Patients undergoing primary PCI appear to draw a benefit from pre treatment with clopidogrel. In other patients, treatment at the time of PCI appears to be sufficient.

**P4847 | BEDSIDE**

**Clinical outcomes for prasugrel versus clopidogrel in a large cohort of the study.**

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**Purpose:** In the TRITON-TIMI 38 study prasugrel reduced the primary endpoint (PEP) - a composite of cardiovascular death, nonfatal myocardial infarction (MI) or nonfatal stroke - as compared to clopidogrel in patients (pts) with acute coronary syndromes (ACS). While separate outcomes for pts with ST-elevation (STE) MI have been published, data for unstable angina (UA) and NSTEMI pts are only available for the PEP and for these 2 groups pooled.

**Methods:** This analysis presents data of PEP, all-cause death, non-CABG TIMI major bleeding and net clinical benefit (NCB, which includes major bleeding) for the DE-induced bleed of 10.074 NSTEMI-ACS pts, as well as for 2582 STEMI NSTEMI pts separately. Additional analyses were performed excluding pts not recommended by the prasugrel label in Europe, e.g. with previous TI/STO, age ≥75 and weight ≤80kg (core cohort), and with the analyses truncated at 360 days.

**Results:** There was a consistent and significant reduction in the occurrence of PEP compared with clopidogrel in the entire NSTEMI-ACS cohort as well as in the UA and NSTEMI groups (p interaction 0.390). While NCB was improved with prasugrel (HR 0.89, p=0.043), non-CABG TIMI major bleeding was increased (HR 1.40, p=0.022), and these findings were consistent for the 2 groups analyzed here (p interaction 0.724 and 0.839).

Limiting the analysis to pts according to the prasugrel label, this drug consistently showed superiority over clopidogrel with regards to PEP and NCB (p interaction 0.980 and 0.989), without significant difference in non-CABG TIMI major bleeding (p interaction 0.522).

**Conclusion:** Main clinical outcomes in separate UA or NSTEMI patient groups from the TRITON-TIMI 38 study are consistent with data for the entire NSTE-ACS cohort of the study.

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**P4848 | BEDSIDE**

**Reversal of dabigatran clotting activity in the rat ex vivo by a specific and sensitive antibody fragment antidote: are there non-specific effects on warfarin, rivaroxaban and apixaban?**

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**Purpose:** The new oral anticoagulants have demonstrated efficacy and safety in preventing stroke in patients with AF; however, they all lack a specific antidote in cases of emergency. A fully humanized monoclonal antibody fragment (Fab) against dabigatran is currently in development. Reversal of dabigatran etexilate (DE) induced blood loss in a rat tail bleeding model by this Fab has been shown at 754±1 NSTEMI pts separately. Additional analyses were performed excluding pts not recommended by the prasugrel label in Europe, e.g. with previous TI/STO, age ≥75 and weight ≤80kg (core cohort), and with the analyses truncated at 360 days.

**Methods:** This analysis presents data of PEP, all-cause death, non-CABG TIMI major bleeding and net clinical benefit (NCB, which includes major bleeding) for the DE-induced bleed of 10.074 NSTEMI-ACS pts, as well as for 2582 STEMI NSTEMI pts separately. Additional analyses were performed excluding pts not recommended by the prasugrel label in Europe, e.g. with previous TI/STO, age ≥75 and weight ≤80kg (core cohort), and with the analyses truncated at 360 days.

**Results:** There was a consistent and significant reduction in the occurrence of PEP compared with clopidogrel in the entire NSTEMI-ACS cohort as well as in the UA and NSTEMI groups (p interaction 0.390). While NCB was improved with prasugrel (HR 0.89, p=0.043), non-CABG TIMI major bleeding was increased (HR 1.40, p=0.022), and these findings were consistent for the 2 groups analyzed here (p interaction 0.724 and 0.839).

Limiting the analysis to pts according to the prasugrel label, this drug consistently showed superiority over clopidogrel with regards to PEP and NCB (p interaction 0.980 and 0.989), without significant difference in non-CABG TIMI major bleeding (p interaction 0.522).

**Conclusion:** Main clinical outcomes in separate UA or NSTEMI patient groups from the TRITON-TIMI 38 study are consistent with data for the entire NSTE-ACS cohort of the study.

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**P4849 | BEDSIDE**

**Pharmacodynamic effects of atorvastatin vs. rosuvastatin in coronary artery disease patients with normal platelet reactivity while on dual antplatelet therapy**

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**Purpose:** In the TRITON-TIMI 38 study prasugrel reduced the primary endpoint (PEP) - a composite of cardiovascular death, nonfatal myocardial infarction (MI) or nonfatal stroke - as compared to clopidogrel in patients (pts) with acute coronary syndromes (ACS). While separate outcomes for pts with ST-elevation (STE) MI have been published, data for unstable angina (UA) and NSTEMI pts are only available for the PEP and for these 2 groups pooled.

**Methods:** This analysis presents data of PEP, all-cause death, non-CABG TIMI major bleeding and net clinical benefit (NCB, which includes major bleeding) for the DE-induced bleed of 10.074 NSTEMI-ACS pts, as well as for 2582 STEMI NSTEMI pts separately. Additional analyses were performed excluding pts not recommended by the prasugrel label in Europe, e.g. with previous TI/STO, age ≥75 and weight ≤80kg (core cohort), and with the analyses truncated at 360 days.

**Results:** There was a consistent and significant reduction in the occurrence of PEP compared with clopidogrel in the entire NSTEMI-ACS cohort as well as in the UA and NSTEMI groups (p interaction 0.390). While NCB was improved with prasugrel (HR 0.89, p=0.043), non-CABG TIMI major bleeding was increased (HR 1.40, p=0.022), and these findings were consistent for the 2 groups analyzed here (p interaction 0.724 and 0.839).

Limiting the analysis to pts according to the prasugrel label, this drug consistently showed superiority over clopidogrel with regards to PEP and NCB (p interaction 0.980 and 0.989), without significant difference in non-CABG TIMI major bleeding (p interaction 0.522).

**Conclusion:** Main clinical outcomes in separate UA or NSTEMI patient groups from the TRITON-TIMI 38 study are consistent with data for the entire NSTE-ACS cohort of the study.

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**P4850 | BEDSIDE**

**Clotigregrel resistance in patients with S-elevation myocardial infarction is associated with high body mass index**

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**Background:** The degree of platelet inhibition on clopidogrel in coronary heart disease patients may be of importance for the risk for new thrombotic events. Degree of inhibition can be evaluated by different laboratory methods.

**Aims and methods:** Patients with S-elevation myocardial infarction (n=466), all treated with PCI, were investigated after a median of 24 hours after symptom onset and 16 hours after PCI. A loading dose of 600 mg clopidogrel in combination with aspirin was given prior to hospital admission. Maintenance dose (75mg od) was given the following morning. The response to clopidogrel was tested by use of the VASP method. In a subset of randomly selected patients analyses with the VerifyNow method were also performed (n=268).

The aims were to assess the frequency of clopidogrel resistance and to compare the two methods. In addition we wanted to explore any differences in subgroups of patients. VASP was determined by flowcytometry (PLT VASP/P2Y12, Biocy- tex, France) and expressed as Platelet Reactivity Index (VASP-PRI). The platelet reaction unit (PRU) was determined by use of VerifyNow assay (Accumetrics, San Diego, California). It was measured before and at the end of each 30-day treatment period. High platelet reactivity after clopidogrel was defined as a PRI value >208.

**Results:** After the 30-day treatment with atorvastatin, platelet reactivity did not significantly change as compared with baseline, pre-treatment evaluation (119 ± 59 PRU, NS), with 2 patients only showing a PRI >208. Similarly, after 30-day treatment with rivaroxaban, platelet reactivity was unchanged as compared with baseline (135 ± 54 vs 128 ±62 PRI, NS), with PRI >208 occurring in 3 patients.

**Conclusion:** Atorvastatin does not negatively affect DAPT as compared with rivaroxaban when is given to stable CAD patients with baseline normal platelet reactivity while on DAPT.

ClinicalTrials.gov Identifier: NCT10567774.

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**References:**


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