cardiac function in patients with acute MI and received primary percutaneous coronary intervention.

**Methods:** This retrospective cohort study included patients presented during January 2008 to March 2011 to Peking University Third Hospital with ST-segment elevation MI. All patients received successful primary PCI.

**Results:** MPV was measured serially from admission to day 7 after MI. In 375 patients, MPV reached its peak value (10.16±1.05 fl) at the admission, and then reduced by 16% within the 24 hours. Patients with poorer ventricular function, estimated by high Killip Class (≥2, n=96), had higher MPV values at all-time points studied. By logistic regression model and after adjusting for related confounders, high MPV remained as an independent predictor of Killip Class score≥2 [odds ratio (OR) = 1.873, 95% confidence interval (CI) 1.373-2.673; p = 0.001].

**Conclusions:** MPV undergoes rapid and dynamic changes during the acute phase of MI, and was higher in patients with high Killip Class, suggesting a predictive value of MPV in ventricular dysfunction and clinical outcome of acute phase of MI.

**GW25-e6197**

**Reliability Analysis on Cystatin C and vulnerable plaque of coronary artery disease**

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**Objectives:** To study the relationship between Cys C and vulnerable plaque of coronary artery disease.

**Methods:** Patients accepted coronary angiography were consecutively enrolled in our research, including 60 cases without any coronary stenosis as control group. The coronary artery lesions was divided into type I, II and III plaque group by the morphology of atherosclerotic plaque. Serum Cys C, hs-CRP and lipid were measured in 60 control subjects, 85 typeIplaque, 139 type II plaque and 76 type III plaque. Then we compared Cys C, hs-CRP, LDL-C the independent risk factors of vulnerable plaque, respectively, the relative ratio (RR) were 2.759, 1.453, 1.708 in type II plaque group. The level of Cys C was correlated positively with hs-CRP in type II group (r=0.635, P<0.01), but there are not same correlation between Cys C and hs-CRP.

**Conclusions:** Cys C may have a correlation with the occurrence of coronary vulnerable plaques, which may lead to changes in plaque stability through direct effects and inflammatory factors.

**GW25-e7265**

**The Improvement of Ticagrelor for Clopidogrel Resistance on Patients with Acute Coronary Syndrome**

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**Objectives:** Aim to investigate whether ticagrelor is associated with significant improved platelet activity after PCI in patients with clopidogrel resistance suffering with acute coronary syndrome and to evaluate its efficacy and safety.

**Methods:** Consecutive patients admitted to Department of Cardiology in the General Hospital of Armed Police were enrolled between December 2012 and January 2014. They were patients with a diagnosis of NSTE-ACS who were scheduled for PCI. Blood samples were obtained by venipuncture of the antecubital vein respectively between 6-12 h after the clopidogrel 600mg. The VASP phosphorylation analysis of blood collection was performed with BD FACS Calibur flow cytometer. According to the definition of High Platelet Reactivity (PRI≥95%) accepted internationally which was correlated well with clinical prognosis of patients undergoing PCI, confirmed by several studies and ROC curve analysis, 76 patients with high platelet reactivity (PRI>50%) were included, and were randomized to clopidogrel 75mg qd group, clopidogrel 150mg qd group, ticagrelor 90mg bid group. The VASP assay was performed 2days, 7days and 28days after PCI respectively. Meanwhile, the MACE, bleeding events and adverse reactions were recorded. All patients received aspirin 100mg qd.

**Methods:** 365 consecutive patients admitted for PCI were prospectively screened for inclusion in this study. A total of 289 patients were not included. Therefore, 76 patients were included and randomized to clopidogrel 75mg qd group (n=26) or clopidogrel 150mg qd group (n=25) or ticagrelor 90mg bid group (n=25). 2 patients in clopidogrel 75mg qd group, 3 patients in clopidogrel 150mg qd group and 2 patients ticagrelor 90mg bid group were drop-outs, which refused to test the platelet function. Ultimately, there were 24, 22 and 23 patients respectively finished the whole study. (2) After 28 days antiplatelet treatment, the PRI decreased in three groups, meanwhile, it was significantly lower in patients receiving ticagrelor 90mg bid group compared with other two groups. The PRI of clopidogrel 75mg qd group, clopidogrel 150mg qd group, ticagrelor 90mg bid group were 52.1±11.2, 45.5±9.7, 22.4±9.4, respectively (P<0.001). After pairwise comparison they all have statistic difference (P<0.03, P<0.01, P<0.001). (3) After 28 days antiplatelet treatment, the MACE decreased compared with other two groups (P<0.001, P<0.003). (4) During 28 days follow-up, 2 cardiovascular adverse events and 2 minor bleeding in clopidogrel 75mg qd group; 1 cardiovascular adverse events and 2 minor bleeding in clopidogrel 150mg qd group; 4 minor bleeding in ticagrelor 90mg bid group, not resulting in a statistically difference (MACE:P=0.4; minor bleeding:P=0.6), with no major bleedings recorded.

**Conclusions:** (1) Ticagrelor 90mg bid and clopidogrel 150mg qd can obviously suppress platelet reactivity by VASP phosphorylation analysis compared with clopidogrel resistance group, and ticagrelor 90mg bid was more significantly. (2) There is a tendency that ticagrelor 90mg bid and clopidogrel 150mg qd can reduce MACE in ACS patients with clopidogrel resistance. (3) Ticagrelor 90mg bid and clopidogrel 150mg qd did not increase bleeding events.