# Mean platelet volume: When the size does matter

To the Editor,

Cardiovascular diseases are known to be associated with unstable atherosclerotic plaques matching with platelet reactions, which lead to thrombus formation and finally clinical events (1). Platelets are heterogeneous blood components, differing in size, density, and reactivity. It is recognized that several substances released from alpha-granules, dense granules, lysosomes, or the cytosol in larger platelets are either vasoactive and prothrombotic (thromboxane A2, coagulation factors), adhesion proteins (P-selectin), growth factors (TGF-beta), chemokines (platelet factor 4), or cytokine-like factors (CD40 ligand). These proteins act in a collaborative way to determine biological functions. In other words, activated platelets are larger, and the mean platelet volume (MPV), a measure of platelet size, could be an accurate and easily available marker of platelet activation. Several studies have reported an increasing MPV associated with the prognosis of either acute coronary syndromes (2) or cerebrovascular diseases (3). However, until this issue in which Kalkan et al. (4) entitled "Mean platelet volume is associated with aortic intima-media thickness in patients without clinical manifestation of atherosclerotic cardiovascular disease." published in Anatol J Cardiol 2015; 15: 753-8 report an association between MPV and the extent of subclinical thoracic aortic atherosclerosis in patients without a clinical manifestation of atherosclerotic cardiovascular disease, we did not know the role of this potential marker in the general population without cardiovascular events, namely, in people whose prothrombotic status is unknown or supposedly inactivated. The authors showed how the extent of thoracic aorta intima to media thickness, as a marker of diffuse atherosclerotic disease, is significantly related to an increasing MPV, supporting the role of systemic thrombocyte activation over the course of atherosclerosis, a relationship that has been previously reported, though in a different scenario such as coronary or carotid arteries (5) and again, in patients with atherosclerotic disease present.

The results reported by Kalkan et al. (4) are interesting, though some questions remain to be answered. MPV is uncomplicated and cheap to obtain, easy to elucidate, and is conventionally measured by automated cell counters. Its increase should suggest a careful assessment of cardiovascular risk; however, more studies are necessary in the general population to confirm the findings by Kalkan et al. (4), and new studies investigating the relationship of MPV with future cardiovascular events in healthy people beyond the wall of their arteries.

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