**BASIC SCIENCE, ANIMAL MODELS AND PRECLINICAL STUDIES (TCTAP A-026 TO TCTAP A-029)**

**TCTAP A-026**

The Angiotensin Converting Enzyme-2 Local Activated in Infarct-Related Coronary Accelerated Myocardic Reverse Remodeling After Myocardial Infarction

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**BACKGROUND**

Growing evidence exists for local Angiotensin Converting Enzyme-2 (ACE2) can be highly activated independent, which is little affected by circulation renin-angiotensin system (RAS). But there is rare information about changes in ACE2 activity in acute myocardial infarction and in reverse remodeling.

**METHODS**

All patients admitted with acute myocardial infarction (n = 20), were under urgent percutaneous coronary intervention therapy. Biochemical markers (rein, ACE1, ACE2 and aldosterone activity) were detected, which were from vein, femoral artery and Infarct-related coronary. All patients were arranged with cardiac magnetic resonance imaging (CMR) and echocardiography, CMR and echocardiographic parameters (EF, left ventricular end-diastolic (EDD) and end-systolic diameter (ESD)) were measured. Routine ACEIs were given to all the patients.

**RESULTS**

Coronary’s local ACE2 level were highly activated compared to the circulation1’s, but not observed in rein, ACE1 and aldosterone level. Furthermore, the coronary’s local ACE2 level can’t be inhibited by ACEIs (two weeks). The local ACE2 activity negatively correlated with EF and positively with EDD and ESD in all patients’ populations. And then local ACE2 activities especially increased in patients with definitive heart failure, meanwhile the fibrosis or scarring within infarcted myocardium aggravated with the local increasing ACE2 level detected by CMR.

**CONCLUSION**

The local ACE2 activity appeared to be new biomarker in heart reverse remodeling, whereas heart failure might be imminent by the high local ACE2 levels, which cannot be decreased by ACEIs. Our data suggested that local ACE2 but not RASS was involved in the pathomechanism of heart failure in short term after infarction. The new target drug of inhibition of local ACE2 will be issued in the future.

**TCTAP A-027**

Panax Notoginseng Saponins Safely Boost Cardiac Function and Anti-apoptosis in a Myocardial Infarction Model in the Rat via the MEK/ERK Pathway

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**BACKGROUND**

Panax notoginseng saponins (PNS) are one of the most important compounds derived from roots of the herb Panax notoginseng which are frequently used in the area of cardiovascular protection. Meanwhile, anti-apoptosis offers the potential for treating ischemic cardiomyopathy. The aim of this study was to examine whether PNS can boost cardiac function and anti-apoptosis after myocardial infarction and to determine the role of MEK/ERK pathway.

**METHODS**

A month after left anterior descending coronary artery ligation, rats received either intraperitoneal injection of saline or the same volume of saline. Cardiacfunction was assessed echocardiographically, and anti-apoptosis was assessed by western blot four weeks after therapy.

**RESULTS**

Reductions in infarction area and scar collagen content were observed by improvements in left ventricular function and attenuation of left ventricular remodeling that were observed histologically in the infarct region compared with the saline control. Echocardiography also showed significant improvements in ejection fraction and fractional shortening in the PNS group. Meanwhile, PNS significantly decreased apoptosis relative to control group, evidenced by influencing the expression of death domain protein 3 (CASP3) in the infarct myocardial. Furthermore, the phosphorylation of MEK/ERK was decreased after the treatment with PNS.

**CONCLUSION**

In conclusion, these results show for the first time that PNS can decrease apoptosis and preserve cardiac function at least in part through the MEK/ERK pathway, which provides a novel explanation for the multi-function of PNS on cardiovascular system.