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论文

羟乙葛根素对大鼠脑缺血再灌注损伤后TNF-*α*表达及NF-*κ*B活性的影响

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摘要：

观察羟乙葛根素对大鼠局灶性脑缺血再灌注损伤后TNF-*α*表达及NF-*κ*B活性的影响。采用大鼠大脑中动脉内栓线阻断法(MCAO)建立大鼠脑缺血再灌注损伤模型，分别于缺血前30 min及再灌注即刻由尾静脉注射羟乙葛根素(10，20及40 mg·kg^{-1})，缺血2 h再灌注24 h后取缺血侧脑组织，HE染色观察大鼠脑组织病理学变化并计数海马CA1区存活神经元数目，放射免疫分析测定脑组织匀浆中TNF-*α*含量，逆转录聚合酶链式反应(RT-PCR)测定脑组织中TNF-*α* mRNA表达情况，凝胶电泳迁移率实验(EMSA)观察NF-*κ*B DNA结合活性改变，Western blotting检测观察I*κ*B*α*蛋白表达情况。羟乙葛根素可明显改善大鼠海马CA1区损伤程度，升高锥体存活神经元数目，减少TNF-*α*蛋白及mRNA表达，抑制NF-*κ*B DNA结合活性。羟乙葛根素可减轻大鼠脑缺血再灌注损伤后炎症反应，这可能是其发挥脑保护作用的机制之一。

关键词： 羟乙葛根素 脑缺血 肿瘤坏死因子-*α* 核因子-*κ*B

Hydroxyethylpuerarin attenuates focal cerebral ischemia-reperfusion injury in rats by decreasing TNF-*α* expression and NF-*κ*B activity

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Abstract:

This study is to investigate the effect of hydroxyethylpuerarin on the expression of tumor necrosis factor-alpha (TNF-*α*) and activity of nuclear factor kappa B (NF-*κ*B) after middle cerebral artery occlusion (MCAO) in rats. Rats were subjected to cerebral ischemia-reperfusion injury induced by MCAO. Hydroxyethylpuerarin (10, 20, 40 mg·kg^{-1}, iv) was administered just 30 min before occlusion and immediately after reperfusion. After a 24 h reperfusion following 2 h of MCAO, the number of viable neurons in hippocampal CA1 region was counted by hematoxylin and eosin (HE) staining. TNF-*α* protein and its mRNA expression were examined with radioimmunoassay (RIA) and reverse transcriptase-polymerase chain reaction (RT-PCR) respectively. NF-*κ*B activity was observed by electrophoretic mobility shift assay (EMSA), and inhibition of NF-*κ*B *α* (I*κ*B*α*) protein expression was evaluated by Western blotting analysis. Animals treated with hydroxyethylpuerarin had a significant increase in neuronal survival in comparison with vehicle-treated group. Hydroxyethylpuerarin significantly reduced the protein and mRNA expression of TNF-*α* following 2 h of ischemia with 24 h of reperfusion. NF-*κ*B DNA binding activity and the degradation of I*κ*B*α* in the cytoplasm also decreased by hydroxyethylpuerarin treatment. The protective effects of hydroxyethylpuerarin against ischemia-reperfusion injury may be mediated by decreasing the expression of TNF-*α* and the activity of NF-*κ*B in rats.

Keywords: cerebral ischemia tumor necrosis factor-alpha nuclear factor kappa B hydroxyethylpuerarin

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