

Letter to the Editor

Inflammatory status in patients with coronary artery disease

Dear Editor,

We intentionally read the article "Comparison of inflammatory biomarkers for detection of coronary stenosis in patients with stable coronary artery disease (CAD)" written by Uydu et al with great interest¹. They aimed to investigate lipoprotein-associated-phospholipase A2 (Lp-PLA2), high-sensitivity C-reactive protein (hs-CRP), and myeloperoxidase (MPO) levels in the detection of coronary stenosis in patients with CAD and healthy controls. They concluded that serum Lp-PLA2 levels are more compatible than hs-CRP and MPO levels in the detection of coronary stenosis. The study is successfully designed and presented. Thank to the authors for their contribution.

Coronary artery disease (CAD) is the most common cause of hospitalization and mortality in many industrialized countries². The pathogenesis of atherosclerosis is multifactorial; however, it is considered to be an inflammatory disease³. Increased inflammatory markers have been demonstrated to be associated with negative clinical outcomes in patients with heart failure, previous myocardial infarction, and stable coronary artery disease⁴. Elevated levels of inflammatory molecules are markers of atherosclerotic disease activity and also indicate an increased risk for the progression of atherosclerosis. So, there is a significant relationship between the endothelial dysfunction, inflammatory parameters and cardiovascular risk factors such as hypertension, diabetes mellitus, smoking and hypercholesterolemia⁵.

Obstructive sleep apnoea syndrome (OSAS) and non-alcoholic fatty liver disease (NAFLD) are common in clinical practice. Cardiovascular complications are common in patients with OSAS have been linked to morbidity and mortality in these patients based on endothelial dysfunction⁶. NAFLD refers to a wide picture of liver damage, ranging from steatosis to steatohepatitis, fibrosis and cirrhosis. Also, the presence and the degree of NAFLD are associated with higher inflammatory parameters in nonhypertensive, nondiabetic individuals, especially in those individuals without metabolic syndrome. Additionally, common pathways involved in the pathogenesis of NAFLD includes hepatic insulin resistance, subclinical inflammation, and atherosclerosis⁷. The inflammatory process was demonstrated in NAFLD patients is initiated at the endothelial dysfunction, inflammation and atherosclerosis, stressing the key role of this active. Another previous study had shown the presence of vascular diseases in patients with NAFLD based on this physiopathology⁸. Peripheral arterial disease (PAD) is also commonly observed with CAD⁹. In this point of view, because PAD, NAFLD and OSAS are associated with inflammatory status, if the authors had mentioned these factors, it is stronger.

Conclusions

Inflammatory status can be affected by the higher inflammatory status such as an inflammatory disease, cardiac syndrome X and infection¹⁰. In this point of view, in the present study, the authors did not mention some of these possible contributing factors. It would be better, if the authors gave information about these factors. These markers are described to assess inflammatory status and it can be affected by many factors. So, these markers themselves without other inflammatory markers may not provide information to clinicians about the CAD. For this reason, we think that it should be evaluated together with other serum inflammatory markers such as mean platelet volume¹¹, red cell distribution width¹², neutrophil lymphocyte ratio in routine clinical practice¹³. We believe that these findings will act as a guide for further studies that will assess inflammatory status as a surrogate marker of endothelial dysfunction and its relationship with CAD.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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