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Original Research Article

Assessment of correlation of mean arterial pressure and left ventricular mass with left atrial volume index in hypertensive patients

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ABSTRACT

Background: Hypertension leads to left ventricular remodeling and left atrial enlargement (LAE), which are critical indicators of hypertensive heart disease. Left atrial volume index (LAVI) is a sensitive marker of LAE and diastolic dysfunction. This study aims to assess the correlation between mean arterial pressure (MAP), left ventricular mass (LVM), and LAVI in hypertensive patients to improve cardiovascular risk stratification.
Methods: This prospective observational study included 50 hypertensive patients admitted to Chettinad Super Speciality Hospital over three months. Using 2D echocardiography, LAVI was measured via the area-length method, while LVM was calculated using the Teichholz cubed formula. Relative wall thickness (RWT) was also determined to characterize left ventricular hypertrophy (LVH). Hemodynamic parameters, including MAP, were recorded. Patients were categorized based on the presence or absence of LVH, and correlations between MAP, LVM, and LAVI were analysed.
Results: Patients aged 60–80 years were predominant (42%), followed by 40–60 years (38%) and 20–40 years (20%). Males constituted 58% of the study group. LVM was higher in the LVH group (0.74) than in non-LVH (0.72). RWT was greater in LVH patients (0.59 versus 0.48), while MAP was lower in LVH patients (101.4 versus 113.5). LAVI was lower in LVH patients (19.4 versus 25.5). A weak positive correlation was found between LAVI and MAP (r=0.3984), indicating that while MAP influences LAVI, other factors may contribute significantly.
Conclusions: Chronically elevated blood pressure leads to structural cardiac remodelling, with LAVI being an early marker of hypertensive heart disease. Monitoring LAVI can aid in risk assessment and treatment planning for hypertensive patients.

Keywords: Hypertension, Mean arterial pressure, Left ventricular mass, Left atrial volume index, Echocardiography, Cardiac remodelling

INTRODUCTION

Hypertension is a global health concern associated with cardiovascular morbidity and mortality.[1] It leads to left ventricular (LV) remodelling, manifesting as concentric remodelling or hypertrophy.[1] Left atrial enlargement (LAE) is a marker of hypertensive heart disease, with prevalence ranging from 16-83% in hypertensive populations.[2] LAE is an independent predictor of cardiovascular events and is more prevalent in patients with left ventricular hypertrophy.[2,3] Left atrial volume index (LAVI) is the most sensitive parameter for detecting LAE.[3] Hypertensive heart disease involves pathological myocardial remodelling, including apoptosis, fibrosis, and microcirculatory changes, affecting not only the left ventricle but also the left atrium and right ventricle.[4] Early detection and management of these cardiac changes are crucial for risk assessment and treatment in hypertensive patients.[4]

Mean arterial pressure (MAP) is the average arterial pressure during one cardiac cycle, influenced by cardiac

output and systemic vascular resistance.[5] It is calculated as given in the formula, representing tissue perfusion pressure.[6]

$$MAP = DBP + 1/3(SBP - DBP)$$

MAP is considered a stable hemodynamic parameter, less affected by monitoring methods and catheter characteristics than other measures.[7] Recent research suggests that MAP may be more accurate in identifying cerebrovascular impacts of hypertension compared to systolic or diastolic blood pressure alone, with classification accuracies up to 95.2%.[8] This underscores the pathophysiological significance of MAP in hypertension diagnosis and research. However, it's important to note that satisfactory MAP values do not necessarily indicate adequate peripheral tissue perfusion, emphasizing the need for comprehensive clinical assessment alongside hemodynamic monitoring.[7]

Research has shown significant relationships between MAP, LVM, and LAVI in hypertensive patients. Studies have found that LVM index is higher in hypertensive patients and correlates with MAP. [9,10] Central sympathetic activations is associated with increased LVM in hypertension, with a positive correlation observed between sympathetic activity and LVM index.[11] LAVI has been identified as an indicator of left ventricular diastolic dysfunction in hypertensive patients with left ventricular hypertrophy.[12] While these studies have established links between MAP and LVM, and between LVM and LAVI, there appears to be a gap in the literature regarding direct correlations between MAP and LAVI. Hence we aimed to assess the correlation of the MAP and LVM with LAVI in hypertensive patients.

This study is significant as it explores the interrelationship between MAP, LVM, and LAVI in hypertensive patients, offering valuable insights into early cardiac remodelling. By identifying correlations among these parameters, the research aims to enhance early detection of diastolic dysfunction and structural heart changes. This can improve risk stratification and guide more personalized management strategies beyond standard blood pressure control. Furthermore, the study contributes to the growing body of knowledge on hypertensive heart disease by addressing a gap in current literature and supporting more comprehensive cardiovascular assessment in hypertension.

METHODS

This prospective observational study was conducted on 50 patients with known case of SHTN admitted in Chettinad Super Speciality Hospital, Kelambakkam for a period of 3 months between February 2024 to April 2024. The study has been approved by Institutional Ethics Committee (IEC) and inform consent was obtained before initiation of the study.

Inclusion and exclusion criteria

The study included patients diagnosed with hypertension, diabetes mellitus, dyslipidemia, hypothyroidism, and diastolic dysfunction. Conversely, patients who were normotensive, had a left atrial mass, valvular heart disease, or cardiomyopathy were excluded from the study.

Methodology

Using 2D conventional echocardiography with a Philips Affinity 50C machine, standard apical views—apical four-chamber (A4C) and apical two-chamber (A2C)—were utilized for the assessment of LA volume and LV mass. The area-length method was applied to measure LA volume, which involves planimetry of the LA area in both A4C and A2C views, typically at end-systole. The linear dimension, or length, is measured from the center of the mitral annulus to the superior border of the LA. LA volume (LAV) is then calculated using the formula where A1 and A2 are the measured areas in one plane and area in the orthogonal plane, L is the major-axis linear dimension.

$$LAV = 0.85 \times (A1 \times A2) \div L$$

For the assessment of LV mass, M-mode echocardiographic measurements were used to obtain interventricular septal thickness (IVS), posterior wall thickness (PW), and left ventricular internal diameter in diastole ($LVID_d$). These measurements are incorporated into the Teichholz cubed formula, which assumes the LV is spherical in shape. The formula for LV mass is given below, which estimates the outer and inner dimensions of the LV to determine myocardial volume.

$$LV\ mass = 1.05 \times [(IVS + PW + LVID_d)^3 - LVIDd^3]$$

Furthermore, LVH was evaluated and characterized as concentric, eccentric, or physiologic based on structural changes. The relative wall thickness, a key parameter in this characterization, was calculated as given below to distinguish whether hypertrophy was due to increased wall thickness or chamber enlargement.

$$Relative\ wall\ thickness = (PW + IVS) \div LVID_d$$

RESULTS

Patients in the 60–80-year age group were predominant, with 21 (42%) patients, followed by the 40–60-years age group in 19 (38%) patients and the 20–40-years age group with 10 (20%) patients. Male patients were more common, representing 29 (58%), while female patients accounted for 21 (42%) (Table 1).

The mean of LV mass was higher in LVH group (0.74) compared to those without LVH (0.72). Relative wall thickness (RWT) was also higher in the LVH group (0.59) compared to the non-LVH group (0.48). Conversely, MAP was lower in patients with LVH (101.4) compared to those

without LVH (113.5). LAVI was lower in the LVH group (19.4) compared to the non-LVH group (25.5) (Table 2).

Table 1: Demographic characteristics.

Variables	N (%)
Age (in years)	
20-40	10 (20)
40-60	19 (38)
60-80	21 (42)
Sex	
Male	29 (58)
Female	21 (42)

Table 2: Comparison of echocardiographic and hemodynamic parameters between patients with and without LVH.

Variables	Mean	
	With LVH	Without LVH
LV MASS	0.74	0.72
RWT	0.59	0.48
MAP	101.4	113.5
LAVI	19.4	25.5

The correlation analysis between the LAVI and MAP revealed a weak positive correlation (r=0.3984). This suggests that as MAP increases, LAVI tends to increase as well, but the relationship is not strong. While there is a positive association, the relatively low correlation coefficient indicates that other factors may also significantly influence LAVI apart from MAP (Table 3).

Table 3: Correlation between LAVI and MAP.

Variable	Value R	Correlation
LAVI and MAP	0.3984	Weekly positive

DISCUSSION

In our study, the majority of patients belonged to the older age groups, with a higher proportion of males than females. This demographic pattern supports the results of multiple epidemiological studies that indicate male sex and age are significant predictors of the development of LVH as a result of long-term exposure to cardiovascular risk factors like metabolic syndrome and hypertension. The idea that ageing and male sex are linked to increased myocardial remodelling tendencies is supported by Gehlken et al report that older adults with heart failure and reduced ejection fraction had significantly higher left LVMI, particularly among male patients.[13]

In our study, patients with LVH exhibited greater myocardial thickness and increased relative wall thickness, indicating structural cardiac remodelling. This is consistent with studies on hypertensive heart disease, which show that concentric remodelling is frequently reflected in increased myocardial thickness and RWT as an adaptive response to long-term pressure overload. According to research by Abdalla et al and Guiz et al, concentric hypertrophy was linked to poor cardiovascular outcomes and was more common in patients with high blood pressure.[14,15] This association is not supported by some studies particularly that increased wall thickness can happen without the conventional pressure-overload mechanisms of LVH, and thus does not always indicate classic structural remodelling. These studies are especially relevant to patients with hypertrophic cardiomyopathy or genetic predispositions.[16,17]

Patients with LVH in our study had lower MAP than those without, which may indicate haemodynamic adaptations. This contradicts the widely held belief that LVH is frequently linked to chronic hypertension and, consequently, elevated MAP. The LVH group also had a lower left atrial volume index (LAVI), which could be a result of variations in filling pressures or left atrial remodelling. A weak positive correlation between LAVI and MAP was confirmed by the correlation analysis, indicating that although higher MAP may be linked to higher LAVI, changes in left atrial volume are probably influenced by other contributing factors. According to Rojek et al, there was a weak but positive correlation between LAVI and MAP in those with lower LV mass index, which may indicate compensatory or adaptive mechanisms that cause some LVH patients to have lower MAP.[18] Blood pressure and left ventricular mass have a positive relationship in patients without hypertrophy but a negative relationship in those with hypertrophy, according to a study by Drayer et al suggesting that other factors control left ventricular mass in hypertensive patients with hypertrophy.[19] According to Mittal et al, higher mean arterial pressure is linked to an increased left ventricular mass index, even in people with normal clinic blood pressure.[20]

CONCLUSION

A chronically high blood pressure load might cause compensatory hypertrophy, and the gradual progression of hypertension to hypertensive heart disease is inevitable. LAE occurring before LVH is an early sign of hypertensive heart disease. The left atrial volume index is independently correlated with the outcome of cardiovascular disease and offers a sensitive morphologic expression of the degree of left ventricular diastolic dysfunction. The left atrial volume index also appears to be a good indicator of cardiovascular risk and burden.

In hypertensive heart disease, left atrial enlargement is a frequent but often disregarded syndrome. Assessment of left atrial enlargement could be a valuable proxy indicator for tracking the success of medical treatment, as well as intense risk factor management and reversal remodelling. Simple measurement of LA diameter by echo may be an effective method for identifying those high risk individuals. In addition, isolated LAE (without LVH) was also frequently seen in hypertensive individuals. The

frequency of LAE was highest in patients with increased LV mass. When compared to the individuals with LVH in this study, the patients without LVH who had higher MAP also showed slightly higher LAVI. It demonstrates that RWT and reduced LV mass will also cause LAE to develop. Elevated LAVI indicates first alterations in reaction to an increase in systemic blood pressure.

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