

基础医学

兔SAH后海马ICAM-1、NF-κB、p38MAPK表达

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摘要:

目的 探讨兔蛛网膜下腔出血(subarachnoid hemorrhage, SAH)脑血管痉挛(cerebral vasospasm, CVS)后丝裂原活化蛋白激酶p38(p38 Mitogen-activated kinase protein, p38MAPK)、核转录因子-κB(nuclear transcription factor-κB, NF-κB)及细胞间黏附分子-1(intercellular adhesion molecule-1, ICAM-1)在海马的表达。方法 将新西兰纯种健康级大白兔60只分为对照组(n=5)、生理盐水(NS)注射组(n=5)、SAH组(n=30)、干预组(n=20)。SAH组又分为SAH后1、3、5、7、9、11d 6个亚组。干预组分为NS治疗组、ICAM-1单克隆抗体治疗组、NF-κB拮抗剂治疗组、p38MAPK抑制组4个亚组。枕大池二次注射制作兔SAH后CVS模型。分离海马,应用免疫组化观察海马p38MAPK、NF-κB及ICAM-1表达变化。结果 p38MAPK、NF-κB及ICAM-1在对照组和NS注射组的海马神经细胞上仅有微弱染色。伴随着血管腔痉挛的加重,ICAM-1的表达逐渐增强,第7天表达最为强烈;p38MAPK、NF-κB的表达也呈逐渐增强的趋势,但在第5天时表达最为强烈,之后逐渐降低。经拮抗剂治疗后,p38MAPK、NF-κB染色信号明显减弱,ICAM-1仅有微弱表达;经单克隆抗体治疗后,p38MAPK、NF-κB有较强表达,ICAM-1有微弱表达。结论 兔SAH及CVS后海马神经细胞存在着与上述因子相关的免疫炎症反应。抑制上述因子的表达,可以较好地控制损伤脑组织的免疫炎症反应。

关键词: 海马; 丝裂原活化蛋白激酶p38; 核转录因子-κB; 细胞间黏附分子-1; 蛛网膜下腔出血; 脑血管痉挛

Expressions of ICAM-1, NF-κB and p38MAPK in the hippocampus of rabbits after SAH

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Abstract:

Objective To explore the expressions of p38MAPK, NF-κB and ICAM-1 in the hippocampus of the rabbits suffering from the cerebral vasospasm (CVS) caused by subarachnoid hemorrhage (SAH). Methods The 60 New Zealand purebred healthy rabbits were divided into control group (n=5), NS injection group (n=5), SAH group (n=30) and the intervention group (n=20), then the SAH group was divided into 6 subgroups (1, 3, 5, 7, 9, 11d) according to the days after having got the SAH, the intervention group was divided into 4 subgroups which were treated by the NS, the ICAM-1 monoclonal antibody, the NF-κB antagonist and the p38MAPK inhibitor respectively. The CVS models caused by the SAH were made by the secondary blood injection into the major cistern. Then the hippocampus was separated and the Immuno-histochemistry was employed to observe the change of the expression of p38MAPK, NF-κB and ICAM-1 in it. Results The slight dyeing of p38MAPK, NF-κB and ICAM-1 was observed by immuno-histochemistry in hippocampus of the rabbits in the control group and NS injection group. With the CVS aggravating, the expression of the ICAM-1 was enhanced gradually and got the top in the 7th day. The expression of the p38MAPK, NF-κB also had the trend, but got the top in the 5th day, then decreased gradually. After the treatment of the NF-κB antagonist, the dyeing signal of the p38MAPK, NF-κB was weakened obviously, and the ICAM-1 was only expressed slightly, then after the treatment of the ICAM-1 monoclonal antibody, the p38MAPK, NF-κB were expressed strongly while the ICAM-1 was expressed slightly. Conclusion There was the immuno-inflammatory reaction related to the above factors in the hippocampus of the rabbits having got the CVS caused by SAH. The immuno-inflammatory reaction after the cranio-cerebral trauma could be controlled better if the expression of the above factors were inhibited.

Keywords: Hippocampus; p38 Mitogen-activated kinase protein; Nuclear transcription factor κB; Intercellular adhesion molecule-1; Subarachnoid hemorrhage; Cerebral vasospasm

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