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论文

前胡丙素对高血压大鼠血管肥厚、细胞内钙、胶原及NO的影响

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摘要:

目的 研究前胡丙素对自发性高血压大鼠SHR及肾型高血压大鼠RHR的血管肥厚、细胞内钙、胶原、NO及血管收缩的反应性影响。方法用显微测微仪测定血管中膜层厚度,细胞大小,用Fura-2/AM为荧光指示剂,测定单细胞内 [Ca²⁺] i,以测定羟脯氨酸含量反映胶原含量,用Griess法测定NO含量,以血管环观察收缩反应。结果 前胡丙素抑制血管中膜层增厚,维持细胞内 [Ca²⁺] i稳态。减少胶原形成,增加SMCs释放NO。抑制血管环高反应状态。结论 前胡丙素抑制高血压血管肥厚,降低胶原含量及血管异常反应。

关键词: 前胡丙素 血管平滑肌肥厚 细胞内游离钙 一氧化氮

EFFECTS OF PRAERUPTORIN C ON VASCULAR HYPERTROPHY, $\lceil \text{Ca}^{2+} \rceil$ i, COLLAGEN CONTENT AND NO IN RENOVASCULAR AND SPONTANEOUSLY HYPERTENSIVE RATS

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Abstract:

AIM To study the effects of praeruptorin C (pra-C), a pure constituent isolated from "Qian-Hu", the roots of Peucedanum praeruptorum Dunn. (Umbelliferae), on vascular hypertrophy, collagen content, transient [Ca²⁺] i, NO and vascular response of the thoracic aorta of renovascular and spontaneously hypertensive rats (RHR, SHR). METHODS RHR and SHR were given pra-C 20 mg.kg⁻¹.d⁻¹ for 9 weeks, ig. Blood pressure of both rats were measured using tail cuff manometry. Under inverted microscopy the length and width of the smooth muscle cells were measured by using computer software MICC (Dongnan University). $[Ca^{2+}]$ i of smooth muscle cell (SMCs) was measured with Fura-2/AM. By measuring the specific aminoacid hydroxyproline content, the collagen content was obtained. By using Griess reagent, the NO in the smooth muscle cells (SMCs) was measured. RESULTS The intermedia of the thoracic aorta in RHR was enlarged than that of the normal and pra-C groups. The size (length×width) of the SMCs of thoracic aorta from RHR increased 73.4 µm vs nomal 34.5 µm and pra-C 34 µm. The collagen content of thoracic aorta was 39%±6.8% dry weight in RHR, they were 26.5%±3% dry weight in normal and 25.6%±1.1% dry weight in pra-C, RHR vs pra-C. The resting [Ca²⁺] i of single cell of SMCs was (62 ± 6) nmol.L⁻¹. In Hanks solution containing CaCl₂ 1 mmol.L⁻¹, the resting $[Ca^{2+}]$ i of SMCs was (150 ± 8) nmol.L⁻¹ in normal. (226 ± 11) nmol.L⁻¹ in RHR. In presence of KCl 60 mmol.L⁻¹, NE 10 μ mol.L⁻¹ 1 , ANG II 100 nmol.L $^{-1}$ and ATP 30 μ mol.L $^{-1}$ the [Ca $^{2+}$] i of SMCs were increased by 128%; 132%; 233% and 152% in RHR, respectively. The pra-C group was similar to the normal group. The resting [Ca²⁺] i of SMCs was (71±6) nmol.L⁻¹ in control of SHR, in Hanks solution containing CaCl₂ 1 mmol.L⁻¹ ¹. The resting $[Ca^{2+}]$ i of SMCs was (160 ± 8) nmol.L⁻¹ in normal, and (362 ± 18) nmol.L⁻¹ in SHR. In presence KCI 60 mmol.L⁻¹ and NE 10 μ mol.L⁻¹ the [Ca²⁺] i of SMCs were increased by 235% and 200% in SHR, respectively. Pra-C group was similar to normal group. NO of SMCs was decreased 76% in SHR, pra-C group was nearly normal. The pra-C improved vascular responses of the thoracic aorta of RHR. CONCLUSION These results indicate that pra-C improved the vacular hypertrophy by decreasing the size of SMCs cells, collagen content. SMCs $[Ca^{2+}]$ i and increasing NO production.

Keywords: vascular smooth muscle hypertroply smooth muscle cells (SMCs) $\left[\operatorname{Ca}^{2+}\right]$ i NO praeruptorin C

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