Comparative Study between the Effects of Angiotensin-Converting Enzyme Inhibitors and Beta Blockers on Left Ventricular Function and Geometry in Mild Hypertensive Patients

Thesis

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Abstract

Background*:** Hypertension is a leading non-communicable disease and the most prevalent cardiovascular disease. Adaptation of the left ventricle to systemic hypertension is complex and it is characterized by functional and structural changes. Increased LV mass is a recognized complication of systemic hypertension and has been shown to be an important predictor of cardiovascular morbidity and mortality. The current study was undertaken to determine whether antihypertensive therapy reduce LVM, and whether these changes are accompanied by improved diastolic function. ***Methods: 60 patients (pts.) with mild hypertension were divided into two groups: Group (I) included 30 pts. who received ACE inhibitors; 15 pts. received Ramipril 5-10 mg once daily, and constituted subgroup Ia, and 15 pts. received Fosinopril 20 mg once daily, and constituted subgroup Ib. Group (II) included 30 pts.who received beta blockers (BB); 15 pts.received Atenolol 50-100 mg once daily, and constituted subgroup IIa, and 15 pts. received Metoprolol 100 mg once daily, and constituted subgroup IIb. Twenty healthy volunteers comprised a control group. To assess the effects of antihypertensive therapy on the heart; LVM, systolic and diastolic function, by M-mode, 2-D and pulsed wave Doppler echocardiography were evaluated. Echocardiographic examinations were performed before therapy, after 2 weeks from the start of therapy and at 2, 4 and 6 months intervals. ***Results**:* At baseline, there were 13 pts. with LVH in a ratio 21.6% of total hypertensive pts. while the rest of the hypertensive pts., 47 pts (78.4%), had normal geometric pattern. The concentric LVH was the predominant form (53.8%) followed by eccentric LVH (30.7%) and finally concentric remodeling (15.3%). Indeces of systolic function in studied pts. were normal while indeces of LV diastolic filling were significantly abnormal in pts. with hypertensive LVH compared to control. After therapy, normalization of blood pressure was achieved in all hypertensive patients. A significant reduction of LVM, RWT and improvement of LV diastolic filling was observed earlier with ACE inhibitors than with beta-blockers. Of particular interest was the fact that improvement in diastolic performance appeared earlier, and was preceded regression of LVM. ***Conclusion**:* 1-Therapy with ACEI and BB results in prevention/ regression of development of LVH in hypertensive patients. 2-The regression of LVM, LVMI, RWT and the improvement of LV diastolic function appears more evident and earlier with ACEI therapy than with BB therapy. 3-Improvement of LV diastolic function appears earlier, preceding the regression of LVH.

Key words: hypertension + LVH + ACE inhibitors +beta blockers.

Content

List of abbreviation

% FS:	Percent fractional shortening.
ABPM:	Ambulatory blood pressure monitoring.
ACE:	Angiotensin-converting enzyme.
AII:	Angiotensin II.
AR :	Pulmonary venous peak atrial contraction reversed velocity.
ASE:	American Society of echocardiography.
AT1RBAs:	Angiotensin II type I receptor blocking agents.
A-velocity:	Peak-filling velocity in atrial systolic phase.
AVP:	Arginine vasopressin.
BB:	Beta Blockers.
BP:	Blood pressure.
CHD:	Coronary heart disease.
CHF:	Congestive heart failure.
CNS:	Central nervous system.
CO:	Cardiac output.
CT:	Computed axial tomography.
CV:	Cardiovascular.
DT :	Early left ventricular filling deceleration time.

E/A : The ratio of early to late peak filling velocity.

ECG: Electrocardiogram.

ECX: Extracellular matrix.

EDRF: Endothelium-derived relaxing factor.

EDV: End diastolic volume.

EF: Ejection fraction.

EPSS: E-Point septal separation.

ESV: End systolic volume.

E-Velocity: Early peak filling velocity.

HR: Heart rate.

IVRT: Isovolumic relaxation time.

IVSd: Interventricular septal thickness at end diastole.

IVSs: Interventricular septal thickness at end systole.

JNC: Joint National Committee.

LV: Left ventricle.

LVH: Left ventricular hypertrophy.

LVIDd: Left ventricular internal dimension diastole.

LVIDs: Left ventricular internal dimension at systole.

LVM: Left ventricular mass.

LVMI: Left ventricular mass index.

MI: Myocardial infarction.

MRI: Magnetic resonance imaging.

NHP: Egyptian National Hypertension Project.

NO: Nitric Oxide.

NYHA: New York Heart Association.

PNS: Peripheral nervous system.

PVR: Peripheral vascular resistance.

PWTd: Posterior wall thickness at diastole.

PWTs: Posterior wall thickness at systole.

RAAS: Renin-angiotensin-aldosterone system.

RWT: Relative wall thickness.

S/D: Systolic-to-diastolic pulmonary venous flow ratio.

SPECT: Single-photon emission computed tomography.

TOD: Target organ damage.

WHO-ISH: World Health Organization-International Society of Hypertension.

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INTRODUCTION

Hypertension is a leading non-communicable disease. It has been recognized as the most prevalent cardiovascular disease. Adaptation of the left ventricle to systemic hypertension is complex and it is characterized by functional and structural changes in the left ventricle (Casale et al., 1986, Levy et al., 1990). Hypertension may result in left ventricular hypertrophy (LVH) and impairment of diastolic and systolic function (Cuocolo et al., 1990). At some point in the natural history of hypertension, the compensatory increase in LVM ceases to be beneficial and becomes a preclinical disease and an independent risk factor for congestive heart failure, ischemic heart disease, arrhythmia, sudden death, and stroke. Left ventricular hypertrophy is associated with adverse heart consequences, including reduction in systolic and diastolic function, reduced coronary reserve, an increased incidence of arrhythmias (Frohlich et al., 1999, Agabiti-Rosei et al., 2006).

Hypertension is known to be associated with a wide spectrum of LV geometric patterns (**Zabalgoitia et al., 2001**). The hemodynamic predominance between pressure and volume overload plays an important role in the determination and development of various LV geometric patterns. Concentric remodeling tends to occur early in hypertension due to pressure overload but the left ventricular mass is normal while eccentric hypertrophy is due to volume overload with increased left ventricular mass. Various geometric patterns are also influenced significantly by LV systolic function parameters **(de Simone et al., 1999**). Persistent pressure overload in concentric LV geometry with