
To the Editor:

We read with great interest the article by Price et al in a recent issue of Circulation. The authors concluded that achievement of on-treatment platelet reactivity (OTR) <208 P2Y12 reaction units at 12 to 24 hours after percutaneous coronary intervention (PCI) or during follow-up was associated with a lower risk for cardiovascular events. Considering the potential clinical implications of this attractive study, addressing some methodological issues would be appreciated.

The GRAVITAS trial enrolled patients after PCI with 1 or more drug-eluting stents for the treatment of stable coronary artery disease or non–ST-elevation acute coronary syndrome (ACS). Did the authors find differences in OTR mean values between patients with ACS and patients with stable coronary artery disease? It has been previously described that OTR is higher in patients with ACS in comparison with patients with stable coronary artery disease. The OTR threshold to identify high-risk patients for adverse cardiovascular events may vary in different clinical settings. In addition, the low overall event rates in patients with stable angina after PCI in comparison with patients with ACS makes it even more difficult to establish a standardized OTR threshold. It should be noticed that certain cardiovascular risk factors such as diabetes enhance platelet reactivity irrespective of the clinical condition.

As the authors explain, OTR varies over time. Despite that it is widely accepted to assess platelet reactivity 12 to 24 hours after PCI, it still remains unknown when is the best moment to perform a platelet function test. This concept is supported by the fact that in the GRAVITAS trial, OTR <208 at 12 to 24 hours or at 30 days after PCI was a significant and independent predictor of lower risk for cardiovascular events. In ACS patients, we identified an OTR threshold of 175 P2Y12 reaction units as an independent predictor of 1-year adverse cardiovascular events; the prolonged period (69±30 hours) of intensive treatment before the angiography (and assessment of platelet reactivity) may explain the differences between the OTR threshold obtained in our study and that proposed in the GRAVITAS trial. In the acute phase of ACS, there is a high platelet reactivity (involving not only ADP pathways, but also thrombin, epinephrine, and collagen) and a thrombin generation environment. Platelet reactivity and OTR threshold assessed in the subacute phase may differ from those in the acute phase.

As recently reported in a consensus document, available evidence supports the concept of an OTR threshold that may be used to stratify patient risk for ischemic events following PCI, including stent thrombosis. However, a unique OTR threshold value for all the clinical subsets of coronary artery disease seems not to be the best option.

Disclosures

None.

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References


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