Author’s Reply

To the Editor,

We are grateful for the kind comments to our manuscript entitled “The association between platelet-lymphocyte ratio and coronary artery disease severity,” published in Anatol J Cardiol 2015; 15: 640-7 (1). In their Letter to the Editor, the authors emphasize the importance of the recently emerged inflammatory marker, platelet-to-lymphocyte ratio (PLR), in atherosclerotic pathogenesis and request for the comparable PLR values of the mild atherosclerosis and control groups in our study.

There may be a few reasons to find similar PLR values between the mild atherosclerosis and control groups. Firstly, as we mentioned, our study sample size is not large enough to statistically demonstrate the significance of such a minor difference in PLR values of these two groups. Secondly, atherosclerotic plaques develop and progress within the arterial wall long before to protrude into the lumen, which cannot be detected by conventional angiography (lumenography). Because of the nature of the atherosclerotic process, we might misdiagnose patients with positive remodelling as normal coronary arteries, which could only be diagnosed by intravascular ultrasound. Finally, although statistically insignificant, there were more smokers in the control group than in the mild atherosclerosis group, which might affect our results.

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The role of platelet-to-lymphocyte ratio in saphenous vein graft disease

To the Editor,

I read with great interest the article by Kundi et al. (1) entitled “Association between platelet-to-lymphocyte ratio and saphenous vein graft disease in patients with stable angina pectoris,” published online in Anatol J Cardiol 2015 May 5. In their study, authors reported that there was a significant association of platelet-to-lymphocyte ratio (PLR) with saphenous vein graft disease (SVGD) and suggested that PLR could be used as a marker of SVGD. I have the following comments and concerns.

Numerous clinical trials showed the relation between PLR and poor cardiovascular outcomes in cardiovascular disease. Because most of the stenosis and occlusion of saphenous vein grafts after the first year is caused by atherosclerosis, is there any difference in the meantime from coronary artery bypass grafting to the last coronary angiogram between the two groups? Also, the patency of bypass grafts on functionally significant lesions is higher than that on nonsignificant lesions (2). Yüksel et al. (3) reported that high PLR appears to be additive to conventional risk factors and commonly used biomarkers in predicting severe atherosclerosis. I was wondering if there was any difference between patients with or without SVGD in terms of severity of coronary artery disease.

Finally, obesity is a chronic inflammatory disease characterized by an increase in the levels of inflammatory cytokines (4). It has been shown that metabolic disorders such as obesity and insulin resistance are related to the progression of coronary atherosclerosis and increased incidence of cardiovascular events such as saphenous vein graft occlusion and acute coronary syndrome (5). Because PLR is a novel biomarker showing inflammation in cardiac and non-cardiac patients, authors should state the body mass index for each group.

In my opinion, the findings from this study could be related to the abovementioned limitations.

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References


