Med Princ Pract 2014;23:390
DOI: 10.1159/000358222

Pentraxin-3 as a Marker of both the Presence and the Severity of Metabolic Syndrome

Uğur Canpolat, Osman Turak, Derya Tok, Kumral Çağlı, Sinan Aydoğdu

Cardiology Clinic, Türkiye Yüksek İhtisas Training and Research Hospital, Ankara, Turkey

Dear Editor,

In a recent issue of *Medical Principles and Practice*, we were grateful to read an interesting article by Karakas et al. [1] investigating the relationship between the level of pentraxin-3 (PTX-3) and the severity of metabolic syndrome (MS) in a total of 203 subjects. They reported that PTX-3 levels were significantly higher in patients with 5 MS criteria compared to healthy controls and groups with 3 and 4 MS criteria, and that PTX-3 was well correlated with serum hs-CRP levels ($r = 0.49$, $p < 0.001$). We thank the authors for their structural and complementary contributions to the area of inflammation and its relationship with MS.

Inflammatory pathway activation and the secretion of various proinflammatory molecules like PTX-3 are important in the pathogenesis of several pathologic conditions including MS and heart failure (HF), and they are also associated with an adverse prognosis in these patients [2, 3]. Also, it has been shown that insulin resistance might link these two common phenomena [4]. It is known that inflammatory pathways are significantly involved in cardiac remodeling and the subsequent development of left ventricular diastolic dysfunction. In a recent study, Matsubara et al. [3] showed that the level of PTX-3, but not hs-CRP, TNF, and IL-6, in subjects without HF is significantly associated with the ratio of mitral E/E', a quantitative echocardiographic measure of left ventricular diastolic dysfunction. Also, they identified the presence of significantly elevated levels of PTX-3, but not hs-CRP, in HF with normal ejection fraction patients compared to non-HF

patients. It has been shown that some components of MS, such as hypertension and dysglycemia, are strongly associated with HF with normal ejection fraction, which leads to stiffening of the left ventricle, resulting in diastolic dysfunction [5]. So, due to the high rate of MS and HF with normal ejection fraction coprevalence, the authors should comment on the echocardiographic data of their study population which may have impacted the levels of PTX-3 concomitantly. Additionally, the authors excluded patients with apparent coronary artery disease from the study. However, because of the higher risk profile of MS patients for coronary artery disease, further noninvasive tests may be useful to exclude subclinical atherosclerosis in those patients.

Finally, as epicardial adipose tissue is a type of visceral adipose tissue that functions as a metabolically active endocrine organ and the association of increased epicardial adipose tissue with the severity of MS has been shown previously [2], higher levels of PTX-3 in patients with MS may be linked to increased epicardial adipose tissue in further studies.

References

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Editor's Note: The authors of the paper were asked to respond to the comment, but they chose not to do so.