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LETTER

Some aspects need to be considered in assessment and treatment of sarcopenia

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Dear editor

We have read the review article by Ilaria Liguori et al[1] with great interest. In this article, authors aimed to review sarcopenia in terms of assessment of its burden and strategies to improve outcomes. We have some comments that would contribute on this extensive review.

Authors reported that the diagnosis of sarcopenia requires the presence of both low muscle mass and low muscle function. Authors defined low muscle function as gait speed lower than 0.8 m/s and/or a grip strength lower than 26–30 kg for men and 16–20 kg for women. However, it should be noted that European Working Group on Sarcopenia in Older People (EWGSOP) recommends use of normative data of the study population for muscle mass and muscle strength cutoff points. After EWGSOP publication, population-specific cutoff values, including the Turkish and Korean population, have been published and better to be included in a paper aimed to review sarcopenia in this context.[2]

The authors noted that, starting from skeletal muscle mass calculated using the Janssen BIA equation, skeletal muscle mass index (SMI) = (skeletal muscle mass/body mass) × 100; low muscle mass is defined by an SMI ≤8.87 kg/m^2 for men and ≤6.42 kg/m^2 for women. However, these cutoff values are SMI cutoff values when the skeletal muscle mass is adjusted by height and not body mass. The cutoff values of skeletal muscle mass adjusted by body mass determined by this methodology are 29.0% for men and 20.5% for women, and this should be corrected.[3]

The authors reported that testosterone is the most effective and safest drug among the drugs investigated for the treatment of sarcopenia, because its adverse effects are dose dependent and associated with very high doses of 300–600 mg/week. However, the dose of 300–600 mg/week testosterone mentioned in this study is well above the recommended dose for standard hypogonadism treatment (at least 2–4 times). In 2017 study derived from the major Testosterone Treatment Trials (TTT), it is stated that testosterone-related side effects may occur even at the recommended low doses for hypogonadism, that is, increase in coronary artery noncalcified plaque volume.[4] Additionally, in another study derived from TTT, it is shown that testosterone does not increase physical function. Because of these reasons, it is difficult to say that testosterone is the safest and most effective drug.

Regarding future therapeutic approaches, authors denoted some agents, that is, selective androgen receptor modulator enobosarm, ghrelin receptor agonist anamorelin, monoclonal antibodies, and stem-cell approaches. However, there are many more

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agents that should be stated in this regard, that is, espindolol, ruxolitinib, peroxisome proliferator-activated receptor delta agonists, AICA (5-aminoimidazol-4-karboksamid-1-beta-4-ribafuranosid), biguanids, tripartite motif-containing 32 inhibitors, growth differentiation factor (GDF11), ciliary neurotrophic factor agonist, myokines activators and inhibitors, CisD protein replacement, sirtuins/reservatol/polyphenols, nitric oxid (isorbide dinitrite), microRNAs (miR-1, miR-29, miR208, and miR486) modulators, RNA antisense, PGCI α agonist, and serum and glucocorticoid inducible kinase 1 (SGK1). It would be better to be included in a paper aimed to review sarcopenia in this context.[5]

Disclosure

The authors declare no conflicts of interest in this communication.

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