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GLP-1-BASED OBESITY THERAPIES AND THE CRITICAL ROLE OF NUTRITION CARE: PREVENTION OF SARCOPENIA, MALNUTRITION, AND WEIGHT REGAIN – A NARRATIVE REVIEW

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ABSTRACT

Background: GLP-1-based therapies such as semaglutide and tirzepatide produce substantial weight loss, but reduced appetite and gastrointestinal adverse effects may compromise dietary adequacy, lean mass, and long-term weight maintenance. This narrative review examines how nutrition care can prevent sarcopenia, malnutrition, and weight regain during GLP-1-based obesity treatment.

Methods: Literature published from January 2018 to May 2026 was searched in PubMed, Scopus, and Web of Science. Trials, observational studies, systematic reviews, clinical guidance, and mechanistic studies were synthesized thematically without meta-analysis. Findings were interpreted contextually.

Results: Pivotal trials reported mean weight reductions of 14.9% with semaglutide 2.4 mg and up to 20.9% with tirzepatide, while body-composition substudies showed reductions in absolute lean mass; approximately one quarter of weight lost with tirzepatide was lean mass. Key risks include sarcopenia, inadequate protein and micronutrient intake, dehydration, and gastrointestinal intolerance. Reduced intake may create an energy gap and place iron, vitamin B12, folate, vitamin D, and thiamine at risk. Withdrawal studies show substantial weight regain after discontinuation. The proposed framework combines baseline nutritional

screening, GLIM-based diagnosis of malnutrition, individualized energy and protein prescriptions, a nutrient-dense dietary pattern, hydration and micronutrient monitoring, resistance exercise, and behavioral support, adapted for older adults, type 2 diabetes, bariatric surgery, pregnancy, adolescents, and Middle Eastern and North African settings, including Ramadan.

Conclusion: Nutrition care is not adjunctive but essential to GLP-1-based obesity treatment. Prospective trials of nutrition interventions in treated patients are still needed. Dietitian-led assessment, monitoring, and maintenance support can help protect muscle, nutritional status, and long-term outcomes.

KEYWORDS: GLP-1 receptor agonists; semaglutide; tirzepatide; sarcopenia; sarcopenic obesity; malnutrition; lean body mass

1.INTRODUCTION

Obesity is a global public health challenge characterized by excessive or abnormal adiposity that can impair health and quality of life. Its development involves complex interactions among genetic, biological, environmental, socioeconomic, and behavioral factors rather than a simple imbalance between energy intake and expenditure. Obesity is associated with numerous chronic conditions, including type 2 diabetes mellitus (T2DM), cardiovascular disease, certain cancers, osteoarthritis, and other musculoskeletal disorders, contributing to substantial morbidity, premature mortality, and healthcare expenditure [1,2]. Accordingly, obesity management requires a comprehensive, long-term approach that addresses its biological mechanisms, associated complications, and the individual circumstances influencing treatment outcomes. Advances in the understanding of appetite regulation, energy homeostasis, and incretin physiology have contributed to the development of effective pharmacological treatments for obesity. Among these, glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have become increasingly important because of their effects on appetite, satiety, and glucose regulation [3,4]. GLP-1 is an endogenous incretin hormone secreted primarily by intestinal enteroendocrine cells in response to nutrient intake. It stimulates glucose-dependent insulin secretion, suppresses inappropriate glucagon secretion, and influences central pathways involved in appetite and food intake. GLP-1-based medications can therefore reduce hunger and energy intake, while some agents also delay gastric emptying to varying degrees [4]. These mechanisms provide a physiological basis for their therapeutic effects in obesity and T2DM.

Semaglutide, a long-acting GLP-1 RA, has demonstrated substantial efficacy in the treatment of obesity. In the STEP 1 randomized controlled trial, adults with overweight or obesity without diabetes who received once-weekly semaglutide 2.4 mg alongside lifestyle intervention achieved a mean weight reduction of 14.9% over 68 weeks, compared with 2.4% in the placebo group [5]. Tirzepatide, a dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonist, has also produced substantial weight reduction in clinical trials. In SURMOUNT-1, mean weight changes at 72 weeks were −15.0%, −19.5%, and −20.9% with tirzepatide 5 mg, 10 mg, and 15 mg, respectively, compared with −3.1% with placebo [6]. These findings demonstrate the potential of incretin-based pharmacotherapy to improve obesity outcomes. However, differences in study populations, treatment duration, doses, and trial designs mean that results from separate trials should not be interpreted as direct head-to-head comparisons.

Despite their efficacy, GLP-1-based therapies do not eliminate the need for comprehensive obesity care. Obesity is a chronic, heterogeneous condition, and treatment should be individualized according to medical history, comorbidities, nutritional status, preferences, and treatment response [7]. Furthermore, weight reduction does not necessarily indicate that all aspects of health are improving equally. Changes in body composition, dietary intake, gastrointestinal tolerance, physical function, and the sustainability of weight management are also important considerations. Evidence from semaglutide withdrawal research indicates that substantial weight regain can occur after treatment discontinuation, emphasizing the need to consider long-term management rather than focusing exclusively on weight loss during active pharmacotherapy [8].

An important clinical consideration is that reduced appetite and energy intake during treatment may make it difficult for some patients to consume adequate amounts of protein, essential fatty acids, vitamins, minerals, and dietary fiber. Gastrointestinal adverse effects—including nausea, vomiting, diarrhea, constipation, and early satiety—may further restrict food and fluid intake. If these challenges persist, they can increase the risk of nutritional inadequacy and may complicate the preservation of lean mass and physical function during weight reduction [9,10]. These risks should not be interpreted as inevitable consequences of GLP-1-based treatment; rather, they highlight the importance of identifying vulnerable patients and monitoring nutritional status throughout therapy.

Nutrition care is therefore an important component of integrated obesity management. A structured approach can include baseline nutritional assessment, individualized energy prescriptions, adequate protein intake, a nutrient-dense dietary pattern, appropriate hydration,

symptom-directed dietary adjustments, and targeted micronutrient assessment when clinically indicated [9,10]. Resistance exercise and other suitable physical activity may complement nutrition interventions by supporting muscle strength and physical function. Nevertheless, the optimal protein prescription, micronutrient monitoring schedule, and most effective nutrition strategies specifically for patients receiving GLP-1-based therapies remain incompletely established. Some current recommendations are consequently informed by broader evidence from obesity management, clinical nutrition, and muscle-preservation research rather than definitive trials of nutrition interventions in GLP-1-treated populations [9,10].

In addition, effective nutrition care must reflect individual clinical needs and social circumstances. Age, T2DM treatment, previous bariatric surgery, pregnancy planning, and other comorbidities may alter nutritional priorities and the safety of treatment. The feasibility of dietary recommendations is also influenced by food affordability, cultural preferences, food availability, and access to professional support. These considerations are particularly relevant in diverse settings, including the Middle East and North Africa, where dietary patterns, household resources, and healthcare access may differ from those represented in many major clinical trials. Although evidence regarding the efficacy of GLP-1-based therapies continues to expand, further synthesis is needed to clarify how nutrition care can help prevent inadequate intake, identify malnutrition risk, support muscle preservation, and promote sustainable weight management. Nutritional risk should be assessed systematically rather than inferred from body weight alone; for example, the Global Leadership Initiative on Malnutrition (GLIM) criteria emphasize the combined assessment of phenotypic and etiologic factors when diagnosing malnutrition [11]. Integrating such assessment principles into obesity treatment may help clinicians identify individuals who require additional dietetic support.

Therefore, this narrative review examines the nutritional implications of GLP-1-based obesity therapies, with particular emphasis on the prevention and management of reduced dietary intake, malnutrition, loss of lean mass, and weight regain. It reviews the mechanisms and clinical outcomes of these medications, discusses potential nutritional risks and vulnerable populations, and evaluates practical approaches to nutritional assessment, energy and protein prescription, dietary quality, hydration, and micronutrient monitoring [12]. It also identifies current evidence gaps and considers how nutrition care can be adapted to different clinical and socioeconomic contexts. By integrating pharmacological evidence with clinical nutrition principles, this review aims to inform a more comprehensive approach to obesity treatment in

which the goal extends beyond weight reduction to include nutritional adequacy, preservation of physical function, and long-term health.

2. METHODS: Literature Search Strategy

2.1 Review Design

This study was designed as a narrative review to synthesize current evidence on glucagon-like peptide-1 (GLP-1)-based therapies for obesity, with particular emphasis on nutritional adequacy, preservation of lean mass and physical function, prevention of malnutrition, and the maintenance of weight loss after treatment discontinuation. A narrative approach was selected to integrate findings from clinical trials, observational studies, clinical guidance, and relevant mechanistic research, allowing the topic to be discussed across interconnected clinical and nutritional domains [13,14].

2.2 Information Sources and Search Period

The literature search covered publications from January 2018 through May 2026. PubMed, Scopus, and Web of Science were identified as the principal bibliographic databases. The search was intended to identify evidence concerning GLP-1 receptor agonists and dual incretin-based therapies, their effects on body weight and body composition, dietary intake, protein and micronutrient adequacy, gastrointestinal adverse effects, physical function, and weight regain following treatment withdrawal.

2.3 Search Strategy

Search terms were developed around the main concepts of obesity pharmacotherapy, nutritional care, body composition, and long-term weight management. Terms and their combinations included “GLP-1 receptor agonist,” “semaglutide,” “liraglutide,” “tirzepatide,” “obesity,” “nutrition,” “protein intake,” “lean mass,” “muscle loss,” “sarcopenia,” “malnutrition,” “micronutrient deficiency,” “gastrointestinal adverse events,” “weight regain,” and “treatment discontinuation.” Search strategies were adapted to the indexing and search functionality of each database. Reference lists of relevant publications were also considered to identify additional pertinent studies. Transparent reporting of databases, search concepts, eligibility criteria, and reference selection is recommended to strengthen the clarity and reproducibility of narrative reviews [13,14].

2.4 Eligibility Criteria

Publications were considered relevant when they addressed GLP-1-based obesity treatment and one or more of the review’s principal themes: weight-loss efficacy, changes in body composition, preservation of muscle mass or function, energy and protein intake, nutritional

deficiencies, gastrointestinal tolerability, nutrition interventions, or weight maintenance after treatment cessation. Randomized controlled trials, observational studies, systematic reviews, clinical practice guidance, expert consensus statements, and foundational mechanistic studies were considered according to their relevance to the review question.

Publications were excluded when they were unrelated to obesity treatment or nutritional outcomes, duplicated another record, or did not provide sufficient relevant information for the review's objectives. The selection of literature was guided by relevance, study design, clinical applicability, and the contribution of each publication to the synthesis rather than by a formal quantitative pooling process.

2.5 Evidence Synthesis

Findings were organized thematically into sections addressing treatment efficacy, lean-mass and muscle-related outcomes, nutritional inadequacy and malnutrition, nutrition assessment and intervention, physical activity and behavioral support, weight regain, and considerations for special populations. Clinical trial findings were interpreted in the context of their study populations, treatment duration, comparators, and reported outcomes. Where available and relevant, numerical results and measures of uncertainty were retained to avoid obscuring differences between individual studies.

Because this was a narrative review, no meta-analysis or pooled effect estimate was undertaken. The methodological approach was informed by published guidance on narrative-review development and quality assessment, including recommendations to state the review's aims, describe the literature search, support claims with appropriate references, and critically interpret the evidence [13–15].

2.6 Methodological Considerations

The review aimed to provide a clinically relevant synthesis rather than an exhaustive systematic evaluation of every eligible publication. Accordingly, the findings should be interpreted in light of the narrative design and the possibility of selection bias. The search period, information sources, search concepts, and eligibility criteria are reported to make the review process more transparent. The quality of the final manuscript should also be assessed against established considerations for narrative reviews, such as clarity of aims, adequacy of the literature search, appropriate referencing, and critical presentation of evidence [15].

A. Search strategy

Table 1. Search strategy and PICO framework.

Component	***Description***
Review design	*Structured narrative literature review*
Databases	*PubMed/MEDLINE, Scopus, and Web of Science*
Search period	*January 1, 2018–May 31, 2026*
Population	*Individuals with overweight or obesity receiving GLP-1-based pharmacotherapy*
Pharmacological interventions	*Semaglutide, liraglutide, tirzepatide, and relevant investigational incretin-based therapies, including retatrutide*
Primary outcome domains	*Changes in lean mass and muscle function; nutritional inadequacy and malnutrition; weight regain following treatment discontinuation*
Secondary outcome domains	*Gastrointestinal adverse effects, micronutrient status, hydration, dietary protein intake, physical activity, and nutritional monitoring*
Search techniques	*Controlled vocabulary where available, free-text terms, Boolean operators (AND/OR), phrase searching, and supplementary reference-list screening*
Eligible publication types	*Randomized controlled trials, observational studies, secondary analyses, clinical guidelines, consensus statements, and relevant evidence syntheses*
Language	*English*
Synthesis method	*Critical thematic narrative synthesis; no statistical meta-analysis planned*

B. PICO framework

PICO element	***Definition for this review***
P — Population	*Individuals with overweight or obesity receiving GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonist therapy. Relevant subgroups include older adults, individuals with type 2 diabetes, and patients at increased risk of muscle loss or malnutrition.*
I — Intervention / exposure	*GLP-1-based obesity pharmacotherapy, including semaglutide, liraglutide, and tirzepatide, alongside nutritional interventions such as individualized energy and protein prescriptions, dietary counseling, micronutrient monitoring, and resistance exercise.*
C — Comparator	*Placebo, alternative pharmacotherapy, usual care, lifestyle intervention, continued treatment versus withdrawal, or baseline measurements, depending on study design.*

O — Outcomes	*Primary: lean mass and muscle function, nutritional status and adequacy, and weight regain after treatment discontinuation. Secondary: fat mass, dietary energy and protein intake, gastrointestinal tolerability, micronutrient status, hydration, physical function, and cardiometabolic outcomes.*

C. Search concepts and representative keywords.

Search domain	***Representative terms***
GLP-1-based therapies	*"GLP-1 receptor agonist", semaglutide, liraglutide, tirzepatide, retatrutide*
Muscle health	*sarcopenia, "sarcopenic obesity", "lean body mass", "muscle strength", frailty*
Nutritional risk	*malnutrition, "protein intake", "micronutrient deficiency", "nutritional assessment"*
Nutrition care	*"medical nutrition therapy", dietitian, "protein supplementation", "resistance training"*
Weight maintenance	*"weight regain", discontinuation, withdrawal, "weight maintenance"*

3. GLP-1-Based Therapies in Brief: What the Nutrition Clinician Must Know

3.1. Agents compared: semaglutide, liraglutide, tirzepatide, and retatrutide

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) and related incretin-based therapies have transformed obesity pharmacotherapy by producing greater weight reduction than was generally achieved with earlier anti-obesity medications. Their clinical effects, however, differ according to molecular targets, dose, treatment duration, population characteristics, and trial design. Understanding these differences is essential when developing nutritional strategies to preserve muscle, prevent inadequate nutrient intake, and support long-term weight maintenance[5,6,12,16–19].

Semaglutide and liraglutide selectively activate the GLP-1 receptor, whereas tirzepatide activates both glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptors. Retatrutide is an investigational triple agonist targeting GIP, GLP-1, and glucagon receptors. These differences in pharmacological action may contribute to differences in appetite regulation, metabolic responses, and observed weight-loss trajectories [12,16,18,19].

In the STEP 1 trial, adults with overweight or obesity without diabetes who received once-weekly semaglutide 2.4 mg alongside lifestyle intervention achieved a mean body-weight reduction of 14.9% at week 68, compared with 2.4% in the placebo group [5]. In the SCALE Obesity and Prediabetes trial, once-daily liraglutide 3.0 mg produced a mean weight

reduction of approximately 8.0% at week 56, compared with 2.6% with placebo [16]. A direct comparison in STEP 8 further demonstrated greater weight loss with semaglutide 2.4 mg than with liraglutide 3.0 mg at week 68: 15.8% versus 6.4%, respectively [17].

Tirzepatide produced mean weight reductions of 15.0%, 19.5%, and 20.9% at weekly doses of 5, 10, and 15 mg, respectively, over 72 weeks in SURMOUNT-1, compared with 3.1% with placebo [6]. Retatrutide produced a mean weight reduction of up to 24.2% at 48 weeks in a phase 2 obesity trial using a 12-mg weekly dose, compared with 2.1% with placebo [18]. These findings suggest substantial weight-loss potential, but the numerical results should not be interpreted as a definitive ranking across all agents because the trials differed in duration, populations, estimands, and study procedures.

The clinical distinction between approved obesity treatments and investigational therapies is important. Retatrutide's results are based on phase 2 evidence within the specified search window; they should not be presented as equivalent in maturity to the phase 3 evidence available for semaglutide and tirzepatide. Furthermore, the nutritional implications of greater weight loss require evaluation independently of its magnitude, particularly where evidence on muscle function, dietary adequacy, and long-term nutritional outcomes remains limited [20–22].

3.2. Mechanisms relevant to nutrition: appetite, gastric emptying, food preferences, and energy intake

The nutritional consequences of GLP-1-based therapies arise primarily from their effects on appetite regulation, satiation, satiety, and energy intake. GLP-1 receptor activation influences central pathways involved in hunger and food reward while also affecting gastrointestinal function. The resulting reduction in food consumption is a major contributor to pharmacological weight loss [22,23].

Semaglutide and liraglutide have been associated with reductions in hunger, food cravings, and ad libitum energy intake. In a controlled study summarized in the mechanistic literature, semaglutide reduced energy intake at a test meal by approximately 35% relative to placebo, while liraglutide reduced energy intake by approximately 16% in a separate experimental comparison [24]. These findings should not be interpreted as a direct comparison between the two agents because the underlying studies differed in design and treatment conditions.

Gastric emptying is another relevant mechanism. GLP-1 receptor activation can delay gastric emptying, particularly during early treatment and dose escalation, contributing to earlier fullness, prolonged postprandial satiety, nausea, and reduced meal size. However, the magnitude of this effect varies by agent, treatment duration, dose, and individual response.

With sustained exposure to some GLP-1RAs, the gastric-emptying effect may diminish, meaning that delayed gastric emptying should not be assumed to be the sole or dominant mechanism of long-term weight reduction [25,26].

Changes in food preferences may also influence dietary quality. Experimental studies have reported reductions in cravings for certain energy-dense foods and changes in food-reward responses during semaglutide treatment [27]. Although these effects may support reduced energy intake, appetite suppression does not necessarily guarantee adequate protein, vitamin, mineral, or fluid consumption. A patient may consume substantially less food while continuing to select a diet with limited nutrient density.

For nutrition clinicians, the practical implication is that reduced appetite should be assessed not only in terms of weight-loss effectiveness but also in terms of the quality and distribution of the remaining dietary intake. Smaller meals, prioritization of protein-rich and micronutrient-dense foods, and symptom-responsive meal planning may help patients meet nutritional requirements despite reduced appetite. Persistent vomiting, poor oral intake, dehydration, or inability to meet nutritional needs warrants clinical assessment rather than being accepted as an inevitable consequence of successful weight loss [27,28].

Tirzepatide combines GIP and GLP-1 receptor agonism, while retatrutide additionally activates the glucagon receptor. Although these agents produce substantial weight loss, their precise effects on food preferences, gastrointestinal physiology, and nutrient selection should not automatically be assumed to be identical to those of selective GLP-1RAs. Direct nutritional investigations remain important for determining whether differences in receptor activity translate into clinically meaningful differences in dietary adequacy or muscle preservation [29–31].

3.3. Body-composition changes: fat mass versus lean mass—what STEP and SURMOUNT really show

The clinical value of pharmacological weight reduction cannot be evaluated solely by the percentage of total body weight lost. Body composition is also relevant because weight reduction generally involves changes in both fat mass and fat-free or lean mass. The magnitude of these changes varies according to baseline body composition, age, sex, degree of weight loss, physical activity, protein intake, and the method used to measure body compartments [32–34].

An exploratory dual-energy X-ray absorptiometry (DXA) analysis of the STEP 1 trial included 140 participants. At week 68, semaglutide 2.4 mg was associated with a 19.3% reduction in total fat mass and a 9.7% reduction in total lean body mass. The proportion of

total body weight represented by lean mass increased despite the absolute reduction in lean mass, reflecting the greater relative reduction in fat mass [5]. This distinction is essential: an increase in the proportion of lean mass does not mean that absolute lean mass increased.

A DXA substudy of SURMOUNT-1 similarly reported reductions in both fat and lean mass during tirzepatide treatment. At week 72, the pooled tirzepatide groups experienced mean reductions of 33.9% in fat mass and 10.9% in lean mass, compared with reductions of 8.2% and 2.6%, respectively as seen in Table.2, in the placebo group. Approximately 75% of the weight lost in the tirzepatide group was attributable to fat mass and 25% to lean mass [35]. These estimates are derived from a substudy involving 160 participants with available baseline and follow-up DXA measurements, rather than the full SURMOUNT-1 trial population.

Several interpretive limitations must be considered. First, DXA-derived lean mass is not a direct measure of skeletal muscle alone; it includes non-fat tissues and can be influenced by hydration. Second, a reduction in lean mass is not equivalent to a clinical diagnosis of sarcopenia, which requires consideration of muscle strength and, where appropriate, muscle quantity or quality and physical performance [36,37]. Third, body-composition substudies generally involve smaller samples than their parent trials and may not represent all treated patients.

Nevertheless, the findings establish an important clinical point: substantial pharmacological weight loss can reduce absolute lean mass even when the overall body-composition profile improves. For nutrition care, this supports the need to assess muscle function and nutritional adequacy alongside weight, waist circumference, and fat mass. Adequate protein intake, progressive resistance exercise, and individualized monitoring are reasonable components of comprehensive care, although the optimal GLP-1-specific nutritional prescription requires further direct evaluation [36–39].

The available evidence also highlights a research gap. Major obesity trials have primarily emphasized total body weight and cardiometabolic outcomes, while muscle function, dietary protein adequacy, and longer-term consequences of lean-mass changes have been less consistently evaluated. Future trials should incorporate standardized body-composition measurements and functional outcomes to establish which patients are most vulnerable and which interventions best preserve clinically meaningful muscle function during treatment [37–39].

Table 2. Comparative pharmacology, weight-loss efficacy, and body-composition outcomes of selected incretin-based therapies.

Agent	Mechanism	Regimen studied for obesity	Trial and duration	Mean body-weight change	Lean-mass findings
Liraglutide [5,17]	GLP-1 receptor agonist	3.0 mg subcutaneously once daily	SCALE, 56 weeks	−8.0% [16]	No directly comparable lean-mass percentage established from the pivotal trial
Semaglutide [5]	GLP-1 receptor agonist	2.4 mg subcutaneously once weekly	STEP 1, 68 weeks	−14.9% [5]	Total lean body mass decreased by 9.7% from baseline in the DXA substudy [5]
Tirzepatide [6,35]	Dual GIP/GLP-1 receptor agonist	5, 10, or 15 mg subcutaneously once weekly	SURMOUNT-1, 72 weeks	−15.0%, −19.5%, and −20.9%, respectively [6]	Total lean mass decreased by 10.9% from baseline in the DXA substudy; approximately 25% of total weight lost was lean mass [35]
Retatrutide [18]	Triple GIP/GLP-1/glucagon receptor agonist	Investigational weekly subcutaneous doses up to 12 mg in the phase 2 trial	Phase 2 obesity trial, 48 weeks	Up to −24.2% at 12 mg [18]	A directly comparable, adequately established lean-mass percentage was not available from the pivotal phase 2 report

Table 2. The lean-mass percentages represent changes from baseline, not the percentage of total weight lost that consisted of lean mass. These are different measures. For example, the trispartide substudy reported a 10.9% reduction in lean mass from baseline and

approximately 25% of total weight lost as lean mass [35]. The corresponding percentage of weight loss attributable to lean mass should not be inferred from the semaglutide 9.7%.

4. Risk 1: Sarcopenia and Sarcopenic Obesity – The Central Concern

4.1. Definitions: sarcopenia, low muscle mass, impaired function (EWGSOP2, ESPEN/EASO)

Sarcopenia: It is defined as a progressive musculoskeletal disorder, often associated with an increased risk of adverse events such as falls, fractures, physical disability, and mortality [40]. EWGSOP2 Criteria (European Working Group on Sarcopenia in Older People – 2nd Update): According to the criteria established by the European Working Group on Sarcopenia in Older People [41], the diagnosis is reached through three stages:

The first stage involves assessing the likelihood of the condition; low muscle strength is a key indicator, measured via handgrip strength or the "chair-stand" test as seen in (Fig 1) [42,43]. The diagnosis is then confirmed by observing a reduction in muscle quantity or quality, which can be assessed through medical imaging or bioelectrical impedance analysis (BIA). Furthermore, poor physical performance and diminished muscle function are considered in the third stage, which indicates the severity of the condition[44]. Sarcopenic Obesity: According to ESPEN/EASO (European Society for Clinical Nutrition and Metabolism / European Association for the Study of Obesity) guidelines, this condition is characterized by high body fat (obesity) combined with low muscle mass, leading to reduced muscle strength (sarcopenia). It is considered a serious condition because the presence of excess fat can mask the underlying muscle weakness [45].

4.2. Prevalence and Magnitude: The Risk of Weight Loss

Total weight loss can consist of 25% to 40% muscle mass [46]. This often occurs when individuals follow extreme diets or use appetite-suppressing medications—such as GLP-1 receptor agonists—without engaging in physical exercise or adhering to a structured nutritional plan, which negatively impacts overall health [47].

4.3. Groups at Highest Risk

Older adults are particularly susceptible to sarcopenia, as aging is associated with a decline in hormone levels and a reduction in muscle mass [48].Postmenopausal women are also at risk due to hormonal changes and the loss of muscle mass [49]. Furthermore, an insufficient daily protein intake, a lack of physical activity, and the absence of resistance training increase the likelihood of developing sarcopenia. Individuals with a low body mass index (BMI)—who lack adequate muscle reserves—are also at an increased risk [50].

4.4. Clinical and Health Consequences

The loss of muscle mass exacerbates medical issues such as frailty and physical vulnerability, impairing the body's ability to withstand physical demands, stress, and the requirements of daily activities.

Moreover, this loss of muscle can lead to an increased incidence of falls and fractures, often resulting from a compromised sense of balance [51]. Additionally, the reduction in muscle mass leads to a decrease in energy expenditure—lowering the daily metabolic rate—or triggers a state of metabolic adaptation as the body attempts to compensate for an energy deficit [52].

4.5. Assessment and Diagnosis in Routine Practice

Various techniques and tools are employed to diagnose and promptly detect cases. A simple five-question questionnaire, known as SARC-F, is used; it asks the patient about their strength, the level of assistance required for walking, the ability to rise from a chair, the capacity to climb stairs, and the frequency of falls [53]. A handgrip dynamometer is also used to assess hand muscle strength, serving as a reliable indicator of overall body strength [54].

Furthermore, patients are evaluated using a 4-meter gait speed test, as individuals with sarcopenia typically exhibit a decline in physical performance as body composition is assessed using methods such as Bioelectrical Impedance Analysis (BIA) or Dual-energy X-ray Absorptiometry (DXA)—the latter being the gold standard for accurately measuring muscle and fat mass [55]. Additionally, the ratio of serum creatinine (a byproduct of muscle tissue) to serum cystatin C is examined; clinicians consider this an indirect biomarker for total body muscle mass [56].

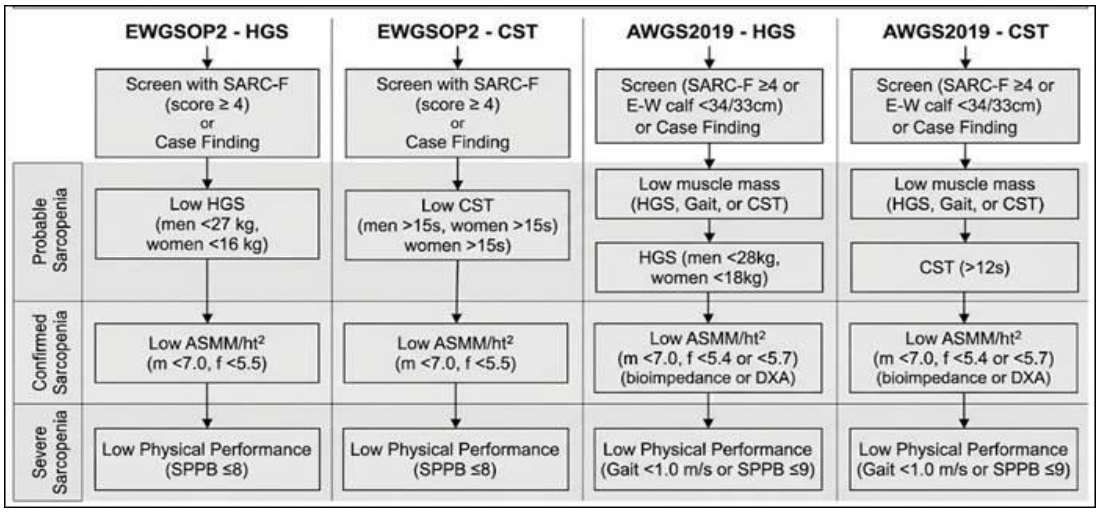


Fig 1. Flowchart outlines the sequential diagnostic algorithms used to identify and categorize sarcopenia severity across four distinct clinical pathways: EWGSOP2 using Handgrip Strength (HGS), EWGSOP2 using the Chair Stand Test (CST), AWGS2019 using HGS, and AWGS2019 using CST Modified from (Verstraeten et al., 2022; EWGSOP2 & AWGS2019 guidelines).

5. Risk 2: Malnutrition, Micronutrient Deficiencies, and GI Intolerance

GLP-1-based obesity therapies can substantially reduce energy intake, improve glycemic regulation, and facilitate clinically meaningful weight loss. However, appetite suppression, early satiety, nausea, vomiting, altered food preferences, and other gastrointestinal adverse effects may also compromise dietary adequacy in some patients. The clinical challenge is therefore not simply to achieve weight reduction, but to ensure that the reduction in energy intake does not result in inadequate protein consumption, micronutrient deficiencies, dehydration, or clinically significant malnutrition [9,11,57–59].

Nutritional risk should not be assumed to occur in every patient receiving semaglutide, liraglutide, tirzepatide, or other incretin-based therapies. Rather, it depends on baseline nutritional status, treatment dose and escalation, the magnitude and speed of weight loss, dietary quality, gastrointestinal tolerance, comorbidities, and the individual's capacity to maintain adequate food and fluid intake. Patients with pre-existing deficiencies, older adults, individuals with low protein intake, and those experiencing persistent gastrointestinal symptoms warrant particular attention.

An important distinction must be maintained between expected appetite reduction and clinically significant nutritional compromise. Reduced hunger may be an intended pharmacological effect, whereas persistent vomiting, progressive dietary restriction, dehydration, or deterioration in physical function requires clinical assessment. Nutritional monitoring should consequently be integrated into obesity treatment from initiation rather than reserved for patients who develop overt complications [9,10,57–59].

5.1. Reduced intake, vomiting, and food aversions: the energy gap

GLP-1-based therapies reduce food intake through effects on appetite, satiety, and, to varying degrees, gastric emptying. These effects may make conventional meal sizes uncomfortable and reduce the frequency of eating occasions. Nausea, vomiting, food aversions, and early satiety may further restrict dietary variety and total energy consumption, particularly during dose escalation [60,61].

An intake of 800–1,200 kcal/day is sometimes discussed in clinical practice as a potential consequence of pronounced appetite suppression. However, this range should not be presented as a universal intake level among GLP-1-treated patients or as an established threshold for malnutrition. Individual requirements vary considerably according to age, sex, body size, activity level, baseline nutritional status, and clinical circumstances. For some adults, prolonged intake at the lower end of this range may be insufficient to support adequate protein, essential fatty acid, vitamin, mineral, and fluid intake.

The relevant clinical concept is the energy gap: the difference between an individual's estimated energy needs and actual intake. For example, if an individual's estimated energy requirement is 2,000 kcal/day and their recorded intake is 1,000 kcal/day, the apparent deficit is 1,000 kcal/day. This is an illustrative calculation, not a recommendation to prescribe a particular intake or a reliable prediction of subsequent weight loss. Energy requirements and actual intake should be assessed individually, and estimates should be interpreted alongside weight trajectory, symptoms, dietary quality, and functional status.

The nutritional significance of reduced intake also depends on food composition. A diet may provide a substantial energy deficit while remaining relatively adequate in selected nutrients, or it may become deficient in both energy and essential nutrients if food selection is limited. Patients who tolerate only small portions may benefit from prioritizing nutrient-dense foods and protein-containing meals before consuming foods with lower nutritional value. Smaller, more frequent meals, slower eating, and avoidance of individual foods that trigger symptoms may improve tolerance, although persistent symptoms require clinical review rather than dietary management alone [60,61].

Particular concern arises when reduced intake occurs alongside recurrent vomiting, diarrhea, or inadequate fluid consumption. In these circumstances, the risk extends beyond an energy deficit to include dehydration, electrolyte disturbances, reduced medication tolerance, and an inability to meet nutritional requirements. A structured dietary history should therefore assess actual food and fluid intake, meal frequency, symptom timing, food avoidance, supplement use, and recent changes in body weight.

5.2. Protein–energy malnutrition, hair loss, fatigue, and immune function

Protein–energy malnutrition is a potential concern when prolonged appetite suppression, gastrointestinal intolerance, or restrictive food choices prevent patients from meeting their nutritional requirements. However, reduced appetite or rapid weight loss alone does not establish malnutrition. Diagnosis requires assessment of nutritional status, weight trajectory, dietary intake, body composition, and relevant clinical findings. The Global Leadership Initiative on Malnutrition (GLIM) framework provides a useful diagnostic approach based on phenotypic criteria, including unintentional weight loss, low body mass index, and reduced muscle mass, together with an etiologic criterion such as reduced intake or disease-related inflammation [11].

In patients receiving GLP-1-based therapy, nutritional compromise may develop when energy intake falls substantially while protein-rich foods become difficult to tolerate or are displaced by a narrow range of preferred foods. The resulting deficit may be particularly

relevant in older adults, individuals with pre-existing muscle depletion, and patients undergoing rapid weight reduction. A nutritional assessment should distinguish the intended reduction in adipose tissue from clinically important deterioration in muscle mass, strength, or physical function [62–64].

Hair shedding and fatigue may indicate inadequate nutritional intake, but neither symptom is specific to protein–energy malnutrition. Hair loss may be associated with rapid weight reduction, inadequate protein or energy intake, iron deficiency, thyroid dysfunction, and other conditions. Similarly, fatigue may reflect dehydration, anemia, inadequate energy intake, micronutrient deficiencies, sleep disturbance, or an underlying illness. These symptoms should prompt a targeted clinical assessment rather than automatic attribution to medication use [65,66].

Protein inadequacy is especially important because dietary protein supplies essential amino acids needed for tissue maintenance, immune-cell synthesis, and recovery from illness. Severe protein–energy malnutrition can impair immune function and wound healing; however, direct evidence that GLP-1-based therapies independently cause clinically significant immune impairment through protein deficiency remains limited. The more defensible concern is that sustained inadequate intake may increase nutritional vulnerability, particularly in individuals who already have poor dietary quality or increased physiological requirements [9,10,67].

The practical response is to assess protein intake, meal distribution, dietary variety, recent weight changes, and functional status. When food volume is limited, protein-containing foods should be prioritized within tolerated meals. Oral nutritional supplements may be considered when ordinary foods are insufficient, with the formulation and quantity individualized to the patient's nutritional needs, kidney function, gastrointestinal tolerance, and overall dietary pattern. Persistent hair loss, unexplained fatigue, muscle weakness, or poor wound healing warrants medical evaluation and targeted laboratory investigations rather than empirical high-dose supplementation.

5.3. Micronutrients at risk: iron, vitamin B12, folate, vitamin D, calcium, magnesium, zinc, and thiamine

Micronutrient inadequacy is a plausible consequence of sustained reductions in food intake and dietary diversity during pharmacological weight loss. Individuals with obesity may already have suboptimal micronutrient status before treatment, meaning that baseline nutritional vulnerabilities can persist or become more clinically relevant when energy intake declines. The 2025 joint advisory on nutritional priorities during GLP-1 therapy highlights

iron, calcium, magnesium, zinc, and several vitamins as nutrients requiring attention when dietary intake is substantially reduced [10].

Nevertheless, three distinct concepts must be separated: inadequate dietary intake, biochemical deficiency, and clinically manifest deficiency. Evidence that patients consume less of a nutrient does not automatically demonstrate a corresponding decline in its blood concentration or the development of a clinical deficiency. The incidence of GLP-1-associated micronutrient deficiencies remains insufficiently characterized, and many recommendations are based on nutritional physiology, observational evidence, and established clinical nutrition principles rather than large prospective deficiency-monitoring trials [9,10].

Iron deficiency may arise when intake of iron-rich foods decreases, particularly in individuals with pre-existing low iron stores, menstrual blood loss, or other causes of iron deficiency. Vitamin B12 and folate inadequacy may become relevant when dietary variety declines or when other predisposing conditions are present. Vitamin D and calcium warrant attention because of their roles in bone health, especially in patients experiencing substantial weight loss or at risk of skeletal fragility. Magnesium and zinc intake may also decline when overall dietary intake is persistently inadequate [9,10].

Thiamine deserves particular attention in patients with prolonged vomiting or severely restricted intake. Thiamine stores are limited, and deficiency can develop rapidly in vulnerable individuals. Persistent vomiting accompanied by neurological symptoms, confusion, gait disturbance, or ocular abnormalities requires urgent medical assessment because thiamine deficiency can cause serious neurological complications. Treatment should not be delayed while awaiting laboratory confirmation when clinical suspicion is high as in Table.3 [68].

A targeted approach to micronutrient monitoring is preferable to indiscriminate supplementation. Depending on the clinical presentation, dietary history, comorbidities, and duration of inadequate intake, investigations may include a complete blood count, ferritin and other iron studies, vitamin B12, folate, 25-hydroxyvitamin D, electrolytes, magnesium, renal function, and selected additional tests. Thiamine assessment or empirical treatment may be indicated in patients with prolonged vomiting or other substantial risk factors. Routine measurement of every micronutrient in every patient has not been established as necessary by direct evidence specific to GLP-1 therapy [9,10,68].

Supplementation should be based on documented deficiency, a strong clinical indication, or an individualized assessment of inadequate intake. Excessive supplementation can cause adverse effects, interact with medications, or be inappropriate in patients with renal

impairment or other comorbidities. The objective is to maintain nutritional adequacy while avoiding unnecessary testing and indiscriminate use of high-dose products.

Table 3. Quantitative Evidence on Weight Loss, Body Composition, Dietary Intake, and Weight Regain During GLP-1-Based Obesity Treatment.

Study	Design and sample	Intervention and follow-up	Main quantitative findings	Statistical interpretation
Wilding et al. (2021), STEP 1 [5]	Randomized, double-blind, placebo-controlled trial; 1,961 adults with overweight or obesity without diabetes	Semaglutide 2.4 mg weekly plus lifestyle intervention versus placebo plus lifestyle intervention; 68 weeks	Mean body-weight change was −14.9% with semaglutide versus −2.4% with placebo.	Between-group difference: −12.4 percentage points (95% CI −13.4 to −11.5; $p < 0.001$).
Look et al. (2025), SURMOUNT-1 body-composition substudy [35]	DXA substudy of 160 participants from SURMOUNT-1; pooled tirzepatide groups, n = 124, and placebo, n = 36	Tirzepatide versus placebo; 72 weeks	With tirzepatide, body weight decreased 21.3%, fat mass 33.9%, and lean mass 10.9%. With placebo, corresponding reductions were 5.3%, 8.2%, and 2.6%. Approximately 25% of weight lost was lean mass in both groups.	All reported between-group comparisons were statistically significant at $p < 0.001$. For lean mass, the estimated treatment difference was −8.3 percentage points (95% CI −10.6 to −6.1).
Wilding et al. (2022), STEP 1 extension [8]	Exploratory extension; 327 participants from the original trial	One-year off-treatment follow-up after 68 weeks of semaglutide or placebo and lifestyle intervention	At week 68, mean weight loss was 17.3% (SD 9.3) with semaglutide versus 2.0% (SD 6.1) with placebo. By week 120, the semaglutide group had regained 11.6 percentage points (SD 7.7) of body weight; net weight change from baseline was −5.6% (SD 8.9).	The extension analyses were exploratory. These descriptive results should not be interpreted as a randomized comparison of different weight-maintenance nutrition strategies.

The influence of liraglutide on dietary patterns and nutrient intakes in patients with obesity and prediabetes (2025) [69]	Secondary analysis of a randomized trial in adults with obesity and prediabetes; dietary intake assessed using 24-hour recalls	Liraglutide 1.8 mg/day versus dietitian-supported energy restriction (−390 kcal/day) and a weight-neutral control; 14 weeks	Evaluated changes in energy and nutrient intake, dietary quality, food sources, and the proportion meeting nutrition recommendations.	The abstract confirms the study design and outcomes but does not provide sufficient numerical results to populate a reliable results row with exact means, SDs, confidence intervals, or p-values. These should be extracted from the full article before final submission.

5.4. Dehydration, electrolyte disturbances, constipation, diarrhea, gastroesophageal reflux, and gallbladder risk

Gastrointestinal adverse effects are among the most clinically relevant tolerability concerns associated with GLP-1-based obesity therapies. Nausea, vomiting, diarrhea, constipation, abdominal discomfort, dyspepsia, and reflux-like symptoms may occur, particularly during dose escalation. Most gastrointestinal adverse effects are mild to moderate and may improve over time; nevertheless, persistent or severe symptoms can compromise food intake, hydration, medication adherence, and nutritional status [9,57].

Dehydration and electrolyte disturbances. Vomiting and diarrhea can cause fluid and electrolyte losses, as seen in Table.4, while reduced thirst or a diminished desire to drink may further limit fluid intake in some patients. Clinically significant volume depletion can contribute to hypotension and acute kidney injury, particularly in individuals with pre-existing renal impairment or those taking medications that increase susceptibility to dehydration. Product labeling for semaglutide specifically warns about acute kidney injury associated with volume depletion, especially when gastrointestinal symptoms occur during treatment initiation or dose escalation [58].

Hydration advice should be individualized according to clinical status, environmental conditions, activity, renal function, and any prescribed fluid restriction. Patients experiencing nausea may tolerate small, frequent sips better than large volumes at once. Oral rehydration solutions may be appropriate when clinically significant gastrointestinal fluid losses occur,

but persistent vomiting, reduced urine output, orthostatic symptoms, or suspected electrolyte abnormalities require medical assessment rather than dietary advice alone.

Constipation and diarrhea. Constipation may be associated with altered gastrointestinal motility, reduced food volume, inadequate fluid intake, and lower consumption of fiber-containing foods. Management may include gradual increases in tolerated dietary fiber, adequate fluid intake when medically appropriate, regular physical activity, and an individualized bowel-management plan. Rapidly increasing fiber intake in a patient with marked nausea, abdominal distension, or substantial early satiety may worsen discomfort; therefore, the intervention should be adjusted to symptom severity and tolerance [9,57].

Diarrhea may arise during treatment and can contribute to dehydration and electrolyte loss. Dietary management should prioritize fluid replacement, tolerable foods, and temporary adjustment of foods that worsen symptoms. Persistent diarrhea requires evaluation for medication-related effects and alternative causes, particularly when accompanied by fever, blood in the stool, severe abdominal pain, or evidence of dehydration.

Gastroesophageal reflux and upper gastrointestinal symptoms. Early satiety, nausea, dyspepsia, and reflux-like symptoms may interfere with eating and drinking. Smaller meals, slower eating, avoidance of individually identified triggers, and avoiding lying down immediately after meals may improve tolerance. Large meals and high-fat foods may aggravate symptoms in some individuals. Persistent reflux, dysphagia, recurrent vomiting, or symptoms suggestive of delayed gastric emptying warrant clinical evaluation. GLP-1-based therapies are not appropriate for unsupervised management of suspected severe gastroparesis [9,57,58].

Gallbladder disease and pancreatitis. Gallstones and gallbladder inflammation are important clinical considerations during pharmacological weight loss. Risk may reflect both treatment-associated factors and the magnitude or rapidity of weight reduction. Tirzepatide prescribing information reports acute gallbladder events and recommends appropriate diagnostic assessment when cholecystitis is suspected [59]. Patients should seek prompt medical evaluation for persistent right-upper-quadrant or upper abdominal pain, particularly when accompanied by fever, jaundice, or vomiting.

Severe, persistent abdominal pain, especially if radiating to the back, may indicate acute pancreatitis and requires urgent medical assessment. If pancreatitis is suspected, the prescribing information for tirzepatide directs discontinuation of treatment and appropriate clinical management [59]. These warning signs should not be managed with dietary modification alone.

5.5. Red flags and when to adjust the dose, pause treatment, or refer

The management of nutritional complications requires coordination between the prescribing clinician and the nutrition professional. A dietitian can identify inadequate intake, evaluate dietary quality, provide symptom-responsive meal planning, and monitor nutritional status. However, medication dose adjustment, treatment interruption, and evaluation of serious adverse effects require the involvement of the authorized prescriber and, when necessary, urgent medical services.

Symptoms that warrant prompt clinical review include persistent nausea that prevents adequate intake, recurrent vomiting, prolonged diarrhea, worsening constipation, progressive food avoidance, unintended excessive weight loss, unexplained fatigue, hair loss accompanied by other nutritional symptoms, and deterioration in muscle strength or physical function. These findings should trigger reassessment of dietary intake, hydration, recent weight trajectory, medication escalation, and relevant underlying conditions [9,57].

Urgent warning signs include inability to retain fluids, markedly reduced urine output, fainting or significant orthostatic symptoms, severe or persistent abdominal pain, pain radiating to the back, gastrointestinal bleeding, jaundice, and new neurological symptoms in a patient with prolonged vomiting or severe dietary restriction. These presentations may indicate dehydration, acute kidney injury, gallbladder disease, pancreatitis, gastrointestinal complications, or severe micronutrient deficiency and require urgent medical assessment [58,59,68].

When gastrointestinal symptoms or inadequate intake become clinically significant, the prescriber should review the dose-escalation schedule and consider whether escalation should be delayed, the dose reduced, or treatment temporarily interrupted or discontinued. The appropriate decision depends on symptom severity, duration, hydration status, comorbidities, and the product-specific prescribing information. Patients should not independently change their dose or discontinue prescribed medication without appropriate clinical advice, except where emergency care is required.

The nutrition professional should document the frequency and duration of symptoms, estimated energy and protein intake, fluid consumption, recent weight changes, bowel patterns, supplement use, and relevant laboratory results. Depending on the presentation, assessment may include electrolytes, renal function, a complete blood count, iron studies, vitamin B12, folate, vitamin D, and targeted micronutrient testing. Patients with persistent poor intake, suspected malnutrition, recurrent gastrointestinal intolerance, or complex

nutritional needs should be referred for dietitian-led care and coordinated medical evaluation [9,10].

The overarching principle is that successful pharmacological weight loss should not be pursued at the expense of hydration, nutritional adequacy, or physical function. Early recognition and management of adverse effects may allow treatment to continue safely when appropriate, while timely dose reassessment or referral can prevent avoidable complications.

Table 4. Gastrointestinal adverse effects associated with GLP-1-based therapies: nutritional mechanisms, practical dietary solutions, and referral criteria.

Symptom or risk	Potential nutritional consequence	Nutrition intervention	When to escalate
Nausea and early satiety	Reduced energy, protein, and micronutrient intake	Offer smaller, more frequent meals; prioritize protein-rich foods; avoid large meals and individual food triggers	Persistent symptoms, inadequate intake, or progressive weakness; prescriber review if symptoms interfere with nutrition
Vomiting	Fluid and electrolyte loss; possible thiamine deficiency with prolonged inadequate intake	Encourage small sips of fluids if tolerated; assess intake and symptoms; consider oral rehydration when clinically appropriate	Persistent vomiting, inability to retain fluids, reduced urine output, neurological symptoms, or suspected dehydration requires prompt clinical assessment
Diarrhea	Fluid and electrolyte losses; reduced intake	Maintain tolerated fluids; assess severity and duration; consider oral rehydration when appropriate	Severe or persistent diarrhea, dizziness, reduced urine output, or suspected electrolyte disturbance
Constipation	Discomfort, reduced appetite, and difficulty maintaining intake	Gradually increase tolerated fiber; maintain appropriate fluid intake and physical activity; review constipating medications where relevant	Severe pain, marked abdominal distension, vomiting, or persistent constipation requires medical assessment
Reflux or post-meal discomfort	Meal avoidance and lower dietary variety	Use smaller meals; avoid lying down immediately after eating; modify high-fat foods or other triggers if they worsen symptoms	Persistent symptoms, swallowing difficulty, bleeding, or other concerning features

Very low food intake or multiple food aversions	Energy and protein inadequacy; possible micronutrient deficiencies	Assess actual intake; use nutrient-dense, tolerated foods; consider an appropriate oral nutrition supplement when indicated	Persistent inability to meet needs, suspected malnutrition, rapid weight loss with functional decline, or abnormal laboratory results
Severe or persistent abdominal pain	Potential serious gastrointestinal or biliary adverse event	Do not attempt to manage unexplained severe pain with diet alone	Urgent medical evaluation, particularly for severe persistent pain or pain radiating to the back
Nausea and early satiety	Reduced energy, protein, and micronutrient intake	Offer smaller, more frequent meals; prioritize protein-rich foods; avoid large meals and individual food triggers	Persistent symptoms, inadequate intake, or progressive weakness; prescriber review if symptoms interfere with nutrition

Sources: Almandoz et al. [9]; Gorgojo-Martínez et al. [57]; current product prescribing information for semaglutide and tirzepatide [58,59]. Dietary measures are individualized supportive strategies and do not replace medical evaluation or prescribed treatment.

6. The Core Nutrition Care Framework: Assessment to Intervention

Nutrition care should be integrated into treatment with glucagon-like peptide-1 receptor agonists (GLP-1 RAs) and dual GIP/GLP-1 receptor agonists, such as tirzepatide, from treatment initiation through maintenance and, where appropriate, medication withdrawal. The principal aim is not simply to maximize weight loss, but to improve fat-mass reduction while preserving muscle function, preventing malnutrition, maintaining dietary adequacy, and supporting long-term weight management.

A structured framework should include baseline nutritional risk screening, individualized energy and protein prescriptions, a nutrient-dense dietary pattern, symptom-directed dietary adjustments, and repeated monitoring. The intensity of care should reflect baseline nutritional status, age, comorbidities, medication tolerability, rate of weight loss, and the patient's ability to meet nutritional requirements. Although these principles are supported by clinical nutrition guidance, the optimal monitoring frequency and specific macronutrient targets have not been established in large trials specifically involving patients receiving GLP-1-based obesity therapies. Therefore, recommendations should be presented as individualized clinical targets rather than universal prescriptions [9–11,70,71].

6.1. Baseline Nutrition Assessment: Screening, Diagnosis, Diet History, and Laboratory Evaluation

6.1.1. Initial screening and comprehensive assessment

Nutrition assessment should begin before treatment or as early as possible after initiation. Screening instruments can identify individuals who require more comprehensive assessment, but they should not replace clinical judgment or a diagnostic evaluation.

The Malnutrition Universal Screening Tool (MUST) is suitable for many community and outpatient settings. It evaluates body mass index (BMI), unintentional weight loss, and the effect of acute disease likely to prevent nutritional intake. The Nutritional Risk Screening 2002 (NRS-2002) was developed primarily for hospitalized patients and incorporates nutritional impairment, disease severity, and age. Accordingly, MUST is generally more appropriate for routine outpatient screening, whereas NRS-2002 is particularly relevant when a patient is hospitalized or receiving inpatient care [70,71].

Screening should be followed by a detailed assessment when the patient has recent unintentional weight loss, low BMI, reduced muscle strength, poor dietary intake, persistent gastrointestinal symptoms, a history of malnutrition, or other factors that increase nutritional risk. Assessment should include: Anthropometry and weight history: current weight, height, BMI, waist circumference, previous weight trajectory, recent unintentional weight loss, and the rate of weight reduction after medication initiation; Body composition and muscle function: when feasible, assessment of fat mass, fat-free mass, skeletal muscle mass, handgrip strength, chair-rise performance, or other validated functional measures. Changes in body weight alone cannot distinguish fat loss from muscle loss; Dietary intake: a 24-hour dietary recall, food record, or structured diet history covering total intake, protein sources, fruit and vegetable consumption, fiber, fluids, meal frequency, supplements, alcohol, food avoidance, and dietary restrictions; Medication and symptom history: current agent and dose, dose-escalation schedule, nausea, vomiting, reflux, early satiety, constipation, diarrhea, and the impact of these symptoms on food and fluid intake; Clinical context: comorbidities, renal and hepatic function where relevant, gastrointestinal disorders, prior bariatric surgery, food insecurity, eating-disorder risk, physical activity, resistance-training participation, and medications that may influence nutritional status.

6.1.2. Diagnosing malnutrition using GLIM

The Global Leadership Initiative on Malnutrition (GLIM) framework provides a standardized approach to diagnosing malnutrition after a positive nutrition-risk screen. Diagnosis requires at least one phenotypic criterion and one etiologic criterion [11]. This distinction is

particularly important in patients with obesity. A high BMI does not exclude malnutrition, and a reduction in body weight during pharmacotherapy does not automatically indicate pathological nutritional deterioration. The clinician must establish whether weight loss is intended, whether intake is sufficient, and whether muscle mass or physical function is being compromised.

GLIM also should not be treated as a stand-alone sarcopenia diagnostic tool. Where muscle impairment is suspected, assessment should include muscle strength and, where possible, muscle quantity or quality using validated methods. Sarcopenia and malnutrition overlap but are not interchangeable conditions.

6.1.3. Dietary history and laboratory evaluation

The baseline dietary assessment should identify whether the patient's usual diet can supply adequate protein, essential fatty acids, fiber, vitamins, minerals, and fluids after appetite decreases. Particular attention should be paid to people who already follow restrictive diets, consume little protein, avoid multiple food groups, or report chronic gastrointestinal symptoms.

Laboratory investigations should be risk-based rather than an identical panel for every patient. Depending on the clinical history, reasonable baseline tests may include a complete blood count, electrolytes, renal function, liver enzymes, glucose or HbA1c, and a lipid profile. Ferritin and iron studies, vitamin B12, folate, 25-hydroxyvitamin D, calcium, magnesium, and other tests can be added when dietary history, symptoms, comorbidities, medications, or examination findings indicate a need.

Albumin and prealbumin should not be interpreted as isolated measures of dietary protein adequacy or used alone to diagnose malnutrition; both can be influenced by inflammation and other clinical factors. Likewise, routine measurement of every micronutrient in every patient is not necessarily evidence-based or cost-effective. The purpose of laboratory testing is to identify clinically relevant abnormalities and guide appropriate treatment.

6.2. Energy Prescription: Individualized Deficits Rather Than Fixed Calorie Thresholds

The energy prescription should support clinically meaningful fat loss without creating an excessive energy gap that compromises protein intake, micronutrient adequacy, hydration, or physical function. A fixed 1,200-kcal diet should not be treated as a universal target or minimum for all adults. Energy requirements vary according to body size, age, sex, activity, medical status, treatment response, and the extent to which appetite is suppressed.

A practical approach is to estimate energy requirements using a validated predictive equation or indirect calorimetry when indicated, then individualize the deficit according to the patient's

clinical circumstances and progress. The prescription should be reviewed if weight loss is unusually rapid, intake becomes persistently inadequate, or symptoms interfere with eating. Very-low-energy diets, commonly defined as providing less than approximately 800 kcal/day, should not be used as an unsupervised consequence of medication-induced appetite suppression; they require an appropriate clinical indication and structured professional supervision [9,10]. Three principles are particularly important: Prioritize nutritional adequacy. The plan should provide enough energy to accommodate adequate protein, essential fats, fiber, and micronutrient-rich foods. A diet that produces rapid weight loss but cannot meet these needs is not a successful nutrition intervention; Respond to actual intake, not only the prescribed intake. A patient may be prescribed an appropriate calorie target but consume substantially less because of early satiety, nausea, vomiting, or food aversion. Periodic dietary records can help identify this discrepancy; Adjust the plan over time. Energy needs and appetite can change during dose escalation, weight reduction, and weight maintenance. Reassessment should consider weight trajectory, body composition where available, hunger, fatigue, strength, and dietary adequacy.

For individuals who cannot consume usual meal volumes, the first response should generally be to improve the nutrient density and tolerability of meals rather than to impose a more restrictive diet. Small, regular meals containing a protein source, a tolerated fruit or vegetable, and an appropriate energy source may be more feasible than large meals. Liquid or semi-solid options may help some patients, but should not routinely replace balanced meals when solid foods are tolerated.

Clinical interpretation: There is no single calorie threshold that reliably prevents malnutrition in all patients receiving GLP-1-based therapies. Energy prescriptions should be individualized and monitored, with escalation of nutrition support or review of medication tolerability when intake remains insufficient.

6.3. Protein Prescription: A Cornerstone of Lean-Mass Preservation

Protein is a central component of nutrition care during pharmacologically supported weight reduction because it supplies essential amino acids for muscle protein synthesis and helps meet the body's requirements during an energy deficit. Nevertheless, adequate protein intake alone cannot guarantee preservation of muscle mass. Resistance exercise, sufficient total energy, adequate recovery, and management of disease-related factors are also important [9,10,72,73].

6.3.1. Setting an individualized daily target

A practical starting range of approximately 1.2–1.6 g protein/kg/day may be considered for selected adults undergoing intentional weight reduction when preservation of lean mass is a priority. This range is an extrapolated clinical target, not a dose established by definitive trials in all GLP-1-treated populations. Requirements should be individualized according to age, physical activity, nutritional status, degree of energy restriction, kidney function, and other clinical considerations.

The choice of body weight denominator is important. Calculating protein solely from actual body weight in severe obesity may yield a very high target that is impractical or inappropriate. Adjusted body weight, ideal body weight, or a clinically justified reference weight may be preferable in some cases, according to local dietetic practice and the patient's clinical condition. The calculation method should be documented so that targets can be interpreted consistently at follow-up.

Higher protein targets require particular caution in patients with chronic kidney disease, especially advanced disease not treated with dialysis. In such patients, the prescription should be coordinated with the treating clinician or renal dietitian rather than applying a high-protein weight-loss target automatically [72].

6.3.2. Protein distribution and meal structure

For patients who tolerate several small meals, distributing protein across the day may make the daily target more achievable. A practical approach is to aim for approximately 25–35 g of protein per meal across three or four eating occasions, adjusting portions to individual requirements and appetite. Smaller patients or those with low requirements may need less per meal, whereas larger or highly active individuals may need a different distribution.

Leucine is an essential amino acid involved in signaling pathways related to muscle protein synthesis. A meal containing approximately 2.5–3 g of leucine has been discussed as a potentially useful target in some muscle-nutrition contexts, particularly in older adults. However, this is not a universal requirement for every meal, and the evidence does not establish a specific leucine threshold that prevents muscle loss in patients receiving GLP-1 therapy. A varied diet supplying adequate complete protein and essential amino acids is generally more useful than focusing on isolated leucine targets [72,73].

6.3.3. Selecting protein sources

Protein sources should be selected according to protein quality, nutrient density, gastrointestinal tolerance, dietary preferences, affordability, and the volume of food the patient can comfortably consume. Suitable animal-based sources include eggs, fish, poultry,

lean meat, milk, yogurt, and other dairy products. Dairy foods can also contribute calcium and vitamin B12, while fish, eggs, and meat provide additional nutrients such as vitamin B12, zinc, and iron in varying amounts. Among plant-based options, soy foods, beans, lentils, peas, nuts, seeds, and whole grains can contribute to daily protein intake while supplying other nutrients and, in many cases, dietary fiber. A varied combination of plant and animal protein sources can help improve dietary adequacy and accommodate different dietary patterns [9,10].

For patients experiencing reduced appetite or early satiety during GLP-1-based treatment, priority should be given to protein-rich foods that provide substantial protein in a manageable portion. Examples include eggs, fish, strained yogurt, cottage cheese, tofu, and appropriately portioned protein-rich soups or smoothies. Protein foods can be consumed early in a meal when appetite is greatest. If food intake alone is insufficient, a protein supplement or nutritionally complete oral supplement may be considered following assessment of the patient's dietary intake and clinical needs. Such products should complement, rather than routinely replace, a varied diet [9,10].

Whey and casein are high-quality dairy proteins that can be useful when convenient protein sources are needed. However, product selection should account for tolerance and composition. Individuals with lactose intolerance may tolerate lactose-free milk or yogurt, whey protein isolate, or suitable plant-based alternatives better than conventional milk or some whey concentrates; tolerance varies between individuals and products. Soy protein is a useful plant-based alternative, while blended plant-protein products may provide a practical option for individuals avoiding dairy. Supplementation should be individualized, particularly in patients with chronic kidney disease or other conditions requiring modification of protein intake [9,10].

Importantly, the evidence supporting specific protein-source choices during GLP-1 therapy remains limited. These recommendations draw substantially on established nutrition research and clinical guidance for weight reduction rather than trials directly comparing protein sources in GLP-1-treated populations. Adequate total protein intake should therefore be combined with appropriate resistance exercise and ongoing assessment of muscle strength and function; increasing protein intake alone cannot guarantee preservation of muscle mass [9,10,72,73].

6.3.4. Supplements when food intake is inadequate

Food-first strategies remain preferable where feasible. If reduced appetite prevents the patient from meeting the individualized protein target, a protein supplement may be used to fill the

documented gap. Whey isolate, lactose-free dairy products, soy protein, or another suitable formulation can be considered according to tolerance and preference. Patients with suspected malnutrition may need a broader oral nutrition supplement containing both protein and energy rather than a protein-only product.

Supplementation should not be prescribed solely because a patient uses a GLP-1-based medication. The decision should follow assessment of actual intake, dietary quality, clinical status, and any relevant contraindications. Persistent inability to meet nutritional requirements should prompt reassessment of gastrointestinal symptoms, medication tolerability, and the need for specialist nutrition support.

6.3.5. Protein and resistance exercise

Protein planning should be integrated with an appropriate physical-activity program, including resistance exercise when medically suitable. Resistance training provides a stimulus for muscle adaptation that protein intake alone cannot replicate. Exercise should be adapted for baseline fitness, musculoskeletal limitations, age, and comorbidities. Patients experiencing weakness, dizziness, marked fatigue, or a substantial decline in physical function should be assessed before exercise intensity is increased.

The overall clinical objective is to protect muscle strength and function, not simply to maintain a particular lean-mass measurement. Changes in body composition should be interpreted alongside functional outcomes and the patient's broader clinical condition.

6.4. Dietary Pattern: A Nutrient-Dense, Mediterranean-Type Model Adapted to Low Appetite

A Mediterranean-type dietary pattern offers a useful foundation because it emphasizes vegetables, fruit, legumes, whole grains, nuts and seeds, olive oil and other unsaturated fats, and varied protein sources, while limiting reliance on highly processed foods rich in salt, added sugars, and saturated fats. This pattern should be adapted to individual needs, preferences, affordability, and gastrointestinal tolerance rather than imposed as a rigid template [9,10].

For patients with markedly reduced appetite, the priority is to preserve dietary quality despite smaller portions. Large salads, bulky whole grains, and high-volume meals may be difficult to tolerate during periods of pronounced early satiety. Smaller portions of nutrient-dense foods may be more practical, with fiber increased gradually according to symptoms and fluid intake. Examples include: A small serving of strained yogurt or lactose-free yogurt with oats, fruit, and seeds, as tolerated, eggs, fish, tofu, or poultry paired with a small portion of vegetables and a tolerated starch, lentil or bean dishes introduced in manageable portions

when bloating is not problematic, fruit, soft-cooked vegetables, and modest servings of whole grains to support fiber intake and unsaturated oils, nut butters, or other energy-dense foods in small amounts when additional energy is needed and fat-containing foods are tolerated.

Dietary fat is not inherently undesirable, but large, high-fat meals can worsen nausea or fullness in some patients. Smaller portions and adjustments to meal composition may improve tolerability. Similarly, spicy foods, caffeine, acidic foods, and particular textures should be limited only when they consistently trigger symptoms; unnecessary broad restrictions can reduce dietary variety.

Ultra-processed foods should not be treated as a single category with identical nutritional effects, nor should all convenience foods be automatically excluded. For patients with limited time, appetite, finances, or food access, suitable fortified foods and convenient protein options can help meet requirements. The central goal is a sustainable pattern that supplies essential nutrients and can be maintained during both active weight reduction and weight maintenance.

6.5. Micronutrient Monitoring and Supplementation Protocol

Reduced food intake can lower the amount of vitamins and minerals consumed even when the diet appears balanced in broad terms. The 2025 joint advisory on nutrition during GLP-1 therapy emphasizes baseline assessment, follow-up of dietary intake, and clinically directed investigation of suspected deficiencies [10]. However, evidence does not currently support a universal schedule requiring every patient to undergo a complete micronutrient panel at fixed intervals.

A risk-stratified monitoring plan is therefore more appropriate. The schedule below is a proposed clinical framework for organizing care, not a validated protocol established by a GLP-1-specific randomized trial. Baseline: before treatment or as early as feasible: Review dietary variety, habitual protein intake, restrictive eating patterns, food allergies, food insecurity, supplements, and prior deficiencies; Obtain a baseline complete blood count and other routine tests guided by comorbidities and the clinical indication, such as renal function, electrolytes, liver enzymes, glucose or HbA1c, and lipid profile; Consider ferritin and iron studies, vitamin B12, folate, and 25-hydroxyvitamin D when dietary risk, symptoms, prior results, malabsorption, menstrual blood loss, or other clinical factors warrant testing; Consider magnesium, calcium-related evaluation, zinc, thiamine, or other targeted investigations when persistent vomiting, prolonged inadequate intake, relevant medication use, malabsorption, or specific clinical findings raise concern.

Three months

At approximately three months, review weight trajectory, food records or dietary recall, protein intake, hydration, bowel symptoms, fatigue, weakness, hair loss, and the patient's ability to consume a varied diet. Repeat laboratory tests when baseline results were abnormal, symptoms have emerged, intake remains poor, weight loss has been unusually rapid, or the patient's comorbidities require monitoring. In a clinically stable patient with adequate intake and no specific risk factors, a repeat of every baseline micronutrient test may not be necessary.

Six months

At six months, reassess the overall nutritional pattern, body composition or muscle function where feasible, and adherence to the protein and physical-activity plan. Review whether ongoing appetite suppression or gastrointestinal symptoms are restricting food choice. Repeat targeted laboratory tests according to prior abnormalities, new symptoms, treatment intensity, dietary limitations, and relevant medical conditions.

Twelve months and ongoing treatment

At twelve months, perform a comprehensive review of weight trajectory, dietary adequacy, functional status, medication tolerability, and identified nutrient risks. Continue annual or more frequent testing according to clinical need, rather than assuming that annual testing is sufficient for every patient. Any significant change in health status, persistent vomiting, prolonged low intake, new anemia, neurological symptoms, or suspected malnutrition should trigger earlier assessment.

Supplementation principles

A standard multivitamin and mineral supplement may be considered when dietary variety is persistently limited, but formulation and duration should be individualized. It does not substitute for sufficient energy, protein, or a varied diet. Confirmed deficiencies should be treated with the appropriate nutrient, dose, duration, and follow-up plan. Avoid indiscriminate high-dose supplementation, especially where excess intake may be harmful or where renal, hepatic, or medication-related factors affect safety as in (Fig 3).

Thiamine requires particular attention when vomiting is prolonged or intake is severely restricted. In a patient with persistent vomiting, rapid nutritional deterioration, or neurological signs, urgent clinical assessment is needed; suspected thiamine deficiency should not be managed by waiting for a routine three- or six-month laboratory review.

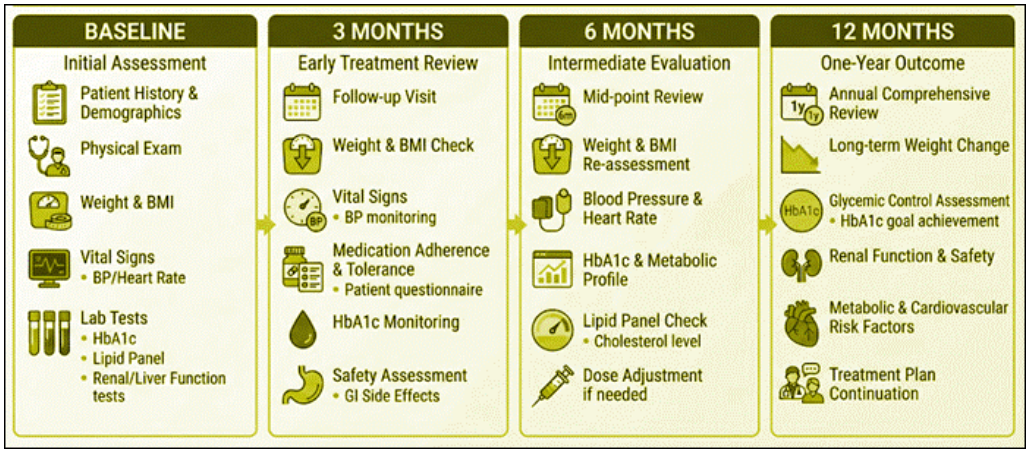


Fig 3. This clinical framework provides a structured 12-month risk-stratified monitoring plan for GLP-1 therapy patients. It is divided into four chronological phases—Baseline Assessment, 3 Months Assessment, 6 Months Assessment, and 12 Months Assessment—focusing on patient safety, metabolic tracking, gastrointestinal tolerance, nutritional adequacy, and targeted laboratory evaluations to prevent deficiencies.

6.6. Hydration, Fiber Titration, and Electrolyte Management

6.6.1. Hydration

A daily fluid intake of approximately 1.8–2.5 L can serve as an initial planning range for many adults, but it should not be interpreted as a universal prescription. Requirements vary with body size, climate, physical activity, pregnancy or lactation, fever, gastrointestinal losses, and medical conditions. Patients with heart failure, advanced kidney disease, or another condition requiring fluid restriction need individualized advice.

Hydration deserves particular attention when nausea, vomiting, diarrhea, or reduced thirst leads to lower fluid consumption. Patients may find small, frequent sips easier to tolerate than drinking large volumes at once. Fluids can be taken between meals if drinking with meals worsens fullness. Where symptoms permit, water and other suitable unsweetened beverages should form the foundation of intake.

Monitoring should include thirst, urine frequency and color, dizziness, postural symptoms, and the ability to maintain normal intake. These observations are not definitive diagnostic tests, but they can help identify patients who require further evaluation. Persistent vomiting, markedly reduced urine output, fainting, confusion, or signs of significant dehydration warrant prompt medical assessment. Oral rehydration solutions may be appropriate for some patients with gastrointestinal fluid losses, but severe dehydration or inability to retain fluids requires medical care rather than dietary advice alone [9,58].

6.6.2. Fiber and bowel function

Fiber can support bowel regularity and long-term dietary quality, but rapid increases may aggravate bloating, abdominal discomfort, or early satiety. The plan should therefore emphasize gradual titration, rather than immediately prescribing a high-fiber diet to a patient who is struggling to eat. A practical sequence is to: Assess the patient's usual fiber intake, bowel pattern, fluid intake, and tolerance of legumes, whole grains, fruit, and vegetables; Increase fiber gradually using tolerated foods, such as oats, fruit, cooked vegetables, or small servings of legumes; Encourage adequate fluid intake when medically appropriate; Reassess constipation, abdominal discomfort, stool frequency, and the effect of fiber on total food intake.

If severe early satiety or marked bloating is present, temporarily emphasizing softer, lower-volume foods may be more practical than increasing bulky foods. Fiber supplements may be considered when food-based strategies are insufficient, but their use should be individualized, with appropriate fluids and consideration of medication timing and gastrointestinal symptoms. Persistent or severe constipation, abdominal distension, severe pain, or vomiting should not automatically be attributed to an ordinary dietary problem.

6.6.3. Electrolytes and escalation of care

Routine electrolyte supplementation is not required simply because a patient is receiving a GLP-1-based medication. Electrolyte testing and replacement should be guided by clinical status, gastrointestinal losses, medication use, renal function, and laboratory results. Vomiting and diarrhea can contribute to volume depletion and electrolyte abnormalities; in susceptible individuals, dehydration can also worsen renal function [58].

The nutrition professional should communicate with the prescriber when symptoms interfere with adequate eating or drinking, or when weight loss is accompanied by progressive weakness, dizziness, persistent vomiting, or evidence of nutritional deterioration. The prescriber should determine whether medication dose escalation should be delayed, the dose adjusted, or treatment interrupted. Severe or persistent abdominal pain, particularly pain radiating to the back, requires urgent assessment for potentially serious causes, including pancreatitis; suspected serious adverse reactions should not be managed by dietary modification alone. The Table.5 is a proposed practice framework. The timing and selection of measurements should be individualized according to baseline risk, clinical findings, treatment stage, and local protocols. Not every patient requires every laboratory test at each visit.

Table 5. Monitoring Checklist for Nutrition Care During GLP-1-Based Obesity Treatment.

Assessment domain	Baseline	Around 3 months	Around 6 months	Around 12 months and ongoing	Supporting references
Weight and anthropometry	Weight, height, BMI, prior weight trajectory; waist circumference where useful	Review weight change and rate of loss	Review trajectory and clinical appropriateness of continued loss	Assess maintenance, continued loss, or regain	[9,10,58]
Nutrition-risk screening	MUST in suitable outpatient settings; NRS-2002 particularly in hospital settings	Repeat if clinical condition or risk changes	Repeat if indicated by new concerns	Continue risk-based screening	[70,71]
Malnutrition assessment	Apply GLIM diagnostic criteria when appropriate after screening	Assess if intake falls, weight loss is concerning, or clinical status deteriorates	Repeat when indicated	Reassess with new symptoms or clinical deterioration	[11,74]
Dietary intake	Diet history, protein sources, food variety, fiber, fluid intake, supplements, and food access	Review actual intake and barriers to meeting needs	Reassess dietary adequacy and tolerability	Review sustainability and dietary adequacy during maintenance	[9,10]
Muscle mass and function	Assess strength and function; consider body-composition measurement when available and useful	Reassess if rapid weight loss, weakness, or functional decline occurs	Repeat if risk persists or findings may change care	Follow longer-term trends where feasible	[10,72,73]
Gastrointestinal symptoms	Document nausea, vomiting, reflux,	Review symptom severity,	Reassess persistent or recurrent	Continue symptom review and reassess	[10,57]

	constipation, diarrhea, and relevant history	duration, and impact on intake	symptoms	after treatment changes	
Routine clinical laboratory tests	Select according to comorbidities, medications, and baseline findings	Repeat selected tests for abnormal results, symptoms, or clinical indications	Review according to prior findings and clinical risk	Continue disease-specific monitoring	[9,10,58]
Micronutrient status	Order targeted tests based on dietary and clinical risk	Repeat targeted tests when indicated	Reassess known deficiencies or ongoing risks	Continue individualized monitoring	[9,10]
Hydration and electrolytes	Review fluid intake, fluid restrictions, and risk factors	Assess symptoms and test when clinically indicated	Repeat if losses, symptoms, or relevant risks persist	Continue risk-based assessment	[10,58,75]
Supplements and nutrition support	Record current products, indications, contraindications, and known deficiencies	Review actual use, tolerance, and ongoing need	Adjust according to intake, laboratory results, and clinical response	Avoid unnecessary supplementation; reassess continued need	[9,10]
Escalation and referral	Identify malnutrition risk, food insecurity, and complex comorbidities	Refer for persistent poor intake, dehydration, or declining function	Escalate unresolved concerns	Review ongoing dietetic and medical support needs	[9–11,57]

The checklist should be implemented as a flexible clinical workflow rather than a rigid testing schedule. In a patient with stable intake, no symptoms, and no relevant abnormalities, a focused review may be sufficient. In contrast, persistent vomiting, rapid weight loss accompanied by weakness, an emerging micronutrient deficiency, or a decline in muscle function justifies earlier and more intensive assessment.

7. Beyond Diet: Exercise and Behavioral Synergy to Preserve Muscle

7.1. Resistance Training Prescription

Physical exercise and specific lifestyle behaviors help maintain muscle mass and prevent atrophy, providing both a scientific and behavioral foundation for stimulating and protecting muscle growth Therefore, it is generally recommended to select exercises that target major muscle groups, while establishing an assessment program that takes into account individuals' fitness levels, physical capabilities, and personal differences [76]. Resistance training serves as the most potent biological signal for the body, facilitating the development of muscle tissue; it is recommended to perform such exercises 2 to 3 times per week [77]. To ensure continuous muscle stimulation and growth, the intensity—whether through increased weight or a higher number of repetitions—should be gradually progressed and determine the right time to intensify your training or extend its duration [78].

Furthermore, the focus should be on the "minimum effective dose" to avoid overexertion and ensure the sustainability of the exercise regimen ,sufficient rest periods should be allocated between training sessions to help muscles recover and rebuild [79].

7.2. The Synergy Between Aerobic Exercise, Walking, and Protein Timing

Combining physical activity with a healthy, well-timed diet supports the process of muscle recovery [80]. Choosing aerobic activities that are appropriate for one's physical condition is one of the best ways to achieve health benefits and sustainable results, moderate aerobic exercise (cardio) promotes cardiovascular health and enhances blood circulation to the extremities without placing excessive strain on the muscles [81].

Additionally, maintaining a consistent daily level of walking (e.g., 7,000–10,000 steps) helps prevent physical inactivity and supports fat burning as the number of daily steps should be increased gradually, based on the individual's capacity; one should not start with an overly ambitious goal that might be difficult to sustain.[82]. It is recommended to consume a variety of protein sources—such as different types of meat, eggs, and legumes—in order to meet the body's nutritional needs and protein intake should be distributed throughout the day—ideally every 3–4 hours, or within 1–2 hours following resistance training—to facilitate muscle protein synthesis (MPS) [83].

7.3. Sleep, Alcohol, Smoking, and Eating Behaviors

Daily habits and environmental factors directly influence the body's hormonal and structural balance,The body requires sufficient sleep—ideally 7–9 hours per night—which facilitates the release of growth hormone and helps prevent elevated cortisol levels It is also advisable to avoid eating heavy meals right before bedtime, as this can exacerbate acid reflux or indigestion in some individuals [84]. Avoiding toxins such as alcohol and smoking supports tissue recovery, promotes

muscle building, enhances oxygen utilization efficiency and improve circulatory health, which in turn supports the ability to engage in physical activity [85]. Adopting a strategy of "mindful eating"—consuming food slowly and paying attention to satiety and fullness cues—is beneficial. Furthermore, distributing daily food intake into smaller, well-spaced meals aids nutrient absorption and prevents indigestion [86]. Gradually adjusting the texture of food can help in facilitate digestion and help avoid gastrointestinal issues and it is advisable to consult with specialists if digestive symptoms persist [87].

7.4. The Role of Health Sciences and Behavioral Support

Health, psychological, and behavioral support play a crucial role in ensuring long-term adherence by helping individuals overcome physical and psychological obstacles through dedicated counseling as it works to enhance the quality of life in a comprehensive way enabling individuals to face setbacks with wisdom [88].

Motivational interviewing—conducted via effective dialogue—helps individuals identify their intrinsic motivations and boosts their self-confidence and it helps in understanding the challenges patients face, as well as the hesitation they may feel toward positive change as in Fig 2, these interviews contribute to the process of transforming personal goals into realistic and sustainable action plans [89].Additionally, smart tracking applications assist in monitoring exercise duration and caloric intake, while also tracking performance and regulating sleep schedules, all of which strengthen an individual's commitment to their goals [90].

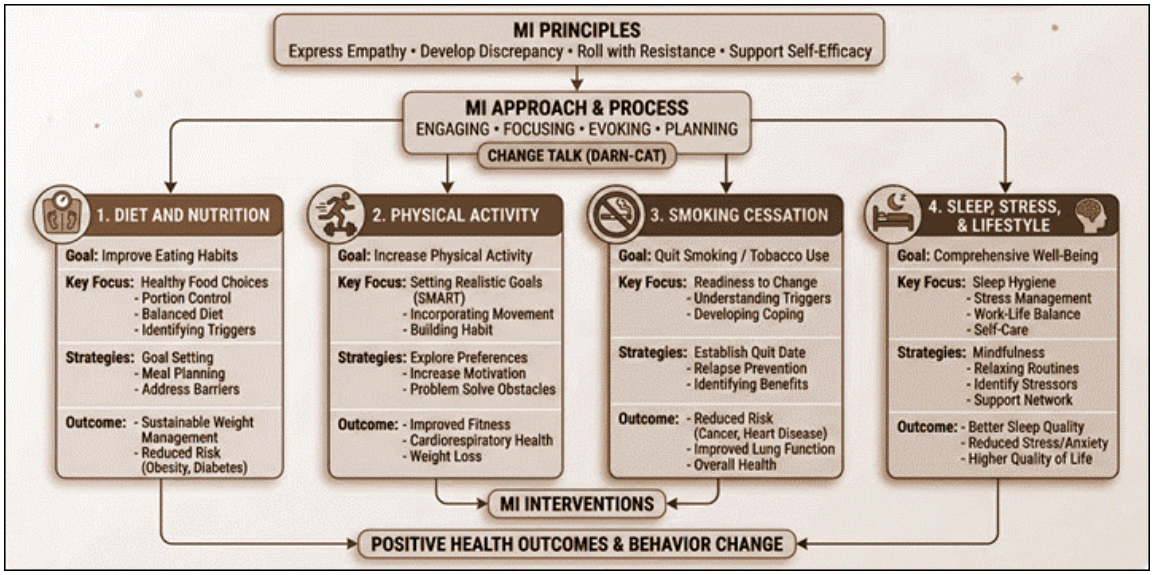


Fig 2. Schematic overview illustrating the multi-domain application of motivational interviewing (MI), encompassing core principles and structured intervention strategies across diet and nutrition, physical activity, smoking cessation, and lifestyle modifications, modified from (Bahri, 2025).

8. Risk 3: Treatment Discontinuation, Weight Regain, and Metabolic Rebound

8.1. What happens after stopping: STEP-1 extension, SURMOUNT-4 withdrawal data – 50-70% regain at 1 year

STEP-1 Extension Study (Semaglutide): Approximately one year after discontinuing treatment, it was observed that two-thirds of the participants had regained about 66% of the weight they had previously lost [8]. While cardiovascular health remained improved compared to baseline, these studies demonstrate that the metabolic changes induced by the medication are not permanent [91]. Research suggests that relying solely on pharmacological intervention is insufficient; long-term, fundamental behavioral lifestyle changes are required, and continued treatment is often necessary to maintain positive health outcomes [92].

SURMOUNT-4 Study (Tirzepatide): In this study, patients who stopped taking Tirzepatide and were switched to a placebo regained, on average, 14% of their body weight by week 36, in contrast to those who continued the medication [93]. It is crucial to monitor patients over the long term—even after initial goals have been met—to prevent weight regain (relapse) [94].

8.2. Mechanisms: appetite return, reduced energy expenditure, loss of food control

The body may return to its previous weight due to a biological defense response, characterized by the resurgence of intense appetite—a phenomenon known as hyperphagia—linked to the decline of the medication's satiety-inducing effects [95]. Furthermore, the absence of the drug's chemical suppression in the brain can lead to a loss of control over food intake and an increased desire to consume larger quantities as weight loss often causes the metabolic rate to slow down, making it more difficult for the body to burn calories efficiently [96]. The brain attempts to restore the body to its previous, higher weight and in the same time disruptions in hormonal balance—particularly involving fat-related hormones like leptin, which is secreted by fat cells and signals satiety—cause the brain to receive constant alerts regarding low energy levels and hunger so the brain will perceive the weight loss as a dangerous state of famine [97].

8.3. Prevention strategy: tapering vs. maintenance dose, structured transition to lifestyle maintenance

A clinical management plan must be established for patients to avoid the shock of "metabolic rebound " This involves a "tapering" strategy, where the maintenance dosage of medication is gradually reduced while keeping the obesity under control so the slow reduction—carried out over several months—allows the brain and digestive system time to adapt [98]. Body composition should be reassessed periodically (using techniques such as InBody or DXA

scans) to ensure that the loss of metabolically active muscle tissue is minimized as regular monitoring of metabolic and vital markers—such as blood glucose levels and hormones that regulate satiety and hunger (e.g., leptin and ghrelin)—facilitates early intervention should any signs of metabolic rebound emerge[99]. And when the medication dosage is reduced, a strict nutritional plan and calorie monitoring should be implemented, accompanied by resistance training to preserve muscle mass and maintain the body's metabolic rate. Additionally, scientifically supported dietary supplements may be used to help control appetite and stabilize weight [10].

8.4. Long-term follow-up model: dietitian-led maintenance program

At this stage, the patient requires ongoing support and a structured approach and this involves regular consultations with a nutritionist to establish a firm, scheduled plan that includes periodic—and eventually monthly—monitoring of vital body indicators. So this ensures a smooth and safe transition from the weight-loss phase to the weight-maintenance phase [100]. A weight increase of up to 2–3 kg is permissible; however, if this limit is exceeded, interventions are required—such as re-evaluating pharmacological options and adjusting the treatment and nutritional plan—to prevent weight regain and a loss of control[101]. A "tapering" protocol should be discussed with the patient in the clinic, alongside the incorporation of physical exercise to preserve muscle mass after discontinuing medication and to prevent relapse [102]. Furthermore, the focus should remain on behavioral and cognitive support, teaching the patient self-management techniques for appetite control, helping them avoid triggers for emotional eating, and instructing them on self-assessment methods to distinguish between psychological hunger and genuine physical hunger [103].

9. Special Populations and Clinical Considerations

Although GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists can provide substantial benefits in obesity management, their nutritional implications differ across populations. Age, diabetes treatment, reproductive status, previous bariatric surgery, and socioeconomic context influence the safety and feasibility of nutrition interventions. The core principles of adequate protein and micronutrient intake, preservation of muscle function, hydration, and symptom management therefore require adaptation to each patient's clinical circumstances. Current guidance emphasizes individualized nutritional care alongside pharmacotherapy rather than a uniform dietary prescription [104,105].

9.1. Older Adults Aged 65 Years and Above: Anabolic Resistance and Fall Risk

Older adults require particular attention because obesity may coexist with age-related muscle loss, reduced physical function, frailty, and multiple chronic conditions. Age-related anabolic resistance—the reduced muscle protein synthetic response to a given protein intake or anabolic stimulus—may make it more difficult to preserve skeletal muscle during an energy deficit. Consequently, weight reduction that is beneficial for metabolic health may also create functional risks if substantial muscle loss occurs or if nutritional intake becomes inadequate [72,106].

The nutrition plan should prioritize adequate energy and protein, distributed across tolerable meals, together with resistance exercise adapted to physical capacity. Protein targets should be individualized according to nutritional status, renal function, activity, and clinical goals; higher protein intake should not be prescribed indiscriminately to people with advanced chronic kidney disease. Soft, nutrient-dense foods and oral nutrition supplements may be useful when chewing difficulties, low appetite, or early satiety restrict food intake. Vitamin D, calcium, and other nutrients should be assessed and supplemented according to clinical need rather than assumed to be deficient in every older patient [72,106].

Monitoring should extend beyond weight and BMI. Relevant outcomes include handgrip strength, chair-rise performance, walking ability, falls, activities of daily living, and body composition when suitable measurement methods are available. New weakness, recurrent falls, declining mobility, or an inability to meet nutritional requirements should trigger reassessment of the weight-loss strategy and consideration of multidisciplinary care. In older adults with frailty or established malnutrition, preventing further nutritional and functional decline may take precedence over pursuing additional weight loss [72,106].

9.2. Type 2 Diabetes: Hypoglycemia, Medication Review, and Glycemic Monitoring

GLP-1-based therapies are widely used in people with type 2 diabetes, but their glucose-lowering effects must be considered alongside other medications and changes in food intake. GLP-1 receptor agonists have a relatively low intrinsic risk of hypoglycemia when used alone; however, the risk can increase when they are combined with insulin or sulfonylureas. When a GLP-1-based therapy is initiated or escalated, the treating clinician should review the existing diabetes regimen and determine whether doses of insulin or insulin secretagogues need adjustment. Patients should not independently change prescribed medication doses [60,104].

Reduced appetite and skipped meals can complicate glycemic management, particularly in patients who continue taking insulin or sulfonylureas without an appropriate treatment plan.

Nutrition counseling should therefore address regular access to suitable meals, recognition and treatment of hypoglycemia, and individualized glucose monitoring. Depending on the medication regimen and clinical circumstances, monitoring may involve capillary blood glucose measurements or continuous glucose monitoring. HbA1c remains useful for longer-term glycemic assessment but does not identify every episode of hypoglycemia [60,107].

Carbohydrate recommendations should emphasize quality, portion awareness, fiber-rich foods, and consistency with the individual's medication regimen rather than indiscriminate carbohydrate elimination. Whole grains, legumes, vegetables, fruit, and other minimally processed carbohydrate sources can be incorporated according to glycemic response, gastrointestinal tolerance, preferences, and affordability. Patients with chronic kidney disease or other diabetes-related complications may require additional modification of protein, sodium, potassium, or fluid intake. The overall plan should integrate glycemic management with the prevention of inadequate energy intake, micronutrient deficiencies, and loss of muscle function [60,104,107].

9.3. Post-Bariatric Surgery, Pregnancy Planning, Lactation, and Adolescents

9.3.1. Post-bariatric surgery

Patients who have undergone bariatric surgery may already have reduced meal capacity, altered nutrient absorption, food intolerance, or established micronutrient deficiencies. If GLP-1-based therapy is considered for persistent or recurrent obesity after surgery, nutritional assessment should account for the specific procedure, time since surgery, current intake, weight trajectory, and previous complications. Existing bariatric follow-up and supplementation requirements remain relevant; pharmacotherapy should not replace them [108].

Particular attention should be given to protein intake, iron, vitamin B12, folate, vitamin D, calcium, and other nutrients indicated by the surgical procedure and clinical history. Persistent vomiting or severely restricted intake warrants prompt evaluation, including consideration of thiamine deficiency. The combination of limited gastric capacity and medication-related appetite suppression may make adequate intake more difficult. Coordination between the obesity-treatment prescriber, bariatric team, and dietitian is therefore advisable.

9.3.2. Pregnancy planning and lactation

Weight-loss pharmacotherapy should not be used as a routine weight-management strategy during pregnancy. Individuals planning pregnancy should discuss treatment discontinuation and alternative management strategies with their prescriber before conception. The required

washout period is agent-specific; for example, US prescribing information recommends discontinuing semaglutide at least two months before a planned pregnancy because of its long half-life. The relevant product information should be checked for the particular medication [104,109].

Obesity medications are generally not recommended during breastfeeding under current obesity-treatment guidance. Decisions should consider the specific agent, product labeling, maternal clinical needs, and potential infant exposure. Nutritional care during pregnancy and lactation should prioritize appropriate maternal and fetal nutrition rather than intentional pharmacologically supported weight loss. Patients who become pregnant while taking a weight-loss medication should contact their healthcare professional promptly to discuss discontinuation and appropriate follow-up [104,109].

9.3.3. Adolescents

Adolescents require age-specific assessment because they are still growing and developing. Adult calorie targets and protein prescriptions should not simply be applied to this population. Care should consider growth trajectories, pubertal development, dietary adequacy, physical activity, psychosocial well-being, and the risk of disordered eating or excessive dietary restriction.

Clinical evidence supports the efficacy of semaglutide for obesity in selected adolescents. In the STEP TEENS randomized trial, adolescents aged 12 to under 18 years with obesity, or overweight with a weight-related condition, received once-weekly semaglutide 2.4 mg or placebo alongside lifestyle intervention for 68 weeks [110]. However, evidence of efficacy does not remove the need for careful growth and nutritional monitoring. Eligibility, prescribing, and follow-up must comply with local regulatory approval and specialist guidance.

Nutrition care should be family-centered when appropriate, emphasizing adequate nourishment, enjoyable physical activity, healthy eating patterns, and body image. Monitoring should include height and weight trajectories, growth and development, dietary adequacy, gastrointestinal adverse effects, and psychological well-being. Pediatric or adolescent obesity specialists and dietitians should be involved where possible.

9.4. MENA and Egyptian Context: Affordability, Dietary Staples, Ramadan, and Local Protein Sources

Nutrition guidance developed in high-income settings may not transfer directly to Egypt and other Middle Eastern and North African (MENA) countries without adaptation. Food prices, household food security, cultural eating patterns, access to dietitians, and the availability and

cost of anti-obesity medication can all influence whether a nutrition plan is feasible. A clinically sound prescription that depends on expensive supplements or foods that are difficult to obtain is unlikely to be sustainable. The World Health Organization has similarly highlighted cost, health-system readiness, and equity as important considerations in the wider implementation of GLP-1-based obesity treatment [105].

9.4.1. Affordability and familiar foods

A practical Egyptian nutrition plan can use locally familiar foods while improving protein adequacy and dietary variety. Depending on household budget, preferences, and clinical requirements, protein sources may include eggs, milk and yogurt, cottage cheese, beans, lentils, chickpeas, fava beans, fish, and poultry. Red meat can be included when affordable and appropriate, but it need not be the primary protein source. Soy foods and commercial protein supplements may be useful for some patients, although their availability, cost, and suitability should be assessed individually.

Traditional staples such as baladi bread, rice, pasta, potatoes, and other cereal-based foods need not be completely eliminated. Instead, portion size, preparation methods, fiber content, and the overall composition of meals should be adapted to the patient's energy needs, glycemic status, appetite, and gastrointestinal tolerance. For example, a meal based on fava beans can be paired with vegetables and an appropriate portion of bread; rice or pasta can be served alongside a protein source and vegetables. These approaches may be more practical than prescribing unfamiliar foods or costly specialty products.

Because GLP-1-based therapies can reduce appetite, patients may eat less without necessarily improving the nutritional quality of what remains in their diet. Counseling should therefore focus on making smaller meals more nutrient-dense, preserving adequate protein, and retaining variety across food groups. When food insecurity limits dietary options, clinicians should recognize that low intake may reflect affordability and access—not simply poor adherence. Nutrition recommendations should be realistic, culturally acceptable, and consistent with the household's resources [10,104].

9.4.2. Ramadan fasting

Ramadan presents a specific consideration for patients using GLP-1-based therapies, particularly those with type 2 diabetes who also take insulin or sulfonylureas. Although GLP-1 receptor agonists generally have a lower intrinsic risk of hypoglycemia than these medications, the risk is not absent when treatment is combined with glucose-lowering agents that can cause hypoglycemia. Changes in meal timing, prolonged fasting, reduced fluid

intake, and gastrointestinal symptoms can further complicate diabetes and nutrition management [60,107].

Patients considering fasting should receive individualized pre-Ramadan assessment. This should include review of diabetes control, hypoglycemia history, kidney function where relevant, medication regimen, prior fasting experience, and the ability to maintain adequate food and fluid intake during non-fasting hours. Medication timing or dosage may require clinician-directed adjustment. Patients should not independently alter insulin or sulfonylurea doses, and those at high risk of complications should be advised about the potential need not to fast in accordance with clinical and religious guidance [9,107]. Persistent vomiting, dehydration, symptomatic hypoglycemia, or inability to consume adequate food and fluids warrants prompt clinical reassessment. Ramadan-specific advice should not be reduced to a fixed meal plan: safe fasting depends on the patient's overall medical status, medications, and individual risk [9,107].

Evidence limitation: Recommendations for Ramadan in this population draw largely on diabetes fasting guidance and general GLP-1 nutrition principles. Direct clinical trials establishing an optimal Ramadan nutrition protocol specifically for patients using GLP-1-based obesity pharmacotherapy remain limited.

10. Research Gaps and Future Directions

Despite rapid growth in the use of GLP-1-based therapies, important uncertainties remain about the optimal nutrition-care strategies to accompany pharmacological weight reduction. Much of the available evidence establishes effects on body weight and metabolic outcomes, whereas evidence for specific dietetic interventions, preservation of muscle function, prevention of micronutrient deficiencies, and long-term weight maintenance is less developed. Future research should distinguish between recommendations extrapolated from general obesity and clinical nutrition research and interventions demonstrated to improve outcomes specifically among GLP-1-treated populations [104,105].

11. CONCLUSION

GLP-1-based therapies have changed obesity treatment by producing large and clinically meaningful weight reductions. However, weight loss alone is not a sufficient measure of success. Body-composition studies show that absolute lean mass can fall alongside fat mass, reduced appetite and gastrointestinal symptoms may limit intake of protein, micronutrients, and fluids, and weight regain is common after treatment is stopped. These risks are not

inevitable, but they justify identifying vulnerable patients early and monitoring nutritional status throughout treatment.

Nutrition care should therefore be an integral component of treatment rather than an optional addition. A practical framework includes baseline screening and GLIM-based assessment, individualized energy and protein prescriptions, a nutrient-dense dietary pattern adapted to low appetite, symptom-directed dietary adjustments, hydration and targeted micronutrient monitoring, resistance exercise, and behavioral support, with a structured plan for maintenance or medication withdrawal. Care should be adapted for older adults, people with type 2 diabetes, pregnancy planning, previous bariatric surgery, and adolescents, and to food access and cultural context, including Ramadan in the Middle East and North Africa.

Many of these recommendations rely on evidence from broader obesity and clinical nutrition research rather than trials in GLP-1-treated populations. Prospective studies with standardized body-composition, functional, and nutritional outcomes are needed to define optimal protein targets, monitoring schedules, and maintenance strategies.

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Author Contributions

Momen Jomaa A. M. Hussein conceived the idea, led the team, and drafted, wrote and edited the manuscript.

Reem Al-Amoudi and Rahaf Al-Amoudi contributed to the writing.

Conflicts

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