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血脂康对自发性高血压大鼠心肌重构的作用及其可能机制

Effects of Xuezhikang on Ventricular Remodeling in Spontaneously Hypertensive Rats and the Possible Mechanism

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中文摘要：

目的 观察血脂康(Xuezhikang，XZK)对自发性高血压大鼠(spontaneously hypertensive rats，SHR)左室肥厚和心肌间质纤维化的影响，探究其可能机制。方法 30只SHR，♂，随机分为3组，血脂康(300 mg·kg^{-1}·d^{-1})治疗组(XZK组，n=10)，辛伐他汀(5 mg·kg^{-1}·d^{-1})治疗组(SIM组，n=10)，SHR对照组(SHR组，n=10)，同时入组同龄Wistar-Kyoto大鼠为正常对照组(WKY组，n=10)，均予等容量0.9%生理盐水灌胃。12周后，分离心脏，左室称重并计算左室重量指数(LVWI)，心肌组织固定包埋后Masson染色并计算心肌胶原容积分数(CVF)及心肌血管周围胶原面积(PVCA)，测定血清Ⅰ型前胶原羧基末端前肽(PⅠCP)浓度、心肌组织超氧歧化酶(SOD)活性及丙二醛(MDA)浓度。结果 与SHR组相比，XZK组和SIM组可以显著降低LVWI(P<0.05)、CVF(P<0.01)、PVCA (P<0.01)及血清PⅠCP浓度(P<0.01)，但SOD活力及MDA浓度差异无统计学意义。结论 血脂康能够显著减轻SHR的左室肥厚，降低心肌间质纤维化程度，并与血压和胆固醇水平的变化无关，血脂康表现出与辛伐他汀相同的抗心肌重构作用，作用机制可能与心肌SOD活性和MDA浓度相关不大。

英文摘要：

OBJECTIVE To observe the effects of Xuezhikang on left ventricular hypertrophy and myocardial interstitial fibrosis in spontaneously hypertensive rats (SHR) and the possible mechanism. METHODS Thirty male SHRs were randomly treated with Xuezhikang (300 mg·kg^{-1}·d^{-1}) (XZK group, n=10), simvastatin (5 mg·kg^{-1}·d^{-1}) (SIM group, n=10) or 0.9% normal saline (SHR group, n=10). Age-matched Wistar-Kyoto rats (WKY) (WKY group, n=10) were also gavaged by 0.9% normal saline for 12 weeks. Left ventricular weight index (LVWI) was calculated. In addition, the serum lipid concentrations, the blood pressure levels, the myocardial collagen volume fraction (CVF) and perivascular collagen area (PVCA), the serum levels of procollagen type Ⅰ C-terminal peptide (PⅠCP), the levels of myocardial superoxide dismutase (SOD) and malondialdehyde (MDA) were also measured in this study. RESULTS After 12 weeks of treatment, the LVWI (P<0.05), CVF (P<0.01), PVCA (P<0.01) and serum PⅠCP concentrations (P<0.01) of XZK group and SIM group were significantly lower than those of SHR group. Myocardial SOD activity and MDA concentrations had no significant changes among four groups. CONCLUSION This study demonstrats that Xuezhikang significantly inhibited left ventricular hypertrophy and myocardial interstitial fibrosis was independent of its antihypercholesterolemic role and exhibited the same effects as simvastatin. The levels of myocardial SOD activity and MDA concentrations seem to be uninvolved in the reverse effects of Xuezhikang.

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