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Adipose tissue is an energy storing organ with endo-


Serum Visfatin Levels Should Be Evaluated with Further Markers of Endothelial Inflammation

Dear Editor:

We read the article “Elevated Serum Visfatin Levels in Patients with Acute Myocardial Infarction” written by Mazaherioun et al. with interest. The authors aimed to assess any potential relationship between blood visfatin levels, anthropometric variables, and known risk factors of atherosclerosis and acute myocardial infarction (AMI). They determined that serum visfatin levels were significantly higher in AMI patients compared to controls. This finding was consistent with other studies that have shown a contribution of visfatin to atherosclerosis and plaque destabilization which, in turn, leads to myocardial infarction. We believe that these findings will act as a guide for further studies about risk factors of atherosclerosis and AMI.

Coronary artery disease is a major public health problem in Iran and one of the leading causes of death worldwide. Obesity is one of the most important risk factors for atherosclerosis of the coronary arteries, and consequently, increases the risk of myocardial infarction. Adipose tissue is an energy storing organ with endocrine properties which has a role in systemic vascular inflammation. Various pro- and anti-inflammatory mediators and cytokines are secreted from adipose tissue. Adiponectin, a peptide hormone and member of the family of adipokines, is absolutely expressed in and secreted by adipocytes and adipose tissue. It has also been shown that adipokines regulate different stages of atherosclerotic, from endothelial dysfunction to plaque destabilization and rupture. Visfatin is a novel adipokine with different functions, for which exist a plethora of research on its characteristics and roles. Visfatin levels may change in patients with inflammation such as diabetes mellitus, hypertension, obesity, metabolic syndrome, cardiovascular disease, hyperlipidemia, hormonal abnormality, known malignancy and smoking habit.

In addition, any abnormality in thyroid function tests, known malignancy, anemia, smoking and any medication such as statins can potentially interfere with inflammation and might change visfatin levels. Thus the authors should have excluded all of these factors in this study. Secondly, the selection of control group was not correctly defined in this study. For example, they have not explained whether or not the control group underwent coronary angiographies. Therefore some control group patients might have coronary artery disease. It would be better if the authors had defined these factors in the study group.

Finally, visfatin alone in the absence of other inflammatory markers may not give information to clinicians regarding the patient’s endothelial inflammatory condition. In our opinion we believe it should be evaluated together with other serum inflammatory markers.

There is no conflict of interest.

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