PLATELET REACTIVITY: JOURNEY TO THE END OF THE NIGHT

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We read with great interest the paper by Stratz et al. (1) on a new automated method to quantify reticulated platelets through several markers, including the immature platelet count (IPC), to predict platelet reactivity (PR). In this well-designed study, the authors observed, using a validated platelet assay, that IPC correlated with on-treatment PR in patients with stable coronary artery disease under clopidogrel and prasugrel. Among several parameters, absolute IPC appeared to be a strong independent predictor of PR. They conclude that IPC was a simple and inexpensive tool to easily assess the on-treatment PR.

However, the study has some limitations. First, in stable coronary artery disease patients the rate and clinical impact of HTPR (High on-Treatment Platelet Reactivity) are reduced (2). Second, the correlation between IPC and PR, although significant, was only observed in the clopidogrel and prasugrel 30-mg groups. Third, very few patients had HTPR (n = 4 of 300), which does not allow to evaluate the sensitivity and specificity of IPC. Indeed, 2 of these 4 HTPR patients had an IPC in the same range as good responders, suggesting a low specificity. Finally, results regarding IPC remains conflicting because studies in acute coronary syndrome with a moderate correlation between IPC and PR under prasugrel and no correlation under ticagrelor (3). Therefore, concluding that IPC may become useful to predict PR seems premature.

An automated, easy-to-perform, rapid, inexpensive, and standardized method to predict PR would be of interest. However, it must be acknowledged that the determination of IPC is not a routine but an optional parameter that is only available in labs using XE-Series Sysmex (Sysmex Europe GmbH, Norderstedt, Germany) hematology analyzers. Furthermore, it is not a platelet function assay and it only reflects thrombopoiesis and the rate of platelet turnover that are contributors rather than predictors of PR for drugs with short-lived active metabolites such as clopidogrel.

Because identifying the factors involved in HTPR is of major interest, studies have investigated its mechanisms, which include intrinsic PR, platelet turnover, body mass index, diabetes, genetic polymorphisms, and more importantly acute coronary syndrome. However, the factors contributing to HTPR differ between patients and may vary over time. Therefore the end-product PR under therapy, which was constantly associated with adverse outcome across several large studies, appeared more relevant than all these individual factors to determine the clinical outcome and predict both bleeding and ischemic events under $P2Y_{12}$–adenosine diphosphate receptor antagonists (2).

REFERENCES

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