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Acute Coronary Syndromes

NOVEL ROLE OF PLATELET REACTIVITY AND INFLAMMATORY RESPONSE IN LEFT VENTRICULAR REMODELING FOLLOWING ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION: THE REMODELING STUDY

Poster Contributions
Hall C
Saturday, March 29, 2014, 3:45 p.m.-4:30 p.m.

Session Title: Clinical Aspects of Anti Platelet Therapy in Acute Coronary Syndrome
Abstract Category: 3. Acute Coronary Syndromes: Therapy
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Authors: *Young-Hoon Jeong, Yongwhi Park, Jin Sin Koh, Jeong Rang Park, Choong Hwan Kwak, Gyeongsang National University Hospital, JINJU, South Korea*

Background: We aimed to determine the linkage of platelet reactivity (PR) and inflammatory response to post-infarct LV remodeling (LVR) following STEMI.

Methods: REMODELING was a prospective, single-center registry of first STEMI patients (n = 150) treated with primary PCI, and given standard aspirin + clopidogrel therapy (600mg loading and 75mg/d). Transthoracic echocardiography was performed at baseline and at 1-month F/U. Biochemical measurements and PR with VerifyNow P2Y12 assay were performed on admission. Primary endpoint was the risk of LVR according to PRU-based quartile distribution. LVR was defined as a relative >20% increase in end-diastolic LV volume (LVEDV) seen at 1-month F/U compared with the baseline on admission.

Results: LVR rate was 10.8% in the 1st quartile, 23.1% in the 2nd, 27.0% in the 3rd, and 35.1% in the 4th (p= 0.015). Optimal cutoff for LVR was ≥248 PRU (AUC: 0.643, p=0.010) by ROC curve analysis. LVR rate increased proportionally according to high-sensitivity CRP-based quartile distribution (p=0.012) and the optimal cutoff was ≥1.4 mg/L (AUC: 0.645, p=0.009). In multivariate analysis, Combined estimation of PRU (≥248) and CRP (≥1.4 mg/L) significantly increased the predictive value for LVR occurrence (OR: 14.1, p < 0.001) (Fig.).

Conclusions: Both platelet reactivity and inflammatory response were associated with the risk of LVR in STEMI patients. The role of intensified antiplatelet or anti-inflammatory therapy in post-infarct LV complication deserves further study.