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The Role of Sarcopenic Obesity in Hyperlipidemia and Its Impact on Metabolic Health

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ABSTRACT

Sarcopenic obesity, a condition characterized by the simultaneous presence of reduced muscle mass and increased adiposity, has emerged as a critical health concern in the context of metabolic disorders. This review examines the complex interplay between sarcopenic obesity and hyperlipidemia, focusing on the implications for metabolic health. The coexistence of diminished muscle mass and excess fat alters lipid metabolism, leading to elevated circulating lipid levels and exacerbating cardiovascular risks. Mechanisms contributing to this dysregulation include insulin resistance, chronic low-grade inflammation, and altered adipokine secretion, all of which are exacerbated in sarcopenic obesity. Moreover, the condition predisposes individuals to metabolic syndrome, type 2 diabetes, and cardiovascular diseases, highlighting the need for targeted therapeutic strategies. This article discusses the underlying pathophysiology, the role of lifestyle factors, and potential interventions aimed at mitigating the impact of sarcopenic obesity on hyperlipidemia and overall metabolic health.

Keywords: Sarcopenic obesity, hyperlipidemia, metabolic health, insulin resistance, inflammation, adipokines, cardiovascular disease, metabolic syndrome

INTRODUCTION

Sarcopenic obesity is an increasingly recognized condition that presents a dual burden of muscle loss (sarcopenia) and excessive fat accumulation (obesity) [1, 2]. While obesity alone is a well-established risk factor for hyperlipidemia and other metabolic disorders, the additional presence of sarcopenia complicates the metabolic landscape, exacerbating health risks [3]. Hyperlipidemia, characterized by elevated levels of lipids in the blood, is a key driver of cardiovascular diseases (CVD) and metabolic syndrome. In sarcopenic obesity, the dysregulation of lipid metabolism is particularly pronounced, leading to a higher prevalence of hyperlipidemia compared to obesity or sarcopenia alone [4, 5]. This review aims to explore the relationship between sarcopenic obesity and hyperlipidemia, highlighting its impact on metabolic health and discussing potential therapeutic strategies. Sarcopenic obesity is a condition characterized by the simultaneous occurrence of sarcopenia, an age-related decline in muscle mass, strength, and function, and obesity, an excessive fat accumulation [6]. This dual condition poses a significant challenge to public health, as it exacerbates individual risks associated with both conditions. Sarcopenia and obesity coexist and have interactive effects that worsen metabolic and functional health outcomes, especially in aging populations. Sarcopenic obesity is more detrimental than either condition alone, as the decrease in muscle mass impairs functional capacity, while the increase in adiposity worsens metabolic health, contributing to conditions like insulin resistance, systemic inflammation, and cardiovascular diseases [7]. The prevalence of sarcopenic obesity varies based on the population, diagnostic criteria, and methods used to assess muscle mass and fat. Estimates suggest that sarcopenic obesity affects anywhere between 5% and 20% of older adults, but these figures can rise depending on the specific population under study, such as those with chronic diseases, sedentary lifestyles, or metabolic disorders [8,9]. As global populations age, particularly in high-income countries, the prevalence of sarcopenic obesity is increasing. Sarcopenic obesity tends to be more common in women due to lower initial muscle mass and hormonal changes post-menopause, which accelerate muscle loss and fat gain. Risk factors include sedentary behavior, poor nutrition, chronic diseases, and inflammatory conditions that promote muscle wasting and fat accumulation [10, 11]. The interaction between muscle loss

and fat gain creates a vicious cycle: reduced muscle mass leads to a lower basal metabolic rate (BMR), making it easier for excess calories to be stored as fat, worsening obesity. Excess adiposity contributes to chronic low-grade inflammation, known as "metaflammation," and worsens insulin resistance, exacerbated by sarcopenia and obesity. Sarcopenic obesity represents a complex intersection of aging, muscle loss, and fat gain that requires a multifaceted approach for prevention and treatment. Public health strategies aimed at promoting physical activity, improving nutritional intake, and addressing underlying metabolic conditions could help mitigate the growing burden of this condition in aging populations [12, 13].

Pathophysiology of Sarcopenic Obesity and Hyperlipidemia

The metabolic disturbances observed in sarcopenic obesity are driven by multiple interconnected mechanisms that impact lipid metabolism, including [3, 14–16].

Insulin Resistance: Insulin resistance is a hallmark of both sarcopenia and obesity and plays a central role in the development of hyperlipidemia. In sarcopenic obesity, muscle loss diminishes glucose uptake, while excess adipose tissue contributes to insulin resistance through the secretion of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF- α) and interleukin-6 (IL-6). Insulin resistance impairs the regulation of lipoprotein lipase (LPL) activity, resulting in elevated triglycerides and low high-density lipoprotein cholesterol (HDL-C).

Chronic Low-Grade Inflammation: Obesity is associated with chronic low-grade inflammation, characterized by elevated circulating levels of inflammatory markers. In sarcopenic obesity, this inflammatory state is further exacerbated due to muscle atrophy. Inflammation promotes dyslipidemia by influencing the expression of enzymes involved in lipid metabolism and by altering the lipid composition of lipoproteins, leading to an atherogenic lipid profile.

Adipokine Imbalance: Adipokines, such as leptin and adiponectin, play crucial roles in the regulation of lipid metabolism. In sarcopenic obesity, adipokine levels are dysregulated, with increased leptin and decreased adiponectin levels. Leptin resistance is common in obesity, and in the context of sarcopenia, this resistance worsens, contributing to hyperlipidemia. Adiponectin, which has anti-inflammatory and lipid-lowering effects, is typically reduced in sarcopenic obesity, further aggravating metabolic disturbances.

Lipid Metabolism Dysregulation: Sarcopenic obesity disrupts the normal metabolism of lipids, leading to an imbalance in lipoprotein synthesis and clearance. The reduced muscle mass decreases fatty acid oxidation, while adipose tissue expansion increases free fatty acid release into circulation. This results in an accumulation of triglycerides and low-density lipoprotein cholesterol (LDL-C), key markers of hyperlipidemia.

Impact on Metabolic Health: Sarcopenic obesity has profound effects on metabolic health, particularly by increasing the risk of developing metabolic syndrome, type 2 diabetes, and cardiovascular diseases. The presence of hyperlipidemia in sarcopenic obesity further exacerbates these risks:

Metabolic Syndrome and Type 2 Diabetes: Sarcopenic obesity significantly increases the likelihood of metabolic syndrome, a cluster of conditions including hyperlipidemia, hypertension, insulin resistance, and abdominal obesity. The synergistic effect of reduced muscle mass and increased fat mass creates a pro-diabetic environment, heightening the risk of developing type 2 diabetes [4, 17].

Cardiovascular Diseases: Hyperlipidemia is a well-established risk factor for cardiovascular diseases, and in sarcopenic obesity, this risk is amplified. The combination of dyslipidemia, chronic inflammation, and insulin resistance leads to accelerated atherosclerosis, increasing the incidence of coronary artery disease, stroke, and heart failure.

Therapeutic Strategies: Managing sarcopenic obesity and its associated hyperlipidemia requires a multifaceted approach that addresses both muscle loss and fat accumulation. Potential therapeutic strategies include:

Exercise Interventions: Resistance training and aerobic exercise are essential components of managing sarcopenic obesity. Resistance training helps to preserve and enhance muscle mass, while aerobic exercise promotes fat loss and improves lipid profiles. Exercise also improves insulin sensitivity and reduces inflammation, mitigating the risk of hyperlipidemia.

Dietary Interventions: Nutritional strategies targeting both muscle preservation and fat reduction are crucial in treating sarcopenic obesity. A high-protein diet, combined with caloric restriction, can support muscle maintenance while promoting fat loss. Additionally, dietary interventions that include omega-3 fatty acids, fiber, and antioxidants can improve lipid metabolism and reduce inflammation.

Pharmacological Approaches: Pharmacotherapy may be considered in individuals with severe hyperlipidemia or those who do not respond to lifestyle modifications. Statins, fibrates, and other lipid-

lowering agents can be used to manage dyslipidemia. However, the potential impact of these drugs on muscle mass must be considered, as some lipid-lowering agents have been associated with muscle-related side effects.

CONCLUSION

Sarcopenic obesity is a complex condition that significantly impacts lipid metabolism and metabolic health. The interaction between muscle loss and fat accumulation creates a pro-inflammatory, insulin-resistant environment that promotes hyperlipidemia and increases the risk of metabolic syndrome, type 2 diabetes, and cardiovascular diseases. Addressing this condition requires a comprehensive approach that combines exercise, dietary modifications, and pharmacological interventions. Further research is needed to develop targeted therapies that can effectively manage sarcopenic obesity and its metabolic complication.

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