

were similar ( $p>0.05$ ). There were no significant differences in the incidence of MACE between two groups.

**Conclusion** ACS patients with loading dose clopidogrel combined with simvastatin or fluvastatin could decrease the MACE, the results in two groups are similar. Neither simvastatin with clopidogrel nor fluvastatin with clopidogrel decreases the platelet activity of clopidogrel.

**e0631** **OBSTRUCTIVE SLEEP APNOEA SYNDROME IS ASSOCIATED WITH THE INCREASED RISK OF LOW-ANTIPLATELET RESPONSE OF CLOPIDOGREL IN PATIENTS WITH UNSTABLE ANGINA**

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**Objective** To address the relationship between low antiplatelet response of clopidogrel and Obstructive Sleep Apnoea Syndrome (OSAS) in patients with unstable angina pectoris.

**Methods** Total of 112 patients hospitalised with unstable angina pectoris from February 2008 to December 2009 were enrolled in this randomised consecutive study. All patients accepted routine treatment including clopidogrel, aspirin, low molecular weight heparin daily. Platelet aggregation (PAR) parameters were measured on samples obtained at baseline and 2nd, 4th, 6th day. All patients were examined for the presence of sleep-disordered breath into 4 quartiles by AponeaLink. The concentration serum adrenaline and norepinephrine were measured in the morning at 06:00 after the sleep study.

**Result** There were no significant differences in the baseline data in all 4 quartiles. However, there was a significant differences in the number of diabetes patients in the first quartiles, healed ( $p=0.0038$ ) compared with other quartiles. At day 2 PAR were inhibited to 63.91% of baseline ( $p<0.01$ ) and 88.38% ( $p>0.05$ ) of baselinctively, in the first quartile. At each of these time points, platelet activity was significantly higher than in patients in other quartiles. At day 6 platelet aggregation were reduced to 32.37%, and 29.75% of baseline respectively in group 2 through 4 ( $p<0.01$  for all). PAR was reduced significantly in patients in the second through fourth quartiles at day 6, but, it showed a lower reduction in the first quartile ( $p>0.05$ ). Compared with that in thebedity of OSAS in the second and third were 25.0% and 14.3% ( $p<0.05$ ), only 3.6% in the fourth group ( $p<0.01$ ). Meanwhile, the concentration first group (60.7%), the mor n of serum adrenaline and nine were higher in the first quartile than others ( $p<0.05$ ).

**Conclusion** OSAS is aicator of low clopidogrel response in unstable angina patients, and higher concentration of epinephrine and norepinephrine in OSAS pa reliable inpatients plaorepinephry a more important role in this situation.

**e0632** **A RANDOMISED COMPARISON STUDY OF RECOMBINANT STAPHYLOKINASE VS RECOMBINANT TISSUE-TYPE TISSUE PLASMINOGEN ACTIVATOR FOR SAFETY AND CORONARY ARTERY PATENCY IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION**

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**Objective** To evaluate the efficacy and safety of r-SAK (recombinant staphylokinase) for acute ST-segment elevation myocardial infarction (STEMI).

**Methods** A total of 48 patients with acute STEMI randomised into r-SAK group and r-tPA group (each with 24 patients). In r-SAK

group, 10mg r-SAK diluted up to 50ml with saline before administration, 2mg bolus over 2 min, followed by an infusion of the remaining 8mg over 30 min. While in r-tPA group, first 8mg bolus over 6 min, then 42 mg over a 90-min period. A 75U/kg heparin bolus was given as r-SAK or r-tPA was infusing for anti-coagulation treatment. CAG were performed at 90 min to confirm infarction location and IRA, stenosis was analysed by QCA, IRA flow was evaluated by TIMI grades, myocardial tissue reperfusion was assessed by TMPG. Acute complications and adverse events were recorded during 30 days after thrombolysis.

**Results** There was no significant difference in baseline data between r-SAK and r-tPA group. There was no difference in IRA distribution between the two groups, the IRA repatency rate ( $p=0.308$ ), TIMI 3 flow ( $p=0.355$ ), myocardial tissue reperfusion ( $p=0.530$ ) in r-SAK group are slightly higher than those in r-tPA group, but the differences was not significant. The acute complications during 30-day period after thrombolysis, include allergic reaction ( $p=0.317$ ), serious arrhythmias ( $p=0.775$ ), heart failure ( $p=0.530$ ), cardiac shock ( $p=1.000$ ), IRA re-occluded ( $p=0.555$ ), postinfarction angina ( $p=0.734$ ) and death ( $p=0.317$ ), have no significant difference between the two groups. The bleeding complications of r-SAK group were slightly less ( $p=0.125$ ). No statistic difference in adverse events was found between the two groups.

**Conclusions** r-SAK proved to be at least as effective as alteplase in inducing early coronary artery patency for STEMI with higher fibrin specificity than r-tPA, r-SAK, and less bleeding complications. The safety of r-SAK thrombolysis therapy is at about the same level of that of r-tPA, not associate with excess mortality and complications of arrhythmia, postinfarction angina and haemorrhage.

**e0633** **THE EFFECT ON LEFT VENTRICULAR FUNCTION AND SAFETY OF HIGH MAINTENANCE DOSE OF CLOPIDOGREL IN PATIENTS WITH ACUTE ANTERIOR MYOCARDIAL INFARCTION UNDERGOING SELECTIVE PCI**

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**Objective** To assess the beneficial efficacy and safety of the high maintenance dose of clopidogrel in acute anterior myocardial infarction (AMI) patients undergoing selective percutaneous coronary intervention (PCI).

**Methods** Fifty two patients were enrolled into this study. These cases were randomly divided into the high maintenance dose group ( $n=26$ , 14 males, 150 mg clopidogrel per day) and the control group ( $n=26$ , 15 males, 75 mg clopidogrel per day). QCA and TIMI Myocardial perfusion grading (TMPG) were used to analyse the lesion and reperfusion of the culprit vessel and myocardium. Record the information of patients in-hospital, in the 1 month and 6 months including the level of BNP, left ventricular ejection fraction (LVEF), the left ventricular peak ejection rate (LPER), the left ventricular peak filling rate (LPFR), the left ventricular time to peak ejection rate (LTPER) and left ventricular time to peak filling rate (LTPFR).

**Result** 1. The CTFC of the high maintenance dose group after PCI was smaller than the standard dose group. The percentage of TMPG 3 grade was higher in the high maintenance dose group. 2. The left ventricular peak ejection rate (LPER), the left peak filling rate (LPFR) 6 months after PCI in the high maintenance dose group was higher than the control group. The left ventricular time to peak ejection rate (LTPER), left ventricular time to peak filling rate (LTPFR) 1 month after PCI in the high maintenance were lower than the control group. 3. There were less acute and subacute thrombosis cases in the high maintenance dose group than the standard dose group. There was no significant difference in haemorrhage events between two groups.