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论著

川芎嗪对大鼠压力超负荷心肌细胞外信号调节激酶1 mRNA表达的抑制作用

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摘要 目的 观察川芎嗪(ligustrazine)对大鼠压力超负荷所致心肌肥厚时细胞外信号调节激酶1（ERK-1）mRNA表达的影响。方法 雄性SD大鼠随机分为正常对照组、假手术组、模型组及川芎嗪(25, 50及100 mg·kg^{-1}）组。后4组采用缩窄腹主动脉（AAC）造模，术后次日开始ig给药，每天1次，连续3周。给药结束后监测大鼠血流动力学指标，以左心室肥厚指数（LVHI）和左心室重/右心室重（LVW/RVW）为左心室肥厚参数，实时荧光定量PCR法检测心肌肥厚标志心房利钠因子(ANF)和ERK-1 mRNA的表达。结果 AAC术后3周，与正常及假手术组比较，模型组血流动力学指标中颈总动脉收缩压(SBP)、左心室内收缩压(LVSP)及左心室舒张末压(LVEDP)明显升高，而左心最大收缩/舒张速率(±dp/dt_{max})明显降低；左心室肥厚参数LVHI与LVW/RVW显著增加，ANF和ERK-1 mRNA表达明显上调。川芎嗪ig给药3周不影响SBP及LVSP，但显著降低LVEDP，增加±dp/dt_{max}，减轻LVHI和LVW/RVW，降低ANF和ERK-1 mRNA的表达。结论 川芎嗪能抑制大鼠压力超负荷心肌肥厚，其机制可能部分与抑制有丝分裂原激活蛋白激酶信号通路中ERK-1 mRNA的表达有关。

关键词 川芎嗪 肥厚, 左心室 腹主动脉缩窄 有丝分裂原激活蛋白激酶类 细胞外信号调节激酶

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Inhibitiory effect of ligustrazine on extracellular signal-regulated kinase-1 mRNA expression in pressure overload rat heart

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Abstract

AIM To investigate the effects of ligustrazine (Lig) on extracellular signal-regulated kinase-1 (ERK-1) mRNA expression in pressure overload rat heart. **METHODS** SD rats were randomly divided into 6 groups, 4 groups among which were accepted an operation of abdominal aorta coarctation to elicit left ventricular hypertrophy (LVH), the others were normal control and sham operation group. Lig (25, 50 and 100 mg·kg^{-1}) were administered (ig) to rats in 3 of these 4 groups, respectively, once daily, for 3 weeks. At the end of administration, hemodynamics of the rats were assessed, and the LVH index 〔left ventricular weight(LVW)/body weight〕 and LVW/right ventricular weight (RVW) were used as hypertrophic parameters; the expressions of atrial natriuretic factors (ANF) and ERK-1 mRNA were determined by iCycler iQ real-time polymerase chain reaction. **RESULTS** Compared with abdominal aorta constricted model group, Lig significantly reduced in LVHI, LVW/RVW and left ventricular end-diastolic pressure, but increased ±dp/dt_{max}. The expressions of ANF and ERK-1 mRNA were significantly suppressed by Lig. **CONCLUSION** Lig has an inhibitory effect on the left ventricular hypertrophy induced by overload pressure in rats, which may be, in partly, mediated by its inhibitory effect on expression of ERK-1 mRNA in mitogen-activated protein kinase signaling pathway.

Key words

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