Impact of kidney function, ticagrelor and genetic polymorphism on high platelet reactivity

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Background: High platelet reactivity (HPR) is associated with worse clinical outcomes. Patients with chronic kidney disease (CKD) respond poorly to clopidogrel. The purpose was to investigate what is major determinant on HPR in CKD patients.

Methods: A total of 219 (139 normal kidney function, 80 CKD) patients who were treated with clopidogrel or ticagrelor were assessed platelet reactivity and genetic polymorphisms. Platelet function was evaluated by the VerifyNowTM P2Y12 assay. Genes tested were ABCB1, PON1, cytochrome P450 (CYP) 2C19, CYP2C9 and P2Y12. HPR was defined as P2Y12 reaction units ≥ 230.

Results: Platelet reactivity were significantly higher in patients with CKD compared to normal kidney function (288 ± 96 vs 223 ± 69, p < 0.01). But, there were no significant differences of genetic distribution between 2 groups. In patients with CKD and normal kidney function, CYP2C19 carriers had greater platelet reactivity during clopidogrel therapy but there were no differences during ticagrelor treatment regardless of CYP2C19 status (Figure). In multivariate analysis, CKD [hazard ratio (HR): 4.67 (2.27 – 9.78), p < 0.01], CYP2C19 carriers [HR: 2.81 (1.36 – 5.82), p < 0.01] and ticagrelor treatment [HR: 0.29 (0.18 – 0.48), p < 0.01] were significantly correlated with HPR.

Conclusion: Ticagrelor treatment achieved greater platelet inhibition in patients with CKD whereas CKD and CYP2C19 genotype were significantly correlated with HPR.