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Review

Impact of Obesity on Non-Alcoholic Fatty Liver Disease: A Brief Review

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ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is a chronic condition characterized by fat accumulation in the liver in individuals without a history of excessive alcohol consumption. Obesity plays a crucial role in the development and progression of NAFLD, being considered one of the main metabolic risk factors associated with the disease. This article explores the impact of obesity on NAFLD, highlighting the underlying pathophysiological mechanisms, such as insulin resistance, chronic low-grade inflammation and mitochondrial dysfunction. Additionally, clinical implications and management strategies aimed at preventing and treating the disease are discussed. The conclusion reinforces the need for integrated approaches to control obesity and mitigate the impacts of NAFLD on global health.

Keywords: Non-alcoholic fatty liver disease; Obesity; Liver; Alcohol

Introduction

Obesity is a global epidemic affecting millions of people and is associated with various metabolic and cardiovascular complications[1,2]. Among these complications, non-alcoholic fatty liver disease (NAFLD) stands out, a condition characterized by excessive fat accumulation in the liver without significant alcohol consumption. The global prevalence of NAFLD is estimated to be increasing, following the rise in obesity and metabolic syndrome rates[3]. This condition can progress to more severe forms, such as non-alcoholic steatohepatitis, liver cirrhosis. The relationship between obesity and NAFLD is well documented in scientific literature, with studies indicating that more than 75% of obese individuals present some degree of hepatic steatosis[4,5]. Obesity, particularly visceral obesity, plays a central role in the metabolic dysfunction underlying NAFLD. Factors such as insulin resistance, chronic low-grade inflammation and mitochondrial dysfunction are frequently observed in obese individuals and are closely related to the development and progression of NAFLD. Epidemiological studies indicate that NAFLD affects approximately 25% of the global population, with regional variations[6]. In Western

countries, prevalence is even higher, with estimates suggesting that up to 30% of the population may be affected. Among obese individuals, this prevalence is significantly higher, reaching up to 90% in patients with morbid obesity. Additionally, NAFLD is currently the leading cause of chronic liver disease in children and adolescents, reflecting the rising obesity rates in this age group[7]. In the context of obesity, excessive adipose tissue acts as a dysfunctional endocrine organ, releasing pro-inflammatory cytokines, free fatty acids and other metabolic mediators that promote liver damage. Furthermore, obesity alters lipid metabolism and energy homeostasis, favoring fat deposition in the liver and subsequent fibrosis development. Understanding the relationship between obesity and NAFLD is crucial because both conditions are highly prevalent and often coexist, significantly impacting public health. Early identification and effective obesity management can potentially reduce NAFLD progression and its associated complications[8].

Objectives

This article aims to review the available scientific evidence on the impact of obesity on NAFLD, exploring the main pathophysiological mechanisms involved, clinical implications and management strategies.

Materials and Methods

A bibliographic review of articles published in the PUBMED, ScienceDirect and Scielo databases was conducted to support the study.

Discussion

Obesity, especially visceral obesity, is one of the main determinants of non-alcoholic fatty liver disease (NAFLD). The relationship between these conditions is mediated by a series of complex pathophysiological mechanisms involving insulin resistance, chronic low-grade inflammation, lipid metabolism alterations and mitochondrial dysfunction[9]. One of the main mechanisms involved in this condition is insulin resistance. Obese individuals often present compensatory hyperinsulinemia, which contributes to increased hepatic lipogenesis and reduced fatty acid oxidation. These processes result in triglyceride accumulation in the liver, characterizing hepatic steatosis. Additionally, insulin resistance negatively affects lipoprotein secretion, further exacerbating hepatic fat deposition[10].

Another crucial aspect is chronic low-grade inflammation, characteristic of obesity. Hypertrophied adipose tissue releases pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which contribute to the activation of inflammatory cells in the liver, promoting NAFLD progression to more severe forms, such as non-alcoholic steatohepatitis[11]. Inflammation is also associated with increased oxidative stress, which damages cellular structures and worsens liver injury. Moreover, mitochondrial dysfunction plays an important role in NAFLD pathogenesis. Studies show that obesity is associated with reduced mitochondrial fatty acid oxidation capacity, leading to the accumulation of toxic metabolites and increased oxidative stress. This dysfunction contributes to the activation of apoptotic pathways and the progression of hepatic fibrosis[12,13].

Obesity complicates NAFLD management, as excess weight aggravates insulin resistance and exacerbates inflammatory processes. Management strategies include lifestyle interventions, such as diet and physical exercise, aimed at weight loss and improved insulin sensitivity[14]. Pharmacological treatments, such as GLP-1 agonists, have shown promising results in reducing hepatic fat and improving inflammatory markers. However, challenges persist, particularly in early diagnosis and personalized treatment of NAFLD in obese individuals[15]. The need for integrated approaches that consider the metabolic complexity of these patients is crucial for improving clinical outcomes.

Conclusion

It can be concluded that obesity has a significant impact on the development and progression of non-alcoholic fatty liver disease (NAFLD), mediated by complex pathophysiological mechanisms that include insulin resistance, chronic inflammation and mitochondrial dysfunction. This interaction exacerbates associated metabolic risks and complicates clinical management. Lifestyle interventions remain the cornerstone of treatment, highlighting the importance of weight loss and physical activity. However, pharmacological approaches have emerged as promising complementary strategies.

Despite advances, there are still gaps in early diagnosis and personalized treatment, particularly in patients with severe obesity. Given the high prevalence of obesity and NAFLD in the global population, investing in public health policies that promote prevention and integrated management of these conditions is essential. Strategies combining nutritional education, weight control programs and access to medical interventions can mitigate NAFLD impacts and improve patients' quality of life. Future research should focus on elucidating the underlying mechanisms of the obesity-NAFLD relationship and developing new targeted therapies. The integration of different therapeutic approaches is essential to address the challenges posed by these metabolic conditions, ensuring better public health outcomes.

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