AUTOANTIBODIES AGAINST $\beta_3$-ADRENOCEPTOR REDUCE THE SUSCEPTIBILITY TO MYOCARDIAL ISCHEMIC/REPERFUSION INJURY OF HEART FAILURE RATS

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10.1136/heartjnl-2011-300867.194

Objective Choosing chronic heart failure rats as starting point, to observe whether Autoantibodies against $\beta_3$-adrenoceptor ($\beta_3$AA) can reduce the susceptibility to myocardial ischemic/reperfusion injury of heart failure rats, then to study whether $\beta_3$AA have cardio protective effect to the heart failure rats.

Methods $\beta_3$AA was produced through actively immunised method; The level of $\beta_3$AA in the sera was detected by ELISA, $\beta_3$AA positive/negative IgG were purified by Mab Trap Kit (Amersham, USA), BCA Protein Assay kit (Pierce, USA) was used for protein quantitation, the purities of extractions were assessed by conventional SDS-PAGE; Aortic banding surgery was used to prepare the heart failure model, Ligating the left anterior descending coronary artery for 30 min, then loose the ligature for reperfusion 3 h to make the myocardial ischemia/reperfusion injury, TTC coloration is used to detect the myocardial infarct size, caspase 3 ability assay and TdT-mediated dUTP nick end labelling were used to detect the apoptosis.

Results After $\beta_3$AA was administrated to the rats, the myocardial infarct size induced by ischemia/reperfusion injury was smaller than that in the control group (48.81±6.45% vs 60.25±2.52%, p<0.05); after the heart failure rats undergo the myocardial ischemia/reperfusion injury, the TUNEL-positive cells and the apoptosis index both were significantly lower in $\beta_3$AA positive group than in $\beta_3$AA negative group (24.33±1.82% vs 31.24±1.31%, p<0.01); moreover compared with the $\beta_3$AA negative group, the caspase-3 activity in myocardicocytes was also lower in $\beta_3$AA positive group (1941±150.0 vs 1485±89.39, p<0.01).

Conclusion $\beta_3$AA can decrease the myocardial infarct size and cardiomyocyte apoptosis induced by ischemia/reperfusion injury in heart failure rats, so $\beta_3$AA reduce the susceptibility to myocardial ischemic/reperfusion injury of heart failure rats.