Significant role of obesity in inflammation and cardiovascular diseases.

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Introduction

Although the mechanism behind this association is unknown, obesity is a substantial risk factor for atherosclerosis. Adipose tissue was once assumed to be little more than a storage space for extra energy, but it is now recognised as a fully working endocrine organ. Adipokines regulate fat metabolism, energy balance, and insulin sensitivity, and so influence obesity-related metabolic illnesses [1]. Several adipokines have recently been recognised as direct linkages between obesity and atherosclerosis, regardless of their effects on glucose and fat metabolism, due to their influence on the activity of endothelial cells, arterial smooth muscle cells, and macrophages in vessel walls.

The identification of a novel adipokine that regulates the atherosclerotic process could pave the way for more effective cardiovascular disease prevention techniques. This study will look at adipokines such adiponectin, resistin, Adipocyte Fatty Acid Binding Protein (A-FABP), omentin-1, and chemerin, which play a role in obesity and atherosclerosis.

Systemic inflammation

Adipose tissue undergoes molecular and cellular changes as people gain weight and their adipocytes become larger, affecting systemic metabolism. To begin, macrophages accumulate in adipose tissue, creating inflammation in the area. Adipose tissue produces a number of proinflammatory mediators when obesity increases [2]. The adipose tissue of obese persons expresses more proinflammatory proteins, such as TNF- and IL-6, than that of lean people. Obesity boosts the number of macrophages in adipose tissue, which appear to scavenge apoptotic adipocytes. Obese people have been demonstrated to have an increased number of these scavengers. Macrophage increase and subsequent local inflammation are assumed to be the source of several metabolic dysfunctions associated with obesity, such as systemic inflammation and atherosclerosis.

Endothelial dysfunction

According to clinical and experimental evidence, endothelial dysfunction is connected to systemic inflammation. According to growing evidence, impaired endothelium function could be a precursor to the progression of atherosclerosis. As a result, endothelial dysfunction is increasingly being recognised as a factor in a wide range of disorders associated with a high prevalence of atherosclerotic CVD [3]. Inflammatory cytokines have a critical role in the formation of atherosclerotic plaques, with repercussions that can be felt throughout the atherosclerotic artery. Importantly, regardless of risk factors (such as diabetes, hypertension, or obesity), endothelial cell dysfunction is what causes atherosclerotic lesions to form.

Ischemia/reperfusion injury is one of the many causes of coronary endothelial dysfunction. Smoking, obesity, hypertension, diabetes, physical inactivity, and hypercholesterolemia are all atherogenic risk factors. Atherosclerosis, a chronic inflammatory disease, is thought to be preceded by endothelial dysfunction. Chronic inflammation is a major factor in the development of atherosclerosis, and those with the condition have greater levels of inflammation, fibrinolysis, and coagulation indicators [4].

Endothelial cells are important for vascular homeostasis because they produce a variety of mediators, surface proteins, and autacoids that help in vasomotion, coagulation, and inflammation. Adipose tissue expresses both the angiotensin system (RAS) (renin, Angiotensin-Converting Enzyme (ACE)) and the Nonrenin-Angiotensin System (NRAS) (cathepsin D, cathepsin G, tonin, chymase). The identification of elevated CRP as a transitory independent risk factor for endothelial dysfunction could be a critical step toward linking a systemic inflammatory marker to atherosclerosis progression.

CRP is now recommended as a measure for determining CVD risk in the general population. Low-grade inflammation is linked to decreased endogenous NO bioavailability, and TNF- may play a role in these processes. Leptin, Serum Amyloid A (SAA), and apelin are among the additional vasoactive hormones identified in adipose tissue. Adipose tissue appears to have a substantial role in cardiovascular physiology via a network of local and systemic signals because most adipocyte-derived chemicals have receptors in blood vessels.

Subclinical atherosclerosis and CVD

Obesity triggers a series of interrelated proatherogenic mechanisms that eventually result in atherosclerosis. A higher BMI is associated with subclinical inflammation, as seen by increased CRP levels and systemic oxidative stress, which is independent of blood glucose and diabetes. According to new research, leptin boosts macrophage cholesterol absorption, especially when hyperglycemia is high. This causes the formation of foam cells and atheromatic lesions. Obesity-induced hypoadiponectinemia may contribute to endothelial dysfunction, increased vascular ROS production, and overall proatherogenic effects [5]. Finally, increased adipose tissue

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production of proinflammatory cytokines including IL-6, IL-1, and TNF- boosts pro-atherogenic gene expression and keeps vascular walls inflamed.

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