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Left ventricular hypertrophy does not prevent heart failure in experimental hypertension.

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Abstract

BACKGROUND:

Left ventricular hypertrophy (LVH) secondary to hypertension has been accepted to prevent heart failure (HF) while paradoxically increasing cardiovascular morbi-mortality.

OBJECTIVES:

To evaluate whether antihypertensive treatment inhibits LVH, restores beta-adrenergic response and affects myocardial oxidative metabolism.

METHODS:

Ninety spontaneously hypertensive rats (SHR) were distributed into groups and treated (mg/kg, p.o.) with: losartan 30 (L), hydralazine 11 (H), rosuvastatin 10 (R), carvedilol 20 (C). Hypertension control group comprised 18 normotensive rats (Wistar-Kyoto, WKY). Following euthanasia at 16months, contractility was measured in 50% of rats (Langendorff system) before and after isoproterenol (Iso) 10^{-9}M, 10^{-7}M and 10^{-5}M stimulation. Left ventricular weight (LVW) was measured in the remaining hearts, and normalized by BW. Expression of thioredoxin 1 (Trx-1), peroxyredoxin 2 (Prx-2), glutaredoxin 3 (Grx-3), caspase-3 and brain natriuretic peptide (BNP) was determined.

RESULTS:

Systolic blood pressure (mmHg): 154±3 (L), 137±1 (H), 190±3 (R)*, 206±3 (SHR)*, 183±1 (C)**, and 141±1 (WKY) (*$p<0.05$ vs. L, H, WKY, **$p<0.05$ vs. L, H, WKY, SHR). LVW/BW was higher in SHR and R ($p<0.05$). Groups SHR, R and C evidenced baseline contractile depression. Response to Iso 10^{-5}M was similar in WKY and L. Expression of Trx-1, Prx-2 and Grx-3 increased in C, H, R and L ($p<0.01$).

CONCLUSIONS:

Present findings argue against the traditional idea and support that LVH might not be required to prevent HF. Increased expression of thioredoxins by antihypertensive treatment might be involved in protection from HF.

KEYWORDS:

BNP; Carvedilol; Caspase-3; Grx-3; Heart failure; Hydralazine; Left ventricular hypertrophy; Losartan; Oxidative stress; Prx-2; Rosuvastatin; Trx-1

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