Review Article

Clinical Approach to Nutritional Deficiencies After Sleeve Gastrectomy and Roux-en-Y Gastric Bypass: A Review Article

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ABSTRACT

Bariatric surgery is one of the most effective and durable treatments for severe obesity, with sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB) among the most commonly performed procedures worldwide. Both operations, however, substantially alter gastrointestinal anatomy and physiology, predisposing patients to a broad spectrum of macro- and micronutrient deficiencies that, if unrecognized, can progress to severe and sometimes irreversible complications such as anaemia, peripheral neuropathy, Wernicke's encephalopathy, metabolic bone disease, and protein-energy malnutrition. This narrative review synthesizes current evidence on the mechanisms, clinical presentation, and management of nutritional deficiencies following SG and RYGB. While SG is primarily restrictive and RYGB combines restriction with malabsorption, both procedures carry distinct risk profiles: RYGB is generally associated with a greater long-term risk of iron deficiency and may confer a higher risk of vitamin B12 deficiency than SG, while both procedures require lifelong surveillance for micronutrient abnormalities. Deficiencies in folate, thiamine, and protein occur across both procedures and warrant vigilant, procedure-tailored surveillance. The review outlines evidence-based preoperative and postoperative screening protocols, prophylactic supplementation regimens, and stepwise management strategies for established deficiencies, and presents a practical clinical algorithm to guide decision-making. Given the often insidious and non-specific nature of these deficiencies, lifelong biochemical monitoring, structured dietetic counselling, and a high index of clinical suspicion are essential to prevent long-term morbidity in bariatric surgery patients.

Keyword: Bariatric surgery; Sleeve gastrectomy; Roux-en-Y gastric bypass; Nutritional deficiencies; Micronutrient deficiency; Vitamin B12 deficiency.

Introduction

Obesity represents a major global public health challenge, with approximately 890 million adults living with obesity worldwide in 2022 and contributing to substantial morbidity, including type 2 diabetes mellitus, cardiovascular disease, obstructive sleep apnea, non-alcoholic fatty liver disease, and various malignancies [1]. Metabolic and bariatric surgery remains an established and highly effective treatment for severe obesity, providing substantial and durable weight loss together with clinically important metabolic benefits [2,3]. Contemporary pharmacological therapies, including glucagon-like peptide-1 receptor agonists and dual glucose-dependent insulinotropic polypeptide/glucagon-like peptide-1 receptor agonists, have also substantially

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expanded the available treatment options for Obesity [4]. Global metabolic and bariatric surgery volumes have increased substantially over recent decades. Recent data from the International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO) confirm that sleeve gastrectomy (SG) remains the most commonly performed bariatric procedure worldwide, with Roux-en-Y gastric bypass (RYGB) continuing to represent a major component of contemporary bariatric practice [5,6]. This widespread adoption of SG is attributed to its technical simplicity, favorable safety profile, and the absence of intestinal rerouting, whereas RYGB remains the benchmark against which novel procedures are evaluated [7]. While demonstrated to be highly effective, SG and RYGB fundamentally alter gastrointestinal anatomy and physiology, rendering patients susceptible to both macro- and micronutrient deficiencies. Such nutritional deficits often manifest insidiously; if left unaddressed, they may precipitate severe and irreversible clinical sequelae, including anemia, peripheral neuropathy, Wernicke's encephalopathy, metabolic bone disease, and protein-energy malnutrition [8]. Consequently, the proactive detection, prevention, and management of these complications are essential components of long-term postoperative bariatric care [8–10]. This narrative review outlines a systematic clinical framework for addressing nutritional deficiencies following SG and RYGB, detailing underlying mechanisms, clinical presentations, diagnostic screening, and therapeutic management strategies.

Methods

A narrative literature review was conducted using PubMed, Scopus, and Google Scholar to identify relevant studies on nutritional deficiencies following sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB). Search terms included combinations of "sleeve gastrectomy," "Roux-en-Y gastric bypass," "bariatric surgery," "nutritional deficiencies," "micronutrient deficiencies," "iron deficiency," "vitamin B12," "folate," "vitamin D," "calcium," "thiamine," and "protein malnutrition." English-language, peer-reviewed studies addressing the prevalence, mechanisms, clinical manifestations, screening, prevention, or management of nutritional deficiencies after SG or RYGB were considered eligible. Priority was given to recent systematic reviews, meta-analyses, randomized controlled trials, clinical practice guidelines, and large observational studies, while selected older landmark studies were retained when clinically relevant. Titles and abstracts were initially screened for relevance, followed by full-text review of potentially eligible articles. The selected literature included studies published between 2004 and 2025. Evidence was narratively appraised according to study design, recency, clinical relevance, and consistency with contemporary bariatric nutrition guidelines.

Results

1. Differences between Sleeve Gastrectomy and Roux-en-Y Gastric Bypass. 1.1 Restrictive vs Malabsorptive Mechanisms: SG functions primarily as a restrictive intervention involving the resection of approximately 80% of the gastric fundus and greater curvature, which creates a tubular gastric pouch with a capacity of 60–150 mL [11]. Although this volumetric reduction limits food intake, the anatomical continuity of the gastrointestinal tract remains preserved. Because the pylorus and the entire absorptive surface area of the small intestine remain intact, malabsorption is typically not considered an intrinsic feature of the procedure under standard clinical conditions [8]. Conversely, the RYGB integrates both restrictive and malabsorptive mechanisms. The procedure involves the creation of a small proximal gastric pouch (approximately 15–30 mL) and the anastomosis of the jejunal Roux limb directly to this pouch, effectively bypassing the gastric remnant, the duodenum, and the proximal jejunum. Digestive secretions traverse the biliopancreatic limb, which joins the Roux limb at the jejunojejunostomy, typically situated 75–150 cm distal to the gastrojejunostomy. This anatomical reconfiguration excludes key sites of nutrient absorption, specifically the duodenum and proximal jejunum, thereby limiting the uptake of essential micronutrients, including iron and calcium [8,10,12].

1.2 Clinical Implications: The distinct anatomical alterations inherent to SG and RYGB directly influence patients' nutritional profiles [8]. Long-term randomized evidence indicates a greater risk of iron deficiency after RYGB than after SG [13]. Meta-analytic evidence also suggests a higher risk of vitamin B12 deficiency after RYGB than after SG [14,15]. Nevertheless, SG also carries clinically relevant micronutrient risks because of reduced gastric volume, altered gastric acid secretion, and reduced dietary intake. Both procedures may therefore predispose patients to nutritional deficiencies and require procedure-specific supplementation and long-term biochemical surveillance. Consequently, a thorough comprehension of these distinct nutritional risks is critical for developing tailored surveillance and supplementation strategies [10,12]. 2. Mechanisms of Nutritional Deficiencies: 2.1 Reduced Dietary Intake. Restriction of gastric capacity fundamentally limits

the volume and diversity of food intake, particularly during the immediate postoperative phase. The frequent presentation of nausea, food intolerances, and early satiety further compromises caloric density and dietary variety [16]. Protein consumption is particularly precarious during the initial postoperative weeks, as the reliance on liquid and soft-food regimens frequently precludes the intake of protein-rich options [17,18]. Additionally, the adverse effects of reduced intake on micronutrient status are exacerbated by inconsistent adherence to recommended supplementation protocols [19]. 2.2 Malabsorption: While malabsorptive phenomena are inherent to RYGB due to the exclusion of the duodenum, this bypass compromises the uptake of micronutrients contingent upon gastric and duodenal acidity, such as iron, calcium, and zinc [8]. Furthermore, hypochlorhydria—resulting from either the resection of the gastric fundus in SG or the creation of a restrictive pouch in RYGB—inhibits the reduction of dietary ferric iron to its ferrous form, a critical step for non-haem iron absorption [8,20]. Additionally, the risk of fat-soluble vitamin malabsorption is augmented in surgical interventions characterized by extensive biliopancreatic limb diversion [15,21]. 2.3 Altered Gastrointestinal Anatomy and Physiology: Bariatric surgical interventions induce profound modifications to gastrointestinal transit, secretory processes, and endocrine signalling. Accelerated gastric emptying and attenuated intestinal transit times curtail the duration available for nutrient absorption [22]. Diminished intrinsic factor secretion—a common sequela of both SG and RYGB—compromises vitamin B12 assimilation, a process reliant upon the ileal uptake of the intrinsic factor–B12 complex [9,23]. Concurrently, alterations in bile acid metabolism, shifts in gut microbiota composition, and modified incretin secretion further modulate nutrient bioavailability [24,25]. These multifactorial pathophysiological changes underscore the necessity for vigilant, lifelong nutritional surveillance in all bariatric surgery patients [10]. 3. Common Nutritional Deficiencies: 3.1 Iron: Iron deficiency is one of the most clinically important long-term nutritional complications after bariatric surgery and may occur following both SG and RYGB [26]. Long-term randomized evidence indicates a greater risk after RYGB; at 10-year follow-up, iron deficiency defined by low ferritin was observed in 41% of patients after RYGB compared with 14% after SG [13]. This is primarily attributed to decreased gastric acidity, which is essential for converting dietary ferric iron into its absorbable ferrous state [20]. For RYGB patients, the additional bypass of the duodenum, a crucial site for iron absorption, substantially heightens this risk [27]. Individuals at elevated risk include menstruating women, those with pre-existing iron deficiency, and patients whose diets are low in haem iron [28]. The clinical manifestations of iron deficiency encompass microcytic hypochromic anaemia, fatigue, diminished exercise tolerance, koilonychia, and, in severe instances, Plummer-Vinson syndrome [29]. 3.2 Vitamin B12: Vitamin B12 deficiency may occur after both SG and RYGB; however, comparative evidence suggests a greater risk following RYGB. A meta-analysis of randomized controlled trials found that RYGB was associated with a higher risk of postoperative vitamin B12 deficiency than SG (RR 1.86, 95% CI 1.15–3.02) [14]. Reduced gastric acid secretion, altered intrinsic factor availability, and changes in gastrointestinal anatomy may impair vitamin B12 absorption after bariatric surgery [8,23]. Dietary B12 absorption requires binding to intrinsic factor in the stomach, followed by ileal uptake; this physiological process is hindered by the reduced gastric secretory function characteristic of these surgeries. Moreover, rapid intestinal transit and dysbiosis may further compromise B12 bioavailability [8]. In the absence of timely intervention, this deficiency can result in insidious neurological sequelae, such as peripheral neuropathy, cognitive dysfunction, and subacute combined degeneration of the spinal cord, which may become irreversible [15]. 3.3 Folate: Folate deficiency following bariatric surgery typically arises from a combination of inadequate dietary intake and, specifically in RYGB procedures, the bypass of the proximal jejunum, which serves as the primary site for folate absorption [23,26]. The reported prevalence of folate deficiency varies across studies and is influenced by dietary intake, supplementation adherence, procedure type, and duration of follow-up [15]. Clinical sequelae include megaloblastic anaemia, and, crucially for women of reproductive age, a heightened susceptibility to neural tube defects in future pregnancies [10]. 3.4 Calcium and Vitamin D: Calcium malabsorption represents a hallmark complication of RYGB, stemming from duodenal bypass, attenuated gastric acidity, and frequent comorbid vitamin D deficiency, which synergistically impair active intestinal calcium transport [21,30]. Vitamin D insufficiency is common among candidates for bariatric surgery and may persist after surgery [30]. Alterations in calcium and vitamin D metabolism can contribute to secondary

hyperparathyroidism, reduced bone mineral density, osteomalacia, and increased fracture risk, with several studies demonstrating more pronounced adverse bone-related changes after RYGB than after SG [30,31]. A meta-analysis found higher parathyroid hormone concentrations after RYGB than after SG at more than 2 years of follow-up (SMD 0.68, 95% CI 0.31–1.05) [32]. Acute manifestations of profound calcium deficiency encompass hypocalcaemic tetany, perioral paraesthesias, and QT interval prolongation [33]. 3.5 Thiamine (Vitamin B1): Thiamine deficiency is an underappreciated yet potentially catastrophic complication of bariatric surgery, occurring most frequently in patients with persistent postoperative emesis, protein-energy malnutrition, or excessive alcohol intake [34]. In the absence of adequate consumption, physiological thiamine reserves may be depleted within two to three weeks. The primary neurological presentation is Wernicke's encephalopathy, characterized by the clinical triad of ophthalmoplegia, ataxia, and confusion, which represents a neurological emergency [35]. Additional sequelae include peripheral neuropathy and cardiovascular beriberi. A recent meta-analysis estimated the pooled prevalence of thiamine deficiency at approximately 7% before bariatric surgery, with the highest postoperative prevalence occurring during the early postoperative period. Patients with persistent vomiting, rapid weight loss, poor nutritional intake, or other risk factors require particular clinical vigilance [34]. 3.6 Protein: Protein malnutrition is an uncommon but clinically important long-term complication after bariatric surgery, particularly following RYGB, and may present with muscle wasting, hypoalbuminaemia, oedema, impaired wound healing, and alopecia. This condition is primarily driven by restrictive anatomical changes that limit the consumption of protein-dense foods, combined with early satiety, and, specifically in RYGB, impaired protein digestion due to reduced pancreatic and biliary secretions [36]. Consequently, patients often struggle to achieve the recommended minimum protein intake of 60–80 g/day during the early postoperative period in the absence of structured dietary counselling and supplementation [17,18]. 3.7Other Micronutrient Deficiencies: Other clinically relevant micronutrient deficiencies after bariatric surgery include copper, zinc, selenium, and vitamins A, E, K, and B6 [22,37]. Copper and zinc deficiencies may occur after both SG and RYGB but are generally more common after procedures with a malabsorptive component [22,38]. Copper deficiency may present with anaemia, neutropenia, myelopathy, and peripheral neuropathy, whereas zinc deficiency may contribute to alopecia, impaired wound healing, and altered taste [22]. Selenium deficiency is less common but may be associated with cardiomyopathy, muscle weakness, and impaired immune function [39]. Fat-soluble vitamin deficiencies, particularly vitamins A, E, and K, are more frequently reported after procedures associated with greater malabsorption; clinical manifestations may include night blindness, neurological dysfunction, and coagulopathy, respectively [15]. Vitamin B6 deficiency is less frequently reported but may contribute to dermatitis, anaemia, and neurological symptoms [22]. These micronutrients should be assessed when clinical symptoms, dietary history, procedure type, or biochemical findings raise suspicion of deficiency [37]. 4. Clinical Presentation: The clinical presentation of post-bariatric nutritional deficiencies is frequently insidious, multifactorial, and non-specific, which necessitates a high index of clinical suspicion. Haematological sequelae are prominent, with anaemia—whether microcytic, macrocytic, or mixed in origin—often manifesting as fatigue, pallor, dyspnoea on exertion, and palpitations [26]. Furthermore, patients may present with neurological deficits that are potentially irreversible; these include distal symmetrical peripheral neuropathy, subacute combined degeneration of the spinal cord, and Wernicke's encephalopathy, the latter of which constitutes an acute neurological emergency [40]. Metabolic bone disease may remain clinically occult for an extended period before radiographic detection, often manifesting with diffuse bone pain, muscle weakness, or pathological fractures [31,32]. Secondary hyperparathyroidism, which is biochemically characterized by elevated parathyroid hormone levels accompanied by hypocalcaemia and low 25-hydroxyvitamin D, serves as a sensitive early indicator of calcium and vitamin D dysregulation [32]. Protein deficiency manifests through muscle wasting, peripheral oedema, and telogen effluvium—a form of diffuse hair loss frequently noted as an early postoperative complaint [36]. Given the considerable overlap in symptomatology across different nutritional deficiencies, a systematic biochemical approach is essential for accurate clinical evaluation. 5. Clinical Approach: 5.1 Screening Protocols: In accordance with contemporary American and British bariatric clinical practice guidelines, systematic nutritional screening is indicated both preoperatively and postoperatively [10,12]. Preoperative assessment

should include a complete blood count, ferritin, folate, vitamin B12, 25-hydroxyvitamin D, calcium, and parathyroid hormone, with additional biochemical and micronutrient testing guided by the planned procedure, clinical history, and individual risk factors [41]. Diagnostic interpretation should consider laboratory-specific reference ranges and clinical context. Iron deficiency is commonly defined by a serum ferritin concentration <15 µg/L, while additional indices such as transferrin saturation may support diagnosis when ferritin is equivocal. Vitamin B12 deficiency should be assessed using serum B12 concentrations, with methylmalonic acid measurement considered when serum B12 is borderline or discordant with clinical findings. Vitamin D deficiency is generally assessed using serum 25-hydroxyvitamin D, with concentrations <50 nmol/L (20 ng/mL) indicating deficiency; calcium and PTH should be interpreted concurrently when secondary hyperparathyroidism is suspected [10,12]. Routine preoperative thiamine measurement is not universally recommended; however, assessment should be considered in patients with relevant risk factors or symptoms [10]. Furthermore, bone mineral density screening is recommended for patients with an elevated risk of osteoporosis [12]. Postoperative biochemical surveillance is recommended at 3, 6, and 12 months during the initial year, followed by annual assessments for the duration of the patient's life [10,12]. Patients undergoing RYGB necessitate more rigorous monitoring, reflecting their elevated risk of malabsorption. Accelerated or more frequent diagnostic testing is indicated for individuals presenting with persistent emesis, suboptimal dietary adherence, gastrointestinal symptomatology, or those planning pregnancy [37,42]. Furthermore, any patient exhibiting symptoms of nutritional deficiency warrants immediate clinical evaluation, irrespective of scheduled follow-up intervals. 5.2 Prevention Strategies: Prophylactic micronutrient supplementation is standard for all post-bariatric patients, initiated in the immediate postoperative period [12]. This regimen typically includes a comprehensive multivitamin-mineral supplement, additional iron as required, calcium supplementation, vitamin D3 adjusted according to serum 25-hydroxyvitamin D concentrations, and routine vitamin B12 replacement according to procedure-specific guidelines [43]. Calcium citrate is the preferred formulation, as its absorption is independent of gastric acid, unlike that of calcium carbonate [10]. Additional iron supplementation should be individualized according to procedure, sex, baseline iron status, and biochemical monitoring, with higher therapeutic doses or intravenous iron considered for established deficiency when oral supplementation is inadequate or poorly tolerated [12,44]. Postoperative management necessitates structured dietary counselling facilitated by a registered dietitian. Comprehensive nutritional education is imperative, focusing on achieving protein targets, optimizing food consistency progression, ensuring strict adherence to supplementation protocols, and mitigating the intake of nutrient-poor, energy-dense foods [17,45,46]. Patients should be counselled regarding the need for lifelong micronutrient supplementation and the importance of long-term adherence [19]. For women planning pregnancy, appropriate preconception folic acid supplementation should be ensured. Higher-dose folic acid (5 mg/day) is recommended for women with additional risk factors such as obesity or diabetes, according to relevant guidelines [10]. The recommended postoperative doses and supplementation regimens for key nutrients after sleeve gastrectomy and Roux-en-Y gastric bypass are summarized in (Table 1) [10,12,46]. 5.3 Management of Established Deficiencies: The clinical management of established nutritional deficiencies necessitates a structured, stepwise approach that considers the severity of the deficiency, the patient's symptomatic profile, and their capacity for oral nutrient absorption. For iron deficiency anaemia, oral iron is generally used initially when absorption is expected to be adequate and treatment is tolerated; ferrous sulphate remains a commonly used formulation because of its availability and low cost [47]. Intravenous iron should be considered when oral iron is not tolerated, when ferritin fails to improve despite an adequate oral trial, or when gastrointestinal anatomy is expected to impair oral iron absorption [48]. Vitamin B12 deficiency should be treated promptly, with the route of replacement guided by the severity of deficiency, clinical manifestations, and local guideline recommendations; intramuscular hydroxocobalamin is particularly appropriate when neurological involvement is present [49]. Vitamin D deficiency typically necessitates initial repletion with high-dose oral cholecalciferol, subsequently followed by daily maintenance doses that are titrated according to serum 25-hydroxyvitamin D levels [30]. Thiamine deficiency with neurological manifestations constitutes a medical emergency, and treatment should be initiated immediately without waiting for laboratory confirmation. In patients with suspected

Table 1: Suggested postoperative supplementation regimens after sleeve gastrectomy and Roux-en-Y gastric bypass.

Nutrient	Sleeve Gastrectomy (SG)	Roux-en-Y Gastric Bypass (RYGB)
Multivitamin-mineral	Complete bariatric multivitamin-mineral supplement daily.	Complete bariatric multivitamin-mineral supplement daily; closer biochemical surveillance is generally required.
Iron	45-60 mg elemental iron/day, especially in menstruating women or patients at increased risk; adjust to biochemical monitoring and tolerance.	45-60 mg elemental iron/day; adjust to biochemical monitoring and tolerance, with particular attention to long-term iron status.
Vitamin B12	350-500 mcg orally/sublingually daily, or 1,000 mcg IM monthly; route may follow local protocol.	350-500 mcg orally/sublingually daily, or 1,000 mcg IM monthly; closer surveillance may be appropriate because of altered absorption.
Calcium + Vitamin D3	Calcium 1,200-1,500 mg/day in divided doses. Vitamin D3 about 3,000 IU/day, titrated to serum 25(OH)D.	Calcium 1,200-1,500 mg/day in divided doses. Vitamin D3 about 3,000 IU/day, titrated to serum 25(OH)D and PTH response.
Thiamine (B1)	At least 12 mg/day; 50-100 mg/day may be used in higher-risk patients, particularly with poor intake or vomiting.	At least 12 mg/day; 50-100 mg/day may be used in higher-risk patients, particularly with poor intake or vomiting.
Folic acid	400-800 mcg/day; 800-1,000 mcg/day in women of childbearing age. Higher-dose folic acid is reserved for specific pregnancy risk factors according to guideline recommendations.	400-800 mcg/day; 800-1,000 mcg/day in women of childbearing age. Higher-dose folic acid is reserved for specific pregnancy risk factors according to guideline recommendations.
Protein	At least 60 g/day or about 1.0-1.5 g/kg ideal body weight/day; individualize to intake, tolerance, and nutritional status.	At least 60 g/day or about 1.0-1.5 g/kg ideal body weight/day; individualize to intake, tolerance, and nutritional status.
Zinc + Copper	Zinc 8-11 mg/day and copper 1 mg/day as part of routine supplementation.	Zinc 8-22 mg/day and copper 2 mg/day as part of routine supplementation.
Other micronutrients	Vitamin A 5,000-10,000 IU/day, vitamin E 15 mg/day, and vitamin K 90-120 mcg/day. Vitamin B6 and selenium should be supplied within a complete multivitamin; add extra supplementation only when clinically indicated.	Vitamin A 5,000-10,000 IU/day, vitamin E 15 mg/day, and vitamin K 90-120 mcg/day. Vitamin B6 and selenium should be supplied within a complete multivitamin; assess selenium more closely when clinically indicated.

Table 2: The mechanisms, clinical manifestations, management, and procedure-specific risks of each deficiency after sleeve gastrectomy and Roux-en-Y gastric bypass.

Nutrient	Mechanism and key clinical manifestations	Management	Procedure risk / quantitative evidence
Iron	Reduced intake and gastric acidity; duodenal/proximal jejunal bypass after RYGB. May cause fatigue, reduced exercise tolerance and microcytic anaemia.	Oral iron with biochemical monitoring; use IV iron when oral therapy is not tolerated, ineffective, or absorption is impaired.	Higher long-term risk after RYGB. At 10 years, low ferritin was reported in 41% after RYGB versus 14% after SG [13].
Vitamin B12	Reduced gastric acid and intrinsic-factor availability with altered gastrointestinal anatomy. Deficiency may cause macrocytic anaemia, peripheral neuropathy and subacute combined degeneration.	High-dose oral or IM replacement according to severity; IM treatment is particularly appropriate when neurological involvement is present.	Both procedures are affected but randomized-trial meta-analysis showed higher postoperative risk after RYGB than SG (RR 1.86, 95% CI 1.15-3.02) [14].
Folate	Reduced intake and altered intestinal absorption. Deficiency may cause megaloblastic anaemia	Oral folic acid according to biochemical status; ensure appropriate preconception and pregnancy supplementation	Comparative randomized evidence found no significant difference in folate deficiency between RYGB and

Nutrient	Mechanism and key clinical manifestations	Management	Procedure risk / quantitative evidence
	and increases fetal neural-tube-defect risk during pregnancy.	according to guideline-defined risk.	SG; risk is strongly influenced by intake and supplement adherence [14].
Vitamin D / Calcium	Pre-existing vitamin D insufficiency, reduced intake, altered absorption and duodenal exclusion after RYGB may impair calcium-vitamin D homeostasis. Consequences include secondary hyperparathyroidism, reduced bone mineral density, osteomalacia and fracture.	Vitamin D3 titrated to serum 25(OH)D; calcium in divided doses with correction of vitamin D deficiency and ongoing biochemical monitoring.	Long-term pooled vitamin D deficiency after bariatric surgery was 35.8% (Chen et al., 2024). PTH was higher after RYGB than SG at >2 years (SMD 0.68, 95% CI 0.31-1.05) [32].
Thiamine (B1)	Risk increases with persistent vomiting, rapid weight loss and poor intake. Deficiency may cause Wernicke encephalopathy, peripheral neuropathy and beriberi.	Treat immediately when deficiency is suspected; suspected Wernicke encephalopathy requires immediate high-dose IV thiamine.	Pooled prevalence was 7% preoperatively, rising to 19% at 3 months, then 9% at 6 months and 6% at 1 year after bariatric surgery [34].
Protein	Inadequate intake, food intolerance, persistent vomiting and reduced digestion/absorption after procedures with a malabsorptive component. May cause muscle wasting, hypoalbuminaemia, oedema and impaired wound healing.	Individualized dietary intervention and oral protein supplementation; specialized enteral or other nutrition support may be required in severe cases.	Uncommon after standard procedures but more strongly associated with RYGB. A scoping review reported a median protein-malnutrition incidence of 1.7% after RYGB (range 0-8.9%) [36].
Zinc / Copper	Reduced intake and absorption. Zinc deficiency may cause alopecia, impaired wound healing and dysgeusia; copper deficiency may cause anaemia, neutropenia, myelopathy and neuropathy.	Replace documented deficiency and maintain an appropriate zinc-to-copper balance; monitor biochemistry in symptomatic or higher-risk patients.	Pooled postoperative prevalence was 18.3% for zinc and 14.4% for copper; subgroup analysis showed fewer deficiencies after SG than RYGB [38].
Selenium	Low intake and altered absorption may contribute to muscle weakness, impaired immune function and, rarely, cardiomyopathy.	Assess and replace when deficiency is documented or strongly suspected; routine additional supplementation beyond a complete bariatric multivitamin should be individualized.	Meta-analysis found no significant overall change in serum selenium after bariatric surgery, but subgroup analysis showed fewer deficiencies after SG than RYGB [38].
Vitamins A, E and K	Fat-soluble vitamin deficiency is associated with reduced intake and greater malabsorption. Clinical effects include night blindness (A), neurological dysfunction (E) and coagulopathy (K).	Assess when symptoms or malabsorptive risk is present and replace documented deficiencies according to biochemical response and clinical guidance.	Long-term pooled prevalence after bariatric surgery was 13.4% for vitamin A, 16.5% for vitamin E and 9.6% for vitamin K [15].
Vitamin B6	Reduced intake and altered postoperative dietary patterns may contribute to deficiency, which can present with dermatitis, anaemia and neurological symptoms.	Assess when clinically suspected and replace documented deficiency; avoid unnecessary high-dose chronic supplementation.	Reported after bariatric surgery, but current comparative evidence is limited and insufficient for firm SG-versus-RYGB estimates [23].

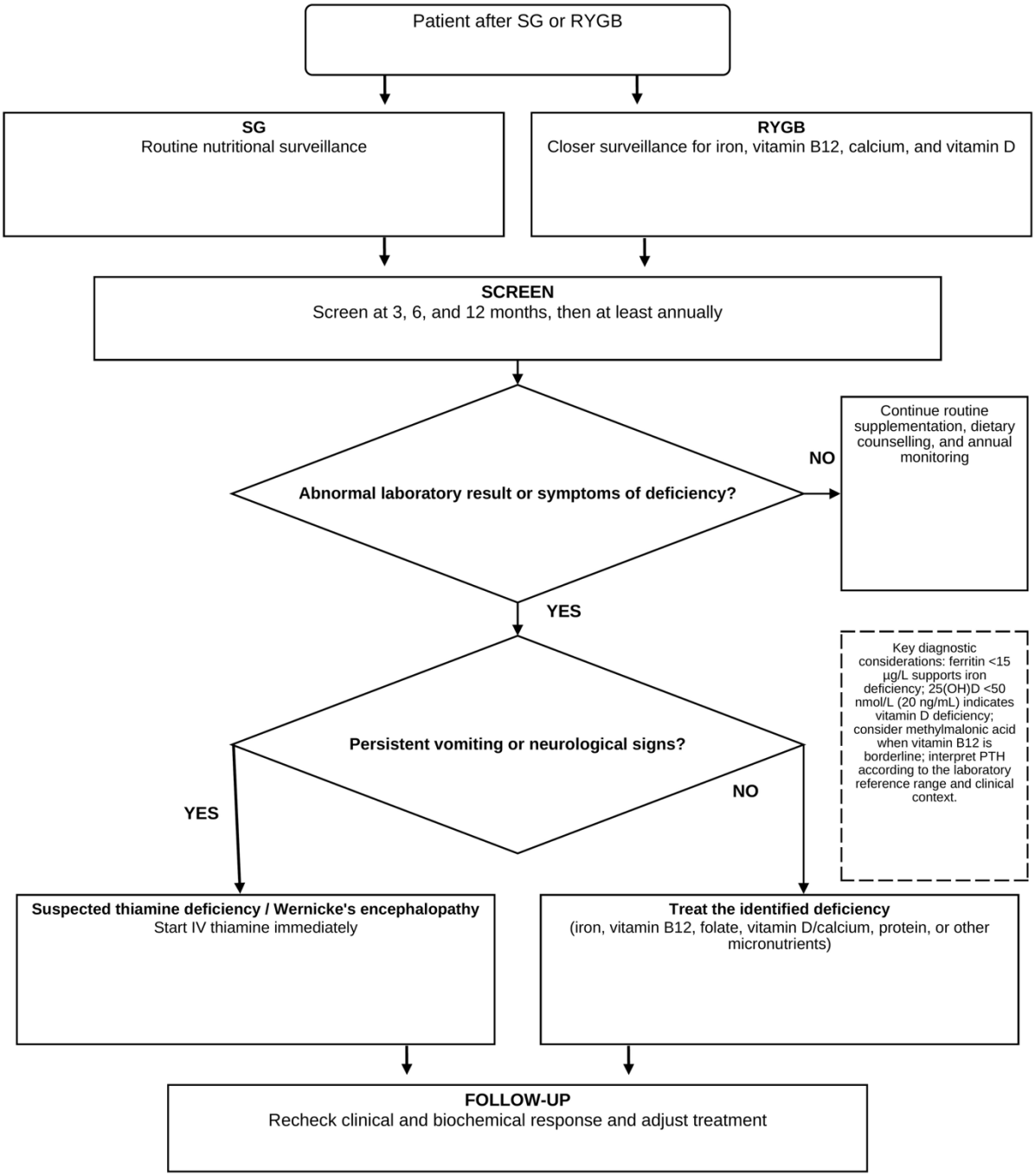


Figure 1:Clinical algorithm for suspected nutritional deficiency after SG and RYGB.

Wernicke's encephalopathy, high-dose intravenous thiamine should be administered immediately at 500 mg three times daily for 3–5 days, followed by 250 mg intravenously daily for an additional 3–5 days or until clinical improvement, with subsequent oral supplementation as clinically indicated [35,50]. The management of protein malnutrition demands substantial dietary intervention, including oral protein supplementation to achieve daily targets of 60–80 g. In severe instances, temporary enteral nutrition may become necessary [18]. 6. Comparison of Nutritional Outcomes Between Sleeve Gastrectomy and RYGB: SG and RYGB have distinct long-term nutritional risk profiles, although RYGB is generally associated with a greater risk of selected micronutrient deficiencies and therefore may require more intensive nutritional surveillance [13,15]. Long-term evidence indicates that iron deficiency is more frequent after RYGB than after SG, although the magnitude of this difference varies according to follow-up duration, supplementation adherence, and the definition of deficiency [28]. Furthermore, deficiencies in calcium and vitamin D are markedly more pronounced after RYGB due to duodenal exclusion, resulting in higher rates of secondary hyperparathyroidism and consequential bone loss [31,32]. Additionally, fat-soluble vitamin deficiencies are observed more frequently post-RYGB, particularly in configurations involving extended biliopancreatic limbs [21]. Nonetheless, SG is associated with a distinct and often underestimated nutritional risk profile. Although SG may impair vitamin B12 absorption through reduced gastric acid secretion and altered intrinsic factor availability, comparative evidence suggests that vitamin B12 deficiency may be more frequent after RYGB. However, long-term randomized data found no significant difference in vitamin B12 deficiency between SG and RYGB at 10 years [13]. Moreover, thiamine deficiency has been documented across both bariatric interventions, particularly in patients experiencing protracted postoperative emesis; consequently, it should not be viewed exclusively as a complication of malabsorptive procedures [34]. Protein malnutrition is uncommon after standard bariatric procedures but is more strongly associated with RYGB and other procedures with a malabsorptive component; inadequate protein intake, persistent vomiting, and dysphagia are important risk factors [36]. These findings underscore the imperative for systematic nutritional surveillance following both procedures, incorporating tailored, procedure-specific supplementation protocols [10]. The mechanisms, clinical manifestations, management, and procedure-specific risks of each deficiency are summarized (Table 2). 7. Clinical Algorithm for Managing Suspected Nutritional Deficiency: This algorithm serves as a clinical decision-making framework and necessitates tailoring to specific patient profiles, local institutional mandates, and prevailing clinical practice guidelines [10,12,47]. The proposed clinical decision-making framework is illustrated (Figure 1). 8. Limitations: This narrative review has several limitations. The included studies varied in design, follow-up duration, definitions of nutritional deficiency, supplementation protocols, and patient characteristics, which limits direct comparison across studies. The review was restricted to English-language literature and did not employ a formal systematic-review or meta-analytic methodology. In addition, evidence for some less frequently studied micronutrients remains limited, and long-term comparative data between SG and RYGB are not uniformly available.

Conclusion

Nutritional deficiencies after SG and RYGB are common but preventable. RYGB carries a greater long-term risk of iron deficiency and may confer a higher risk of vitamin B12 deficiency, while both procedures can affect vitamin D, folate, thiamine, and protein status. Effective management requires preoperative assessment, regular biochemical monitoring, evidence-based supplementation, and dietary counselling. Because neurological and metabolic complications may develop insidiously, lifelong multidisciplinary follow-up is essential. Future research should refine procedure-specific supplementation and monitoring strategies.

Conflict of Interest

None

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None

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