Indian Journal of
Nutrition

Open Science Publications

Volume 13, Issue 1 - 2026
© Bellani D, et al. 2026
www.opensciencepublications.com

Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments

Review Article

Bellani D*, Desai A, Anand S, Maldahiar SP, Mishra S and Hegde R

Department of Medical Affairs, IntelliMed Healthcare Solutions Sakhi House 2nd Floor, Corporate Park, VN Purav Marg, Swastik Chamber, Chembur, Mumbai, Maharashtra India

***Corresponding author:** Depti Bellani, Department of Medical Affairs, IntelliMed Healthcare Solutions Sakhi House 2nd Floor, Corporate Park, VN Purav Marg, Swastik Chamber, Chembur, Mumbai, Maharashtra India. E-mail Id: depti.bellani@intellimed.org

Article Information: **Submission:** 09/04/2026; **Accepted:** 24/04/2026; **Published:** 28/04/2026

Copyright: © 2026 Bellani D, et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Background: Obesity management has rapidly expanded with glucagon-like peptide-1 receptor agonists (GLP-1 RAs), dual incretin therapies, alongside established bariatric surgery. Although these interventions induce durable weight loss, emerging evidence indicates, rapid weight reduction carries nutritional and metabolic consequences inadequately addressed in current clinical frameworks.

Objective: To synthesize existing evidence on the mechanisms, prevalence, and clinical impact of nutritional deficiencies associated with GLP-1–based therapies and bariatric surgery, and to propose an integrated, evidence-based approach to nutritional monitoring and supplementation.

Methods: This narrative review integrates data from randomized controlled trials, meta-analyses, observational studies, and international guidelines, including those from the American Diabetes Association (ADA), Endocrine Society, American Heart Association/American College of Cardiology/The Obesity Society (AHA/ACC/TOS), American Society for Metabolic and Bariatric Surgery (ASMBS), International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO), British Obesity and Metabolic Surgery Society (BOMSS), and Indian Council of Medical Research (ICMR).

Results: Pharmacologic and surgical weight-loss modalities converge on pathways of reduced intake, altered gastrointestinal physiology, and systemic metabolic adaptation, resulting in a high protein and micronutrient deficiency risk. Evidence demonstrates that one-fourth of weight loss may derive from fat-free mass, with clinically significant deficiencies emerging within the first year of therapy. While bariatric care incorporates structured nutritional surveillance and supplementation, pharmacologic pathways lack comparable protocols despite achieving similar magnitudes of weight loss. This gap is particularly relevant in Indian populations, where high baseline prevalence of micronutrient deficiency and distinct body composition profiles amplify vulnerability.

Conclusion: Rapid weight loss should be reframed as a state of heightened nutritional risk requiring proactive, structured management. Bariatric-style monitoring, individualized supplementation, and body composition assessment must be integrated into GLP-1–based care pathways to preserve metabolic benefits while preventing nutritional compromise. Future research should prioritize trials evaluating supplementation strategies and long-term nutritional outcomes in treated populations..

Keywords: GLP-1 receptor agonists; Dual incretin therapy; Bariatric surgery; Rapid weight loss; Nutritional deficiency; Sarcopenia

ISSN: 2395-2326

Introduction

Rationale for Focusing on GLP-1 Therapies and Bariatric Surgery

Obesity care has entered an era where we can transform body weight faster than we are prepared to protect nutritional health. Targeted metabolic interventions now enable sustained, clinically meaningful weight loss, marking a new landmark in obesity management. GLP-1 receptor agonists (GLP-1 RAs) and dual incretin therapies achieve 15-25% reductions in body weight with broad metabolic, cardiovascular, and renal benefits [1,2]. Modern bariatric procedures deliver similar or greater effects, with durable >20% weight loss and substantial reductions in long-term morbidity and mortality [3,4].

However, this degree of weight loss reflects systemic metabolic adaptation rather than selective adiposity loss. Approximately 25% of total weight reduction arises from loss of fat-free mass [5]. Both therapeutic modalities constrain nutrient intake and absorption—GLP-1 agents through appetite suppression, delayed gastric emptying [6,7], and bariatric surgery via anatomical alteration that impairs uptake of iron, calcium, vitamin B12, and fat-soluble vitamins [8].

Deficiencies emerge early and are clinically meaningful. Observational data report up to 12.7% deficiency in six months of GLP-1 therapy, rising to 22.4% in one year, predominantly involving protein, vitamin D, and iron, with downstream effects on strength, bone health, and metabolic resilience [9].

These risks are amplified in Indian populations, characterized by high baseline micronutrient deficiency and the "thin–fat" phenotype visceral adiposity with low lean mass at comparatively lower BMI thresholds [10,11]. Vegetarian dietary patterns further predispose to vitamin B12, iron, and vitamin D insufficiency, heightening vulnerability during rapid treatment-induced weight loss.

Despite parallel metabolic efficacy, pharmacologic guidelines, including those from the American Diabetes Association (ADA), the Endocrine Society Clinical Practice Guidelines, and the American Heart Association/American College of Cardiology/The Obesity Society (AHA/ACC/TOS), rarely mandate structured nutritional surveillance. This stands in contrast to bariatric protocols from the American Society for Metabolic and Bariatric Surgery (ASMBS), the International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO), and the British Obesity and Metabolic Surgery Society (BOMSS), which embed lifelong monitoring and supplementation.

Scope

This review examines adults undergoing rapid weight loss with GLP-1 RAs, dual incretin therapies, and bariatric surgery, focusing on the interplay between metabolic efficacy and nutritional risk. Particular attention is given to population-specific risk in Indian cohorts, where baseline micronutrient insufficiency and body-composition patterns modify vulnerability.

Methods

Evidence was synthesized narratively from randomized controlled trials, meta-analyses, observational cohorts, and major international and national guidelines, including AHA/ACC/TOS, Endocrine Society, ADA Standards of Care, ASMBS, IFSO, BOMSS, and ICMR. Particular emphasis was placed on DEXA-based body-composition studies and longitudinal assessments of micronutrient status to capture the qualitative as well as quantitative dimensions of weight loss. Integration prioritized mechanistic coherence, clinical relevance, and applicability to real-world practice, with explicit consideration of the Indian context.

Pathophysiology of Nutritional Risk

GLP-1 RAs and bariatric surgery induce weight loss through distinct but converging biological pathways that establish a sustained state of negative energy and nutrient balance. In GLP-1–based therapy, weight reduction is mediated by coordinated central and peripheral mechanisms. Activation of hypothalamic and brainstem circuits suppresses appetite and enhances satiety, while modulation of reward pathways reduces hedonic feeding [12,13]. Peripherally, delayed gastric emptying prolongs gastric distension and alters meal patterns, contributing to sustained reductions in intake [6,14].

These effects extend beyond caloric restriction to influence nutrient exposure and gastrointestinal processing. Reduced gastric motility alters the timing and distribution of nutrients delivered to the small intestine, potentially modifying the absorptive environment for minerals such as iron and calcium [14]. Concurrent hormonal adaptations, including changes in insulin and glucagon signaling, further influence substrate utilization and nutrient partitioning [15,16].

Bariatric procedures achieve weight loss through structural modification of the gastrointestinal tract, combining restrictive, malabsorptive, and hormonal mechanisms. Restrictive components reduce intake, while malabsorptive procedures such as Roux-en-Y gastric bypass exclude the duodenum and proximal jejunum—primary sites of absorption for iron, calcium, and several micronutrients [17]. Altered bile acid circulation and reduced pancreatic enzyme mixing impair fat digestion and absorption, affecting fat-soluble vitamins [8]. In addition, diminished gastric acid and intrinsic factor secretion limit vitamin B12 availability [18].

Despite differing mechanisms, both modalities converge on a shared physiological state characterized by reduced nutrient availability, altered absorption dynamics, and metabolic adaptation. Sustained negative nitrogen balance, increased proteolysis, and attenuated anabolic signaling collectively predispose to loss of lean tissue when nutrient intake is inadequate [11,19]. Hormonal shifts involving insulin, leptin, and sex steroids further influence bone remodeling and metabolic regulation, linking weight loss to skeletal vulnerability [1,20].

Collectively, these mechanisms establish the biological substrate for downstream nutritional deficiency, sarcopenia, and metabolic adaptation observed during rapid weight loss.

Guideline Landscape: Obesity, GLP-1 RAs, and Bariatric Surgery

The management of obesity is anchored in major international

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.

guidelines that frame obesity as a chronic, multifactorial disease requiring long-term, staged intervention. The AHA/ACC/TOS and Endocrine Society recommendations endorse a stepwise escalation from lifestyle therapy to pharmacotherapy and, when indicated, bariatric surgery, guided by treatment response and comorbidity burden [21,22]. ADA Standards of Care position GLP-1 RAs as core agents for patients with obesity and type 2 diabetes, citing robust evidence for weight reduction and cardiovascular and renal risk modification [23,24].

Potent incretin-based therapies, including semaglutide and dual GLP-1/GIP agonists, now achieve 15–25% sustained weight loss with broad cardiometabolic benefit, approaching outcomes traditionally associated with bariatric surgery [2,25]. This convergence in efficacy extends the relevance of surgical-style risk considerations to pharmacologic pathways.

By contrast, bariatric surgery is embedded within detailed nutritional guidance from ASMBS, IFSO, and BOMSS, which explicitly recognize micronutrient deficiency as an inherent consequence of altered gastrointestinal anatomy [26,27]. These guidelines mandate preoperative nutritional assessment, procedure-specific supplementation, and lifelong biochemical surveillance of iron, vitamin B12, calcium, vitamin D, and fat-soluble vitamins, supported by data demonstrating persistent deficiencies and progressive bone loss [3,27,28]. Recommended schedules typically include monitoring at 3–6 month intervals in the first postoperative year and annually thereafter [27].

Pharmacologic guidelines, in contrast, exhibit minimal integration of structured nutritional risk management. ADA, Endocrine Society, and AHA/ACC/TOS documents offer detailed metabolic algorithms but do not require routine micronutrient testing or systematic supplementation for patients treated with GLP-1–based therapies; nutritional advice remains largely generic and non-protocolized [21-23]. This is increasingly misaligned with emerging evidence that GLP-1–induced weight loss is accompanied by reduced intake of protein and micronutrient-rich foods, gastrointestinal intolerance, and quantifiable deficits in vitamin D, iron, and protein status, with deficiency prevalence rising from 12.7% at six months to 22.4% at one year [29-31] Concurrent DEXA data showing 30-40% of total weight loss attributable to fat-free mass loss parallel observations in bariatric cohorts [32,33].

In this context, the absence of structured nutritional frameworks within pharmacologic obesity care pathways represents a critical gap. Bariatric guidelines anticipate and manage nutritional risk through protocolized monitoring and supplementation, whereas pharmacologic pathways remain predominantly focused on glycemic and cardiometabolic endpoints [3,22,27].

The Indian context further accentuates this asymmetry. ICMR and related data highlight the coexistence of obesity with widespread micronutrient deficiency and the "thin-fat" or Asian Indian phenotype—characterized by higher visceral adiposity and lower lean mass at comparatively lower BMI levels [34]. High baseline prevalence of vitamin B12, iron, and vitamin D deficiency amplifies vulnerability to additional nutritional depletion during rapid, GLP-1–mediated weight loss, yet current Indian frameworks do not embed structured nutritional surveillance within pharmacologic pathways [35].

Overall, the guideline landscape reveals a pronounced asymmetry: bariatric surgery is supported by comprehensive, nutrition-centered protocols, whereas pharmacologic regimens achieving comparable weight loss lack analogous structures. Systematic integration of nutritional monitoring, risk stratification, and targeted supplementation into GLP-1–based care is therefore essential to align practice with evolving evidence and safeguard the long-term benefits of weight reduction.

Nutritional Deficiencies with GLP-1–Based Weight Loss

Nutritional deficiency during GLP-1 RA –mediated weight loss reflects the clinical manifestation of sustained reductions in dietary intake and altered eating patterns. Incretin-based therapies are associated with significant decreases in total caloric intake, accompanied by disproportionate reductions in protein consumption and micronutrient-rich food groups [9,30]. These intake patterns frequently fall below recommended thresholds required to maintain physiological function.

Observational data consistently demonstrate inadequate intake of protein, calcium, and iron, alongside widespread vitamin D insufficiency, often superimposed on pre-existing deficiency [30]. These deficits are compounded over time, with the prevalence of clinically significant deficiency increasing from 12.7% at six months to 22.4% at one year of therapy [9].

The clinical consequences of these deficiencies extend beyond biochemical abnormalities. Patients frequently exhibit fatigue, reduced exercise tolerance, and impaired metabolic resilience, reflecting the systemic impact of inadequate nutrient availability. Of particular importance is the loss of lean body mass. Dual-energy x-ray absorptiometry (DXA) body analysis demonstrates that approximately one-fourth of total weight loss during GLP-1 therapy is attributable to reductions in fat-free mass [5]. This loss is associated with diminished strength, reduced resting energy expenditure, and increased risk of sarcopenia, particularly in older individuals and those with baseline vulnerability [31,32].

In the Indian context, these risks are amplified by high baseline prevalence of micronutrient deficiency and dietary patterns characterized by variable protein intake. Deficiencies in vitamin B12, iron, and vitamin D are common even in otherwise healthy individuals, increasing susceptibility to further nutritional compromise during treatment [35].

Despite this growing body of evidence, current pharmacologic guidelines provide limited direction on routine nutritional monitoring or supplementation in GLP-1–treated populations, highlighting a critical gap between emerging clinical data and established care pathways.

Nutritional Deficiencies after Bariatric Surgery

Nutritional deficiency after bariatric surgery is a predictable, multifactorial consequence of profound anatomical and physiological

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.

alterations in the gastrointestinal tract. In contrast to pharmacologic weight loss, where deficits predominantly reflect reduced intake, bariatric procedures directly disrupt digestion and absorption, creating a sustained and often lifelong risk of nutrient depletion.

Pre-existing deficiencies are frequent at baseline, particularly for vitamin D, iron, and vitamin B12, reflecting dietary inadequacy and obesity-related metabolic disturbances, with an even higher burden in Indian populations characterized by pervasive micronutrient insufficiency and limited dietary diversity [35,36]. Procedure type critically shapes deficiency profiles: sleeve gastrectomy mainly restricts intake and lowers gastric acid, while Roux-en-Y gastric bypass combines restriction with malabsorption via exclusion of the duodenum and proximal jejunum—key sites for iron, calcium, and micronutrient absorption; more extensive operations such as biliopancreatic diversion confer even greater malabsorptive burden [3,8].

These structural changes disrupt multiple stages of nutrient assimilation. Bypass of the proximal intestine impairs iron and calcium uptake; altered bile acid circulation and limited pancreatic enzyme mixing reduce fat-soluble vitamin absorption; and diminished gastric acidity compromises liberation and subsequent intrinsic factor–mediated absorption of vitamin B12 [3]. Deficiencies evolve over time; postoperative phases are dominated by low intake and rapid weight loss, whereas long-term deficits reflect persistent malabsorption and poor adherence to supplementation, with longitudinal data confirming that abnormalities can persist or progress, necessitating lifelong surveillance [37].

Clinical consequences are systemic and clinically significant: iron deficiency anemia reduces functional capacity; vitamin B12 deficiency contributes to neurological dysfunction; and calcium and vitamin D deficiency drive increased bone turnover, osteopenia, and elevated fracture risk, with documented reductions in bone mineral density after surgery [37,38]. Additional deficits in fat-soluble vitamins and trace elements further impair immune competence and metabolic function. These risks underpin ASMBS and BOMSS recommendations for routine biochemical monitoring and lifelong supplementation with multivitamins, iron, calcium, vitamin D, and vitamin B12, though long-term adherence remains challenging [26,37].

The contrast with pharmacologic weight-loss pathways is striking: bariatric care embeds standardized nutritional surveillance and supplementation, whereas GLP-1–based therapy lacks comparable frameworks despite overlapping physiological consequences. This disparity supports extending bariatric principles of structured monitoring and supplementation to patients undergoing GLP-1–mediated weight loss.

Monitoring Frameworks and Risk Stratification

The management of rapid weight loss requires structured monitoring frameworks that extend beyond weight-centric metrics to include nutritional status, body composition, and functional reserve. Given the established risk of nutritional compromise across both pharmacologic and surgical modalities, systematic surveillance is essential to ensure safe and sustainable outcomes [32].

Bariatric surgery provides a well-defined model for such monitoring. Guidelines from ASMBS and BOMSS mandate comprehensive biochemical assessment beginning in the preoperative period and continuing lifelong, reflecting the predictable trajectory of nutritional risk [26,27]. Monitoring protocols are aligned with clinically relevant parameters, including iron indices, vitamin B12, calcium, vitamin D, and, where appropriate, parathyroid hormone, enabling early identification of deficiency and targeted intervention [8,20]. Recommended intervals include assessment every three to six months during the first year and annually thereafter [27].

Conversely, structured monitoring for GLP-1–based therapy remains insufficiently integrated into clinical practice. Emerging expert consensus supports baseline nutritional assessment followed by periodic evaluation of key micronutrients and protein status, particularly in individuals experiencing significant or sustained weight loss [39]. Adoption of such protocols represents a necessary extension of existing pharmacologic care pathways.

Body composition assessment provides an important adjunct to biochemical monitoring. Techniques such as DEXA enable quantification of lean and fat mass, offering insight into qualitative changes in body composition that are not captured by total weight alone [19]. Incorporation of these measures facilitates early identification of sarcopenia and informs targeted nutritional and exercise interventions.

Risk stratification enables tailoring of monitoring intensity. Individuals undergoing malabsorptive bariatric procedures represent the highest-risk group and require intensive surveillance. However, patients receiving GLP-1 therapy may similarly warrant closer monitoring in the presence of rapid weight loss, reduced intake, advanced age, or comorbid conditions [19].

In the Indian context, high baseline prevalence of micronutrient deficiency necessitates routine baseline screening and lower thresholds for ongoing monitoring and supplementation [10,11] . In resource-constrained settings, simplified laboratory panels and integration into primary care pathways may enhance feasibility, while preserving the core principle that nutritional monitoring is integral to obesity management.

Collectively, a unified, risk-stratified monitoring approach across both surgical and pharmacologic pathways enables early detection of deficiency, timely intervention, and preservation of the metabolic benefits of weight loss.

Evidence-Based Supplementation Strategies: Core Nutrients

An evidence-based supplementation framework for rapid weight loss should parallel established bariatric guidance while integrating emerging data from GLP-1–treated populations. Core principles include early initiation, risk- and procedure-specific tailoring, and sustained adherence across the course of weight loss and maintenance.

Protein is foundational for preserving lean mass in the context of rapid, catabolic weight loss. GLP-1– and surgery-induced weight reduction is consistently associated with substantial loss of fat-free mass, driven by increased proteolysis and reduced muscle protein

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.

synthesis [19]. Bariatric and nutrition guidelines support protein intakes of 1.0–1.5 g/kg/day, with higher targets of 1.2–2.0 g/kg/day increasingly advocated in GLP-1–treated and high-risk patients to mitigate sarcopenia and maintain function[40]. Given frequent shortfalls due to appetite suppression and gastrointestinal intolerance, high-quality protein supplements (e.g., whey, casein, or equivalent alternatives) are often required to meet these thresholds [41].

Targeted anabolic support can further optimise muscle preservation. Leucine, via mTOR activation, and its metabolite β-hydroxy β-methylbutyrate (HMB) have demonstrated attenuation of muscle protein breakdown and improved lean mass retention in catabolic settings, supporting their use as adjuncts during rapid weight loss[42]. Creatine, through augmentation of intramuscular phosphocreatine stores, enhances ATP availability, strength, and lean mass, particularly when combined with resistance training—an approach that may be especially valuable in GLP-1–treated individuals at risk of functional decline [43] .

Micronutrient supplementation remains a cornerstone of bariatric care and is directly applicable to pharmacologic weight loss. Routine use of a complete multivitamin–mineral preparation, together with targeted vitamin D, calcium, iron, and vitamin B12, is strongly recommended in bariatric guidelines due to high prevalence and clinical impact of deficiencies [8,26]. Vitamin D deficiency is ubiquitous and exacerbated by reduced intake and pre-existing insufficiency; supplementation supports bone and muscle health but may not fully prevent bone mineral density loss. Calcium is essential to counteract negative calcium balance and bone resorption, particularly in malabsorptive states [28].

Iron deficiency is frequent across both bariatric and GLP-1 pathways, driven by reduced intake, impaired absorption, and increased physiological demands; declines in ferritin and hemoglobin in GLP-1 cohorts emphasize the need for individualized oral or parenteral iron strategies [31]. Vitamin B12 deficiency, arising from reduced gastric acid, intrinsic factor–dependent absorption, and suboptimal intake, warrants routine monitoring and oral or parenteral replacement, especially in vegetarian and Indian populations with high baseline risk [44]. Additional micronutrients such as zinc, selenium, and fat-soluble vitamins contribute to immune and metabolic function and are appropriately covered within comprehensive multivitamin regimens recommended by bariatric societies [27].

Overall, supplementation should begin early, be guided by baseline status and anticipated risk, and be adapted to treatment modality and patient characteristics. Extending bariatric-style, protocolized supplementation strategies to GLP-1–treated patients represents a necessary evolution to safeguard metabolic benefits while preventing avoidable nutritional compromise [8]. A consolidated comparison of nutritional risk, deficiency profiles, and evidence-based supplementation strategies across GLP-1–based therapies and bariatric surgery is summarized in (Table 1).

Targeted Nutraceuticals and Adjunctive Therapies

Omega-3 Fatty Acids

Omega-3 fatty acids provide complementary metabolic and anti-inflammatory benefits. Meta-analytic data show dose-dependent reductions in triglycerides, supporting their role in cardiovascular risk modification during weight loss. Additional effects on inflammation and lipid metabolism further justify omega-3 supplementation in patients with elevated cardiometabolic risk [50,51].

Fiber Supplements and Probiotics

Rapid weight loss often reduces fiber intake due to lower food volume and tolerance, contributing to constipation and microbiota perturbation. Soluble fiber supplementation improves bowel regularity and may enhance satiety and glycemic control [47]. Probiotics can favourably modulate gut microbial composition, with emerging evidence for benefits in metabolic regulation and immune function [48].

Integrating GLP-1 and Bariatric Frameworks: A Practical Algorithm

The evolving obesity-treatment landscape supports a shift from modality-specific pathways to an integrated metabolic framework in which pharmacologic and surgical strategies lie on a single continuum. Both GLP-1 RAs and bariatric surgery converge on shared mechanisms—reduced intake, altered gastrointestinal physiology, and systemic metabolic adaptation—yet only bariatric care is underpinned by structured nutritional protocols [52]. Bariatric guidelines from ASMBS and BOMSS provide comprehensive models of baseline assessment, scheduled monitoring, and lifelong supplementation [26,27], whereas ADA, Endocrine Society, and AHA/ACC/TOS guidance largely prioritizes metabolic outcomes and under-addresses nutritional surveillance, despite rising evidence of deficiency in GLP-1–treated cohorts [21-23]. This divergence is increasingly incongruent with data showing comparable weight-loss magnitude and physiological impact between pharmacologic and surgical approaches [2,3].

An integrated algorithm should begin with a comprehensive baseline assessment, including diet history, micronutrient biochemistry, and, where feasible, body composition analysis. Baseline deficiencies are common across both groups and are particularly prevalent in Indian populations, where vitamin B12, iron, and vitamin D deficiency are highly prevalent [35]. Subsequent risk stratification then guides monitoring intensity: malabsorptive bariatric procedures occupy the highest-risk tier [27], but GLP-1–treated patients may transition into higher-risk strata with rapid or sustained weight loss, marked intake reduction, advanced age, frailty, or chronic disease [32].

Monitoring intervals can reasonably mirror bariatric protocols, three to six monthly reviews in the first year, followed by annual surveillance[27] with emerging consensus supporting similar schedules in GLP-1–treated individuals experiencing substantial or prolonged weight loss [49]. Supplementation should be proactive and individualized: core use of multivitamins, vitamin D, calcium, iron, and vitamin B12 in high-risk patients, titrated against biochemical results, with lower thresholds for initiation in GLP-1 cohorts given the rapid emergence of deficiencies [29]. Integration of body composition metrics is essential, as DEXA data show a substantial proportion of GLP-1–mediated weight loss derives from fat-free

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.

Table 1: Comparative Nutritional Risk, Deficiency Profiles, and Evidence-Based Supplementation Strategies in GLP-1/GIP–GLP-1 Therapy and Bariatric Surgery

Domain	GLP-1 / GIP–GLP-1 Therapy	Bariatric Surgery	Clinical Consequence	Evidence-Based Strategy
Primary mechanism of Nutritional risk [1]	Reduced caloric intake due to central satiety, delayed gastric emptying	Reduced intake + anatomical malabsorption (procedure-dependent)	Quantitative undernutrition vs combined undernutrition + malabsorption	Early nutritional assessment and anticipatory supplementation
Protein intake & lean mass [19,40]	Reduced intake → inadequate protein despite preserved absorption	Reduced intake ± impaired digestion/absorption	Loss of lean body mass, sarcopenia, functional decline	≥1.2 g/kg/day (up to 1.5–2.0 g/kg/day in high-risk); protein supplementation if intake inadequate
Muscle preservation adjuncts [45]	No direct evidence; extrapolated from sarcopenia literature	Limited direct evidence; similar extrapolation	Accelerated muscle loss in high-risk patients	Creatine (3–5 g/day), HMB (1.5–3 g/day) in selected populations
Iron deficiency [46]	Reduced intake, food aversion (especially red meat)	Impaired absorption (duodenal bypass, hypochlorhydria)	Iron deficiency anemia	Targeted screening and supplementation (oral or IV based on severity)
Vitamin B12 deficiency [44]	Reduced intake + metformin interaction	Impaired intrinsic factor-mediated absorption	Neuropathy, anemia	Monitoring and replacement (oral/high-dose or parenteral)
Folate deficiency [37]	Reduced intake	Reduced intake ± absorption	Megaloblastic anemia	Multivitamin or targeted supplementation
Vitamin D and Calcium [28]	Reduced intake	Reduced intake + impaired absorption	Bone loss, secondary hyperparathyroidism	Vitamin D + calcium (prefer calcium citrate post-surgery)
Fat-soluble vitamins (A, E, K) [27]	Rare; intake-related	High risk in malabsorptive procedures	Vision impairment, coagulopathy	Targeted supplementation based on procedure and labs
Thiamine (Vitamin B1) [27]	Reduced intake + vomiting (rare but important)	Vomiting + malabsorption	Neurological complications (Wernicke's)	Early recognition; urgent supplementation in symptomatic patients
Gastrointestinal tolerability [47,48]	Nausea, vomiting, constipation → reduced intake	Dumping, intolerance, altered digestion	Poor dietary adherence, worsening deficiency	Fibre (5–15 g/day), probiotics (symptom-based)
Overall nutritional approach [49]	Intake-driven deficiency risk	Combined intake + absorption-driven risk	Multisystem deficiency risk	Risk-stratified monitoring + individualized supplementation

mass, enabling early recognition of sarcopenia and timely nutritional and exercise intervention [5,32].

Contextual adaptation is crucial in India, where high baseline micronutrient deficiency and variable protein intake amplify risk [35,53] . Here, baseline screening carries heightened priority, empiric supplementation may be appropriate even without overt biochemical deficiency, and simplified monitoring panels integrated into routine practice can enhance feasibility. A unified, risk-stratified algorithm spanning GLP-1 therapy and bariatric surgery thus aligns care with emerging evidence, embedding structured monitoring, early supplementation, and body composition assessment to preserve the metabolic benefits of weight loss while minimizing nutritional harm.

Special Clinical Scenarios

The use of weight-loss therapies in heterogeneous clinical populations introduces additional complexity, as comorbidities, physiological states, and demographic factors modify both efficacy and safety. In these settings, nutritional management assumes heightened importance to preserve physiological reserve alongside metabolic benefit.

In heart failure, GLP-1 RAs may improve metabolic and cardiovascular profiles but operate within a complex pathophysiological milieu characterized by altered energy metabolism and, in advanced stages, cardiac cachexia with skeletal muscle wasting [54,55]. Rapid weight loss in this setting may further exacerbate loss of lean body mass unless protein intake, anabolic support, and resistance exercise are optimized, with concurrent attention to fluid balance and electrolyte homeostasis, particularly in patients receiving diuretic therapy.

Frailty represents a high-risk state in which low physiological reserve, baseline sarcopenia, and micronutrient deficits converge. In older adults, rapid weight loss can precipitate functional decline unless protein intake, vitamin D status, and structured resistance training are actively intensified [18].Women of child-bearing potential require additional precaution: bariatric guidance recommends deferring pregnancy until weight stabilizes and nutritional adequacy is confirmed, and analogous caution is warranted with GLP-1 therapy given the potential impact of folate, iron, and vitamin B12 deficiency on fetal development. Preconception counseling and targeted supplementation are essential [56,57].

In the Indian context, vegetarian dietary patterns, variable protein intake, and high baseline prevalence of vitamin B12, iron, and vitamin D deficiency magnify nutritional risk during GLP-1–induced appetite suppression or post-bariatric restriction [35]. Socioeconomic constraints and variable access to nutrient-dense foods further reinforce the need for proactive supplementation and culturally tailored dietary counseling.

Implementation in Multidisciplinary Care

Effective implementation of nutritional strategies in rapid weight loss requires a multidisciplinary model that integrates medical, nutritional, and behavioral expertise. The complexity of nutritional risk across GLP-1–based therapy and bariatric surgery exceed the scope of single-discipline care, necessitating coordinated involvement of physicians, dietitians, nurses, and allied health professionals.

Clinicians are responsible not only for initiating pharmacologic or surgical treatment but also for longitudinal oversight of nutritional status, including baseline assessment, interpretation of biochemical

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.

results, and dynamic adjustment of supplementation. Dietitians operate these strategies by translating guidance into individualized meal plans, troubleshooting intake barriers, and reinforcing adherence through structured follow-up.

Behavioral support is integral. Appetite suppression, gastrointestinal symptoms, and lifestyle constraints frequently undermine adherence to diet and supplementation. Targeted interventions such as counseling, motivational interviewing, and digital tools help sustain behavioral change and optimize long-term adherence.

In the Indian context, implementation must account for variable access to specialists, resource limitations, and heterogeneous care pathways. Pragmatic solutions include embedding nutritional care in primary practice, leveraging telemedicine, and using community-based programs to extend reach and continuity.

Patient education remains pivotal. Individuals undergoing rapid weight loss should clearly understand their risk of nutritional deficiency and the rationale for monitoring and supplementation. Informed, engaged patients are more likely to participate proactively and achieve durable clinical benefits.

Knowledge Gaps and Future Research Agenda

Despite major advances in obesity therapeutics, the evidence base for nutritional management remains incomplete, particularly for GLP-1–based weight loss. Bariatric surgery is supported by extensive data on mechanisms, prevalence, and treatment of deficiencies, whereas evidence for GLP-1 therapies is comparatively nascent.

A key gap is the absence of randomized controlled trials testing structured supplementation in GLP-1–treated populations. Current practice largely extrapolates from bariatric cohorts or observational data, limiting the precision of guidance. Future trials should evaluate protein supplementation, micronutrient replacement, and targeted nutraceuticals with endpoints that include lean mass preservation, functional capacity, and quality of life, not only weight change.

Long-term nutritional trajectories under prolonged GLP-1 therapy are also poorly defined. Although deficiencies within the first year have been documented [9], their persistence, progression, and clinical consequences over multi-year treatment require clarification. Integration of body composition outcomes, particularly DEXA-derived lean and fat mass changes, is essential to capture qualitative aspects of weight loss beyond total weight alone [19].

In India, population-specific research is a priority. Heterogeneity in dietary patterns, high baseline micronutrient deficiency, and socio-cultural determinants of diet demand locally generated evidence to inform context-appropriate guidelines and algorithms.

Finally, translational and implementation research is needed to bridge evidence and practice. The effectiveness of multidisciplinary care models, digital health solutions, and community-based strategies in improving nutritional monitoring, adherence, and outcomes remains underexplored and should form a core component of the future research agenda.

Conclusion

GLP-1–based pharmacotherapy and bariatric surgery have irrevocably raised the ceiling of what is achievable in obesity care, shifting the field from incremental weight reduction to profound, disease-modifying benefit. However, their true success cannot be measured in kilograms alone. The same interventions that normalize glycemia and reduce cardiovascular risk also reshape body composition and nutrient reserves, with consequences that extend to strength, resilience, and long-term functional independence.

What emerges from the current evidence is a simple but transformative insight: rapid weight loss is not merely a metabolic triumph; it is a nutritionally vulnerable state. Surgical care has already internalized this reality through mandatory monitoring and lifelong supplementation; pharmacologic pathways must now catch up. Treating GLP-1–induced weight loss with the same rigor accorded to bariatric surgery through structured surveillance, early supplementation, and protection of lean mass redefines "best practice" for the next era of obesity medicine.

Nowhere is this shift more urgent than in settings with high baseline micronutrient deficiency and low lean mass, where unguarded weight loss risks exchanging cardiometabolic disease for sarcopenia, frailty, and fracture. The imperative is clear: obesity therapies must be judged not only by the magnitude of weight they remove, but by the strength, function, and quality of life they preserve.

A future-ready obesity paradigm will therefore be one in which every prescription for weight loss is implicitly a prescription for nutritional stewardship where metabolic success and nutritional integrity are pursued as inseparable, co-equal goals.

References

1. Wilding JPH, Batterham RL, Calanna S, Davies M, Van Gaal LF, et al. (2021) Once-Weekly Semaglutide in Adults with Overweight or Obesity. N Engl J Med. 384: 989-1002.
2. Jastreboff AM, Aronne LJ, Ahmad NN, Wharton S, Connery L, et al. (2022) Tirzepatide Once Weekly for the Treatment of Obesity. N Engl J Med 387: 205-216.
3. Arterburn DE, Telem DA, Kushner RF, Courcoulas AP (2020) Benefits and Risks of Bariatric Surgery in Adults: A Review. JAMA 324: 879-887.
4. Sjöström L (2013) Review of the key results from the Swedish Obese Subjects (SOS) trial - a prospective controlled intervention study of bariatric surgery. J Intern Med 273: 219-234.
5. Heymsfield SB, Gonzalez MCC, Shen W, Redman L, Thomas D (2014) Weight loss composition is one-fourth fat-free mass: a critical review and critique of this widely cited rule. Obes Rev Off J Int Assoc Study Obes 15: 310-321.
6. Misra A, Vikram NK, Ghosh A, Ranjan P, Gulati S (2025) India Obesity Commission Members. Revised definition of obesity in Asian Indians living in India. Diabetes Metab Syndr 19: 102989.
7. Prokopidis K, Daly RM, Suetta C (2025) Weighing the risk of GLP-1 treatment in older adults: Should we be concerned about sarcopenic obesity? J Nutr Health Aging 29: 100652.
8. Farhana A, Rehman A (2026) Metabolic Consequences of Weight Reduction. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 Apr 6].
9. Scott Butsch W, Sulo S, Chang AT, Kim JA, Kerr KW, et al. (2025) Nutritional

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.

deficiencies and muscle loss in adults with type 2 diabetes using GLP-1 receptor agonists: A retrospective observational study. Obes Pillars 15: 100186.

10. Johnson BVB, Milstead M, Kreider R, Jones R (2025) Dietary supplement considerations during glucagon-like Peptide-1 receptor agonist treatment: A narrative review. Obes Pillars 16: 100209.

11. Madhu SV, Kapoor N, Das S, Raizada N, Kalra S (2025) ESI Clinical Practice Guidelines for the Evaluation and Management of Obesity in India - An Update (2025). Indian J Endocrinol Metab 29: 355-365.

12. Holst JJ (2007) The physiology of glucagon-like peptide 1. Physiol Rev 87: 1409-1439.

13. Blundell J, Finlayson G, Axelsen M, Flint A, Gibbons C, et al. (2017) Effects of once-weekly semaglutide on appetite, energy intake, control of eating, food preference and body weight in subjects with obesity. Diabetes Obes Metab 19:1242-1251.

14. Chao AM, Paul A, Hodgkins JV, Wadden TA (2024) A Guideline-Directed Approach to Obesity Treatment. Diabetes Spectr Publ Am Diabetes Assoc 37: 281-295.

15. Rosenbaum M, Leibel RL (2010) Adaptive thermogenesis in humans. Int J Obes 34: S47-S55.

16. Baggio LL, Drucker DJ (2007) Biology of incretins: GLP-1 and GIP. Gastroenterology 132: 2131-2157.

17. Arterburn DE, Telem DA, Kushner RF, Courcoulas AP (2020) Benefits and Risks of Bariatric Surgery in Adults: A Review. JAMA 324: 879-887.

18. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, et al. (2019) Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing 48: 16-31.

19. Bikou A, Dermiki-Gkana F, Penteris M, Constantinides TK, Kontogiorgis C (2024) A systematic review of the effect of semaglutide on lean mass: insights from clinical trials. Expert Opin Pharmacother 25: 611-619.

20. Karakasis P, Patoulias D, Fragakis N, Mantzoros CS (2025) Effect of glucagon-like peptide-1 receptor agonists and co-agonists on body composition: Systematic review and network meta-analysis. Metabolism. 164: 156113.

21. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, et al. (2014) AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. Circulation 129: S102-S138.

22. Apovian CM, Aronne LJ, Bessesen DH, McDonnell ME, Murad MH, et al. (2015) Pharmacological management of obesity: an endocrine Society clinical practice guideline. J Clin Endocrinol Metab 100: 342-362.

23. American Diabetes Association Professional Practice Committee for Diabetes*. 8 (2026) Obesity and Weight Management for the Prevention and Treatment of Diabetes: Standards of Care in Diabetes. Diabetes Care. 49: S166-S182.

24. Kristensen SL, Rørth R, Jhund PS, Docherty KF, Sattar N, et al. (2019) Cardiovascular, mortality, and kidney outcomes with GLP-1 receptor agonists in patients with type 2 diabetes: a systematic review and meta-analysis of cardiovascular outcome trials. Lancet Diabetes Endocrinol 7: 776-785.

25. Davies M, Færch L, Jeppesen OK, Pakseresht A, Pedersen SD, et al. (2021) Semaglutide 2·4 mg once a week in adults with overweight or obesity, and type 2 diabetes (STEP 2): a randomised, double-blind, double-dummy, placebo-controlled, phase 3 trial. Lancet 397: 971-984.

26. Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, et al. (2017) American Society for Metabolic and Bariatric Surgery Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient 2016 Update: Micronutrients. Surg Obes Relat Dis Off J Am Soc Bariatr Surg 13: 727-741.

27. O'Kane M, Parretti HM, Pinkney J, Welbourn R, Hughes CA, et al. (2020) British Obesity and Metabolic Surgery Society Guidelines on perioperative and postoperative biochemical monitoring and micronutrient replacement for patients undergoing bariatric surgery-2020 update. Obes Rev Off J Int Assoc Study Obes 21: e13087.

28. Gagnon C, Schafer AL (2018) Bone Health After Bariatric Surgery. JBMR Plus 2: 121-133.

29. Urbina J, Salinas-Ruiz LE, Valenciano C, Clapp B (2026) Micronutrient and Nutritional Deficiencies Associated With GLP-1 Receptor Agonist Therapy: A Narrative Review. Clin Obes 16: e70070.

30. Christensen S, Robinson K, Thomas S, Williams DR (2024) Dietary intake by patients taking GLP-1 and dual GIP/GLP-1 receptor agonists: A narrative review and discussion of research needs. Obes Pillars 11: 100121.

31. Ben-Porat T, Sherf-Dagan S, Côté M, Miner CJ, Buch A (2025) Nutritional Challenges of Incretin-Based Obesity Management Medications: Implications for Clinical Practice. Adv Nutr 16:100522.

32. Neeland IJ, Linge J, Birkenfeld AL (2024) Changes in lean body mass with glucagon-like peptide-1-based therapies and mitigation strategies. Diabetes Obes Metab 4:16-27.

33. Heymsfield SB, Wadden TA (2017) Mechanisms, Pathophysiology, and Management of Obesity. N Engl J Med. 376: 254-266.

34. Kurpad AV, Varadharajan KS, Aeberli I (2011) The thin-fat phenotype and global metabolic disease risk. Curr Opin Clin Nutr Metab Care 14: 542-547.

35. Venkatesh U, Sharma A, Ananthan VA, Subbiah P, Durga R (2021) CSIR Summer Research training team. Micronutrient's deficiency in India: a systematic review and meta-analysis. J Nutr Sci 10: e110.

36. van der Klaauw AA, Farooqi IS (2015) The hunger genes: pathways to obesity. Cell 161:119–132.

37. Stein J, Stier C, Raab H, Weiner R (2014) Review article: The nutritional and pharmacological consequences of obesity surgery. Aliment Pharmacol Ther 40: 582–609.

38. O'Leary F, Samman S (2010) Vitamin B12 in health and disease. Nutrients 2: 299-316.

39. Chavez AM, Carrasco Barria R, León-Sanz M (2025) Nutrition support whilst on glucagon-like peptide-1 based therapy. Is it necessary? Curr Opin Clin Nutr Metab Care 28: 351-357.

40. Bauer J, Biolo G, Cederholm T, Cesari M, Cruz-Jentoft AJ, et al. (2013) Evidence-based recommendations for optimal dietary protein intake in older people: a position paper from the PROT-AGE Study Group. J Am Med Dir Assoc 14: 542-559.

41. Deutz NEP, Bauer JM, Barazzoni R, Biolo G, Boirie Y, et al. (2014) Protein intake and exercise for optimal muscle function with aging: recommendations from the ESPEN Expert Group. Clin Nutr 33: 929-936.

42. Wu H, Xia Y, Jiang J, Du H, Guo X, et al. (2015) Effect of beta-hydroxy-beta-methylbutyrate supplementation on muscle loss in older adults: a systematic review and meta-analysis. Arch Gerontol Geriatr 61:168-175.

43. Wang Z, Qiu B, Li R, Han Y, Petersen C, et al. (2024) Effects of Creatine Supplementation and Resistance Training on Muscle Strength Gains in Adults <50 Years of Age: A Systematic Review and Meta-Analysis. Nutrients 16: 3665.

44. Singla R, Garg A, Surana V, Aggarwal S, Gupta G, et al. (2019) Vitamin B12 Deficiency is Endemic in Indian Population: A Perspective from North India. Indian J Endocrinol Metab 23: 211-214.

45. Rossi G, Bucciarelli L, Mananguite CL, Giovarelli M, Fiorina P (2026) Muscle loss and GLP-1R agonists use. Acta Diabetol 63: 333-342.

46. Almuammar SA, Alzahrani HK (2025) GLP-1 analog therapy and hemoglobin levels: Insights from a retrospective study. Saudi Med J 46: 907-912.

47. van der Schoot A, Drysdale C, Whelan K, Dimidi E (2022) The Effect of Fiber Supplementation on Chronic Constipation in Adults: An Updated Systematic Review and Meta-Analysis of Randomized Controlled Trials. Am J Clin Nutr 116: 953-969.

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.

48. Koutnikova H, Genser B, Monteiro-Sepulveda M, Faurie JM, Rizkalla S, et al. (2019) Impact of bacterial probiotics on obesity, diabetes and non-alcoholic fatty liver disease related variables: a systematic review and meta-analysis of randomised controlled trials. BMJ Open 9: e017995.

49. Mozaffarian D, Agarwal M, Aggarwal M, Alexander L, Apovian CM, et al. (2025) Nutritional priorities to support GLP-1 therapy for obesity: A joint Advisory from the American College of Lifestyle Medicine, the American Society for Nutrition, the Obesity Medicine Association, and The Obesity Society. Obesity 33:1475-1503.

50. Wang T, Zhang X, Zhou N, Shen Y, Li B, et al. (2023) Association Between Omega-3 Fatty Acid Intake and Dyslipidemia: A Continuous Dose-Response Meta-Analysis of Randomized Controlled Trials. J Am Heart Assoc 12: e029512.

51. Mozaffarian D, Wu JHY (2011) Omega-3 fatty acids and cardiovascular disease: effects on risk factors, molecular pathways, and clinical events. J Am Coll Cardiol 58: 2047-2067.

52. Schauer PR, Bhatt DL, Kirwan JP, Wolski K, Aminian A, et al. (2017) Bariatric Surgery versus Intensive Medical Therapy for Diabetes - 5-Year Outcomes. N Engl J Med 376: 641-651.

53. Swaminathan S, Vaz M, Kurpad AV (2012) Protein intakes in India. Br J Nutr 2: S50-S58.

54. Rahmani AR, Dhaliwal SK, Pastena P, Kazakov E, Jayaseelan K, et al. (2025) GLP-1 Receptor Agonists in Heart Failure. Biomolecules 15: 1403.

55. Wang W, Green D, Ibrahim R, Abdelnabi M, Pham HN, et al. (2025) Navigating Sarcopenia Risks in GLP-1RA Therapy for Advanced Heart Failure. Biomedicines 13: 1108.

56. Różańska-Walędziak A, Walędziak M, Mierzejewska A, Skopińska E, Jędrysik M, et al. (2023) Nutritional Implications of Bariatric Surgery on Pregnancy Management-A Narrative Review of the Literature. Medicina (Mex) 59:1864.

57. Morton A, He J (2025) Pregnancy outcomes following first trimester exposure to semaglutide. Obstet Med. 4: 1753495X251346330.

Citation: Bellani D, Desai A, Anand S, Maldahiar SP, Mishra S, et al. Hidden Nutritional Risks of Rapid Weight Loss: A Clinical and Nutraceutical Framework for Prevention in Pharmacological and Surgical Obesity Treatments. Indian J Nutri. 2026;13(1): 341.