as shown in the table. Multivariate logistic regression analysis, DM was independent predictor of intra-stent thrombi on OCT (OR 14.031, 95% CI 2.556-77.033, p=0.002), adjusting ARU, PRU and major risk factors, but ARU, PRU and aspirin / clopidogrel resistance were not related to intra-stent thrombi on OCT.
Conclusions: This OCT study demonstrated that ARU, PRU and aspirin / clopidogrel resistance were not related to intra-stent thrombi on OCT.

	All Patients (n=313)	p	DM (n=94)	P
AR (+)	3 (8.6%)	0.288	2 (11.1%)	1.000
AR (−)	12 (4.4%)		8 (11.1%)	
CR (+)	5 (3.0%)	0.107	5 (8.8%)	0.466
CR (−)	10 (7.0%)		5 (13.5%)	

HTN (n=184)	p	Age≥75 (n=18)	p
0	0.341	0	1.000
8 (4.8%)		1 (7.1%)	
5 (4.6%)	0.823	1 (7.7%)	1.000
3 (3.9%)		0	

OCT-D≥6month (n=170)	p
3 (11.1%)	0.408
9 (6.3%)	
3 (3.9%)	0.234
9 (9.2%)	

AR : Aspirin resistance (ARU≥550), CR ; Clopidogrel resistance (PRU≥250), DM ; Diabetes mellitus, HTN ; Hypertension, OCD-D ; OCT follow-up duration, p ; p-value

TCT-746

Physicians' Response To Platelet Function Testing After Percutaneous Coronary Therapy: Does High On-Treatment Platelet Reactivity Testing Impact Antiplatelet Strategy?

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Background: Although high on-treatment platelet reactivity (HoTPR) predicts future ischemic events after percutaneous coronary intervention (PCI), the clinical applicability of platelet function testing remain unclear. This study aims to evaluate the impact of platelet function testing results on the managing physician's response to escalate antiplatelet therapy after PCI.
Methods: The study included 465 consecutive patients undergoing PCI who were on ≥5 days of maintenance clopidogrel therapy. We performed platelet function testing with the Verify Now P2Y12 (Accumetrics) assay before the PCI. The result of this assay was made known to the managing physician after PCI. HoTPR was defined as >230 P2Y12 reaction units (PRU). Antiplatelet therapy at baseline and at hospital discharge post-PCI was recorded. We also evaluated the 30-day major adverse clinical events (MACE: all-cause mortality, myocardial infarction, and target vessel revascularization) in this population.
Results: The mean age of the study population was 63 years; 73% were male and 22% were African-American. HoTPR was present in 143 patients (30.8%). Of these, only 21(14.7%) were managed with an escalation of antiplatelet therapy (increased clopidogrel dose or switched to prasugrel). Of the remaining 322 patients with appropriate platelet inhibition of ≤230PRU, 55 (17.1%) received more intensive antiplatelet therapy. Patients more likely to receive intensified antiplatelet therapy were those with a history of coronary artery bypass grafting (p=0.03), diabetes mellitus (p=0.03), and dialysis (p=0.04), as well as those presenting with myocardial infarction (p=0.014), receiving vein-graft PCI and treatment to a complex (ACC/AHA type C) lesion. At 30 days, 4 MACE events occurred. The group with HoTPR who received intensified antiplatelet therapy had the highest proportion of events (4.7% vs 2.2%, p=0.04).
Conclusions: The results of platelet function testing do not significantly impact decisions to alter amtiplatelet therapy post-PCI. Rather, baseline clinical and angiographic parameters were primary factors considered when intensifying antiplatelet therapy.

TCT-747

Efficacy and safety of intensified antiplatelet therapy on the basis of platelet reactivity testing in patients after percutaneous coronary intervention: Systematic review and meta-analysis

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Background: ADP-specific platelet function assays were shown to predict thrombotic events, and might be helpful to select candidates for more potent antiplatelet therapy. We aimed to determine the efficacy and safety of giving intensified antiplatelet therapy on the basis of platelet reactivity testing for patients undergoing percutaneous coronary intervention (PCI).
Methods: Electronic databases were searched to find prospective, randomized trials that reported the clinical impact of using an intensified antiplatelet protocol (repeated loading or elevated maintenance doses of clopidogrel, prasugrel or glycoprotein IIb/IIIa inhibitor) on the basis of ADP-specific platelet reactivity testing (using VerifyNow, Multiplate, VASP or light transmission aggregometry) compared to standard-dose clopidogrel. Evaluated efficacy measures included cardiovascular death, non-fatal myocardial infarction and definite/probable stent thrombosis (ST), while major bleeding events were recorded as safety endpoint.
Results: Between 2008 and 2011, 10 clinical trials comprising 4,213 randomized patients were identified. Compared to standard antiplatelet therapy, the intensified protocol was associated with a significant reduction in cardiovascular mortality, ST and myocardial infarction (p<0.01 for all). There was no difference in the rate of major bleeding events between intensified and standard groups (P=0.44). Although the observed effects regarding mortality, ST and bleeding were not heterogeneous, meta-regression analysis revealed that the net clinical benefit of the more potent antiplatelet strategy in patients with HPR significantly depended on the risk of ST of the control group using standard-dose clopidogrel (p=0.023).
Conclusions: Intensifying antiplatelet therapy on the basis of platelet reactivity testing might reduce cardiovascular mortality and ST after PCI, without increasing major bleeding complications. However, the net benefit of giving more potent agents based on platelet function testing depends on the risk of ST with standard-dose clopidogrel.

TCT-748

Double The Dose Of Clopidogrel Or Switch To Prasugrel To Antagonize Proton Pump Inhibitor Interaction

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Background: The potential negative metabolic interaction between proton pump inhibitors (PPI) and clopidogrel is of particular concern. We have hypothesized that doubling the maintenance dose (MD) of clopidogrel (150mg, double) was less effective than switching to prasugrel 10mg MD (switch) to attenuate this potential negative interaction.
Methods: In a prospective randomized placebo-controlled double blind study, we assigned 82 stable coronary artery disease patients treated with 75 mg clopidogrel MD dose and aspirin to receive lanzoprazole (30mg/day) or placebo in addition to either clopidogrel double MD (150 mg/jour) or standard prasugrel MD (10 mg/jour). The primary endpoint was the relative change in residual platelet reactivity (RPA, assessed by light transmission aggregometry, 20μMol ADP) over the 14 days study period [(RPA-baseline-RPA14day)/RPAbaseline]. All the measures were performed 4 hours after drug intake. Exclusion criteria were instability, contraindication to prasugrel and a mandatory use of PPI.
Results: Baseline characteristics were balanced between the 4 groups. The effect of a double clopidogrel MD on RPA was significantly blunted by the co-administration of lansoprazole (-53.6±48.6% versus 0.8±53.7% without and with lansoprazole, respectively, p<0.001) whereas 10 mg of prasugrel MD dramatically reduced RPA irrespective of lanzoprazole co-administration (-81.8±24.8% versus -72.9±32.9%, without and with lanzoprazole, respectively, p=NS). In patients receiving double clopidogrel MD and lansoprazole, RPA was not significantly different from that of clopidogrel 75mg MD alone. These findings were consistent irrespective of the platelet function test used to assess RPA. PPI use was the only parameter with a significant interaction with RPA among clopidogrel groups. CYP2C19 metabolizer status displayed non-significant interaction with RPA irrespective of the thienopyridine strategy.
Conclusions: The effect of a double clopidogrel MD on platelet inhibition is significantly attenuated by the co-administration of lansoprazole as opposed to prasugrel 10mg.