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Editorial on Obesity and Endothelial Function

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Editorial

Obesity is a major public health issue that is associated with rising rates of cardiovascular morbidity and mortality. Overweight or obese adults account for over 1.9 billion adults worldwide, and the prevalence of obesity is rising. Endothelial function is influenced by obesity-related complications such as hypertension, dyslipidemia, diabetes, metabolic syndrome, and obstructive sleep apnea syndrome. Obesity causes adipocyte dysfunction as well as oxidative stress, insulin resistance, and inflammation, which leads to endothelial dysfunction. Several anthropometric indices and imaging modalities used to assess obesity have found a link between obesity and endothelial function. For the prevention and treatment of cardiovascular events, there has been a great deal of emphasis in recent decades on the mechanisms underlying endothelial dysfunction caused by obesity.

Endothelial dysfunction is the first step in the pathogenesis and development of atherosclerosis. Obesity is known to cause endothelial dysfunction. As a result, much attention has been paid to the mechanisms underlying endothelial dysfunction caused by obesity in the prevention and treatment of cardiovascular events [1-3]. This review focuses on the pathophysiological mechanisms of obesity-induced endothelial dysfunction and potential obesity therapeutic targets for endothelial dysfunction prevention.

The vascular endothelium connects circulating blood to blood vessels. As a paracrine organ, the vascular endothelium detects shear stress caused by blood flow and secretes vasodilating (NO), prostacyclin, and endothelium-derived hyperpolarizing factor) and vaso constricting (endothelin-1, angiotensin II, and thromboxane A2) substances. Shear stress is required for the endothelium to regulate cellular signaling [4,5]. Among vasoactive agents, NO is important in maintaining vascular homeostasis, which includes endothelial function, blood pressure, and blood flow. The vascular endothelium regulates vasoconstriction and vasodilation, growth promotion and growth inhibition, pro-thrombosis and anti-thrombosis, pro-inflammation and anti-inflammation, and pro-oxidation and anti-oxidation to maintain vascular homeostasis.

Endothelial dysfunction is an early stage of atherosclerosis that increases the risk of cardiovascular events. Endothelial function is known to be impaired in patients with coronary risk factors. Endothelial dysfunction is an independent predictor of cardiovascular events, according to growing evidence. Inflammation, an imbalance between vasodilators and vasoconstrictors, endogenous endothelial NO synthase (eNOS) uncoupling, and low shear stress are all important mechanisms that lead to endothelial dysfunction. In a clinical setting, a significant relationship between BMI and endothelial function has been demonstrated. A high BMI has been linked to endothelial dysfunction in children, adolescents, healthy people, patients with suspected coronary artery disease, and patients with cardiovascular disease. A large cross-sectional study (n = 7682) was conducted to assess the relationship between obesity and endothelial function. According to the findings, BMI is a reliable predictor of endothelial dysfunction. Indeed, a link has been found between the risk of endothelial dysfunction and obesity and overweight in younger adults (60 years of age).

BMI calculation is a simple method for assessing obesity. BMI, on the other hand, does not accurately predict body fat distribution. It has been demonstrated that abdominal obesity is more strongly associated with cardiovascular events than overall obesity. As a result, assessing abdominal obesity in addition to BMI is recommended to assess cardiovascular risk. Indeed, a number of studies have looked at the relationships between abdominal obesity indices and endothelial function. Waist circumference, waist-to-hip ratio, waist-to-height ratio, body adiposity index, and a body shape index (ABSI) have all been linked to endothelial function. We recently reported that ABSI is inversely related to FMD and that high ABSI is an independent predictor of endothelial dysfunction.

Advances in imaging techniques such as Computed Tomography (CT) and magnetic resonance imaging have resulted in a better understanding of the relationship between body fat distribution and cardiovascular risk. It has been demonstrated that the regional distribution of body fat has a greater influence on cardiometabolic risk than fat mass itself. There are two types of adipose tissue in the abdomen: visceral adipose tissue and subcutaneous adipose tissue. According to accumulating evidence, visceral adiposity is linked to chronic inflammation, insulin resistance, and cardiovascular disease, whereas subcutaneous adiposity may be protective in this context. Endothelial dysfunction has been linked to an increase in visceral adipose tissue volume. However, it was discovered that the volume of subcutaneous adipose tissue was not related to Ectopic fat accumulation, such as fat accumulation in the liver, heart, and blood vessels, is frequently associated with excess visceral adiposity. Endothelial function has been demonstrated to be impaired in patients with non-alcoholic fatty liver disease, patients with increased epicardial adipose tissue, and subjects with increased perivascular adipose tissue. Ectopic fat deposition has been linked to insulin resistance and an increased risk of cardiovascular complications. The liver is the primary organ of ectopic fat accumulation, and fatty liver is thought to play an important role in the pathogenesis of cardiovascular disease complications. However, it is unknown whether fatty liver is associated with cardiovascular events on its own.

Conflict of Interest

The author declares that there is no conflict of interest associated with this paper.

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