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Sarcopenia and sarcopenic obesity in type 2 diabetes mellitus and cardiovascular disease: current perspective and narrative review

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Sarcopenia, initially described as a disease of the elderly and its coexistence with obesity, now termed as 'sarcopenic obesity' (SO), are now emerging to be important health problems worldwide. Their relatively recent recognition as distinct clinical entities with growing research has shown an increasing prevalence of both sarcopenia and SO. There is a strong independent association, which exists between sarcopenia/SO, type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD). Studies investigating prevalence of sarcopenia/SO indicate considerable heterogeneity, likely due to the lack of a universally accepted definition, differences in age groups, gender, ethnicity, and socio-demographic factors of the population studied, apart from use of distinct diagnostic criteria and methods of estimation based on either muscle mass or strength.

Keywords: cardiovascular diseases, muscle strength, sarcopenia, type 2 diabetes mellitus

Sarcopenia

Introduction and diagnostic criteria. The term 'sarcopenia' initially coined by Rosenberg (1989) is derived from the Greek words - 'sarx' meaning flesh and 'penia' meaning loss, which literally translates to 'poverty of flesh'. In today's era, sarcopenia is defined as a skeletal muscle disorder characterized by a decline in skeletal muscle mass, strength and/or physical performance (Kim and Choi 2013). Sarcopenia coupled with obesity is now well-established as a distinct form of obesity known as 'sarcopenic obesity' (SO) (Batsis and Villareal 2018). With an ever-increasing geriatric population, sarcopenia and SO are now emerging public health problems worldwide. Moreover, with increasing clinical attention to these disease entities, different organizations have put forward certain criteria for diagnosing sarcopenia and SO (Table 1).

Prevalence. According to a systematic review by International Sarcopenia Initiative (ISI) (adults aged ≥50 years), the prevalence of sarcopenia was found out to be between 1–29% in community-dwelling populations, 14–33% in long-term care populations and 10% in acute hospital-care setting with prevalence being highest in the geriatric population (Cruz-Jentoft et al. 2014). Another large study, which included data from over 28 European countries projected that the occurrence of sarcopenia is likely to rise from 20.2% (2016) to 22.3% (2045) in the elderly (>65 years), which corresponds to an overall increase in the number of sarcopenic individuals by 63.8%. Hence suggesting the importance of sarcopenia as a major public health concern (Ethgen et al. 2017). Furthermore, a large multicentric study from over nine countries (n=18363; age ≥65 years) found the prevalence of sarcopenia to be ranging from 12.6% in Poland to a maximum of 17.5% in India (Tyrovolas et

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al. 2016) indicating possible geographical and ethnic differences in prevalence of sarcopenia. It is clear that the prevalence of sarcopenia varies greatly when different diagnostic criteria are used. Hence this necessitates the need for either formulating universal diagnostic criteria for sarcopenia or adopting specific criteria, which is indigenous to a particular population based on specific ethnic, racial, geographical, and physiological attributes.

Sarcopenic obesity

Prevalence. Batsis et al. (2017) have defined sarcopenia by the FNIH criteria (see Table 1) and obesity using a body fat percentage (males >25%, females >35%) using the National Health and Nutrition Examination Survey (NHANES) data and find out the prevalence of sarcopenic obesity (SO) to be 33.5% in females and 12.6% women in subjects aged over 80 years indicating the strong association of SO with increasing age. The Korean sarcopenic obesity study (Kim et al. 2009) (n=526, 20–80 years) found out the prevalence of SO to be between 1.3%–15.4% in men and 0.8%–22.3% in women using different diagnostic criteria used to define and estimate sarcopenia and obesity, respectively.

Furthermore, in a large-scale multicentric study, the prevalence of SO was found to be ranging from 1.3% in India to 11.0% in Spain in people aged ≥65 years (Tyrovolas et al. 2016). The prevalence of

Table 1
Comparison of different diagnostic criteria of sarcopenia and sarcopenic obesity

Study	Reference	Criteria for Sarcopenia	Criteria for Obesity
New Mexico Aging Process Study (1998)	Baumgartner 2000	RSMI=muscle mass (kg)/stature (kg/m^2) less than -2 SD below reference values in young healthy individuals <7.26 kg/m^2 (M) and <5.45 kg/m^2 (F) predicted/measured by DXA	Body fat percentage: >27% (M) and >38% (F)
International Working Group on Sarcopenia (IWGS) (2011)	Fielding et al. 2011	- AFLM/height^2: ≤7.23 kg/m^2 (M) and ≤5.67 kg/m^2 (F); - GS <1 m/s with less than 20 percentile of lean mass values for healthy adults	NA
National Health and Nutrition Examination Survey (NHANES-III) (2014)	Davison et al. 2002	- Two lower quintiles of muscle mass; - ALM/height^2: <9.12 kg/m^2 (M) and <6.53 kg/m^2 (F)	Two highest quintiles of fat mass: >37.16% (M) and >40.01% (F)
Foundation for the National Institutes of Health (FNIH) (2014)	Studenski et al. 2014	- ALM: <19.75 kg (M) and <15.02 kg (F) measured by DXA, - Grip strength: <26 kg (M) and <16 kg (F)	NA
European Working Group on Sarcopenia in older People (EWGSOP2) (2019)	Cruz-Jentoft et al. 2019	- ASM/height^2: <7.0 kg/m^2 (M) and <5.5 kg/m^2 (F); - Muscle grip strength: <27 kg (M) and <16 kg (F); - GS: ≤0.8 m/s	NA
Asian Working Group for Sarcopenia (AWGS) (2019)	Chen et al. 2020	**Possible sarcopenia**: Handgrip muscle strength: <28 kg (M) and <18 kg (F) **OR** 5-time chair stand test: ≥12 s **Sarcopenia**: low ASM: <7.0 kg/m^2 (M) and <5.4 kg/m^2 (F) (DXA) or <7.0 kg/m^2 (M) and <5.7 kg/m^2 (F) (BIA) + low handgrip muscle strength: <28 kg (M) and <18 kg (F) **OR** low physical performance: 6-m walk GS <1.0 m/s or 5-time chair stand test ≥12 s or short physical performance battery ≤9 **Severe sarcopenia**: low ASM + low handgrip muscle strength **AND** low physical performance	NA

Abbreviations: AFLM – appendicular fat lean mass; ALM – appendicular lean mass; ASM – appendicular skeletal muscle mass; BIA – body impendence analyzer; DXA – dual-energy X-ray absorptiometry; F – females; GS – gait speed; M – males; NA – not applicable; RSMI – relative skeletal muscle index, SD – standard deviation.

SO varies depending upon the criteria used to define either sarcopenia-based on low muscle mass and/or low hand grip strength, or obesity, and for this it was studied that the prevalence of SO ranged from 4% to 12% (Baumgartner 2000; Davison et al. 2002; Zoico et al. 2004; Cruz-Jentoft et al. 2019).

According to a recent meta-analysis done in 2021 (Gao et al. 2021), it is studied that SO affects more than one in ten individuals in the older age group and that the overall prevalence of SO is 11% globally, which ranges from 4% when muscle mass and strength and/or physical performance are considered vs. 15% when only muscle mass is used to diagnose sarcopenia, and 7% when body impendence analyzer (BIA) is used vs. 15% when dual-energy x-ray absorptiometry (DXA) is used to measure the muscle mass. This indicates that there should be more defined criteria, which makes uses of either muscle mass and/or muscle strength for defining and measuring sarcopenia optimally using standardized instruments.

The recent meta-analysis by Gao et al. (2021) has also shown that the pooled prevalence of SO ranged from 10% when body fat criteria was used, 13% when body mass index (BMI) was considered, and 16% when waist circumference was used to diagnose obesity. Thus, this indicates that even though all three criteria may be good parameters to diagnose SO, future studies might be able to justify the most sensitive and accurate criteria for defining SO.

Hence studies investigating prevalence of sarcopenia/SO indicate considerable heterogeneity, likely due to the lack of a universally accepted definition, differences in age groups, gender, ethnicity, and socio-demographic factors of the population studied, apart from use of distinct diagnostic criteria and methods of estimation based on either muscle mass or strength.

Pathophysiology. The pathophysiology of SO is multifactorial and the possible contributing factors include: 1) ageing resulting in ectopic fat accumulation and decrease in both muscle mass and strength (Goodpaster et al. 2006); 2) qualitative changes in muscles-muscle atrophy and switch from type-II to type-I fibers (Nilwik et al. 2013); 3) metabolic derangements - increased oxidative stress, inflammation, and catabolic cytokines like tumor-necrosis factor-α (TNF-α), interleukins IL-6 and IL-1, C-reactive protein (CRP) (Furukawa et al. 2004), and insulin resistance (Carrascosa et al. 2011); 4) cellular-mitochondrial and stem cell dysfunctions (Dahlmans et al. 2016); 5) endocrinal-decreased levels of growth hormone (GH) insulin-like growth factor-1 (IGF-1) (Hermann and Berger 2001), testosterone in both males and females (Feldman et al. 2002; Morley and Perry 2003), estrogen (Collins et al. 2019) and irisin (Sakuma and Yamaguchi 2012), and increased the myostatin levels (Wehling et al. 2000); 6) diet-inadequate protein intake, vitamin D deficiency (Oh et al. 2017); and 7) physical inactivity (Oh et al. 2017). Moreover, with increasing research in this field, more and more new risk factors are being defined.

Association with T2DM and cardiovascular disease

Sarcopenia and T2DM. Studies, including some from Asian countries, indicate an increased prevalence of sarcopenia in subjects with T2DM (Wang et al. 2016; Murata et al. 2018; Trierweiler et al. 2018; Fung et al. 2019; Wasir et al. 2024). A Chinese study (n=1090; age ≥60 years) using the AWGS-19 definition (see Table 1) found out the prevalence of sarcopenia and pre-sarcopenia to be higher in patients with T2DM than in healthy controls (sarcopenia: 14.8% vs. 11.2%; p=0.035, pre-sarcopenia: 14.4% vs. 8.4%; p=0.002) (Wang et al. 2016). According to a study in the Brazilian population (n=240, ≥60 years), it was found that the prevalence of sarcopenia was more prevalent in patients with T2DM than without T2DM (21% vs. 17%) and that men had a higher prevalence of sarcopenia (75%) (de Paula et al. 2019).

A large population-based study of Korean middle-aged adults, who were followed up for an average of 2.9 years, clearly showed a strong inverse relationship between reduced muscle mass and incident diabetes even after adjusting for important confounding metabolic parameters (Hong et al. 2017). A prospective study (~5 years) in 219 subjects showed that age-related muscle mass decline significantly predicts incident diabetes and this association is independent of insulin resistance (HOMA-IR) (Maliszewska et al. 2019). This inverse relationship between low muscle mass and incident diabetes may also hold true in individuals with a non-obese BMI (Son et al. 2017). This strongly points towards the importance of sarcopenia in being an independent risk factor for developing T2DM.

Importantly, an inverse relationship between skeletal mass and pre-diabetes was shown in a cross-sectional study (NHANES 3; n~ 14000 adults, >20 years), which found a 10% increase in skeletal mass index, associated with a 11% relative reduction in insulin resistance (HOMA-IR) and 12% relative reduction in pre-diabetes prevalence (Srikanthan and Karlamangla 2011). This implies that possible health

interventions focused on building muscle mass may have a protective impact on future risk of T2DM.

Sarcopenic obesity and T2DM. There is ever-increasing evidence regarding the strength of SO as an important risk factor for T2DM. The association of SO with T2DM involves many overlapping causal risk factors, which have been discussed earlier, and which play the key role in the etiopathogenesis of this relationship.

Korean sarcopenic obesity study by Kim et al. (2010) (n=810, ≥60 years) has shown a higher prevalence of sarcopenia in both men and women with T2DM than in those without T2DM (15.7% vs. 6.9%, respectively). Son et al. (2017) have conducted a 2-year prospective study (n=6895, 40–69 years) as a part of the Korean genome and epidemiology study (KoGES) and showed that during the follow up of 9.06 years, 1336 participants (19.4%) developed T2DM (incidence rates; males=20.8%, females=18.1%), which was attributed to low muscle mass (measured using muscle mass index) independent of obesity. Interestingly, they also found that the lowest tertile of muscle mass index for developing T2DM increased by 11.9% in the non-obese group and 19.7% in the obese group; thereby, implying both sarcopenia and obesity as strong determinants for T2DM development. On similar grounds, Senechal et al. (2012) in a cross-sectional study (n=3, 007; ≥50 years) has shown that subjects with SO had the maximum odds for developing T2DM (22.9%) as compared to the sarcopenic non-SO, non-obese, and non-sarcopenic non-obese groups. Ma et al. (2016) in a retrospective study investigating the prevalence of T2DM over a period of 3 decades have shown that subjects with SO had the maximum prevalence of T2DM at 10 years (21.4%) and 20 years (4.0%) as compared to the non-SO groups. Therefore, future studies should also aim at studying the relationship of sarcopenia/SO with the duration of T2DM to effectively identify sarcopenia/SO at its early incubating stages and limit its progression if possible. Lastly, Khadra et al. (2019) in a meta-analysis have shown that the presence of SO increased the future risk of T2DM in individuals by 38% (OR=1.38, 95% CI: 1.27–1.50) in comparison to those without SO, and emphasized on the importance of screening of individuals with SO because of its strong association with T2DM. Hence strongly justifying the inclusion of SO as a strong predictor for T2DM.

Cardiovascular disease. In this section, we briefly discuss the association of sarcopenia/SO with cardiovascular disease (CVD). The prevalence of sarcopenia and SO was found to be significantly higher among T2DM patients with CVD (n=21; 70% vs. n=9; 30%, p=0.222, respectively) as compared to those patients only having T2DM (n=12; 40% vs. n=5; 16.7%, p=0.222, respectively) indicating the role that CVD might independently contribute to an increased risk and prevalence of sarcopenia/SO in T2DM patients, the opposite of which may also hold true (Chin et al. 2013; Chung et al. 2013; Atkins et al. 2014; Kim et al. 2015; Wasir et al. 2024).

Kim et al. (2015) (n=3320, ≥40 years) have shown that adults with SO had a significantly greater 10-year CVD risk (overall ≥20%; males=43.8%, and females=14.6%) when compared to non-sarcopenic adults (males=24.1%, females=11.1%) and non-obese adults (males=42.7%, females=12.2%). This study also showed that the CVD risk was higher in males than in females (15.04% vs. 7.91%, respectively) and that males with SO had a higher 10-year CVD risk than females with SO possibly because during the ageing process, males seem to lose more muscle mass than females (Dey et al. 2009). However, the long-term impact of gender on SO in CVD patients remains to be known. Interestingly, it was also found that the chances of having CVD were increased when the individuals had both sarcopenia and metabolic syndrome, but surprisingly even when they only had sarcopenia and no metabolic syndrome, these individuals were still likely to experience CVD events (Chin et al. 2013), suggesting the possibility of an independent association of sarcopenia with CVD.

The magnitude of importance of any health risk factor is defined by its association with mortality (all-cause and/or CVD-related) as an outcome measure. Atkins et al. (2014) (n=4,252; 60–79 years) have shown that sarcopenia and obesity had a strong association with increased CVD-related and all-cause mortality. The results also showed that men with SO had the highest risk (HR) for an all-cause mortality (HR=1.72) as compared to the sarcopenic group (HR=1.41) or the obese group (HR=1.21) (Atkins et al. 2014). A meta-analysis including data from 50,866 subjects (50–82.5 years) indicated that SO is associated with a HR of all-cause mortality among adult people having a HR of 1.21 (HR being 1.14 in community dwelling adult people and 1.65 in hospitalized patients). Zhang et al. (2019) have suggested an increased risk for fatal events, especially in the aged population, also having other problems.

Muscle mass vs. muscle strength: what is better?

Emerging data suggest a heterogeneity in associations of sarcopenia/SO with important health

outcomes like T2DM, CVD, and mortality, which possibly results from using different criteria used to define sarcopenia and obesity. Stephen and Janssen (2009) in a prospective study (n=3366 aged ≥65 years) have shown that irrespective of how SO has been classified the crude event rates for CVD were higher in the SO group than in the sarcopenic and obese groups and that on comparison with the normal group, the CVD event rates increased by 23% when obesity and muscle mass were used as criteria, whereas these rates increased by 33% when obesity and muscle strength were used. This implies that the association of SO with CVD becomes stronger when muscle strength is used as a defining parameter of SO instead of muscle mass.

Similarly, Atkins et al. (2014) (n=4,252; age 60–79 years) have shown lower hand grip strength to be strongly associated with an increased risk of CVD and all-cause mortality and which is also a good prognostic indicator for the mortality risk in adults with or without obesity. Also, the fact that muscle strength decline precedes and exceeds the decline in muscle mass with ageing, inferring that muscle strength may be included as a mandatory parameter for defining sarcopenia/SO (Goodpaster et al. 2006). Thus, in a community where state-of-the-art-technologies like DXA scans and others for assessing muscle mass are not available and/or applicable, muscle strength may be used as a cheaper and more effective diagnostic tool for assessing sarcopenia.

Conclusion

There is a higher prevalence of sarcopenia and SO in patients with type T2DM and CVD. CVD may also be independently associated with sarcopenia/SO in T2DM patients. However, the magnitude of this association and risk it may have in patients with T2DM needs to be studied further. Positive efforts in this direction will effortlessly reflect in the betterment of patient health with better outcomes.

Conflicts of interest: *The authors declare no conflicts of interest.*

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