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Research Progress on the Relationship between Dynapenic Obesity and Type 2 Diabetes

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Abstract

Sarcopenic obesity (SO) is a disease state characterized by a decline in muscle mass and function, combined with obesity. In the elderly, SO often coexists with type 2 diabetes (T2D), as chronic inflammation and insulin resistance are common pathophysiological mechanisms for both. The coexistence of them can lead to significant burdens for older adults, including increased frailty, elevated risk of falls, reduced daily living capabilities, and potential cognitive impairments, as well as the challenges associated with polypharmacy. However, the diagnostic criteria for SO are complex and not uniform, and research suggests that muscle function, rather than muscle mass, seems to be more closely related to adverse outcomes in elderly patients with T2D. Moreover, the measurement of muscle strength is simpler. Replacing SO with dynapenic obesity (DO) as a biological marker for the early prediction of functional decline in patients with T2D appears to be more effective and convenient. Therefore, this paper elaborates on the hazards and epidemiology of DO, and reviews its relationship with T2D and the management of comorbidities.

Keywords

Dynapenic Obesity, Type 2 Diabetes, Elderly

1. Introduction

The global issue of population aging is escalating to a critical level. As reported by the Population Division of the United Nations, it is projected that by 2050, the global population of individuals aged 65 and above will surpass 2.1 billion [1]. With advancing age, the human body is prone to developing multiple chronic conditions, including type 2 diabetes (T2D), cognitive impairment, and mobility disabilities [2]. Concurrently, there are alterations in body composition, characterized by an

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increase in fat mass and a reduction in muscle mass, thereby leading to sarcopenic obesity (SO) [3]. SO not only precipitates metabolic derangements such as hyperlipidemia, insulin resistance, and inflammatory responses but also contributes to a decline in the elderly's activities of daily living, an increased risk of disability, and the onset of depression and other adverse events [4]. Ultimately, this culminates in a significant deterioration of the quality of life and may even result in mortality.

However, the diagnostic criteria for SO remain inconsistent. The diagnosis of sarcopenia generally necessitates the evaluation of both muscle mass and muscle function. The Asian Working Group for Sarcopenia (AWGS) [5] and the European Working Group on Sarcopenia [6] have both recommended the use of the Appendicular Skeletal Muscle Mass Index (ASMI) for assessing muscle mass, along with grip strength and gait speed for evaluating muscle function. Notwithstanding, the diagnostic thresholds proposed by these two groups exhibit subtle differences. In addition, there exists a multiplicity of approaches for diagnosing obesity. Commonly employed metrics include the Body Mass Index (BMI), total body fat percentage (BF%), waist circumference (WC), and visceral fat area, among others. The diagnosis of sarcopenia is intricate, as the assessment of muscle mass often relies on techniques such as dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA). Notably, a meta-analysis encompassing 50 prospective studies has demonstrated that, compared to low muscle mass, low muscle strength (manifested as low grip strength) is more strongly correlated with a decline in physical function [7]. Consequently, dynapenic obesity (DO), a disease state in which reduced muscle function coexists with obesity, seems to represent a more straightforward and efficacious biological marker for predicting the physical function of the elderly population.

Aging is the shared pathophysiological mechanism underpinning both DO and T2D. Insulin resistance and systemic inflammatory responses instigate a vicious cycle between these two conditions. Ultimately, this cycle culminates in adverse consequences, including a decline in the quality of life among the elderly and an elevation in hospitalization rates. Therefore, this paper reviews the recent research advancements concerning the relationship between DO and T2D, including the mutual influences between the two, and elaborates on the comprehensive management of their comorbidity.

2. Definition and Prevalence of DO

DO denotes a condition characterized by the co-occurrence of obesity and a reduction in muscle strength. The diagnostic criteria are presented in **Table 1**. Dynapenia can be appraised via measures such as handgrip strength, the Time Up and Go (TUG) test, and the Short Physical Performance Battery (SPPB). In clinical settings, handgrip strength represents one of the most frequently utilized approaches for assessing muscle strength. This is typically measured using either a spring dynamometer or an electronic dynamometer. Additionally, the assessment of knee-extension strength is also commonly employed. Notably, the criteria for

determining muscle-strength decline demonstrate ethnic specificity. Herein, we present only the diagnostic cut-off values applicable to the Asian population. Specifically, we have adopted the criteria proposed by the AWGS (2019), which are more strongly and independently associated with adverse health outcomes in elderly individuals with T2D, as the diagnostic threshold for low handgrip strength in the context of dynapenia [8].

Table 1. The diagnostic criteria for dynapenic obesity.

		Male	Female
Dynapenia	Asian Working Group for Sarcopenia	<28 kg	<18 kg
Obesity	Working Group on Obesity in China	BMI ≥ 28 kg/m^2	
		BMI ≥ 25 kg/m^2	
	World Health Organization	BF% ≥ 25%	BF% ≥ 35%
		WC ≥ 90 cm	WC ≥ 85 cm
	Chinese Medical Association	Visceral fat area ≥ 100 cm^2	

BMI, Body Mass Index; BF%, total body fat percentage; WC, waist circumference.

In a cross-sectional study carried out in China, the prevalence of DO among the elderly aged over 60 was found to be 6.17% [9]. Another study from Brazil reported that the prevalence of DO among individuals aged over 60 was 10.73% [10]. In contrast, among community-dwelling residents in the United Kingdom (UK) aged over 50, this prevalence was merely 2.2% [11]. The prevalence rates of DO exhibit substantial variations among diverse studies. This may be attributed to several factors. Firstly, the sample sizes in each study differ, and the methods and thresholds used for diagnosing obesity and dynapenia also show considerable discrepancies. Secondly, there are disparities in the ethnic groups and the overall age ranges of the populations enrolled in these studies. A study by Mori [12] *et al.*, which involved 645 patients with T2D, revealed that the prevalence of DO among these diabetic patients was 7.9%. Intriguingly, this value did not exceed the prevalence of DO observed in the general population. Nevertheless, in this study, obesity was defined using BMI, a metric that may have failed to identify certain patients with abdominal obesity. It is well-recognized that abdominal obesity is more strongly associated with metabolic derangements compared to general obesity. Typically, the T2D population exhibits a more pronounced inflammatory response and heightened insulin resistance relative to the general population. Moreover, they often contend with diabetic complications and are more predisposed to comorbid chronic conditions such as hypertension and obesity. Consequently, it is reasonable to postulate that the prevalence of DO might be higher within the T2D population.

3. Relationship between DO and T2D

3.1. Mutual Prevalence Promotion in DO & T2D

DO and T2D share common disease-related mechanisms. They are reciprocally

causal, each promoting the development of the other and thus establishing a vicious cycle. A Japanese investigation demonstrated that DO is associated with an elevated prevalence of T2D in middle-aged and elderly men (odds ratio [OR] = 0.64; 95% confidence interval [CI]: 0.49 - 0.83) [13]. Another study, carried out among individuals aged over 50, defined dynapenia by evaluating lower-limb muscle strength via the five-repetition chair-stand test (CST-5), also corroborated the aforementioned finding (relative risk [RR] = 2.45; 95% CI: 2.25 - 2.67) [14]. Moreover, a 10-year longitudinal study of the elderly population in the UK indicated that DO significantly augments the risk of developing T2D (OR = 5.87; 95% CI: 3.13 - 11.03) [15].

On the contrary, a cross-sectional study carried out in the UK incorporated 5290 community-dwelling individuals aged over 50. These participants were categorized into four distinct groups: non-diabetic individuals (characterized by no self-reported diabetes and a glycated hemoglobin [HbA1c] level < 6.5%), those with undiagnosed diabetes (absence of self-reported diabetes yet HbA1c ≥ 6.5%), patients with controlled diabetes (self-reported diabetes accompanied by an HbA1c < 7%), and those with uncontrolled diabetes (self-reported diabetes and HbA1c ≥ 7%) [16]. The resultant findings demonstrated that, in comparison to non-diabetic subjects, those in the uncontrolled diabetes group were associated with the incidence of dynapenia (for men: OR = 2.37, 95% CI: 1.36 - 4.14; for women: OR = 1.67, 95% CI: 1.01 - 2.79). However, this association was not evident in either the undiagnosed diabetes group or the controlled diabetes group. Subsequent linear regression analysis further revealed a significant correlation between low HbA1c levels and dynapenia. Notably, this relationship remained robust even after adjusting for various factors, including sociodemographic, behavioral, and clinical characteristics. Additionally, a gender-specific difference was observed, with men having a lower HbA1c threshold associated with dynapenia compared to women (HbA1c ≥ 6.5% for men and ≥8.0% for women). This finding suggests that the systemic inflammatory response and metabolic derangements inherent in T2D are independently correlated with the development of dynapenia, and males appear to be more susceptible to a decline in muscle function. Another research endeavor, which involved 2729 middle-aged and elderly patients presenting with abdominal obesity, also indicated a close association between diabetes and dynapenia [17].

Consequently, a substantial body of research indicates that there exists a reciprocal relationship between DO and T2D, whereby each condition promotes an elevation in the prevalence of the other. This association holds significant clinical implications. In the clinical setting, when managing patients with DO, healthcare providers must remain vigilant regarding the risk of T2D and initiate screening procedures proactively. Conversely, during the diagnosis and treatment of T2D patients, due attention should be paid to the potential development of DO, enabling timely intervention.

3.2. Biomarkers in DO-T2D Interactive Progression

The interaction mechanisms between DO and T2D are intricate, and certain

clinical studies may offer valuable insights. A study involving 166 T2D patients discovered that the accumulation of advanced glycation end-products (AGEs) in these patients constituted an independent risk factor for dynapenia [18]. Another investigation, which incorporated 1442 diabetes patients and assessed dynapenia via knee-extension strength, revealed a significant association between diabetic polyneuropathy and dynapenia in middle-aged and elderly T2D patients [19]. In a prospective study by Sivritepe [20], 122 elderly female T2D patients were recruited, and their serum vitamin D levels and handgrip strength were measured. The results indicated a significant negative correlation between vitamin D levels and dynapenia. A survey conducted by the Department of Geriatrics at Tokyo Medical University in Japan demonstrated that, in comparison to elderly T2D patients with Alzheimer's disease, those with diabetes-related dementia (DrD) exhibited a significant reduction in upper-limb strength and gait speed, yet no apparent decline in muscle mass was observed [21]. It has been reported that the pathophysiological mechanisms of DrD diverge from those of Alzheimer's disease or vascular dementia. Specifically, cerebrovascular diseases or parietotemporal hypoperfusion are not detectable through imaging and nuclear medicine techniques [22]. This finding seemingly implies that T2D may induce dementia via its unique pathophysiological pathway, ultimately resulting in the development of dynapenia.

Conversely, Sénéchal [23] *et al.* defined dynapenia as a decline in leg strength. Their findings indicated that, relative to the control group, the DO group presented with elevated plasma triglyceride and blood glucose levels, along with reduced high-density lipoprotein cholesterol levels. Simultaneously, the prevalence of metabolic syndrome, T2D, and cardiovascular diseases was notably higher in the case group. This observation prompts the conjecture: does DO elevate the incidence of T2D via metabolic pathways? In a study encompassing 483 non-diabetic individuals, a significant correlation between DO and insulin resistance was identified (OR = 4.98, 95% CI: 1.46 - 6.88) [24]. A Brazilian investigation among elderly patients with T2D suggested that DO was associated with heightened inflammation levels in these patients. Specifically, subjects in the DO group demonstrated increased levels of pro-inflammatory factors (e.g., tumor necrosis factor-*α*) and decreased concentrations of anti-inflammatory cytokines (e.g., interleukin-10) [25].

In general, T2D, a complex chronic metabolic disease, is prominently characterized by metabolic derangements and suboptimal nutritional status. Throughout the disease course, microvascular complications and diabetes-associated comorbidities may act upon the body, creating a conducive environment for the onset and progression of DO. DO, in turn, with its inherent features of chronic low-grade inflammation, dyslipidemia, and profound insulin resistance, promotes the progression of T2D.

3.3. Impact of DO on Function among T2D Patients

In elderly individuals with T2D, the disease is frequently complicated by multiple

comorbidities, including cognitive impairment, frailty, and cardiovascular and cerebrovascular diseases. The emergence of body composition alterations, coupled with the development of DO, further exacerbates the health burden. The study conducted by Oba [26] *et al.* enrolled 417 elderly patients with cardiometabolic diseases, excluding those with severe cognitive impairment. Among these participants, 52.7% were diagnosed with T2D. The Japanese version of the Montreal Cognitive Assessment (a score of ≤ 25) was employed to identify mild cognitive impairment (MCI). After adjusting for relevant covariates, it was revealed that patients with DO had a significantly elevated risk of developing MCI (OR = 3.98, 95% CI: 1.15 - 13.77). Subsequent logistic regression analysis demonstrated that both low grip strength (OR = 2.19, 95% CI: 1.11 - 4.29) and high waist circumference (OR = 2.03, 95% CI: 1.03 - 3.99) were independently associated with MCI. A Japanese prospective study included 204 elderly T2D male patients aged 65 years and above. The Tokyo Metropolitan Institute of Gerontology Index of Competence (TMIG-IC) was utilized to evaluate their advanced functional capacity. The findings provided evidence of a statistically significant association between dynapenia and the decline in advanced functional capacity among elderly T2D male patients (regression coefficient = −1.26, 95% CI [−2.35, −0.17], P = 0.024) [27].

In the geriatric cohort with T2D, the physiological decline of multiple bodily functions, coupled with the inherent influence of the disease, has established a profound association with cognitive impairment and a decrement in activities of daily living (ADL). Cognitive decline typically manifests as impairments in memory, attention, and executive function. These deficits introduce substantial obstacles to the elderly's daily decision-making processes and social interactions. Concurrently, the decline in ADL is evident in difficulties encountered during fundamental activities such as dressing, eating, and ambulation, which severely undermines their independence and self-sufficiency. When geriatric T2D patients develop comorbid DO, the situation deteriorates significantly. DO-induced muscle weakness and compromised balance lead to a marked increase in the incidence of accidental incidents, including falls and fractures. These adverse events not only inflict direct physical trauma upon patients but also substantially degrade their quality of life, imposing substantial physical and psychological distress. Simultaneously, there is a concomitant surge in the demand for medical care among these patients. Prolonged treatment and care regimens not only exacerbate the financial burden on families but also place increased strain on social financial resources. Moreover, the deterioration of health status is associated with an elevated mortality rate, further underscoring the formidable challenges presented by this comorbidity to elderly T2D patients, their families, and society at large.

3.4. Interaction Mechanisms between DO and T2D

Firstly, a vicious cycle appears to exist between the two physical components of DO: muscle and adipose tissue. Reportedly, obesity, particularly the accretion of

visceral fat, can instigate chronic inflammation [28]. Elevated levels of pro-inflammatory cytokines, such as interleukin-6 and C-reactive protein, may then precipitate muscle atrophy [29]. Simultaneously, an abundance of free fatty acids in the context of obesity can induce mitochondrial dysfunction, augment the generation of reactive oxygen species, give rise to lipotoxicity and insulin resistance, and enhance the secretion of certain pro-inflammatory myokines. Cumulatively, these effects may ultimately lead to a reduction in muscle mass [30]. A decrease in muscle mass, in turn, can result in diminished physical activity capacity and reduced energy expenditure, thereby exacerbating obesity. On the other hand, changes in the secretion of pro-inflammatory myokines, characterized by an upregulation of myostatin levels and a downregulation of irisin levels [31], impede the growth and differentiation of muscle cells [32]. Moreover, these alterations are closely linked to suboptimal browning of white adipose tissue [33], ultimately contributing to an increase in adiposity. Notably, the insulin resistance and chronic inflammation engendered during these processes represent crucial pathophysiological mechanisms of T2D. Conversely, the chronic hyperglycemia, AGEs, and micro- and macrovascular complications inherent in diabetes can further exacerbate DO, thus perpetuating a new vicious cycle [34].

4. Comprehensive Management of DO and T2D

When T2D presents in conjunction with DO, hypoglycemic therapy serves as the cornerstone of the overall treatment strategy. This is attributable to the fact that persistent hyperglycemia inflicts damage on multiple systemic functions, precipitating a spectrum of acute and chronic complications that substantially compromise the patient's health. Nonetheless, the scientific management of body composition is of equal significance. In individuals with DO, the coexistence of excessive fat accumulation and muscle mass reduction is prevalent. This dysregulated body composition exacerbates metabolic derangements and insulin resistance, creating a vicious cycle. Consequently, proactive reduction of fat mass can mitigate the body's inflammatory response and enhance insulin sensitivity. Simultaneously, augmenting muscle mass bolsters the body's metabolic capabilities, thereby promoting the uptake and utilization of blood glucose. Employing these dual-targeted strategies is essential for a comprehensive improvement in the patient's condition, enhancement of their quality of life, and minimization of the risk of complications.

4.1. Regulation of Lifestyle

Energy restriction and physical activity are of paramount importance in ameliorating DO. A randomized controlled trial carried out in Taiwan Region, China, implemented interventions of resistance exercise, aerobic exercise, or a combination of both, twice weekly over 8 weeks, among subjects afflicted with DO. A control group was concurrently established. The findings revealed that the elderly participants in the training groups all exhibited an increment in muscle mass, along with a decrease in total fat mass and visceral fat area. Notably, the resistance-

exercise group demonstrated superior grip-strength performance [35]. A meta-analysis further corroborated that calorie and fat control, in conjunction with resistance exercise, can effectively address DO [36].

4.2. Pharmacological Interventions

At present, there is no globally accepted pharmacological treatment protocol for DO. Nevertheless, a substantial number of clinical trials and basic research studies have been conducted on medications aimed at improving body composition. Liraglutide, an antidiabetic agent, has been reported to potentially reduce body weight while simultaneously increasing the skeletal muscle mass index [37]. Vitamin D supplementation has demonstrated efficacy in improving muscle function [38]. Anamorelin, a ghrelin receptor agonist primarily utilized in the management of cancer cachexia, was the subject of a Phase III clinical trial. The results indicated that Anamorelin significantly augmented the lean body mass of patients with advanced non-small-cell lung cancer; however, it did not lead to an increase in grip strength [39]. In a Phase II clinical trial involving 75 overweight or obese patients with T2D, Bimagrumab, which acts by blocking the activin type II receptor, was found to significantly decrease fat mass, increase muscle mass, and enhance metabolic parameters within 48 weeks [40].

5. Limitations

This study has several limitations. Firstly, when discussing the prevalence of DO, it was found that there is still a lack of a unified diagnostic standard for DO in this field. This deficiency has resulted in substantial disparities in the reported prevalence rates across different studies, rendering it challenging to draw consistent conclusions. Secondly, concerning the exploration of the interaction mechanisms between DO and T2D, there is a shortage of research on more specific signal transduction pathways. Looking ahead, there is an urgent need for further research endeavors. These should focus on establishing a unified diagnostic standard for DO, which would enhance the comparability and reliability of future studies. Additionally, efforts should be made to identify novel signal transduction pathways associated with DO and T2D. Such advancements would not only deepen our understanding of the underlying pathophysiology but also enable the development of more targeted and precise management strategies for individuals affected by these conditions.

6. Summary

In general, DO refers to a condition characterized by reduced muscle function in combination with obesity. Compared to SO, the detection methods for DO are simpler, and DO can better predict poor prognoses in elderly patients with T2D. There is a mutually promoting relationship between DO and T2D, as they share common pathophysiological mechanisms such as reduced insulin sensitivity and chronic low-grade inflammation. DO can also exacerbate cognitive impairment

and the decline in activities of daily living in elderly patients with T2D. Therefore, controlling the occurrence and progression of DO through diet, exercise, and relevant drug therapies plays a crucial role in the prognosis of elderly patients with T2D.

Conflicts of Interest

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

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