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芪参益气滴丸对心肌梗死大鼠心肌的保护作用

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中文摘要:目的：探讨芪参益气滴丸对心肌梗死大鼠心肌的保护作用及其机制。 方法：成功构建大鼠心肌梗死模型后,随机分为模型组、氟伐他汀对照组、芪参益气滴丸3个不同剂量组各12只,另设假手术组12只。芪参益气滴丸组分别给予低、中、高剂量(15,30,45 mg·kg^{-1}·d^{-1}ig);氟伐他汀组(30 mg·kg^{-1}·d^{-1}ig);模型组和假手术组生理盐水(30 mL·kg^{-1}·d^{-1} ig)。8周后测定血清中乳酸脱氢酶(LDH)、肌酸磷酸激酶(CPK)和心肌组织中超氧化物歧化酶(SOD)、过氧化氢酶(CAT)、谷胱甘肽过氧化物酶(GPx)活性及还原型谷胱甘肽(GSH)含量;应用HE染色分析心肌细胞组织形态学变化;并应用免疫组化法检测心肌毛细血管密度变化,采用TUNEL试剂盒检测心肌细胞凋亡。 结果：模型组大鼠血清中LDH,CPK活性均明显高于假手术组(P<0.05)和芪参益气滴丸组(P<0.05)及氟伐他汀组(P<0.05)，各治疗组间无统计学差异;而心肌组织中SOD,CAT,GPx活性和GSH的含量,模型组均明显低于假手术组(P<0.05)和各治疗组(P<0.05),芪参益气滴丸高剂量组明显高于氟伐他汀组(P<0.05)。模型组心肌细胞肥大增生明显,而芪参益气滴丸和氟伐他汀组细胞肥大程度明显降低。免疫组化分析表明,和假手术组相比,模型组毛细血管密度略显升高,芪参益气滴丸组和氟伐他汀组新生毛细血管明显增多,密度显著增加(P<0.05)。芪参益气滴丸组和氟伐他汀组心肌细胞凋亡指数(AI)显著低于模型组(P<0.05)。 结论：芪参益气滴丸可有效对抗大鼠心肌梗死后氧化应激反应、促进心肌毛细血管增生、保持心肌细胞密度、减轻心肌细胞凋亡,从而对梗死大鼠心肌起到较好的保护作用。

中文关键词:芪参益气滴丸　心肌梗死　氧化应激　凋亡

Protection of Qisheng Yiqi Pills on Rats with Myocardial Infarction

Abstract: Objective: To study the protection of Qisheng Yiqi pills on rats with myocardial infarction (MI). Method: Left coronary artery of Sprague-Dawley rats were ligated to make MI model. The rats were randomly subjected to MI model group, three different Qisheng Yiqi pills dose(15, 30, 45 mg·kg^{-1}·d^{-1}) groups, fluvastatin control group(30 mg·kg^{-1}·d^{-1}) and sham-operated group,each group consisted of 12 rats. The MI group and normal control group were fed 0.9% sodium chloride 30 mL·kg^{-1}·d^{-1}. Eight weeks later, the rats were sacrificed, blood was collected, and the serum separated was used for assaying the activity of lactate dehydrogenase (LDH) and creatine phosphokinase (CPK). Immediately after the sacrifice, the heart was excised and homogenized. The homogenate was used for assaying the activity of antioxidant enzymes superoxide dismutase (SOD), catalase (CAT), glutathione-peroxidase (GPx), and glutathione (GSH). Segmental heart samples were used for hematoxylin and eosin stain and histological evaluation. For measurement of capillary density, sections were immunohistochemically stained with a specific primary antibody. Capillary density was defined as the capillary to cardiomyocyte ratio. Apoptosis was determined using TUNEL kit. Result: Compared with the Sham-operated group, Qisheng Yiqi pills and fluvastatin group, the activities of marker enzymes, LDH, CPK in the serum of the MI group were significantly increased(P<0.05). While the activities of SOD,CAT,GPx and GSH in the heart of MI group were significant decreased than the normal control group, Qisheng Yiqi pills and fluvastatin group$P$<0.05), compared with sham operated mice, the MI group indicated compensatory cardiac hypertrophy, and the capillary density was significantly decreased(P<0.05). While in the Qisheng Yiqi pills group, the capillary density was significantly increased(P<0.05)and the number of TUNEL positive cells was significantly lower than the MI group (P<0.05). Conclusion: Qisheng Yiqi pills can significantly reduce myocardial damage, counteract the oxidative stress induced by myocardial infarction in rats, increase microvessel density, and decrease cardiomyocyte apoptosis.

keywords: Qisheng Yiqi pills　myocardial infarction　oxidative stress　apoptosis

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