ysis (backward logistic regression) revealed age ≥75 years and recent/recurrent gastrointestinal (GI) bleeding as predictive factors of in-hospital bleeding BARC of any type (OR=2.14, 1.21-3.80 95% CIs, p=0.009 and OR=6.48, 1.13-37.92 95% CIs, p=0.04 respectively). In 215 propensity-matched pairs of patients, rate of in-hospital bleeding BARC of any type did not differ significantly between clopidogrel vs prasugrel or ticagrelor-treated patients (3.7% vs 7.9%, p=0.098).

Conclusions: In a real-world PCI-treated ACS population, older age and previous GI bleeding are associated with an increased risk of low to moderate severity in-hospital bleeding events. A trend towards more bleeding with the newer agents is apparent.

P4914 | BEDSIDE Pharmacodynamic effect of adjunctive cilostazol vs. high-dose clopidogrel in acute coronary syndrome patients according to the CYP2C19 genotype (ACCEL-GENOTYPE) study

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Purpose: Cilostazol use in ACS patients has been reduced the risks of ischemic events and restenosis in East Asians. Compared with Caucasians, East Asians have higher frequency of CYP2C19 loss-of-function allele (*2 and *3). We compared the pharmacodynamic effect of adjunctive cilostazol vs. high-dose clopidogrel in PCI-treated East Asians with ACS according to the CYP2C19 genotype.

Methods: ACS patients were assigned to either clopidogrel 150 mg/d (DOUBLE group; n=136) or cilostazol 100 mg twice a day (TRIPLE group; n=139) or cilostazol 100 mg twice a day + clopidogrel 75 mg/d (TRIPLE group; n=130) on top of aspirin. Platelet aggregation (PA) was measured at baseline and at least 30-day follow-up with light transmittance aggregometry (20 μM ADP and 6 μg/ml collagen). PAs were adjusted with known clinical covariates. CYP2C19*2 or *3 genotype was determined. Primary endpoint was the level of 20 μM ADP-induced PA at follow-up. HPR was defined as 20 μM ADP-induced PA >50%.

Results: During standard-dose clopidogrel treatment, platelet measurements did not differ according to individual CYP2C19 genotype between the groups (Fig. 1A). PA levels and HPR risk increased across the CYP2C19 genotype (p=0.174). At follow-up, the TRIPLE vs. DOUBLE group showed significantly lower level of PAs irrespective of the CYP2C19 genotype (Fig. 1B). In the DOUBLE group, the risk of HPR was observed in 27.3% of patients and increased across the CYP2C19 genotype (adjusted p=0.042), whereas the effect of TRIPLE regimen was not significantly influenced with the CYP2C19 genotype (adjusted p=0.289).

Similar trend was observed for collagen-induced PA.

Conclusion: In PCI-treated East Asians presented with ACS, adjunctive cilostazol exhibited favorable platelet measurements compared with high-dose clopidogrel irrespective of the CYP2C19 genotype.

P4915 | BEDSIDE Pharmacodynamic comparison of pitavastatin versus atorvastatin on platelet reactivity in patients with coronary artery disease treated with dual antplatelet therapy

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Background: Levels of platelet reactivity in patients on Dual Antiplatelet Therapy (DAPT) can be influenced by concomitant treatment with medications (i.e. statins) that inhibit the CYP3A4 system involved in the activation of clopidogrel. Atorvastatin and simvastatin are metabolized by CYP3A4, while pitavastatin is mostly excreted unchanged in bile and undergoes minimal biotransformation through the cytochrome P450 system. The primary objective of this study was to compare the pharmacodynamic effects of a CYP3A4-metabolized statin (atorvastatin) versus a non-CYP3A4-metabolized statin (pitavastatin) in patients with coronary artery disease (CAD) treated with DAPT.

Methods: A total of 102 CAD patients receiving DPAT (clopidogrel) 75 mg plus aspirin 100 mg) after percutaneous coronary intervention entered the PORTO trial. After a 1-week statin wash-out period, patients were randomly assigned to atorvastatin (20 mg/day, N=51) or pitavastatin (4 mg/day, N=51) for 30 days. After another 1-week wash-out period to avoid any carryover effect, cross-over was performed, and patients were switched to the other drug which was continued for 30 days. Platelet reactivity (expressed as P2Y12 reaction units (PRU) by the point-of-care VerifyNow assay) was measured before and at the end of each 30-day treatment period. High platelet reactivity after clopidogrel was defined as a PRU value >208.

Results: After the 30-day treatment period with atorvastatin, platelet reactivity was significantly higher as compared with pre-treatment values (212±96 vs 186±79 PRU, p=0.010), with a more common occurrence of patients showing a PRU>208 (57% vs. 36%, p=0.047). Conversely, after the 30-day treatment period with pitavastatin, platelet reactivity was unchanged as compared with pre-treatment values (178±81 vs 189±73 PRU, NS), with no difference in the frequency of patients showing a PRU>208 before and after treatment (41% vs. 37%, NS).

Conclusion: Pitavastatin, a non-CYP3A4-metabolized statin, does not negatively affect levels of platelet reactivity as compared with atorvastatin in CAD patients on DAPT. ClinicalTrials.gov Identifier: NCT01648829.

INVASIVE ELECTROPHYSIOLOGICAL STUDIES

P4917 | BEDSIDE Location and ratio of high and low contact force during left atrial mapping

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Purpose: The aim of this study was to evaluate the location and the ratio of high/low contact force (CF) during left atrial (LA) mapping.

Methods: CF during point-by-point LA mapping were assessed in 50 patients. In 30 patients, the operators were blinded to the CF during the procedure (Group A). In 20 patients, the CF was displayed to the operators (Group B). As a parameter of the catheter stability, relative standard deviation (RSD=standard deviation of CF*100/mean CF) was calculated at each mapping point. Data were analyzed according to 11 predefined areas (Figure). In Group B, the optimal CF was defined to be 10-40g and the operators attempted to acquire points (pts) in this optimal range. We compared the CF and the catheter stability between 2 groups.

Results: A total of 5866 mapping pts were analyzed (3467 pts in Group A and 2399 pts in Group B). Mean CF was 20±19g in Group A and 19±11g in Group B. Low CF<10g was observed at 1301 pts (36%) in Group A and 398 pts (17%) in Group B (P<.001) (Figure). High CF>40g was noted at 414 pts (12%) in Group A and 67 pts (3%) in Group B (P<.001). While extremely high CF>100g was noted.