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Letter to the Editors-in-Chief

Mean platelet volume may not play a role in determining thrombosis development in patients with antiphospholipid syndrome

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Dear Editor

We have read with great interest the retrospective study of Llorente-Chávez et al. examining the relationship between mean platelet volume (MPV) and thrombosis and thrombocytopenia in patients with antiphospholipid syndrome (APS) [1]. The authors suggested that the baseline MPV value may be helpful in determining the development of thrombosis in APS patients. We think that there are other factors that may have negatively affected MPV results in this study.

The authors claimed that MPV value is a marker of cardiovascular risk, high MPV values are found in patients with venous thrombosis and ischemic stroke, it is a prognostic factor for mortality in venous thrombosis, and that MPV values increase in many diseases accompanying inflammation. Contrary to the claims of the authors, it was emphasized that MPV values should not be used for reasons such as diagnosis and prognosis in acquired diseases, as measurement standardization of MPV values has not been achieved yet [2]. The main variables affecting MPV measurement standardization are which anticoagulant was used in complete blood count measurements, how long after blood was taken MPV measurement was made, and with which devices MPV measurements were performed [3–6]. A rapid increase in the diameter of platelets begins after they are placed in a blood tube. Ethylenediaminetetraacetic acid (EDTA) is the most widely used anticoagulant and has been shown to cause shape changes in platelets by electron microscopy [3]. After the blood is put into the tube with EDTA, the MPV increase can reach 30% in the first 5 min and up to 40–45% in the first 2 h [3]. It has been shown in different studies that the variability in MPV values can occur between 2 and 50% [3,4]. MPV results also differ according to the anticoagulant used in complete blood count. Whichever anticoagulant is chosen, the increase in diameters of platelets should be expected to stabilize. This is after 120 min for EDTA and 60 min for citrate after blood is drawn [7]. According to the measurement devices used, deviations of up to 40% have been reported in MPV values [3–6]. The fact that MPV measurement methodology was not defined at all in the study of Llorente-Chávez et al. significantly negatively affects the reliability of MPV values.

An important deficiency of this study is that it was designed retrospectively. In retrospective studies, it is not possible to exclude pre-analytical and analytical errors, and it has been emphasized that analytical errors are unacceptable, especially for MPV values [8].

Another problem related to the design of this study is the absence of a healthy control group. The fact that the data of the patient groups could not be compared with the data of the healthy control group leads to the inability to understand whether the MPV data in the patient groups are indeed abnormal. Moreover, the comparison of the compared patient groups in terms of age and gender was not made, and it was reported that age and gender differences may also cause differences in MPV values [2].

Another issue is that the authors claimed that MPV values are a reliable marker of platelet function in order to explain the physiopathological significance of MPV results. The gold standard test used in the measurement of platelet function is light transmission platelet aggregation, and in studies using this method, no correlation has been shown between platelet aggregation results and platelet indices, including MPV [9,10]. Essentially, today, any of the platelet parameters are not included in the platelet function tests.

In conclusion, contrary to the authors' claims, baseline MPV values may not play a role in determining thrombosis development in patients with APS.

Authors' contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Cengiz Beyan and Esin Beyan. The first draft of the manuscript was written by Cengiz Beyan and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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