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论著

信号转导及转录激活因子3在高血压左室肥厚心肌中的变化及作用

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摘要 目的：探讨信号转导及转录激活因子3（Stat3）在高血压左室肥厚心肌中的变化及作用。 方法：采用两肾一夹法建立Goldblatt大鼠高血压模型，56只健康雄性SD大鼠随机分为高血压非用药组（H组，n=11）；螺内酯治疗组（S组，n=12）；缬沙坦治疗组（V组，n=11）；螺内酯和缬沙坦联合治疗组（S+V组，n=11）；假手术组（sham组，n=10）。每周测1次尾动脉压，每两周测1次超声心动图，术后第10周处死大鼠。免疫组化检测左室心肌Stat3水平变化及c-Myc蛋白表达，放免检测左室心肌血管紧张素Ⅱ(AngⅡ)的含量。结果：术后10周Stat3活化H组明显高于sham组（P<0.01）； V组、S+V组明显低于H组（P<0.01），接近于sham组水平；S组显著高于V组、S+V组（P<0.01），与H组差别无显著（P>0.05）。Stat3活化与术后10周大鼠颈动脉压、左室重量指数及c-Myc蛋白表达呈正相关，与心肌局部AngⅡ的含量无相关性。 结论：Goldblatt大鼠左室肥厚心肌中，Stat3的活化增加，Stat3可能是参与左室肥厚发生发展的一个新的信号转导及转录激活因子；持续的压力负荷对Stat3活化起着重要的作用；缬沙坦降低压力负荷逆转左室肥厚发生的同时，抑制了Stat3活化及c-Myc蛋白表达。

关键词 高血压； 信号转导及转录激活因子3； 蛋白质c-Myc； 肾素-血管紧张素系统

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Role of Stat3 in the left hypertrophic ventricle in hypertension rats

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Abstract

<FONT face=Verdana>AIM: To investigate the role of Stat3 in the left hypertrophic ventricle in rats. METHODS: The Goldblatt model of renovascular hypertension was induced in forty-six male Sprague-Dawley rats. The rats were randomized to untreated hypertension group (group H, n=11), spironolactone treatment group (group S, n=12), valsartan treatment group (group V, n=11), spironolactone and valsartan treatment group (group S+V, n=11). Ten sham-operated rats served as the control group (group sham, n=10). The tail cuff blood pressure and echocardiogram were detected every week and every other week, respectively. After eight weeks' treatment, the rat left ventricle (LV) was collected. The concentration of angiotensin Ⅱ (AngⅡ) in LV, the activation of Stat3 and protein expression of c-myc were examined. RESULTS: Ten weeks after operation, LVH was detected in group H and group S (P<0.05) but not in group V, S+V and sham. Activation of Stat3 was significantly higher in group H and S than that in group V, S+V and sham. Activation of Stat3 was positively related to blood pressure, left ventricular mass index, and protein expression of c-myc. CONCLUSION: In the left hypertrophic ventricle of Goldblatt rats, the activation of Stat3 increases significantly. Sustained pressure overload plays an important role in the activation of Stat3. Stat3 may participate in the development and progression of cardiac hypertrophy and is probably a new activator of transcription in signal transduction of cardiac hypertrophy. </FONT>

Key words Hypertension Signal transduction and activator of transcription 3 Protein c-Myc Renin-angiotensin system

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