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Sarcopenic Obesity: A Growing Public Health Concern – A Literature Review

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Abstract

Background: Sarcopenic obesity (SO) is a growing clinical and public health concern characterized by the coexistence of excess adiposity and reduced skeletal muscle mass and function. It is increasingly prevalent across various age groups, particularly among individuals with chronic conditions.

Methods: This review is based on a narrative synthesis of peer-reviewed literature published between 2015 and 2024. Articles were identified through PubMed, limited to free full-text resources in English. Recent consensus guidelines from the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) were also included.

Results: SO is a multifactorial condition driven by aging, physical inactivity, chronic inflammation, insulin resistance, hormonal changes, oxidative stress, and lipotoxicity. Its prevalence varies widely due to inconsistent diagnostic standards but is notably higher in older adults and those with comorbidities such as type 2 diabetes and cardiovascular disease. Sarcopenic obesity contributes to the development of metabolic disorders, physical limitations, reduced life quality, and an elevated risk of death. Effective management requires a combination of lifestyle interventions, including resistance training, adequate protein intake, and weight management.

Conclusion: Early diagnosis and targeted interventions are essential to mitigate the impact of sarcopenic obesity. Further research is necessary to refine diagnostic criteria and develop novel therapeutic approaches to improve outcomes and reduce healthcare burdens.

Keywords: Sarcopenic Obesity, Muscle Mass Loss, Body Composition, Lifestyle Intervention, Sarcopenia, Obesity.

1. Introduction

Sarcopenic obesity (SO) is a medical condition defined by the coexistence of two significant pathophysiological factors: sarcopenia, the progressive loss of muscle mass and function, and obesity, characterized by excessive body fat accumulation [1,2]. SO is increasingly recognized as a major public health issue, particularly in aging populations, where it contributes to adverse metabolic, functional, and physiological outcomes [3,4,5]. Unlike obesity or sarcopenia alone, SO presents a unique challenge due to the simultaneous presence of low muscle mass and excessive fat, which exacerbates metabolic dysfunction, insulin resistance, and physical frailty [6,7,8]. The synergistic interaction between excessive adiposity and muscle degradation amplifies metabolic disturbances, creating a vicious cycle that exacerbates physical decline and insulin resistance [9,10].

The pathophysiology of SO is multifactorial, involving factors such as aging, physical inactivity, poor nutrition, metabolic disturbances, and hormonal imbalances [11,12,13,14]. This article provides a comprehensive overview of SO, including its prevalence, risk factors, clinical implications, treatment strategies, and the impact of medications on muscle and bone health [15,16,17,18]. The aim of this review is to synthesize current knowledge on the prevalence, pathophysiology, clinical implications, and management strategies of sarcopenic obesity.

2. Epidemiology

The global prevalence of sarcopenic obesity remains difficult to determine due to variations in diagnostic criteria and the lack of standardized definitions for sarcopenia and obesity [1,4]. The prevalence of SO varies considerably across studies due to differences in populations, diagnostic methods, and definitions of obesity and sarcopenia [1,5].

A 2021 meta-analysis incorporating 50 studies and 86,285 adults aged 60 years and older estimated the global prevalence of SO at 11% (95% CI 10–13%) [8]. However, high heterogeneity was observed, with estimates ranging from 0.1% to 48%. This variability reflects the diverse study designs and diagnostic criteria employed [4,5]. Notably, the prevalence of SO increases with age, with a marked rise in individuals aged 75 years and older, where approximately one in four individuals is affected [3,19].

2.1 Gender and Geographical Variations

The prevalence of sarcopenic obesity differs between sexes, with some studies indicating notable variation. Some studies indicate higher rates in women, while others report similar prevalence in both sexes. For instance, data from the National Health and Nutrition Examination Survey (NHANES) indicated an SO prevalence of 12.6% in men and 33.5% in women [19]. A broader meta-analysis across 48 studies reported comparable rates in men and women (9%) [5,8].

Geographically, the prevalence of SO is higher in South America (21%) and North America (19%) compared to Europe (12%) [4,5]. These differences are likely attributed to variations in dietary patterns, lifestyle behaviors, and healthcare access [4].

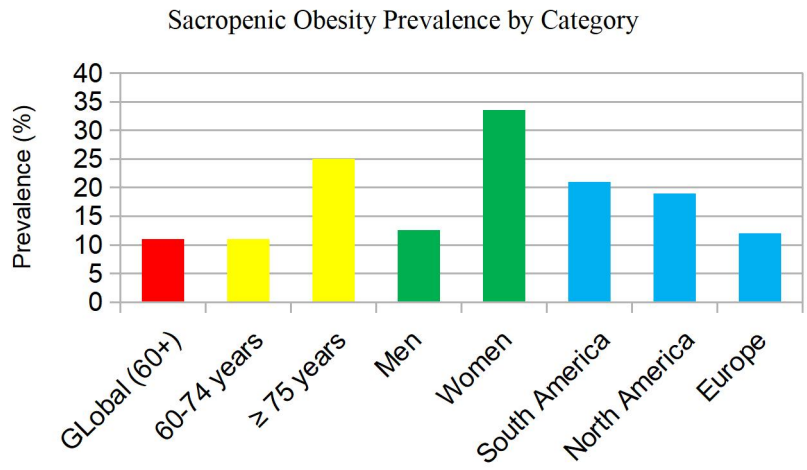

Chart 1. Epidemiology of Sarcopenic Obesity [3,4,5,8,19].

2.2 Impact of Diagnostic Criteria on Prevalence Estimates

The definition and diagnostic criteria used to identify sarcopenia and obesity play a crucial role in determining prevalence estimates [1,5]. Studies that adjust muscle mass by body weight tend to report higher rates of SO (approximately 23%), whereas studies that adjust for height squared report lower rates (around 8%) [5]. Adjusting muscle mass by body weight may overestimate SO in individuals with higher adiposity, while height-based adjustments may underestimate it in shorter individuals with preserved BMI but low lean mass [1]. The variability in prevalence underscores the need for standardized diagnostic criteria, and recent guidelines from the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) are expected to improve diagnostic consistency [1].

3. Diagnostic Criteria

The diagnostic criteria for sarcopenic obesity (SO) have been evolving, and a lack of universal consensus has historically made diagnosis challenging [1,4]. However, recent efforts, particularly the consensus statement from the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO), provide a structured approach [1].

According to ESPEN and EASO guidelines, the diagnostic process for SO involves three distinct stages: initial screening, formal diagnosis, and clinical staging. It is shown in Table 1.

Step	Component	Details
1. Screening [1,4]	**Elevated Adiposity Indicator**	- **BMI**: WHO cut-offs - **Waist Circumference (WC)**: Ethnicity-specific cut-offs (e.g., NIH for Caucasians, Misra et al. for Asians)
	Sarcopenia Risk Indicators	- Clinical symptoms or risk factors - SARC-F questionnaire (especially in older adults)
	Criteria to proceed	**Both elevated BMI/WC and sarcopenia risk indicators must be present**
2. Diagnosis [1,4,5]	**Muscle Function Assessment**	- **Hand-Grip Strength (HGS)** - **Knee Extensor Strength** - **Chair Stand Test (e.g., 5-times sit-to-stand)** *(No specific test is preferred; all require population-specific reference values)*
	Body Composition Assessment	**If muscle function is impaired**, assess body composition: **DXA (preferred)**: - Measurement: ALM/W (Appendicular Lean Mass / Weight) - Advantages: High precision, low radiation, regional assessment. - Disadvantages: High cost, not portable. **BIA (alternative)**: - Measurement: ALM/W or SMM/W (Skeletal Muscle Mass / Weight) - Advantages: Portable, low cost. - Disadvantages: Sensitive to hydration status; potential for misclassification in individuals with BMI > 34 kg/m². **CT/MRI (optional):** - MRI: Precise, no radiation - CT: Opportunistic muscle analysis - Disadvantages: High cost, limited availability in routine clinical practice.
	Obesity Definition	Based on **% Fat Mass (%FM)**, ideally adjusted for height; ALM/W or SMM/W used for sarcopenia
3. Staging [1,4]	**Stage I**	No complications from altered body composition/muscle function
	Stage II	Presence of complications (e.g., metabolic disease, cardiovascular/respiratory issues, disability)

Table 1. Diagnostic criteria for sarcopenic obesity (SO). BMI – Body Mass Index; DXA – Dual-Energy X-ray Absorptiometry; BIA – Bioelectrical Impedance Analysis; CT – Computed Tomography; MRI – Magnetic Resonance Imaging.

4. Risk Factors and Pathophysiology

Several interconnected factors contribute to the development of sarcopenic obesity, including aging, physical inactivity, inadequate nutrition, low-grade inflammation, and metabolic dysregulation [9,10,11,12,13].

Category	Key Factors	Mechanisms / Implications
1. Aging-Related Changes [9,13]	- Natural loss of muscle mass & strength - Increase in visceral fat mass - Decline in basal metabolic rate (BMR)	- Reduced anabolic response - ↑ Insulin resistance risk - Promotes fat infiltration into muscle (myosteatosis)
2. Lifestyle Factors [9,14]	- Physical inactivity - Poor nutrition (e.g., low protein, micronutrient deficiencies) - Excess calorie intake	- Muscle atrophy from disuse - Impaired muscle protein synthesis - Fat accumulation due to sedentary lifestyle and energy imbalance
3. Metabolic & Inflammatory Factors [10,11]	- Insulin resistance - Chronic low-grade inflammation - Ectopic fat deposition - Oxidative stress, mitochondrial dysfunction	- Impaired glucose uptake by muscle - Muscle breakdown from pro-inflammatory cytokines (e.g., TNF-α, IL-6) - Decreased muscle quality and metabolic health
4. Hormonal Dysregulation [12,13]	- Declines in testosterone, estrogen, GH, IGF-1 - Elevated cortisol - Leptin resistance	- ↓ Muscle protein synthesis - ↑ Fat deposition - Hormonal imbalance exacerbates sarcopenia and obesity
5. Biological & Environmental Factors [9,12,13]	- Genetic predisposition (e.g., obesity-related gene variants) - Gut microbiota dysbiosis - Social/environmental factors (e.g., food access, exercise opportunities)	- Genetic influence on body composition - Microbial metabolites affect inflammation and insulin sensitivity - Socioeconomic factors impact lifestyle and health behaviors

Table 2. Risk factors and pathophysiology. BMR – Basal Metabolic Rate; TNF-α – Tumor Necrosis Factor-alpha; IL-6 – Interleukin 6; GH – Growth Hormone; IGF-1 – Insulin-like Growth Factor 1.

5. Clinical Implications

Sarcopenic obesity carries significant health risks, including increased disability, frailty, and the development of metabolic disorders such as type 2 diabetes and cardiovascular disease [3,4,6,7]. Individuals with SO are at greater risk for mobility impairments, poor physical function, and falls, which may lead to fractures and long-term disability [4,8,20]. Additionally, SO is associated with increased all-cause mortality, making it a critical area of concern for healthcare providers [3,8]. Some of the clinical implications of SO are shown in Table 3.

Category	Key Clinical Implications
Mortality & Frailty [3,8]	- ↑ All-cause mortality (51% higher risk) - ↑ Risk in hospitalized patients - Strong risk factor for frailty
Cardiovascular Disease (CVD) [4,6]	- ↑ Risk of CAD, stroke, and heart disease - ↑ CVD-related mortality - Linked to coronary artery calcification, lower event-free survival in STEMI, and possibly atrial fibrillation
Metabolic Disorders [4,7]	- ↑ Risk of insulin resistance, dyslipidaemia, atherosclerosis - ↑ Risk of type 2 diabetes, especially in "pre-SO" individuals - In diabetics: ↑ risk of death, fragility fractures, CKD, and CVD events - Possible link with metabolic syndrome
Physical Disability & Function [4,9,20]	- ↑ Risk of disability and limitations - ↑ Risk of falls and osteoarthritis - Muscle loss contributes to inactivity and mobility issues
Bone Health [4,8]	- Coexistence with osteoporosis (OP) ↑ fracture risk - May reduce motor function and mobility
Hospitalization & Recovery [4,8]	- ↑ Risk of prolonged hospital stays and infections - ↓ Quality of life and work capacity - Psychological and economic burden
Cancer Outcomes [21]	- ↑ Risk of cancer recurrence and mortality (e.g., pancreatic cancer) - ↑ Post-op complications - Sarcopenia is a negative prognostic factor
Cognitive Health [4]	- Associated with cognitive impairment, especially in the elderly
Clinical Challenges [1,4]	- Poor clinical management due to inconsistent definitions - Need for standardized criteria to improve prevalence estimates, risk prediction, and treatment strategies

Table 3. Clinical implications of SO. CAD – Coronary Artery Disease; STEMI – ST-segment Elevation Myocardial Infarction; CKD – Chronic Kidney Disease; OP – Osteoporosis; ↑ – increases.

5.1 Impact of Medications on Muscle and Bone Mass

Medication/Compound	Effects on Muscle/Bone	Clinical Notes
Myostatin & Activin Receptor Inhibitors (e.g., Bimagrumab) [16]	↑ Muscle mass & strength, ↑ BMD, ↓ Fat mass	Promising; shown to reverse atrophy and improve body composition
Denosumab [16]	↑ Insulin sensitivity, potential muscle benefits	Needs more data on direct muscle impact; used in osteoporosis
Creatine + Resistance Training [16]	↑ Muscle strength, ↓ Falls & fractures	Effective in older adults
Metformin [14]	↓ Inflammation, may restore muscle function	Dual effects; may also ↑ myostatin, leading to muscle atrophy
Ghrelin Mimetics [16]	↑ Lean mass, ↓ Inflammation	Potential in catabolic states
Semaglutide [15,16]	↓ Fat mass, preserves lean mass	Muscle-sparing in T2DM patients
Tirzepatide [16]	↓ Fat mass, improved fat-to-lean ratio	Muscle mass preservation unclear
Leptin [12]	Conflicting: ↑ muscle in rodents, but ↓ density in older humans	Linked to muscle atrophy in aging
Testosterone [16]	↑ Muscle mass/function in hypogonadal men	Benefits vary; long-term safety uncertain
Vitamin D [22]	Potential ↑ in muscle strength, mass & BMD	Mixed evidence; commonly deficient in elderly
Incretin-Mimetics (IMDs) [18]	↓ Weight, but may also ↓ muscle mass	Exercise recommended alongside to preserve muscle
Osteocalcin [16]	↑ Muscle mass, ↑ Exercise capacity	Shown effective in animal models
RANKL/RANK/OPG Modulators [16]	↓ Muscle atrophy, ↑ Insulin sensitivity	Denosumab may benefit via this pathway
Meteorin-like protein (METRNL) [16]	↑ Bone regeneration, fracture healing	Early research stage
Dihydromyricetin [17]	Protects mitochondria, prevents muscle wasting	Promising in preclinical models
Selective Androgen Receptor Modulators (SARMs), GH, Vitamin K, Adiponectin [7,16]	Experimental ↑ in muscle/bone mass	Require more human studies

Table 4. Impact of medications on muscle and bone mass. T2DM – Type 2 Diabetes Mellitus; BMD – Bone Mineral Density; ↑ – increases; ↓ – decreases.

It is important to note that the effects of medications can vary depending on the individual's condition, age, and other factors, and further research is often needed to fully understand their long-term impact on muscle and bone health [7,16,18].

5.2 Economic Impact

The healthcare burden associated with SO includes increased hospitalizations, prolonged rehabilitation, and higher healthcare costs, underscoring the need for early diagnosis and intervention [4,8].

6. Treatment of Sarcopenic Obesity

Management of sarcopenic obesity requires a comprehensive, individualized approach that combines exercise, nutrition, pharmacological interventions, and lifestyle changes [4,9,14].

6.1 Combined Exercise and Nutritional Interventions

Exercise remains the cornerstone of treatment for SO, particularly resistance training, which is essential for stimulating muscle protein synthesis and increasing muscle mass and strength [10,23]. Aerobic exercises also play a vital role in managing body weight, enhancing muscle function, and reducing inflammation [9].

Nutritional strategies are equally important, with an emphasis on adequate protein intake (1.0–1.5 g/kg body weight/day) to support muscle maintenance and function. High-quality proteins, particularly those rich in leucine, such as whey protein, are recommended [14].

6.2 Pharmacological Interventions

Though no specific drugs are approved for SO treatment, pharmacological agents under investigation include GLP-1 receptor agonists, myostatin inhibitors, and recombinant irisin [16,17,18]. These medications may assist in reducing fat mass, increasing muscle mass, and improving overall body composition [15,16,17].

6.3 Physical Stimulation

For individuals who are unable to participate in traditional exercise, therapies like Electrical Muscle Stimulation (EMS) and Whole-Body Vibration (WBV) may improve muscle strength and function [4,16].

6.4 Lifestyle Interventions

Lifestyle changes, including smoking cessation and stress reduction, are also important components of SO management. Smoking accelerates muscle loss, while chronic stress negatively affects metabolism and body composition [9,12,13].

7. Conclusion

Sarcopenic obesity is a complex and growing public health concern, particularly among older adults [3,4,5]. The combination of low muscle mass and excessive fat leads to significant metabolic, functional, and physiological challenges [6,7,9]. Effective management requires a multifaceted approach that includes exercise, nutritional optimization, and potential pharmacological interventions [9,14,16]. Ongoing research is essential to develop standardized diagnostic criteria and refine treatment strategies for this increasingly prevalent condition [1,4,18].

As research into sarcopenic obesity continues, novel therapeutic targets and more precise guidelines for its management are likely to emerge, offering new hope for improving the quality of life and survival of affected individuals [8,10].

DISCLOSURE

Authors' contribution

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Software: not applicable
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References

1. Donini LM, Busetto L, Bischoff SC, Cederholm T, Ballesteros-Pomar MD, Batsis JA, Bauer JM, Boirie Y, Cruz-Jentoft AJ, Dicker D, Frara S, Frühbeck G, Genton L, Gepner Y, Giustina A, Gonzalez MC, Han HS, Heymsfield SB, Higashiguchi T, Laviano A, Lenzi A, Nyulasi I, Parrinello E, Poggiogalle E, Prado CM, Salvador J, Rolland Y, Santini F, Serlie MJ, Shi H, Sieber CC, Siervo M, Vettor R, Villareal DT, Volkert D, Yu J, Zamboni M, Barazzoni R. Definition and Diagnostic Criteria for Sarcopenic Obesity: ESPEN and EASO Consensus Statement. Obes Facts. 2022;15(3):321-335. doi: 10.1159/000521241. Epub 2022 Feb 23. PMID: 35196654; PMCID: PMC9210010.
2. Vogele D, Otto S, Sollmann N, Haggenmüller B, Wolf D, Beer M, Schmidt SA. Sarcopenia - Definition, Radiological Diagnosis, Clinical Significance. Rofo. 2023 May;195(5):393-405. English, German. doi: 10.1055/a-1990-0201. Epub 2023 Jan 11. PMID: 36630983.
3. Wannamethee SG, Atkins JL. Sarcopenic Obesity and Cardiometabolic Health and Mortality in Older Adults: a Growing Health Concern in an Ageing Population. Curr Diab Rep. 2023 Nov;23(11):307-314. doi: 10.1007/s11892-023-01522-2. Epub 2023 Aug 11. PMID: 37566368; PMCID: PMC10640508.
4. Wei S, Nguyen TT, Zhang Y, Ryu D, Gariani K. Sarcopenic obesity: epidemiology, pathophysiology, cardiovascular disease, mortality, and management. Front Endocrinol (Lausanne). 2023 Jun 30;14:1185221. doi: 10.3389/fendo.2023.1185221. PMID: 37455897; PMCID: PMC10344359.
5. Danielewicz AL, Marra A, Tringali G, Micheli R, Abbruzzese L, Fanari P, Codecasa F, Lazzer S, Mendonça VA, Lacerda ACR, de Avelar NCP, Sartorio A. Analysis of sarcopenic obesity prevalence and diagnostic agreement according to the 2022 ESPEN and EASO Consensus in hospitalized older adults with severe obesity. Front Endocrinol (Lausanne). 2024 Jun 20;15:1366229. doi: 10.3389/fendo.2024.1366229. PMID: 38966224; PMCID: PMC11222587.
6. Mirzai S, Carbone S, Batsis JA, Kritchevsky SB, Kitzman DW, Shapiro MD. Sarcopenic Obesity and Cardiovascular Disease: An Overlooked but High-Risk Syndrome. Curr Obes Rep. 2024 Sep;13(3):532-544. doi: 10.1007/s13679-024-00571-2. Epub 2024 May 16. PMID: 38753289; PMCID: PMC11306598.
7. Wang M, Tan Y, Shi Y, Wang X, Liao Z, Wei P. Diabetes and Sarcopenic Obesity: Pathogenesis, Diagnosis, and Treatments. Front Endocrinol (Lausanne). 2020 Aug 25;11:568. doi: 10.3389/fendo.2020.00568. PMID: 32982969; PMCID: PMC7477770.
8. Veronese N, Ragusa FS, Pegreffi F, Dominguez LJ, Barbagallo M, Zanetti M, Cereda E. Sarcopenic obesity and health outcomes: An umbrella review of systematic reviews with meta-analysis. J Cachexia Sarcopenia Muscle. 2024 Aug;15(4):1264-1274. doi: 10.1002/jcsm.13502. Epub 2024 Jun 19. PMID: 38897937; PMCID: PMC11294015.
9. Bilski J, Pierzchalski P, Szczepanik M, Bonior J, Zoladz JA. Multifactorial Mechanism of Sarcopenia and Sarcopenic Obesity. Role of Physical Exercise, Microbiota and Myokines. Cells. 2022 Jan 4;11(1):160. doi: 10.3390/cells11010160. PMID: 35011721; PMCID: PMC8750433.

10. Axelrod CL, Dantas WS, Kirwan JP. Sarcopenic obesity: emerging mechanisms and therapeutic potential. Metabolism. 2023 Sep;146:155639. doi: 10.1016/j.metabol.2023.155639. Epub 2023 Jun 26. PMID: 37380015; PMCID: PMC11448314.
11. Jung UJ. Sarcopenic Obesity: Involvement of Oxidative Stress and Beneficial Role of Antioxidant Flavonoids. Antioxidants (Basel). 2023 May 8;12(5):1063. doi: 10.3390/antiox12051063. PMID: 37237929; PMCID: PMC10215274.
12. Mazurkiewicz Ł, Czernikiewicz K, Grygiel-Górniak B. Immunogenetic Aspects of Sarcopenic Obesity. Genes (Basel). 2024 Feb 5;15(2):206. doi: 10.3390/genes15020206. PMID: 38397196; PMCID: PMC10888391.
13. Gross DC, Cheever CR, Batsis JA. Understanding the development of sarcopenic obesity. Expert Rev Endocrinol Metab. 2023 Sep-Nov;18(6):469-488. doi: 10.1080/17446651.2023.2267672. Epub 2023 Nov 28. PMID: 37840295; PMCID: PMC10842411.
14. Faria I, Samreen S, McTaggart L, Arentson-Lantz EJ, Murton AJ. The Etiology of Reduced Muscle Mass with Surgical and Pharmacological Weight Loss and the Identification of Potential Countermeasures. Nutrients. 2024 Dec 31;17(1):132. doi: 10.3390/nu17010132. PMID: 39796566; PMCID: PMC11723338.
15. Xiang J, Ding XY, Zhang W, Zhang J, Zhang YS, Li ZM, Xia N, Liang YZ. Clinical effectiveness of semaglutide on weight loss, body composition, and muscle strength in Chinese adults. Eur Rev Med Pharmacol Sci. 2023 Oct;27(20):9908-9915. doi: 10.26355/eurrev_202310_34169. PMID: 37916360.
16. Žižka O, Haluzík M, Jude EB. Pharmacological Treatment of Obesity in Older Adults. Drugs Aging. 2024 Nov;41(11):881-896. doi: 10.1007/s40266-024-01150-9. Epub 2024 Nov 8. PMID: 39514148; PMCID: PMC11554829.
17. Old VJ, Davies MJ, Papamargaritis D, Choudhary P, Watson EL. The Effects of Glucagon-Like Peptide-1 Receptor Agonists on Mitochondrial Function Within Skeletal Muscle: A Systematic Review. J Cachexia Sarcopenia Muscle. 2025 Feb;16(1):e13677. doi: 10.1002/jcsm.13677. PMID: 39815782; PMCID: PMC11735953.
18. Mechanick JI, Butsch WS, Christensen SM, Hamdy O, Li Z, Prado CM, Heymsfield SB. Strategies for minimizing muscle loss during use of incretin-mimetic drugs for treatment of obesity. Obes Rev. 2025 Jan;26(1):e13841. doi: 10.1111/obr.13841. Epub 2024 Sep 19. PMID: 39295512; PMCID: PMC11611443.
19. Murdock DJ, Wu N, Grimsby JS, Calle RA, Donahue S, Glass DJ, Sleeman MW, Sanchez RJ. The prevalence of low muscle mass associated with obesity in the USA. Skelet Muscle. 2022 Dec 21;12(1):26. doi: 10.1186/s13395-022-00309-5. PMID: 36539856; PMCID: PMC9769063.
20. Jurdana M, Žiberna L. Sarcopenic obesity and hypertension in elderly patients: a narrative review of pathophysiology and management strategies. Ann Ist Super Sanita. 2023 Jul-Sep;59(3):231-239. doi: 10.4415/ANN_23_03_10. PMID: 37712242.