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羟基红花黄色素A对脑缺血大鼠皮层炎症信号转导途径相关因子的抑制作用

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羟基红花黄色素A(hydroxysafflor yellow A, HSYA)是红花中的单体有效成分。本研究采用线栓法制备大鼠永久 性脑缺血模型,观察HSYA对永久性脑缺血大鼠炎症信号转导途径相关因子的抑制作用。于缺血后3、 6、 12和24 h取大脑皮层。Western blotting 检测细胞核及胞浆核转录因子κB(NF-κB) p65及胞浆磷酸化IκB-α(pIκB-α)蛋白 水平表达; Trans AM试剂盒检测NF-κB DNA结合活性; RT-PCR检测炎性因子TNF-α、 IL-1β、 IL-6和IL-10转录 ▶加入引用管理器 水平表达。结果表明,大鼠脑缺血后多次静脉注射HSYA(10 mg·kg $^{-1}$),能显著抑制p65核转位以及I κ B-a的磷酸化,降低NF- κ B DNA结合活性,并降低促炎因子TNF-a、 IL-1 β 和IL-6 mRNA表达,升高抗炎因子IL-10 mRNA表 达水平。提示HSYA的抗脑缺血作用可能与其抑制炎症信号途径中NF-κB激活及炎性因子转录水平表达有关。 关键词: 羟基红花黄色素A 脑缺血 核转录因子KB 炎性因子

Inhibitory action of hydroxysafflor yellow A on inflammatory signal transduction pathway related factors in rats with cerebral cortex ischemia

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Abstract:

Hydroxysafflor yellow A (HSYA) is a main active monomer purified from Carthamus tinctorius L. The research is to study the inhibitory effect of HSYA on the inflammatory signal transduction pathway related factors which were induced by permanent cerebral ischemia in rats. By using the successive administration at a 30 min interval of HSYA and the rats permanent focal cerebral ischemia model established by a intraluminal suture occlusion method. After cerebral artery occlusion 3, 6, 12 and 24 h, cortex was removed for the next experiments. Western blotting was used to detect the expression of p65 protein and the phospho-I κ B-a (pI κ B-a) in the cytoplasm and nucleus. Nuclear factor- κ B (NF- κ B) DNA binding activity was measured by Trans-AM transcription factor assay kits. mRNA expression of cytokines TNF-a, IL-1 β , IL-6 and IL-10 was measured by the RT-PCR method. The result showed that intravenous injection of HSYA (10 mg kg⁻¹) to rats after cerebral occlusion, the p65 translocation activity Article by and the phosphorylation of IkB-a were significantly inhibited. At the same time, HSYA suppressed p65 binding activity and the transcriptional level of pro-inflammatory cytokines including TNF-a, IL-1 β and IL-6, and promoted the mRNA expression of anti-inflammatory cytokine IL-10. In conclusion, the anticerebral ischemic mechanism of HSYA may be due to its inhibition of NF-kB activity and the mRNA expression of cytokines in the inflammatory transduction pathway.

Keywords: cerebral ischemia nuclear factor-κB inflammatory cytokine hydroxysafflor yellow A (HSYA)

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