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Opinion Article

Sarcopenia in Hemiplegia

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Abstract

Sarcopenia is a disease characterized by quantitative and qualitative degeneration of the skeletal muscles and it primarily presents with a decline in the muscle strength. It frequently occurs in patients after a cerebrovascular accident due to a combination of various factors, such as the brain injury, structural adaptations, limited physical activity as long as malnutrition. Most of the articles and reviews concerning stroke-related sarcopenia are limited and usually are discussing about the factors and causes that may lead to the muscle wasting and the particular characteristics that distinguish it from age-related sarcopenia. As a result, even though sarcopenia is described as a medical entity, it appears to lack the attention that requires, limiting the maximum therapeutic effect a patient can obtain. Suggestions have been made concerning general treatment and management of sarcopenia, associated with exercise, diet and the use of medical preparations, lacking though disease-specific guidelines for management, treatment and possibly prevention of stroke-related sarcopenia. Nowadays, it is considered of major importance that the medical community should be properly informed and to raise awareness on this particular issue aiming to a better and holistic management of the patients after a cerebrovascular accident, in order to reduce morbidity and disability that both are sequelae that reduce quality of life.

Keywords: Disability, Muscle strength, Muscle wasting, Sarcopenia, Stroke

Introduction

Sarcopenia is a medical entity in the form of muscle disease or muscle failure. It is characterized by prominent decline of muscle strength with simultaneous low muscle mass. It is a progressive and generalized disease which after the revision of the European consesus in the meeting of the European Working Group on Sarcopenia in Older People 2 (EWGSOP2) in 2018 can be identified by three criteria. Low muscle strength is the first criterion and the key characteristic which allows the identification of sarcopenia, low quantity and quality of the muscle units is the second criterion and the one to confirm diagnosis, and the third criterion and also the one to evaluate the severity of sarcopenia is poor physical performance[1,2].

The adaptive and adverse changes of the muscle can occur during a lifetime and at any age. There are multiple causative factors that may lead to muscle failure. The aetiology can be of primary or secondary origin. The most common cause of sarcopenia as well as the primary cause is the advanced age, often recognized by the definition of age-related sarcopenia. Secondary sarcopenia can arise due to many underlying conditions such as in diseases (inflammatory conditions, osteoarthritis, neurologic conditions), inactivity (sedentary lifestyle, physical inactivity) and malnutrition (malabsorption, under-nutrition, obesity, drug-related anorexia)[1,2].

Cerebrovascular accidents (stroke) are the second leading cause of death and the third leading cause of disability worldwide according to World Health Organization (WHO)[3] as well as a disease that is being accompanied by sarcopenia. The brain injury is considered the primary cause of disability[4] leading to hemiparesis, which thereafter attributes to changes to the skeletal muscles due to a combination of the neurologic deficits and the limited mobilization, in addition to more factors that are mentioned below.

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Stroke-related sarcopenia - characteristics

Stroke-related sarcopenia is a specific type of sarcopenia that shares some same characteristics as age-related sarcopenia but differs in others[5]. In contrast to the muscle wasting observed in elderly, in a patient after a cerebrovascular accident the following features are observed[6]:

A. Rapid reduction of the muscle units post-accident: the patient can have a decline of muscle mass as early as four hours after stroke in the hemiplegic limb[7]. As the patient goes from the acute to the chronic stage this reduction is even more prominent.

B. Changes in the skeletal muscle structure: a specific characteristic of stroke-related sarcopenia is the shift of slow to fast muscle fibers. Normally during aging muscle fibers switch from fast-twitch (myosin heavy chain (MHC) type IIa and IIx) to mitochondria-rich slow-twitch (MHC type I) muscle fibers. After a cerebrovascular accident a converse change is seen with an increase of the fast-twitch MHC type II[8]. This is caused due to denervation which is followed by reinnervation by neibhoring muscle fibers[9].

C. The disability is determined and affected by the brain lesion itself, leading to differences between the hemiplegic and the healthy side, although alterations are observed in the non paretic limb aswell[10,11].

D. The structural and functional changes of the muscle leading to wasting are not dependant to the age of the patient.

E. Catabolic activation due to an imbalance in the autonomic nervous system: the brain infarction is followed by systemic effects (stress, pain, inflammation etc.) as well as local damage (impaired signaling) which lead to decreased anabolic and increased catabolic signals[8].

Factors leading to Stroke-related sarcopenia

The changes of the skeletal muscles after a cerebrovascular accident happen due to many factors. The phenotypic alterations arise from the effects of a set of procedures that are denervation and reinnervation, the diminished use, the spasticity that results after stroke, the structural adaptations as well as systemic effects due to the brain injury altogether in combination. Thus sarcopenia after the event can be considered of multifactorial origin[5,8].

Concerning the structural adaptations and changes of the skeletal muscle after a stroke, most of them arise due to the motor unit denervation and reinnervation from intangible muscle fibers, in combination with the disturbed production balance of the skeletal muscle protein, that favors the destruction against its production[12]. The phenomenon of the inverse shift of slow to fast muscle fibers takes place after the event, which also distinguishes this type of sarcopenia from the age-related. The fast-twich fibers have a greater vulnerability to denervation and reinnervation in comparison to the slow-twich fibers[9]. In studies it has been observed that this shift of the muscle fibers from slow to fast-twitch MHC affects the paretic limb, without effect on the non paretic, in which after measurements and comparison of the fibers in healthy individuals , no significant differences were noticed[13]. This particular inversed shift has also been observed in cases of muscular disuse. Therefore in combination with the pathological neurologic background of the patient, which the more extensive it is the more restricted the mobility, characteristics of the patient such as the age, the muscle power, physical activity prior to the event, as well as the cardiovascular profile, affect the metabolic characteristics and the molecular phenotype of the skeletal muscle in the hemiplegic side[9,14].

Following a cerebrovascular accident the patient is hospitalized and in an extend determined by the neurologic and motor deficits becomes immobilized. This renders the patient to an inactive state with muscle unloading and to sequelae of the physical inactivity and limited mobilization such as insulin resistance, decline of muscle mass, atrophy and fat deposition intramuscularily[8,15].

Impaired feeding is a possible sequence after stroke leading the patient after the event to be malnurished. The result is worsened by the fact that the local brain lesion may give rise to an imbalance between anabolic and catabolic signals with an increased systemic catabolic activation with the consequence of weigth loss and muscle wasting. Dysphagia and in general feeding difficulties disrupts the proper consumption, both qualitatively and quantitatively, of calories and nutrients that an organism, especially under those circumstances, needs. As a result both muscle and fat tissue wasting occurs as the malnurished state aggravates sarcopenia. A significant remark, called the “obesity paradox”, has been observed in which studies showed that overweight and obese patients had better survival rates, lower morbidity and less chances for a new cerebrovascular accident in the future[16]. All these parameters not only affect muscle and fat tissue but also skeletal tissue, thus impairing much further the functional units of the musculoskeletal system, contributing with a greater complexity to the stroke-related sarcopenia.

Methods to evaluate muscle mass and function

The gold standard technique used to assess body composition granting the possibility to evaluate the muscle mass, the fat mass as well as the body’s mineral content is the Dual-energy X-ray Absorptiometry or DXA[17]. The DXA method estimates the body composition in a molecular level, while at the same time has the ability to differentiate between the normal decline in the muscle mass as a natural process from a pathologic decline. Additionally DXA is considered a great tool that serves in the decision-making process concerning the choice of treatment as well as its follow up[18]. Some other ways to evaluate body composition is with computed tomography (CT) and magnetic resonance tomography (MRT) as well as ultrasound by measuring the

cross-sectional area of the limbs. Another method also exists called bioelectrical impedance analyses (BIA) which is used in order to estimate the body composition as well as the muscle mass[8,18]. DXA is the prefered method as it is simple, quick, non-invasive and inexpensive, with extremely small amount of radiation and no side effects.

For the purpose of evaluating the muscle function, methods exist that can be performed in stroke patients although they face numerous limitations due to their deficits. Muscle strength can be evaluated by Hand grip strength, Finger pinch strength and Quadriceps isometric strength test. A range of clinical tests such as gait speed in patients tha have relearned to walk again, as well as results from evaluation with Short Physical Performance Battery Test and Fugl-Meyer Assessment, are used to estimate the physical performance of the patient. The assessment of functional capacity and the ability to perform the abilities of the daily living is also useful and can be performed by the Barthel Index and Functional Independence Measure (FIM)[1,6].

Therapeutic approach and management

The management of sarcopenia according to recent research and data demands a multidisciplanery approach, especially when the muscle wasting results after a cerebrovascular event. The physical rehabilitation of the patient with physiotherapy, kinesiotherapy and retraining in order to achieve exercises of functional ability, so that each individual will be able to integrate more easily to activities of the everyday living, always in accordance to the neurologic and motor deficits, is essential to overcome the factors related to the physical inactivity and the limited physical performance, that are able to intensify further sarcopenia. In clinical practice, the perspective of the alteration of the inverse shift of the muscle fibers from slow to fast-twitch with the application of muscle stimulation devices, appears to be of great importance in the management of the disease[9]. In combination with the physical rehabilitation, primary role has the restoration of the nutritional supply as long as a proper diet according to the demands of the patient's needs, as one of the main factors that aggravate sarcopenia after stroke is malnutrition. An appropriate diet plan accompanied with vitamins and supplements is of great importance in the management and presumably in the prevention of the development of stroke-related sarcopenia. Foods rich in proteins, essential amino acids, fatty acids, antioxidants such as vitamin C and E, as well as supplementation of vitamin D are some of the components of a diet that seems to have a positive influence in the treatment of sarcopenia. In cases of existing limitations due to dysphagia and feeding difficulties, additional adjustments can be made and used in order to facilitate the patient. Furthermore, a variety of medical preparations have been studied and evaluated for their efficiency to contribute positively in the management of sarcopenia such as testosterone, selective estrogen receptor modulators (SERMs), angiotensin converting enzyme inhibitors (ACEI), and myostatin inhibitors. This is aiming in the preservation and the increase of the muscle and skeletal tissue with simultaneous decrease of the fat tissue, with ultimate goal to improve muscle strength and functionality[19].

Conclusions

Stroke-related sarcopenia is a phenomenon arising secondarily to a cerebrovascular accident, contributing together with the brain injury itself to great disability and morbidity, creating a huge burden towards the decision-making concerning treatment and rehabilitation. Disuse atrophy, spasticity, inflammation, denervation and reinnervation, impaired feeding as well as impaired intestinal absorption play an important role on aggravating the reduction of the muscle bulk, making the results of sarcopenia even more prominent as the patient goes from acute to chronic phase. It is essential all data existing and hopefully future studies and their results to be taken under consideration and to be evaluated in order to create specific guidelines about management and treatment of sarcopenia, which will aid for a better and holistic approach towards the stroke patients.

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