

Review Article

SARCOPENIC OBESITY IN INDIA: AN UPATED REVIEW OF PATHOPHYSIOLOGY, DIAGNOSIS, AND MANAGEMENT

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ABSTRACT

Sarcopenic obesity (SO) is an emerging, but under-recognized public health problem in India and is defined as having a low level of skeletal muscle mass or function with excessive adipose tissue. There is a complex epidemiological situation in the country, and the nutrition transition, sedentary urbanisation and insufficient protein intake are causing an increasing obesity epidemic and population aging. The prevalence of SO has been reported from various epidemiological studies in India as 5.4% and 8.7% in older people, and is basically higher in type 2 diabetes mellitus (T2DM) conditions. Pathophysiology is complex, with various serious condition such as myosteatosis, chronic low grade inflammation, insulin resistance, hormonal imbalance and sometimes mitochondrial membrane potential alteration and dysfunction. In this review, it underlines the current Indian evidence available for SO and its need for population specific diagnostic cut-offs and recommends for policy driven, multidisciplinary interventions to address this geriatric multi-compounding syndrome.

Keywords: Sarcopenic obesity, Aging, Type 2 diabetes, Hormonal changes.

INTRODUCTION

The combination of low skeletal muscle mass/low skeletal muscle function and excess fat, Sarcopenic obesity (SO), is an increasing recognised, clinically relevant phenotype. The concepts of SO were initially introduced in 1998 by Baumgartner and further developed in a number of international consensus statements; SO is an increasing metabolic and functional deterioration and the risk of SO is greater than the risk of metabolic or functional deterioration alone. It is important to note that the European Association for the Study of Obesity (EASO) and the European Society for Clinical Nutrition and Metabolism (ESPEN) reached consensus on new criteria for diagnosis in 2022, a step towards standardisation of diagnosis in the field all over the globe.^[1,2]

India is an epidemiological challenge that is different. There are currently over 140 million in the world over the age of 60 and this number will double by 2050. The implications of the effects of aging muscle loss are colossal with more than 140 million people aged over 60 and this number is expected to double by

2050. Concurrently, India's nutritional transition (decreased protein intake, increased carbohydrate intake, and decreased physical activity) has led to an obesity epidemic especially in urban settings. Likewise, as India's country is transitioning to a less protein rich, more carbohydrates rich and less active lifestyle has contributed to an obesity epidemic, particularly in urban areas. This convergence of trends makes SO an important public health problem in the Indian context that requires immediate attention from both clinicians as well as researchers and policy makers.^[3,4]

This brief review is intended to summarise the epidemiology of SO in India, discuss pathophysiological mechanisms, explain the clinical context of various diagnostic approaches that have been found to be useful in the Indian setting, explore implications of an SO diagnosis and discuss effective strategies to manage SO.

Epidemiology of Sarcopenic Obesity in India

India has several deficit areas with regards to systematic epidemiological data on SO, which is slowly improving. The largest nationally representative study of older adults, the Longitudinal

Aging Study of India (LASI) Wave 1 (2017–18) has sampled 65,562 individuals aged ≥ 45 years. The prevalence of possible sarcopenia and obesity were reported as 41.9% and 27.1%, respectively, and 8.7% was the prevalence of SO according to the AWGS-2019 criteria. Adults with SO had significantly higher mean age, BMI and blood pressure. Geographic clustering in the west and south of India, urbanization, tobacco use, alcohol use, physical inactivity and comorbid diabetes were identified as independent factors for SO.^[5,6]

In SO-CUBES (Chandigarh) study, prevalence of SO (based on EWGSOP2 criteria with indigenous cut-off) was seen to be 5.4%–6.3% in apparently healthy elderly Indians (≥ 65 years). The study demonstrated the usefulness of BMI along with WC and fat mass percentage derived from DXA as screening tools for SO, which makes BMI-based screening possible in a resource-limited setting.^[4]

A high prevalence of SO is noted in high-risk groups. In a cross sectional study conducted on 750 participants including 250 diabetic, 250 nondiabetic healthy and 250 obese nondiabetic people from Gujarat in 2024, Patel et al found that 40% of the diabetic group had SO, compared to 11% in the nondiabetic healthy group, implying a 2.4-fold increase (95% CI: 1.1–5.0) in the risk of SO in diabetic patients over controls after adjusting for covariates. In this study, there was strong association between the severity of the SO and duration of diabetes and glycaemic control, highlighting the metabolic relationship between T2DM and SO. In addition, SO rates in recent pilot data from a 2025 study in patients with established cardiovascular disease showed even higher rates of SO, indicating that there is a synergistic relationship between cardiovascular disease and musculoskeletal disease.^[6]

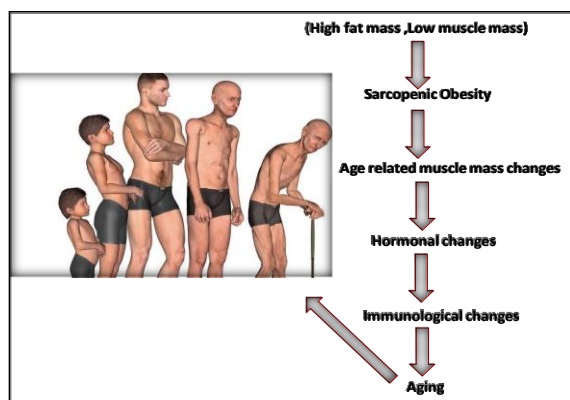


Figure 1: Age associated changes during sarcopenic obesity represented.

Pathophysiology

Aging and unhealthy lifestyle habits common in India further potentiates the cascade of complex, interacting events involved in SO with the adipose-muscle crosstalk, systemic inflammation, hormonal regulation and mitochondrial dysfunction. Intramuscular and perimuscular fat infiltration not

only compromises myocyte contractility but also causes ectopic fat deposition, which in turn leads to a reduction in metabolic efficiency. The standard BMI thresholds used in the western populations are insufficient to diagnose the problem of obesity in Indian individuals especially with metabolically obese normal weight (MONW) or “thin-fat” phenotype.^[8]

Chronic low-grade inflammation: Inflammatory cytokines secreted by hypertrophied adipocytes such as TNF- α , IL-6 and IL-1 β stimulate ubiquitin-proteasome pathway, thus promoting proteolysis. In Indian SO cases, there is a consistent increase in C reactive protein, inflammatory adipokines (leptin, resistin) and a decrease in adiponectin, which further accelerates muscle catabolism.^[9,10]

Insulin resistance: Skeletal muscle is the main tissue with insulin-dependent glucose uptake. Insulin resistance in SO leads to a decrease in anabolic signalling through the PI3K/Akt/mTOR pathway which slows muscle protein synthesis. Concurrent hyperinsulinaemia promotes adipogenesis, establishing a vicious cycle of fat accumulation and muscle wasting.^[11]

Elevated cortisol and relative decrease in growth hormone (GH), insulin-like growth factor-1 (IGF-1), and testosterone (T) results in a net increase in muscle catabolism and fat deposition. Men and women of the Indian origin, who have SO, always have low levels of IGF-1. The hormonal milieu is further compromised by hypothyroidism, which is common in India, especially subclinical hypothyroidism in women.^[12]

Dietary protein insufficiency: Although Indians typically eat foods that are good for their health, they lack sufficient amounts of essential amino acids, especially leucine, a critical mTORC1 stimulator of protein synthesis. Low protein consumption (less than 0.8 g/kg/day in a significant number of elders in India) exacerbates the negative nitrogen balance that is a characteristic of aging.^[13,14]

Pathological Perspective: Low-grade chronic inflammation is the common link between the processes of sarcopenic obesity, and the pathology of the disease is characterized by structural and molecular changes in both skeletal muscle and adipose tissue.^[15]

Skeletal Muscle Histopathology: Myosteatorsis is the most consistent finding in muscle biopsy specimens and is represented by two forms: intramyocellular lipid droplets (within myofibers) and intermyocellular adipose tissue (between muscle fascicles). The fat infiltration is well appreciated with Oil Red O staining on frozen sections and fiber atrophy/architectural disorganization is well appreciated with H&E staining. FTA shows selective atrophy of fibres of type II (fast-twitch, glycolytic); type I (slow-twitch, oxidative) fibres are relatively spared, a differential susceptibility that can also be verified by immunohistochemical staining of the isoforms of myosin heavy chain.^[16]

Adipose Tissue Pathology: Adipocyte hypertrophy is especially seen in visceral and ectopic fat depots and causes relative local hypoxia (up-regulation of HIF-1 α). The hallmark of this process is the accumulation of M1-polarized macrophages around necrotic or dying adipocytes, which can be detected with CD68 immunostaining and are referred to as "crown-like structures". These structures generate a chronic inflammatory microenvironment.^[17]

Molecular and cellular mechanisms: Adipose tissue produces too much of the inflammatory biomarkers (TNF- α , IL-6, CRP, and leptin) which trigger muscle catabolism, and there is a reduction of the protective, anti-inflammatory biomarker, adiponectin. At the molecular level, protein degradation pathways are activated to a much greater extent, especially the ubiquitin-proteasome system, as evidenced by increased expression of muscle-specific E3 ubiquitin ligases (MuRF1 and MAFbx/Atrogin-1) by qRT-PCR or western blot studies. At the same time, the entire autophagy-lysosomal system is also impaired.^[18]

Dysfunction of the mitochondria and the regeneration cells: Electron microscopy findings report fewer mitochondria and fewer cristae (mitochondrial oxidative phosphorylation) that are indicative of impaired ATP production, which is sometimes described as "mitochondrial dysfunction of sarcopenic obesity. At the same time, the satellite cell pool (Pax7+ cells) becomes smaller and less regenerator, thus severely affecting muscle repair and regeneration.^[15,19]

Extracellular Matrix Remodelling: Progression of fibrosis occurs in the interstitium of the muscle, characterized by collagen deposition (as detected by Masson's trichrome) and activation of fibroblasts to increase tissue stiffness and impair further muscle contraction. There have also been some recent reports of increased bone marrow adiposity, which could influence the hematopoietic microenvironment and increase systemic inflammatory burden — an area which still needs to be fully pathologically characterised.^[20]

Diagnostic Challenges in India: One basic problem is the lack of a universally accepted, India-specific diagnostic definition. There are two sets of international criteria available, which use different thresholds for appendicular lean mass (ALM), handgrip strength and gait speed: EWGSOP2 (European) and AWGS-2019 (Asian). Although AWGS-2019 has a somewhat more ethnical appropriateness due to the similarity in body composition between south and east Asians, it was not created from Indian population.^[21]

The current reference standard for body composition assessment is dual-energy X-ray absorptiometry (DXA), but this is not suitable for population level screening due to limited availability in district hospitals and peripheral health centres. A low cost alternative is bioelectrical impedance analysis (BIA) which is validated in Indian adults with the use of

population specific equations to avoid systematic bias.^[22,23]

The Low Appendicular Lean Mass (ALM) indices were low and visceral fat was high, even in individuals with a normal BMI, making it difficult to accurately estimate the prevalence of SO using the BMI-based definition of obesity (BMI ≥ 30 kg/m²). Some of this is addressed by revised BMI criteria for Asian populations (BMI ≥ 25 kg/m² for obesity), however further normative data for Asian populations is urgently required.^[24]

Clinical Consequences: SO confers greater morbidity and mortality risk than either sarcopenia or obesity alone. In India, SO is closely related to:

Cardiometabolic Disease: SO has been shown to be independently associated with T2DM, hypertension, dyslipidaemia and non-alcoholic fatty liver disease (NAFLD). Some studies in India reported that the prevalence of metabolic syndrome in SO patients is more than 60%.^[25]

Physical Disability and Falls: The physical disability in SO is associated with loss of muscle strength and altered gait mechanics, which significantly increases fall risk, fracture incidence, activity limitation, which has significant implications on the healthcare cost and caregiver burden of the aging population in India.^[26]

Cognitive Decline: There is emerging evidence that SO is associated with an increased risk of cognitive decline and dementia, in part through common inflammatory pathways and lower cerebral blood flow. The mortality risk of SO is around 1.5-fold higher than for isolated sarcopenia or obesity, especially for cardiovascular and all-cause mortality.^[27]

Management Strategies: Integrated, multidisciplinary interventions are needed to address both muscle preservation and fat reduction in the management of SO. Progressive resistance training is still considered the mainstay of the SO management, known as Resistance Exercise Training (RET).^[28] RET increases protein synthesis, decreases intramuscular fat and increases insulin sensitivity, all of which are mediated by mTORC1. Even low intensity RET (2-3 sessions per week) over 12 weeks has shown to improve muscle strength and functional capacity in older adults in India.^[29] Aerobic and resistance programmes are favoured over aerobic or resistance programmes alone.

Nutritional Optimisation: The protein level for older adults with SO is 1.2–1.6 g per kg of body weight per day, with a preference for proteins that contain leucine. In the Indian context, adding dals, soy, dairy and eggs to the diet is culturally appropriate and effective. Omega-3 fatty acids have been found to be anti-inflammatory and anabolic; they may have special significance for the people of India who consume a plant-food diet. Since hypovitaminosis D is widely prevalent in India and muscle weakness and adiposity are aggravated, vitamin D supplementation is strongly recommended.^[30]

Pharmacological Approaches: No drug has been approved for SO. GLP-1 receptor agonists (e.g., semaglutide) have been found to be beneficial for adiposity reduction and partially preserve lean mass, with differential effects on muscle in SO yet to be investigated. There are clinical trials for myostatin inhibitors and selective androgen receptor modulators (SARMs). Early pharmacological glycaemic management might be enough to reduce the progression of sarcopenia in Indian patients with T2DM and SO.^[31]

Research gaps and future directions: There are multiple critical gaps to progress towards addressing SO in India. First, a nationally representative cohort data with standardised SO definitions is lacking, and second, the data is not prospective. Second, India-specific diagnostic cut-off points for the ALM, handgrip strength and gait speed are urgently needed as these were identified with large, ethnically diverse sample populations. Third, there are virtually no intervention trials testing for SO in Indians, including community-based and scalable interventions. Fourth, the interplay of SO and country-specific risk factors (protein-energy malnutrition in rural areas and metabolic obesity in urban areas, high carbohydrate diet, physical inactivity) needs to be explored separately. Incorporating SO screening into the current India's national geriatric health programmes, specifically under the framework of the Longitudinal Aging Study of India, suggests a feasible approach.^[32,33]

CONCLUSION

Due to these factors, sarcopenic obesity is a new and growing public health problem in India where aging is adding an increasing burden, nutritional transition is contributing to this burden, and metabolic disease is a public health epidemic in the country. The clinical and health system implications are significant since the prevalence of SO is nearing 8.7% in the general population of older Indians in a community setting and much more prevalent in high-risk groups. An India specific, consolidated diagnostic framework with support from big epidemiological studies and evidence-based intervention trials is an urgent priority. In the meantime, the AWGS-2019 criteria must be used with Asian-appropriate cut-offs, and a low threshold for screening should be used in older adults with obesity, diabetes or physical inactivity. The present best evidence for modifying the trajectory of SO in India is through multidisciplinary management that involves resistance training, protein-optimised nutrition and targeted pharmacotherapy.

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