[gw22-e0506]

AUTOANTIBODIES AGAINST β_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 β_3 -adrenoceptor (β_3 AA) can reduce the susceptibility to myocardial ischemic/reperfusion injury of heart failure rats, then to study whether β_3 AA have cardio protective effect to the heart failure rats.

Methods β_3 AA was produced through actively immunised method; The level of β_3 AA in the sera was detected by ELISA, β_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 activity assay and TdT-mediated dUTP nick end labelling were used to detect the apoptosis.

Results After β_3 AA was administrated to the rats, the myocardial infarct size induced by ischemia/reperfusion injury was smaller than that in the control group ($48.81 \pm 6.45\%$ vs $60.25 \pm 2.52\%$, $p < 0.05$); after the heart failure rats undergo the myocardial ischemia/reperfusion injury, the TUNEL-positive cells and the apoptosis index both were significantly lower in β_3 AA positive group than in β_3 AA negative group ($24.33 \pm 1.82\%$ vs $31.24 \pm 1.31\%$, $p < 0.01$); moreover compared with the β_3 AA negative group, the caspase-3 activity in myocytes was also lower in β_3 AA positive group (1941 ± 150.0 vs 1485 ± 89.39 , $p < 0.01$).

Conclusion β_3 AA can decrease the myocardial infarct size and cardiomyocyte apoptosis induced by ischemia/reperfusion injury in heart failure rats, so β_3 AA reduce the susceptibility to myocardial ischemic/reperfusion injury of heart failure rats.