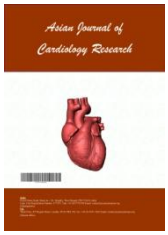

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Impact of Central Aortic Pressures on Left Ventricular Mass Index

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Original Research Article

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ABSTRACT

Aims: To measure central aortic pressures using Brachial cuff pulse volume plethysmography (PVP) and left ventricular mass index by echocardiography using standard DEVEREUX & REICHEK CUBE and BSA in hypertensive patients. To correlate central aortic and brachial cuff pressures with the left ventricular mass in hypertensive patients.
Study Design: cross-sectional, observational study.
Place and Duration of Study: From September 2018 to July 2020 at G Kuppuswamy Naidu Memorial Hospital, Coimbattore, Chennai.
Methodology: A total of 228 patients were included in the study, their written informed consent were obtained. With the help of a structured questionnaire basic demographic profile of the systemic hypertension patients such as Age, Gender, Family history of hypertension/CAD , smoking, duration of hypertension, number of hypertensive medications patient is on and other comorbid conditions, electrocardiogram, previous Echocardiogram if available were recorded. Newly detected hypertensives satisfying AHA/ACC definition and hypertensives fulfilling inclusion criteria were included. Patients who are satisfying inclusion criteria were subjected for measurement of Central aortic blood pressures, peripheral blood pressures, mean pulse wave velocity & augmentation index measured using non-invasive brachial oscillometry with a Mobil-O-Graph PWA device -ARC slover (IEM GmbH).Using Philips EPIQ 7 Cardiac ultrasound machine baseline transthoracic echocardiogram, Left ventricular mass index assessment by a linear method

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using DEVEREUX & REICHEK CUBE formula of patients was recorded. Data compiled and the statistical analysis done.

Results: We analysed 228 selected hypertensive subjects, and our observations were increased age (> 50 years), smoking, increased duration of hypertension (> 2 years), obesity, very high central Aortic Systolic, and Diastolic Pressure, high Mean Pulse Wave Velocity, and patients on Non (CCB + ACEI + ARB) group of hypertensive drugs were found to be an independent predictor for abnormal LVMI in hypertensive patients, which could be an early marker of myocardial dysfunction, major cardiovascular events and death. Central Aortic pressure (aortic systolic and diastolic pressure) was found to be the strongest predictor of abnormal LVMI among hypertensive patients in our study followed by Mean Pulse Wave Velocity.

Conclusion: Elevated central aortic pressures measured non-invasively by brachial cuff pulse volume plethysmography in hypertensive individuals are associated and statistically correlated with an increase in LVMI.

Keywords: Central Aortic Pressures (CAP); Left Ventricular Mass (LVM); Mean Pulse Wave Velocity (aPWV); Left ventricular mass index (LVMI); Augmentation index; Hypertension.

1. INTRODUCTION

Hypertension is the leading cause of death and disability worldwide [1,2]. The use of a sphygmomanometer for measuring brachial artery pressures was introduced into medical practice well over ten decades ago, which enabled the routine non-invasive measurement of arterial blood pressure. Data from RCTs over the last fifty years demonstrates that lowering blood pressure in hypertensive individuals, reduces major cardiovascular events [1,3] substantially. It's well known that systolic pressure varies throughout the arterial tree, such as the measured central aortic pressure (CAP) is lower than the corresponding brachial values, although this difference has high variability between individuals [1,2,4,5]. Over the last 5 decades, studies have clearly shown that CAP compared to brachial cuff pressures is a better predictor of cardiovascular events [6,7] and it responds differently to certain drugs [8,9]. CAP acts as a key factor over hemodynamic changes that affect myocardial muscle [6,10]. With the advent of new applanation tonometer's & brachial cuff pulse volume plethysmography, central aortic pressures (CAP) can now be assessed non-invasively with the same ease as brachial pressures [11]. CAP is derived using peripheral pulse waveforms typically of brachial or radial arteries [12,13]. CAP is more closely correlated with the wide accepted surrogate of left ventricular mass (LVM) [14,15] and carotid intimal medial thickness [16]. Hypertrophy of the left ventricle can be a marker for vascular injury in hypertensives[17]. Studies have shown that regression of LVM was more strongly related to change in central pressures when compared with brachial pressures [14,15]. Several longitudinal observations provide greater support for the potential value of CAP measurement and its impact on LVM as well as hypertension drug optimization [14,15].

2. METHODOLOGY

Based on inclusion and exclusion criteria, about 228 patients were included in the study. Written informed consent from the eligible patients or their legal representatives were obtained, the data of the patients were collected with the help of a structured questionnaire, which was filled up after interviewing the patients or his/her family members. Basic demographic profile of the systemic hypertension patients such as Age, Gender, Family history of hypertension/CAD, smoking, duration of hypertension, number of hypertensive medications patient is on, and other comorbid conditions were recorded. Previous blood pressure recordings, Electrocardiogram, previous Echocardiogram if available, were recorded. Newly detected hypertensives satisfying AHA/ACC definition and hypertensives fulfilling criteria were included. Patients who are satisfying inclusion criteria were subjected for measurement of Central aortic blood pressures, peripheral blood pressures, mean pulse wave velocity & augmentation index measured using noninvasive brachial oscillometry with a Mobil-O-Graph PWA device -ARC slover (IEM GmbH). Using Philips EPIQ 7 Cardiac ultrasound machine, baseline transthoracic echocardiogram of patients was recorded. Left ventricular mass index assessment by a linear method using DEVEREUX & REICHEK CUBE formula and Body surface area were measured [16]. The formula used for estimation of LVM from LV linear dimensions is based on modeling the LV

as a prolate ellipse, assumed that the major/minor axis ratio is 2: 1. The upper limits of normal ranges in the ASE chamber quantification update are >95 g/m^2 in women and >115 g/m^2 in men [18]. Data compiled and the statistical analysis was done [19].

3. RESULTS AND DISCUSSIONS

We analysed 228 selected hypertensive subjects who were divided into two major groups, group-1 includes individuals with normal LV mass and group-2 includes persons with increased LV mass. Group -1 had 163 participants (71.49%) with a mean of 89.12 g/m^2 and Group-2 had 65 participants (28.51%) with a mean of 126.09 g/m^2. Group-2 was subdivided based on outcome variable- based on the European Association of Cardiovascular Imageing classification: Mildly abnormal (n=30, 46.15%), Moderately abnormal (n=20, 30.77%) and Severely abnormal (n=15, 23.08%).

The age distribution analysis showed that in Group -1 the majority were in the 51-60 years age group (n=58, 35.58%) followed by the 41-50 years age group (n=48, 29.45%) with a mean age of 51.32 years. In Group -2 the majority of the subjects belonged to the 51-60 years age group (n20, 30.77%) followed by the 41-50 years age group (n=19, 29.23%) with a mean age of 56.88 years. The association between age distribution among study groups is considered to be statistically significant. In group -2 as patient age increases the LVMI also increases, moderately correlated with age (r = 0.512, P< 0.004). Males were predominant in both groups with no statistical significance related to gender. Incidence of premature CAD, diabetes mellitus, dyslipidemia, pulse rate, hemodynamic parameters like peripheral SBP, peripheral DBP, and Augmentation Index showed no significant difference and effects on outcomes. The mean LVMI distribution was seen to be less among non-smokers compared to smokers (92.62 vs 105.64). The association between smoking status and incidental increase in LVMI was noted with statistical significance.

The majority of Group -1 subjects belonged to less than 2 years duration of hypertension (n=102, 50.25%) with a mean duration of hypertension of 4.58 years and Group-2 subjects belonged to 2-5 years duration of hypertension (n=46, 50.55%) with a mean duration of hypertension of 6.26 years. Increase in LVMI was positively correlated with duration of hypertension (r = 0.694, P< 0.05). The majority of Group 1 subjects belonged to the overweight class interval (n=85, 52.15%) and the majority of Group -2 subjects belonged overweight and obese class interval (n=27, 41.54%). The increase in LVMI was positively but moderately correlated with BMI (r = 0.576, P< 0.044).

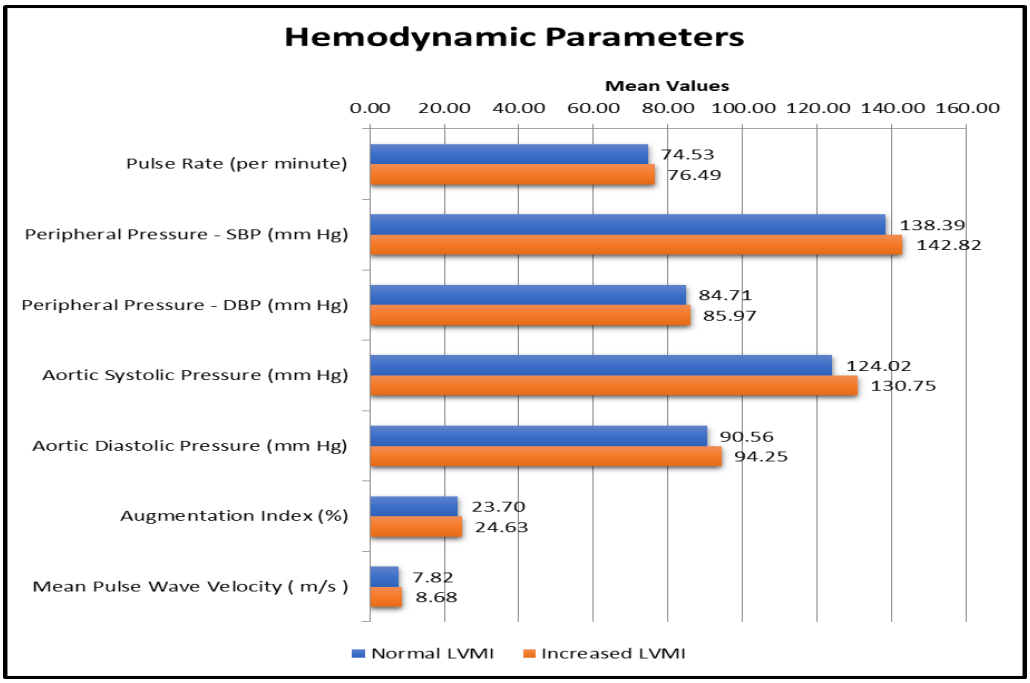

Fig. 1. Correlation of hemodynamic parameters

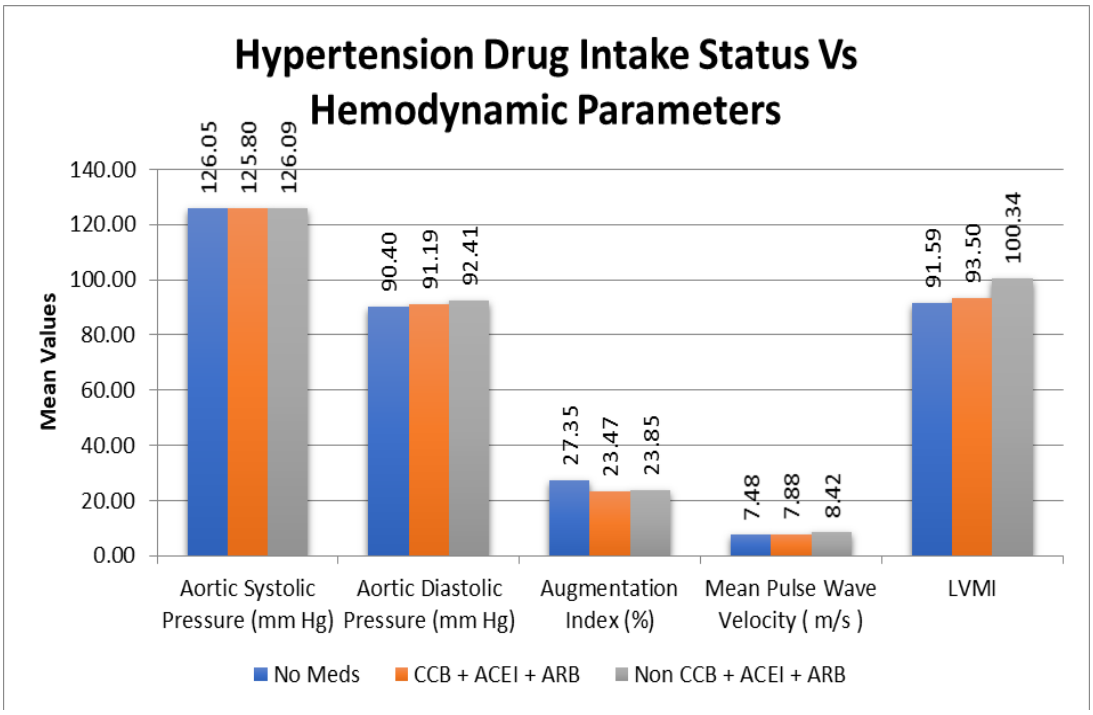

Fig. 2. Correlation of hypertension drug intake with hemodynamic parameters

It was observed that the mean values were correlating with central Aortic Systolic Pressure (124.02 vs 130.75), central Aortic Diastolic Pressure (90.56 vs 94.25), and Mean Pulse Wave Velocity (7.82 vs 8.68).

(Fig. 1 & 2). There was an association between LVMI and central Aortic Systolic Pressure /Aortic Diastolic Pressure/ Mean Pulse Wave Velocity among study groups is considered to be statistically significant. Comparing the different hypertension drug intake groups with key hemodynamic parameters showed that the mean values were smaller in no medicines group when compared CCB + ACEI + ARB group which was smaller than non CCB + ACEI + ARB group concerning Aortic Systolic Pressure (126.05 vs 125.80 vs 126.09), Aortic Diastolic Pressure (90.40 vs 91.19 vs 92.41) and Augmentation Index (27.35 vs 23.47 vs 23.85). There was no association observed between hypertensive medications and the Aortic Systolic Pressure/ Aortic Diastolic Pressure/Augmentation Index. The mean values were smaller in no medicines group when compared to CCB + ACEI + ARB group which was smaller than non CCB + ACEI + ARB group in relation to Mean Pulse Wave Velocity (7.48 vs 7.88 vs 8.42) and LVMI (91.59 vs 93.50 vs 100.34). It was observed that there was an association between hypertensive medications and mean pulse wave velocity, LVMI among the study groups with statistical significance. The LVMI was positively but strongly correlating with central aortic systolic pressure ($r = 0.727$, $P < 0.001$) & central Aortic Diastolic Pressure ($r = 0.673$, $P < 0.001$). The LVMI was positively but very poorly correlating with Augmentation Index ($r = 0.172$, $P < 0.281$). The LVMI was positively but highly correlating with Mean aortic Pulse Wave Velocity ($r = 0.699$, $P < 0.001$), Table 1.

In summary, increased age (> 50 years), smoking, increased duration of hypertension (> 2 years), obesity, very high central Aortic Systolic, and Diastolic Pressure, high Mean Pulse Wave Velocity, and patients on Non (CCB + ACEI + ARB) group of hypertensive drugs were found to be an independent predictor for abnormal LVMI in hypertensive patients, which could be an early marker of myocardial dysfunction, major cardiovascular events and death. central Aortic pressure (aortic systolic and diastolic pressure) was found to be the strongest predictor of abnormal LVMI among hypertensive patients in our study followed by Mean Pulse Wave Velocity.

Table 1. Multivariable analysis – independent predictors of abnormal Lvmi In Hypertensives

Parameter	HR	95% CI	P-value
Age	2.07	1.04 to 4.09	< 0.001
Gender	1.67	1.03 to 3.09	0.526
Premature CAD	1.02	0.32 to 1.97	0.783
Smoking	1.67	1.01 to 3.78	0.017
Duration of Hypertension	2.24	1.04 to 4.83	< 0.001
Diabetes Mellitus	1.10	1.03 to 1.17	0.419
Dyslipidemia	1.05	0.94 to 1.07	0.875
BMI	2.76	1.24 to 4.78	< 0.001
Pulse Rate	1.56	1.08 to 1.84	0.187
Peripheral SBP	1.02	1.01 to 1.10	0.256
Peripheral DBP	1.03	0.99 to 1.06	0.224
Central Aortic Systolic Pressure	4.02	2.68 to 6.41	< 0.001
Central Aortic Diastolic Pressure	3.65	2.53to 5.59	< 0.001
Augmentation Index	1.20	1.07 to 1.35	0.343
Mean Pulse Wave Velocity(aPWV)	3.26	1.40 to 5.37	< 0.001
Hypertension Drug Intake –Non (CCB + ACEI + ARB)	2.70	1.26 to 4.34	0.018

The above table analysis of independent variables like age, gender, premature CAD, smoking, hypertension duration, diabetes, dyslipidemia, BMI, pulse rate, peripheral and central blood pressures , augmentation index, mean pulse wave velocity and hypertension drug intake were done and significant P valves were highlighted in yellow

4. CONCLUSIONS

Elevated central aortic pressures measured non-invasively by brachial cuff pulse volume plethysmography in hypertensive individuals are associated and statistically correlated with an increase in LVMI. Elevated Mean pulse wave velocity in hypertensive individuals measured non-invasively by brachial cuff pulse volume plethysmography is associated and statistically correlated with an increase in LVMI. Even though age, sex, duration of hypertension, BMI were found to be independent predictors for LVMI, CAP and aPWV were found to have the strongest influence. The use of CCB as an antihypertensive agent as monotherapy or in combination with ACE/ARB was seen to be significantly associated with decreased LVMI and aPWV.

5. RECOMMENDATION

- The routine measure of central aortic pressures non-invasively by approved devices (radial tonometers or brachial cuff PVP or supra systolic brachial cuff PVP) in hypertensive individuals with aids in better prognostication prevention of end-organ damage and medication optimization.
- Echocardiographic assessment of LVMI in all hypertensive subjects and periodic follow up of LVMI by echo imaging, preventing early onset of CV events.

CONSENT AND ETHICAL APPROVAL

The study is approved by institutional ethical committee and scientific committee. All patients were included after informed written consent.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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