

论著

大黄素甲醚对大鼠脑缺血再灌注损伤的拮抗作用

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收稿日期 2004-10-8 修回日期 2005-1-13 网络版发布日期 2010-1-13 接受日期 2005-1-13

摘要 目的: 探讨大黄素甲醚对脑缺血再灌注后IL-1 β 含量和ICAM-1及caspase-3表达的影响。方法: 91只SD大鼠随机分为正常组(normal), 假手术组(sham), 模型组(model), 大黄素甲醚大剂量(PHD)及小剂量(PLD)组。采用线栓法复制大鼠右侧大脑中动脉缺血再灌注模型, 用放射免疫法测定病变侧脑组织IL-1 β 的含量, 用免疫组织化学方法测定ICAM-1和caspase-3表达的变化, 并进行组织病理学观察。结果: Model组再灌注6 h病变侧IL-1 β 含量明显升高且达高峰, 再灌注24 h病变侧ICAM-1、caspase-3表达明显升高, 中性粒细胞附壁浸润明显; 大黄素甲醚PHD组再灌注12 h、24 h病变侧IL-1 β 、ICAM-1和caspase-3表达明显低于model组相应时段($P < 0.05$ 或 $P < 0.01$), 中性粒细胞附壁浸润较少。结论: 大黄素甲醚可降低脑缺血再灌注后IL-1 β 、ICAM-1和caspase-3水平, 减轻脑缺血再灌注损伤。

关键词 [大黄素甲醚](#); [脑缺血](#); [再灌注损伤](#); [白细胞介素1](#); [胞间粘附分子1](#); [半胱氨酸天冬氨酸蛋白酶3](#)

分类号 [R363](#)

Protective effects of physcion against cerebral injury induced by ischemia-reperfusion in rats

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Abstract

AIM: To explore the effect of physcion (P) on the level of IL-1 β and expression of ICAM-1 and caspase-3 during cerebral ischemia-reperfusion injury. METHODS: The 91 healthy adult SD rats were selected, and were randomly divided into normal group, sham-operated group, cerebral ischemia-reperfusion group (model), low-dose physcion (PLD) and high-dose physcion (PHD) treatment group. The level of IL-1 β was detected by radioimmunoassay. The expression of ICAM-1 and caspase-3 was detected by immunohistochemistry. The changes of tissue pathology were also investigated. RESULTS: The level of IL-1 β reached the peak at 6 h after ischemia-reperfusion (IR). The protein expression of ICAM-1 and caspase-3 reached the peak at 24 h after IR. The level of IL-1 β and the protein expression of ICAM-1 and caspase-3 in PHD group decreased obviously compared with those in model group ($P < 0.05$ or $P < 0.01$), infiltration and adhesiveness of neutrophils were less serious at the same time. CONCLUSION: Physcion decreases the level of IL-1 β and the protein expression of ICAM-1 and caspase-3 to protect brain tissue from cerebral ischemia-reperfusion injury.

Key words [Physcion](#) [Brain ischemia](#) [Reperfusion injury](#) [Interleukin-1](#) [Intercellular adhesion molecule-1](#) [Caspase 3](#)

DOI: 1000-4718

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