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论著

肢体缺血后处理对兔急性心肌缺血再灌注损伤的影响

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摘要 目的:探讨肢体缺血后处理对兔急性心肌缺血再灌注损伤的影响及其可能机制。 方法:健康新西兰大白兔30只,随机分为3组(每组10只):对照组(Con)、心肌缺血后处理组(MIP)和肢体缺血后处理组(LIP)。缺血前、缺血后及再灌注结束后分别测定血浆磷酸肌酸激酶(CK)活性和丙二醛(MDA)含量;实验结束后,测心肌梗死面积并检测心肌组织髓过氧化物酶(MPO)活性。 结果:MIP和LIP组心肌梗死面积均明显低于Con组(P < 0.01);再灌注180 min末血浆CK活性检测证实心肌梗死面积的这种差异;MIP和LIP组再灌注180 min末MDA含量明显低于Con组(P < 0.01);MIP和LIP组中性粒细胞在缺血心肌的聚集程度,即组织MPO活性(U/100 g)均明显轻于Con组 (P < 0.01)。 结论:心肌缺血再灌注前肢体短暂缺血具有显著的心肌保护作用。这种远隔器官缺血后处理心脏保护作用可能与减轻活性氧的损伤及抗氧化作用加强有关。

关键词 缺血后处理 再灌注损伤 活性氧

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Impact of remote postconditioning on rabbit hearts with acute myocardial ischemia-reperfusion injury

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Abstract

<FONT face=Verdana>AIM:In this study, we tested the hypothesis that remote postconditioning induced by a single 5-min episode of femoral artery occlusion and reperfusion applied immediately before the onset of coronary artery reperfusion protects the myocardium from reperfusion injury.
METHODS:Thirty healthy New Zealand white rabbits were randomly divided into three groups (n=10 in each group):control, myocardial ischemic postconditioning (MIP) and Limb ischemic postconditioning (LIP). Myocardial infarct size and tissue myeloperoxidase (MPO) activity were determined at the end of the experiment. Plasma creatine kinase (CK) activity and malondialdehyde (MDA) levels were measured at baseline, the end of ischemia, and after 180 min of reperfusion, respectively.
RESULTS:Myocardial infarct size was significantly reduced in MIP and LIP as compared to control (P<0.01), also was confirmed by plasma CK activity. Plasma MDA, a product of lipid peroxidation, was significantly lower at 180 min of reperfusion in MIP and LIP than that in control (P<0.01). Neutrophil accumulation (MPO activity) in the area at risk was lower in MIP and LIP than that in control (P<0.01).
CONCLUSION:Limb postconditioning provides potent myocardial infarct size reduction. The potential mechanism of remote postconditioning might be associated with decreasing the injury caused by oxygen free radicals and strengthening the action of antioxidation. </FONT>

Key words Ischemic postconditioning Reperfusion injury Reactive oxygen species

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