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苦参素注射液对实验性结肠炎大鼠 结肠黏膜保护机制的研究

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中文摘要:目的:研究NOD2在实验性大鼠结肠炎发病机制中的作用,并探讨苦参素注射液对实验性大鼠结肠炎的治疗作用和治疗机制。方法:将40只SD大鼠随机分为4组:正常对照组、模型组、美沙拉唑组和苦参素注射液(OMT)组,每组10只。对照组未行造模,其余3组均采用三硝基苯磺酸造模。OMT组大鼠给予苦参素注射液肌肉注射,美沙拉唑组大鼠给予美沙拉唑混悬液灌胃。模型组和对照组大鼠均以蒸馏水灌胃,治疗15 d。观察实验大鼠结肠黏膜大体形态及组织病理评分,并用免疫组化检测大鼠结肠黏膜NOD2、NF-κB p65蛋白表达,ELISA检测实验大鼠结肠黏膜IL-6的表达。结果:与对照组相比,模型组大鼠结肠黏膜NOD2、NF-κB p65蛋白、IL-6表达较对照组明显升高($P<0.01$)。美沙拉唑组、OMT组大鼠结肠黏膜NOD2、NF-κB p65蛋白、IL-6表达较模型组显著降低($P<0.01$, $P<0.05$)。结论:结肠黏膜NOD2、NF-κB p65蛋白过度表达,IL-6分泌增多参与了溃疡性结肠炎的发生发展过程,而OMT可以通过抑制NOD2、NF-κB p65蛋白过度表达,降低IL-6分泌,起到减轻结肠黏膜炎症、保护结肠黏膜的作用。

中文关键词: NOD2蛋白 NF-κB p65 TNBS诱导的结肠炎 苦参素

Study on protective mechanism of kushenin injection on colonic mucosa of experimental colitis rats

Abstract: Objective: To study the effect of NOD2 on colitis pathogenesis in experimental rats, and discuss therapeutical effect and mechanism of kushenin injection(OMT) on colitis in experimental rats. Method: Forty Sprague-Dawley(SD) rats were randomly divided into four groups: the normal control group, the model group, the SASP group, and the OMT group, with 10 rats in each group. Except the normal control group, models were established in the remaining three groups with TNBS. The OMT group was injected with kushenin injection, the SASP group was orally administered with mesalazine suspension, the model group and the normal group were orally administered with distilled water for 15 days. Colon lesion score and histological score of experimental rats were observed. Expression of NOD2, NF-κB p65 protein in rats colonic mucosa was detected by immunohistochemistry. Expression of IL-6 in rat colon mucosa was detected by ELISA. Result: Compared with normal control group, the expression of NOD2, NF-κB p65 and IL-6 in colonic mucosa of the model group were significantly increased ($P<0.01$). The SASP group and the OMT group showed lower expressions of NOD2, NF-κB p65 and IL-6 in colonic mucosa than the model group ($P<0.01$, $P<0.05$). Conclusion: The over expression of colonic mucosa proteins NOD2 and NF-κB p65 and increasing secretion of IL-6 take part in the appearance and development of ulcerative colitis. OMT can attenuate ulcerative colitis and protect colonic mucosa by inhibiting expression of NOD2, NF-κB p65 and decreasing IL-6.

keywords: NOD2 protein NF-κB p65 TNBS induced colitis kushenin

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