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基础医学

rhEPO对氧化应激时心肌细胞及心肌梗死后心脏的保护作用

王福1，刘善文1，李彬2，耿海华3，孙帅1，李睿1，肖洁1，季晓平1

1.山东大学齐鲁医院心内科， 济南 250012; 2.山东大学附属济南市中心医院保健科， 济南 250013; 3.南通大学附属医院心内科， 江苏 南通 226001

摘要：

目的 初步探讨重组人促红细胞生成素(rhEPO)对氧化应激时乳鼠心肌细胞及心肌梗死后小鼠心脏的保护作用。方法 建立乳鼠心肌细胞的氧化应激损伤模型后，加入rhEPO进行干预。以噻唑蓝(MTT)比色法和流式细胞术(FCM)检测rhEPO对心肌细胞凋亡的影响。同时应用Western blotting检测凋亡效应酶胱天蛋白酶-3(caspase 3)、Bax、Bcl-2蛋白的表达水平。将小鼠随机分为假手术组(Sham组)、模型组(MI组)和rhEPO组，通过结扎左冠状动脉前降支建立小鼠心梗模型，14d后取心脏组织，HE染色观察rhEPO对心肌梗死面积的影响。结果 在氧化应激损伤过程中，rhEPO可以明显降低心肌细胞的凋亡率，下调caspase-3和Bax蛋白的表达，上调Bcl-2蛋白的表达。HE染色结果显示，rhEPO可以明显缩小小鼠心肌梗死面积。结论 在氧化应激时，rhEPO可对抗过氧化氢引起的心肌细胞损伤；rhEPO还可缩小小鼠心肌梗死面积。

关键词： 重组人促红细胞生成素；心肌细胞；氧化应激损伤；心肌梗死

Protective effects of rhEPO on oxidative stress damage in cardiomyocytes and on heart after myocardial infarction

WANG Fu1, LIU Shan-wen1, LI Bin2, GENG Hai-hua3, SUN Shuai1, LI Rui1, XIAO Jie1， JI Xiao-ping1

1.Department of Cardiology, Qilu Hospital of Shandong University, Jinan 250012, China;
2. Department of Health, Jinan Central Hospital Affiliated to Shandong University, Jinan 250013, China;
3. Department of Cardiology, Affiliated Hospital of Nantong University, Nantong 226001, Jiangsu, China

Abstract:

Objective To study the protective effects of recombinant human erythropoietin(rhEPO)on cardiomyocytes against oxidative damage and on mice hearts after myocardial infarction. Methods Isolated neonatal rat cardiomyocytes were treated with rhEPO in vitro after the establishment of oxidative damage experimental models. Thiazolyl blue (MTT) assay and flow cytometry (FCM) were used to detect the apoptosis rate of cardiomyocytes. Protein expression levels of caspase-3, Bax and Bcl-2 were detected by Western blotting. Mice were randomly divided into the sham group, the myocardial infarction group and the rhEPO treatment group. Myocardial infarction models were induced by the ligation of the left anterior descending artery in mice. Mouse heart was harvested after 14 days. HE staining was used to investigate the effects of rhEPO on the myocardial infarct size. Results In the process of oxidative damage, rhEPO could significantly reduce the apoptosis rate of cardiomyocytes and the expressions of caspase-3 and Bax, but could raise the expression of Bcl-2. HE staining showed that rhEPO could significantly reduce the myocardial infarct size of mouse heart. Conclusion RhEPO can protect cardiomyocytes from oxidative stress damage induced by H2O2 and reduce the myocardial infarct size in mice after myocardial infarction.

Keywords: Recombinant human erythropoietin; Cardiomyocytes; Oxidative stress damage; Myocardial infarction

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通讯作者: 季晓平， E-mail：xpj64@163.com

作者简介:

作者Email:

参考文献：

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