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Acute Coronary Syndromes

A PHARMACOGENOMIC APPROACH TO ANTIPLATELET THERAPY IN STEMI PATIENTS: REASSESSMENT OF ANTI-PLATELET THERAPY USING AN INDIVIDUALIZED STRATEGY IN PATIENTS WITH ST-ELEVATION MYOCARDIAL INFARCTION (THE RAPID STEMI STUDY)

Oral Contributions
West, Room 3001
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Background: Patients with common at-risk genetic variants have increased MACE following percutaneous coronary intervention (PCI). Ultra rapid genotyping may facilitate a pharmacogenomic approach to anti-platelet therapy in STEMI patients receiving PCI.

Method: We utilized a point-of-care genetic device, designed to identify CYP2C19*2, ABCB1 3435 C → T and CYP2C19*17 carriers, to evaluate the potential of a pharmacogenomic strategy in STEMI patients. Following successful PCI, CYP2C19*2 carriers and ABCB1 3435 TT homozygotes were randomized to a strategy of prasugrel 10mg daily for 1 month or clopidogrel 150mg daily for 7 days then 75mg daily for 3 weeks. The primary endpoint is the proportion of CYP2C19*2 and/or ABCB1 3435 TT carriers with high on-treatment platelet reactivity (HPR) (defined by P2Y12 reactivity unit (PRU) >234) in the prasugrel compared to the clopidogrel arm at 1 month.

Results: Among 102 patients, 59 (57.8%) were randomized following identification of at least one at-risk genotype. Overall, 36.3% and 32.3% were CYP2C19*2 and ABCB1 3435 TT carriers, respectively. The baseline PRU was 183.5±90.6 among carriers compared with 147.3±84.7 in non-carriers, p=0.04. Primary and secondary outcomes are shown below:

Primary and Secondary Platelet Function Outcomes According to Assigned Groups

	Patients without at-risk genetic variants (N=43)	At-risk patients randomized to Prasugrel (N=30)	At-risk patients randomized to High-dose Clopidogrel (N=29)	p-value†
Primary Outcome				
Patients with PRU>234 at Day 30 - no.(%)	2(4.7)	0(0)	7(24.1)	0.005
Patients with PRU>208 at Day 30 - no.(%)	4(9.3)	1(3.3)	10(34.5)	0.003
Secondary Outcomes				
Baseline PRU	147.3±84.7	192.6±100.5	174.1±80.6	0.440
PRU at Day 30	110.4±85.1	53.8±60.3	157.1±94.7	<0.001
% Platelet Inhibition at Day 30	59.6±28.5	80.0±21.6	42.3±31.9	<0.001

Plus-minus values are mean ±SD, PRU = P2Y12 Reactivity Unit
† - p-values between patients randomized to prasugrel vs. high-dose clopidogrel

Conclusions: Point-of-care genetic testing permitted rapid identification of STEMI patients with an increased propensity for HPR. Alteration to prasugrel in carriers of at-risk genetic variants decreased HPR compared to a strategy of high-dose clopidogrel.