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Relationship Between Mean Platelet Volume and Hypertrophic Cardiomyopathy: Reply

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We would like to thank Taşolar et al[1] for their kind interest and considerations on our article.[2] As they addressed in their letter, we considered that platelets may be activated in patients with hypertrophic cardiomyopathy (HCM), and this activation may contribute to the thromboembolic events in such patients. In our study, mean platelet volume (MPV), a marker of platelet activation, was elevated in patients with both obstructive and nonobstructive HCM. Moreover, MPV was independently associated with septal thickness ($\beta = 0.66$, 95% confidence interval: 0.42-0.89, $P = .001$).

Previous studies have showed that thromboembolic vascular events such as stroke and arterial embolism can occur in patients with HCM.[3-5] These events are frequently associated with atrial fibrillation (AF).[5] However, the presence of AF cannot entirely account for such an association. Other potential confounders can be older age, left atrial (LA) dilatation, and obstruction of the left ventricular outflow tract (LVOT). Moreover, it may be considered that HCM itself will lead to prothrombotic state.[3] Dimitrow et al have reported that LVOT obstruction was independently associated with enhanced thrombin generation (thrombin–antithrombin complex and prothrombin fragment 1 + 2) and platelet activity (soluble CD-40 ligand, β-thromboglobulin, and P-selectin) in patients with HCM having sinus rhythm.[3] Accordingly, HCM itself may predispose to the prothrombotic state and platelet activation regardless of the presence of AF. We agree with Taşolar et al[1] that patients with HCM should be followed to determine the thrombotic potential of MPV.

In our analyses,[2] MPV was significantly correlated with LVOT obstruction and septal thickness. Septal thickness was only independently associated with MPV levels ($\beta = .66$, 95% confidence interval [CI]: 0.42-0.89, $P = .001$) in linear regression analysis. The "coefficient β" and its 95% CIs were written incorrect by mistake. Its correct values are $\beta = .66$ (95% CI: 0.42-0.89, $P = .001$).

In our study,[2] MPV was significantly correlated with LA diameter, systolic and diastolic diameter of the left ventricle (LV), thickness of LV posterior and septal wall, LVOT gradient, and platelet count. All of these variables were included in the multivariable linear regression analysis to determine the independent variables associated with MPV. Only LV septal thickness was independently associated with MPV. Furthermore, HCM itself may contribute to the prothrombotic state and platelet activation via non-AF mechanisms as mentioned earlier. We did not find an independent association between MPV and LA size, although it was correlated with LA size. On the other hand, LA enlargement can trigger the development of AF and possibly subsequent thromboembolic events.

Finally, our study is cross-sectional in design. We have no follow-up outcomes for thromboembolic events. Our population is limited to evaluate such hard clinical end points. Therefore, we agree that long-term follow-up studies are needed to document the relationship between arterial thromboembolic events and MPV in patients with HCM.

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