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论文

赤芍801通过抑制NF-KB抗脑缺血-再灌注损伤的机制

郑建明1, 2, 陈晓春1*, 林 敏3, 张 静3, 林智颖1, 郑关毅3, 李康增2

(1. 福建医科大学附属协和医院神经内科, 福建 福州 350001; 2. 福建省宁德市闽东医院神经内科, 福建 宁德 355000; 3. 福建省老年医学研究所, 福建 福州 350001)

摘要:

探讨赤芍801减轻在体大鼠脑缺血—再灌注损伤的可能机制。采用线栓模型制作大鼠局灶性脑缺血—再灌注损伤模型,在缺血后给予赤芍801,用Western blotting方法检测脑缺血周边区的NF- κ B活性、COX-2和HSP70的表达量,用ELISA方法测定TNF- α 的表达量,用RT-PCR方法和免疫荧光染色法检测TLR-4的转录和蛋白表达。结果显示:赤芍801能抑制脑缺血周边区NF- κ B活性,减少COX-2和TNF- α 的表达,同时抑制NF- κ B的上游TLR-4的转录和蛋白表达,对TLR-4的内源性配体HSP70的蛋白表达也有抑制作用。赤芍801作为一种抗氧化剂,有可能通过抑制脑缺血周边区的NF- κ B活性,减少局部的COX-2和TNF- α 的表达,达到减轻脑缺血—再灌注损伤的目的,同时对NF- κ B的上游HSP70和TLR-4产生影响。

关键词: 赤芍801 NF-κB 脑缺血-再灌注损伤

Mechanism of the reduction of cerebral ischemic-reperfusion injury through inhibiting the activity of NF- κ B by propyl gallate

Abstract:

The probable mechanism of the reduction of rat cerebral ischemic-reperfusion injury by propyl gallate was studied. Intraluminal suture middle cerebral artery occlusion model of rat was employed. Propyl gallate was injected immediately after the ischemia was happened. The activity of NF- κ B, and the expression of COX-2 and HSP70 on the peripheral ischemia were determined by Western blotting. The expression of TNF- α was determined by ELISA assay. RT-PCR and immunofluorescence staining were employed to detect the transcription and expression of TLR-4. Results showed that propyl gallate could inhibit the activity of NF- κ B in the peripheral ischemia, and reduce the expression of COX-2 and TNF- α . As the upstream of NF- κ B, the transcription and expression of TLR-4 decreased, as well as HSP70, the endogenic ligand of TLR-4. As an antioxidant, propyl gallate could reduce the cerebral ischemic-reperfusion injury through inhibiting the activity of NF- κ B and decreasing the COX-2 and TNF- α in the peripheral ischemia. It also could influence HSP70 and TLR-4.

Keywords: propyl gallate NF-kB cerebral ischemic-reperfusion injury

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