Mean platelet volume and impaired myocardial reperfusion: Risk factor or innocent bystander?

Mean platelet volume (MPV) has emerged in recent years as a potential independent risk factor for poor clinical outcomes among patients with unstable angina and ST-segment elevation myocardial infarction (STEMI) (1, 2). Since MPV is an indicator of platelet activation and correlates with agreeability, larger and hyperreactive platelets could accelerate the formation of intracoronary thrombus and therefore play an essential role in the pathophysiology of coronary artery disease. Additionally, it has been also related with culprit lesion severity in acute coronary syndromes (3) and left ventricular systolic function in STEMI patients (4).

With the purpose of justifying a pathophysiological mechanism that correlates MPV with major cardiovascular complications, some studies have evaluated the association between MPV and impaired myocardial reperfusion in patients with STEMI (5, 6). Microvascular impairment after STEMI in the presence of infarct-related artery patency could be attributable to small platelet aggregates, among other factors, which could mediate the presence of microvascular injury and endothelial dysfunction in both coronary arterioles and capillaries. High MPV may correspond with an increased number of these platelet aggregates and thus explain the phenomena as slow coronary flow or no reflow of an infarct-related coronary artery (7, 8). This point could be the key to explaining why MPV acts as a risk factor in these patients.

In this context, Kırbaş et al. (9), published in this issue, conducted a retrospective analysis of patients with a first STEMI who underwent reperfusion therapy with thrombolysis to assess the association of MPV with ST-segment resolution, a widely used electrocardiographic variable of successful reperfusion. The study found that higher MPV on admission was associated with impaired ST-segment resolution, defined by the lack of at least 50% ST-segment resolution in the single lead with maximal ST elevation, measured 90 minutes after thrombolytic therapy. These findings could help to strengthen the evidence that correlates higher MPV with impaired reperfusion in STEMI, although some aspects of the study should be considered.

Firstly, as the authors acknowledge in the limitations section, prior use of antiplatelet drugs was not reported because of the retrospective study design, which could modify the MPV values and response to thrombolysis. This is especially relevant if we take into account that MPV is a quantitative variable with a relatively narrow range of values, the regulation of which is multifactorial, and because antiplatelet agents are a significant factor in the modulation of platelet size (10).

Furthermore, the absence of imaging tests for the analysis of the effects of unsuccessful reperfusion should also be considered a major limitation of the study, especially given that other studies have indeed considered this aspect. In this regard, Şarlı et al. (11), reported a significant association of higher MPV with poor postinterventional myocardial blush grade, which is considered a reliable marker for microvascular patency, in patients with STEMI who underwent primary percutaneous coronary intervention. Other studies went further and confirmed the relationship between elevated MPV and a greater area of necrosis and microvascular obstruction, estimated by cardiac resonance (12).

Since MPV is a biochemical value that is easy to interpret, inexpensive, and routinely measured in clinical practice both in the inpatient and outpatient setting, its potential role as a new cardiovascular biomarker is certainly attractive. In this regard, the evidence to date (13) suggests that patients with STEMI have higher MPV values than those without myocardial infarction, that elevated MPV is associated with increased mortality following acute coronary syndromes, and that higher MPV seems to be related with impaired reperfusion, irrespective of the localization of the myocardial infarction, time of ischemia, or reperfusion criteria used. However, as so often occurs in medicine, the solid association between two variables does not imply a cause-effect relationship.

So, the key point is whether routine assessment of MPV on admission could modify our clinical management, as "statistically significant" does not necessarily imply "clinically significant." Therefore, could the value of MPV guide our clinical practice? Or, is it only a "population" prognosis marker without "individual" clinical impact? Unfortunately, evidence progresses in small steps, and these questions probably can not be responded to yet; so, further studies are needed to find a place for MPV in the clinical evaluation of patients with acute coronary syndromes.

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