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Why Obesity Risk Factor for Atherosclerosis Disease?

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Abstract

Obesity is a multifactorial chronic disease characterized by an accumulation of visceral and subcutaneous fat, which leads to a predisposition toward cardiometabolic diseases. A plethora of mechanisms, including abnormalities in lipid metabolism, insulin resistance, inflammation, endothelial dysfunction, and inflammasome activation have been suggested to underlie the relationship between obesity and atherosclerosis.

Keywords: Obesity; Cardiometabolic disease; Cholesterol; Body mass index

Introduction

Obesity has been an serious health problem of growing significance all over the world. Its prevalence is increasing in both developed and developing countries [1]. According to WHO data, 39% of the worlds population above 18 years of age are overweight and of these, 13% are obese. A number of studies have demonstrated a strong relationship between obesity and cardiovascular diseases (stable coronary disease, acute myocardial infarction, heart failure, cardiac arrhythmias, and sudden cardiac death). The association between obesity and hypertension, diabetes mellitus, dyslipidaemias, and sleep apnoea syndrome has also been shown to increase the incidence of cardiovascular disorders [2].

Body Mass Index (BMI) is used for measuring the extent of obesity. However, it gives no information on fat distribution, which is of high significance in cardiovascular risk. Therefore, novel clinical measurements (e.g., abdominal circumference and the calculation of waist/hip ratio) have been introduced with the aim of characterizing central or abdominal obesity. Abdominal circumference above 102 cm in the case of men and above 88 cm in the case of women qualifies as central obesity and involves increased cardiovascular risk. A waist/hip ratio above 0.9 in the case of men and above 0.85 in the case of women indicates central obesity [3].

The Relationship between Obesity and Atherosclerosis

In the past decades, many details of the pathophysiological processes of obesity and atherosclerosis have been revealed so far. Previously, both diseases had been regarded as lipid storage disorders with triglyceride accumulation in the fat tissue and cholesterol esters in atherosclerotic plaques. Nowadays, both obesity and atherosclerosis are considered chronic inflammatory conditions, in which the activation of both nonspecific and adaptive immune processes is assigned a significant role [4].

The pathogenesis of obesity and atherosclerosis has several common factors. In both cases, lipids, oxidized LDL particles, and free fatty acids activate the inflammatory process and trigger the disease. Inflammation is responsible for all the steps towards atherosclerosis, from early endothelial dysfunction to the atherosclerotic plaques causing complications, and is related to obesity, insulin resistance, and type 2 diabetes. The fatty tissue releases adipocytokines, which induce insulin resistance, endothelial dysfunction, hypercoagulability, and systemic inflammation, thereby facilitating the atherosclerotic process. In visceral obesity, inflammatory adipocytokines (e.g., TNF-α, IL-6, MCP-1, leptin, and resistin) rise to higher levels. Moreover, the increased level of C-reactive protein is associated with an increased risk of myocardial infarction, peripheral vascular disease, and diabetes mellitus. Interestingly, a clinical study performed on obese women confirmed that body weight reduction achieved through lifestyle changes reduces the level of inflammatory biomarkers and insulin resistance. In the course of the process, adiponectin, an anti-inflammatory and insulin-sensitizing adipocytokine, is released . It is important to understand the relationship between the inflammatory process and atherosclerosis and the accelerating role of obesity [5,6].

Excess weight and obesity are associated with an increased risk of cardiovascular diseases. This is a consequence on the one hand of obesity itself and on the other hand of associated medical conditions (hypertension, diabetes, insulin resistance, and sleep apnoea syndrome). In case of already established cardiovascular diseases, the mortality of overweight and obese patients is often lower than that of people with a normal body weight, which is known as "obesity paradox." The exact mechanism of the latter is not clear yet. Considering the increased cardiovascular risk, the regular cardiology screening, and control of still symptom-free obese patients is important for the early diagnosis and treatment of subclinical medical conditions.

Conclusion

The processes that lead to CVD in obesity are multiple, complex and only partly understood, although the evidence base is ever increasing.

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