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Early Aerobic Exercise Combined with Hydrogen-Rich Saline as Preconditioning Protects Myocardial Injury Induced by Acute Myocardial Infarction in Rats

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Abstract

It has been reported that hydrogen-rich saline (HRS) water reduces oxidative stress, and early aerobic exercise (eAE) acts an efficient exercise preconditioning (EP) against cardiac I/R injury. However, whether early aerobic exercise combined with hydrogen-rich saline (eAE-HRS) water can more effectively protect myocardial damage induced by acute myocardial infarction (MI) is still unknown. This study was aimed to evaluate the effect of eAE-HRS in preventing MI-induced myocardial damage and explore the possible underlying mechanisms. After Sprague-Dawley (SD) rats were given a intragastric administration of HRS (1.6 ppm) at a dosage of 10 mL/kg weight daily for 3 weeks and/or the SD rats were performed a eAE program with 3 weeks running training, the left anterior descending coronary artery was ligated to induce MI. We assessed the effects of eAE-HRS on myocardial injury and oxidative damage in the MI model of rats and detected the effects of eAE-HRS on the expressions of cardiac OGG1 and Tom40, Tom20, and Tim23. The eAE-HRS increased significantly left ventricular systolic pressure, reduced left ventricular end-diastolic pressure, and potentiated + dp/dtmax, -dp/dtmax, heart coefficient and pH after MI injury. The eAE-HRS reduced MIinduced CK-MB level, c-Tnl level, h-FABP level, infarct size. The eAE-HRS enhanced MI-induced levels of the superoxide dismutase and total antioxidant capacity, attenuated MI-induced levels of malondialdehyde and catalase. The eAE-HRS increased expressions of OGG1, Tom20 and Tim23 proteins after MI injury, but not Tom40. The eAE-HRS has the potential to be a novel precautionary measure to protect myocardial injury after MI via partially regulating expressions of antioxidantrelated proteins and mitochondrial-associated proteins.

Keywords: Aerobic exercise; Cardioprotective; Hydrogen-rich saline; Myocardial infarction.

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