Commentary

Mean platelet volume: ready for prime time?

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Coronary artery disease (CAD) represents a leading cause of death worldwide despite current advances in drug and revascularization therapies. Given it adverse prognosis, risk stratification of patients with an acute coronary syndrome (ACS) remains a challenging issue. In this regard, different biomarkers are becoming more and more important in diagnosis assessment and outcome prediction.

Platelets play a key role in the pathogenesis of ACS, especially in the setting of atherothrombotic acute myocardial infarction (AMI) with occlusion of the infarct-related artery by a platelet-rich clot (1). Circulating platelets are heterogeneous in size, density and reactivity, and changes in these variables are important for the development of an ACS (2).

Mean platelet volume (MPV) represents a quick approach to the variability in size of circulating platelets and is an easy measurable and inexpensive parameter usually included in routine blood test (3). MPV may reflect platelet load of containing granules and is deemed to be an indicator of platelet function and reactivity (4). Larger platelets seem to be more active from a metabolic and enzymatic point of view, thus having a higher thrombotic potential (5). Specifically, larger platelets were found to be denser and have enhanced aggregability, releasing greater amounts of thromboxane A2 and B2, serotonin and β-thromboglobulin, and expressing more adhesion surface proteins (6-9).

Regarding ischemic heart disease (IHD), MPV was proved to be higher in patients with ACS, with an apparent stepwise decrease from AMI to unstable angina to stable chronic coronary disease or non ACS patients (10). This finding can be explained as an expression of enhanced platelet reactivity in the acute setting of IHD and suggests that MPV could be useful in the evaluation of patients with chest pain (11).

Furthermore, MPV has shown prognostic value in patients with CAD. Goncalves *et al.* reported that elevated MPV was a strong independent predictor of long-term outcomes (death and AMI) after percutaneous coronary intervention, with a comparable predictive value to troponin in patients with an ACS (12).

Huczek *et al.* informed that high MPV was associated with impaired angiographic reperfusion (no-reflow phenomenon) and increased 6-month mortality in patients undergoing primary percutaneous coronary intervention (13). Estévez-Loureiro *et al.* published that increased MPV was an independent predictor of infarct-related artery patency in patients with acute ST-segment elevation myocardial infarction treated with primary angioplasty (14). On the contrary, platelet count itself did not show any diagnostic nor prognostic value in patients with ACS (15). All these findings have been confirmed in later studies and systematic reviews. Chu *et al.* pooled results from three cohort studies involving 3,184 patients with AMI and concluded that elevated MPV (two studies used a cut-off MPV of ≥10.3 fL and the other >9 fL) increased the odds of death as compared with a normal MPV (11.5% *vs.* 7.1%; OR, 1.65; 95% CI, 1.12–2.52, P=0.012) (16). Moreover, recent studies showed prognostic implications of raised MPV beyond IHD (17).

Sun *et al.* enrolled more than eighteen hundred Chinese patients with ST-segment elevation AMI along almost 6 years (18). In this study, patients were classified in four groups according to their MPV in the laboratory tests on admission, with most cases within established normal values (9.5–12.5 fL). Subjects with higher MPV tended to be older and have higher levels of triglyceride, low density lipoprotein cholesterol and white blood cell count, and presented greater prevalence of prior AMI. These findings already point out that elevated MPV was related to a higher-risk population. During a mean follow up of over 4 years, worst outcomes occurred among patients with

larger MPV, either above the normal range (>12.5 fL) or in higher values within the normal range (11.1–12.5 fL). After adjustment of other factors independently associated with survival, increased MPV remained as an independent predictor of all-cause mortality in Cox regression model (MPV =11.1–12.5 fL, HR, 1.38, 95% CI, 1.20–1.68; MPV >12.5 fL, HR, 1.72, 95% CI, 1.41–1.96). Although relationship of MPV and cardiac death was not specifically assessed in this study, most of documented events (144 out of 197) during follow-up were due to cardiovascular issues. As authors already commented in their article, this study is limited due to single-center and retrospective enrollment. Moreover, some well-known prognostic factors in the scenario of an ACS, such as troponin, complete revascularization or left ventricular ejection fraction were not included in the analysis and may potentially acted as confounding factors.

Pathological mechanisms underlying increased adverse outcomes after AMI in patients with elevated MPV remains uncertain and multiple mechanisms may be involved. It has been reported the larger platelets have an increased metabolic activity and expression of inflammatory and coagulatory mediators, giving place to a proaggregating stage (6-9). This may therefore lead to an increased thrombogenic instability. Plaque rupture leading to intracoronary thrombus formation is commonly the precipitating event in ACS and, in addition to any other prothrombotic changes occurring in the atherosclerotic plaque, the presence of large and more reactive platelets is also likely to contribute to thrombosis and recurrent events (19).

From a clinical perspective, these results, alongside with prior evidence, support the role of MPV as a predictor of outcomes in AMI with ST segment elevation. MPV is easy to obtain and interpret, and routinely measured by automated cell counters as compared with other more complex markers of platelet activity. However, further studies are requested to compare MVP and other thrombosis and inflammatory biomarkers, define the exact cut-off value to predict an adverse outcome, assess if MPV might guide antiplatelet therapy selection and define if MPV improves risk stratification beyond current available scores.

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Footnote

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