Editorial

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Metabolic Syndrome and Fetuin-A: Framework of Situation

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ABSTRACT

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Introduction

Metabolic syndrome (MetS), often known as syndrome X, is a type of metabolic illness that is becoming more widespread as the population becomes increasingly fat [1]. Hypertension, central obesity, insulin resistance, and atherogenic dyslipidemia (poor high-density lipoprotein cholesterol, hypertriglyceridemia) are all closely linked to visceral adiposity [1,2]. Pathology in numerous target tissues, including as the cardiovascular system, pancreas, and liver, is relatively common in those who have MetS [3]. Insulin resistance (IR) is well considered as a factor in several metabolic disorders, including obesity, dyslipidemia, MetS, hypertension, and atherosclerosis, nonalcoholic fatty liver disease (NAFLD), type 2 diabetes mellitus (T2DM), and some cases of type 1 diabetes mellitus (T1DM). Infact, IR has been identified as a crucial main pathophysiological pathway in the onset of MetS, referring to a complex pathological deficiency in insulin signaling pathways that results in an inadequate cellular response to insulin hormone in insulin-dependent tissues such as skeletal muscle, adipose, and liver tissues [2,4,5].

Several cytokines and inflammatory mediators, such as tumor necrosis factor- (TNF-), monocyte chemotactic protein-1 (MCP-1), C-reactive protein (CRP), and interleukins, have been shown to be greatly increased in the course of IR. Endocrine adipocytes, however, are more likely to allow the release of inflammatory mediators including leptin, TNF-, and adiponectins, as well as plasminogen-activator inhibitor-1, angiotensinogen, and active steroid hormones, all of which have a role in the progression of IR [6]. Furthermore, several studies have suggested that oxidative stress, a common hallmark of obesity, is linked to the persistent low-grade inflammation that distinguishes MetS. Thus, systemic oxidative stress may play a role in the disordered production of adipokines that promote to the pathophysiology of MetS [7]. IR has also shown cellular dysfunctions caused by a variety of inflammatory mediators that may connect with one another, such as protein kinases and phosphatases. Decreased insulin-stimulated glucose transporter 4 (GLUT-4) expression or translocation is predicted to occur in altered glucose transport, reduced glycogen storage, and inhibited protein synthesis when the phosphorylated signaling pathway is disrupted [8].

The resulting hyperinsulinemia is therefore sufficient to induce serine/threonine phosphorylation of the insulin receptor substrate (IRS), which is necessary to stimulate IRS degradation and block the phosphorylation of tyrosine, which is a crucial step in proceeding the surviving phosphorylation cascades of downstream targets, in the PI3K/protein kinase B (Akt)/Forkhead box transcription factors of the class O (FOXO1) signaling pathways, possibly phosphatidylinositide 3-kinases (PI3K) or a class of small GTPases (RAS) are implicated. [9].

Fetuin-A (Fet-A) is a hepatokine and is also known as 2-Heremans-Schmid glycoprotein (AHSG) in humans. It relates to the protease inhibitors cystatin superfamily. Fet-A protein is involved in a number of physiological cellular functions, including cellular protein and fatty acid metabolism, acute inflammatory

response regulation, bone mineralization and calcified matrix metabolism, neutrophil degranulation, lymphocyte recruitment, thyroid hormones, and calcium ion homeostasis, to mention a few [10]. The glycoprotein, however, has lately been hypothesized as a molecular relationship between obesity, NAFLD, IR, and MetS, since it has been linked to insulin receptor activity suppression, which leads to a malfunction in insulin cascade pathways [11]. During the last several years, sophisticated techniques in biochemical, immunochemical, and molecular genetics have greatly enhanced our understanding of the Fet-A protein, leading to important conclusions about its utility as a biomarker in the diagnosis and treatment of human disorders [11].

Therefore, MetS is a serious and growing public health and clinical concern around the world, covering a variety of basic dysfunctional processes such insulin resistance, inflammation, hormonal changes, and physical activity [12]. Obesity - related lipolysis causes an excess of free fatty acids to be produced and released into the bloodstream, leading to an increase in chronic subclinical inflammation and exacerbation of pre-existing insulin resistance in numerous target tissues [13-15]. Non-alcoholic fatty liver disease (NAFLD), which is one of the most frequent hepatic expressions of the metabolic syndrome, is one of a group of chronic liver illnesses that includes anything from simple steatosis (NASH) to liver cirrhosis [16,17].

Organokines are well-known for their autocrine, paracrine, and endocrine effects on cellular metabolism. Recent studies have shown that liver tissue regulates glucose and lipid metabolism through the secretion of several hepatokines, including Fet-A protein, and thus performs a central role in the regulation and management of whole-body energy homeostasis, implying that the liver and its associated hepatokines are heavily responsible for the development of some metabolic diseases [18]. Cardiovascular disease (CVD), which includes disorders affecting the cardiovascular system such as coronary artery disease, angina, and heart failure, has become one of the most common causes of death globally; as a result, more people die from CVD each year than perhaps with any other reason. Several research has found that determining coronary calcification can help predict cardiovascular risk without relying on other traditional risk markers. Fet-A, as among the most prominent calcification inhibitors, has lately received more attention as an useful factor connected to cardiovascular mortality [11,19]. As a result, circulating fetuin-A levels are reported to be significantly higher in the development of obesity, metabolic syndrome, and type 2 diabetes, and are linked to hepatic steatosis and CVD in humans [11,20].

In conclusion, early atherosclerosis, MetS, obesity, IR, NAFLD, and low-grade adipose tissue inflammation may all have a positive correlation with fetuin-A. High levels of circulating fetuin-A have also been demonstrated to be a powerful predictor of incident T2DM, albeit the exact molecular pathways are yet unknown. There is evidence that fetuin-A could become a new molecular target in the elucidation of complicated pathophysiological pathways implicated in the start of metabolic illnesses, as well as a valuable marker in clinical practice for the early diagnosis of these conditions in the future.

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