

**Conclusion** The high maintenance dose clopidogrel can improve cardiac function. There is potential benefit in increasing coronary blood flow and improving myocardium perfusion. High maintenance dose clopidogrel decreases the acute and subacute thrombosis but do not increase the haemorrhage events.

#### e0634 THE RELATIONSHIP BETWEEN HYPOKALAEMIA AT THE EARLY STAGE OF ACUTE MYOCARDIAL INFARCTION AND MALIGNANT VENTRICULAR ARRHYTHMIA

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**Objective** To investigate the relationship between hypokalaemia at the early stage of acute myocardial infarction (AMI) and malignant ventricular arrhythmia (MVA) as well as the features of hypokalaemia.

**Methods** Total of 302 patients were involved in this study and conformed to the following conditions: getting AMI primarily; onset was within 24 hours, accepted serum potassium test and Holter monitoring on admission, didn't use diuretics before, hyperthyroidism, diabetes, vomiting or diarrhoea resulted from gastrointestinal diseases. Relevant data including types of AMI, namely STEMI or NSTEMI; infarct sites of STEMI; time interval from onset of AMI to admission; whether or not hypokalaemia (serum potassium  $\leq 3.5$  mmol/l) and MVA were recorded. The relationships between hypokalaemia and MVA, the time interval and hypokalaemia, types of AMI and hypokalaemia, infarct sites and hypokalaemia were analysed. SPSS 13.0 was used for statistical analysis. The categorical data was processed with chi-square test and p values below 0.05 were considered significant.

**Results** The incidence of hypokalaemia for 24 patients within 3 h from onset of AMI to admission was 37.5%. The incidence of MVA between the group with and without hypokalaemia had significant difference (10.47% vs 3.36%,  $p < 0.05$ ). The incidence of hypokalaemia between the group within 3h and group within 3 h to 24 h of time interval from onset of AMI to admission had significant difference (37.5% vs 15.47%,  $p < 0.05$ ). There was no significant difference in incidence of hypokalaemia between the group of STEMI and NSTEMI (20.35% vs 12.68%,  $p > 0.05$ ). There was no significant difference in incidence of hypokalaemia between groups with anterior wall AMI and non-anterior wall AMI (25.88% vs 18.81%,  $p > 0.05$ ).

**Conclusion** At the early stage of AMI, hypokalaemia is often present. MVA was close associated with hypokalaemia at the early stage of AMI, which indicated that hypokalaemia was a cause of death.

#### e0635 THE EVALUATION TO THE EFFICACY AND SAFETY OF TIROFIBAN IN ACUTE CORONARY SYNDROME PATIENTS WITH CLOPIDOGREL RESISTANCE DURING PERCUTANEOUS CORONARY INTERVENTION

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**Objective** To assess the efficacy and safety of the tirofiban in acute coronary syndrome patients with clopidogrel resistance undergoing selective percutaneous coronary intervention (PCI).

**Methods** A total of 90 acute coronary syndrome patients with clopidogrel resistance were randomised into two groups, the high

maintenance clopidogrel group (HMCG,  $n=50$ ) and the tirofiban group (TG,  $n=40$ ). All the patients underwent PCI after 7-10 day's medical treatment. Clinical information was collected. The platelet aggregation rate (PAR) were measured, and the markers of platelet activation, PAC-1 and CD62P were measured.

**Result** There was no significant difference in baseline data between two groups. The expression rate of CD62P and PAC-1 in HMCG and TG were higher than the normal control group, but no difference between clopidogrel group and the tirofiban group. After the medical treatment the expression rate of CD62P and PAC-1 in TG is higher than that in HMCG ( $p < 0.05$ ). At the time of 0.5 h after PCI, the expression rate of CD62P and PAC-1 is higher than that before PCI ( $p < 0.05$ ). Until 12 h after PCI the expression rate of CD62P and PAC-1 is dropped down to the level before PCI. There were less MACE cases in TG than that in HMCG in hospital ( $p < 0.05$ ), but no significant difference in haemorrhage events between two groups.

**Conclusion** 150 mg/d clopidogrel can inhibit the activation of platelet but 75 mg/d clopidogrel can't in patients of ACS with clopidogrel resistance. Tirofiban can decrease the MACE cases of patient with clopidogrel resistant during PCI but do not increase the haemorrhage events.

#### e0636 THE RELATIONSHIP STUDY BETWEEN BNP LEVELS AND CK-MB, CTNI CONCENTRATIONS, THE DEGREE OF CORONARY ARTERY DISEASE, HEART FUNCTION IN PATIENTS WITH ST-SEGMENT ELEVATION ACUTE MYOCARDIAL INFARCTION

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**Objective** To analysis the relationship between BNP levels and CK-MB, cTNI concentrations, the degree of coronary artery disease and heart function in patients with acute ST-segment elevation myocardial infarction (STEMI).

**Methods** A total of 86 patients with AMI got intravenous thrombolysis within 6 h after myocardial infarction were divided into group A (BNP  $< 100$  pg/ml), group B (BNP 100–500 pg/ml), C group (BNP  $> 500$  pg/ml) according to the BNP peak level. The BNP level, CK-MB, and the cTNI peak concentrations within 24 h were examined. The heart function was examined by UCG within one week, cardioangiography was performed within 7–10 days after AMI, so that to evaluate the relationship between BNP levels and CK-MB, cTNI concentrations, the degree of coronary artery disease, heart function.

**Result** There were no statistical differences in baseline data among A, B, C groups, the higher the BNP level. The higher the CK-MB level ( $p < 0.05$ ), so is the cTNI peak level ( $p < 0.05$ ). There was a significantly correlation between BNP peak levels and CK-MB, cTNI peak concentrations, while the higher the BNP level, the lower the LVEF was ( $p < 0.05$ ), there was a significantly negatively correlation between BNP peak level and LVEF. Leaman coronary score show that scores in group C are higher than that in B, A groups ( $p < 0.05$ ) caused a positively significantly correlation between BNP peak level and leaman coronary score. There was increased trend of left ventricular end diastolic pressure (LVEDP) ( $p < 0.05$ ). Spearman correlation analysis showed significantly correlation between BNP peak levels and CK-MB, cTNI, LVEF, LVEDP levels. The linear regression equation between BNP peak levels and LVEFs, cTNI levels were  $Y = 0.5466 - 0.00015X$ ,  $Y = 5.6314 + 0.0023X$  (all  $p < 0.05$ ).

**Conclusion** The higher the BNP peak levels, the higher the CK-MB, cTNI levels and the lower the LVEF in acute myocardial infarction patients. The between BNP peak levels had positive with CK-MB, cTNI peak concentrations and the degree of coronary artery disease, negative correlation-ship with LVEF.