SARCOPENIC OBESITY IN POSTMENOPAUSAL WOMEN: ASSOCIATION WITH INSULIN RESISTANCE AND COMPONENTS OF METABOLIC SYNDROME

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Abstract

Background:

The menopausal transition is accompanied by substantial endocrine and metabolic alterations that influence body composition and cardiometabolic health. Declining estrogen concentrations are associated with increased visceral adiposity, unfavorable changes in glucose and lipid metabolism, and progressive alterations in skeletal muscle mass and function. The coexistence of excess adiposity and impaired muscle mass and/or function, referred to as sarcopenic obesity, has emerged as an important clinical phenotype in aging women. Increasing evidence suggests that sarcopenic obesity may be closely associated with insulin resistance and the development of metabolic syndrome.

Objective:

To summarize current evidence concerning the pathophysiological relationship between sarcopenic obesity, insulin resistance, and components of metabolic syndrome in postmenopausal women, with particular emphasis on changes in body composition associated with estrogen deficiency.

Materials and Methods:

A narrative review of contemporary scientific literature was conducted. Publications addressing sarcopenic obesity, postmenopausal women, estrogen deficiency, visceral adiposity, insulin resistance, skeletal muscle dysfunction, and metabolic syndrome were analyzed. International consensus documents concerning the definition and diagnosis of sarcopenic obesity were also considered.

Results:

Current evidence indicates that postmenopausal women are exposed to several interacting mechanisms that may promote sarcopenic obesity. Estrogen deficiency may contribute to increased visceral fat accumulation, altered lipid metabolism, impaired insulin sensitivity, and changes in skeletal muscle metabolism. Aging, physical inactivity, inadequate protein intake, and reduced anabolic responsiveness may further accelerate the loss of muscle mass and strength.

The combination of increased visceral adiposity and reduced skeletal muscle mass may have important metabolic consequences. Skeletal muscle is a major site of glucose uptake and disposal; therefore, a reduction in functional muscle mass may impair glucose utilization and contribute to insulin resistance. At the same time, visceral adipose tissue is metabolically active and is associated with chronic low-grade inflammation and altered adipokine secretion. These processes may adversely affect skeletal muscle metabolism, creating a bidirectional relationship between adiposity, insulin resistance, and muscle deterioration.

Sarcopenic obesity may consequently be associated with several components of metabolic syndrome, including abdominal obesity, impaired glucose regulation, elevated blood pressure, and atherogenic dyslipidemia. However, the reported prevalence and clinical associations vary considerably

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among studies, partly because different diagnostic criteria and body composition assessment methods have been used.

Recent international consensus recommendations emphasize that sarcopenic obesity should not be diagnosed on the basis of body mass index alone. Assessment should incorporate measures of adiposity together with muscle mass and muscle function. Bioelectrical impedance analysis and dual-energy X-ray absorptiometry can be used to evaluate body composition, while handgrip strength and standardized physical performance tests provide information about muscle function. The 2025 Asia–Oceania consensus additionally emphasizes central obesity and proposes a stepwise approach incorporating screening, diagnosis, and intervention. (PubMed)

Conclusion:

Sarcopenic obesity represents an important and potentially underrecognized phenotype among postmenopausal women. The interaction between estrogen deficiency, visceral adiposity, insulin resistance, inflammation, and skeletal muscle loss may contribute to an unfavorable cardiometabolic profile. Comprehensive assessment of body composition, muscle function, and metabolic parameters may improve early recognition of women at increased risk. Further prospective studies in postmenopausal populations are needed to clarify the epidemiology, diagnostic thresholds, and longitudinal relationship between sarcopenic obesity, insulin resistance, and metabolic syndrome.

Keywords: postmenopausal women; sarcopenic obesity; sarcopenia; obesity; insulin resistance; metabolic syndrome; visceral adiposity; skeletal muscle; estrogen deficiency; cardiometabolic risk.

1. Introduction

Menopause represents an important biological transition associated with profound changes in endocrine regulation, body composition, and metabolic health. The decline in ovarian estrogen production during the menopausal transition is accompanied by changes in adipose tissue distribution, glucose homeostasis, lipid metabolism, and skeletal muscle physiology. These alterations occur against a background of progressive biological aging and are frequently accompanied by reductions in physical activity.

One of the most characteristic changes in postmenopausal women is a shift toward central and visceral fat accumulation. At the same time, age-related reductions in skeletal muscle mass and strength may become increasingly apparent. When excessive adiposity coexists with reduced skeletal muscle mass and/or impaired muscle function, the resulting phenotype is termed sarcopenic obesity.

Sarcopenic obesity has attracted increasing scientific attention because it combines two major adverse changes in body composition. The condition is not simply the simultaneous presence of obesity and sarcopenia; rather, adipose tissue dysfunction and impaired skeletal muscle function may interact through metabolic, inflammatory, hormonal, and behavioral mechanisms.

The ESPEN/EASO consensus statement published in 2022 highlighted the absence of a universally accepted definition and established a structured approach for screening and diagnosing sarcopenic obesity. (PubMed) More recently, a 2025 Asia–Oceania consensus proposed a three-step algorithm involving screening, diagnosis, and intervention, reflecting the continuing evolution of diagnostic standards. (PubMed)

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Postmenopausal women represent a particularly relevant population for studying this phenotype because menopause-associated hormonal changes occur simultaneously with age-related changes in muscle and adipose tissue. A 2024 review specifically addressing sarcopenic obesity in postmenopausal women emphasized the importance of early screening, prevention, and comprehensive management. (PubMed Central (PMC))

2. Pathophysiological Relationship Between Menopause and Sarcopenic Obesity

The pathogenesis of sarcopenic obesity is multifactorial. Estrogen deficiency, aging, physical inactivity, nutritional factors, insulin resistance, inflammation, and alterations in muscle protein metabolism may interact to produce progressive changes in body composition.

Estrogen has multiple metabolic effects beyond its reproductive functions. Reduced estrogen exposure during menopause is associated with changes in adipose tissue distribution and increased accumulation of visceral fat. Visceral adipose tissue differs metabolically from subcutaneous adipose tissue and is closely associated with insulin resistance and an adverse cardiometabolic profile.

At the same time, skeletal muscle undergoes age-related changes characterized by reductions in muscle mass, strength, and functional capacity. Reduced physical activity may accelerate these changes. Inadequate protein intake and impaired anabolic responsiveness may further compromise the maintenance of muscle tissue.

The simultaneous increase in visceral adiposity and decline in muscle mass creates a metabolically unfavorable environment. Because skeletal muscle represents one of the principal tissues responsible for postprandial glucose disposal, loss of functional muscle tissue may reduce glucose utilization and contribute to insulin resistance.

Conversely, insulin resistance can adversely affect skeletal muscle metabolism. Hyperinsulinemia and altered intracellular signaling may interfere with normal muscle protein metabolism, while chronic low-grade inflammation associated with visceral adiposity may contribute to muscle catabolism.

Therefore, the relationship between sarcopenic obesity and insulin resistance may be bidirectional rather than representing a simple causal sequence.

3. Sarcopenic Obesity and Insulin Resistance

Insulin resistance is one of the central metabolic abnormalities potentially linking obesity and skeletal muscle dysfunction. In postmenopausal women, the accumulation of visceral adipose tissue may increase free fatty acid flux and alter adipokine secretion, contributing to impaired insulin signaling.

Skeletal muscle plays a central role in maintaining glucose homeostasis. A reduction in muscle mass, particularly when accompanied by reduced muscle quality and physical activity, may decrease glucose disposal capacity. Consequently, the combination of excess visceral adiposity and reduced functional muscle mass may increase the likelihood of insulin resistance.

Evidence from population-based research has demonstrated an association between sarcopenic obesity and insulin resistance. For example, an Italian cohort study applying the ESPEN/EASO diagnostic algorithm specifically examined insulin resistance as a metabolic risk factor associated with sarcopenic obesity. (PubMed)

This relationship is particularly relevant during postmenopause because hormonal changes, adipose tissue redistribution, and age-related muscle loss occur concurrently. However, the precise contribution of each factor remains difficult to determine, and longitudinal studies in postmenopausal populations remain necessary.

4. Relationship with Components of Metabolic Syndrome

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Metabolic syndrome represents a cluster of interrelated cardiometabolic abnormalities that generally includes central obesity, elevated blood pressure, impaired glucose regulation, and atherogenic dyslipidemia.

Sarcopenic obesity may contribute to several of these components simultaneously. Increased visceral adiposity is closely related to abdominal obesity and insulin resistance. Impaired skeletal muscle glucose disposal may contribute to abnormal glucose regulation. Changes in lipid metabolism associated with visceral adiposity and insulin resistance may promote elevated triglyceride concentrations and reduced HDL cholesterol.

Furthermore, the coexistence of reduced muscle function and obesity may reduce physical activity and exercise capacity. This can create a positive feedback loop in which decreased activity promotes additional fat accumulation and further loss of muscle function.

An umbrella review published in 2024 summarized evidence linking sarcopenic obesity with multiple adverse health outcomes and emphasized the clinical importance of this phenotype. (PubMed Central (PMC))

Nevertheless, interpretation of the existing evidence requires caution because studies have used different definitions of sarcopenia, obesity, and sarcopenic obesity. Consequently, reported prevalence rates and associations with metabolic outcomes are not directly comparable across all populations.

5. Diagnostic Considerations

An important limitation of using body mass index alone in postmenopausal women is its inability to distinguish fat mass from lean mass. A woman may have a BMI within or near the normal range while experiencing substantial changes in body composition.

Current consensus approaches therefore recommend a multidimensional assessment. The ESPEN/EASO framework incorporates the assessment of obesity, muscle strength, and muscle mass. (PubMed)

Anthropometric parameters such as waist circumference may provide information regarding central adiposity. Bioelectrical impedance analysis can be used to estimate body composition, while DXA provides a more detailed assessment of fat and lean mass distribution.

Muscle strength may be evaluated using handgrip dynamometry. Physical performance can be assessed using standardized tests such as gait speed, the Short Physical Performance Battery, or the five-times sit-to-stand test. The 2025 Asia–Oceania consensus incorporates these approaches into a structured diagnostic algorithm and places particular emphasis on central obesity. (PubMed)

For research purposes, body composition assessment should ideally be combined with metabolic measurements. Fasting glucose, fasting insulin, HOMA-IR, lipid profile, blood pressure, and waist circumference can provide complementary information regarding cardiometabolic status.

6. Clinical and Research Implications

Recognition of sarcopenic obesity in postmenopausal women has important implications for preventive medicine. Traditional approaches focused exclusively on weight reduction may be insufficient because excessive caloric restriction can potentially result in additional loss of lean tissue.

Management strategies should therefore aim to reduce excess adiposity while preserving or improving muscle mass and function. Resistance exercise is particularly relevant for maintaining muscle strength, while adequate dietary protein and overall nutritional quality may support muscle preservation.

The 2025 Asia–Oceania consensus emphasizes a combination of dietary intervention, increased physical activity, resistance training, and individualized management. (PubMed)

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From a research perspective, several questions remain unresolved. These include the optimal diagnostic thresholds for postmenopausal women, the prevalence of sarcopenic obesity in different populations, the contribution of menopausal stage and duration, and the longitudinal relationship between sarcopenic obesity and cardiometabolic events.

Population-specific research is particularly important because body composition, dietary patterns, physical activity, healthcare access, and genetic and environmental factors may influence the phenotype.

7. Conclusion

Sarcopenic obesity represents an emerging clinical and research challenge in postmenopausal women. The simultaneous accumulation of visceral adipose tissue and deterioration of skeletal muscle mass and function may create a metabolic environment characterized by insulin resistance, chronic low-grade inflammation, and an increased burden of metabolic syndrome components.

Estrogen deficiency during menopause may contribute to this process by promoting adverse changes in adipose tissue distribution and metabolic regulation, while aging and reduced physical activity may accelerate skeletal muscle loss. The resulting interaction between adipose tissue dysfunction and muscle impairment may contribute to a self-reinforcing cycle of metabolic deterioration.

Early identification of sarcopenic obesity requires more than conventional BMI assessment. A comprehensive approach incorporating adiposity, muscle mass, muscle strength, physical performance, and metabolic parameters may provide a more accurate characterization of cardiometabolic risk.

Further prospective studies specifically involving postmenopausal women are warranted to establish population-specific diagnostic thresholds, determine the prevalence and clinical significance of sarcopenic obesity, and clarify its longitudinal relationship with insulin resistance and metabolic syndrome.

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