

## GW25-e3172

**The evaluation of short-term prognosis after PCI of unstable angina pectoris patients for triple antiplatelet therapy**

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**Objectives:** Investigate the triple antiplatelet therapy on the evaluation of short-term prognosis of unstable angina pectoris patients after PCI, in order to supply a clinical evidence for patients who were treated by PCI with preoperative and postoperative antiplatelet choices.

**Methods:** Selective PCI patients with UAP 302 cases (male 172 cases, females 130 cases) in our hospital were randomly divided into control group which was double antiplatelet treatment therapy group (aspirin and clopidogrel, 151 cases) and triple antiplatelet therapy group (aspirin, clopidogrel and cilostazol, 151 cases). The day before PCI the dual antiplatelet therapy group was given aspirin and clopidogrel loading capacity 300 mg, after PCI aspirin was 100 mg once a day and clopidogrel 75 mg. While the day before PCI the triple antiplatelet therapy group was given aspirin and clopidogrel load capacity 300 mg, load capacity for cilostazol was 200 mg, after PCI patients in TT were given aspirin 100 mg once a day, clopidogrel 75 mg once a day and cilostazol 100 mg twice a day. Observing in patients with high sensitivity C reactive protein (hs-CRP), cardiac markers, adverse cardiovascular and cerebrovascular events (MACCE) and bleeding events.

**Results:** (1) There were no obvious differences in clinical characteristics (age, sex, hypertension, diabetes, smoking history, proton pump inhibitors (PPI), the average number of vascular lesions, the average number of stent implantation between the double groups) ( $P > 0.05$ ). (2) Using hs-CRP as a sign of inflammatory injury, there was no striking difference between the two groups before PCI  $24\text{h}$  ( $4.67 \pm 0.56$  vs  $4.63 \pm 0.35$ ,  $P = 0.633$ ). The follow-up  $24\text{h}$  hs-CRP there was a clear difference between the conventional treatment group and the TT group ( $7.69 \pm 0.75$  vs  $6.03 \pm 0.63$ ,  $P < 0.001$ ). (3) Detect CK-MB and cTnI after PCI  $16\text{h}$ , the values were significantly higher than the preoperative ones (double antiplatelet group CK-MB:  $16.80 \pm 3.26$  vs  $11.30 \pm 2.38$ ; cTnI:  $0.18 \pm 0.02$  vs  $0.06 \pm 0.02$ ; triple antiplatelet group CK-MB:  $14.30 \pm 2.78$  vs  $10.90 \pm 3.02$ ; cTnI:  $0.17 \pm 0.04$  vs  $0.06 \pm 0.02$ ). But after  $16\text{h}$  triple antiplatelet group compared with dual antiplatelet group CK-MB and cTnI were significantly decreased (CK-MB:  $16.80 \pm 4.326$  vs  $14.30 \pm 2.78$ ,  $P < 0.001$ ; cTnI:  $0.18 \pm 0.02$  vs  $0.17 \pm 0.04$ ,  $P = 0.017$ ). (4) After PCI  $30$  days the two groups had no serious bleeding to stop anti-platelet therapy in patients. In the dual antiplatelet therapy group there were 6 cases of a little bleeding and 9 cases of minor bleeding, while 6 cases of a little bleeding and 15 cases of minor bleeding in the triple antiplatelet group. There was no obvious difference between the dual groups ( $10\%$  vs  $14\%$ ,  $P = 0.538$ ). (5) Within PCI  $30$  days, there were 3 cases of cardiac death, 9 cases of myocardial infarction, 6 patients with revascularization and 6 patients with cerebral stroke in the dual antiplatelet therapy group. While in the triple antiplatelet group, there were 3 cases of myocardial infarction and 3 patients with revascularization. The incidence of MACCE in the triple anti-platelet group was lower than dual antiplatelet therapy group ( $16\%$  vs  $4\%$ ,  $P = 0.046$ ).

**Conclusions:** Compared with dual antiplatelet therapy, preoperative PCI triple antiplatelet therapy based on the load capacity of cilostazol can reduce postoperative inflammatory reaction and the incidence of MACCE after PCI. At the same time PCI does not increase the incidence of postoperative hemorrhage.

## GW25-e3192

**Evidence-based Comparative Safety of Atorvastatin 10mg versus 80mg in Chinese Atherosclerosis Patients**Li Jianping<sup>1</sup>, Hui Wang<sup>2</sup>, Na Qin<sup>2</sup>, Mengxi Chen<sup>2</sup>, Shaowen Tang<sup>2</sup><sup>1</sup>Department of Cardiology, Peking University First Hospital, <sup>2</sup>Department of Epidemiology & Biostatistics, Nanjing Medical University

**Objectives:** To compare the safety profile of atorvastatin 80mg with that of atorvastatin 10mg in Chinese atherosclerosis patients based on systematic review.

**Methods:** Literatures were retrieved from PubMed, EMBASE, CENTRAL, CBMDisc, CMCI, CNKI, VIP and Wanfang database (March 2013). The quality assessment, data extraction and verification of collected literatures were conducted by two reviewers independently. Review Manager 5.2.1 and Indirect Treatment Comparisons (ITC) software were applied to compute direct and indirect effects of adverse reactions. Then the results were described by Odds ratios (OR) and 95% confidence intervals (95% CI) in each comparison group.

**Results:** In total 19 qualified literatures with 1761 atherosclerosis patients were included. The maximum treatment period was 5 years. Adverse reactions such as drug-induced abnormal liver function, myalgia, creatine phosphokinase and gastrointestinal reactions were investigated in 19, 12, 6 and 6 studies respectively. The numbers of direct comparisons in drug-induced abnormal liver function of 10mg vs 80mg, 10mg vs 20mg, 20mg vs 80mg, 10mg vs 40mg, and 40mg vs 80mg were 2, 6, 4, 7, and 1, respectively. The OR of direct effect of drug-induced abnormal liver function in 10mg vs 80mg was 0.43 (95% CI 0.06-2.97). Similarly, the OR of direct effect in 10mg vs 20mg, 20mg vs 80mg, and 10mg vs 40mg were 0.54 (95% CI 0.23-1.26), 0.60 (95% CI 0.15-2.41), and 0.32 (95% CI 0.10-1.01), respectively. No drug-induced abnormal liver function was observed in direct comparisons of 40mg vs 80mg. To compare the indirect effects, setting 20mg as a common comparator in ITC, the OR of indirect effect in 10mg vs 80mg group was 0.324 (95% CI 0.064-1.651). Changing the common comparator from 20mg to 40mg, the OR of indirect effect was dropped to 0.298 (95% CI 0.005-17.263). No drug-induced myalgia was described in

direct comparisons of 10mg vs 80mg, and setting 20mg as a common comparator, the OR of indirect effect of drug-induced myalgia in 10mg vs 80mg was 0.105 (95% CI 0.006-1.753).

**Conclusions:** Our results did not find significant differences between 10mg atorvastatin group and 80mg atorvastatin group in Chinese atherosclerosis patients for drug-induced abnormal liver function and myalgia based on the results of direct and indirect comparison. Similarly, no significant difference was found between 10mg and 40mg in drug-induced abnormal liver function based on the results of direct comparison.

## GW25-e4270

**Effects of heart rate control with ivabradine plus beta-blocker on exercise tolerance in patients after Q-wave myocardial infarction with early left ventricular systolic dysfunction**

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**Objectives:** To investigate the effects of heart rate (HR) control with combination of ivabradine and beta-blocker versus uptitration of beta-blocker on exercise tolerance in patients after Q-wave myocardial infarction (MI) with early left ventricular ejection fraction (LVEF)  $< 45\%$  at 6-month follow-up.

**Methods:** In single-blind parallel-group study 28 patients with a first Q-wave MI and early LVEF 30-45%, sinus rhythm  $> 80$  bpm, Killip class I-II, after reperfusion therapy by percutaneous coronary intervention in 12 patients (42.9%) and thrombolysis in 16 (57.1%) were randomized into 2 groups. As part of standard therapy, all patients at the time of hospital admission received metoprolol tartrate 12.5 mg bid. Apart from metoprolol tartrate (up-titrated to 25 mg bid at day 5, average daily dose  $55.3 \pm 1.7$  mg), at day 5 patients in group 1 ( $n = 16$ ) received ivabradine 2.5 mg bid (up-titrated to 7.5 mg bid, average daily dose  $11.3 \pm 1.5$  mg). In group 2 ( $n = 12$ ) metoprolol tartrate was up-titrated to 75 mg bid (average daily dose  $105.4 \pm 5.2$  mg). The two groups were similar for clinical characteristics. No difference was evidenced in initial resting HR and LVEF between two groups at entry ( $87.3 \pm 1.6$  vs  $86.7 \pm 1.5$  bpm,  $38.5 \pm 1.4$  vs  $38.9 \pm 1.5\%$ , respectively, all  $P > 0.05$ ). Besides LVEF at admission, day 28 and after 6 months, symptom-limited treadmill test (Bruce protocol) to conduct stress tests at the trough of drug activity (12 h after last intake of ivabradine and metoprolol tartrate) was performed at day 28 and after 6 months.

**Results:** Resting HR decreased similarly in both groups (at day 28 -  $62.5 \pm 1.5$  vs  $63.7 \pm 1.4$  bpm, after 6 months -  $61.3 \pm 1.5$  vs  $62.8 \pm 1.6$  bpm, all  $P > 0.05$ ). Group 1 showed a significant increase in LVEF (at day 28 - increased to  $46.3 \pm 1.5\%$ , after 6 months - up to  $49.1 \pm 1.7\%$ , all  $P < 0.01$ ). In group 2 LVEF didn't change significantly (at day 28 -  $42.1 \pm 1.5\%$ , after 6 months -  $43.2 \pm 1.6\%$ , all  $P > 0.05$ ). The difference in LVEF at day 28 after initial treatment between the 2 groups was significant ( $P < 0.01$ ). After 6 months, threshold load capacity, exercise duration and chronotropic reserve significantly improved in both groups in comparison to basal levels (in group 1 -  $6.3 \pm 0.4$  vs  $5.0 \pm 0.3$  MET,  $257.3 \pm 12.6$  vs  $209.4 \pm 12.4$  s,  $54.2 \pm 2.8$  vs  $42.3 \pm 2.5$  bpm, all  $P < 0.01$ ; in group 2 -  $5.2 \pm 0.3$  vs  $4.1 \pm 0.3$  MET,  $211.3 \pm 12.5$  vs  $168.2 \pm 12.2$  s,  $43.8 \pm 2.9$  vs  $34.5 \pm 2.8$  bpm, respectively, all  $P < 0.05$ ). However, in group 1 compared to group 2 higher threshold load capacity, exercise duration and chronotropic reserve (all  $P < 0.01$ ) were attained in spite of higher HR at peak exercise (at day 28 -  $107.3 \pm 2.6$  vs  $98.6 \pm 2.5$  bpm, after 6 months -  $129.5 \pm 2.7$  vs  $106.8 \pm 2.6$  bpm, respectively, all  $P < 0.01$ ).

**Conclusions:** In patients after Q-wave MI with early LVEF  $< 45\%$ , ivabradine in combination with low dose metoprolol tartrate, besides improvement in left ventricular systolic function, increased exercise tolerance at 6-month follow-up.

## GW25-e5270

**The systematic review on Wendan decoction's clinical efficacy in the treatment of Arrhythmia**

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**Objectives:** Although the research about WenDan decoction treatment of arrhythmia a lot, it is lack of rigor, system evaluation, also cannot form the objective evidence. Therefore this research will evaluate the clinical efficacy and safety of Wendan decoction in the treatment of arrhythmia, provide new ideas and the new method for clinical treatment of arrhythmia.

**Methods:** According to the method of Cochrane systematic review. Literatures from WANFANG database, VIP database, the full text of the CNKI database, CBM database and Medline database regarding randomized controlled trials and half a randomized controlled trials of Wendandecocotion in the treatment of arrhythmia were reviewed. As all searches end in 2014. Related literatures were selected and analyzed according to the standards. The method ological quality of the trials was assessed by quality evaluation standard which was recommended by the Cochrane systematic review manual 5.2 and evaluation was performed with software RevMan5.2. And heterogeneity test, sensitivity and bias analysis was carried on.

**Results:** Eleven randomized controlled trials meeting the inclusion criteria were selected and reviewed. All selected trials are in Chinese. A total of 990 patients, the treatment group of 534 people, the control group of 456 people. Only 2 trial is in high quality, the others are not. Meta-analysis results shown: in the comparison with control group, the clinical total effective rate of the Wendan decoction in the treatment