EXOGENOUS RECONSTITUTED HIGH-DENSITY LIPOPROTEIN (RHDL) ATTENUATES THE MYOCARDIAL ISCHEMIC-REPERFUSION INJURY

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Background: High-density lipoprotein (HDL) is well known to promote cholesterol efflux to prevent the atherosclerosis. In addition, the anti-oxidant properties of HDL are important to protect the cardiovascular system as well. We investigated the protective effect of HDL through pharmacological postconditioning against myocardial I/R injury and its underlying mechanisms.

Methods: In male Sprague-Dawley rats (weight of 200-230mg), 100mg/Kg of rHDL or placebo was infused intravenously at the onset of reperfusion. Area of necrosis (AN by TTC stain), the area at risk (AAR by Evans blue stain), and LV size were measured after 30 min. of coronary occlusion and 24 hours of reperfusion. For the analysis of oxidative stress and anti-oxidant effect, related parameters, as malondialdehyde (MDA), thiobarbituric acid reactive substances (TBARS) and superoxide dismutase (SOD), were measured in AAR, normal left LV area, RV and plasma. To evaluate the role of inflammation on I/R injury and response to rHDL, we measures the CRP in plasma.

Results: rHDL administration reduced infarct size in AN/AAR by 43% (control vs. rHDL; 51.3±10.4 vs. 29.4±18.3, p<0.05) and showed the trend of decrease in AN/LV by 33% (control vs. rHDL; 32.1±10.1 vs. 21.5±12.3, p=0.06), while the size of AAR/LV was not altered. rHDL enhanced the enzymatic anti-oxidant level (SOD level of control vs. rHDL; 0.45 ± 0.24 U vs. 1.39 ± 0.52 U in LV, 0.29 ± 0.13 U vs. 1.07 ± 0.52 U in AAR, and 0.77 ± 0.13 U vs. 1.02 ± 0.18 U in plasma, p<0.05) and showed the trends of decrease in oxidative stress level (control vs. rHDL in AAR; 51.91 ± 6.47 U vs. 33.24 ± 6.02 U for MDA, and 0.24 ± 0.08 U vs. 0.19 ± 0.07 U for TBARS). rHDL also decreased the CRP level by 28.4% (control vs. rHDL; 352.4±44.7 ug/ml vs. 252.4±29.6 ug/ml, p<0.05) in plasma.

Conclusions: These results suggest that rHDL might have a protective postconditioning effect and play an important role on myocardial I/R injury through anti-oxidant and anti-inflammatory mechanisms and imply new clinical application of rHDL for myocardial protection from ischemia-reperfusion injury conjunction with reperfusion therapy for myocardial salvage.