

between MPV and AHI and minimum O₂ saturation. Nena et al. (3) studied 610 non-diabetic subjects with suspected OSA. MPV (12.1 femtoliters) was significantly higher in patients with severe OSA defined by an AHI greater than 30 events per hour than in controls (9.8 femtoliters). They found significant correlations between MPV and AHI and between MPV and the percent of time the O₂ saturations were below 90%. This study suggested that there are significant correlations between MPV and important variables in patients with severe OSA. Varol et al. (4) studied 31 patients with severe OSA and measured MPV before and after treatment with CPAP for 6 months. The median MPV was significantly higher in patients with severe OSA than in control subjects, and there was a significant reduction in this volume after 6 months of CPAP therapy.

In our view, MPV is an easily available laboratory test that may identify patients with an increased risk for cardiovascular events and may represent a response parameter to monitor during treatment of these patients. It seems important to develop large prospective studies on its utility in patients with OSA.

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The role of platelet-lymphocyte ratio in the severity of coronary artery disease assessed by the angiographic Gensini score

To the Editor,

I am grateful to have read with great interest the article entitled "The association between platelet-lymphocyte ratio and coronary artery disease severity" by Yüksel et al. (1), published

in *Anatol J Cardiol* 2015; 15: 640-7. In this well-presented study, the authors aimed that the platelet-lymphocyte ratio (PLR) was associated with the severity of coronary artery disease, assessed by the Gensini score, because a high PLR was shown to be closely related with inflammation and atherosclerosis. They found that a high PLR was significantly and independently related with the severity of coronary artery disease.

Prior studies investigated that PLR, a combination of both platelet and lymphocyte counts, is a novel inflammatory marker and predictor of adverse cardiovascular outcomes (2-6). Yüksel et al. (1) showed that PLR was significantly higher in the group of severe atherosclerosis than in the other control and mild atherosclerosis groups. As known, the mild atherosclerosis group has a more severe inflammation than the control group; however, there was no difference between the mild atherosclerosis and control groups ($p=0.729$).

In conclusion, according to these results, it was not clear to highlight the pathogenesis role of PLR in the severity of coronary artery disease. According to me, further larger studies are needed to show and clarify this situation.

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