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Cyclooxygenase-2 pathway in reversion of myocardial fibrosis by Salvia Miltiorrhiza Bge (SMB) in senescent spontaneously hypertensive rats

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中文摘要:

目的 探讨丹参对于老年自发性高血压大鼠（SHR）心肌纤维化的逆转作用和可能的作用机制—— 花生四烯酸环氧化酶途径。方法 实验采用17个月龄的SHR分别连续给予12周的丹参注射液和生理盐水对照, 选取同周龄的WKY大鼠作为正常对照, 同时选用花生四烯酸环氧化酶-2（COX-2）的特异性抑制剂塞来昔布灌胃处理后注射丹参。测定血压、血流动力学、左心室质量指数, HE染色和Masson染色分析心肌细胞的大小或心肌纤维化水平, 左心室心肌COX-2的蛋白表达和活性水平。结果 与老年WKY大鼠相比, 老年SHR高血压对照组血压显著增高[收缩压:（171±12）vs（125±3）mmHg; 舒张压:（115±9）vs（87±3）mmHg; 平均动脉压:（139±10）vs（106±5）mmHg; P＜0.05], 心功能恶化[LVEDP:（13.9±1.7）vs（7.6±1.3）mmHg; LVdP/dtmax:（2528±167）vs（3015±217）mmHg/s; －LVdP/dtmax:（1957±134）vs（2501±175）mmHg/s, P＜0.05], 左室质量指数增加[（3.45±0.07）vs（2.23±0.06）mg/g, P＜0.05], 心肌细胞增大[心肌细胞直径:（23.5±0.4）vs（14.3±0.4）μm, P＜0.05], 纤维化严重[心肌间质纤维化指数:（1.66±0.05）% vs（0.64±0.05）%; 心肌血管周围纤维化指数:（139±9）% vs（68±7）%, P＜0.05], 心肌COX-2的蛋白表达水平增加[（125.8±7.2）vs（47.6±3.8）, P＜0.05]以及活性增强[（73.9±5.6）vs（56.7±4.4）kU/g, P＜0.05]。应用丹参的SHR组, 收缩压无显著变化, 其他指标均有显著下降（P < 0.05）, 塞来昔布能够部分消除丹参的作用。结论 丹参能显著逆转老年SHR的心肌纤维化水平, 该作用可能是通过花生四烯酸环氧化酶途径。

英文摘要:

Objective To explore the roles of Salvia Miltiorrhiza Bge (SMB) in improving myocardial fibrosis in aged spontaneously hypertensive rats and the possible mechanisms —— cyclooxygenase pathway of arachidonic acid. Methods The 17-month-old SHR rats were given consecutive SMB injection or saline control for 12 weeks, while the age-matched WKY rats were selected as normal control. Meanwhile, celecoxib, the specified arachidonic acid cyclooxygenase-2 (COX-2) inhibitor, was administrated intragastrically before SMB injection. The blood pressure, hemodynamic index and left ventricular mass index were determined. HE staining and Masson staining were performed to analyze the size of myocardial cells or the level of myocardial fibrosis, as well as left ventricular myocardial expression and activity level of COX-2 protein. Results Compared with the elderly WKY rats, senescent SHR had higher blood pressure[systolic blood pressure: (171±12) vs (125±3)mmHg; diastolic blood pressure: (115±9) vs (87±3)mmHg; mean artery pressure: (139±10) vs (106±5)mmHg , P＜0.05], deterious heart function[LVEDP: (13.9±1.7) vs (7.6±1.3)mmHg; LVdP/dtmax: (2528±167) vs (3015±217)mmHg/s; ?LVdP/dtmax: (1957±134) vs (2501±175)mmHg/s, P＜0.05], increased left ventricular mass index[(3.45±0.07) vs (2.23±0.06)mg/g, P＜0.05], increased myocardial cells[diameter: (23.5±0.4) vs (14.3±0.4)μm, P＜0.05], severe fibrosis[myocardial interstitial fibrosis index: (1.66±0.05)% vs (0.64±0.05)%; myocardial perivascular fibrosis index: (139±9)% vs (68±7)%, P＜0.05], elevated expression[(125.8±7.2) vs (47.6±3.8), P＜0.05] and activity level of myocardial COX-2 protein[(73.9±5.6) vs (56.7±4.4)kU/g, P＜0.05]. Except the systolic blood pressure, other indicators in SMB-treated SHR were significantly reduced. The administration of celecoxib partially abolished the effect of SMB. Conclusion SMB therapy can reverse myocardial fibrosis in senescent spontaneously hypertensive rats, which may be through cyclooxygenase pathway.

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