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Review Article

Nutritional Deficiencies following Bariatric Surgery: What Have We Learned?

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Deficiencies in vitamins and other nutrients are common following the Roux-en-Y gastric bypass (RYGBP), biliopancreatic diversion (BPD) and biliopancreatic diversion with duodenal switch (BPD-DS), and may become clinically significant if not recognized and treated with supplementation. This paper presents a review of the current literature and evidence of the most commonly deficient vitamins and minerals following weight loss surgery, including protein, iron, vitamin B_{12}, folate, calcium, the fat-soluble vitamins (A, D, E, K), and other micronutrients. The deficiencies appear to be more substantial following malabsorptive procedures such as BPD, but occur with restrictive procedures as well. The review suggests that further studies are needed to evaluate the clinical significance of the nutritional deficiencies, and to determine guidelines for supplementation.

Key words: Morbid obesity, Roux-en-Y gastric bypass, biliopancreatic diversion, laparoscopic surgery, nutrition, deficiency, malabsorption, protein, iron, vitamin, calcium

Introduction

The Roux-en-Y gastric bypass (RYGBP), biliopancreatic diversion (BPD) and biliopancreatic diversion with duodenal switch (BPD-DS) are common surgical weight loss operations. These surgeries, which are now routinely performed laparoscopically, induce weight loss by a combination of restriction and malabsorption. Despite the desired weight loss achieved with these operations, postoperative deficiencies in protein, iron, and other vitamins and minerals are common and require supplementation to maintain normal levels. The current recommended dietary allowances of these vitamins and nutrients are published by the United States Department of Agriculture (USDA) (Table 1). Deficiencies mostly occur from malabsorption secondary to bypassing segments of gastrointestinal tract, where the various nutrients are absorbed (Figure 1). It is possible, but not proven, that a fur-

Table 1. Recommended dietary allowances (RDA)

Vitamin/Nutrient	RDA
Protein	46-56 g/day
Calcium	1000-1200 mg/day
Iron	8-18 mg/day
Thiamine	1.1-1.2 mg/day
Folate	400 ug/day
Vitamin B_{12}	2.4 ug/day
Vitamin D	5-15 ug/day
Vitamin A	700-900 ug/day
Vitamin E	15 mg/day
Vitamin K	90-120 ug/day
Zinc	8-11 mg/day
Magnesium	310-420 mg/day
Selenium	55 ug/day
Copper	900 ug/day
Vanadium	Not Determined

Source: United Sates Department of Agriculture, 2001 Guidelines

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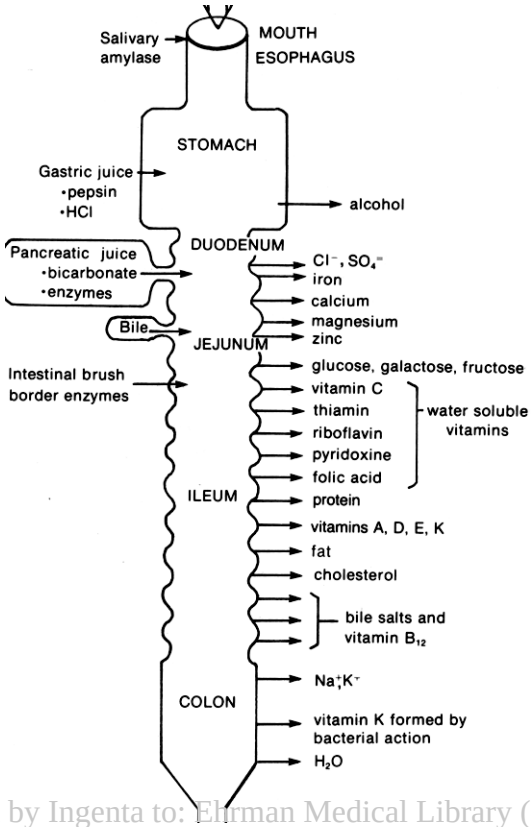

Figure 1. Sites of absorption of common vitamins and nutrients in the gastrointestinal tract.
Adapted from: Mahan and Escott-Stump: *Krause's Food, Nutrition and Diet Therapy,* 9/e, p. 13, ©1996, with permission from Elsevier.

ther deficiency in some nutrients can occur with restrictive operations resulting from decreased intake and a tendency toward avoidance of certain nutrient-rich foods postoperatively, due to intolerance. While surgeons who routinely perform these operations are expected to understand the resulting nutritional deficiencies and use appropriate supplementation accordingly, metabolic abnormalities are often undiagnosed or misdiagnosed. Brolin and Leung[1] noted a wide variation in performance of laboratory tests and use of supplements following RYGBP and BPD in a survey of 109 bariatric surgeons. In fact, not all of the respondents recommended prophylactic multivitamins, and the incidence of deficiencies after RYGBP was considerably underestimated. The majority of the literature on nutritional deficiencies resulting from bariatric surgery consists of case reports or series, retrospective reviews and occasional prospective studies.

This paper provides a review of the most recent literature, and objectively presents the deficiencies that can result from bariatric surgery, focusing on the BPD, BPD-DS and RYGBP. The principle deficiencies including protein, iron, vitamin B_{12}, folate, calcium, vitamin D, thiamine, and the fat-soluble vitamins are discussed.

Protein Deficiency

It is well known that protein is one of the main nutrients affected by bariatric surgery. Protein malnutrition, characterized by hypoalbuminemia, anemia, edema, asthenia and alopecia represents a serious potential late complication of BPD. The pathogenesis is multifactorial, but is most commonly related to excessive malabsorption from bypassing segments of small intestine where protein is absorbed (Figure 1), and to a lesser degree from food limitation.[2] Protein deficiency (albumin <3.5 g/dL) can occur after the RYGBP, but is more commonly seen in malabsorptive procedures, such as the BPD (Table 2).

In order to assess protein deficiency occurring after RYGBP, Brolin performed a prospective randomized study of super-obese patients (BMI ≥50). At a minimum of 2 years after surgery, 13% of the patients who underwent a distal RYGBP were found to have hypoalbuminemia, some requiring TPN for severe protein malnutrition. In contrast, protein deficiency was not noted in the group of patients with a shorter Roux-limb of <150 cm.[3] Another study had similar findings with a 5.9% protein deficiency rate 20 months after the distal RYGBP, but no deficiency after the standard RYGBP or vertical banded gastroplasty.[4] A retrospective study determined that the peak incidence of hypoalbuminemia, which occurred at 1-2 years postoperatively, was negligible, with mean albumin values >4 g/dL. After the distal RYGBP and standard RYGBP, only 3% and 1.4% of patients respectively, had an albumin level <3 g/dL.[5] In another prospective randomized study done by Brolin et al,[6] no cases of protein deficiency were found in patients after the RYGBP. The mean follow-up was 43 months.

Several studies looked at protein deficiency in BPD and BPD-DS. Marceau et al[7] found a high incidence of protein deficiency in his study. At a follow-up of 79 months after BPD, 11% of the patients had

Table 2. Studies of protein deficiency

Study	Study Type	N=	Operation	Protein Deficiency Rate and Length of Follow-up
Brolin, 2002[3]	Prospective	298	Distal RYGBP	13% at 2 years
Skroubis, 2002[5]	Retrospective	79	RYGBP	1.4% at 1 year
			Distal RYGBP	3% at 1 year
Kalfarentzos, 1999[4]	Retrospective	38	RYGBP	0% at 20 mo.
		17	Distal RYGBP	5.9% at 20 mo.
Brolin, 1992[6]	Prospective	45	RYGBP	0% at 43 mo.
Dolan, 2004[10]	Retrospective	134	BPD/BPD-DS	18% at 28 mo.
Rabkin, 2004[11]	Retrospective	589	BPD-DS	0% at 3 years
Skroubis, 2002[5]	Retrospective	95	BPD	3% at 2 years
Nanni, 1997[9]	Retrospective	59	BPD	3.4%
Marceau, 1995[7]	Retrospective	92	BPD	11% at 79 mo.

serum albumin levels <3.5 g/dL, and 2.4% had serum albumin levels <3.2 g/dL. Revisions were done on 6% of the patients to improve low serum albumin values.[7] In another study, questionnaires were mailed and completed by 858 patients who underwent BPD.[8] At 5 years, there was approximately a 40% follow-up rate. Of those who were followed up, rehospitalization was needed for severe protein deficiency in 3.7% of patients. Revisions were performed in 6% of the total patient population, and were mainly for recurrent protein malnutrition.[8] Nanni et al[9] reviewed their experience with BPD by ad hoc stomach resection, and found that the main specific complication was protein deficiency, which occurred in 3.4% of the patients.

Two other studies addressed protein deficiency following BPD-DS. Dolan et al[10] compared BPD with and without duodenal switch and found that 18% of patients were hypoalbuminemic, with no significant differences between the two surgeries. The results were on average 37 months after BPD and 23 months after BPD-DS, using a common channel length of 50 cm.[10] A contrasting study looked at 589 patients who underwent BPD-DS, and found that albumin and total protein remained within normal limits within the first 3 postoperative years, although the common channel was 100 cm in this study.[11]

Whether protein deficiency occurred after the BPD or RYGBP, approximately 3 weeks of total parenteral nutrition (TPN) helped to correct the acute problem. Dietetic counseling and increased protein intake helped to prevent recurrences in several studies.[4,5,9]

Iron Deficiency

Studies of both restrictive and malabsorptive operations indicate that patients undergoing bariatric surgery are at risk of developing iron deficiency (Table 3). The bypass of the primary site of absorption in the duodenum and proximal jejunum (Figure 1) may contribute to the development of anemia postoperatively. Anemia is usually only seen in the setting of other chronic sources of bleeding, such as menstruation or stomal ulceration.[2] At 1-year follow-up, the incidence of iron deficiency has been estimated anywhere from 6% to 33%.[12,13]

Iron deficiency has been shown to occur following RYGBP. Skroubis et al[5] followed patients undergoing RYGBP from the preoperative period to 5 years postoperatively, and demonstrated an increasing percentage of serum iron and ferritin deficiency over time. Iron deficiency increased from 26% of the sample preoperatively to 39% at 4 years, and then decreased again to 25% at 5 years. Ferritin deficiencies increased from 16% of the sample preoperatively to 44% at 4 years and then decreased to 25% at 5 years. With the distal RYGBP, iron deficiency rates were more prevalent than in previous studies (45-52% at 4 years follow-up).[3] In the above studies, multivitamin supplements were given to patients, with additional oral iron prescribed for

Table 3. Studies of iron deficiency

Study	Study Type	N=	Operation	Iron Deficiency Rate and Length of Follow-up
Brolin, 2002[3]	Retrospective	298	RYGBP	45-52% at 2 years
			Distal RYGBP	49% at 2 years
Scroubis, 2002[5]	Retrospective	79	RYGBP	39% at 4 years
				25% at 5 years
Kalfarentzos, 1999[4]	Retrospective	38	RYGBP	13.1% at 20 mo.
		17	Distal RYGBP	5.9% at 20 mo.
Dolan, 2004[10]	Retrospective	134	BPD/BPD-DS	22.9% at 28 mo.
Rabkin, 2004[11]	Retrospective	589	BPD-DS	0% at 3 years
Scroubis, 2002[5]	Retrospective	95	BPD	44% at 4 years
				100% at 5 years
Kalfarentzos, 2001[15]	Retrospective		VBG	32% at 4 years
Cooper, 1999[16]	Retrospective	26	VBG	0% at 1 year

menstruating women.

While some studies have demonstrated iron deficiency over time following BPD, others have not observed this trend. In a small-sample study by Skroubis et al,[5] ferritin deficiency increased from 6.45% in the preoperative period to 55.6% at 4 years and 100% at 5 years. Iron deficiency rates were 32.6% preoperatively and increased to 44.4% at 4 years and 100% at 5 years. Marceau and co-workers[14] found abnormal ferritin levels (<20 ug/L) preoperatively in 4% of the total sample, and up to 25%-40% at follow-up. Iron deficiency did not differ in BPD with or without duodenal switch, and occurred at a rate of 22.9% at mean follow-up of 28 months.[10] Other studies have not demonstrated iron deficiency, including a larger scale study of 589 patients who underwent laparoscopic BPD-DS with a common channel length of 100 cm, which showed that mean serum iron levels remained within the normal range over 3 years.[11] Most BPD studies had protocols for multivitamin supplementation, including iron for menstruating women.[5,10]

There is inconsistent data regarding iron deficiency after purely restrictive operations such as the vertical banded gastroplasty (VBG). One study reported a 20% rate of iron deficiency before VBG, increasing to 32% at 4 years after VBG.[15] However, in another small-sample study of VBG patients, mean serum ferritin remained within normal ranges at 5 months and 1 year.[16] Patients were advised to take multivitamin supplements, with a 90% reported adherence at 5 months and 55% at 12 months.[16]

Therapeutic studies for iron deficiency are few, but one suggests that the addition of vitamin C supplementation to iron supplementation significantly increases ferritin levels, more than would be seen with iron alone.[17]

Vitamin B_{12} and Folate Deficiency

Vitamin B_{12} and folate deficiencies are often evaluated together. Studies indicate that these deficiencies are fairly prevalent after bariatric operations (Table 4). The sites of vitamin B_{12} and folate absorption in the small intestine are shown in Figure 1. Despite the likelihood of malabsorption, other possibilities in the pathogenesis of this deficiency may exist. Marcuard et al[18] identified a 36% rate of vitamin B_{12} deficiency after gastric bypass at a mean of 22 months postoperatively. Schilling tests, a common test performed to evaluate vitamin B_{12} absorption in which urinary excretion of radiolabelled vitamin B_{12} is assessed over a 24-hour period, were abnormal in just over half of those with B_{12} deficiency. Intrinsic factor (IF) was absent in the gastric juice of the bypassed gastric segment in 90% of those with deficiency and 40% of those without. Interestingly, IF was identified when parietal cell biopsies were performed in those showing lack of IF in the gastric juice of the bypassed segment. The investigators therefore concluded that an inadequacy of IF secretion is the mechanism for vitamin

Table 4. Studies of vitamin B_{12} deficiency

Study	Study Type	N=	Operation	Vitamin B_{12} Deficiency Rate and Length of Follow-up
Scroubis, 2002[5]	Retrospective	79	RYGBP	33% at 4 years
Brolin, 2002[3]	Prospective	298	RYGBP	33-37% at 3 years
			Distal RYGBP	8% at 3 years
Marcuard, 1989[18]	Retrospective	429	RYGBP	36% at 22 mo.
Halverson, 1986[12]	Retrospective	74	RYGBP	33% at 1 year
Brolin, 1991[13]	Retrospective	140	RYGBP	37% at 2 years
Scroubis, 2002[5]	Retrospective	95	BPD	22% at 4 years
Cooper, 1999[16]	Retrospective	26	VBG	0% at 1 year

B_{12} deficiency.[18]

Halverson et al[12] demonstrated that 33% of patients had vitamin B_{12} deficiency (<250 pg/ml) 1 year after gastric bypass. Folate deficiency (<3 ng/ml) in the same group occurred at a rate of 63%, despite the patients being advised to follow a multivitamin regimen. Another study of gastric bypass patients demonstrated vitamin B_{12} deficiency in 37% of patients and folate deficiency in 22% of patients by 2 years follow-up.[13] These patients had no deficiency preoperatively, and postoperative multivitamin use was shown to prevent folate and B_{12} deficiency when taken regularly. Surprisingly, during the evaluation of different Roux limb lengths, Brolin demonstrated that the distal RYGBP had significantly lower postoperative B_{12} deficiency than shorter limb procedures (8% vs 33%-37%) at 3 years.[3]

Purely restrictive operations may not demonstrate the same vitamin B_{12} and folate deficiency rates.[16] VBG patients followed to 5 months postoperatively showed no deficiency in B_{12} or folate. By 12 months, however, despite all patients having normal serum folate readings, the mean had fallen by a significant amount. Vitamin B_{12} levels did not alter significantly over 1 year.[16]

Studies have also investigated the appropriate dosing of vitamin B_{12} supplements following bariatric surgery. Rhode et al[19] conducted a 9-month study evaluating different doses of oral vitamin B_{12} supplement and the impact on patients 7 months to 4 years after either VBG or gastric bypass. All patients had vitamin B_{12} deficiency (<100 pmol/L) at the onset of study. After receiving doses of B_{12} ranging from 100 ug to 600 ug per day, a dose of at least 350 ug was needed to maintain a serum level >150 pmol/L, and therefore recommended.

Calcium and Vitamin D Deficiency

It is well known that calcium and vitamin D deficiency can occur in patients undergoing bariatric surgery. The duodenum and proximal jejunum are selective sites for calcium absorption, while vitamin D is absorbed preferentially in the jejunum and ileum (Figure 1). Malabsorption of calcium and vitamin D occurs from bypassing these segments of intestine during bariatric operations. The malabsorption of vitamin D contributes further to calcium malabsorption. With a relative lack of calcium, the production of parathyroid hormone (PTH) is increased, which leads to release of calcium from bone, potentially causing bone loss and long-term risk of osteoporosis.

It is important to keep in mind that morbidly obese individuals often have a pre-existing degree of nutritional deficiency before surgery. Secondary hyperparathyroidism and osteomalacia can occur in obese individuals who have not undergone any type of gastric bypass surgery. A prospective review of 213 patients presenting for surgical treatment of morbid obesity showed that 25% of the patients had hyperparathyroidism which was positively correlated with BMI, and 21% of patients had abnormally low levels of 25-hydroxyvitamin D.[20] Despite possible pre-existing nutritional deficiencies, studies continue to show further abnormalities in calcium and vitamin D metabolism that occur follow-

Table 5. Studies of calcium and vitamin D deficiency

Study	Study Type	N=	Operation	Calcium Deficiency Rate Vitamin D Deficiency Rate and Length of Follow-up
Brolin, 2002[3]	Prospective	298	Distal RYGBP	10% at 2 years (Ca) 51% at 2 years (Vit D)
Slater, 2004[25]	Retrospective	170	BPD	48% at 4 years (Ca) 63% at 4 years (Vit D)
Dolan, 2004[10]	Retrospective	73 61	BPD/BPD-DS	25% at 28 mo.(Ca) 50% at 28 mo. (Vit D)
Newbury, 2003[26]	Retrospective	82	BPD	25.9% at 32 mo. (Ca) 50% at 32 mo. (Vit D)
Hamoui, 2003[27]	Retrospective	165	BPD-DS	25% at 9-18 mo. (Ca) 17% at 9-18 mo. (Vit D)

ing the RYGBP and BPD or BPD-DS (Table 5).

Many retrospective studies have established a deficiency of both calcium and vitamin D following gastric bypass surgery, and some have shown evidence of postoperative metabolic bone disease. Evidence comes from observations of increased serum and urine markers of bone turnover, including elevated alkaline phosphatase (ALP) and PTH (secondary hyperparathyroidism) and markers of bone resorption and formation. Calcium and vitamin D deficiency occurs more frequently with the malabsorptive operations compared to purely restrictive operations. Chapin et al[21] compared a group of morbidly obese patients who underwent BPD to a similar group who underwent VBG, and found that the BPD group had significantly lower serum calcium, 25-hydroxyvitamin D and urine calcium excretion. The BPD group also had higher PTH, ALP and urinary hydroxyproline/creatinine ratios, suggesting increased bone turnover.

Calcium and vitamin D deficiency rates following distal RYGBP have been reported at rates of 10% and 51% respectively by Brolin.[3] Other studies evaluating RYGBP have concentrated on the metabolic consequences of calcium deficiency. Coates et al[22] studied patients for abnormalities in bone metabolism, 3 months after laparoscopic RYGBP. Markers of bone turnover including urinary N-telopeptide cross-linked Collagen type I and osteocalcin were significantly elevated, despite calcium and vitamin D supplementation. At 9 months, the trends continued, and bone mineral density and content were significantly diminished at the hip, trochanter and total body. Decreased bone mass can be particularly problematic in post-menopausal women with other risk factors for osteoporosis. Ott et al[23] looked at women 10 years after RYGBP for evidence of metabolic bone disease and compared them to obese controls. Decreased serum calcium and vitamin D, and increased markers of bone turnover including ALP and osteocalcin were significantly more prevalent in the postoperative group. When stratified by age <50 compared to age >50, the difference between the two groups did not reach significance, although the sample size was small. Goode et al[24] performed bone density screening on 44 women (23 premenopausal and 21 postmenopausal) 3 years or more following RYGBP, and compared them to similar controls. While bone mass did not differ between premenopausal women with and without RYGBP, it was altered in postmenopausal women who underwent RYGBP, with evidence of lower bone mineral content at the femoral neck. Secondary hyperparathyroidism (PTH levels >3 times the control group) and evidence of bone resorption occurred in the RYGBP group, and could not be suppressed by supplementing dietary calcium (1.2 g/day) and vitamin D (8 ug/d) for 6 months.

In order to address the impact of BPD on calcium and vitamin deficiency, a review of 170 patients by Slater et al[25] showed significant deficiencies in the fat-soluble vitamins including vitamin D, and corrected calcium levels, despite supplementation. Abnormal vitamin D levels (<50 nmol/L) were detected in 57% of patients 1 year after surgery, and 63% at 4 years. The incidence of hypocalcemia

(corrected calcium <2.25 mmol/L) increased from 15% to 48% within the 4 years. Secondary hyperparathyroidism was present in 69% of patients, and 3% showed evidence of increased bone resorption. Similar results were reported by Newbury et al,[26] who reviewed 82 patients, with a follow-up of 32 months after BPD. One-quarter of the patients were hypocalcemic and half had low vitamin D levels. Moreover, 24% showed elevated ALP levels and 63% had secondary hyperparathyroidism, despite the majority taking vitamin supplements.

In order to address whether the length of the common channel in the BPD would have an impact on the degree of vitamin D and calcium deficiency, Hamoui et al[27] looked at 165 patients who underwent BPD, comparing a common channel length of 75 cm vs 100 cm. Both groups showed an incidence of hypocalcemia of 25%; however, subnormal vitamin D levels were more prevalent in the group with the shorter common channel, and new onset hyperparathyroidism was significantly more common in this same group. This suggests that vitamin D malabsorption is more prevalent in patients with a shorter common channel, leading to a higher incidence of hyperparathyroidism and bone turnover.

Dolan et al[10] compared the nutritional side-effects of BPD alone vs BPD-DS, by performing a nutritional screen at a median follow-up of 28 months in both groups of patients. One-quarter of the patients were hypocalcemic, and almost half showed evidence of low vitamin D, despite more than 80% taking vitamin supplements. There were no significant differences between the BPD and BPD-DS, suggesting that duodenal switch does not lessen the nutritional side-effects of BPD.

Thiamine (Vitamin B_1) Deficiency

Thiamine is absorbed in the proximal small intestine (Figure 1), and may become deficient after a combination of reduced intake, frequent vomiting, and malabsorption. The incidence of thiamine deficiency after bariatric surgery has been reportedly low. Chang et al[28] found only 29 cases (0.0002%) of thiamine deficiency in a total of 168,010 bariatric cases reported in a survey by members of the American Society for Bariatric Surgery. There have been several case reports in the literature, mostly regarding consequences of thiamine deficiency. The majority of these studies concluded that vomiting was a precipitating factor for Wernicke's encephalopathy.[29-32] A retrospective study of 50 initial patients who underwent RYGBP identified one patient with Wernicke-Korsakoff encephalopathy within 2-3 months after surgery.[33] A large study looking at 1,663 patients who underwent BPD demonstrated that 0.18% developed Wernicke-Korsakoff encephalopathy within 3-5 months after surgery.[34] Although relatively rare, when thiamine deficiency is suspected, there are reports that have shown resolution of symptoms with prompt treatment of 50-100 mg/day of intravenous or intramuscular thiamine. Resolution has occurred as early as 24 hours and up to 4 months.[29-31,33,34] Since thiamine plays a role in carbohydrate metabolism, the administration of glucose and other carbohydrates without thiamine can be dangerous in a patient with thiamine deficiency.[33]

Other Fat-Soluble Vitamin Deficiencies

Fat-soluble vitamin deficiencies are both common and predictable following bariatric surgery. Malabsorption of the fat-soluble vitamins is most often seen following operations such as BPD which limit the exposure of food to the biliopancreatic secretions necessary for fat breakdown. Whereas fat-soluble vitamins are normally absorbed in the ileum (Figure 1), absorption following operations such as BPD becomes limited to the common channel, where the alimentary limb meets the biliopancreatic limb, and is consequently decreased.

Vitamin A

Several studies have demonstrated vitamin A deficiency after bariatric surgery. Dolan and co-workers[10] evaluated vitamin A levels and found them to be low (<1.6 μmol/L) in 61% of patients undergoing BPD with or without duodenal switch at 28 months follow-up. This was despite an 80% compliance rate with multivitamin supplementation. Similar findings were found in a dual-center study of 170 patients following BPD-DS, in which the incidence

of vitamin A deficiency was 69% by the fourth year after surgery, with a clear increase in deficiency over time.[25] A larger sample study demonstrated a lesser degree of vitamin A deficiency in both BPD with distal gastrectomy (12% at 8 years, n=233) and BPD with sleeve gastrectomy and duodenal switch (5% at 4 years, n=457).[14] These rates were despite supplementation protocols that included vitamin A. Brolin et al[3] observed that prophylactic supplementation of vitamin A did not prevent deficiency after distal RYGBP for 10% of patients after 4 years. While the clinical consequences of vitamin A deficiency following bariatric surgery are few, case studies have demonstrated the rare occurrence of ophthalmologic complications such as night blindness or ocular xerosis.[35-37]

Vitamin E

Vitamin E can be deficient following malabsorptive operations, most notably the BPD. Nevertheless, vitamin E deficiency is uncommon, and has not been shown to be clinically significant in patients who receive supplementation in the form of multivitamins. Slater et al[25] showed that serum vitamin E levels were normal in all study patients at 1 year after BPD, and remained normal in 96% of the patients up to 4 years after surgery. These patients were taking fat-soluble vitamin supplements. Another study showed that only 5% of patients at an average of 28 months after BPD and BPD-DS had low levels of vitamin E (<7 umol/L), although the majority were taking supplements.[10]

Vitamin K

Few clinical studies have been published regarding the deficiency of fat-soluble vitamin K postoperatively. While altered clotting and increased bleeding tendency may be seen with vitamin K deficiency, there is no reported evidence of this occurring after bariatric surgery. A study of 170 patients following BPD and BPD-DS found that the incidence of vitamin K deficiency (<0.3 nmol/L) was 68% by the fourth year. There was no clinical manifestation of increased bleeding.[25] Dolan[10] compared the BPD with and without the duodenal switch and found that half of the patients were deficient in vitamin K, with no significant differences between the two operations. The results are from an average follow-up of 37 months after BPD and 23 months after BPD-DS.

Other Deficiencies

Magnesium

Studies specifically looking at magnesium deficiency (<0.7 mmol/L) following gastric bypass operations are lacking. Marceau et al[38] found no significant abnormalities in magnesium levels before BPD, after 4 years and after 10 years. Dolan[10] found that on average 5% of patients 28 months after surgery had low magnesium levels, but the difference between those who underwent BPD and those who underwent BPD with duodenal switch was not significant. There is no reported evidence of clinical complications of magnesium deficiency following bariatric surgery.

Zinc

After BPD-DS, a two-center study showed that the incidence of zinc deficiency (<12 μmol/L) was close to 50% during 4 years of follow-up, and did not increase with time after surgery.[25] In another study, zinc deficiency occurred in only 10.8% of patients after BPD with or without the duodenal switch, despite an 80% compliance rate with multivitamin supplementation.[10] Clinical consequences of zinc deficiency have been reported. In a sample of 130 patients after VBG, 36% developed alopecia while on multivitamin supplementation. Hair regrowth occurred in all patients with additional supplementation of zinc sulphate.[39] Despite these findings, the transient hair loss typically occurring several months following bariatric surgery is a common phenomenon and its pathogenesis remains controversial.

Selenium

Few clinical studies have been published regarding selenium deficiency after bariatric surgery. When comparing BPD with and without duodenal switch, Dolan[10] found that 14.5% of patients were deficient in selenium (<0.7 umol/L), with no significant differences between the two surgeries, and no clinical consequences.

Other Micronutrients

There is no evidence in the literature to support the development of chromium, vanadium, or copper deficiencies after bariatric surgery. Nevertheless, these micronutrients are included in most multivitamin supplements.

Summary

The majority of the studies in the literature support the fact that nutritional deficiencies develop following bariatric surgery. These deficiencies are seen to a greater extent with the more malabsorptive procedures, such as BPD, although the RYGBP is also implicated. Although low nutrient levels are discovered following surgery, there is a lack of consensus in the dosing and form of supplements. The potential to develop complications exists if the patients are not adequately followed and prescribed supplements. Further prospective and randomized studies are required to evaluate the clinical significance of the nutritional deficiencies following bariatric surgery, and provide appropriate guidelines for supplementation.

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