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论著

黄芩苷对局灶性脑缺血再灌注损伤大鼠海马神经细胞凋亡的影响

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摘要 目的 探讨黄芩苷对大鼠局灶性脑缺血再灌注损伤的保护作用及其机制。方法 Wistar大鼠随机分为假手术组、脑缺血再灌注模型组、黄芩苷组(50，100和200 mg·kg^{-1})，以及尼莫地平0.4 mg·kg^{-1}组。利用大鼠大脑中动脉内栓线阻断法制备局灶性脑缺血再灌注损伤模型，通过HE染色、流式细胞术、免疫组化及RT-PCR等方法，观察黄芩苷对缺血再灌注大鼠脑组织形态学改变、神经细胞凋亡率及半胱氨酸天冬氨酸蛋白酶(caspase)-3表达的影响。结果 黄芩苷50，100和200 mg·kg^{-1} iv均可明显改善缺血再灌注损伤所致的大鼠脑组织病理形态学改变，神经细胞凋亡率从模型组的(20.6±5.4)%分别降至（14.5±3.1）%，（11.12±4.2）%及（10.2±2.6）%，促凋亡基因caspase-3 mRNA表达比模型组分别降低23.2%，36.5%及41.0%，其蛋白表达比模型组分别降低25.6%，41.2%及49.3%。结论 黄芩苷对 大鼠局灶性脑缺血再灌注损伤具有保护作用，其作用机制可能与黄芩苷抑制caspase-3表达有关。

关键词 黄芩苷 海马 脑缺血 再灌注损伤 细胞凋亡 半胱氨酸天冬氨酸蛋白酶

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Effects of baicalin on hippocampus neuronal apoptosis in focal cerebral ischemia reperfusion injury rats

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Abstract

AIM To investigate the protection of baicalin from ischemia-reperfusion(I-R) damage in rat hippocampus neurons and its mechanisms. **METHODS** Ninety-six male Wistar rats were randomly divided into six groups（*n*=16）: sham operated group, I-R group, nimodipine group and three baicalin groups, to which baicalin of 50, 100 and 200 mg·kg^{-1} was administered, respectively. The models of focal brain I-R injury induced by middle cerebral artery occlusion were used. HE was used to stain hippocampal slices. Flow cytometry was used for determination of neuronal apoptosis. Caspase-3 protein expression of the neurons was detected with immunohistochemistry. Reverse transcription polymerase chain reaction was used to detect the expression of the mRNA level of caspase-3. **RESULTS** Baicalin（50, 100 and 200 mg·kg^{-1}, iv） was shown to be able to significantly inhibit the neuronal apoptosis from （20.6±5.4）% in I-R group to （14.5±3.1）%, （11.12±4.2）% and （10.2±2.6）% in baicalin treated groups and improved the pathological changes in hippocampus CA1 area. At the same time, baicalin（50, 100 and 200 mg·kg^{-1}, iv） could reduce significantly the transcription of caspase-3 mRNA and the expression of the protein，the percentage of reducton of caspase-3 mRNA was 23.2%, 36.5% and 41.0%, and the protein was 25.6%, 41.2% and 49.3%, respectively. **CONCLUSION** Baicalin can relieve brain damage induced by focal I-R in rats, which may be related to inhibit the process of the neuronal apoptosis resulted from the inhibition of caspase-3 mRNA transcription.

Key words baicalin hippocampus brain ischemia reperfusion injury apoptosis caspase

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