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论著

黄芩苷对大鼠局灶性短暂性脑缺血再灌继发炎性损伤的保护作用

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摘要 目的 研究黄芩苷对大鼠短暂性脑缺血再灌损伤的保护作用是否与其调节炎症因子和粘附分子的表达有关。方法 线栓法制备大脑中动脉阻断短暂局灶性脑缺血模型，缺血2 h，再灌注24 h。评价神经功能状态和脑梗死体积；用免疫组化、分光光度法测定组织细胞间粘附分子(ICAM)1表达和髓过氧化物酶活性；HE染色观察组织炎性细胞浸润；RT-PCR、免疫印迹和放免法分别测定大脑缺血皮质诱导型一氧化氮合酶（iNOS）、核因子κB（NF-κB）和白细胞介素1(IL-1)的表达。结果 黄芩苷能显著降低大鼠短暂性脑缺血后皮质梗死体积，改善神经功能状态，抑制ICAM-1，iNOS和NF-κB表达，降低缺血皮质IL-1含量，与模型组相比具有显著性差异。结论 黄芩苷通过抑制炎性介质的表达和释放对大鼠短暂性脑缺血损伤具有保护作用。

关键词 黄芩苷 脑缺血 细胞间粘附分子 一氧化氮合酶 NF-κB 白细胞介素1

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Protective effect of baicalin on inflammatory injury following transient focal cerebral ischemia reperfusion in rats

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Abstract

AIM To investigate if the protective effect of baicalin on cerebral injury induced by transient focal ischemia is related to modulation of expressions of inflammatory cytokines or adhesive molecules. **METHODS** Transient focal cerebral ischemia injury model in rats was induced by occlusion of the right middle cerebral artery for 2 h, followed by 24 h reperfusion. The infarct volume and neurological deficit were determined by TTC staining and the scoring method of Longa *et al*. The expression of intracellular adhesion molecule-1 (ICAM-1), neutrophils infiltration, and myeloperoxidase (MPO) activity in brain were measured by immunohistochemistry, hematoxylin-eosin staining, and spectrophotometer, respectively. Semiquantitative RT-PCR was employed to assess the expression of inducible nitric oxide synthase (iNOS) mRNA. The level of interleukin-1 (IL-1) in brain was assayed by radioimmunoassay. The expression of nuclear factor-κB (NF-κB) protein was evaluated by Western blot. **RESULTS** After transient cerebral ischemia, MPO activity and the expression of ICAM-1 in the periphery of ischemic cortex were significantly increased. Increase in iNOS mRNA and NF-κB protein expression was also shown in the ischemic area. Treatment with baicalin markedly reduced brain infarct volume and neurological deficit induced by ischemic insult, inhibited MPO activity, inflammatory cell infiltration, as well as expression of ICAM-1, iNOS and NF-κB, and decreased IL-1 level. **CONCLUSION** Baicalin may play a protective effect on cerebral ischemic injury through inhibiting the expression and release of the inflammatory mediators after cerebral ischemia.

Key words baicalin cerebral ischemia intracellular adhesion molecule-1 nitric oxide synthase NF-kappa B interleukin-1

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