



中国药学杂志 » 2013, Vol. 48 » Issue (7) :521-526 DOI: 10.11669/cpj.2013.07.008

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原花青素对大鼠自身免疫性慢性非细菌性前列腺炎的治疗作用及其机制的研究

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摘要 目的 研究原花青素对前列腺炎的治疗作用及其作用机制。方法 建立慢性非细菌性前列腺炎大鼠模型, 动物随机分成对照组、模型组、原花青素高、中、低剂量组(50 、 25 、 $10\text{ mg}\cdot\text{kg}^{-1}$)。各组于第 1 、 2 、 6 、 10 天进行药物干预。光镜下观察各组前列腺组织病理学变化, 测定前列腺组织和血浆中白细胞介素-10和巨噬细胞炎性蛋白-1 α 的表达, 并测定超氧化物歧化酶活力和丙二醛水平。结果 原花青素能有效改善慢性非细菌性前列腺炎造成的病理改变, 以中、低剂量效果更佳。原花青素各剂量均能降低慢性非细菌性前列腺炎大鼠组织白细胞介素-10和巨噬细胞炎性蛋白-1 α 基因水平表达, 并降低慢性非细菌性前列腺炎大鼠血浆白细胞介素-10和巨噬细胞炎性蛋白-1 α 蛋白水平表达。原花青素各剂量均能显著降低组织丙二醛的生成, 但对超氧化物歧化酶活性没有显著影响。**结论** 原花青素具有抗慢性非细菌性前列腺炎治疗效果, 其机制可能部分与抗炎抗氧化有关。

关键词: 原花青素 慢性非细菌性前列腺炎 白细胞介素-10 巨噬细胞炎性蛋白-1 α 超氧化物歧化酶 丙二醛

Abstract: OBJECTIVE To study the therapeutic effect of procyandin (PC) on rats with chronic abacterial prostatitis (CAP) and its underlying mechanisms. METHODS The experimental rat model of autoimmune nonbacterial prostatitis was established. The animals were randomly divided into control group, model group, PC high dose, medium dose and low dose group (50 , 25 and $10\text{mg}\cdot\text{kg}^{-1}$), and received the corresponding treatment on day 1, 2, 6 and 10. A placebo normal control group was also set. HE staining was used to observe the histopathological changes in prostate. The expressions of interleukin-10 (IL-10) and macrophage inflammatory protein-1 α (MIP-1 α) in prostate tissue and plasma were detected. The activity of superoxide dismutase (SOD) and content of malondialdehyde (MDA) were observed. RESULTS The pathological change in CAP rats was improved after treating with PC, especially at medium or low dose. All the three doses of PC could reduce the gene expressions of IL-10 and MIP-1 α in prostate tissue and the protein expressions of IL-10 and MIP-1 α in serum of CAP rats. The three doses of PC all decreased the content of MDA in prostate tissue of CAP rats, and showed no significant effect on the activity of SOD in tissue or plasma. CONCLUSION PC has potent therapeutic effect for chronic abacterial prostatitis, and its mechanism of action is partly related to its anti-inflammatory and anti-oxidation effect.

Keywords: procyandin, chronic abacterial prostatitis, interleukin-10, macrophage inflammatory protein-1 α , superoxide dismutase, malondialdehyde

收稿日期: 2012-04-19;

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引用本文:

孙鑫波, 刘朝东*, 王咸钟 .原花青素对大鼠自身免疫性慢性非细菌性前列腺炎的治疗作用及其机制的研究[J] 中国药学杂志, 2013,V48(7): 521-526

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