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SARCOPENIC OBESITY IN THE ERA OF INCRETIN-BASED THERAPIES: DIAGNOSTIC CHALLENGES, THE RISK OF MUSCLE MASS LOSS, AND PREVENTION STRATEGIES

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ABSTRACT

Introduction and purpose. Sarcopenic obesity (SO) is defined by the coexistence of excess adiposity and reduced muscle mass and function and is associated with disability, frailty, cardiometabolic disease, and increased mortality. This review aims to summarize current evidence on the definition and diagnosis of SO, assess the effects of incretin-based therapies on body composition, with a focus on practical strategies to reduce the risk of clinically relevant muscle loss during obesity treatment.
Material and methods. This manuscript was prepared as a structured narrative review based on publications identified in PubMed/MEDLINE, Google Scholar, and the Cochrane Library.
Results. Current evidence suggests that semaglutide and tirzepatide produce significant and sustained reductions in body weight and fat mass, including visceral adipose tissue, while also being associated with a decrease in absolute lean body mass. These changes should not be interpreted automatically as clinically relevant skeletal muscle loss. However, older adults, frail individuals, and patients with low muscle reserve are more susceptible, especially without adequate protein intake, resistance exercise, and regular monitoring.
Conclusions. Incretin-based therapies represent a major advance in obesity treatment; however, in patients at risk of SO, they should be used within a multimodal strategy aimed at preserving muscle mass and function.

KEYWORDS

Sarcopenic Obesity; Sarcopenia; Obesity; Semaglutide; Tirzepatide; Incretin-Based Therapy

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1. Introduction

To discuss sarcopenic obesity, it is necessary to begin with the two conditions that underlie this phenotype: sarcopenia and obesity. Sarcopenia is currently recognized as a progressive and generalized skeletal muscle disorder characterized by loss of muscle strength and muscle mass, and it is associated with adverse outcomes such as falls, disability, loss of independence, poorer quality of life, and mortality [1,32]. According to the revised consensus of the European Working Group on Sarcopenia in Older People 2 (EWGSOP2), low muscle strength is the key criterion for diagnosis. This condition is considered probable when muscle strength is low, confirmed when low muscle quantity or quality is also present, and classified as severe when poor physical performance coexists [1]. In clinical practice, sarcopenia is recognized not merely as a reduction in muscle mass, but as a disorder of muscle function with important clinical consequences. It occurs predominantly in older adults and is particularly associated with age-related muscle loss, physical inactivity, malnutrition, frailty, disability, multimorbidity, and recent hospitalization [1,20,32].

Obesity is a chronic disease characterized by excessive body weight and associated adverse health effects. It may contribute to cardiometabolic complications, reduced physical function, and poorer long-term outcomes. In adults, it is commonly defined as a body mass index (BMI) of 30 kg/m² or more. However, BMI alone does not adequately reflect fat distribution, muscle mass, or related cardiovascular risk [31]. This limitation becomes particularly important when obesity coexists with impaired muscle health. In such cases, elevated body weight may mask clinically relevant deficits in muscle strength, physical performance, and muscle quality, making risk assessment more difficult when weight-based indices are considered alone [2,4,5,7].

When excess adiposity coexists with reduced muscle mass and impaired function, the condition is referred to as sarcopenic obesity, a phenotype increasingly recognized as clinically distinct rather than merely as the coexistence of obesity and sarcopenia [1,2]. In clinical practice, the diagnosis of sarcopenic obesity remains more difficult than the diagnosis of sarcopenia or obesity alone, mainly because of the absence of

universally accepted diagnostic criteria and the variability in methods used to assess adiposity, muscle mass, strength, and physical performance [2,6,7]. Current evidence indicates that this remains one of the main unresolved challenges in the field. The EWGSOP2 consensus identifies low muscle strength as the key criterion for diagnosis, whereas the European Society for Clinical Nutrition and Metabolism/European Association for the Study of Obesity (ESPEN/EASO) consensus additionally requires the coexistence of excess adiposity and low muscle mass and/or impaired muscle function [1,2]. Compared with older diagnostic approaches based mainly on BMI and appendicular lean mass normalized to height, these criteria provide a more accurate assessment because obesity may mask clinically relevant reductions in muscle strength, impairments in physical performance, and deterioration in muscle quality [2,4,5,7].

Despite this, substantial heterogeneity persists across studies. Different investigations use different measures of adiposity, muscle mass indices, normalization methods, and thresholds for muscle strength and physical performance, resulting in substantial variation in the classification of sarcopenic obesity [2,6,7]. As a consequence, the true scale of the phenomenon remains difficult to determine, even within the same population. In the Canadian Longitudinal Study on Aging, where multiple definitions were applied to the same cohort, the prevalence of sarcopenic obesity ranged from 0.1% to 85.3% in men and from 0% to 80.4% in women [6]. By contrast, a global meta-analysis estimated the pooled prevalence of sarcopenic obesity in older adults at 11% [3]. This discrepancy illustrates how strongly prevalence estimates depend on the diagnostic criteria and assessment methods used. Inconsistent definitions therefore make it more difficult to identify high-risk individuals, select patients for intervention, and compare treatment outcomes across studies [2,5-7].

Current evidence indicates that sarcopenic obesity is associated with a wide range of clinically relevant adverse outcomes. In addition to increased all-cause mortality, it has been linked with a higher risk of cardiovascular disease and related mortality, as well as metabolic disorders, cognitive impairment, arthritis, functional limitation, and respiratory disease. Overall, the available evidence suggests that it should be regarded as a multidisciplinary problem at the intersection of obesity medicine, geriatrics, rehabilitation, clinical nutrition, and cognitive health, rather than merely as an abnormality of body composition [4,5,8].

The clinical relevance of SO has become particularly evident in the era of incretin-based therapies. Semaglutide and tirzepatide belong to the group of incretin-based medications that have been incorporated into obesity treatment and have substantially changed the therapeutic approach to this disease. Semaglutide is a glucagon-like peptide-1 receptor agonist, whereas tirzepatide is a dual glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1 receptor agonist. Large clinical trials have demonstrated that both medications produce marked and sustained reductions in body weight, with an efficacy previously difficult to achieve with non-surgical treatment, representing a major advance in contemporary obesity pharmacotherapy [10,12,15,16,30]. In the STEP 1 trial, semaglutide 2.4 mg once weekly produced a mean weight reduction of 14.9% at 68 weeks, whereas in the SURMOUNT-1 trial tirzepatide produced mean reductions ranging from 15.0% to 20.9% at 72 weeks, depending on dose [10,12]. Their clinical importance extends beyond the magnitude of weight loss alone, because their increasing use has focused attention on the composition of weight loss, particularly the balance between reduction in fat mass and preservation of lean tissue and muscle function in individuals at risk of sarcopenic obesity [11,13-16,30].

2. Material and methods

This manuscript was prepared as a structured narrative review. A focused literature search was conducted in PubMed/MEDLINE, Google Scholar, and the Cochrane Library and was complemented by hand-searching the reference lists of key eligible papers. Publications from 2008 onward were considered. The search strategy included terms related to sarcopenia, sarcopenic obesity, obesity, semaglutide, tirzepatide, incretin-based therapy, body composition, lean mass, muscle loss, protein intake, and resistance exercise. Relevant consensus statements, systematic reviews, meta-analyses, randomized controlled trials, and body-composition substudies were included. The evidence was reviewed and synthesized narratively, with emphasis on diagnostic challenges, changes in body composition, and strategies aimed at preserving muscle mass and function during pharmacological treatment of obesity.

3. Results

3.1. Effects of incretin-based therapies on body composition

The effects of incretin-based therapies on body composition are particularly well illustrated by studies evaluating semaglutide. In the STEP 1 trial, once-weekly semaglutide 2.4 mg produced a mean weight reduction of 14.9% at 68 weeks, clearly outperforming placebo and confirming its high efficacy in obesity treatment [10]. Exploratory body-composition analysis from the same trial showed a 19.3% reduction in total fat mass, a 27.4% reduction in visceral fat mass, and a 9.7% reduction in lean body mass [11]. As a result, the proportion of lean mass relative to total body mass increased, because adipose tissue was reduced to a greater extent. Similar findings were also reported in the SUSTAIN 8 body-composition substudy and in a prospective real-world study, where semaglutide reduced fat mass and visceral adipose tissue, without clinically relevant changes in skeletal muscle index, fat-free mass index, or muscle strength over 26 weeks of treatment [18,19]. A systematic review confirmed that semaglutide is associated with a reduction in lean mass across studies, although the proportion of lean tissue lost relative to total weight reduction varies considerably [14].

A similar pattern has been observed with tirzepatide, although the magnitude of weight loss appears to be even greater. In the SURMOUNT-1 trial, once-weekly tirzepatide produced mean weight reductions of up to 20.9% at 72 weeks, together with broad improvement in cardiometabolic risk [12]. Body-composition analysis from the SURMOUNT-1 substudy showed that tirzepatide reduced body weight by 21.3%, fat mass by 33.9%, and lean mass by 10.9%; approximately three quarters of total weight loss consisted of fat mass, whereas about one quarter consisted of lean mass [13]. A systematic review likewise showed that tirzepatide was associated with reductions in total fat mass, visceral adipose tissue, and waist circumference, whereas its effects on fat-free mass remained less clearly defined because of the limited number of available studies [17].

3.2. Clinical interpretation of reductions in lean tissue

An important question arising from these data is how reductions in lean mass should be interpreted in clinical practice. Available evidence indicates that such changes should not be automatically interpreted as evidence of clinically relevant skeletal muscle loss. This is because the lean compartment includes not only skeletal muscle, but also organs, extracellular fluid, and the lean component of adipose tissue. For this reason, changes in total lean mass do not directly reflect changes in skeletal muscle mass or function. Current evidence suggests that, in many patients, these body-composition changes may represent an adaptive response to substantial weight loss, particularly when they occur together with marked reductions in visceral adiposity, improved insulin sensitivity, and favorable changes in muscle fat infiltration [15,16].

However, in older adults, particularly those with frailty, reduced muscle reserve, impaired mobility, poor nutritional status, or multiple chronic conditions, even partly adaptive losses of lean tissue may become clinically unfavorable. This is particularly relevant when they are accompanied by declines in muscle strength, balance, and mobility, because such changes may further increase the risk of falls, loss of independence, and reduced ability to recover from acute illness. In this group, even relatively small reductions in lean tissue may result in clinically meaningful deterioration in physical function and overall resilience [5,15,16,28,30]. Therefore, the clinical significance of lean-mass reduction should always be interpreted in conjunction with muscle strength, physical performance, nutritional status, and baseline risk profile, rather than solely on the basis of dual-energy X-ray absorptiometry or bioimpedance measurements [1,2,5,7,15,16].

3.3. Prevention strategies for preserving muscle mass and function

The prevention of muscle loss during obesity treatment should rely on an integrated approach combining resistance-based exercise with adequate dietary support, particularly sufficient protein intake. International clinical practice guidelines for sarcopenia identify resistance-based exercise as a core component of management and emphasize that, together with nutritional support, it plays a central role in the prevention of muscle loss [20,21]. Nutritional management should be based on a balanced, high-protein diet that provides adequate energy intake. The PROT-AGE recommendations suggest a daily protein intake of approximately 1.0-1.2 g/kg in healthy older adults and 1.2-1.5 g/kg in individuals with acute or chronic disease, provided that such intake is clinically appropriate [21]. During anti-obesity pharmacotherapy, regular nutritional assessment is particularly important in high-risk patients, especially older adults and individuals with frailty, reduced muscle reserve, or poor nutritional status, because incretin-based medications may decrease total energy and protein intake as a result of appetite suppression and gastrointestinal adverse effects, particularly when dietary intake is not systematically monitored [5,26-28,30].

Exercise interventions further support this strategy. A combination of aerobic and resistance exercise improved functional status more effectively than either exercise modality alone [22]. Strength training attenuated the decline in lean mass during caloric restriction and contributed to the maintenance of muscle

strength [23,24]. According to the World Health Organization guidelines on physical activity and sedentary behaviour, adults, including older adults, should perform at least 150-300 minutes per week of moderate-intensity aerobic activity or 75-150 minutes per week of vigorous-intensity aerobic activity, or an equivalent combination of both [33]. For adults in general, muscle-strengthening activity is recommended on at least 2 days per week, whereas in older adults additional multicomponent exercise emphasizing balance and strength is recommended on at least 3 days per week [33].

Overall, the available evidence supports an integrated strategy combining obesity pharmacotherapy, adequate protein intake, structured exercise, and regular assessment of muscle strength and physical performance to improve the quality of weight loss and reduce the risk of muscle deterioration [20-30,33].

4. Discussion

The findings presented in this review indicate that sarcopenic obesity should be regarded as a clinically important and multidimensional problem, particularly in the context of increasingly effective pharmacological treatment. On the one hand, incretin-based therapies have substantially expanded the therapeutic options available, because they produce marked and sustained reductions in body weight and fat mass, thereby offering an important non-surgical therapeutic option [10-17]. On the other hand, their use has drawn attention to the composition of weight loss, especially in individuals with reduced muscle reserve, frailty, impaired mobility, or other features associated with increased vulnerability to muscle-related complications [5,15,16,28,30]. In this context, the clinical relevance of sarcopenic obesity extends beyond body composition alone and should also be considered in relation to functional capacity, independence, and the ability to tolerate illness or metabolic stress [4,5,8,9].

A central observation emerging from the available studies is that treatment with semaglutide and tirzepatide is accompanied by a measurable decrease in absolute lean tissue, although fat loss remains the predominant component of weight reduction. In the exploratory body-composition analysis of STEP 1, semaglutide reduced total fat mass by 19.3%, visceral fat mass by 27.4%, and lean body mass by 9.7% [11]. As a result, the proportion of lean mass relative to total body mass increased, because adipose tissue was reduced to a greater extent. A similar pattern was reported in the SURMOUNT-1 substudy, in which tirzepatide reduced body weight by 21.3%, fat mass by 33.9%, and lean mass by 10.9%; approximately three quarters of total weight loss consisted of fat mass and about one quarter of lean mass [13]. Comparable observations were also made in the SUSTAIN 8 substudy and in a prospective real-world cohort, where semaglutide reduced fat mass and visceral adipose tissue [18,19]. There were no clinically relevant changes in skeletal muscle index, fat-free mass index, or muscle strength over 26 weeks in the real-world study [19]. These findings suggest that the predominant body-composition effect of incretin-based therapy is a reduction in adipose tissue, although a decrease in absolute lean tissue is also consistently observed.

This distinction is clinically important, because lean tissue is not equivalent to skeletal muscle alone. It also includes organs, extracellular fluid, and the lean component of adipose tissue [15,16]. Relative body composition may improve during treatment, because the reduction in fat mass often exceeds the decrease in lean tissue [11,13,15,16]. Nevertheless, greater caution is warranted in patients with frailty, low muscle reserve, poor nutritional status, or impaired mobility, in whom even partly adaptive losses of lean tissue may be associated with declines in muscle strength, physical performance, and resilience during illness [5,15,16,28,30]. Therefore, body-composition findings should be interpreted in conjunction with functional assessment rather than in isolation.

At current stage of knowledge, the most justified approach in treatment seems to be an integrated management strategy rather than pharmacotherapy alone. In older adults with obesity, combined dietary weight reduction with aerobic and resistance exercise improved functional status more effectively than either exercise modality alone [22]. Exercise added to weight-loss therapy attenuated the reduction in muscle mass and supported muscle strength in frail older adults with obesity [23]. Resistance exercise appears to be particularly important in this context. Adequate dietary protein intake is equally important. The PROT-AGE recommendations support daily protein intakes of approximately 1.0-1.2 g/kg in healthy older adults and 1.2-1.5 g/kg in most individuals with acute or chronic disease [21]. This is particularly relevant during incretin-based treatment, because they may cause appetite suppression and gastrointestinal adverse effects, which can reduce total energy and protein intake if nutritional status is not systematically monitored [21,26-30]. The reviewed studies supports a model in which obesity pharmacotherapy is combined with adequate protein intake, resistance-based exercise, and regular assessment of muscle strength and physical performance in order to improve the quality of weight loss and reduce the risk of clinically meaningful muscle deterioration.

A common problem across reviews is the lack of uniform diagnostic criteria for sarcopenic obesity. It remains a major challenge, contributing to difficulties in diagnosis, risk stratification, and comparison of study outcomes [2,6,7]. Heterogeneity in the assessment of adiposity, muscle mass, muscle strength, and physical performance further limits the consistency of case identification across studies and clinical settings. This has direct consequences for both epidemiology and clinical decision-making. In the Canadian Longitudinal Study on Aging, where multiple definitions were applied to the same cohort, the prevalence of sarcopenic obesity ranged from 0.1% to 85.3% in men and from 0% to 80.4% in women [6]. By contrast, a global meta-analysis estimated the pooled prevalence in older adults at 11% [3]. Such discrepancies are not merely methodological. They influence which patients are classified as high risk, how readily sarcopenic obesity is recognized in routine care, and how reliably treatment outcomes can be compared across studies.

The current evidence base also has important limitations. Many available data come from exploratory analyses, substudies, or relatively short follow-up, and not all studies include direct assessment of muscle strength or physical performance alongside changes in lean tissue [11,13,14,18,19]. The available evidence is clinically informative, but it does not yet allow precise identification of patients at greatest risk of unfavorable muscle-related outcomes during treatment. Further studies using more standardized diagnostic criteria, longer follow-up, and direct functional outcomes are needed, particularly in older adults and other high-risk populations. It is necessary for better balance the metabolic benefits of effective obesity pharmacotherapy with the preservation of muscle function and physical resilience. [2,5-7,15,16,28,30].

5. Conclusions

Incretin-based therapies represent a major advance in obesity treatment; however, in individuals at risk of sarcopenic obesity, weight loss should be interpreted not only by its magnitude, but also by changes in body composition. Particular caution is needed in older adults, frail individuals, and patients with limited muscle reserve. Current evidence supports an integrated strategy combining pharmacotherapy with adequate protein intake, resistance exercise, and regular assessment of muscle strength and physical performance to improve the quality of weight loss and reduce the risk of functional decline. Further research using more standardized diagnostic criteria, longer follow-up, and direct functional outcomes is needed, particularly in older adults and other high-risk populations. This may help achieve a more favorable balance between the efficacy of obesity pharmacotherapy and the preservation of muscle function and physical resilience.

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