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论文

兔SAH后p38MAPK、NF-κB、ICAM-1在基底动脉的表达

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摘要：

目的　探讨兔蛛网膜下腔出血（SAH）后丝裂原活化蛋白激酶p38（p38MAPK）、核转录因子-κB（NF-κB）及细胞间粘附分子-1(ICAM-1)在脑血管痉挛（CVS）发病机制中的作用及其相互间内在关系。方法　将新西兰纯种健康级大白兔92只分为正常组（n=10）、对照组（n=10）、SAH组（n=72），SAH组又分为SAH后1、3、5、7、9、11d 6个亚组。枕大池二次注血制作兔SAH后CVS模型。分离兔基底动脉（BA），应用形态学观察、免疫组化和原位杂交等方法，观察兔BA管径、血管壁上p38MAPK、NF-κB及ICAM-1表达的动态变化及其与CVS的关系。结果　在SAH后第3天， BA管腔出现狭窄，第5天时狭窄明显，第7天狭窄最为明显。伴随着血管腔管径的变化，血管壁上ICAM-1的表达逐渐增强，第7天时表达最为强烈；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。p38MAPK、NF-κB与ICAM-1在蛋白质和mRNA水平上的强烈表达有时程上的差别。结论　在CVS的早期即出现的p38MAPK、NF-κB高表达以及之后出现的ICAM-1高表达，均提示存在由p38MAPK、NF-κB调控的ICAM-1介导的痉挛血管壁的炎症反应，这一级联反应在CVS的发生和发展过程中起了重要作用。

关键词： 蛛网膜下腔出血；丝裂原活化蛋白激酶p38；核转录因子-κB；细胞间粘附分子-1；脑血管痉挛；模型，动物；兔

Expression of p38MAPK, NF-κB and ICAM-1 in the basilar artery of rabbits after SAH

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Abstract:

Objective　To investigate the presence and relationship of p38MAPK, NF-κB and ICAM-1 in the occurrence and progress of CVS(cerebral vasospasm) in the basilar artery of rabbits. Methods　92 New Zealand pure rabbits were divided into the normal, the control and the SAH(subarachnoid hemorrhage) groups, and the latter was further divided into 6 subgroups: groups of 1 day, 3 days, 5 days, 7 days, 9 days, 11 days after SAH. Models of SAH and CVS were successfully established via double-injection into the major cistern and basilar artery(BA) . Morphological observation, immunohistochemistry and hybridization in situ were employed to observe the variances of the basilar artery and dynamic expressions of p38MAPK, NF-κB and ICAM-1in the wall of the BA. Results　Stenosis of BA were identified on 3rd day, obvious on 5th day, and peaked on 7th day after SAH. With the decreased vascular lumen, expression of ICAM-1increased and peaked on 7th day, while expressions of p38MAPK, NF-κB increased and peaked on 5th day but later fell. Expression of p38MAPK, NF-κB and ICAM-1 in protein and mRNA level showed differences in time order. Conclusion　Inflammation controlled by p38MAPK and NF-κB which are induced by ICAM-1 exists in cerebral vasospasm. These relations play important roles in the onset and progress of CVS.

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

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