Modern interventional and pharmacological treatments significantly reduced the incidence of mortality and other major adverse cardiovascular events (MACE), and improved contractile function in patients with ST-elevation acute myocardial infarction (STEMI). Double antiplatelet therapy (DAPT) with acetylsalicylic acid (ASA) and an antagonist of the platelet P2Y$_{12}$ receptor for adenosine diphosphate contributes substantially to the improvement of clinical outcomes of these patients. There is a large inter-individual variability of response to clopidogrel, a P2Y$_{12}$ antagonist that is still widely used in this clinical setting: about 30–40% of clopidogrel-treated patients display high on treatment platelet reactivity (HTPR) and, for this reason, are not adequately protected from MACE (1).

Adverse left ventricular remodelling (LVR) following STEMI, which leads to alterations in cardiac geometry, impaired ventricular function, heart failure and ventricular arrhythmias, is still a major cause of mortality and morbidity. Persistent and exaggerated inflammatory reaction contributes, among other factors, to the complex pathogenesis of adverse LVR (2): high levels of high-sensitivity C-reactive protein (hs-CRP), a marker of inflammation, are associated with increased risk for adverse LVR.

An interesting study by Park et al. published in the April 2017 issue of *Thrombosis and Haemostasis* demonstrates that HTPR, measured by the VerifyNow system, predicts the risk of adverse LVR in patients with STEMI on DAPT with ASA and clopidogrel (3). In a multivariate analysis, the combination of HTPR and high hs-CRP was associated with a 21-fold increased risk of LVR occurrence, suggesting a synergism between platelets and inflammation. Indeed, platelets, in addition to playing a central role in physiological primary haemostasis and in the pathogenesis of arterial thrombosis, contribute to the inflammatory process and the regulation of immune response (4). They secrete inflammatory mediators and form heteroaggregates with leukocytes, contributing to their activation and recruitment to the inflamed tissues. Interestingly, many pro-inflammatory effects of platelets are mediated by their P2Y$_{12}$ receptors (3).

Therefore, the study by Park et al. has the important clinical implication that inadequate platelet inhibition by clopidogrel not only is associated with MACE, but also with adverse LVR, pointing to the absolute need for protecting STEMI patients with effective inhibition of P2Y$_{12}$-dependent platelet function.

**Conflicts of interest**


**References**