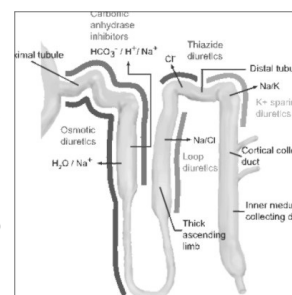


ORIGINAL

(CLINICAL PRACTICE ARTICLE)

PROF-790

REGRESSION OF LEFT VENTRICULAR HYPERTROPHY (LVH); COMPARISON BETWEEN ACE INHIBITORS & DIURETICS



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ABSTRACT ... Left ventricular hypertrophy is an independent risk factor for cardiovascular diseases. Therefore optimal anti-hypertensive therapy should provide the regression of LVH. There are certain group of drugs which cause regression of LVH, such as angiotensin converting enzyme inhibitors and diuretics. **Objectives:** To assess the magnitude regression induced by captopril and indapamide when patients are treated for equal duration of time. **Patients & Methods:** We gave captopril (25–150 mg) to eleven patients with left ventricular hypertrophy for six months and another eight patients, indapamide 2.5 mg (Diuretic) was given for six months. **Results:** There was an overall reduction of left ventricular mass index (LVMI) by 12.5% in patients taking captopril, while left ventricular mass index (LVMI) decreased by 7.5% in patients taking indapamide. The LVMI decreased from 157.2 ± 12.6 to $137.5 \pm 8.7 \text{g/m}^2$ (Mean \pm SEM) and from 156.1 ± 9.5 to $141.3 \pm 8.5 \text{g/m}^2$ (Mean \pm SEM) in patients taking captopril and indapamide respectively. The mean blood pressure in both the groups decreased, systolic blood pressure decreased from 172 to 146 mm Hg and diastolic blood pressure from 101 to 82 mm Hg. **Conclusions:** ACE inhibitors induce more regression than diuretics when patients are treated for equal duration of time.

Key words: LVH, Captopril, Indapamide, Regression, LVMI.

INTRODUCTION

The hypertensive patients are prone to develop left ventricular hypertrophy. Left ventricular hypertrophy is associated with congestive cardiac failure², coronary heart disease¹, extrasystole, ventricular coupletes and ventricular tachycardia¹¹. The Frahingham study has demonstrated the association between left ventricular hypertrophy, angina, myocardial infarction and sudden death¹⁰.

The reversal of left ventricular hypertrophy is associated with improvement of systolic as well as diastolic function¹⁸. A possible improvement of coronary flow reserve has been associated with regression of left ventricular hypertrophy. The other complications such as dysrhythmias also decrease with regression of left ventricular hypertrophy. Antihypertensive agents that modulate the renin angiotensin aldosterone system, sympathetic drive or intracellular free calcium concentration, angiotensin converting enzyme inhibitors,

β blockers, centrally acting adrenergic drugs and calcium antagonist lead to regression³.

ACE inhibitors (Captopril) and Indapamide (diuretic) cause regression. This study was carried out to assess the degree of regression.

SUBJECTS & METHODS

Nineteen hypertensive patients were enrolled for study after informed consent was taken from them. (Eleven men and eight women aged 50 ± 10 years ranging from 36 to 66 years). All were diagnosed as having essential hypertension on the basis of seated diastolic blood pressure of between 95 mm Hg to 115 mm Hg in triplicate measurements over a period of three weeks. Eleven patients took captopril in the range of 25 to 150mg/per day in divided doses while eight patients were prescribed indapamide 2.5mg once daily for six months. Their diastolic blood pressures were maintained less than 95 mm Hg.

Patients with myocardial infarction, severe renal or hepatic function impairment, patients taking neuroleptics, corticosteroids, phenylbutazone and antidepressants were excluded from the study. M-mode echocardiogram was performed by using standard 3.5MHz transducer with Toshiba sonolayer-SAL-38 as echocardiograph.

The patients were kept in left lateral decubitus position which is standard echocardiographic technique. The transducer was placed parasternally in the 3rd and 4th intercostal space. The echocardiogram was taken on day 0 and after six months of treatment. The following parameters were obtained from the echocardiogram.

- * Interventricular septal thickness (mm) IVST.
- * Posterior wall thickness (mm) PWT.
- * Left ventricular internal diameter.

The left ventricular mass index was calculated by dividing left ventricular mass by body surface area (in square meters). The left ventricular mass was calculated as under; $1.04 \times \{[\text{diastolic LV diameter} + \text{septal Thickness} + \text{PWT}]^3 - [\text{Diastolic LV diameter}]^3\}$.

RESULTS AND DISCUSSION

Indapamide, a sulphonamide derivative is an effective antihypertensive agent which causes regression of VH^{3,6,11,15,17}. The regression in our study caused by indapamide was LVMI decreased from 156.1 ± 9.5 to $141.1 \pm 8.5 \text{g/m}^2$ (Mean \pm SEM).

The posterior wall thickness decreased from 12 ± 1.7 to 11.4 ± 2.1 mm (mean \pm SEM). Inter septal thickness decreased from 11.5 ± 1.9 to 10.5 ± 1.8 mm (mean \pm SEM). The left ventricular Internal diameter decreased from 51 ± 3.8 to 50.8 ± 3.1 mm (mean \pm SEM). The overall regression induced by indapamide was 7.5%. While patients treated with captopril, the overall regression was of 12.3% in left ventricular mass index. The posterior wall thickness decreased from 11 ± 2.1 to 10.1 ± 2.5 mm (mean \pm SEM). The IVST and left ventricular internal diameter decreased from 12.6 ± 2.3 to 11.2 ± 1.5 mm (mean \pm SEM) and 5.0 ± 3.6 to 49.7 ± 2.08 mm (mean \pm SEM) respectively. The regression was mainly due to regression in posterior wall thickness and Interventricular septal thickness. It was not due to decrease in LVID. The process of hypertrophy is accentuated in some patients.

The effect of antihypertensive drugs (captopril) and indapamide showed a decrease in mean systolic blood pressure in both the groups from 172 ± 8.6 to 146 ± 5.8 mm Hg (mean \pm SEM) and mean diastolic blood pressure from 101 ± 6.5 to 82 ± 4.8 mm Hg. The diastolic blood pressure was maintained below 95 mm of Hg.

Table-I. Echo cardiographic parameters at baseline and after 6 months captopril treatment (n = 11)

Baseline		After 6 months
LVID (mm)	5.0 ± 3.6	49.7 ± 2.08
PWT (mm)	11 ± 2.1	10.1 ± 2.5
IVST (mm)	12.6 ± 2.3	11.2 ± 1.5
<i>LVID = Left Ventricular Internal Diameter in diastole</i> <i>PWT = Posterior Wall Thickness</i> <i>IVST = Inter Ventricular Septal Thickness.</i> \pm = Standard error of mean.		

LVH is the result of cardiac compensation for the excessive work load imposed by hypertension. The regression of LVH is as important as reduction of blood pressure in hypertensive patients.

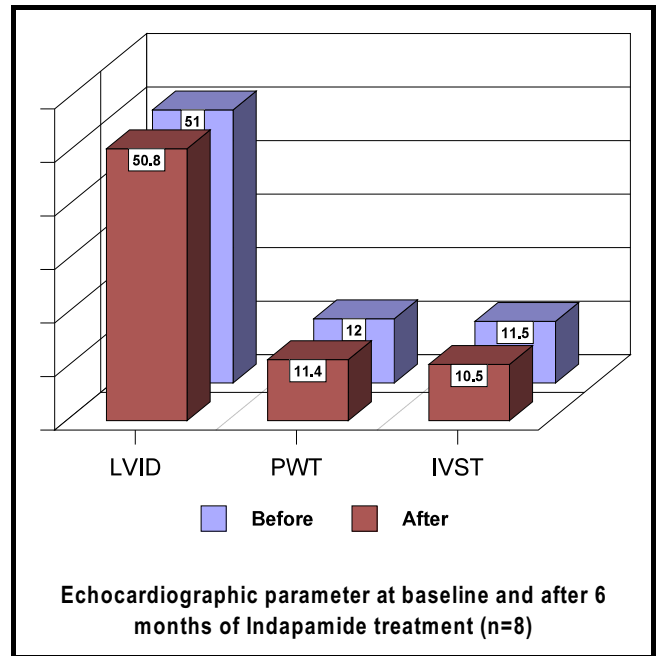
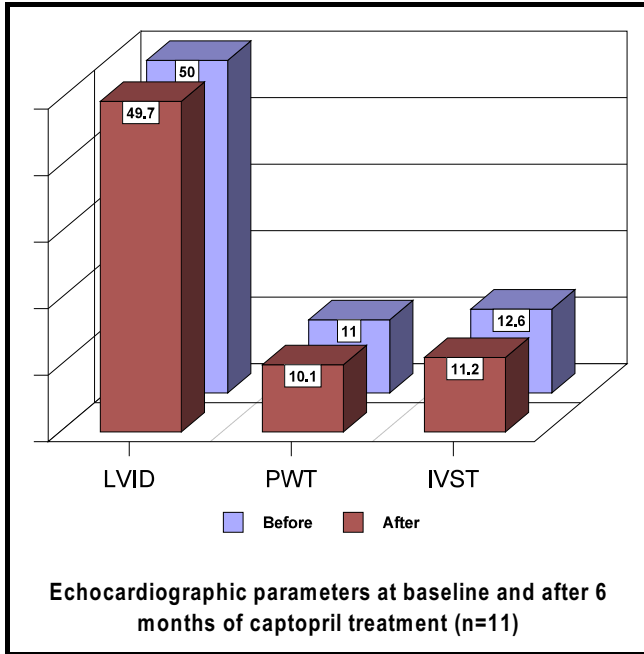


Table-II. Echo cardiographic parameter at baseline and after 6 months of Indapamide treatment (n = 8)

Baseline	After 6 months	
LVID (mm)	51±3.8	50.8±3.1
PWT (mm)	12.0±1.7	11.4±2.1
IVST (mm)	11.5±1.9	10.5±1.8

LVID = Left Ventricular Internal Diameter in diastole
 PWT = Posterior Wall Thickness
 IVST = Inter Ventricular Septal Thickness.
 ± = Standard error of mean.

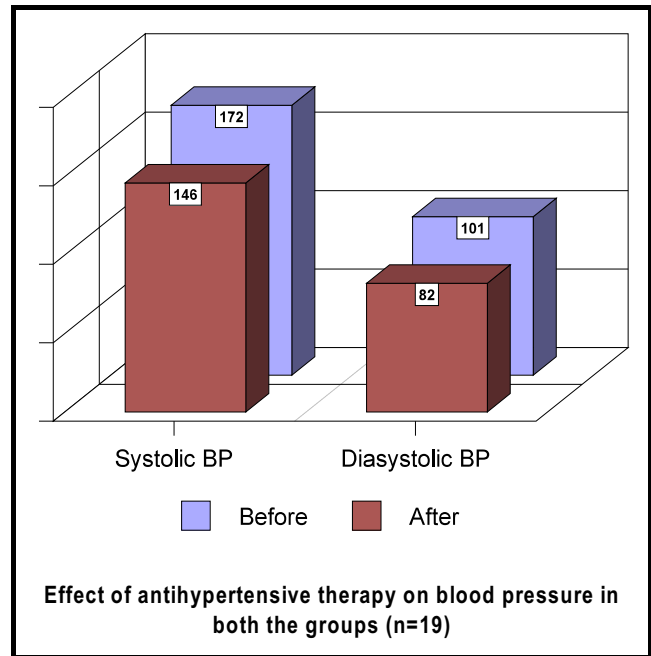


Table-III. Effect of antihypertensive therapy on blood pressure in both the groups (n = 19).

Before treatment	After treatment	
Mean Systolic B. P.	172±8.6	146±5.8
Mean diastolic B. P	101±6.5	82±4.8

mean ± standard error of mean.

The regression of left ventricular hypertrophy is beneficial for cardiac pump performance and it decreases the complication associated with left ventricular hypertrophy. Certain group of drugs cause regression of left ventricular hypertrophy such as angiotensin converting enzyme inhibitors and diuretic indapamide. After reversal of left

ventricular hypertrophy, ventricular contractions and systolic and diastolic blood pressures are improved¹⁸.

CONCLUSION

ACE Inhibitors induce more regression than indapamide (Diuretics) when patients are treated for equal duration of time.

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