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Research Progress on the Correlation between Sarcopenia and Hypertension in Elderly People

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Abstract: *With the gradual aggravation of global aging problem, the prevalence of chronic diseases and mortality in elderly people are rapidly increasing, resulting in a massive disease burden. Managing chronic diseases is an important initiative to promote healthy aging. Sarcopenia and hypertension are both diseases associated with aging, and studies have shown that there is a significant relationship between them. In this paper, we will review the correlation studies on sarcopenia and hypertension in recent years, aiming to explore the possible links between them, and provide more effective strategies for prevention and treatment.*

Keywords: *Sarcopenia; Hypertension; Elderly*

1. Overview of sarcopenia

1.1 Definition of sarcopenia

The term sarcopenia was originally come out in 1989 by Rosenberg, he described sarcopenia as a progressive decline in muscle mass and strength during aging after comparing the thigh skeletal muscles of older and younger women [1]. In 2016, sarcopenia was formally recognized as a muscle disease when it was assigned the disease code ICD-10-CM (M62.84) in International Classification of Diseases [2]. The consensus on sarcopenia has been updated continuously by different regional working groups due to the ongoing development of related clinical and basic research [3]. The European Working Group on Sarcopenia in Older People (EWGSOP) released the most recent consensus in 2018 [4]. In 2019, Asian Working Group for Sarcopenia (AWGS) expanded upon the European consensus by incorporating the specific characteristics of the Asian population and subsequently published the Asian consensus [5].

The AWGS 2019 consensus defines sarcopenia as a progressive and generalized skeletal muscle disease that is characterized by a decline in muscle function and mass associated with aging. Sarcopenia is associated with the risk of adverse health outcomes, including falls, fractures, physical disability, and death [5]. The occurrence of sarcopenia is expected to rise as the population ages [6], resulting in a substantial strain on the healthcare system [7]. This issue is becoming increasingly recognized as a public health concern that demands immediate attention and intervention [8].

1.2 Diagnostic criteria for sarcopenia

The diagnostic criteria for sarcopenia are still in controversy due to variations in anthropometrics, cultural habits, and lifestyles among populations in different regions. The widely accepted international diagnostic criteria include the EWGSOP criteria [4, 9], the AWGS criteria [5, 10], and the Sarcopenia International Working Group (IWGS) criteria [11].

The reduction in muscle mass was the primary focus of early investigations into sarcopenia. The first diagnostic criteria for sarcopenia were proposed by Baumgartner in 1998, he defined loss of height-adjusted appendicular skeletal muscle mass (ASM) as sarcopenia. Additionally, Baumgartner established a diagnostic threshold of two standard deviations below the mean of young controls. With the deepening of research, the consensus on sarcopenia has been continuously updated among regional working groups [12]. In 2010, the European Working Group on Sarcopenia in Older People introduced a novel diagnostic criterion for sarcopenia. This criterion involves a reduction in muscle mass, along with a decrease in muscle strength and/or physical performance. Subsequently, regional working groups on sarcopenia developed the diagnostic criteria for sarcopenia, taking into account the specific characteristics of the

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regional populations [9].

Currently, the prevailing consensus indicates that the assessment of sarcopenia in older persons should primarily focus on the assessment of muscle mass, muscle strength, and physical performance. According to the EWGSOP 2010 criteria, it is recommended to utilize physical performance, specifically step speed, as a screening indicator for sarcopenia. Individuals who have a step speed of ≦0.8 m/s are deemed to be at risk of developing sarcopenia. The AWGS 2014 criteria is similar to the EWGSOP 2010 criteria, but it proposes muscle strength (specifically grip strength) or physical performance (specifically step speed) as potential screening indicators for sarcopenia [10]. Based on more subsequent research evidences, the EWGSOP made an update to the sarcopenia consensus in 2018 [4], and the EWGSOP 2018 criteria emphasized decreased muscle strength and introduced the concept of severe sarcopenia. The criteria suggest that a decrease in muscle strength is suggestive of possible sarcopenia, while an accompanying decrease in muscle mass confirms a diagnosis of sarcopenia. Furthermore, a diagnosis of severe sarcopenia is established when there is a simultaneous drop in physical performance. The AWGS 2019 criteria closely resemble the EWGSOP 2018 criteria, which classify sarcopenia as a condition characterized by reduced skeletal muscle mass, together with diminished muscle strength or physical performance [5]. The diagnostic thresholds for reduced skeletal muscle mass are as follows: 1) Skeletal muscle index (SMI) of the extremities measured by bioelectrical impedance analysis (BIA), with the suggested cutoff values of 7.0 kg/m^2 in men and 5.7 kg/m^2 in women. 2) SMI measured by dual-energy X-ray absorptiometry (DXA), the cutoff values were 7.0 kg/m^2 in men and 5.4 kg/m^2 in women. The diagnostic thresholds for reduced muscle strength were grip strength below 28 kg for males and below 18 kg for females. Diagnostic cutoffs for reduced physical performance: 1) 6-meter step speed <1.0 m/s; 2) 5-sit-up test ≥12 seconds; 3) a score of 9 points on the Short Physical Performance Battery (SPPB).

2. Epidemiology of sarcopenia and hypertension

The prevalence of sarcopenia varies widely among studies due to differences in target populations and diagnostic criteria. A meta-analysis of 151 studies including 692,056 older adults showed that the overall prevalence of sarcopenia in older adults worldwide ranged from 10% to 27% [13]. According to a study employing the EWGSOP2010 diagnostic criteria, the occurrence of sarcopenia among elderly males and females (with a mean age of 67 years) within the UK population was found to be 4.6% and 7.9%, respectively [14]. Another study using EWGSOP2018 diagnostic criteria showed that the prevalence of sarcopenia in older men and women in the Spanish community was 21.1% and 18.3%, respectively [15]. The prevalence of sarcopenia among older adults in Asia was about 6.8%- 25.7% [5]. Specifically, the prevalence of sarcopenia among community-dwelling older adults in Japan was 9.9% (of which 9.8% were males and 10.1% were females) [16], the incidence among the elderly people residing in the community in Singapore was found to be 32.2% (of which 33.7% were males and 30.9% were females) [17], and the prevalence of sarcopenia among community-dwelling elderly in China was about 18.5% (of which 16.3% were males and 19.9% were females) [18].

Hypertension is a prevalent chronic condition that is strongly linked to a higher likelihood of developing cardiovascular illness, such as coronary heart disease, stroke, and peripheral vascular disease [19], which is the leading cause of death in the global population. The prevalence of hypertension in the elderly population exceeds 60% [20] and continues to increase with overall aging and unhealthy lifestyles accompanying rapid economic growth. Consequently, future cardiovascular events can still pose a substantial health risk [21].

Previous studies have suggested that the existence of hypertension is a contributing factor to the heightened likelihood of sarcopenia in elderly individuals. Furthermore, those with hypertension are more prone to developing sarcopenia, with prevalence rates varying between around 18.0% and 49.5% [22, 23]. There is a significant correlation between a decline in muscle mass and hypertension in older individuals [24]. And muscle mass tends to diminish as hypertensive patients age, with a reduction of roughly 0.5%- 1.0% every year after the age of 70 [25]. Previous studies have documented that elderly hypertensive patients exhibit notably reduced muscle strength and physical function compared to the non-hypertensive population [26, 27]. Additionally, a persistent hypertensive state is linked to a decline in walking speed among the elderly, with a decrease of approximately 2.3 cm/s per year in hypertensive patients.

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3. Interaction between sarcopenia and hypertension.

3.1 Changes in muscle in hypertensive patients

An international investigation has shown that hypertensive individuals exhibit a notably reduced skeletal muscle oxygenation capacity compared to the normotensive group. Furthermore, this decrease in skeletal muscle oxygenation capacity was linked to a diminished degree of grip strength [28]. The possible mechanisms that produce this change are as follows: 1) The sustained state of hypertension leads to remodeling of the microvessels because of mechanical stress on the vessel wall as well as inflammation, resulting in narrowing of the lumen of the microvessels and a decrease in the density of the capillaries. Skeletal muscle microvessels with impaired structure and function have a negative impact on the transport and utilization of oxygen, resulting in a reduction in muscle oxygenation capacity. 2) Additionally, myofibrillar mitochondrial dysfunction in hypertensive patients leads to enhanced oxidative stress and mitochondrial oxidative phosphorylation uncoupling, affecting cellular energy utilization and ultimately leading to a decrease in muscle oxygenation capacity.

A longitudinal investigation conducted on African males revealed notably elevated levels of diminished skeletal muscle mass among those who had just developed hypertension, as compared to those who did not have hypertension [29]. Furthermore, intermuscular fat infiltration was significantly higher in patients with new-onset hypertension than in non-hypertensives. The primary factor contributing to the decline in skeletal muscle mass is the decrease in the number of myofibers and the atrophy of myofiber volume, particularly in type II myofibers [25, 30]. The expression of myosin heavy chain (MyHC) undergoes alterations as the disease advances, resulting in an elevation in the percentage of chronic oxidized myofibers (MyHC I) and a reduction in the percentage of rapidly oxidized myofibers (MyHC IIA) and intermediate myofibers (MyHC IIX). The depletion of type II myofibers is also linked to a reduction in skeletal muscle satellite cells, which possess stem cell characteristics and play a role in muscle repair and regeneration following injury and disease [31]. Type II fibers are more prone to atrophy as a result of inflammatory and hypoxic stimuli due to their lower concentration of peroxisome proliferator-activated receptor gamma coactivator-1 alpha (PGC-1α) compared to type I fibers. In addition, fatty degeneration of muscle due to adipose infiltration of muscle tissue may cause local muscle insulin resistance, inflammation, and atrophy. The series of changes described above ultimately result in skeletal muscle dysfunction and sarcopenia [32].

3.2 Effect of muscle changes on blood pressure in hypertensive patients

The compromised structure and function of skeletal muscle microvasculature in individuals with hypertension result in elevated vascular resistance both at rest and during periods of heightened metabolic activity, such as exercise [28]. Hypertensive patients require a more significant increase in blood pressure to attain a level of muscle oxygenation comparable to that observed in individuals without hypertension after exercise. This is primarily due to a reduction in skeletal muscle oxygenation capacity, which is associated with the extent of microvascular congestion and aortic blood pressure. In addition, skeletal muscle cell mitochondrial dysfunction in hypertensive patients leads to increased reactive oxygen species production, reduced endothelial nitric oxide synthase (NOS), and decreased NO bioavailability, which promotes endothelial dysfunction in the vascular wall, further contributing to the vicious cycle of elevated blood pressure. In conclusion, dysregulation of muscle oxygen delivery and utilization as well as impaired skeletal muscle microvascular structure and function in hypertensive patients contribute to high blood pressure during exercise.

Muscle serves as a significant location for insulin-mediated glucose metabolism. In hypertensive individuals, the reduction of skeletal muscle mass and the infiltration of intermuscular fat can result in localized muscle insulin resistance. This condition has the potential to elevate blood pressure due to the induction of inflammation and oxidative stress [29]. In addition, the depletion of skeletal muscle in individuals with hypertension will result in a reduction in the release of myokines, which are cytokines generated during muscle contraction and have the functions of participating in the repair of muscle damage and improving insulin resistance, etc. The decrease in myokines will further aggravate the related damage in hypertensive patients [33].

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4. Etiology and pathogenesis of sarcopenia combined with hypertension

4.1 Imbalance of myosin metabolism

The normal structure and function of skeletal muscle are based on the dynamic balance of myofibrillar protein synthesis and catabolism, and the compromised production of myosin and excessive breakdown of myosin are significant factors that contribute to the development of hypertension in conjunction with sarcopenia.

Growth hormone (GH) and insulin-like growth factor 1 (IGF-1) are important regulators involved in myosin synthesis and metabolism [30]. As individuals age, the activity of the GH/IGF-1 axis diminishes, resulting in a decline in myosin synthesis. Consequently, this leads to a reduction in muscle mass, which is thought to be an important pathogenesis of sarcopenia. Hypertensive patients have notably reduced levels of GH compared to normotensive patients, whereas there is no substantial disparity in IGF-1 levels between the two groups [25]. The administration of GH via subcutaneous injection has been shown to enhance skeletal muscle mass in older males [34]. Hypertension is considered to be a chronic inflammatory disease, and individuals with hypertension exhibit considerably elevated levels of inflammatory factors compared to those with normal blood pressure [35]. Inflammatory factors such as C-reactive protein (CRP), tumor necrosis factor-α (TNF-α), and interleukin 6 (IL-6) have been shown to increase myosin catabolism. In addition, IL-6 can further affect myosin metabolism by down-regulating IGF-1 levels [25].

4.2 Inflammatory response and oxidative stress

There exists a substantial correlation between elevated levels of inflammatory factors and the occurrence of skeletal muscle injury among elderly individuals with hypertension [36]. Increased chronic inflammation and catabolic cytokines in the body are the main risk factors for hypertension, and the level of inflammation is higher in the elderly with hypertension [37]. Inflammatory factors such as CRP, TNF-α, and IL-6 affect the blood supply to the skeletal muscles by damaging the endothelium of the microvessels, which may lead to the occurrence of sarcopenia [38].

Oxidative stress, in concert with chronic inflammation, is involved in the development of hypertension combined with sarcopenia [39]. Systemic levels of oxidative stress are elevated in hypertensive patients, especially those with muscle atrophy. The accumulation of reactive oxygen and nitrogen species (RONS) is a major cause of skeletal muscle mass and functional impairment. On the one hand, RONS disrupts the metabolic balance of myosin, resulting in a loss of muscle mass. On the other hand, it affects the neuromuscular junction by reducing the release of acetylcholine from the synaptic gap, thus affecting the generation of action potentials in the muscle membrane. Additionally, persistent oxidative stress changes the morphology of neuromuscular junctions, affecting the excitatory-contractile coupling and leading to a decrease in calcium release from the sarcoplasmic reticulum.

4.3 Mitochondrial dysfunction

Mitochondria play an important role in energy supply, maintenance of redox homeostasis, and regulation of catabolic pathways. They are also involved in regulating the plasticity of skeletal muscle fibers and maintaining myocyte activity [40]. The primary manifestations of mitochondrial dysfunction in the skeletal muscle cells of hypertensive patients include reduced mitochondrial density, decreased expression of mitochondrial components and transcription factors involved in mitochondrial biogenesis, and impaired assembly of respiratory complexes. These dysfunctions are primarily characterized by the disruption of the respiratory chain, decreased oxidase activity, oxidative stress, and increased apoptosis-related phenomena [28]. The inflammatory response is significantly associated with mitochondrial dysfunction in myocytes, in which TNF-α plays an important role. On the one hand, TNF-α produces a large amount of nitric oxide (NO) by stimulating inducible nitric oxide synthase (iNOS), which leads to cellular damage and tissue necrosis, ultimately promoting the occurrence and progression of inflammatory diseases. On the other hand, TNF-α triggers mitochondrial dysfunction through a cell membrane death receptor mediated signaling pathway triggering apoptosis. In conclusion, mitochondrial dysfunction in hypertensive patients triggers chronic inflammation and oxidative stress, which in turn negatively affect mitochondrial function. These factors collectively contribute to the occurrence of muscle fiber atrophy in hypertensive patients [41].

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4.4 Overactivity of the RAS system

The renin-angiotensin system (RAS) is a major regulator of blood pressure, and its overactivity underlies the development of hypertension. In addition, the RAS system is involved in the regulation of muscle mass. The regulatory function of the RAS system encompasses a wide range of processes, including protein turnover, apoptosis, and collagen metabolism. The classical RAS axis is responsible for inducing protein degradation by accumulating intracellular reactive oxygen species (ROS) and exerting a detrimental impact on the pro-synthetic pathway of the insulin-like growth factor receptor (IGF-R), ultimately resulting in muscle atrophy [42, 43].

4.5 Insulin resistance

Insulin resistance underlies hypertension, and insulin resistance-induced hyperinsulinemia can lead to structural changes in the kidneys, such as glomerular hypertrophy and focal stage glomerulosclerosis, which significantly reduces the glomerular filtration rate and cause elevated arterial blood pressure [29]. Untreated essential hypertension patients exhibit elevated fasting and postprandial insulin levels compared to normal controls. Additionally, there is a clear correlation between plasma insulin concentrations and blood pressure levels. These alterations may be associated with chronic inflammation and oxidative stress. Inflammatory factors, such as TNF-alpha and IL-6, induce serine phosphorylation of insulin receptor substrate-1 (IRS-1), which prevents normal IRS-1 tyrosine phosphorylation of IRS-1, resulting in reduced binding of IRS-1 to the insulin receptor and ultimately inhibiting the insulin downstream signaling pathway. Insulin resistance accelerates muscle loss by interfering with the mTOR signaling pathway, leading to the development of sarcopenia, while muscle is the main tissue for insulin-mediated glucose utilization, and the reduction of skeletal muscle mass exacerbates insulin resistance, thus forming a vicious circle [44].

4.6 Malnutrition and muscle-wasting atrophy

Elderly hypertensive patients suffer from inadequate energy intake and increased protein catabolism due to issues such as loss of taste, loss of appetite, impaired feeding and absorption, and drug-related anorexia. These factors contribute to skeletal muscle atrophy, reduced exercise capacity, and a decline in overall quality of life [45].

Research has demonstrated that hypertensive patients experience a considerably greater decrease in walking pace compared to individuals with normal blood pressure. This indicates a negative association between hypertension and the elderly's capacity to engage in daily physical activity. Hypertension is a risk factor for atherosclerosis, and lesions of the peripheral and coronary arteries lead to a decrease in the body's mobility by affecting the tissue blood supply. In addition, cerebral white matter lesions in hypertensive patients are associated with worse mobility. Hypertensive patients have limited physical activity due to debilitation, malnutrition, and emotional factors. Prolonged bed rest or sedentary behavior can lead to muscle wasting atrophy [26, 46].

5. Co-interventions for sarcopenia and hypertension

5.1 Exercise intervention

Exercise training is a central strategy for preventing and delaying sarcopenia and also significantly improves disease progression in hypertensive patients. Resistance exercise training and endurance exercise training are two prevalent forms of exercise training [47].

Endurance exercise, alternatively referred to as aerobic exercise, mostly encompasses conventional physical activities such as brisk walking, jogging, cycling, swimming, and engaging in ball games. Endurance exercise training not only increases the number of mitochondria in myocytes and improves oxidase activity, but also improves intermuscular fat infiltration and reverses insulin resistance [48]. Furthermore, skeletal muscle contraction can release a range of myokines, including irisin, which has been demonstrated to play a role in controlling blood pressure. There is a prevailing consensus that individuals who attain a maximal power or heart rate of 60% to 80% of their symptom-limiting capacity during endurance exercise training are capable of attaining the intended therapeutic outcome. It is recommended that patients with sarcopenia combined with hypertension participate in endurance exercise training of moderate intensity or higher for 30 minutes on at least 3 days during the week [49].

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Resistance exercise is commonly used to increase muscle strength, and progressive resistance training is the most common form of exercise for older adults, including static squats against a wall, knee extensions, and isometric grip training. Resistance exercise training enhances the cross-sectional area of skeletal muscles and augments the quantity of type II muscle fibers, which improves muscle strength and physical function [47]. In addition, isometric exercise has been found to decrease blood pressure through the reduction of peripheral sympathetic vasoconstrictor activity, enhancement of oxidative stress, and inhibition of vascular endothelial function, among various other pathways [50]. A system overview comprising 121 randomized controlled studies has provided evidence that the implementation of progressive resistance training, conducted two to three times per week, yields considerable enhancements in the performance of activities of daily living among elderly individuals [51]. Hypertensive older adults who performed resistance exercise training had an average reduction in systolic blood pressure of 10-13 mmHg and diastolic blood pressure of 6-8 mmHg. The specific exercise program was as follows: It is recommended that four sets of 2-minute resistance exercise training be performed each time, with a maximum voluntary contraction of 20% to 50%. Each set should be followed by a 1-4-minute rest interval. And the exercise should be repeated 3-5 times a week for 4-10 weeks [50].

5.2 Nutritional support

Nutritional management is an important component of disease management in patients with hypertension combined with sarcopenia. Dietary protein supplementation can increase muscle mass in the elderly, which is an effective intervention for sarcopenia. Additionally, a diet that partially replaces carbohydrates with proteins can alleviate the blood pressure level of patients with hypertension [52]. It is recommended that the daily protein intake of elderly patients with hypertension combined with sarcopenia be increased to 1.2-1.5 g/kg body weight, evenly distributed among three meals [53].

Supplementation with leucine alone can enhance muscle mass in older adults with sarcopenia. Leucine is a key component responsible for stimulating postprandial muscle protein synthesis and is thought to have a specific positive effect on signaling pathways for muscle protein synthesis. In addition, the use of nutrients such as omega-3 fatty acids and vitamin D has the potential to enhance protein anabolism, hence improving muscle development and function in older adults [54, 55].

5.3 Exercise combined with dietary interventions

A study in older adults showed that protein supplementation during prolonged resistance exercise training significantly increased defatted body weight, cross-sectional area of type I and type II muscle fibers, and lower limb strength in older adults compared to resistance exercise training alone [56]. Exercise has been found to augment muscle protein synthesis by enhancing muscle sensitivity to anabolic processes mediated by insulin or amino acids. This effect reaches its maximum within a span of 3 hours following exercise. Therefore, it is advisable to ingest protein promptly after exercise in order to maximize its effects [53].

Research has demonstrated that combining creatine supplementation with progressive resistance training can enhance muscle mass and strength, indicating that creatine supplementation may have a preventive effect against sarcopenia in older individuals. Additionally, creatine has been found to lower homocysteine levels, making it potentially advantageous in the management of hypertension [57].

5.4 Medication

Currently, there are no specific medications approved for the treatment of sarcopenia, and the majority of research has been concentrated on enhancing muscle mass in individuals with sarcopenia. Selective androgen receptor modulators, myostatin antibodies, myostatin receptor blockers, fast-twitch troponin activators, and other drugs have garnered significant attention in the field of pharmaceuticals. These drugs are expected to increase the muscle mass of patients with sarcopenia by acting on the processes of myosin metabolism, myoblast differentiation, and proliferation to improve muscle strength and function. However, most of the results of the clinical studies only show an increase in muscle mass, with limited effects on improving muscle strength or physical function in patients [58].

A study in the United States showed that hypertensive older adults using angiotensin-converting enzyme inhibitors (ACEIs) had more significant improvements in physical function through exercise than those using other antihypertensive medications or those who did not, suggesting that ACEIs have a role in preventing and delaying disease in patients with hypertension and sarcopenia. This implies that

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ACEIs may have a positive role in preventing and delaying disease progression in patients with hypertension combined with sarcopenia [59].

6. Summary

In conclusion, a strong correlation exists between sarcopenia and hypertension, and the presence of hypertension independently increases the risk of sarcopenia in the elderly. Individuals with hypertension are susceptible to decreased skeletal muscle mass and dysfunction, and alterations in muscle structure and function exacerbate the damage associated with hypertension, creating a vicious circle. The pathogenesis of the interaction between sarcopenia and hypertension is still being explored, and more studies are needed to explore the relationship between the two. As the prevalence of sarcopenia and hypertension continues to increase with the aging of the population, it is advisable to promptly conduct screenings for sarcopenia among the elderly demographic. Furthermore, it is crucial to implement dietary and lifestyle interventions to prevent and postpone the development of sarcopenia and hypertension. Pharmacologic interventions for sarcopenia are still in the exploratory phase and need to be supported by more evidence of clinical application.

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