Post-procedural high platelet reactivity with prasugrel loading predicts in-hospital adverse events in ACS patients

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Background/Introduction: High platelet reactivity (HPR) is associated with adverse cardiovascular events, primarily intrastent thrombosis, after a percutaneous coronary intervention (PCI). However, the relationship between hyperacute postprocedural HPR with prasugrel loading and clinical outcomes in acute coronary syndrome (ACS) remains unclear. Moreover, factors contributing to HPR in ACS with prasugrel loading are also unknown.

Purpose: To assess the effects of post-procedural HPR with prasugrel loading on clinical outcomes in ACS during hospitalization, and to define the appropriate cut-off values and identify factors contributing to HPR.

Methods: A single-center, retrospective observational study that enrolled 154 patients who underwent emergent PCI for ACS with prasugrel loading was performed. The P2Y12 reaction unit (PRU) value was measured immediately after PCI using the VerifyNowR system. The primary end-point was major adverse cardiac events (MACE, defined as the composite of death, myocardial infarction, stroke, heart failure, ventricular arrhythmia needing defibrillation).

Results: The mean patient age (standard deviation) was 70.7 (±12.5) years, 76.6% were men, and the average time from the prasugrel intake to PRU calculation was 103.2 (±48.5) min. During the mean hospital stay of 15.6 (±8.5) days, 24 in-hospital MACE (15.5%) and 8 deaths (5.2%) occurred. Thrombosis events, including myocardial infarction recurrence, did not occur (only one case of spontaneous coronary artery dissection was considered as myocardial infarction recurrence). PRU was significantly higher in the MACE group than that in Non-MACE group (287±55 and 232±64, respectively, $p<0.001$). The ROC curve analysis of PRU for discriminating the significant in-hospital MACE showed the cut-off value of 293 (sensitivity: 62.5%, specificity: 83.1% [AUC=0.756, $p<0.0001$]). A total of 37 patients (24%) were thus categorized as HPR (PRU>293) immediately after the emergent PCI. Kaplan-Meier curve showing MACE events occurred in the HPR group than that in the non-HPR group (40.5% vs 7.6%, $p<0.001$). Multiple cox analysis demonstrated that HPR was independent predictors of MACE in patients with ACS who underwent PCI (OR 11.01, 95% CI 2.39–20.2, $p<0.0001$). Multiple logistic regression model showed old age, female sex, low systolic blood pressure, short prasugrel intake to measure time, and large acute gain were independent predictors of HPR.

Conclusion: PRU was significantly higher in the MACE group, with an appropriate cut-off value of HPR of 293 in this study. HPR was an independent predictor of MACE during hospitalization; however, thrombosis events were not significant. HPR predictors were old age, female sex, low systolic blood pressure, short prasugrel intake to measure time, and large acute gain. This study shows the post-procedural HPR with prasugrel loading in patients with ACS can be a useful predictive marker of adverse events during hospitalization.