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解偶联蛋白-2在大鼠缺血预适应心肌中的表达

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摘要:

目的:探讨解偶联蛋白-2(UCP2)在心肌缺血预适应(IPC)心肌保护中的作用。方法:采取结扎左冠状动脉的方法复制大鼠心肌缺血再灌注模型。IPC组行3次缺血5min,再灌注10min的预处理。缺血再灌注(IR)组与IPC组行30min缺血及120min再灌注;对照组不结扎左冠状动脉。电镜观察心肌超微结构。据Rainio评分标准进行心肌超微结构损伤程度的半定量分析,随机选取20个低倍视野,计算平均心肌细胞凋亡数。采用RT—PCR和Western印迹法检测心肌中UCP2的表达。结果:IPC组Rainio评分和心肌细胞凋亡率均低于IR组(氏0.05),IPC组的UCP2 mRNA和蛋白表达水平均较IR组明显增加(P〈0.01)。结论:IPC可减轻心肌超微结构损伤程度和减少细胞凋亡。IPC可诱导UCP2表达,提示UCP2可能参与了IPC的心肌保护作用。

关键词:心肌 缺血预适应 解偶联蛋白2 心肌超微结构

The expression of uncoupling protein-2 on myocardium of rats following ischemic preconditioning

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Abstract:

Objective: To discuss the role of uncoupling protein 2(UCP2) in myocardium with ischemic preconditioning(IPC). Method: The model of myocardial ischemia reperfusion injury in rats was established by ligation of left coronary artery. Myocardium of rats in both ischemic reperfusion(IR) and IPC groups were subjected to 30min of ischemia followed by 120min of reperfusion. And the myocardium in IPC group was subjected to three episodes of 5min of ischemia coupled to 10min of reperfusion. The ultrastructural changes were observed under electron microscopy and the apoptosis ratio of cardiomyocyte was also determined. The content of UCP2 in mitochondria was tested by Western blot and RT-PCR. Result: In IPC group the apoptosis ratio of cardiomyocyte markedly decreased and the ultrastructural damage attenuated (P<0.05). UCP2 was induced following ischemic preconditioning(P<0.05, vs. IR). Conclusion: UCP2 is overexpressed following ischemic preconditioning, and it may be involved in the mechanism of the cardioprotection induced by IPC.

Keywords:myocardium ischemic preconditioning uncoupling protein 2 myocardial ultrastructure

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