[gw22-e0859]

THE INFLUENCE AND FUNCTION MECHANISM OF HYPERBARIC OXYGEN PRETREATMENT ON MYOCARDIAL ISCHEMIA REPERFUSION INJURY IN RATS

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10.1136/heartjnl-2011-300867.78

Background and objective In recent years, coronary atherosclerotic heart disease become the major disease which is harmful to human health with a rising the trend of incidence. The world health organisation (WHO) predicts that by 2020 cardiovascular disease will become the primary cause of death in the world and Acute myocardial infarction as the most serious coronary heart disease type will become a culprit undoubtedly. Opening occlusion blood vessels early can effectively restore myocardial blood reperfusion, and can effectively save myocardial ischemic heart muscle, but ischemic reperfusion injury caused by reperfusion may lead to further myocardial cell death and cardiac function obstacle. Hyperbaric oxygen has been widely used in clinical, and played a good effect in the treatment of many diseases. The aim of this research is mainly to observe and discusses the influence and function mechanism of hyperbaric oxygen pretreatment on myocardial ischemia reperfusion (MIR) injury in rats.

Methods Specific pathogen-free adult male Wistar rats weighing 200 to 250 g were used for the study provided by Laboratory animal center of Bethune medical college of Jilin University, and rats were divided into Control group, Irgroup, HBOpre+IR group randomly. Rats were exposed to 100% O_2 (hyperbaric oxygenation, HBO) for 60 min in a hyperbaric chamber daily. Rats exposed to hyperbaric conditions were allowed to recover for seven days before heart preparation. The rats received intraperitoneal injection of 20% Urethane, hearts were excised and immediately connected to an aortic cannula, and then perfused at a constant pressure in a non-circulating Langendorff mode with Krebs- Henseleit buffer solution, connect to BL-420 Biological function system. We record the data of HR, LVSP, ±dp/dtmax for different periods. After each experiment, we determined the activity of SOD and the quality of MDA, calculated myocardial infarction size with TTC dyeing, and observed the change of myocardial ultrastructure with HE dyeing. Last, we detected the expression of caspase-3, Bcl-2 and Bax in myocardial cells with immunohistochemical, and cardiomyocyte apoptosis with TUNEL.

Results (1) Before ischemia, HR of HBOPre+IR group was a litter lower compared with Control group and IR group with no difference ($p>0.05$); During reperfusion, HR of HBOPre+IR group was much lower compared with Control group and IR group with significant difference ($p<0.05$); after 5 min of reperfusion, LVSP, ±dp/dt of IR group and HBOPre+IR group increased gradually with significant difference ($p<0.05$), but obviously lower than Control group and level of before ischemia. (2) Compared with IR group, the activity of SOD increased and the quality of MDA decreased in HBOpre+IR group with significant difference ($p<0.05$). (3) Compared with

IR group, myocardium infraction size of HBOpre+IR group reduced with significant difference ($p<0.05$). (4) Light microscopy: in Control group, Cardiac structure was complete, clear, myocyte aligned loose, accidentally saw slightly inflammatory cell; in IR group, Myocardial cells were arranged irregular, cell cytoplasm swelling visible myocardial eosinophilic and enhance and there were lots of inflammatory cells; in HBOpre+IR group, Myocardial cell lineaged more rules, cell slightly swollen visible myocardial cell cytoplasmic eosinophilic enhancement, with a little inflammatory cell. (5) Compared with Control group, AI of IR group increased obviously ($p<0.05$); Compared with IR group, AI of HBOPre+IR group decreased significantly ($p<0.05$). (6) Compared with Control group, the expression and apoptosis index of caspase-3 and Bax were significantly higher, the expression of Bcl-2 was reduced in IR group with significant difference ($p<0.05$); Compared with IR group, the expression and apoptosis index of caspase-3 and Bax were decreased, the expression of Bcl-2 was increased in HBOPre+IR group with significant difference ($p<0.05$).

Conclusions (1) Hyperbaric Oxygen Pretreatment plays a role in the improvement of heart function, enhances the ability of antioxidation, reduces infarct size and infiltration of inflammatory cells. (2) Hyperbaric Oxygen Pretreatment may reduce cardiomyocyte apoptosis and MIRI by downer gulating the expression of caspase-3 and Bax and up-regulating the expression of Bcl-2