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LETTER TO THE EDITOR

Whether or not the association of MPV with coronary collateral development is independent of hs-CRP level?

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We have read the article reported by Ege et al. [1] with great interest. The authors reported higher mean platelet volume (MPV) levels in patients with inadequate coronary collateral development (CCD). The authors have emphasized that there is not a casual interest between MPV and CCD. According to the authors, higher MPV reflects activated inflammatory state which is associated with inadequate coronary collateral vessels.

High-sensitive C-reactive protein (hs-CRP) is a well-established marker of inflammation. In the literature, there are three studies which examine the association of hs-CRP with CCD [2–4]. In all these studies, elevated hs-CRP levels were found to be associated with poor CCD. It is well known that coronary collateral flow protects the myocardium against ischemia. So, the presence and adequacy of coronary collateral vessels is closely associated with better prognosis in patients with coronary artery disease [5]. Based on these aforementioned data, the authors of these studies claimed that one of the mechanisms responsible for bad cardiovascular outcomes in higher inflammatory state may be impairment in coronary collateralization. Furthermore, MPV has been shown to be associated with atherosclerosis, coronary events, and bad prognosis in numerous studies [6–9]. Similarly, based on the article of Ege et al. [1], it can be speculated that the association between higher MPV and bad prognosis may be partly due to poor collateral development. This hypothesis must be tested via further studies.

In addition, recently, some studies have suggested that MPV can be used as marker of inflammation and disease activity [10, 11]. Interestingly, it has been noted by Gasparyan et al. [11] that the intensity of systemic inflammation determines the size of circulating platelet size. In that, while high-grade inflammation leads to lower MPV levels, low-grade inflammation is associated with higher MPV levels. However, there is no study in the pertinent literature evaluating the simultaneous effect of both hs-CRP and MPV on CCD. Since Ege et al. hypothesize that higher MPV is related to poor collateral because of higher inflammatory state, hs-CRP may help clarify the relation between inflammation and MPV. If MPV is found independently related to CCD regardless of

hs-CRP levels, then, another hypothesis will be required to explain the results of Ege et al.'s study.

Ege et al. studied neither serum levels of hs-CRP nor the MPV–hs-CRP relation in the study population. We think that further studies are needed to clarify this issue.

We hope that the above-mentioned items would add to the value of the well-written manuscript of Ege et al., which reveals – for the first time in the literature – the association of higher MPV values with inadequate CCD.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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