中国病理生理杂志 2005 21 (5): 966-969 ISSN: 1000-4718 CN: 44-1187/R

论著

NFκB在大鼠局灶性脑缺血预处理抗细胞凋亡中作用的研究

邓艳秋[1，2],赵纲[2],赵政[3],王建枝[1]

1华中科技大学同济医学院病理生理教研室， 湖北 武汉 430030；2哈尔滨医科大学病理教研室， 黑龙江 哈尔滨 150086；3哈尔滨工业大学计算机科学与技术学院， 黑龙江 哈尔滨 150001

收稿日期 2003-9-28 修回日期 2003-12-16 网络版发布日期 2009-9-23 接受日期 2003-12-16

摘要 目的：研究NFκB在大鼠局灶性脑缺血预处理抗细胞凋亡中作用。方法： 采用开颅方法阻断大鼠大脑中动脉（MCAO），通过脑梗塞体积分析及病理形态学变化，观察脑缺血预处理的保护作用。采用TUNEL的方法检测神经细胞的凋亡程度。免疫组化染色和细胞化学方法检测脑组织核转录因子NFκB p65蛋白的表达和超氧歧化酶 (SOD)活性、丙二醛（MDA）水平的变化。结果： 预处理未引起脑组织神经元的病理性损伤，相对于未经预处理的缺血组，经预处理的脑缺血组脑组织梗塞体积显著减小，半影区凋亡细胞数明显减少，且细胞核NFκB p65蛋白表达显著增加，脑组织SOD的活性亦明显增大，MDA值明显减小(均P<0.01)。结论： 缺血预处理能够减轻再次的缺血性损伤所诱导的神经细胞凋亡。NF-κB可能是缺血预处理保护中抗凋亡信号调节的关键步骤之一。

关键词 缺血预处理； 脑缺血； 细胞凋亡； NF-κB； 超氧化物歧化酶； 大鼠

分类号 R363

Effect of nuclear factor-κB on the anti-apoptosis induced by focal ischemia preconditioning in Wistar rats

DENG Yan-qiu[1,2], ZHAO Gang[2], ZHAO Zheng[3], WANG Jian-zhi[1]

1Department of Pathophysiology, Tongji Medical University, Huazhong Science and Technology University, Wuhan 430030, China;2Department of Pathology, Harbin Medical University, Harbin 150086, China;3School of Computer Science and Technology, Harbin Institute of Technology, Harbin 150001, China

Abstract

<FONT face=Verdana>AIM: To explore the effect of nuclear factor-κB (NF-κB) on the anti-apoptosis induced by brain ischemia preconditioning (IP). METHODS: Temporary middle cerebral artery occlusion for 20 min followed three days reperfusion before 6 hours middle cerebral artery occlusion (MCAO) trancranially was used as preconditioning in Wistar rats. The protective role was evaluated by analyzing the infarct volume. The status of neuronal apoptosis was observed by TUNEL. The expression of NFκB p65 protein, the assay of SOD activity and MDA concentration were analyzed by using the methods of immunohistochemistry and cytochemistry. RESULTS: Compared to the control group, 20 min ischemic preconditioning, which did not produce neuronal damage obviously, reduced the infarct volume significantly after MCAO 6 h and obviously decreased the number of neural cell apoptosis in penumbra (P<0.01). The expression of NFκB p65 protein and the activity of SOD were extensively increased. The concentration of MDA was decreased in IP cortex and white matter in ischemic group with preconditioning. CONCLUSION: These results indicate that IP is capable of activating NF-κB that in turn increases the expression of genes and participates in IP-induced protective responses to apoptosis. </FONT>

Key words Ischemic preconditioning Brain ischemia Apoptosis NF-kappa B Superoxide dismutase Rats

DOI: 1000-4718

通讯作者　邓艳秋 dengyanqiu66@yahoo.com.cn