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Mini Review

Sarcopenic obesity

Magdalini A. Nikolaou[1], Dimitris A. Nikolaou[2]

[1]Private Practice Clinical Nutritionist-Dietologist, Athens, Greece;
[2]Hellenic Osteoporosis Foundation, Athens, Greece

Abstract

Sarcopenic obesity is considered to be the unique co-existence of two distinctive pathologies: those of sarcopenia and obesity, which affects primarily older adults. Its manifestation is not afflicted by the individual's age, sex or body weight. There is a number of different diagnostic methods, and that goes for the therapeutic approach as well. Nutrition could be one of the key-factors of tackling sarcopenic obesity, being supported by proper physical exercise. This short review aims at providing current definitions and diagnostic as well as therapeutic interventions.

Keywords: Multifactorial disease, Obesity, Sarcopenia

Sarcopenic Obesity

Clinical definition

The clinical definition of sarcopenic obesity is used in order to deliberate the unique co-existence of two distinctive pathological conditions: that of sarcopenia and that of obesity. As described by Heber et al. (1996) sarcopenic obesity incorporates the deterioration of lean mass with a concurrent increase in total adipose tissue percentage[1] Similarly to the chronic disease of osteoporosis which stands as a silent disease, sarcopenic obesity could be described as a case of silent obesity combined with decreased muscle mass[1]. The reason is that individuals suffering from sarcopenic obesity remain asymptomatic for a reasonable period of time. Actually, their weight could be within the desired range, but their body composition may include excess adipose tissue alongside with reduced muscle mass[2].

Sarcopenic obesity is associated to numerous adverse health outcomes such as a decreased quality of life, reduced or even non-existent physical activity habits and disability – which stands as the inefficiency to perform activities of daily living (ADL). Last but not least sarcopenic obesity may lead to an increased mortality, as well as osteoporosis, type II diabetes etc.[3].

The prevalence of sarcopenic obesity

Two distinctive clinical factors influence vastly the diverge prevalence of sarcopenic obesity: a) The clinical definition and b)The method that was used in order to reach the diagnosis. The accuracy of its prevalence, is greatly affected by the inability of reaching to a universally accepted clinical definition[4]. Evaluation performed via the Bioelectrical Impedance Analysis (BIA) method, highlighted an age-related increase in prevalence[4]. When using the Dual X-ray Absorptiometry (DXA) method, the associated prevalence ranged from 0 to 84.5% and from 0 to 100%, for the female and male population respectively in patients with class II/III obesity[5]. In agreement with the National Health and Nutrition Examination Survey (NHANES), sarcopenic obesity met a prevalence of 33.5% in women and 12.6% in men[6]. Needless to say, the rise of life expectancy indicates an increase of the prevalence of both sarcopenia and obesity[7].

Pathogenesis of sarcopenic obesity

Although the clear-cut biological mechanisms that are responsible for the manifestation of the syndrome remain unexplained to a great extent, there is a number of clinical factors that could be attributable:

- Excessive energy intake, throughout food consumption.
- Shortage or complete lack of physical activity which could lead to atrophy of several muscle groups.

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Corresponding author: *Magdalini A. Nikolaou, Clinical Nutritionist-Dietologist, Athens, Greece*

E-mail: *magdalini.nikolaou94@gmail.com*

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- Inflammation.
- Insulin resistance due to obesity.
- Age-related changes in the hormonal environment and body weight[8].

In an attempt to interpret the bidirectional course of the pathology, we could conclude: sarcopenia leads to a noteworthy reduction of the individual's physical activities, which limits energy consumption and thus entails an increased risk of obesity. Simultaneously the increment of visceral fat triggers a number of inflammatory reactions that could enhance the gradual establishment of sarcopenia[9]. Both muscles and visceral fat share common inflammatory pathways. Obesity activates macrophages and T-lymphocytes, thus helping into the development of a low-level inflammation[3]. The inflammation proceeds into the secretion of Tumor Necrosis Factor Alpha (TNFα), Leptin and Growth Hormone (GH). All of the afore-mentioned mechanisms lead to insulin resistance, which promotes the increase of fat and at the same time the muscle loss[3]. Leptin regulates the pro-inflammatory cytokines IL-6 and TNF, which contribute into the reduction of the anabolic effects of Insulin-like Growth Factor 1 (IGF1). That reduction of IGF1, alongside the age-related reduction of testosterone, set also the ground for the manifestation of frailty syndrome[3].

Diagnosis of sarcopenic obesity

In order to evaluate and confirm the manifestation of sarcopenia the parameters that should be looked upon are the muscle mass, the muscle strength and the physical performance.

Muscle mass could be estimated via the practice of the following methods:

- Computed Tomography (CT)
- Magnetic Resonance Imaging (MRI)
- Dual X-ray Absorptiometry (DXA)
- Bioelectric Impedance Analysis (BIA)

CT as well MRI are considered to be accurate imaging methods, which could easily separate fat from other soft tissues in the human body. These methods are not greatly preferred due to the fact that they are rather expensive (MRI), there is limited accessibility, the assessed individual is exposed to a high dose of radiation (CT) and a specialized personnel is required in order to perform the procedure as well interpret the outcome[11].

DXA on the other hand may be useful in evaluating muscle mass, especially whole body DXA, and may be preferred due to its low radiation than CT. It's a reliable and inexpensive method that is mainly used as part of the osteoporosis screening. Some of the disadvantages of the method is the inability for portable apparatus as well the fact that the individual's hydration levels could affect the measurements[11].

BIA is an easy method of evaluating fat and non-fat mass, whose basic principle lies into the electrical conductance. BIA estimates the rate at which a low-level electrical current transcends throughout the body. According to that rate a calculation of the fat-free mass is made. It is an easy to use non-invasive method, without a great cost or having the assessed individual exposed to radiation. Last but not least the portability of the apparatus enables measurements to be carried out in bedridden patients as well. The measurements via BIA are greatly affected by the patient's hydration level[12].

Muscle strength could be evaluated via the following methodology:

- Handgrip Strength examination
- Knee flexion/extension examination
- Peak Expiratory Flow[10]

Even though the lower extremities seem to be more important when assessing gait and physical function, the handgrip strength has been widely used and correlated with relevant results.

As mentioned before specifications such as the availability of the apparatus, the cost and the ease to use, may determine whether the associated methodology is best suited to everyday clinical practice or a research setting. It is a common knowledge that when evaluating muscle strength clinical factors such as e.g. motivation or cognitive level could hinder the whole process[13]. The upper extremities circumference and the skin fold thickness can be used in order to estimate muscle mass in ambulatory patients. However, several age-related changes in the body such as e.g. the fat deposit, the loss of skin elasticity etc. could elicit dubious results[13]. There is a relatively small number of studies assessing the reliability of the anthropometric measurements when it comes to elderly and obese individuals. The subjectivity that follows these practices is an important limitation[10].

The individual's physical performance could be screened thought the succeeding methods:

- Gait speed
- Timed Up and Go test (TUG)
- Short Physical Performance Battery examination (SPPB)[14].

On the other hand in the case of obesity and the expected/desired categorization, the BMI normative is used. According to the World Health Organization (WHO) guidelines an individual with a BMI of 18.5-24.9 is considered to be of normal weight. In case the BMI-associated value is found to be below 18.5, then the assessed individual is described as underweight. In contrast when BMI values are of 25-29.9, 30-34.9, 35-39.9 and above 40, the individual is at a state of pre-obesity, obesity class I, obesity class II and obesity class III respectively.

Nutritional treatment of sarcopenic obesity

The individual's eating habits constitute a key factor in the manifestation as well as the progression of both sarcopenia and obesity. The distinctive mechanisms linked to sarcopenia and obesity, are presented as entirely opposite. Sarcopenia is correlated to insufficient energy intake, while obesity stands as the consequence of excessive energy

intake and the imbalance in-between the energy intake and expenditure[16].

The outlining of the intervention when it comes to sarcopenic obesity, should comprehend the correction of the intake regarding the various nutrients. Proper intake will increase the skeletal muscle mass, while preventing further loss. Consequently the optimal nutritional and energy intake will lead to the diminishing of excessive fat storage.

When the loss of unnecessary body weight is desired there is a high possibility of applying a hypo-caloric diet. The most appropriate yet safe energy restriction range for a sarcopenic obese older adult is that of 200-700 kcal per day. There is a satisfactory number of evidence regarding the effectiveness of the aforementioned interventions. What every health professional working with sarcopenic obese individuals should be informed about, is that the excessive loss of fat mass is trailed by loss of muscle mass.

According to Villareal et al. an average fat loss of 7, 1 kg (SE: 3.9, p<0,001) is accompanied by a loss of approximately 3.2 kg of muscle mass (SE: 2.0, p<0,001)[17]. Villareal et al. used a sample of obese older adults, after following a low-caloric diet for 52 weeks. It is likely that 25% of the weight loss achieved throughout low-caloric diets in obese elderly is of skeletal mass, which could bring greater problems to sarcopenic obese individuals. For that reason the diet should not be strictly hypo-caloric. An energy deficit of 200-700 kcal per day could be described as a secure approach into losing the minimum possible muscle mass. The other way around, a severe loss of muscle mass could eventually impair the self-sufficiency during activities of daily living e.g. going up and down the stairs or walking for a distance etc. The adjustments of eating habits should always be performed in regard to bone mineral density, as well the several micronutrients[18].

Every nutritional treatment should be backed up by a well-organized physical exercise regime. That personalized plan must aim at maintaining and even improving several distinctive characteristics of the musculature such as muscle strength, muscle mass etc. Simultaneously, the individual throughout his engagement is able to improve his functionality thus performing a greater number of daily living activities.

Clinical studies findings

Like many health-related topics, there are areas when it comes to sarcopenic obesity that have not yet been fully understood or even investigated. The exact mechanism under which insulin resistance, low-grade inflammation and vitamin D deficiency are related to the pathological passages of sarcopenic obesity remains unknown.

Kim et al (2013) estimated the effect of the homeostatic model on the evaluation of insulin resistance (HOMA-IR), high sensitivity C-reactive protein (hsCRP) and 25-hydroxyvitamin D (25[OH]D) levels in a sample of 493 Koreans – of whom 180 were men and 313 were women. The sub-group of sarcopenic women demonstrated higher levels of HOMA-IR and hsCRP, when compared with the non-sarcopenic group.

Respectively the levels of 25(OH) D in sarcopenic men were significantly lower, in comparison to those of non-sarcopenic group. What needs to be ascertained is that the levels of hsCRP and HOMA-IR displayed a negative correlation in regards to the skeletal muscle index (ASM), and a positive one towards the percentage of visceral fat. This particular observation stood independent of the individuals' gender. On the other hand levels of 25(OH) D were positively related to the ASM as well the percentage of visceral fat[19].

Moreover sarcopenia is related to functional difficulties. Functional difficulties may lead to disability and finally to death. Disability could be an outcome of diminished muscle strength, which could easily be assessed via handgrip strength examination. According to E. Kaledkiewicz et al. (2020) the decrease of the handgrip strength was associated to an increased mortality[20].

According to Å von Berens et al. (2020) male individuals over the 75th year of age were much more prominent to the manifestation of sarcopenic obesity than women. Although the sarcopenic obesity retained higher morbidity rates in women, when compared to non-sarcopenic obese women[21].

More studies are currently under way to elucidate the pathogenesis of sarcopenic obesity as well as its various contributing factors, and methods of treatment.

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