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Beyond weight loss: Clinical anabolism to reduce muscle mass loss and physical function decline in patients undergoing pharmacological treatment for obesity

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Abstract

The rapid expansion of anti-obesity pharmacotherapies, such as GLP-1 receptor agonists and GIP/GLP-1 co-agonists, has shifted the focus of obesity management toward substantial weight loss. In older adults and individuals with low lean mass reserve, however, weight loss often includes reductions in fat-free mass, declines in strength and performance, and potential skeletal costs—elements central to autonomy and quality of life. This article presents a clinical and pathophysiological rationale for complementing weight loss interventions with "clinical anabolism" (the therapeutic, protocol-driven, and monitored use of anabolic steroids), always in combination with resistance training and adequate protein-energy nutrition. The proposal is supported by robust indirect evidence from hypercatabolic states and the sarcopenic obesity phenotype, in which agents such as oxandrolone and nandrolone decanoate have demonstrated gains in lean mass and functional improvement under moderate-dose and time-limited regimens. Potential scenarios are specified (older adults undergoing anti-obesity pharmacotherapy, established sarcopenic obesity, disproportionate lean mass loss following more aggressive interventions), along with selection criteria,

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functional targets, and precautions to differentiate legitimate medical use from supraphysiologic abuse. Limitations are acknowledged (scarcity of specific randomized controlled trials in obesity with concomitant pharmacotherapy; androgenic, metabolic, and cardiovascular risks dependent on molecule/dose/duration), as well as the need for systematic clinical and laboratory monitoring. In summary, protecting muscle, bone, and function during weight loss may require combined strategies in which clinical anabolism, in well-selected subgroups, is considered an adjunct to optimize the "quality" of weight loss.
Keywords: Obesity; Sarcopenia; Anabolic Steroids; Weight Loss; Body Composition.

Introduction

Obesity management has entered a new era with the advancement of GLP-1 receptor agonists and dual agonists, such as GIP/GLP-1 co-agonists, capable of inducing substantial weight loss; however, their effects in older adults remain understudied and largely centered on body composition and physical function outcomes (1). In older adults, weight loss, whether intentional or pharmacologically induced, tends to include a concomitant reduction in lean mass, a critical component for mobility, functional capacity, and metabolic health (1–3). Beyond muscle, losses in bone mineral density and an increased risk of fractures have also been described following weight loss in postmenopausal women, with a relative increase in risk observed in analyses from the Women's Health Initiative, suggesting that weight loss may carry a relevant skeletal cost in more vulnerable populations (4).

The notion of "quality" of weight loss, understood as reducing fat while preserving lean mass and functional capacity, requires combined strategies that transcend pharmacological monotherapy, integrating structured exercise, adequate protein-energy nutrition, and agents capable of stimulating anabolism or reducing muscle catabolism (1). The asymmetric pattern of weight regain, which is common after more aggressive weight loss interventions, such as incomplete recovery of lean mass relative to what was lost and disproportionate regain of fat mass, favors a trajectory toward sarcopenic obesity and may impose cumulative functional costs, particularly in an aging population (1,5,6). In cohort studies with follow-up of functional capacity, the dynamics of weight loss and regain cycles were associated with poorer physical

performance and greater vulnerability, supporting the view that multimodal interventions should consider not only “how much” weight is lost, but “how” it is lost and maintained over time (5,6).

Considering the significance of fat-free mass loss accompanying weight loss interventions, particularly when weight reduction is more pronounced, the need for a therapeutic framework that combines anti-obesity strategies with anabolic support appears logically consistent to protect musculoskeletal targets at risk (1–3,6,11). The scientific literature on anabolic-androgenic steroids (AAS) in catabolic states provides robust proof of concept, such as oxandrolone, which has demonstrated efficacy and an appropriate safety profile in various contexts of tissue depletion, with gains in lean mass and functional improvement when properly indicated and monitored (7,12,13). Scientific compilations and several clinical trials in chronic catabolic diseases support this rationale (14,15). In chronic obstructive pulmonary disease, a randomized study reported improvements in body composition and quality of life with anabolic steroids as adjunctive therapy, while in patients with major burns, nandrolone decanoate, administered under controlled regimens, attenuated hypercatabolism and improved clinical outcomes without new relevant safety signals (8,9). Taken together, these data suggest that, in the context of weight loss, therapeutic pharmacological anabolism may be explored as a potential adjunctive strategy to preserve muscle mass and function in individuals at greater risk of sarcopenia, without abandoning the core of obesity treatment (1–3,7–9,11).

Candidate selection for treatment, choice of agents, and dosing regimens, however, should prioritize pathophysiological profiles and clearly defined functional goals, with preference given to anabolic agents that maximize anabolic effects and minimize adverse events (7–11,14,15). Accumulated experience indicates that AAS such as oxandrolone, oxymetholone, and nandrolone decanoate are more aligned with this profile, given that they are less androgenic and more anabolic compared with testosterone (7,10,11,16), although they still require clinical and laboratory monitoring for potential androgenic and metabolic adverse effects. In scenarios of sarcopenic obesity, the proposal to revisit anabolic agents as adjuncts specifically aims to mitigate dynapenia and performance decline, linking gains in lean mass to clinically meaningful outcomes in physical function, autonomy, and quality of life (2,6,11). In this context, the conceptual distinction between replacement, therapeutic use, and abuse should guide both eligibility and scientific communication, minimizing stigma and interpretive bias

and reinforcing that this represents legitimate medical use, with well-defined objectives and limits, such as dose and duration of the clinical anabolism therapeutic process (7,10,11).

Thus, the present clinical reasoning proposal is supported by indirect evidence derived from catabolic states and sarcopenic obesity to suggest that short, well-monitored courses of AAS, in combination with anti-obesity pharmacotherapy, exercise, and nutrition, may help preserve muscle mass and function in more vulnerable subgroups (1–3,7–11,17). Priority populations include older adults at risk of sarcopenia, individuals with disproportionate lean mass loss during weight reduction, and patients already presenting with the sarcopenic obesity phenotype, in whom body composition, muscle strength, and functional performance outcomes assume a central role alongside clinical and laboratory safety (1–3,6,11,17). This opinion article does not intend to offer prescriptive recommendations, but rather to organize the pathophysiological and clinical rationale, as well as possible application scenarios, in order to discuss whether and how clinical anabolism may have an assertive role in contemporary obesity care (1–3,7–11).

Finally, in this context, understanding the mechanisms by which weight loss influences muscle mass, and how clinical anabolism could act to counteract undesirable effects, as well as sarcopenia and dynapenia, is essential to evaluate the possibility of using this strategy as a potential therapeutic adjunct to be considered in specific subgroups of patients with obesity (1–3,6,7–11).

Pathophysiological and Clinical Rationale

The coexistence of excess adipose tissue and reduced muscle mass defines a phenotype of greater clinical vulnerability, associated with poorer physical performance, higher risk of disability, and increased mortality in older adults (2,6,11). Sarcopenic obesity represents precisely this intersection, combining increased fat mass, often visceral, with depletion of lean mass and muscle strength, thereby reducing functional reserve and limiting the capacity to respond to metabolic and inflammatory stressors (2,6,11). Systematic reviews show that weight loss interventions based on caloric restriction, although effective in reducing fat mass and improving cardiometabolic outcomes, are accompanied by significant loss of fat-free mass, whereas the addition of resistance training attenuates, but does not completely eliminate, this effect in

middle-aged and older individuals (3,11,17,18). In patients who already present with a deficit in muscle mass or with a sarcopenic obesity phenotype, the induction of weight loss without an explicit strategy to protect lean tissue tends to deepen musculoskeletal reserve impairment and to increase the risk of frailty and adverse events (1–3,6,11,17).

From a pathophysiological perspective, weight loss in individuals with obesity often evolves within an environment of chronic low-grade inflammation, anabolic resistance to protein intake and physical exercise, and hormonal alterations that include patterns unfavorable to the maintenance of muscle mass, phenomena described at the interfaces between obesity, aging, and sarcopenia (2,3,11). When more intense or prolonged energy restriction is added to this context, such as in structured weight loss programs or with the use of potent pharmacological therapies, the catabolic state is amplified, favoring the mobilization of muscle protein as an energy substrate and accelerating the decline in muscle mass and strength (1,3,11). In clinical states of hypercatabolism, such as wasting diseases, chronic obstructive pulmonary disease, or major burns, the introduction of anabolic steroids under therapeutic regimens has demonstrated the ability to attenuate negative nitrogen balance, increase lean mass, and improve functional outcomes, indicating that muscle remains responsive when appropriate stimuli are combined with targeted pharmacological support (7–9,11–15).

In this scenario, the concept of clinical anabolism emerges as a rational complement to anti-obesity interventions in selected subgroups. Models of therapeutic use of testosterone and other anabolic steroids in contexts of catabolism, dynapenia, or sarcopenic obesity suggest that these agents can be employed in a protocol-driven manner, with moderate dosing, limited duration, and systematic monitoring, clearly distinguishing this legitimate medical practice from recreational abuse at doses far above therapeutic ranges (7–11,14,15). Applied to pharmacologically induced weight loss, especially in older adults, in individuals with disproportionate lean mass loss, or in those presenting with a sarcopenic obesity phenotype, this rationale supports the hypothesis that combinations of anti-obesity pharmacotherapy, structured exercise, preferably resistance training, nutritional support, and anabolic steroids administered under therapeutic regimens may preserve muscle and function without compromising the metabolic benefits of the primary treatment (1–3,7–11,14,15,17,18).

To synthesize the concept of “quality” of weight loss and the possible role of clinical anabolism in this context, **Table 1** contrasts interventions focused solely on

kilograms lost, particularly monotherapies, with approaches oriented toward the preservation of muscle mass and function.

Table 1. Conceptual model contrasting weight loss interventions focused solely on kilograms lost with approaches centered on the quality of weight loss. In the first pathway, caloric restriction and/or anti-obesity pharmacotherapy used in isolation may reduce fat, but also adversely reduce muscle mass and function, favoring progression toward sarcopenic obesity in vulnerable individuals. In the second pathway, the combination of strategies, including resistance training, protein-energy nutrition, and, in selected subgroups, clinical anabolism under therapeutic regimens, seeks to reduce fat while preserving muscle mass and function, with a focus on maintaining autonomy and quality of life.

Aspect	Interventions Focused Only on Body Weight	Interventions Focused on the Quality of Weight Loss
Primary Objective	Reduction in kilograms on the scale, with emphasis on numerical weight loss targets.	Reduction of body fat while preserving or increasing muscle mass and function, with focus on functional capacity and autonomy.
Main Components	Isolated caloric restriction; anti-obesity pharmacotherapy alone (e.g., GLP-1 receptor agonists), with little or no structured exercise or supportive nutritional strategy.	Anti-obesity pharmacotherapy when indicated; structured caloric restriction; resistance training; protein-energy–optimized nutrition; clinical anabolism in selected subgroups (therapeutic use of anabolic steroids in short courses, moderate doses, with systematic monitoring).
Expected Effects on Body Composition	Reduction in body fat accompanied by proportional or disproportionate loss of lean mass.	Reduction in body fat with attenuation of lean mass loss or potential muscle gain, depending on patient profile and intensity of anabolic stimuli.
Probable Functional Effects	Reduction in muscle strength; worsening or only marginal maintenance of performance in functional tests; greater vulnerability to falls and decline in autonomy in higher-risk patients.	Maintenance or improvement of muscle strength and functional performance; greater preservation of mobility, balance, and capacity for activities of daily living.
Risk of Sarcopenic Obesity and Frailty	Increased risk of progression to sarcopenic obesity in individuals with low baseline lean mass; possible acceleration of frailty trajectory.	Reduced risk or attenuation of progression to sarcopenic obesity in vulnerable individuals; potential protection against muscle loss–related frailty.
Clinical Comment	Strategy that may achieve weight loss goals but with relevant musculoskeletal cost in more vulnerable subgroups, especially older adults and patients with low baseline lean mass.	Strategy that aims to balance the metabolic benefits of weight loss with protection of skeletal muscle, integrating clinical anabolism within a well-defined and monitored therapeutic context for selected subgroups.

Based on this rationale, the next step is to identify the clinical situations in which therapeutic anabolism could, in theory, provide protection of muscle mass and function during weight loss without compromising the core of anti-obesity treatment (1–3,6,7–11,14,15).

Clinical Scenarios for the Therapeutic Use of Anabolic Steroids During Weight Loss

The discussion regarding the therapeutic use of anabolic steroids in the context of weight loss should begin with clearly defined clinical scenarios in which the potential benefit in preserving muscle mass and function is relevant and plausibly outweighs the risks (7–11). The literature on sarcopenic obesity and catabolic states suggests that this balance tends to be more favorable in individuals with greater musculoskeletal vulnerability, such as older adults with low lean mass reserve, patients with a sarcopenic obesity phenotype, and individuals exposed to additional stressors, for example chronic diseases, that exacerbate protein catabolism (2,3,6,7–9,11,14,15). In these contexts, clinical anabolism, when indicated in a protocol-driven and monitored manner, is not a cosmetic strategy, but rather a morphological and functional intervention aimed at attenuating potential muscle and bone mass loss, preserving or restoring physical performance, and maintaining autonomy and quality of life (7–11).

A first scenario includes older adults with obesity who initiate anti-obesity pharmacological therapies and present a high risk of sarcopenia, whether due to low baseline lean mass or a prior trajectory of marked weight loss with functional impairment (1–3,6,11). In this population, fat-free mass losses associated with energy restriction and pharmacological treatment may translate into reduced strength, worsening performance on functional tests, and increased risk of falls and fractures, especially in postmenopausal women (1–5,11,14,15,20). The hypothesis is that a short and well-monitored course of an anabolic steroid, combined with resistance training and adequate nutritional support, as well as supplementation when necessary, may attenuate muscle mass loss and preserve functional capacity without compromising the cardiometabolic benefits of weight loss (1–3,7–9,11,14,15,17–19).

A second scenario involves individuals with sarcopenic obesity, characterized by low muscle mass and strength associated with excess adiposity, in whom conventional weight loss interventions tend to aggravate the quantitative and qualitative musculoskeletal deficit (2,3,6,11,20). In this population, revisiting the therapeutic use of anabolic steroids as adjuncts aims to mitigate dynapenia and promote performance gains that translate into greater autonomy, lower risk of disability, and improved quality of life, provided that careful selection and systematic monitoring are ensured (7,11,14,15). The rationale is similar to that adopted in classic catabolic states, such as burns, chronic obstructive pulmonary disease, and wasting diseases, in which therapeutic anabolism has demonstrated increases in lean mass, functional improvement, and an acceptable safety profile under moderate-dose and time-limited regimens (7–9,11–13).

A third potential scenario involves patients undergoing more intensive weight loss interventions, such as aggressive caloric restriction programs or bariatric procedures, including intragastric balloon placement, who experience disproportionate lean mass loss or clinically relevant functional decline in the post-intervention period, despite improvement in cardiometabolic markers (1–3,11). Evidence indicates that bariatric surgery is consistently associated with significant loss of fat-free mass, particularly in the first months following the procedure, potentially accounting for approximately 12–22% of the total weight lost after surgery (21–23). In these cases, individualized consideration of a restricted course of an anabolic steroid as part of a rehabilitation protocol, combining resistance training, protein-energy optimization, and clinical and laboratory monitoring, may be fully justifiable in selected subgroups (3,7–9,11).

A useful conceptual framework to avoid confusion between legitimate medical practice and recreational abuse is a four-scenario taxonomy, distinguishing replacement therapy in hypogonadism, therapeutic use in catabolic states or muscle loss, controlled supraphysiologic use for aesthetic or performance purposes, and chaotic abuse at high doses, often involving polypharmacy and absence of monitoring (10,11,24,25). The proposal discussed in this article explicitly falls within the second scenario, that is, the therapeutic use of anabolic steroids to mitigate catabolism and preserve muscle mass and function in contexts of greater vulnerability, rationally and scientifically extrapolated to patients undergoing obesity treatment (1–3,7–11,14,15). Maintaining this distinction is central to interpreting risks and benefits and to preventing structured interventions, with moderate dosing, limited duration, and systematic monitoring, from being improperly equated with abusive practices that account for the majority of severe adverse events described (7–11,24,25).

These scenarios illustrate that the possible role of clinical anabolism during weight loss interventions should be directed toward profiles of greater musculoskeletal vulnerability, which makes it even more important to clearly and in a balanced manner articulate the potential benefits and risks involved (1–3,6–11,14,15).

Potential Benefits and Risks of the Therapeutic Use of Anabolic Steroids During Weight Loss

The potential benefits in the context of weight loss derive from three main axes: preservation of lean mass, maintenance or improvement of physical function, and a favorable impact on intermediate clinical outcomes, such as risk of falls, disability, and hospitalizations (1–3,6,7–9,11,14,15,26,27). In classic catabolic states, such as wasting diseases, heart failure, chronic kidney disease, cirrhosis, HIV, chronic obstructive pulmonary disease, and extensive burns, reviews and clinical trials have documented increases in fat-free mass, improvements in strength and functional performance, and, in some scenarios, more favorable overall recovery when anabolic steroids were added to nutritional and rehabilitation strategies (7–9,11,15,26,27). In the sarcopenic obesity phenotype, by mitigating muscle loss or facilitating muscle gain in individuals with excess adiposity and low lean mass, therapeutic anabolism may contribute to interrupting the cycle of frailty, inactivity, and progressive functional decline, particularly in older adults (2,6,11,15,26,27). Extrapolated to pharmacologically induced weight loss, the hypothesis is that short and well-monitored courses of anabolic steroids may render the "quality" of weight loss more favorable in higher-risk subgroups, without replacing the primary obesity treatment (1–3,7–9,11,26,27).

These potential benefits require careful consideration in light of recognized risks, even in therapeutic contexts. Changes in lipid profile, elevations in liver enzymes, polycythemia, fluid retention, androgenic effects in women, and possible cardiovascular consequences vary according to the specific molecule, dose, and duration (7–11,26,27). Effects on the hypothalamic–pituitary–gonadal axis, with suppression of endogenous testosterone production and repercussions on fertility, become especially relevant in younger individuals or in proposals involving prolonged or repeated use, reinforcing the need to clearly define indications, duration, and functional targets (7,10,11,26,27). Pharmacological differences among agents imply distinct hepatic and metabolic risk profiles, requiring individualization of the specific drug and avoiding uncritical transposition of standardized protocols across different populations (7,9–11,26,27).

There are also, unfortunately, ethical and regulatory challenges in a field marked by stigma, especially among health professionals, and a history of abuse at doses far above therapeutic ranges without supervision (10,11,28,29–31). The line separating therapeutic use from aesthetic or performance use becomes blurred if clinical criteria, functional goals, and limits regarding dose and duration are not precisely defined and communicated (10,11,26,27). For proposals of clinical anabolism during weight loss interventions to be viewed within an appropriate ethical framework, it is essential to

maintain precise indications, select patients with a genuine risk of functional loss, and ensure informed consent, systematic clinical and laboratory monitoring, and documentation of adverse events, recognizing that the inferences discussed here are largely based on indirect evidence (1,7–11,14,15,26,27).

Table 2 pragmatically organizes selected clinical scenarios in which the therapeutic use of anabolic steroids could be considered as an adjunct during weight loss, taking into account the context of greater musculoskeletal vulnerability.

Table 2. Clinical scenarios in which therapeutic anabolism may be considered during weight loss interventions, highlighting the primary risk in each situation, the functional objectives of clinical anabolism, the required associated components (exercise, nutrition, and monitoring), and the main precautions to differentiate legitimate medical use from supraphysiologic abuse.

Clinical Scenario	Main Risk During Weight Loss	Objective of Therapeutic Use of Anabolic Steroids	Mandatory Associated Components	Comments and Precautions
Older adult with obesity and low baseline lean mass, initiating anti-obesity pharmacotherapy	Disproportionate lean mass loss, reduced strength, falls, functional decline, worsening autonomy	Attenuate loss of muscle mass and strength during weight reduction, preserving physical performance and capacity for activities of daily living	Structured resistance training; adequate protein-energy nutrition; regular clinical and laboratory monitoring; review of concomitant medications	Consider only in patients with clear musculoskeletal vulnerability; use short courses or moderate doses; periodically reassess need; avoid extrapolation for aesthetic or performance purposes
Patient with sarcopenic obesity (excess fat + low muscle mass and strength)	Disability, frailty, functional limitation, increased risk of hospitalization and institutionalization	Facilitate gain or maintenance of lean mass and strength, aiming to improve functional performance, mobility, and quality of life	Rehabilitation program focused on resistance exercise; protein-calorie optimization; management of comorbidities; monitoring of cardiovascular, hepatic, and hematological adverse events	Population with greater potential for functional benefit; careful candidate selection required; clearly distinguish therapeutic use from supraphysiological abuse; clear communication of functional goals to the patient
Post-bariatric surgery patient or individual after aggressive caloric restriction, with marked lean mass loss and functional decline	Significant decline in strength and physical performance despite metabolic improvement; difficulty in musculoskeletal rehabilitation	Support recovery of muscle mass and function in the post-intervention period, facilitating rehabilitation and restoration of autonomy	Supervised physical rehabilitation; adequate protein-energy intake during postoperative phase; micronutrient monitoring; multidisciplinary follow-up	Consider only after clinical stabilization; assess procedure-specific risks and comorbidities; clearly define indication, duration, and discontinuation criteria for clinical anabolism
Middle-aged adult with obesity and repetitive weight loss–regain cycles, with progressive lean mass decline	Repeated cycles of fat loss and regain with progressive muscle depletion; gradual deterioration of physical performance	Break the cycle of cumulative lean mass loss during a new weight loss phase, preserving musculoskeletal reserve	Structured weight loss plan combined with resistance training; protein-energy nutrition; longitudinal body composition monitoring; lifestyle support	Indicate only in cases with clear history of lean mass loss and weight cycling; avoid trivialization of use; emphasize temporary and therapeutic nature of intervention
Patient with multiple chronic comorbidities (e.g., COPD, stable heart failure, chronic kidney disease) and obesity, with high catabolic risk	Increased vulnerability to accelerated muscle loss during weight reduction; potentially severe functional impact in context of reduced physiological reserve	Protect muscle mass and function in high metabolic risk situations, contributing to maintenance of functional capacity and exercise tolerance	Coordination with core medical team (e.g., pulmonology, cardiology, nephrology); physiotherapy and nutrition; rigorous assessment of contraindications; close	Case-by-case evaluation required; heightened caution in dose selection; prioritize maximum safety; detailed recording of adverse events and functional response

Clinical Scenario	Main Risk During Weight Loss	Objective of Therapeutic Use of Anabolic Steroids	Mandatory Associated Components	Comments and Precautions
			monitoring of clinical and laboratory parameters	

Considering this balance between potential morphological and functional gains and a set of associated risks, it is essential to acknowledge the limitations of the available specific evidence and the boundaries of the rationale proposed here in order to avoid undue extrapolations or recommendations (1–3,7–11).

Limitations and Boundaries of the Rationale

The scientific and clinical rationale presented in this article is supported by solid evidence, although predominantly indirect, derived from observational studies on sarcopenic obesity and from clinical trials involving anabolic steroids in classic catabolic states, such as wasting diseases, chronic obstructive pulmonary disease, heart failure, chronic kidney disease, cirrhosis, HIV, and major burns (2,3,6–9,11,15,26,27). To the best of our knowledge, we did not identify randomized trials specifically designed to evaluate the concomitant therapeutic use of anabolic steroids as adjuncts in pharmacological or surgical weight loss interventions in individuals with obesity, which limits the potential for direct causal inference in this context (1–3,7–9,11,15,26,27). Thus, the proposals discussed here should therefore be interpreted as hypotheses for clinical intervention and as an attempt to coherently organize pieces of scientific evidence dispersed across different clinical contexts (1–3,7–11,15,26,27).

Furthermore, the recognized risks of anabolic steroids, even in therapeutic contexts, especially when considered over prolonged periods of use, impose additional boundaries on the rationale (14,15,26,27). Changes in lipid profile, potential cardiovascular effects, androgenic events in women, molecule-dependent hepatic impact, and polycythemia are aspects that require careful patient selection, judicious choice of agents and dosing regimens, and systematic clinical and laboratory monitoring (7–11,26,27). The frequent presence of multiple comorbidities in individuals with obesity, such as diabetes, hypertension, and established cardiovascular disease, increases this complexity and reinforces the prudence required in this context (1,7–11,14,15,26,27). Finally, the history of stigma and recreational abuse of anabolic steroids requires a clear conceptual distinction between legitimate medical use, with

moderate dosing, limited duration, and clinical and laboratory monitoring, and supraphysiologic practices aimed at aesthetic or performance purposes without medical supervision, which account for the majority of severe adverse events described in the literature (7–11,25,30,31).

Conclusion

The combination of obesity, muscle mass loss, and aging creates a context in which the quality of weight loss becomes as important as the quantity of kilograms eliminated. In many patients, particularly those with low lean mass reserve or a sarcopenic obesity phenotype, weight loss interventions may deepen musculoskeletal impairment and accelerate functional decline, even when they produce relevant metabolic benefits.

Accumulated experience with anabolic steroids in chronic diseases characterized by catabolic states and in sarcopenic obesity suggests that, when used in a therapeutic, protocol-driven, and monitored manner, these agents may contribute to preserving or restoring muscle mass and function in contexts of greater vulnerability. Extrapolating this knowledge to obesity treatment implies cautiously and individually considering the possibility of clinical anabolism as a complement to pharmacological and behavioral weight loss strategies, with a focus on functional and quality-of-life outcomes.

The ideas discussed in this article should not be interpreted as a recommendation for the routine use of anabolic steroids in patients with obesity, but rather as a conceptual, clinical, and scientific exercise that critically organizes indirect evidence and suggests possibilities for optimized treatment. Rather than proposing an immediate change in practice, this text seeks to reposition muscle and functionality at the center of the obesity discussion and to open space for a balanced reflection on the role of clinical anabolism in contemporary care. The rigorous distinction between legitimate medical use and recreational abuse of anabolic steroids is an essential element of this discussion, both to protect patients and to allow potential therapeutic benefits to be evaluated without the burden of historical stigma.

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