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Editorial

Metabolic Syndrome and Correlation of Adiponectin, ICAM-1 and VCAM-1

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Obesity, high blood pressure, inadequate glucose tolerance, and dyslipidemia are all components of the metabolic syndrome, which is a risk factor for cardiovascular disease (CVD) [1].

The malfunction of adipose tissue is associated with the prevalence of obesity accompanied by insulin resistance, hypertension, and cardiovascular disease because adipose tissue secretes a number of adipokines that regulate insulin sensitivity, energy metabolism, and vascular homeostasis [2]. Due to the enlargement of adipose tissue, obesity is believed to change the expression of adipokines, such as adiponectin, which has a strong anti-inflammatory and vascular protective effect [3]. Adiponectin is the most abundant adipose-specific adipokine. The heart, liver, pancreatic -cells, brain, bone, kidneys, blood vessels, immune cells, and many more tissues are all affected favorably by the adipokine [4]. Adiponectin provides protection the vasculature by acting pleiotropically on endothelial cells, endothelial progenitor cells, smooth muscle cells, and macrophages to increase insulin sensitivity and metabolic characteristics and inhibit the vascular dysfunction caused by obesity and diabetes [5,6]. Adhesion molecules in artery endothelial cells cause the buildup of monocytes/macrophages and T lymphocytes in atherosclerosis, an inflammatory disease [7]. Oxygen-derived free radicals (ROS) are one of the variables causing endothelial dysfunction, and adipocyte hypertrophy, one of the mechanisms underlying obesity, which is a low-grade inflammatory state, increases their levels [8]. Endoplasmic reticulum (ER) stress and mitochondrial malfunction are caused by this disorder [9].

Vascular Cell Adhesion Molecule-1 (VCAM-1) and Intercellular Adhesion Molecule-1 (ICAM-1) significantly increase in the aortic intima as a result of adiponectin insufficiency [10]. Adiponectin accumulates in the vasculature and is reduced in obesity owing to TNF-α suppression [5,6]. ICAM-1 and VCAM-1 levels have been found to be higher in atherosclerotic patients in several researches [11,12], and baseline ICAM-1 levels have been found to be strongly linked with incident myocardial infarction and carotid atherosclerosis [13,14]. Due to their roles in the onset of atherosclerosis and as early biomarkers of changes in the arterial wall, ICAM-1 and VCAM-1 are recognized as early signs of endothelial dysfunction [15]. Additionally, ICAM-1 and VCAM-1 are inflammatory markers that are connected to other inflammatory markers since they are both members of the immunoglobulin superfamily [16]. To put it succinctly, when atherosclerosis advances, rising levels of tumor necrosis factor (TNF)-α and IL-1 release cause activated monocytes and vascular smooth muscle cells to produce IL-6, which increases the synthesis of hepatic CRP [17]. ICAM-1 and VCAM-1 are produced by macrophages and endothelial cells in response to TNF- and IL-1 [18]. Endothelial dysfunction can be brought on by metabolic syndrome, smoking, and a lack of physical activity [19]. Lesions in the arterial wall can begin to occur gradually during childhood and progress into adolescence and adulthood [20].

In conclusion, metabolic syndrome may occur with an increase in systemic cytokines, but additional research is required to fully understand these correlations.

Disclosure Statement

The authors declare that there are no conflicts of interest.

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