariatric hypoglycaemia and dumping syndrome
All of the phenomena described herein appear post-prandially and do not appear when the patient is fasting.
Hypoglycaemia has been described after bariatric surgery in two scenarios - early and late. Early onset hypoglycaemia after bariatric surgery is related to the dumping syndrome. Dumping syndrome is divided into early and late types. Both emerge from the passage of food from the stomach to the small intestine. The early dumping syndrome appears not long after a meal.

The pathophysiology of this is the passage of water to the small intestine due to osmotic pressure of the food, this causes lowering of blood pressure. This could result in fainting or near fainting, weakness, and tachycardia. The postponed/late type of dumping syndrome can appear early in the course post-surgery. It results from passage of food to the small intestine resulting in insulin secretion and glucose level lowering. This occurs about two hours post a meal, usually with simple carbohydrates. The symptomatology is similar/identical to the early dumping syndrome. Diagnosis relies on history taking with blood pressure and glucose monitoring.

A different sub-set of hypoglycaemia is a hypoglycaemia which appears post prandially a year or more after the operation. This postprandial hypoglycaemia can be severe. Salehi et al stressed this: 'Post bariatric hypoglycemia can be severe and disabling for some patients, with neuroglycopenia (altered cognition, seizures, and loss of consciousness) leading to falls, motor vehicle accidents, and job and income loss. Moreover, repeated episodes of hypoglycemia can result in hypoglycemia unawareness, further impairing safety and requiring the assistance of others to treat hypoglycemia' (26). The aetiology for this kind of hypoglycaemia is perceived to be hyper-sensitive beta cells and/or frank hypertrophy or hyperplasia of the pancreatic beta cells (nesidioblastosis). The incidence of this post-bariatric hypoglycaemia is unknown due to differences in the diagnostic criteria. In a population study post-bariatric hypoglycaemia was found in 2% of patients (27). An incidence as high as 80% was described in pregnant women after RYGB and of ~30% was described in patients with type 2 diabetes in a study using oral glucose tolerance for diagnosis (28, 29).
Treatment first requires awareness. Avoiding simple carbohydrates (high glycaemic index) is of upmost importance in combination with increased consumption of small meals. If these measures are not enough than medications are considered. Intractable hypoglycaemia is an indication for revisional surgery. At times even this is not enough and partial pancreatectomy is performed.

Table 2
Micronutrient deficiency (30)

	LAGB	LSD	RYGB	BPDDS
Thiamin (B_1)	0	0	12	10 – 15
Folate (B_9)	10	10 – 20	15	15
Piridoxine (B_6)	0	0 – 15	0	10
Cobalamin (B_{12})	10	10 – 20	30 – 50	22
Vitamin A	10	10 – 20	10 – 50	60 – 70
Vitamin D (<30 ng/dL)	30	30 – 70	30 – 50	40 – 100
Vitamin E	0	0 – 5	10	10
Vitamin K	0	0	0	60 – 70
Iron	0 – 32	15 – 45	25 – 50	25
Copper	–	10	10	70
Zinc	–	7 – 15	20 – 37	25

(–) indicated that data not available
Abbreviations: LAGB laparoscopic-assisted gastric banding, *LSG* laparoscopic sleeve gastrectomy, *RYGB* Roux-en-Y gastric bypass, *BPDDS* biliopancreatic diversion with duodenal switch

4. Post-operative Follow-up and Supplementation

The best way to treat nutritional deficiencies is to prevent them or identify them early. Every patient who has undergone bariatric surgery should be assessed and treated by a dietician. The frequency of appointments will be more frequent in the immediate post-operative period and is less frequent later on. Laboratory assessment is mandatory as well. There are many protocols for follow-up and supplementation. There are currently no tools to compare existing protocols. **Table 3** provides a summary of recommendations from: 'Practical Recommendations of the Obesity Management Task Force of the European Association for the Study of Obesity for the Post-Bariatric Surgery Medical Management' (31).

Table 3
Minimal periodic surveillance for nutritional deficiencies after bariatric surgery (28, 31)

	AGB	SG	RYGB	BPD – BPD/DC
Timing	every 6 months in the first year every 12 months thereafter	every 3-6 months in the first year every 12 months thereafter	every 3-6 months in the first year every 12 months thereafter	every 3 months in the first year every 6-12 months thereafter
Assessment	CBC, platelets electrolytes iron, ferritine vitamin B12 folate vitamin D PTH	CBC, platelets electrolytes iron, ferritine vitamin B12 folate vitamin D PTH	CBC, platelets electrolytes iron, ferritine vitamin B12 folate vitamin D PTH 24-H U-calcium osteocalcin	CBC, platelets electrolytes iron, ferritine vitamin B12 folate vitamin D PTH 24-H U-calcium osteocalcin vitamin A vitamin E INR albumin prealbumin

AGB = Adjustable gastric banding; SG = sleeve gastrectomy; RYGB = gastric bypass; BPD = biliopancreatic diversion; BPD/DC = biliopancreatic diversion with duodenal switch; CBC = complete blood count; PTH = intact parathyroid hormone; 24-H U-calcium = 24-hour urinary calcium

5. Summary

The number of bariatric surgical procedures performed for morbid obesity is continuously increasing in parallel with the "epidemic" of obesity. Bariatric surgery has been shown to result in long-lasting beneficial effects on obesity, co-morbid disease and mortality. However, bariatric surgery involves a major abdominal surgical procedure with the risk of serious short- and long-term nutritional and metabolic complications. Adherence to a pre- and post-operative nutritional assessment and treatment protocol is essential to minimize the chance of micro- and or macronutrient deficiencies and/or metabolic complications. It is of importance to note that such complications can arise at any time point peri-operatively and even many years after surgery. Prevention and awareness are the key factors to prevent and treat these complications. A multidisciplinary team should collaborate in the preoperative setting with emphasis on information and medical assessment. Good results depend on the patient's strict adherence to a life-long follow up programme.

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