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Mean Platelet Volume May Not Be Changed in Patients with Coronary Artery Ectasia

LETTER TO THE EDITOR

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Dear Editor,

We read the study of Sivri et al. (1) on increased mean platelet volume (MPV) in patients having undergone coronary artery ectasia, which was accepted for publication in your journal, with great interest. In this study, which was conducted with retrospective data, stable coronary artery group without acute coronary syndrome, acute coronary syndrome group, and coronary artery ectasia group without acute coronary syndrome were evaluated. The researchers suggested that MPV values were significantly higher in acute coronary syndrome and isolated coronary artery ectasia groups than in stable coronary artery disease group without acute coronary syndrome and that MPV values only increased in the acute coronary syndrome period in patients with coronary artery disease. On the basis of the elevated MPV value in coronary artery ectasia group without acute coronary syndrome, they defended that platelet activation was higher in these patients, and it was necessary to administer long-term antiplatelet treatment in patients with isolated coronary artery ectasia despite the absence of apparent occlusive stenosis. We would like to determine some points in this research paper.

Platelet function tests are used for revealing the presence of acquired or inherited platelet diseases in patients with a history of hemorrhage and when necessary, for determining the efficiency of treatment in patients receiving antiplatelet therapy. Platelet parameters, including MPV, are not involved among defined platelet function tests. Platelet aggregation, which is based on the principle of light transmission, is still the gold standard method used for determining platelet functions, although it was invented in the 1960s (2). Beyan et al. (3) compared platelet aggregation responses with platelet parameters using platelet aggregation working with the light transmission principle in healthy cases, and they reported that there was no consistency between all platelet parameters, including MPV and platelet aggregation responses. In another study recently published, De Luca et al. (4) examined the relationship between coronary artery disease and MPV in 1016 diabetic patients having undergone coronary angiography. The researchers used platelet aggregation working with the principle of light transmission, and they stated that no parallelism was available between MPV and platelet reactivity, and that no relationship was found between the elevated MPV value and presence of coronary artery disease and severe coronary artery disease.

In short, MPV is not an indicator of platelet function, and it appears right to defend, considering an increase in MPV values, that long-term antiplatelet treatment should not be performed in patients with coronary artery ectasia despite the absence of apparent occlusive stenosis.

Another important issue is that analysis-related standardization of MPV, which is suggested to be used as a diagnostic marker for cardiovascular diseases in some studies, has not yet been solved (5). In complete blood count, following the contact with ethylenediaminetetraacetic acid (EDTA) used as an anticoagulant, an increase in MPV values begins as a function of time. In general, after coming in contact with EDTA, MPV increases up to 30% within the first 5 min and then additionally by 10%–15% in the next 2 h (6). In studies, although it is accepted that the increase in MPV reaches the peak within the first 2 h following bloodletting, it has also been reported that it can last for 39 h. Many researchers expressed the change in MPV, which occurs in cases where EDTA is used as an anticoagulant, with great deviations ranging from 2% to 50%. Lance et al. (7) reported that when EDTA was used as an anticoagulant in complete blood count, timing of MPV measurement was very important, and optimal measurement time had to be adjusted as the 120th minute following bloodletting. In the study of Sivri et al. (1), in which data were retrospectively analyzed, because the measurement times of complete blood counts were non-standardized, MPV values of patients may have displayed remarkable deviations in association with EDTA, and analysis-related error may have played a role in the differences between the groups.

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In conclusion, MPV measurements should not be used as an indicator of platelet functions. It should be kept in mind that it is primarily necessary to carefully standardize MPV measurement in studies planned to be conducted on MPV.

Best regards,

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