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Obesity and Relative Significant Risk Factor for Atherosclerosis

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Introduction

Obesity is a significant risk factor for atherosclerosis, although the mechanism behind this link is unclear. Adipose tissue was once thought to be only a storage facility for excess energy, but it is now recognised as a fully functional endocrine organ. Various adipokines, such as leptin (a protein produced by fat cells), tumour necrosis factor- (TNF-), resistin, and adiponectin, govern fat metabolism, energy balance, and insulin sensitivity, and hence influence obesity-related metabolic diseases. Because of their influence on the activity of endothelial cells, arterial smooth muscle cells, and macrophages in vessel walls, several adipokines have lately been viewed as direct connections between obesity and atherosclerosis, regardless of their effects on glucose and fat metabolism. The discovery of a novel adipokine that controls the atherosclerotic process might open up new avenues for creating more effective cardiovascular disease prevention strategies [1]. Adipokines that mediate obesity and atherosclerosis, such as adiponectin, resistin, adipocyte fatty acid binding protein (A-FABP), omentin-1, and chemerin, will be discussed in this study.

Mechanisms of the relationship amongobesity, inflammation, and CVD

Systemic inflammation:

As people gain weight and their adipocytes get larger, the adipose tissue undergoes molecular and cellular changes that impact systemic metabolism. To begin, macrophages build up in fat tissue, causing local inflammation. As obesity rises, adipose tissue produces a number of proinflammatory mediators. Obese people's adipose tissue expresses more proinflammatory proteins, such as TNF- and IL-6, than lean people's adipose tissue. Obesity increases the amount of macrophages in adipose tissue, which appear to serve as scavengers of apoptotic adipocytes. Obese people have also been shown to have a significant increase in these scavengers [2, 3]. Numerous metabolic dysfunctions associated with obesity, such as systemic inflammation and atherosclerosis, are thought to be caused by macrophage accumulation and consequent local inflammation.

Endothelial dysfunction:

Endothelial dysfunction is linked to systemic inflammation, according to clinical and experimental evidence. Disrupted endothelium function may be an early indication of a continuing atherosclerosis process, according to mounting data. As a result, endothelial dysfunction is becoming more well recognised as a factor in a variety of diseases linked to a high prevalence of atherosclerotic CVD [4]. Inflammatory cytokines play a key role in the development of atherosclerotic plaques, causing consequences all across the atherosclerotic vessel. Importantly, independent of risk factors (e.g., diabetes, hypertension, obesity), the formation of atherosclerotic lesions is defined by the disturbance of endothelial cell function.

Coronary endothelial dysfunction has a variety of causes, including ischemia/reperfusion damage. Atherogenic risk factors include smoking, obesity, hypertension, diabetes, physical inactivity, and hypercholesterolemia. Endothelial dysfunction is thought to be a precursor to atherosclerosis, a chronic inflammatory disease [5]. Chronic inflammation is a key contributor to atherosclerosis, and individuals with established atherosclerotic disease have higher levels of inflammation, fibrinolysis, and coagulation markers.

Endothelial cells are critical for vascular homeostasis, and they generate a range of mediators, surface proteins, and autacoids that are involved in vasomotion, coagulation, and inflammation. The angiotensin system (RAS) (renin, angiotensin-converting enzyme (ACE)) as well as the nonrenin-angiotensin system (NRAS) (cathepsin D, cathepsin G, tonin, chymase) are both expressed in adipose tissue. The discovery of increased CRP as a temporary independent risk factor for endothelial dysfunction might be a crucial step toward connecting a systemic inflammatory marker to the development of atherosclerosis. As a result, CRP has been recommended as a tool for assessing CVD risk in the general population. Evidence shows that low-grade inflammation is associated with reduced endogenous NO bioavailability, and that TNF- may play a role in these processes. Other vasoactive factors found in adipose tissue include leptin, serum amyloid A (SAA), and apelin, among others . Because most adipocyte-derived substances have receptors in blood arteries, adipose tissue appears to play a significant role in cardiovascular physiology via a network of local and systemic signals [6].

Subclinical atherosclerosis and CVD

Obesity causes a cascade of interconnected proatherogenic processes that lead to atherosclerosis. A greater BMI is linked to subclinical inflammation, as seen by elevated CRP levels and increased systemic oxidative stress, which is independent of blood glucose and diabetes. Recent research suggests that leptin increases macrophage cholesterol absorption, especially in the presence of high hyperglycemia. This leads to the production of foam cells and the establishment of atheromatic lesions. Hypoadiponectinemia caused by obesity may potentially play a role in endothelial dysfunction, increased vascular ROS generation, and overall proatherogenic consequences. Finally, increased adipose tissue production of proinflammatory cytokines such as IL-6, IL-1, and TNF- increases pro-atherogenic gene expression and maintains vascular wall inflammation [7].

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