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Bariatric Surgery and Postoperative Endocrine-Metabolic Dysfunction: Main Clinical Syndromes and Complications

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Abstract

Bariatric surgery is a highly effective intervention for the treatment of severe obesity and its cardiometabolic comorbidities; however, its effects are not limited to weight loss. The anatomical and functional reconfiguration of the gastrointestinal tract modifies nutrient transit, enteroendocrine secretion, macro- and micronutrient absorption, body composition, bone metabolism, and several hormonal axes. This review analyzes the main endocrine-metabolic complications following bariatric surgery, with emphasis on their pathophysiology, clinical expression, risk factors, and follow-up strategies. The most relevant entities include post-bariatric hypoglycemia, dumping syndrome, protein-energy malnutrition, sarcopenia, micronutrient deficiencies, metabolic bone disease, gonadal and thyroid alterations, weight regain, and recurrence of metabolic comorbidities. These complications may appear early or late, even years after initial weight-loss success, and require clinical, biochemical, nutritional, muscular, skeletal, metabolic, and psychosocial surveillance. The optimal approach requires a longitudinal, multidisciplinary model stratified by surgical technique, aimed at preventing irreversible damage, preserving the metabolic benefits of surgery, and promptly identifying the need for nutritional, pharmacological, endoscopic, or revisional surgical intervention.

Keywords

Bariatric Surgery, Metabolic Surgery, Endocrine-Metabolic Complications, Post-Bariatric Hypoglycemia, Nutritional Deficiencies, Sarcopenia, Metabolic Bone Disease, Weight Regain

1. Introduction

Obesity is a chronic, progressive, and multifactorial disease whose epidemiological burden has acquired a global public health dimension. In the pooled analysis by the NCD Risk Factor Collaboration, based on 3663 population-based studies and 222 million participants from 200 countries and territories, the age-standardized global prevalence of obesity in adults increased between 1990 and 2022 from 8.8% to 18.5% in women and from 4.8% to 14.0% in men; in absolute terms, an estimated 504 million adult women and 374 million adult men were living with obesity in 2022. This transition has also extended to younger age groups, with obesity among school-aged children and adolescents increasing from 1.7% to 6.9% in girls and from 2.1% to 9.3% in boys during the same period [1]. This population-wide expansion anticipates a sustained increase in candidates for highly effective therapeutic interventions, particularly among individuals with severe obesity, established metabolic disease, or failure of conservative strategies; indeed, it is projected that by 2035 more than 4 billion people will be living with overweight or obesity, while obesity itself may increase from 14% to 24% of the global population [2].

In this context, bariatric surgery has become one of the most effective interventions for achieving clinically significant and sustained weight loss, while also improving cardiometabolic comorbidities such as type 2 diabetes mellitus, arterial hypertension, dyslipidemia, metabolic liver disease, and obstructive sleep apnea. Its global expansion reflects this efficacy: the worldwide survey of the International Federation for the Surgery of Obesity and Metabolic Disorders reported 507,806 surgical and endoluminal procedures in 2020 and 598,834 in 2021, with sleeve gastrectomy being the most frequently performed technique worldwide [3]. In parallel, the 2022 ASMBS/IFSO guidelines redefined the modern indications for metabolic-bariatric surgery, recommending it in individuals with BMI > 35 kg/m^2 regardless of the presence or severity of comorbidities, and considering it in patients with metabolic disease and BMI between 30 and 34.9 kg/m^2; these guidelines also describe a low contemporary perioperative mortality, between 0.03% and 0.2%, which has contributed to its acceptance as a safe and durable therapeutic strategy in selected patients [4].

However, bariatric surgery should not be interpreted solely as a restrictive or mechanical intervention aimed at reducing caloric intake. Its effects depend on an anatomical and functional reorganization of the gastrointestinal tract that modifies nutrient transit, incretin secretion, insulin sensitivity, bile acid availability, the

intestinal microbiome, macro- and micronutrient absorption, and multiple hormonal axes. This reprogramming explains the metabolic superiority of procedures such as Roux-en-Y gastric bypass and sleeve gastrectomy over conventional medical treatment in numerous clinical scenarios; nevertheless, it also creates the pathophysiological conditions for the development of specific endocrine-metabolic complications that may manifest months or years after the intervention [5]. In this regard, the consequences of surgery are not limited to glucose metabolism: they may compromise gonadal and thyroid function, as well as bone health, body composition, and overall nutritional status [6].

The clinical spectrum of postoperative endocrine-metabolic dysfunction includes heterogeneous entities with overlapping mechanisms and variable presentation according to the surgical technique used. Among the most relevant complications are post-bariatric hypoglycemia, dumping syndrome, protein-energy malnutrition, excessive loss of lean mass, sarcopenia, deficiencies of iron, vitamin B12, folate, thiamine, vitamin D, calcium, and other micronutrients, secondary hyperparathyroidism, metabolic bone disease, weight regain, and recurrence of metabolic comorbidities. Some of these alterations are a direct consequence of malabsorption, reduced gastric acidity, or duodenojejunal exclusion; others arise from exaggerated hormonal responses, changes in intake, poor adherence to supplementation, pre-existing nutritional vulnerability, or insufficient clinical follow-up. Therefore, although bariatric surgery offers substantial metabolic benefits, it also entails risks related to malabsorption, hormonal alteration, hypoglycemia, and osteoporosis, which require preoperative assessment and regular long-term surveillance [7].

Prevention of these complications requires shifting the focus from isolated weight-loss success toward longitudinal endocrine-nutritional surveillance. The BOMSS guidelines emphasize that many patients reach surgery with low vitamin and mineral concentrations, and that all bariatric procedures can induce clinically significant micronutrient deficiencies; therefore, preoperative preparation, postoperative biochemical monitoring, supplementation, and early correction of deficiencies are essential components of bariatric care [8]. Accordingly, follow-up after surgery should integrate metabolic, nutritional, bone, muscle, and hormonal assessment, using an individualized strategy according to surgical technique, time elapsed since the intervention, symptoms, comorbidities, risk of malabsorption, and therapeutic adherence.

Consequently, the present review aims to analyze the main endocrine-metabolic complications following bariatric surgery, with emphasis on their pathophysiological mechanisms, clinical expression, risk factors, and follow-up needs. The purpose is to provide an integrated view of postoperative clinical syndromes that may compromise the initial metabolic benefit, impair quality of life, or generate preventable morbidity and mortality if not identified and treated in a timely manner. The evidence was interpreted according to its methodological strength: randomized trials and meta-analyses were prioritized for efficacy and comparative

therapeutic outcomes, prospective studies and cohort studies for incidence and longitudinal risk, clinical guidelines and consensus statements for diagnostic and follow-up recommendations, and case reports mainly for rare, severe, or low-frequency complications.

2. Physiological Basis of Postoperative Endocrine-Metabolic Dysfunction

Bariatric surgery modifies systemic metabolism because it transforms gastrointestinal anatomy and, with it, the way nutrients are received, processed, absorbed, and signaled. Roux-en-Y gastric bypass creates a small gastric pouch and diverts alimentary transit toward the jejunum, excluding much of the stomach, the duodenum, and the proximal jejunum; in contrast, sleeve gastrectomy longitudinally resects the stomach, preserves intestinal continuity, and creates a lower-capacity gastric tube. These differences are not merely surgical but physiological, as each procedure reorganizes nutrient flow, intestinal mucosal exposure, enteroendocrine secretion, bile acid circulation, and microbiota composition in distinct ways. Therefore, postoperative endocrine-metabolic dysfunction should not be understood as a nonspecific consequence of weight loss, but rather as the clinical expression of profound, dynamic gastrointestinal adaptations that depend on the type of procedure performed [9].

One of the most relevant early changes is the alteration of gastric emptying and intestinal transit. After gastric bypass, and to a lesser extent after sleeve gastrectomy, accelerated delivery of food to the small intestine promotes early glucose appearance in the peripheral circulation, intensifies GLP-1 secretion, and stimulates a more pronounced postprandial insulin response. In parallel, intestinal adaptation after bypass may include delayed distal transit, epithelial hypertrophy, increased expression of glucose transporters, expansion of hormone-secreting cells, and modification of the microbiome. This reorganization contributes to improved hepatic, muscular, and adipose insulin sensitivity, but also creates the physiological substrate for postprandial syndromes such as dumping, reactive hypoglycemia, and postoperative glycemic variability, particularly when rapid carbohydrate absorption and the enteroinsular response become disproportionately coupled [10].

Enteroendocrine signaling occupies a central position in this transition between metabolic benefit and clinical risk. Early improvement in glucose tolerance after gastric bypass depends on the initial energy restriction and weight loss, which first improve hepatic insulin sensitivity and later peripheral insulin sensitivity; however, it also depends on increased postprandial insulin secretion induced by exaggerated GLP-1 responses. In turn, reduced appetite and food intake appear to be mediated by increased secretion of anorexigenic intestinal hormones such as GLP-1 and peptide YY, stimulated by accelerated nutrient exposure in the small intestine. The magnitude of these responses is not uniform across procedures: in a randomized trial comparing Roux-en-Y gastric bypass and one-anastomosis gastric

bypass, both procedures produced approximately 25% weight loss and metabolic improvement at one year, but the early postprandial GLP-1 increase was greater after Roux-en-Y gastric bypass, with an early area under the curve 31% higher at six months and 25% higher at twelve months, as well as GLP-1 peaks approximately 32% higher at both follow-up points. These findings reinforce that small anatomical differences may translate into distinct hormonal and glycemic responses, with potential implications for procedure selection and surveillance of postprandial symptoms [11] [12].

Beyond the incretin axis, bile acids and the gut microbiota act as a second layer of metabolic signaling. After metabolic surgery, bile acids are no longer relevant only because of their digestive function; they also behave as endocrine molecules capable of modulating receptors such as FXR and TGR5, with effects on insulin sensitivity, lipid metabolism, GLP-1 secretion, hepatic homeostasis, and energy expenditure. The gut microbiome is also remodeled after gastric bypass and sleeve gastrectomy, modifying bacterial metabolites, interactions with bile acids, and low-grade inflammatory signals. This bile acid-microbiota-metabolic receptor network helps explain why surgery may induce glucolipid benefits that exceed weight loss, but also why some patients develop digestive intolerance, absorptive alterations, changes in colonic fermentation, or persistent metabolic variability [13].

Macronutrient malabsorption must be interpreted precisely and not as a homogeneous phenomenon. Although it was historically assumed that part of the bariatric effect depended on reduced caloric absorption, contemporary evidence suggests that fat, protein, and carbohydrate malabsorption varies according to the procedure and tends to be limited in the most used techniques. In a study using isotope technology, macronutrient malabsorption was slightly greater after Roux-en-Y gastric bypass, but not after sleeve gastrectomy, compared with controls; moreover, colonic protein fermentation increased after both bypass and sleeve gastrectomy. This suggests that macronutrient malabsorption probably does not, by itself, explain weight loss or universally compromise nutritional status, but it may modify substrate availability for the microbiome, colonic metabolite production, and the risk of malnutrition in vulnerable patients with insufficient intake or low functional reserve [14].

By contrast, micronutrient deficiencies represent a more persistent and clinically relevant consequence of gastrointestinal reconfiguration. Reduced gastric acidity, lower intake, duodenojejunal exclusion, altered contact with bile and pancreatic enzymes, food intolerance, and poor adherence to supplementation may affect the absorption of iron, vitamin B12, folate, calcium, vitamin D, and other micronutrients. In a 10-year prospective observation, the prevalence of iron deficiency increased from 9.0% to 18.7%, folic acid deficiency from 1.3% to 11.6%, and vitamin B12 deficiency from 7.1% to 17.4%, with differences in iron metabolism between sleeve gastrectomy and gastric bypass. These data show that post-bariatric endocrine-metabolic dysfunction may emerge long after initial weight-

loss success and justify prolonged biochemical follow-up, even in clinically stable patients [15].

3. Post-Bariatric Hypoglycemia and Dumping Syndrome

Dumping syndrome, or accelerated gastric emptying syndrome, is a frequent complication of gastric, esophageal, and bariatric surgery, caused by the rapid arrival of incompletely digested nutrients into the small intestine. To reduce diagnostic overlap, three scenarios should be distinguished according to chronology, symptom pattern, and the need for biochemical confirmation. Early dumping usually occurs within the first hour after eating and is predominantly manifested by gastrointestinal and vasomotor symptoms, such as abdominal pain, bloating, borborygmi, nausea, diarrhea, flushing, palpitations, sweating, tachycardia, hypotension, fatigue, and, less frequently, syncope, without requiring documentation of hypoglycemia for its clinical diagnosis. Late dumping usually occurs between one and three hours after carbohydrate-rich meals and reflects an exaggerated enteroinsular response, mediated in part by incretins, which may culminate in symptomatic hypoglycemia. Although these terms have been used in a partially overlapping manner in the literature, they should not be employed as equivalents without considering timing, symptoms, and biochemical confirmation [16].

Post-bariatric hypoglycemia should be reserved for postprandial hyperinsulinemic hypoglycemia that typically appears two to four hours after eating in patients who have undergone bariatric surgery, more often when they have already been discharged from specialized surgical follow-up. Its clinical definition requires documentation of low glucose values associated with compatible symptoms and resolution after correction of hypoglycemia, that is, fulfillment of Whipple's triad. This delimitation allows it to be distinguished from early dumping, late dumping without confirmed hypoglycemia, nonspecific postprandial symptoms, pharmacological hypoglycemia, and non-surgical causes such as insulinoma, adrenal insufficiency, severe malnutrition, or critical illness. The appearance of symptoms during fasting, at night, or without a clear temporal relationship with food intake should prompt investigation for alternative diagnoses [17].

The frequency of these syndromes is variable and depends on the procedure, the time elapsed since surgery, and the diagnostic instrument used. In a Swedish cohort evaluated using the Dumping Severity Scale, highly suggestive symptoms of dumping after Roux-en-Y gastric bypass increased from 4.9% before surgery to 26.3% at five years, whereas symptoms compatible with post-bariatric hypoglycemia increased from 1.4% to 19.3%; in contrast, after sleeve gastrectomy, no significant increase in dumping or hypoglycemic symptoms was observed, and persistent type 2 diabetes appeared to behave as a protective factor against symptomatic hypoglycemia. Complementarily, a questionnaire-based study reported that 34.2% of patients undergoing gastric bypass or sleeve gastrectomy had a high suspicion of postprandial hypoglycemic symptoms, with greater likelihood among women, patients without diabetes, individuals with longer time since surgery, and

subjects with preoperative hypoglycemic symptoms [18] [19].

The discrepancy between symptomatic and biochemical prevalence reveals that post-bariatric hypoglycemia does not always behave as a clinically evident syndrome. In a random sample evaluated four years after primary gastric bypass using a mixed-meal test, 48% of patients developed glucose levels below 3.3 mmol/L, but all events were asymptomatic, suggesting a relevant proportion of hypoglycemia unawareness. In a naturalistic assessment using continuous glucose monitoring and ecological momentary self-reporting, approximately 66% of patients presented at least one possible daytime hypoglycemic event one year after gastric bypass, although the total number of reported symptoms did not correlate robustly with sensor-detected events. These findings require post-bariatric hypoglycemia to be considered as a spectrum that includes subclinical glycemic excursions, mild autonomic symptoms, disabling neuroglycopenic episodes, and hypoglycemia unawareness, rather than as an entity with a single and stable prevalence [20] [21].

From a pathophysiological standpoint, the central sequence combines rapid emptying, accelerated carbohydrate absorption, an early glycemic peak, exaggerated GLP-1 secretion, beta-cell hyperresponsiveness, and a late decline in plasma glucose. However, the model is not limited to incretin excess, because increased insulin sensitivity after weight loss, inappropriately sustained insulin secretion, altered glucagon counterregulation, hypoglycemia unawareness, changes in body composition, bile acid modifications, and intestinal adaptation may also participate. The clinical presentation reflects this complexity: autonomic symptoms include tremor, palpitations, anxiety, sweating, and hunger, whereas neuroglycopenic symptoms include fatigue, weakness, blurred vision, difficulty speaking, confusion, behavioral changes, loss of consciousness, or seizures. Therefore, the clinical history should specify timing, relationship with simple carbohydrates, presence of neuroglycopenia, need for assistance, occupational impact, driving risk, and occurrence of symptoms during fasting or at night, since these latter patterns should raise suspicion of alternative diagnoses [22].

Treatment should be stepwise and begin with a structured nutritional strategy aimed at reducing the rate of carbohydrate absorption and attenuating the axis of glycemic peak-hyperinsulinism-late hypoglycemia. Initial recommendations include small and frequent meals, restriction of simple sugars, selection of low-glycemic-index carbohydrates, adequate protein intake, incorporation of soluble fiber, separation of liquids from meals, and education to recognize high-risk episodes. When dietary management is insufficient, acarbose may reduce rapid carbohydrate absorption and attenuate postprandial peaks; in persistent cases, somatostatin analogues, diazoxide, calcium channel blockers, SGLT2 inhibitors, GLP-1 analogues in selected scenarios, or emerging strategies targeting the GLP-1 receptor may be considered, although pharmacological evidence remains limited and requires specialized management. In patients with severe refractory hypoglycemia, the approach may escalate to enteral feeding through an alternative route, anatomical revision, pouch reduction, bypass reversal, or individualized surgical

conversion, whereas pancreatectomy has lost prominence because of inconsistent efficacy and the risk of insulin-dependent diabetes. Consequently, contemporary management should integrate endocrinology, clinical nutrition, bariatric surgery, glycemic education, and prolonged follow-up, with the aim of preventing neuroglycopenia without unnecessarily sacrificing the metabolic benefits of surgery [23].

4. Protein-Energy Malnutrition, Sarcopenia, and Loss of Lean Mass

Weight loss after bariatric surgery should not be interpreted exclusively as a reduction in adipose tissue, because a clinically relevant proportion of weight reduction involves lean compartments of high metabolic and functional importance. In this context, it is useful to distinguish between fat-free mass, lean mass, skeletal muscle mass, sarcopenia, and sarcopenic obesity, since not every reduction in lean mass is equivalent to muscle disease, and not every successful weight-loss outcome implies preservation of function. Sarcopenic obesity is defined as the coexistence of excess adiposity with low muscle mass and function, and its diagnosis requires integration of clinical screening, functional assessment, and body composition measurement. This conceptual precision is particularly important in the post-bariatric patient, who may retain residual adiposity while simultaneously developing muscle loss, reduced strength, lower protein reserve, and impaired physical performance. Therefore, surgical outcome assessment should shift from absolute body weight toward the quality of weight loss, incorporating muscle as an endocrine-metabolic organ rather than as a simple anatomical compartment [24].

The magnitude of lean tissue loss after bariatric surgery is sufficiently consistent to be considered an expected physiological phenomenon, although potentially pathological when it exceeds the patient's adaptive capacity. In a meta-analysis of 59 studies, pooled loss at one year was 8.13 kg for lean mass, 8.23 kg for fat-free mass, and 3.18 kg for skeletal muscle mass, with approximately 55% of lean mass loss occurring during the first three months after surgery. This chronology is critical because the phase of greatest catabolism coincides with intense energy restriction, reduced protein intake, rapid mobilization of reserves, and postoperative hormonal adaptation. Consequently, the early follow-up window should not be limited to monitoring weight loss and food tolerance, but should identify signs of disproportionate muscle loss, functional decline, and nutritional risk before deterioration becomes established [25].

The risk of post-bariatric sarcopenia is not uniformly distributed. In a prospective cohort of 184 patients with severe obesity undergoing sleeve gastrectomy, the prevalence of sarcopenia increased from 8% at the time of surgery to 32% at one year of follow-up; moreover, male sex and baseline muscle mass parameters measured by computed tomography, such as skeletal muscle area and skeletal muscle index, were significantly associated with the development of postoperative sarco-

penia. These data show that sarcopenia is not a complication confined to older adults or to classically malabsorptive procedures, but rather a body composition vulnerability that may emerge even after restrictive techniques when low baseline muscle reserve, accelerated weight loss, or absence of intensive nutritional and functional intervention are present. Early identification of these phenotypes allows selection of patients who require reinforced follow-up, specific dietary intervention, and exercise prescription before loss of mass translates into clinical frailty [26].

Protein-energy malnutrition represents the clinically severe end of this continuum and must be differentiated from the expected loss of fat-free mass. Although it is observed more frequently after highly malabsorptive procedures, it may also appear after Roux-en-Y gastric bypass and even sleeve gastrectomy, indicating that its pathophysiology does not depend solely on the length of excluded intestine. In a scoping review that included 18 studies and 3015 patients undergoing gastric bypass, the median incidence of protein malnutrition was 1.7%, with a range from 0% to 8.9%, and diagnosis occurred between 12 and 120 months after surgery. The most frequent cause was insufficient oral protein intake, although persistent hypoalbuminemia requires ruling out intractable vomiting, dysphagia, food intolerance, small intestinal bacterial overgrowth, protein-losing enteropathy, anatomical complications, liver disease, chronic inflammation, and intestinal limb lengths with a greater malabsorptive component. Mild hypoalbuminemia may remain subclinical, but severe deficiency, especially with albumin below 25 g/L, may be associated with edema, anemia, Kwashiorkor-like injury, liver failure, and the need for advanced nutritional support or surgical revision [27].

Nutritional follow-up must therefore integrate anthropometric measurements, malnutrition screening, dietary assessment, muscle strength, functional tests, and body composition by DXA, bioelectrical impedance analysis, or computed tomography when available. The ESPEN/UEG guidelines emphasize that the goal of any intervention in obesity should be to reduce fat while minimizing loss of muscle mass, and recommend screening for sarcopenia or sarcopenic obesity in at-risk individuals, with assessment of handgrip strength, knee extension, or chair-stand tests, followed by body composition measurement if function is impaired. In the post-bariatric context, this logic implies not relying solely on albumin or prealbumin, since these markers are influenced by inflammation, hydration, and intercurrent disease, but rather combining biochemical data with actual intake, digestive tolerance, weight trajectory, strength loss, physical activity, and adherence to supplementation. When oral intake is insufficient or sustained protein deterioration exists, escalation should include high-protein supplements, enteral nutrition, and, in selected cases, parenteral nutrition or anatomical correction, always within a multidisciplinary bariatric team [28].

Prevention cannot rest solely on "increasing protein," because muscle preservation depends on the interaction between amino acid substrate, mechanical stimulus, energy balance, intensity of weight loss, and prior functional reserve. In a

randomized trial in women undergoing gastric bypass, whey protein supplementation combined with supervised resistance training for 18 weeks did not significantly prevent lean mass loss compared with usual care, but it did produce a greater increase in relative lower-limb strength, suggesting that functional recovery may precede or not be linearly reflected in body mass changes. Complementarily, a meta-analysis of eight clinical trials found that protein intake above the recommended value was associated with greater weight and fat mass loss, but did not demonstrate an overall significant effect on preservation of fat-free mass, except in the sleeve gastrectomy subgroup. Taken together, the evidence supports a combined, early, and personalized strategy: sufficient protein intake, preferably distributed throughout the day and adjusted to ideal body weight, progressive resistance training, correction of digestive intolerances, surveillance of muscle mass and strength, and prolonged follow-up to prevent weight-loss success from becoming postoperative metabolic frailty [29] [30].

5. Micronutrient Deficiencies with Endocrine-Metabolic Impact

Micronutrient deficiencies after bariatric surgery are not an incidental or exclusively malabsorptive phenomenon, but rather the consequence of a convergence between preoperative nutritional vulnerability, sustained reduction in intake, food intolerances, gastric hypoacidity, exclusion of absorptive segments, reduced contact with bile and pancreatic enzymes, vomiting, diarrhea, systemic inflammation, medication use, and variable adherence to supplementation. For this reason, risk begins even before the procedure: the integrated guidelines of the American Society for Metabolic and Bariatric Surgery recommend systematic preoperative screening for thiamine, vitamin B12, folate, iron, vitamin D, calcium, and, according to procedure and clinical risk, fat-soluble vitamins, copper, and zinc, because many candidates already present one or more deficiencies before surgery. This point is fundamental because it prevents attributing every deficit to the surgical act and requires micronutritional follow-up to be conceived as a continuum that begins before surgery and extends indefinitely throughout post-bariatric life [31].

The frequency of deficiencies varies according to surgical technique, follow-up time, supplementation received, and measurement method. In a cross-sectional cohort of 365 post-bariatric patients, the most frequent abnormalities were vitamin D deficiency in 55.3%, iron deficiency in 40.0%, anemia in 38.9%, vitamin B12 deficiency in 30.1%, folate deficiency in 22.2%, and calcium deficiency in 20.8%, with multiple deficiencies in 36.7% of cases. Vitamin D and iron deficiencies were more common after gastric bypass than after sleeve gastrectomy, whereas women had a higher frequency of anemia and vitamin D deficiency; moreover, risk increased with time elapsed since surgery, reinforcing that these complications do not belong only to the early postoperative period, but may consolidate as chronic morbidity if follow-up is interrupted [32].

Iron deficiency is one of the most relevant alterations because of its frequency,

functional impact, and close relationship with fatigue, exercise intolerance, reduced work performance, exertional dyspnea, and anemia. After Roux-en-Y gastric bypass, exclusion of the duodenum and proximal jejunum reduces iron exposure to the main absorptive territory, whereas decreased gastric acidity limits the conversion of ferric iron into more bioavailable forms; in sleeve gastrectomy, although there is no intestinal bypass, lower acid production, reduced intake, intolerance to red meat, and persistent inflammation may also promote iron deficiency. Reported prevalences are broad, ranging from 18% to 53% after gastric bypass and from 1% to 54% after sleeve gastrectomy, reflecting methodological heterogeneity, differences in supplementation, menstrual variability, and difficulty interpreting ferritin in the presence of inflammation. Therefore, assessment should not depend on a single marker, but should integrate complete blood count, ferritin, serum iron, transferrin, transferrin saturation, C-reactive protein, and clinical symptoms [33].

Post-bariatric anemia requires a broader approach than the isolated search for iron deficiency. Iron deficiency usually produces microcytic anemia, whereas vitamin B12 and folate deficiencies are associated with macrocytosis; however, in practice, mixed deficiencies may coexist and generate apparently normocytic erythrocyte indices. Copper deficiency, although less frequent, may also cause anemia, neutropenia, and neurological alterations, so its omission may lead to incomplete diagnoses in patients with persistent cytopenias or progressive neuropathy. A systematic review on iron, vitamin B12, folate, and copper showed that evidence regarding prevalence and the specific contribution to anemia during the first postoperative year remains limited, partly because many studies do not adequately control for inflammation, supplementation, adherence, or functional markers such as methylmalonic acid for vitamin B12. Consequently, post-bariatric anemia should be interpreted as a multifactorial hematometabolic syndrome rather than as a uniform category [34].

Thiamine deficiency occupies a particular place because it may appear early, progress rapidly, and produce irreversible neurological damage. Thiamine has limited body stores, a short half-life, and a central role in neuronal energy metabolism; therefore, persistent vomiting, low intake, rapid weight loss, parenteral nutrition without adequate replacement, or glucose administration in depleted patients may precipitate Wernicke encephalopathy. In a preoperative series of 346 patients, 3.5% had concentrations below the normal limit and an additional 14% were at risk of deficiency; furthermore, the review of 118 cases of post-bariatric Wernicke encephalopathy showed that patients presented with vomiting in 87.3%, ataxia in 84.7%, altered mental status in 76.3%, and oculomotor disorders in 73.7%, with frequent onset during the first months after surgery. Therefore, in the presence of persistent vomiting or compatible neurological symptoms, parenteral thiamine should be administered empirically and urgently, without waiting for biochemical confirmation [35] [36].

Trace elements add a highly complex diagnostic layer because their deficiencies

may mimic other neurological, hematological, or immunological diseases. Copper is absorbed predominantly in the stomach and proximal duodenum; therefore, procedures with intestinal bypass, excessive zinc supplementation, and episodes of diarrhea or poor intake may favor clinically severe depletion. Copper-deficiency myelopathy may resemble subacute combined degeneration due to vitamin B12 deficiency, with paresthesias, sensory ataxia, weakness, hyperreflexia, gait disturbance, and spinal cord lesions. In a case described after one-anastomosis gastric bypass, the patient had copper of 4 μmol/L and ceruloplasmin of 94 mg/L, neurological progression despite intravenous and enteral replacement, and required surgical reversal, with biochemical normalization but incomplete neurological recovery. This example illustrates why delayed detection may leave permanent sequelae and why there should be a low threshold for requesting copper and ceruloplasmin in patients with unexplained anemia, neutropenia, neuropathy, myelopathy, or prolonged zinc supplementation [37].

Biochemical follow-up should be structured but also interpretive. In a prospective cohort with control of supplementation, inflammation, and glycemic control during the first postoperative year, preoperative deficiencies were frequent for vitamin D, iron, and selenium; after gastric bypass, deficiencies of vitamin B1, vitamin A, and selenium increased early, whereas after sleeve gastrectomy, folate was vulnerable at six months. However, the study also showed that inflammation modifies the interpretation of several micronutrients: it may increase ferritin, copper, and vitamin B1, reduce vitamin A, and alter functional selenium markers, meaning that a "normal" result does not always equal biological sufficiency and a low result does not always reflect isolated depletion. Hence, optimal follow-up combines type of surgery, symptoms, actual intake, adherence, inflammation, medication use, reproductive status, postoperative time, and hematological or neurological findings, reserving the in-depth discussion of vitamin D, calcium, and parathyroid hormone for the specific analysis of post-bariatric metabolic bone disease [38].

6. Post-Bariatric Metabolic Bone Disease

Post-bariatric metabolic bone disease should be understood as a chronic and multifactorial endocrine-metabolic complication, not as a simple consequence of "low vitamin D." After bariatric surgery, bone is exposed to a combination of reduced mechanical loading due to accelerated weight loss, reduced lean mass, lower protein intake, decreased intestinal calcium absorption, vitamin D insufficiency, secondary hyperparathyroidism, increased bone turnover markers, and modifications in endocrine signals derived from adipose tissue and the intestine. This convergence may produce a high-turnover bone phenotype, loss of mineral density, microarchitectural deterioration, and a progressive increase in fracture risk. The position statement of the European Calcified Tissue Society summarizes this concern by indicating that bariatric surgery is associated with an approximate 21% - 44% increase in fracture risk, with a time-dependent pattern that usually becomes

more evident around the third postoperative year and with greater impact in procedures with a malabsorptive component, such as Roux-en-Y gastric bypass and biliopancreatic diversion [39].

Skeletal damage is not distributed homogeneously across surgical techniques. In the randomized Oseberg trial, which compared sleeve gastrectomy and Roux-en-Y gastric bypass in patients with severe obesity and type 2 diabetes, bypass was associated at one year with greater reduction in bone mineral density at the femoral neck, total hip, and lumbar spine, whereas increases in P1NP and CTX-1 were approximately 100% higher compared with sleeve gastrectomy. Complementarily, a study of 650 patients assessed at two years showed decreased mineral density after both techniques, but with a significantly greater reduction at the femoral trochanter and lumbar spine after gastric bypass, as well as a more pronounced increase in parathyroid hormone. These findings suggest that post-bariatric skeletal deterioration depends not only on the magnitude of weight loss, but also on surgical anatomy, duodenojejunal exclusion, mineral absorption, and the intensity of bone remodeling induced by each procedure [40] [41].

Sleeve gastrectomy, despite preserving intestinal continuity, should not be considered neutral for mineral metabolism. In a prospective study using a dual stable isotope method, fractional intestinal calcium absorption decreased from 31.4% to 16.1% six months after sleeve gastrectomy, despite robust 25-hydroxyvitamin D concentrations and calcium intake consistent with recommendations. In addition, the decline in absorption was accompanied by a substantial increase in bone turnover markers and decreased mineral density at the proximal femur, with greater loss of total hip BMD among those with lower postoperative calcium absorption. This finding is particularly important because it shifts interpretation away from a purely anatomical logic—"if there is no intestinal bypass, there is no relevant mineral malabsorption"—toward a functional view, in which changes in gastric acidity, intake, emptying, hormonal signals, and intestinal adaptation may alter calcium-bone homeostasis even without intestinal exclusion [42].

The most relevant clinical outcome of this cascade is fracture. In a population-based cohort of 42,345 Medicare beneficiaries, Roux-en-Y gastric bypass was associated with a nonvertebral fracture incidence of 6.6 per 1000 person-years compared with 4.6 per 1000 person-years after adjustable gastric banding, corresponding to an adjusted hazard ratio of 1.73. The risk was particularly elevated for hip fracture, with an HR of 2.81, in addition to increased risk at the wrist and pelvis; importantly, the pattern was similar in older and younger adults, without significant interaction by age, sex, diabetes, or race. This evidence confirms that densitometric and biochemical alterations are not only intermediate markers, but signals of late skeletal fragility with functional implications, especially in postmenopausal patients, men older than 50 years, individuals with low muscle mass, falls, persistent deficiencies, or malabsorptive procedures [43].

Diagnosis must integrate clinical, biochemical, and densitometric stratification. In high-risk patients, especially postmenopausal women and men older than 50

years, assessment should include history of fragility fractures, falls, smoking, alcohol use, osteotoxic medications, hypogonadism, kidney or liver disease, and bone mineral density measurement by DXA at the lumbar spine and hip, ideally with vertebral assessment when silent fracture is suspected. Laboratory evaluation should include calcium, phosphorus, albumin, 25-hydroxyvitamin D, parathyroid hormone, alkaline phosphatase, and, when possible, remodeling markers such as CTX and P1NP, interpreted together with intake, supplementation, adherence, and postoperative time. Contemporary recommendations emphasize that bone-risk screening should be as systematic as cardiometabolic control, but a gap remains between recognition of the problem and its routine incorporation into bariatric follow-up [44].

Prevention and treatment require a combined strategy, because no single axis corrects the biology of post-bariatric high bone turnover. The therapeutic foundation includes correction of vitamin D deficiency, adequate calcium intake—preferably in forms with better absorption in contexts of low acidity—sufficient protein intake, progressive resistance training, fall prevention, and longitudinal surveillance of BMD and metabolic markers. When established osteoporosis, fragility fracture, or high risk exists in postmenopausal patients or men older than 50 years, consensus statements tend to prefer zoledronic acid over oral bisphosphonates because of issues related to gastrointestinal tolerance and absorption, ensuring prior vitamin D and calcium sufficiency to reduce the risk of hypocalcemia; denosumab may be considered when bisphosphonates are not appropriate, although it requires careful planning because of the risk of hypocalcemia and rebound after discontinuation. Within this logic, the BABS Study supports multimodal management: preoperative vitamin D loading, sustained supplementation with vitamin D, calcium, and adjusted protein, together with physical exercise, attenuated the loss of bone mineral density and lean mass and reduced the magnitude of the increase in bone turnover markers. Therefore, post-bariatric bone health should be addressed from the preoperative period and maintained as long-term endocrine-metabolic surveillance, not as a late intervention once fracture has already occurred [44] [45].

7. Gonadal, Reproductive, and Systemic Hormonal Alterations

Bariatric surgery produces endocrine reprogramming that extends beyond weight loss and substantially modifies the hypothalamic-pituitary-gonadal, thyroid, and reproductive axes. In severe obesity, visceral adipose tissue behaves as a dysfunctional endocrine organ capable of amplifying insulin resistance, low-grade inflammation, peripheral aromatization, reduced sex hormone-binding globulin, and alterations in sex steroid secretion. This biology is expressed with clear sexual dimorphism: in women, obesity is associated with hyperandrogenism, anovulation, and polycystic ovary syndrome; in men, with functional secondary hypogonadism, reduced total and free testosterone, decreased SHBG, progressive visceral ad-

iposity, and impaired sexual function. In a meta-analysis of patients with severe obesity undergoing bariatric surgery, obesity-associated gonadal dysfunction was highly prevalent: polycystic ovary syndrome was present in 36% of women and male obesity-associated hypogonadism in 64% of men; after surgery, pooled resolution reached 96% for polycystic ovary syndrome and 87% for male hypogonadism, accompanied by increased SHBG, decreased estradiol in both sexes, and divergent androgenic changes, with reduced testosterone in women and increased testosterone in men [46].

In women with polycystic ovary syndrome, surgical weight loss may restore ovarian function by reducing hyperinsulinemia, inflammatory pressure, ovarian/adrenal androgen production, and visceral adiposity. This effect should not be interpreted merely as cosmetic improvement of hirsutism, but rather as reactivation of the ovulatory axis with immediate reproductive implications. In a systematic review and meta-analysis of nine studies including 234 patients with obesity and polycystic ovary syndrome, bariatric surgery significantly reduced abnormal menstruation, hirsutism, total testosterone, free testosterone, and body mass index, while also improving metabolic comorbidities such as type 2 diabetes and hypertension. This endocrine-reproductive restoration turns the post-bariatric woman into a patient at risk of unplanned pregnancy if early contraceptive counseling is not provided, especially during the phase of rapid weight loss, when catabolism, nutritional instability, and a higher probability of maternal-fetal deficiencies coexist [47].

Fertility after bariatric surgery requires a transition from purely weight-centered surveillance toward structured preconception planning. The international consensus on pregnancy after bariatric surgery recommends specialized reproductive health care, appropriate contraceptive choice, nutritional counseling, supplementation, micronutrient assessment, and individualized antenatal and postnatal follow-up; it also emphasizes that improved fertility through restoration of ovulation may occur before the patient reaches metabolic stability. Along the same line, recent clinical reviews recommend delaying conception for at least 12 months after surgery, ideally until weight stabilization, nutritional optimization, and control of complications have been achieved, because early pregnancy during the rapid weight-loss phase is associated with greater vulnerability to macro- and micronutrient deficiencies, prematurity, fetal growth restriction, small-for-gestational-age newborns, and the need for close obstetric surveillance. Therefore, contraception should not be a secondary recommendation, but an integral part of consent and postoperative follow-up in every woman of reproductive age [48] [49].

Post-bariatric pregnancy clearly illustrates the ambivalent nature of endocrine-metabolic reprogramming: it reduces some risks inherent to maternal obesity, but introduces risks derived from surgical anatomy, altered absorption, postprandial hypoglycemia, and limited nutritional reserve. Women with a bariatric history usually have a lower incidence of preeclampsia and gestational diabetes, but a

higher risk of small-for-gestational-age newborns and, in some series, fetal death; moreover, the oral glucose tolerance test may be poorly tolerated or physiologically inappropriate after procedures such as gastric bypass because of the risk of dumping and reactive hypoglycemia, requiring alternative glucose-monitoring strategies. During pregnancy, specific surgical complications must also be considered: a systematic review by the BARIA-MAT group identified 120 pregnant women who required emergency surgery for complications related to previous bariatric surgery, predominantly after gastric bypass, with internal hernia, intussusception, intestinal obstruction, band slippage, volvulus, and perforation as the main diagnoses, in addition to maternal mortality of 2.5% and fetal mortality of 7.5%. Therefore, abdominal pain, persistent vomiting, or obstructive symptoms in a post-bariatric pregnant patient should trigger urgent surgical evaluation and should not be automatically attributed to benign gestational symptoms [49] [50].

The male gonadal axis deserves independent consideration because obesity-associated secondary hypogonadism is frequent, potentially reversible, and clinically relevant. Male obesity reduces testosterone through multiple interrelated mechanisms: increased aromatization in adipose tissue, functional suppression of the hypothalamic-pituitary-testicular axis, insulin resistance, inflammation, decreased SHBG, expansion of visceral fat, and possible alteration of leptin-kisspeptin-GnRH signaling. In a cohort of 413 men with severe obesity, 62% had biochemical hypogonadism using a total testosterone threshold of 2.64 ng/mL; among those who underwent bariatric surgery, only 20.1% still had hypogonadism at 3 - 6 months, and recovery was independently associated with preoperative testosterone, percentage of excess weight loss, and mixed restrictive-malabsorptive techniques. This pattern suggests that surgery may reverse functional hypogonadism in a substantial proportion of patients, although biochemical normalization should not be assumed to automatically equal recovery of fertility, spermatogenesis, sexual desire, or erectile function without specific clinical assessment [51].

The thyroid axis constitutes another systemic dimension of post-bariatric adaptation. Obesity is associated with a higher prevalence of overt or subclinical hypothyroidism and with elevations in TSH that may reflect both true thyroid dysfunction and neuroendocrine adaptation to excess weight. In a meta-analysis of 28 studies including 1284 patients, bariatric surgery was associated with a significant decrease in TSH, resolution of subclinical hypothyroidism in 87% of cases, and an average reduction in levothyroxine dose among patients with overt hypothyroidism. However, management is not linear: levothyroxine is a narrow-therapeutic-index drug, and its exposure may be modified by reduced gastric acidity, use of proton pump inhibitors or H2 blockers, interaction with calcium and iron, reduced intake, weight changes, and bypass of intestinal segments involved in absorption. Consequently, after bariatric surgery, the hypothyroid patient requires serial monitoring of TSH and free T4, review of formulation, administration timing, separation from mineral supplements, and dynamic dose adjustment, avoid-

ing both undertreatment due to malabsorption and overtreatment secondary to weight loss and reduced requirements [52].

8. Weight Regain and Recurrence of Metabolic Comorbidities

Weight regain after bariatric surgery should not be interpreted as a binary failure or as a simple consequence of poor individual adherence, but rather as a manifestation of obesity as a chronic, relapsing, and biologically defended disease. In this context, it is essential to differentiate among suboptimal initial responses, insufficient weight loss, partial responses, and weight recurrence. The IFSO consensus proposed standardizing this terminology to reduce heterogeneity across studies and improve clinical decision-making: a suboptimal initial clinical response may be defined as total weight loss below 20%, whereas recurrent weight gain is conceptualized as regain exceeding 30% of the initially lost weight or as worsening of an obesity-related complication. This precision is relevant because not every increase in weight after reaching the nadir implies surgical failure, and not every smaller-than-expected loss has the same pathophysiological, anatomical, or therapeutic meaning [53].

The magnitude of the problem depends on the definition used, the procedure performed, follow-up duration, and the outcome considered clinically relevant. In the literature, insufficient weight loss and weight regain have been defined using percentages of excess weight loss, weight regained from the nadir, percentage of maintained total weight loss, or deterioration of comorbidities, which explains the wide variability in reported prevalence. A scoping review indicated that approximately 20% - 25% of patients may experience clinically considerable weight regain, whereas insufficient weight loss, often defined as less than 50% excess weight loss, represents a frequent cause of revisional surgery. Concordantly, in a cohort of 868 patients evaluated five years after Roux-en-Y gastric bypass or sleeve gastrectomy, 87% experienced some degree of weight recurrence, but the frequency of "significant" regain ranged from 16% to 37% depending on the definition applied. These data show that the problem does not lie only in measuring weight, but in identifying what degree of recurrence is associated with functional, metabolic, or quality-of-life deterioration [54] [55].

The mechanisms of weight regain are heterogeneous and rarely obey a single cause. After the nadir, compensatory metabolic adaptations may emerge, including reduced energy expenditure, increased energy efficiency, partial recovery of appetite, changes in intestinal and adipocytic signals, reduced satiety, reappearance of hedonic eating, and vulnerability to liquid or ultra-processed foods with high caloric density. These mechanisms are compounded by behavioral and psychosocial factors such as grazing, night eating, binge eating, alcohol use, depression, anxiety disorders, loss to follow-up, sedentary behavior, and internalized stigma. Anatomical causes should also be considered, including dilation of the pouch or residual fundus after sleeve gastrectomy, dilation of the gastrojejunal

anastomosis, gastrogastric fistula after bypass, a wide sleeve, or technical issues related to the initial procedure. Overall, weight recurrence should be evaluated as a multifactorial reactivation of neuroendocrine, anatomical, and behavioral circuits, not as a moral or disciplinary category [56].

The most important clinical consequence of weight regain is not isolated weight increase, but the progressive loss of metabolic response. Recurrence or worsening of type 2 diabetes, arterial hypertension, dyslipidemia, obstructive sleep apnea, metabolic liver disease, osteoarticular pain, and impaired quality of life may occur even when the patient retains part of the initial weight-loss benefit. In type 2 diabetes, relapse must be interpreted with particular care: metabolic surgery may induce remission, reduce medication use, and improve glycemic control for years, but diabetes remains a progressive disease influenced by beta-cell reserve, preoperative disease duration, previous insulin use, age, weight trajectory, and recurrence of visceral adiposity. A review on maintenance of diabetes remission indicates that only 20% - 50% of patients who achieve remission remain in remission long term, and that approximately 30% may experience relapse; however, relapse should not be considered absolute failure, because a cardiometabolic "legacy effect" may persist, with lower diabetes severity and reduced medication requirements [57].

Long-term randomized evidence reinforces this nuanced interpretation. In the ARMMS-T2D pooled analysis, which integrated four randomized clinical trials with prolonged follow-up, patients undergoing bariatric surgery maintained better glycemic control than those treated with medical and lifestyle management: at seven years, HbA1c reduction was greater in the surgical group, and the between-group difference remained significant even at twelve years. In addition, diabetes remission was more frequent after surgery at both seven and twelve years, although remission rates decreased over time. This observation is central to post-bariatric follow-up: the progressive decline in remission does not invalidate the metabolic superiority of surgery, but it does require longitudinal surveillance, prevention of weight regain, early treatment of recurrent hyperglycemia, and selection of drugs with weight or cardiometabolic benefit when diabetes reappears [58].

Clinical evaluation of weight regain should be structured and should not be limited to recommending "diet and exercise." It must reconstruct the complete weight trajectory, identify the nadir, quantify the percentage regained, review the surgical technique, digestive symptoms, postprandial hypoglycemia, reflux, vomiting, abdominal pain, protein intake, consumption of caloric liquids, alcohol use, physical activity, sleep, mental health, obesogenic medications, and loss of contact with the bariatric team. It should also evaluate body composition, glucose, HbA1c, blood pressure, lipid profile, liver function, nutritional deficiencies, and recurrence of comorbidities. When the history suggests anatomical failure, or when significant weight regain, obstructive symptoms, severe reflux, or loss of restriction is present, endoscopy, contrast studies, or imaging should be considered to assess the pouch, sleeve, anastomosis, fistulas, or dilations. This approach al-

lows therapeutic phenotypes to be distinguished: patients with predominantly behavioral, neurohormonal, pharmacological, anatomical, or mixed drivers.

Management should be stepwise and proportional to the identified phenotype. Nutritional and behavioral intervention remains necessary, but it is rarely sufficient when weight regain is clinically significant; therefore, anti-obesity pharmacotherapy occupies an increasing role as a complement to surgery. Observational evidence in post-bariatric populations supports benefits with liraglutide, topiramate, and phentermine/topiramate, whereas modern GLP-1 receptor agonists and higher-potency incretin-based therapies represent a promising pathway to optimize weight loss or control recurrence, although specific long-term data in postsurgical patients are still required. When a correctable anatomical alteration is present, endoscopic therapies, such as transoral outlet reduction after bypass, or revisional surgical strategies may be considered in expert centers. The IFSO position statement emphasizes that, because obesity may require multiple interventions throughout life, recurrence of weight or obesity-related complications should be managed with individualized treatment, combining intensive follow-up, pharmacotherapy, bariatric endoscopy, and revisional surgery when the risk-benefit balance is favorable [59] [60].

9. Prevention, Screening, and Longitudinal Endocrine-Metabolic Follow-Up

Prevention of endocrine-metabolic complications after bariatric surgery should be conceived as a chronic care model, not as a brief sequence of weight-centered visits. Surgery induces a new digestive, absorptive, hormonal, behavioral, and pharmacokinetic physiology; therefore, follow-up must integrate surveillance of weight loss, food tolerance, nutritional deficiencies, lean mass, bone metabolism, glycemic control, recurrence of comorbidities, mental health, pregnancy, and signs of anatomical failure. The recommendations of the European Association for the Study of Obesity emphasize that the post-bariatric patient may develop specific diagnostic, preventive, and therapeutic needs, and that long-term multidisciplinary follow-up is mandatory, with referral to a bariatric center in complex clinical situations [61].

Prevention begins in the preoperative period. Before surgery, micronutrient deficiencies, poorly controlled diabetes, metabolic liver disease, hypertension, sleep apnea, bone risk, psychological disorders, alcohol or tobacco use, obesogenic medications, and barriers to adherence should be identified. The 2021 ERAS guidelines recommend individualized preoperative information and education, alcohol and tobacco screening, smoking cessation at least four weeks before surgery, strict abstinence in patients with alcohol abuse, and preoperative weight loss with a low- or very-low-calorie diet. In patients with diabetes, this phase requires close monitoring because of the risk of hypoglycemia and the need to adjust glucose-lowering medications [62]. This preparation not only reduces perioperative risk, but also establishes the baseline from which weight loss, metabolic remission, supplementation, and comorbidity evolution will be interpreted.

Early follow-up, particularly during the first year, should focus on the phase of greatest catabolic velocity. During this period, protein intake, hydration, vomiting, food intolerance, dumping or hypoglycemic symptoms, excessive lean mass loss, fatigue, neuropathy, adherence to multivitamins, and signs of malnutrition should be monitored. In practical terms, nutritional reviews are commonly organized at 1, 3, 6, 9, and 12 months during the first year, with at least annual follow-up thereafter. Physical activity should also progress under supervision, avoiding prolonged immobilization while respecting the healing phase, with gradual incorporation of training aimed at preserving fat-free mass and preventing weight regain [63].

Biochemical surveillance should be scheduled and stratified by procedure. As a minimum panel, it should include complete blood count, platelets, electrolytes, renal and liver function, iron, ferritin, folate, vitamin B12, calcium, 25-hydroxyvitamin D, and parathyroid hormone, with greater intensity in malabsorptive procedures. European recommendations propose monitoring every 3 - 6 months during the first year and then annually after sleeve gastrectomy and gastric bypass; in biliopancreatic diversion or duodenal switch, follow-up should be closer, every three months during the first year and every 6 - 12 months thereafter. Complementarily, recent reviews emphasize that micronutrient monitoring should not be limited to the first year: the schedule may include testing every three months during the first year, every six months in the second year, and annually thereafter, with targeted measurements of vitamin A, zinc, selenium, copper, or thiamine in cases of chronic diarrhea, steatorrhea, alopecia, neurological symptoms, unexplained anemia, or poor adherence [61] [64].

Adherence to follow-up must be interpreted critically. Although attendance during the first 0.5 to 3 years is associated with greater excess weight loss, evidence does not show with the same clarity that rigid follow-up models after three years improve total weight loss by themselves; therefore, the goal should not be to multiply indefinite visits, but to design follow-up proportional to risk [65]. In stable patients, a shared model between the bariatric center and primary care may be used, whereas patients with hypoglycemia, persistent vomiting, refractory anemia, hypoalbuminemia, neuropathy, fractures, pregnancy, weight regain, diabetes relapse, or suspected anatomical alteration should re-enter specialized care.

Metabolic follow-up must incorporate the relapsing nature of obesity and type 2 diabetes. The American Diabetes Association standards recognize that metabolic surgery can induce weight losses greater than 20%, substantially improve glycemia, and promote diabetes remission; however, they recommend maintaining weight management as a primary therapeutic goal together with glycemic control, using person-centered, stigma-free language [66]. In the post-bariatric patient, this implies measuring body weight and body composition, but also HbA1c, glucose, blood pressure, lipid profile, liver function, symptoms of sleep apnea, recurrence of medication use, and the need for anti-obesity or antidiabetic pharmacotherapy with a favorable weight profile.

Finally, mental health should be part of longitudinal screening. Umbrella-review evidence shows that bariatric surgery is associated with improvement in depression, anxiety, and eating disorders in many patients, but also with signals of increased risk for suicide, self-harm, and alcohol use disorder, especially in later stages [67]. Therefore, follow-up should include periodic assessment of depression, anxiety, binge eating, loss-of-control eating, alcohol use, suicidal ideation, and adaptation to body changes. The lack of consensus on what constitutes "long-term" follow-up complicates comparison of outcomes across studies; however, in a benign disease with life expectancy extending for decades after surgery, follow-up should continue beyond the first five years and should be oriented toward preserving benefits, detecting recurrences, and escalating treatment in a timely manner [68].

10. Limitations

This review should be interpreted considering some limitations inherent to the available evidence. Methodological quality varies across the different endocrine-metabolic complications analyzed: some outcomes are supported by randomized trials, meta-analyses, or prospective cohorts, whereas others, especially rare complications, severe low-frequency events, and certain revisional strategies, depend primarily on observational studies, consensus statements, or case reports. Likewise, heterogeneity in surgical techniques, diagnostic definitions, follow-up duration, adherence to supplementation, and reporting criteria limits direct comparison across studies and require that recommendations be interpreted within the individual clinical context.

11. Conclusions

Bariatric surgery should be understood as a complex metabolic intervention that induces a new digestive, hormonal, and nutritional physiology. Although it offers substantial and sustained benefits in weight loss, glycemic control, and reduction of comorbidities, it may also generate specific endocrine-metabolic complications that compromise quality of life, muscle function, bone health, nutritional status, glycemic stability, and the durability of the surgical outcome.

Postoperative follow-up should not be restricted to body weight measurement. Longitudinal assessment must integrate postprandial symptoms, body composition, protein intake, micronutrients, calcium-vitamin D-parathyroid hormone metabolism, glycemic control, reproductive health, thyroid function, mental health, therapeutic adherence, and recurrence of comorbidities. This surveillance should begin before surgery, intensify during the first year, and continue indefinitely with risk-adjusted periodicity.

Prevention of complications requires preoperative education, correction of deficiencies, individualized supplementation, physical activity aimed at preserving lean mass, early detection of hypoglycemia and dumping, screening for bone disease, monitoring of diabetes and cardiovascular comorbidities, and psychosocial

intervention when necessary. In patients with weight regain, metabolic relapse, or suspected anatomical failure, management should escalate rationally toward anti-obesity pharmacotherapy, bariatric endoscopy, or revisional surgery in specialized centers.

Overall, the success of bariatric surgery does not depend solely on the initial procedure, but on the capacity of the health care system to sustain comprehensive, anticipatory, and multidisciplinary endocrine-metabolic care. Recognizing these complications as part of the postoperative physiological continuum allows preservation of surgical benefits, reduction of preventable morbidity and mortality, and optimization of long-term clinical outcomes.

Author Contributions

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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