

Platelet to lymphocyte ratio: a novel and simple predictor of slow coronary flow

To the Editor,

We are grateful to have read with interest the article entitled "Relationship between platelet-to-lymphocyte ratio and coronary slow flow" by Oylumlu et al. published in *Anatol J Cardiol* 2015; 15: 391-5 (1). In this well-presented article, the authors hypothesized that the platelet-to-lymphocyte ratio (PLR) is associated with slow coronary flow (SCF) because an increased PLR was shown to be closely associated with inflammation and atherosclerosis. They demonstrated that PLR was significantly and independently associated with SCF. They suggested that increased PLR is an indicator of underlying inflammation in SCF.

Interventional cardiologists are familiar with the phenomenon of delayed opacification at the distal segments of the major epicardial coronary arteries in the absence of significant epicardial coronary artery stenosis, which is termed as SCF (2). The pathophysiological mechanisms underlying the SCF phenomenon have not been explicitly defined. Endothelial and microvascular dysfunction, inflammation, increased platelet activation, and atherosclerosis have been demonstrated to be closely associated with SCF (2, 3). As a combination of both platelet and lymphocyte counts, PLR recently emerged as a new potential inflammatory marker and predictor of major adverse outcomes in various cardiovascular diseases (4, 5). In the study by Oylumlu et al. (1), PLR was significantly higher in patients with SCF than in those in the control group (135.4±54.1 vs 113.4±31.1, p=0.001). However, other direct and indirect indicators of inflammation including white blood cell count, neutrophil count, neutrophil-to-lymphocyte ratio, and red cell distribution width were similar between the study groups. Additionally, the study lacks any data correlating the conventional biomarkers of systemic inflammation such as C-reactive protein (CRP) with PLR. According to all these findings, it was impossible to highlight the patho-

genetic role of PLR in SCF. In a recent study with a relatively large number of SCF patients (n=221), we reported that PLR, white blood cell, neutrophil, and platelet counts and serum CRP levels were significantly higher in the SCF group than those in the control group (5). Furthermore, PLR was also shown to be positively correlated with serum CRP levels confirmatory to its association with systemic inflammation. Therefore, we proposed that the relationship between PLR and SCF is because of the presence of an ongoing low-grade chronic inflammatory status. Chronic inflammation may cause an enhanced PLR, which would result in an increased risk for SCF.

In conclusion, these results suggest that besides its already known effect on prothrombotic status, a higher PLR level represents the impact of low grade chronic inflammatory state on coronary blood flow. As an easily available and cheap parameter of complete blood count, PLR can be calculated in clinical practice for the prediction of SCF. Further studies are needed to confirm our findings and define the pathophysiological role of PLR in SCF.

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