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Sarcopenic Obesity in the Elderly

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Abstract

In this mini-review pathophysiology, comorbidities, diagnosis and management of sarcopenic obesity (SO) are discussed. SO is a high risk geriatric syndrome more associated with osteoarthritis (OA), falls, dementia and increased cardiometabolic risk profiles than obesity alone. Decreased physical activity, low-grade chronic inflammation, oxidative stress and insulin resistance all of which being by-products of obesity and the aging process are involved. Easy diagnostic tools are not yet available and sophisticated DXA and SPECT scans are not always feasible. Prevention and resistance exercise programs combined with protein supplementation are the cornerstone of SO management. When male SO patients can be easily identified, treatment with selective androgen receptor modulators (SARMs) might be considered in the near future.

Introduction

The prevalence of obesity in combination with sarcopenia, the age-related loss of muscle mass and strength or physical function, is increasing in adults aged 65 and older. A major subset of adults over the age 65 is now classified as having sarcopenic obesity, a high risk geriatric syndrome. Moreover, recent estimates suggest that 37% of U.S. adults aged 65 year and older are obese [1]. The specific criteria for defining sarcopenic obesity (SO) are somewhat arbitrary and depend on the study cited [2]. Thus, the prevalence of SO varies from 4% to 84% in men and from 4% to 94% in women [3]. Older adults with SO have higher risks of mobility disability, cardiometabolic disease and mortality [4]. In this mini-review pathophysiology, comorbidities, diagnosis and management of SO are discussed.

Pathophysiology SO

Human aging is associated with a progressive decline of skeletal muscle mass. Several studies have suggested that muscle mass decreases by approximately 6% per decade after midlife [5]. Lower muscle mass results in decreased muscle strength [6]. There are significant differences among individuals in peak muscle mass, the age at which muscle loss starts, and the amount of muscle that is lost over time [7]. At the cellular level sarcopenia is accompanied by a loss of innervation and adaptive changes in the proportion of slow and fast motor units, as well as in the cross-sectional area of muscle fibers [8]. Several mechanisms have been linked to the development of sarcopenia [9]. Most mechanisms are also associated with visceral obesity, leading to a vicious circle of interacting risk factors. Insulin resistance plays an important role in obesity and results in muscle fiber atrophy and mitochondrial dysfunction [9,10]. Age-related changes in hormones play a pivotal role and affect the anabolic and catabolic processes in skeletal muscle [11,12]. Reduced androgen and estrogen levels decrease muscle mass and strength [12]. In addition, sarcopenia is an inflammatory state that is driven by proinflammatory cytokines and oxidative stress [13]. Oxidative stress modulates the expression of transcription factors, such as nuclear factor-kappa B (NF-kB), which enhances proteolytic pathways and increases the production of proinflammatory cytokines [14]. Tumor necrosis factor-alpha (TNF-alpha) impairs protein synthesis in skeletal muscle by altering translation initiation, which may contribute to sarcopenia [15]. Higher levels of interleukin-6 (IL-6) and C-reactive protein (CRP) are associated with a greater decline in muscle strength [16]. Myostatin (growth differentiation factor 8) inhibits muscle cell growth and differentiation and could be a potential mediator of sarcopenia [17].

Excess nutrient availability and tissue delivery, particularly saturated fat and glucose further contribute to the cluster of insulin resistance, inflammation and oxidative stress that occur in obesity. Resulting adipose tissue dysfunction develops in response to the enhanced demand for lipid storage [18-20]. These changes may result in an “anabolic resistance state” to nutrients where the muscle protein synthesis from nutrients is blunted [21-24]. Mtochondrial changes are observed in obese skeletal muscle until late stages [25,26]. Their onset may however exacerbate oxidative stress and related metabolic cascades leading to insulin resistance and catabolism. Potential reduction in ATP production may also result in low muscle strength and endurance capacity [25,26]. Stem cell dysfunction leads to functionally altered muscle stem cells that may undergo adipocyte differentiation and accompanied fat accumulation [27-29]. Low physical contributes to a positive energy balance [26]. Progressive reduction of physical activity is further observed with disease progression due

to worsening obesity and musculoskeletal disorders with direct negative impact on muscle protein turnover and muscle oxidative and performance capacity [30,31].

SO and comorbidities

Osteoarthritis

Individuals with osteoarthritis (OA) may exhibit a higher prevalence of SO compared with rheumatoid arthritis (RA)-[32]. Misra et al. studied a large cohort from the Multicenter Osteoarthritis (MOST) Study, a longitudinal cohort of individuals with or at risk for knee OA. Based on body composition from whole body Dual Energy X-Ray (DXA) subjects were categorized as obese, sarcopenic obese (SO), sarcopenic and non-sarcopenic obese.

Among 1633 subjects with radiographic knee OA at baseline, significant increased risk of incident radiographic knee OA was found among obese (women RR 2,29;95%CI 1,64-3,20;men RR 1,73;95% CI 1,08-2,78) and SO women (RR 1,91;95%CI 1,73-3,11) but not men (RR 1,74;95% CI 0,68-4,46) .Sarcopenia was not associated with knee OA risk (women RR 0,96;95% CI 0,34-1,30). It was concluded that in this large cohort population, body composition based obesity and SO but not sarcopenia was associated with knee OA risk. Weight loss strategies for knee OA should focus on obesity and SO [33].

Sarcopenia obesity (SO) results in more physical disability than sarcopenia alone or obesity alone and has been strongly implicated in both risk of OA and frailty [34-36]. Total joint arthroplasty (TJA) in adults with obesity is associated with increased surgical risk and prolonged recovery. SO is associated with higher infection rates, poorer function and slower recovery in other clinical populations, but not thoroughly investigated in osteoarthritis [37].

Cardiometabolic complications of SO

Ma et al. analyzed a cohort of the Framingham Heart Study's Offspring and Omni 1 cohorts for mid-adulthood cardiometabolic risk profiles in patients with SO [38]. Utilizing BMI and sex-specific 24h urinary creatinine excretion,1019 participants from the Framingham cohorts were categorized as non-sarcopenia non obese (NSNO); non-obese sarcopenia, non-SO and SO. Cardiometabolic risk factors were quantified by standard laboratory assessment cross-sectionally and 10,20 and 30 years before SO assessment. NSNO, sarcopenia, obesity and SO accounted for 30,0%,39,6%,20,0% and 10,4% of study participants, respectively. Cross-sectionally, participants with SO had a higher proportion of hypertension, metabolic syndrome and type 2 diabetes than those with NSNO or sarcopenia (all p<0,03) Similar patterns were observed retrospectively at 10,20 and 30 years. Compared with NSNO or sarcopenia SO was associated with a higher prevalence of type 2 diabetes at 10 years and hypertension and metabolic syndrome at all three points before baseline (all p<0,03). Individuals with SO had more type 2 diabetes than those with obesity alone at baseline and 10 years prior (all p<0,001). The authors conclude that adults with SO had more adverse midlife cardiometabolic risks, particularly diabetes 10 years earlier.

Falls and SO

Pasco et al. examined the association between falls and SO, among elderly individuals in the population [39]. Participants were 353 men and 245 women, aged 65-98 years of the Geelong Osteoporosis Study. Body fat and lean body mass were measured using dual energy X-ray absorptiometry (DXA). Body fat mass was expressed as a percentage of weight (%BF) and appendicular lean mass was adjusted for height (rALM, kg/m2). Poor physical performance was assessed using the timed up & go (TUG) test. Sarcopenic obesity referred to low-rALM (T score<1), poor physical performance (TUG> 10s) and obesity (%BF>25% for men,35% for women) Fallers were identified by self-report as having had at least one fall in the previous 12 months. Associations between SO and falls were determined using logistic regression after adjusting for age and sex. In total 219 (36,6%) had lower rALM,205(34,2%) had poor physical performance,466 (77,9%) were obese and 69(11,5%) had SO. There were 170 (28,4%) fallers. Falls were more common for those with OS than without (28(40,6%) vs 42(26,8%) ;p=0,017). The like li hoof of falls in association with SO were: SO,OR=1,65 (95%CI 0,96-2,85), sarcopenia, OR=1,52 (0,93-2,47),poor physical performance and obesity, OR=1,74 (1,16-2,61),low r-ALM,OR=1,41 (0,96-2,06),poor physical performance, OR=1,88 (1,26-2,80),obesity OR=0,88 (0,57-1,35). The authors conclude that while obesity per se was not associated with falls there was an increased risk of falls in SO individuals that was of borderline statistical significance and appeared largely a consequence of poor physical performance [39].

SO and dementia

Sarcopenia and obesity both negatively impact health including cognitive function. Their coexcistense however, can pose an even higher threat likely surpassing their individual effects. Tolea et al assessed the relationship of SO with performance on global-and subdomain-specific tests of cognition [40]. The study was a cross-sectional analysis of data from a series of community-based aging and memory studies (n=353) with an average age of 69 years with a clinical visit, valid cognitive (Montreal Cognition Assessment) test, functional (grip strength, chair stands) and body composition measurements [40]. The authors found consistent evidence to link SO to poor global cognitive performance in community-dwelling older adults. This effect is best captured by its sarcopenia component with obesity likely having an additive effect. Several mechanisms may explain the obesity-cognitive dysfunction link including decreased participation in physical activity, low-grade chronic inflammation, oxidative stress and insulin resistance all of which being by-products of the aging process [41]. The authors conclude that sarcopenia alone and in combination with SO can be used in clinical practice as indicators of probable cognitive impairment. At risk older adults may benefit from programs addressing loss of cognitive function by maintaining and improving strength and preventing obesity [40].

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SO diagnosis

The current definitions of SO combine sarcopenia as defined through variable criteria in the presence of obesity as defined as BMI>30kg/m2. Simple anthropometric measurements in obese individuals may be biased by confounding adipose depots. Radiological methods that include nuclear magnetic resonance spectroscopy CT scans (SPECT) or dual X-ray absorptiometry have been considered most accurate but are not always available and feasible in this older population. Bio-electrical impedance analysis has been mentioned as an acceptable compromise. Functional measures are heterogenous and include hand-grip, knee-extensor strength and various moblity measurements involving postural and walking tests [42-44]. At this moment there is obviously no ideal methodology to achieve simultaneously maximal precision, safety, and routine applicability.

Management SO

Management strategies for obesity commonly favor diet changes and aerobic ecercise in order to reduce levels of body fat. However, this approach doesn't address the loss of muscle mass that may occur during weight loss and contribute to sarcopenia. It is of critical importance that management strategies focus on maintenance or accretion of muscle mass as well as fat loss in order to maintain strength, function and resting metabolic rate (RMR). Resistance exercise in combination with protein supplementation, prescribed by a dietician, can achieve these goals. In this way an 8 week resistance exercise program with protein supplementation can improve muscle mass significantly in even frail old men and women [45]. Any person starting a resistant exercise program should have at least a dietary protein intake of 1.5 g/kg. This is nearly twice the recommended daily amount (RDA) of 0,8 g/kg per day by the Food and Nutrition Board of the U.S. When male SO patients can be identified routinely in an easy manner, treatment with selective androgen receptor modulators (SARMs) might be considered in the near future [46].

Conclusion

Sarcopenic obesity (SO) is definitely a high risk geriatric syndrome. Decreased physical activity, low grade chronic inflammation, oxidative stress and insulin resistance all of which being by-products of obesity and the aging process are involved [41]. It is obvious SO is more associated with osteoarthritis (OA), falls, dementia and increased cardiometabolic risk profiles than obesity alone. Prevention, resistance exercise programs combined with dietary protein supplementation are the cornerstone of SO management [45]. Ideal easy routine diagnostic tools are not yet available. Radiological techniques measuring total body composition are most accurate but not always present or feasible in the elderly. When male SO patients can be easily identified, treatment with selective androgen receptor modulators (SARMs) might be considered in the near future [46].

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