CONCLUSIONS PPI use concomitant with clopidogrel is associated with increased risk of mortality and myocardial infarction after coronary intervention. Beneficial effect of clopidogrel may be attenuated by drug interaction with PPI.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS Meta-analysis, Myocardial infarction, Proton pump inhibitors

TCT-256

Prognostic value of ACEF (age, creatinine, ejection fraction) score in patients undergoing percutaneous coronary intervention after acute myocardial infarction

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BACKGROUND Recently, ACEF (age, creatinine, ejection fraction) score showed prognostic value predicting major adverse cardiac events (MACEs) in patients undergoing percutaneous coronary intervention (PCI) after acute myocardial infarction (AMI). However, it has not been fully validated yet in large population. We aimed to assess whether ACEF score would improve the ability of the GRACE score to predict MACEs of patients undergoing PCI after AMI.

METHODS Between November 2005 and July 2014, 11,549 patients (8,442 men; 63±13 year-old) underwent PCI after AMI were included from Korean AMI registry. The ACEF score was calculated as follows: age/Left ventricular ejection fraction × 0.2 - 1 if Serum creatinine>2 mg/dL. The 1-year MACEs were stratified according to ACEF score tertiles: ACEFLOW≤1.07 (n=3,828), 1.07 < ACEF MID ≤ 1.44 (n=3,848), and ACEF HIGH>1.44 (n=3,873). The 1-year MACEs were defined as death, non-fatal MI, and revascularizations.

RESULTS During the follow-up, rate of MACE was significantly higher in the highest tertile group compared with patients in the lower 2 tertiles (8.5% versus 10.5% and 24.0%; log-rank p<0.001). In Cox-proportional hazards model, ACEF score (hazards ratio [HR] 1.60, p<0.001) in addition to Killip class >1 (HR 1.39, p=0.001), anterior MI (HR 1.13, p=0.055), diabetes mellitus (HR 1.27, p=0.001), multivessel (HR 1.51, p<0.001), pre TIMI flow 0 or 1 (HR 1.19, p=0.07), and GRACE score (HR 1.01, p=0.001) was an independent predictor of 1-year MACEs. The respective C-statistics from ACEF score were significantly higher compared to GRACE score in terms of 1-year MACEs (0.672 versus 0.63, p=0.0005) and mortality (0.807 versus 0.777, p=0.0001). The ACEF score significantly improved net reclassification of patients compared to GRACE score in terms of 1-year MACEs (0.153, p<0.001) and mortality (0.038, p<0.0001), and also significantly improved integrated discrimination of patients compared to GRACE score in terms of 1-year MACEs (0.085, p=0.005) and mortality (0.028, p=0.001).

CONCLUSIONS The ACEF score improves the discrimination accuracy of conventional risk model to predict MACEs of patients underwent PCI after AMI.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS Acute myocardial infarction, Percutaneous coronary intervention, Risk model

TCT-257

Frequency and clinical impact of prasugrel cessation after primary PCI in STEMI patients: a prospective cohort study

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BACKGROUND STEMI patients are at increased risk for recurrent ischemic events, and guidelines recommend potent P2Y12 inhibitors for the duration of at least one year after primary PCI. The frequency and clinical impact of premature prasugrel cessation or a change to clopidogrel or ticagrelor following primary PCI remains unknown.

METHODS Between September 2009 and June 2012, 1382 patients with STEMI undergoing primary PCI using newer generation DES and discharged on dual antiplatelet therapy with prasugrel and aspirin were enrolled in the framework of the Comfortable trial, SPUM ACS registry and Bern PCI registry. Prasugrel was prescribed for one year. Clinical follow-up information was obtained at discharge, 30 days and one year. Prespecified categories for prasugrel cessation included disruption (non-compliance, bleeding, side effects), physician recommended discontinuation or a change to clopidogrel. All adverse events and information on prasugrel cessation was collected and independently adjudicated according to the 3 categories. Using Cox regression models with time-dependent variables, we assessed the effect of prasugrel cessation on cardiovascular outcomes. The primary outcome measure was defined as cardiac death, reinfarction, and stroke. Secondary endpoints included death, cardiac death, reinfarction, definite stent thrombosis.

RESULTS A total of 1,382 STEMI patients were included; 1,196 (86.5%) patients completed DAPT on prasugrel throughout one year; Prasugrel was disrupted in 48 (3.5%) patients, discontinued in 42 (3%) patients and switched to another P2Y12 inhibitor in 95 (6.9%) patients. The adjusted hazard ratio (HR) for MACE after prasugrel disruption tended to be increased (2.34 (95% CI 0.85-6.38); p=0.098), while no difference was observed after premature discontinuation (HR – 1.43 (95 CI 0.34-5.91); p=0.63) or change in type of P2Y12 therapy (HR=1.20 (95 CI 0.48-3.01); p=0.69); in each case compared to the period the patients completed DAPT on prasugrel throughout one year; Prasugrel disruption was associated with an increased risk of death (6.09, 95% CI 1.72-21.59, p=0.005), cardiac mortality (HR=4.63, 95% CI 1.00-21.54, p=0.05) and stent thrombosis (HR=4.22, 95% CI 1.27-14.05, p=0.019), while there was a trend towards an increased risk of MI (HR=2.78, 95% CI 0.86-8.94, p=0.087).

CONCLUSIONS A high proportion STEMI patients remained on DAPT with prasugrel throughout one year after primary PCI in routine clinical practice. Prasugrel disruption is infrequent but associated with an increased risk for ischemic cardiovascular events compared to physician guided discontinuation or change to clopidogrel.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS PCI - Percutaneous Coronary Intervention, Prasugrel, ST elevation myocardial infarction

TCT-258

Reasons for False ST Elevation Myocardial Infarction activations at a Primary Percutaneous Coronary Intervention Capable Center

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BACKGROUND Several conditions may mimic a true ST Elevation Myocardial Infarction (STEMI) and lead to false activation of the Cardiac Catheterization Laboratory (CCL). We investigated the reasons for false STEMI activations at our institution.

METHODS We reviewed the medical records of all patients presenting to our institution for percutaneous coronary intervention for possible STEMI from July 2012 to November 2014. A false STEMI activation was
defined as a lack of clinical and electrocardiographic evidence of ongoing myocardial infarction. Patients who underwent cardiac catheterization without an obvious culprit lesion corresponding to the ECG changes were also included as false STEMI. The diagnoses were obtained based on the clinical presentation and ECG findings as well as transthoracic echocardiogram and coronary angiography findings in some cases.

RESULTS Of the 431 STEMI activations, (356/431) 82.6% were males and 17.4% (75/431) were females. The overall false STEMI activation rate was 35.3% (152/431). There were 23% (35/152) females and 77% (117/152) males who had false STEMI activations (p < 0.003). The most frequent cause for false STEMI activation was abnormal ECG findings-19% (29/152), of which presumed new Left Bundle Branch Block accounted for 48% of these. 12.5% had non cardiac chest pain (19/152) and 11.8% (18/152) had Non STEMI (NSTEMI). Coronary artery vaso-spasm occurred in 7.9% (12/152) of cases. Pericarditis was diagnosed in (6.6%) 10/152 and severe hypertension in 5.9% (9/152). There were 5.2% cases (8/152) with a diagnosis of acute or decompenated Congestive Heart Failure (CHF), unstable angina and syncope each. 4.6% (7/152) had cardiac arrest. Pericardial effusion was found in 3.3% (5/152) patients. Three patients (2%) were diagnosed each with Bru-gada and Takotsubo. Two patients (1.3%) had pulmonary embolism (PE), aortic dissection and Implantable Cardioverter Defibrillator (ICD) shocks each. Less common causes were endocarditis, Intracranial Hemorrhage (ICH), Subdural Hematoma, severe Aortic Regurgitation (AR), severe anemia, ventricular aneurysm and sepsis where 1 case of each was observed.

CONCLUSIONS The commonest reason for False STEMI activation was the finding of an abnormal ECG. Presumed new LBBB accounted for 48% of the ECG abnormalities.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS ST-segment elevation myocardial infarction

TCT-259 Impact of ECG-Defined Infarct Location on Mortality in Patients with STEMI Undergoing Primary PCI: Insights from the HORIZONS-AMI Trial

Gennaro Giustino,1 Joe Dizon,2 Philippe Genereux,3 Roxana Mehran,4 Undergoing Primary PCI: Insights from the HORIZONS-AMI Trial

BACKGROUND Anterior ST-segment elevation infarction (A-STEMI) is associated with larger infarct size and greater reduction in left ventricular ejection fraction (LVEF) compared with non-anterior STEMI (NA-STEMI). Whether or not ECG-defined STEMI location predicts long-term mortality after primary percutaneous coronary intervention (PPCI) is unclear. We sought to investigate: (i) the clinical and quantitative coronary angiography (QCA) characteristics of patients with ECG-defined A- and NA-STEMI, and (ii) the unadjusted and independent long-term prognostic impact of ECG-defined STEMI location in patients undergoing PCI.

METHODS Participants from the HORIZONS-AMI trial were categorized according to A-STEMI vs. NA-STEMI ECG-defined STEMI location. By ECG core laboratory analysis, A-STEMI was defined as an ST-elevation in the anterior (V2-4) or anteroseptal (V1-3) ECG leads. Primary endpoint of interest was all-cause mortality at 3 years. Adjusted associations with 3-year mortality were estimated by Cox proportional hazards modeling.

RESULTS Among 2,578 patients undergoing PCI with core laboratory determined STEMI location, 765 (29.7%) and 1813 (70.3%) had A-STEMI and NA-STEMI respectively. Patients with A-STEMI were older and had lower baseline LVEF. Left anterior descending (LAD) artery was the culprit artery in 90% of A-STEMIs, while the right coronary artery (RCA) or left circumflex (LCX) were the culprit arteries in 61.7% and 17.4% of NA-STEMIs, respectively. By QCA, lesion length was longer, and thrombus, calcification, and type B2/C lesions were more prevalent in NA-STEMI. At 3 years, patients with A-STEMI had higher unadjusted rates of 3-year all-cause mortality (7.6% vs. 4.8%, p = 0.004). This association persisted after multivariable adjustment for baseline clinical confounders (hazard ratio [HR]: 1.42; 95% confidence interval [CI]: 1.19–1.69; p = 0.0001). However, after including QCA variables in the multivariable model, ECG-defined A-STEMI had no effect on 3-year mortality (HR: 1.01; 95% CI: 0.77–1.37). On the other hand, QCA variables such as LAD as a culprit artery, presence of LCX plaques with > 50% di-meters stenosis (DS), presence of left main plaques with > 50% DS, and total lesion length did.

CONCLUSIONS ECG-defined A-STEMI was associated with greater long-term mortality in patients undergoing primary PCI from HORIZONS-AMI. QCA variables, however, including infarct artery location, had better long-term prognostic value than ECG-defined STEMI location.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS Electrocardiography, Infarct, PCI

TCT-260 Combination of Platelet Count and Neutrophil-Lymphocyte Ratio (COP-NLR) Predicts Short-term and Long-term Clinical Outcomes in Patients with ST-segment Elevation Myocardial Infarction

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BACKGROUND It is known that elevated inflammatory biomarkers were associated with clinical outcomes of patients with ST-segment elevated myocardial infarction (STEMI). This study investigated the usefulness of a novel inflammation-based prognostic system, named the combination of platelet count and neutrophil-lymphocyte ratio (COP-NLR) for predicting prognosis of patients with STEMI.

METHODS We analysis 305 consecutive patients with STEMI treated with primary PCI. The COP-NLR was calculated as follows: patients with both an elevated platelet count (> 300 × 109/l) and neutrophil lymphocyte ratio (> 3) were allocated a score of 2 and patients showing one or neither were allocated a score of 1 or 0, respectively.

RESULTS The patients with COP-NLR score 0, 1, and 2 was 102, 149, and 23 patients, respectively. The peak CK-MB (181.4 ng/ml, 219.7 ng/ml, and 265.1 ng/ml, respectively) ANOVA p = 0.070) and Troponin T (0.63 ng/ml, 0.78 ng/ml, and 1.57 ng/ml, respectively, ANOVA p = 0.059). During the PCLI, no-reflow tended to be observed more frequently in patients with score 2 (2.8%, 4.7%, and 10.3%, respectively, p = 0.117). The in-hospital mortality was highest in COP-NLR score 2 (0%, 4.7%, and 13.8%, respectively, P = 0.002). The Kaplan-Meier curve showed that higher COP-NLR score significantly associated with major cardiovascular adverse events in 12-month follow-up.

Short-term and long-term clinical outcomes according to COP-NLR score

<table>
<thead>
<tr>
<th>COP-NLR score</th>
<th>N = 106</th>
<th>COP-NLR score 1</th>
<th>N = 161</th>
<th>COP-NLR score 2</th>
<th>N = 25</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital death</td>
<td>0, 0%</td>
<td>6, 4.7%</td>
<td>4, 13.8%</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>All cause death</td>
<td>0, 0%</td>
<td>9, 5.9%</td>
<td>4, 13.8%</td>
<td>0.003</td>
<td></td>
</tr>
<tr>
<td>Non-fatal MI</td>
<td>3, 2.8%</td>
<td>3, 1.8%</td>
<td>1, 3.4%</td>
<td>0.771</td>
<td></td>
</tr>
<tr>
<td>TLR</td>
<td>5, 4.7%</td>
<td>3, 1.8%</td>
<td>0, 5%</td>
<td>0.231</td>
<td></td>
</tr>
<tr>
<td>MACE</td>
<td>7, 6.6%</td>
<td>16, 9.4%</td>
<td>6, 20.7%</td>
<td>0.072</td>
<td></td>
</tr>
</tbody>
</table>

Short-term and long-term clinical outcomes according to COP-NLR score

P-value