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miR-145表达状态对大鼠高血压动脉内膜增生的影

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Title: Role of miR-145 expression in arterial intimal hyperplasia of hypertension in rats

作者: 皮燕; 张莉莉; 胡子成; 黎炳护; 尹延伟; 高长越; 李敬诚
第三军医大学大坪医院野战外科研究所神经内科

Author(s): Pi Yan; Zhang Lili; Hu Zicheng; Li Binghu; Yin Yanwei; Gao Changyue; Li Jingcheng
Department of Neurology, Institute of Surgery Research, Daping Hospital, Third Military Medical University, Chongqing, 400042, China

关键词: 高血压; 血管平滑肌细胞; 内膜增生; miR-145

Keywords: hypertension; vascular smooth muscle cell; intimal hyperplasia; miR-145

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摘要: 目的 观察微小RNA-145(microRNA-145，miR-145)在高血压血管内膜增生中的作用。 方法 常规HE染色观察自发性高血压大鼠（SHR）和对照大鼠（WKY）主动脉内膜增生情况，逆转录多聚酶链反应（RT-PCR）方法检测不同病程SHR大鼠主动脉中miR-145的表达水平，并应用miR-145前体premiR-145和抑制剂2’OMe-miR-145分别升高和抑制血管平滑肌细胞（smooth muscle cell， VSMC）中miR-145的表达水平，从分化标志蛋白α平滑肌肌动蛋白（α-SMA）表达水平、VSMC的增殖和迁移能力等方面检测VSMC表型转化情况。 结果 ①SHR大鼠主动脉内膜增生，内膜/中膜面积百分比（39.7±12.1）%显著高于WKY组（3.8±1.2）%（*P*=0.001），α-SMA染色阳性的VSMC是增生内膜的主要细胞成分；②与WKY相比，SHR大鼠主动脉来源的VSMC表型转化明显增多，表现为增殖和迁移能力升高，而α-SMA表达降低（*P*<0.01）；③SHR大鼠主动脉中的miR-145水平在8周龄时开始下降（*P*=0.045 6），至18周龄时降至仅为WKY组的7.5%；④应用premiR-145可上调SHR-VSMC中α-SMA的表达（*P*=0.005 6）并促进VSMC增殖迁移过程（*P*<0.01），而2’OMe-miR-145可抑制VSMC中α-SMA的表达（*P*=0.023 2）和VSMC增殖迁移过程（*P*<0.01）。 结论 高血压时动脉中miR-145水平呈进行性下降，可能是引起VSMC表型转化并促进动脉内膜增生的一个重要机制。

Abstract: Objective To determine the effect of miR-145 on arterial intimal hyperplasia in hypertension. Methods Spontaneous hypertension rats (SHR) and

Wistar-Kyoto (WKY) rats were used in this study. Aortic intimal hyperplasia was evaluated pathologically. The expression of miR-145 in aorta of SHR was detected by reverse transcription polymerase chain reaction (RT-PCR). The phenotypic modulation of vascular smooth muscle cells (VSMCs) was assessed by detecting the expression of α-smooth muscle actin (α-SMA) and the capability of proliferation and migration in the presence of miR-145 precursor, premiR-145, or inhibitor 2′OMe-miR-145. Results SHR aortas exhibited obvious neo-intima formation which was composed primarily of VSMCs, with the ratios of intima/media significant higher than WKY rats (P=0.001). SHR-derived VSMCs exhibited marked phenotypic modulation, however, the expression of α-SMA was lower (P<0.01). The expression of miR-145 in SHR aortas was declined at the 8th week (P=0.045 6) and reduced to 7.5% of that of WKY rats at the 18th week. Expression of α-SMA and proliferative and migratory capacities of VSMCs were respectively upregulated and downregulated by premiR-145 and 2′OMe-miR-145. Conclusion The expression of miR-145 decreases progressively during hypertension, which may at least partly contribute to the excessive VSMCs phenotypic modulation and thus arterial intimal hyperplasia.

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