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REVIEW

Sarcopenic obesity: research advances in pathogenesis and diagnostic criteria

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Abstract

Sarcopenic obesity (SO) refers to an obesity disease accompanied by low skeletal muscle quality, strength and/or function, which is more common in the elderly and seriously affects their quality of life and can lead to falls, unstable walking, balance disorders and fractures in the elderly. The increase in aging populations and the various health problems and medical costs associated with SO have aroused widespread concern in society. However, the pathogenesis of SO has not been fully clarified and the diagnostic criteria are not uniform, meaning that there are inconsistent data on the prevalence of SO and the potential correlation between SO and health outcomes. Therefore, we review the research progress on delineating the pathogenesis and diagnostic criteria of SO, to assist in the early diagnosis and evaluation of SO and subsequent interventions.

Keywords Sarcopenia · Obesity · Sarcopenic obesity · Pathogenesis · Diagnostic criteria

Introduction

Sarcopenia is a common syndrome of middle-aged and elderly people characterized by a decline in muscle mass, strength and function [1]. Sarcopenic obesity (SO) is a combination of sarcopenia and obesity with an average incidence rate of approximately 5–10%, increasing with age and especially prevalent in those aged 80 years or more [2–4].

Sarcopenia and obesity have independent and cumulative adverse effects on the health of the elderly, but SO has a greater effect on the morbidity and mortality of metabolic diseases [5] and cardiovascular diseases [6] than sarcopenia or obesity alone, and is significantly correlated with cognitive impairment [7]. The medical sequelae from SO are far more serious than those resulting from simple sarcopenia or obesity, thus SO is considered a major global threat to health and well-being [8]. The world population is aging rapidly, such that by 2050 it is expected that 22% of the total population will be over 60 years old, and approximately 5% over 80 years old [9]. The consequent increase in people suffering from SO will lead to serious health problems in society. It is, therefore, crucial to gain a deeper understanding of the pathogenesis and diagnostic criteria of SO to provide strategies for its early screening, diagnosis and treatment.

Pathogenesis

The pathogenesis of SO is complex, involving multiple interacting factors such as age-related changes in body composition (increase in visceral fat and muscle fat, decrease in muscle), systemic chronic inflammation, insulin resistance (IR), lifestyle changes (diet, physical activity, etc.), molecular mechanisms (mediated by, for example, leptin, adiponectin, interleukin 6, interleukin 10, or myostatin). However, a

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consistent medical view of the direction and extent of the causal relationships between these factors remains to be determined.

Changes in body composition related to age

SO is closely related to age-related changes in body composition. Aging is accompanied by changes in physiology and body composition, such as redistribution of muscle and adipose tissue. The amount of fat increases gradually with age, reaching a peak between 60 and 75 years old, which may progress to obesity. The proportions of visceral fat and intramuscular fat also increase with age, while the proportion of subcutaneous fat decreases [10, 11]. With the increase of age, the muscle strength of limbs and respiratory muscles will gradually decrease, and the functions of standing up, squatting and jogging will decrease. Generally, muscle quality and strength begin to decline from about 30 years of age, and the rate of decline increases markedly after 60 years of age. In addition, with the increase of body fat content, muscle mass gradually decreases, thus reducing basic energy consumption, which may also be related to hormone changes, decreased physical activity and diet changes. The decrease in muscle mass can, in turn, accelerate the increase in adipose tissue, which tends to accumulate in the abdomen [12]. This phenomenon may also be related to chronic subclinical inflammation, which aggravates SO [13]. Adipose cells also infiltrate muscle tissue, reducing contraction efficiency and muscle strength, which may also lead to a decrease in physical activity level, resulting in neuromuscular dysfunction.

Chronic inflammation

Patients with SO are considered to be in a highly inflammatory state, which increases the risk of chronic diseases and oxidative stress, thus impairing insulin sensitivity and growth hormone secretion and then leading to muscle loss and the subsequent occurrence and progression of sarcopenia [14]. Obesity is the main cause of systemic low-level inflammation, especially visceral fat, which secretes a variety of different proinflammatory cytokines, such as interleukin 6 (IL-6), tumor necrosis factor-α (TNF-α) and C-reactive protein (CRP). These proinflammatory cytokines are related to the occurrence of SO [9]. In obese adipose tissue, adipocytes undergo hypertrophy, proliferation and activation, resulting in the accumulation of pro-inflammatory macrophages and other immune cells, as well as causing a production imbalance of various adipokines. These cells, together with cytokines and chemokines, which are both released by aging cells and immune cells, produce a local pro-inflammatory state. Chronic inflammation may also lead to compound resistance and some cardiovascular and metabolic complications, such as insulin resistance [15].

Inflammatory cytokines play a key role not only in muscle dynamic balance but also in clinical pathogenesis characterized by changes in protein metabolism. The increase in pro-inflammatory cytokines such as TNF-α, IL-1 and IL-6 leads to muscle collapse and increased protein decomposition [16]. A study of 4984 elderly people showed that compared with simple sarcopenia and obesity, the levels of inflammation-related factors in SO patients were significantly higher. This indicated that the SO was closely related to systemic inflammation [17]. Chronic systemic inflammation is considered the most important factor in the pathogenesis of SO.

Insulin resistance (IR)

IR is related to fat hyperplasia and muscle mass reduction. An individual usually develops IR when they have too much fat. The occurrence of IR will lead to hyperinsulinemia and an increase of compensatory mechanisms, which is usually manifest as increased insulin secretion by pancreatic cells. One of the main causes of IR is the accumulation of secondary products of fat metabolism in myocardial cells. Obesity and the ageing process generate high levels of reactive oxygen species (ROS), due to lipid toxicity and mitochondrial dysfunction, further promoting the development of IR [18]. The resulting imbalance between oxidation and antioxidant compounds leads to the activation of the stress pathways of, for example, c-jun N-terminal kinase (JNK), IκB kinase (IKK) and p38-mitogen-activated protein kinase (p38-MAPK), thus leading to inactivation of the insulin receptor and its substrate [19].

In addition, ROS also inhibit mitochondrial function, resulting in lipid toxicity and malignant circulation of IR; this may promote muscle catabolism in obese people and lead to muscle mass loss or reduction because insulin is a powerful signal for protein metabolism [20]. At the same time, muscle loss reduces the quality of insulin-responsive target tissues, thus further aggravating IR and promoting muscle atrophy and obesity [7]. Chronic inflammation can also lead to the occurrence of IR, which promotes the reduction of muscle mass and the increase of fat mass [21]. Research by Park [22] and other scholars has shown that IR is an independent related factor for muscle strength decline and is related to the accelerated loss of leg muscle strength and mass in elderly diabetic patients.

Lifestyle changes

Changes in lifestyle related to the onset of SO mainly include diet and lack of physical exercise. This includes malnutrition、vitamin D deficiency, insufficient protein intake and changes in intestinal microecology, which can

promote the reduction of muscle quality and further cause negative changes in muscle strength and muscle function. Recent evidence shows that there is a potential link between individual, macro- and micro-nutrient intake and SO [23]. Chronic malnutrition may promote the development of SO by inducing hyperglycemia, low insulin levels and low protein synthesis. Therefore, SO may be prevented by increasing dietary fiber, reducing intake of simple carbohydrates and maintaining good protein intake [24].

Vitamin D is believed to play an important role in protein synthesis and muscle contractility regulation of muscle fibers. Low vitamin D results in decreased muscle anabolism and it is correlated with decreased insulin secretion, myofibrillar degradation and subsequent muscle protein turnover [25]. Changes in intestinal microecology cause the occurrence and development of sarcopenia and SO mainly through the effects of intestinal flora on fat metabolism, bile acid produced by intestinal bacterial metabolism, intestinal bacterial translocation, and intestinal bacteria on vitamin synthesis [26, 27]. Only low-level sports activities are undertaken by many otherwise sedentary elderly persons, promoting the development of obesity, which may further aggravate the reduction of muscle strength [28]. Although there is no mechanistic link of lifestyle changes with chronic inflammation and IR and the pathogenesis of SO, such changes can affect chronic inflammation and IR in a variety of ways, thus playing a synergistic role. Changes in lifestyle may also lead to weight cycling, which is a risk factor for low muscle mass and strength in a population of males and females with obesity [29].

Molecular mechanism

Some molecules such as pro-inflammatory factors (IL-6, IL-1, TNF-α) cause SO through chronic inflammation. In addition, other molecules such as adiponectin, leptin, muscle somatostatin, growth hormone, sex hormones (testosterone and estrogen), insulin and glucocorticoid have functions that can be related to SO. For example, adiponectin is released by adipose tissues, and increases the sensitivity of insulin and promotes fatty acid oxidation by promoting glucose uptake in skeletal muscle and adipose tissues [30]. In addition, adiponectin can reduce the formation of type-I oxidized muscle fibers in skeletal muscle. These effects are realized via the activation of the AMP-activated protein kinase (AMPK) signal pathway [31].

There is a strong negative correlation between exercise-induced weight loss and plasma adiponectin concentration and fat mass, while a change of leptin level is positively correlated with exercise-induced weight loss. Leptin can increase the expression of transcription factors related to muscle atrophy in the nucleus of muscle cells, and reduce muscle mass, muscle strength and muscle function [32]. Myostatin is a negative regulator of skeletal muscle growth. The mechanisms by which it inhibits skeletal muscle include the binding activin type II receptors A and B, accelerating phosphorylation of Smad2 and Smad3, up-regulating genes involved in reducing proliferation and differentiation of skeletal muscle precursor cells, and enhancing protein degradation pathways of mature muscle fibers. Muscle somatostatin can also produce ROS in skeletal muscle cells through TNF, thus inducing oxidative stress [33]. In addition, many obesity-related molecules participate in the pathogenesis of SO. These molecules act in the elderly through synergistic or antagonistic pathways, directly or indirectly affecting muscle loss and fat increase.

Diagnostic criteria

Diagnostic criteria for sarcopenia

At present, there are no unified diagnostic criteria for sarcopenia. The commonly used diagnostic criteria for epidemiological investigation include the Baumgartner diagnostic criteria and those of the European Working Group on Sarcopenia in Older People (EWGSOP), the International Sarcopenia Working Group on Sarcopenia (IWGS), the Asian Sarcopenia Working Group for Sarcopenia and the Foundation for the National Institutes of Health Sarcopenia Project (FNIHSP) [34]. Aside from the Baumgartner diagnostic criteria, which evaluate only muscle quality, all the criteria comprehensively evaluate muscle quality, muscle strength and muscle function. Muscle mass can be measured by dual X-ray absorptiometry (DXA), Computed tomography(CT), Magnetic Resonance Imaging (MRI), bioelectrical impedance analysis (BIA), total or partial body potassium per fat-free soft tissue, anthropometric measures, etc. and currently the most commonly used are DXA or BIA [35]. Muscle strength is assessed by handgrip, muscle function is assessed by gait speed or short physical performance battery (SPPB), and the evaluation method for diagnostic criteria are the same [36]. All diagnostic criteria except those of the FNIHSP are evaluated according to levels of relative appendicular skeletal muscle (RASM), which is the ratio of the square of appendicular skeletal muscle and height^2(ASM/ht^2); muscle mass is considered "low" if it is two standard deviations less than the control mean of healthy young people of the same sex [37, 38]. The FNIHSP criteria evaluate muscle mass as the ratio of Appendicular Lean Mass (ALM) to Body Mass Index (BMI) [39]. In October 2018, the EWGSOP revised its consensus and proposed a new operational definition of sarcopenia, the EWGSOP2 [40]. EWGSOP2 focuses on low muscle strength as a key characteristic of sarcopenia, uses detection of low muscle quantity and quality to confirm the sarcopenia diagnosis, and identifies poor

physical performance as indicative of severe sarcopenia. A comparison of the various diagnostic standards is shown in Table 1.

The correct evaluation of the above-recognized sarcopenia diagnostic criteria is time-consuming and costly, so the short-term and inexpensive screening tools are welcomed by clinicians and other personnel working in the field of geriatrics. At present, there is no unified standard for early screening of sarcopenia at home and abroad. Epidemiological investigation and common clinical early screening methods include simple five-item scoring questionnaire(SARC-F), gait speed, Body Mass Index (BMI), SPBB, abdominal routine CT examination, etc. [41–43]. The SARC-F scale [44, 45] is considered to be the first simple step in the grading screening process, and is suitable for simple comparison of clinical therapeutic effects, scientific research experimental observation and community large-scale epidemiological investigation, and can be regarded as another new diagnostic standard of sarcopenia. The latest research on SARC-F shows that [46], SARC-F is an excellent test for eliminating muscle function damage and muscle atrophy. Compared with the diagnostic criteria proposed by authoritative sarcopenia working groups such as EWGSOP, IWGS, FNIHSP, the detection rate of SARC-F has higher overlap and higher sensitivity and specificity for muscle function test. And the research found that [47] that the SARC-F combined with the calf circumference (SARC-CalF) improved the specificity and diagnostic accuracy of SARC-F.

Diagnostic criteria for obesity

Obesity refers to excessive accumulation of fat in the body and/or abnormal distribution, usually with weight gain. International indicators for the diagnosis of obesity include BMI, waist circumference (WC), waist-to-hip ratio (WHR), body fat rate (BF%), etc. [51]. There are three commonly used diagnostic criteria for obesity: Methods 1, 2 and 3. Method 1 uses the obesity determination criteria in the Guidelines for the Prevention and Control of Overweight and Obesity in Chinese Adults, which is BMI $\geq$ 28 kg/m^2; Method 2 is WHO-recommended and uses the BF% to determine obesity, which is defined as BF% $\geq$ 25% for men and $\geq$ 35% for women; Method 3: uses the percentage of BF % exceeding the 60th percentile of a study population. This is a more accurate and important indicator for evaluating obesity than BMI [52, 53], and the cut-off point is especially accurate for diagnosing the obesity prevalence in a study population. Therefore, the percentage of BF% exceeding the 60th percentile of a research population is currently the most commonly used diagnostic criterion for obesity.

Diagnostic criteria for SO

The diagnostic criteria for SO are a combination of the diagnostic criteria of sarcopenia and obesity. The diagnostic criteria for SO vary with the changes in the diagnostic criteria for sarcopenia and obesity. Before 2010, the diagnostic criteria of SO were mainly evaluated by muscle mass combined with obesity. Baumgartner [54] uses DXA and ASM/ht^2 to diagnose SO, SO is diagnosed when the relative skeletal muscle mass index is lower than the average value of healthy young people by 2 standard deviations and the percentage of body fat exceeds 60% of the population of the same age. Davison et al. [55] used anthropometric measures and BIA to measure human body composition and indirectly measured the body fat and fat removal content of human body, thus calculating the body muscle mass and fat mass to define SO: that is, the body fat content exceeds the population level by 60%, and the muscle mass is lower than the population level by 60%. However, since EWGSOP [48] first proposed the comprehensive diagnostic criteria and stages of sarcopenia in 2010, the diagnosis of SO is not limited to the evaluation of muscle mass combined with obesity, but includes the comprehensive evaluation of

Table 1 Comparison of diagnostic criteria for various sarcopenia

Diagnostic criteria	Skeletal muscle mass		Muscle strength	Muscle function
Baumgartner [37]	ASM/ht^2 $\leq$ 2 standard deviations for healthy young people of the same sex		/	/
EWGSOP [48]	DXA:ASM/ht^2 $\leq$ 7.26 kg/m^2 (M) DXA:ASM/ht^2 $\leq$ 5.44 kg/m^2 (F)		Handgrip < 30 kg (M) handgrip < 20 kg (F)	Gait speed < 0.8 m/s (4 m) or < 1.0 m/s (6 m)
IWGS [49]	DXA:ASM/ht^2 $\leq$ 7.23 kg/m^2 (M) DXA:ASM/ht^2 $\leq$ 5.67 kg/m^2 (F)		Can't stand up from a chair	Gait speed < 1.0 m/s (6 m)
AWGS [50]	DXA:ASM/ht^2 < 7.0 kg/m^2 (M) DXA:ASM/ht^2 < 5.4 kg/m^2 (F)	BIA:ASM/ht^2 < 7.0 kg/m^2 (M) BIA:ASM/ht^2 < 5.7 kg/m^2 (F)	Handgrip < 26 kg (M) Handgrip < 18 kg (F)	
FNIHSP [39]	ALM/BMI < 0.789 (M) ALM/BMI < 0.512 (F)		Handgrip < 26 kg (M) Handgrip < 16 kg (F)	/
EWGSOP2 [40]	DXA/BIA:ASM/ht^2 < 7.0 kg/m^2 (M) DXA/BIA:ASM/ht^2 < 5.5 kg/m^2 (F)		Handgrip < 27 kg (M) Handgrip < 16 kg (F)	Gait speed $\leq$ 0.8 m/s (4 m)

muscle mass, muscle strength and muscle function, and is determined in combination with obesity. The prevalence rate of SO obtained by different combination methods and the different diagnostic cut point is quite different, and even if the prevalence rate is approximately the same, the overlap rate is very low [56]. As the latest research shows [57], under the same diagnostic criteria for sarcopenia, BF% is a method with the highest detection rate of SO compared with BMI or WC. Therefore, the SO diagnostic criteria most relevant to the population under study (in terms of, for example, age or ethnicity) should be selected.

In summary, SO is a multi-faceted medical disorder with poor prognosis, which can lead to different health consequences. The increase of aging populations and concomitant increasing incidence of SO has aroused widespread concern in healthcare circles. The pathogenesis of SO is complex, and mainly involves chronic inflammation and IR, but with these and other factors interacting with each other. It is thus very important that the diagnostic criteria for SO are unified as much as possible to ensure that studies of SO, its effects, and ways it may be ameliorated or prevented can be strengthened, to thus improve the health of the elderly.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

Ethical approval This article does not contain any studies with human participants performed by any of the authors.

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