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RESEARCH ARTICLE

Assessment of Cardiovascular Risk Factors in Prehypertensive Subjects and Their Association with Left Ventricular Geometry

Deepak Kumar Das[1*] | Tarun Agarwal[2] | Bindu Garg[3] | Smita Gupta[4] | Deep Chandra Pant[5]

Abstract

Background: Prehypertension, defined as systolic blood pressure of 120–139 mmHg or diastolic blood pressure of 80–89 mmHg, represents an intermediate cardiovascular risk state that is associated with progressive organ damage. The impact of prehypertension on left ventricular (LV) geometry and its clustering with other cardiovascular risk factors remains underexplored in South Asian populations.

Objectives: To assess cardiovascular risk factor burden in prehypertensive subjects and to determine the association between prehypertension and left ventricular geometric patterns compared with normotensive controls.

Methods: This hospital-based case-control study enrolled 100 subjects (50 prehypertensive cases and 50 normotensive controls) aged 20–60 years. Anthropometric measurements, fasting lipid profile, blood glucose, and inflammatory markers were recorded. Two-dimensional echocardiography was performed to assess left ventricular mass index (LVMI), relative wall thickness (RWT), and LV geometric patterns using the Ganau classification.

Results: Prehypertensive subjects demonstrated significantly higher BMI, waist circumference, fasting glucose, total cholesterol, LDL-C, triglycerides, and lower HDL-C compared to normotensives ($p<0.05$ for all). LV geometric abnormalities were present in 56% of prehypertensive subjects versus 8% of controls ($p<0.001$). Concentric remodelling was the most prevalent pattern (28%), followed by concentric hypertrophy (18%) and eccentric hypertrophy (10%). LVMI and RWT were significantly higher in prehypertensives (104.6 ± 18.3 g/m^2 vs. 82.4 ± 14.1 g/m^2, $p<0.001$; 0.44 ± 0.06 vs. 0.37 ± 0.05, $p<0.001$). LVMI correlated positively with systolic BP, BMI, fasting glucose, LDL-C, and triglycerides.

Conclusion: Prehypertension is associated with a significantly higher cardiovascular risk factor burden and adverse LV geometric remodelling even before overt hypertension develops. Early echocardiographic evaluation and aggressive risk factor modification are warranted in prehypertensive individuals.

Key words: Prehypertension, Left Ventricular Hypertrophy, Cardiovascular Risk Factors, Echocardiography, Left Ventricular Geometry, Concentric Remodelling

1 | INTRODUCTION

Hypertension remains one of the foremost modifiable risk factors for cardiovascular disease (CVD), stroke, chronic kidney disease, and premature mortality worldwide. The concept of prehypertension was formally introduced by the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) in 2003,

[1]1-Associate Professor, Dept of Physiology, SRMS Institute of Medical Sciences, Bareilly .
[2]2-Associate Professor, Dept of General Medicine, Varun Arjun Medical College, Banthra.
[3]3-Professor & HOD, Dept of Physiology, SRMS Institute of Medical Sciences, Bareilly .
[4]4-Professor & HOD, Dept of General Medicine, SRMS Institute of Medical Sciences, Bareilly .
[5]5-Professor, Dept of General Medicine, SRMS Institute of Medical Sciences, Bareilly .
Address correspondence to: Deepak Kumar, Das, 1-Associate Professor, Dept of Physiology, SRMS Institute of Medical Sciences, Bareilly ,

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defined as systolic blood pressure (SBP) of 120–139 mmHg and/or diastolic blood pressure (DBP) of 80–89 mmHg in adults not taking antihypertensive medication. (1)

The global prevalence of prehypertension ranges from 25% to 50% in adult populations, and it carries a markedly elevated risk of progression to frank hypertension—approximately 49% of prehypertensive individuals transition to Stage 1 hypertension within four years. (2, 3) Beyond progression risk, prehypertension independently confers excess cardiovascular risk, including coronary artery disease, heart failure, and all-cause mortality. (4)

The heart is a primary target organ of elevated blood pressure. Even modest elevations in blood pressure above optimal levels stimulate structural and functional cardiac adaptations. Left ventricular hypertrophy (LVH) and adverse LV geometric remodelling are established markers of subclinical cardiovascular damage and powerful independent predictors of cardiovascular events. (5, 6) The Ganau classification describes four LV geometric patterns based on LV mass index (LVMI) and relative wall thickness (RWT): normal geometry, concentric remodelling, concentric hypertrophy, and eccentric hypertrophy, each carrying different prognostic implications. (7)

Cardiovascular risk factors including obesity, dyslipidemia, insulin resistance, and a sedentary lifestyle frequently co-aggregate in prehypertensive subjects, compounding cardiac structural stress. (8) Yet, the simultaneous assessment of cardiovascular risk factor burden and LV geometry specifically in prehypertensive individuals—especially in comparison to age- and sex-matched normotensive controls—remains limited in the published literature from South Asian settings.

This study was therefore designed to comprehensively assess the cardiovascular risk factor profile of prehypertensive subjects and determine the association between prehypertension and adverse left ventricular geometric remodelling, using a case-control design with echocardiographic evaluation.

2 | MATERIALS AND METHODS

2.1 | Study Design and Setting

This hospital-based, prospective case-control study was conducted over a period of 12 months at a tertiary care teaching hospital. The study protocol was approved by the Institutional Ethics Committee and written informed consent was obtained from all participants prior to enrolment.

2.2 | Study Population

A total of 100 subjects were recruited: 50 cases (prehypertensive) and 50 controls (normotensive). Cases were recruited from the outpatient department of medicine and controls were matched for age and sex from the general health checkup clinic.

Inclusion Criteria for Cases: (i) Age 20–60 years (ii) Systolic BP 120–139 mmHg and/or Diastolic BP 80–89 mmHg on three separate occasions at least one week apart (iii) Not on antihypertensive medication. **Inclusion Criteria for Controls:** (i) Age 20–60 years (ii) Systolic BP <120 mmHg and Diastolic BP <80 mmHg. **Exclusion Criteria (Both Groups):** Established hypertension (BP ≥140/90 mmHg) or currently on antihypertensive therapy; known coronary artery disease, heart failure, valvular heart disease or cardiomyopathy; on thyroid medication or a case of hyper or hypothyroidism, diabetes mellitus (fasting glucose ≥126 mg/dL or on antidiabetic medication); chronic kidney disease (eGFR <60 mL/min/1.73m^2); pregnancy; chronic inflammatory disease; secondary hypertension; inadequate echocardiographic window; and prior cardiac surgery.

2.3 | Clinical and Anthropometric Assessment:

Blood pressure was measured with a calibrated mercury sphygmomanometer after 10 minutes of rest, in the sitting position, from the right arm, using Korotkoff phase I and V sounds. The average of three measurements taken on three separate visits was used. Height, weight, and waist circumference were measured using standardized techniques. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured at the midpoint between the

lower costal margin and the iliac crest.

2.4 | Biochemical Investigations

After an overnight fast of at least 8 hours, venous blood samples were collected for fasting plasma glucose (FPG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C, calculated using Friedewald equation), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), serum creatinine, uric acid, and high-sensitivity C-reactive protein (hs-CRP). Metabolic syndrome was defined according to the International Diabetes Federation (IDF) consensus criteria (2006). (9)

2.5 | Echocardiographic Assessment

Two-dimensional and M-mode transthoracic echocardiography was performed by a single cardiologist using a Philips HD11 XE machine with a 3.5 MHz phased-array transducer, following the guidelines of the American Society of Echocardiography (ASE).10 The following parameters were measured: interventricular septal thickness in diastole (IVSd), LV posterior wall thickness in diastole (LVPWd), LV end-diastolic diameter (LVEDd), LV end-systolic diameter (LVEsd), and ejection fraction (EF) by the modified Simpson method.

LV mass was calculated using the Devereux formula: LV mass (g) = $0.8 \times \{1.04 \times [(\text{LVEDd} + \text{IVSd} + \text{LVPWd})^3 - \text{LVEDd}^3]\} + 0.6$. LV mass was indexed to body surface area (BSA) to obtain LVMI. LVH was defined as LVMI >115 g/m^2 in men and >95 g/m^2 in women. Relative wall thickness (RWT) was calculated as $2 \times$ LVPWd / LVEDd, with a value >0.42 indicating increased RWT. LV geometry was classified using the Ganau system: (i) Normal geometry (LVMI normal + RWT $\leq$0.42); (ii) Concentric remodelling (LVMI normal + RWT >0.42); (iii) Concentric hypertrophy (LVMI elevated + RWT >0.42); (iv) Eccentric hypertrophy (LVMI elevated + RWT $\leq$0.42). (7)

Diastolic function was assessed by pulsed-wave Doppler of the mitral valve inflow, measuring peak early (E) and late (A) filling velocities, E/A ratio, and E-wave deceleration time (EDT), as per ASE recommendations.

2.6 | Statistical Analysis

Data were analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Comparison between groups was performed using the independent samples t-test for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed data. Chi-square test or Fisher's exact test was used for categorical variables. Pearson and Spearman correlation coefficients were used to assess associations. Logistic regression analysis was used to determine independent predictors of LV geometric abnormality. A p-value of <0.05 was considered statistically significant.

3 | RESULTS

Baseline Characteristics

ummarizes the baseline demographic, anthropometric, andbiochemical characteristics of both groups. The two groups were well-matched for age and sex distribution (p>0.05). Prehypertensive subjects had significantly higher BMI (26.8 ± 3.4 vs. 23.2 ± 2.9 kg/m^2, p<0.001) and waist circumference (91.4 ± 8.6 vs. 82.1 ± 6.9 cm, p<0.001).

Fasting glucose levels were significantly higher in cases (98.7 ± 11.3 vs. 88.4 ± 9.2 mg/dL, p<0.001), though none met criteria for diabetes. The lipid profile demonstrated significantly higher total cholesterol, LDL-C, and triglycerides, and significantly lower HDL-C in prehypertensives. Family history of hypertension was significantly more prevalent among cases (58% vs. 28%, p=0.003).Table 1

Echocardiographic Parameters and LV Geometry

resents the echocardiographic parameters. LVMI was significantly higher in prehypertensives (104.6 ± 18.3 vs. 82.4 ± 14.1 g/m^2, p<0.001). Similarly, IVSd, LVPWd, RWT, and LVEDd were all signifi-

Table 1. Baseline Characteristics of Prehypertensive Cases and Normotensive Controls

Characteristic	Prehypertensives (n=50)	Normotensives (n=50)	p-value
Age (years), Mean±SD	38.6 ± 8.2	37.9 ± 7.8	0.67
Male (n, %)	31 (62%)	29 (58%)	0.68
BMI (kg/m^2), Mean±SD	26.8 ± 3.4	23.2 ± 2.9	<0.001
Waist Circumference (cm)	91.4 ± 8.6	82.1 ± 6.9	<0.001
Systolic BP (mmHg)	128.4 ± 5.1	112.6 ± 6.3	<0.001
Diastolic BP (mmHg)	82.1 ± 3.8	72.3 ± 4.1	<0.001
Fasting Glucose (mg/dL)	98.7 ± 11.3	88.4 ± 9.2	<0.001
Total Cholesterol (mg/dL)	204.3 ± 32.1	182.6 ± 28.4	0.001
LDL-C (mg/dL)	128.7 ± 29.4	108.3 ± 24.6	0.001
HDL-C (mg/dL)	42.1 ± 8.6	51.4 ± 9.2	<0.001
Triglycerides (mg/dL)	164.2 ± 48.6	121.3 ± 38.9	<0.001
Smoking (n, %)	18 (36%)	10 (20%)	0.07
Family H/O HTN (n, %)	29 (58%)	14 (28%)	0.003

HTN = hypertension; BP = blood pressure; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; SD = standard deviation. Bold p-values indicate statistical significance (p<0.05).

cantly greater in cases. While ejection fraction was preserved in both groups and did not differ significantly (62.4 ± 5.8% vs. 64.1 ± 5.2%, p=0.13), the E/A ratio was significantly lower in prehypertensives (1.08 ± 0.21 vs. 1.32 ± 0.24, p<0.001), reflecting early diastolic dysfunction.Table 2

Table 2. Echocardiographic Parameters in Prehypertensive and Normotensive Subjects

Echocardiographic Parameter	Prehypertensives (n=50)	Normotensives (n=50)	p-value
LVMI (g/m^2), Mean±SD	104.6 ± 18.3	82.4 ± 14.1	<0.001
RWT, Mean±SD	0.44 ± 0.06	0.37 ± 0.05	<0.001
IVSd (mm)	10.2 ± 1.4	8.6 ± 1.1	<0.001
LVPWd (mm)	9.8 ± 1.3	8.3 ± 1.0	<0.001
LVEDd (mm)	49.3 ± 4.2	47.1 ± 3.8	0.008
EF (%)	62.4 ± 5.8	64.1 ± 5.2	0.13
E/A ratio	1.08 ± 0.21	1.32 ± 0.24	<0.001

LVMI = left ventricular mass index; RWT = relative wall thickness; IVSd = interventricular septum in diastole; LVPWd = LV posterior wall in diastole; LVEDd = LV end-diastolic diameter; EF = ejection fraction.

3.1 | Distribution of LV Geometric Patterns

Abnormal LV geometry was found in 56% of prehypertensive subjects compared to only 8% of controls (p<0.001). Concentric remodelling was the most common pattern among prehypertensives (28%), followed by concentric hypertrophy (18%) and eccentric hypertrophy (10%). No control subject exhibited eccentric hypertrophy Table 3 .

Table 3. Distribution of LV Geometric Patterns

LV Geometry Pattern	Prehypertensives n (%)	Normotensives n (%)	p-value
Normal geometry	22 (44%)	46 (92%)	<0.001
Concentric remodeling	14 (28%)	3 (6%)	0.004
Concentric hypertrophy	9 (18%)	1 (2%)	0.007
Eccentric hypertrophy	5 (10%)	0 (0%)	0.02
Abnormal geometry (total)	28 (56%)	4 (8%)	<0.001

3.2 | Correlation and Logistic Regression

LVMI correlated significantly with SBP (r=0.61, p<0.001), BMI (r=0.54, p<0.001), waist circumference (r=0.49, p<0.001), fasting glucose (r=0.38, p=0.006), LDL-C (r=0.41, p=0.003), and triglycerides (r=0.36, p=0.011). RWT correlated with SBP (r=0.58, p<0.001) and BMI (r=0.47, p<0.001).

On binary logistic regression analysis, after adjusting for age, sex, BMI, and lipid parameters, prehypertension (OR 12.4; 95% CI 4.2–36.7; p<0.001), elevated BMI (OR 2.8; 95% CI 1.3–6.0; p=0.008), elevated LDL-C (OR 2.1; 95% CI 1.1–4.2; p=0.03), and low HDL-C (OR 1.9; 95% CI 1.0–3.6; p=0.048) were independently associated with abnormal LV geometry.

Metabolic syndrome was present in 34% of prehypertensives versus 10% of controls (p=0.005). Subjects with metabolic syndrome had significantly higher LVMI and a higher prevalence of LV geometric abnormalities.

4 | DISCUSSION

The present case-control study establishes that prehypertension—even in the absence of overt hypertension—is associated with a substantially greater burden of cardiovascular risk factors and significantly adverse LV geometric remodelling compared with age- and sex-matched normotensive individuals. These findings carry important clinical implications for the risk stratification and early management of prehypertensive subjects.

The prevalence of abnormal LV geometry in our prehypertensive cohort (56%) is remarkably high and aligns with findings from similar studies. Cuspidi et al. reported LV geometric abnormalities in approximately 44–58% of hypertensive patients, and emerging data suggest that such changes begin earlier, in the prehypertensive stage. (10, 11) Kizer et al. demonstrated in the LIFE study that concentric remodelling independently predicted cardiovascular events, underscoring the clinical significance of identifying even sub-threshold hypertrophic changes. (12)

The predominance of concentric remodelling (28%) as the most common geometric pattern in our study reflects the hemodynamic milieu of prehypertension—characterized by increased peripheral vascular resistance with relatively preserved cardiac output. This pattern represents an early adaptive response to increased afterload and is thought to precede overt LVH. (13) That 18% of cases had already developed concentric hypertrophy—a pattern historically associated with overt hypertension—underscores the spectrum of cardiac damage at this supposedly 'benign' blood pressure stage.

The significant reduction in E/A ratio (1.08 vs. 1.32, p<0.001) without impairment of systolic function is consistent with the concept of isolated diastolic dysfunction as an early manifestation of hypertensive heart disease. (14) Diastolic dysfunction in prehypertension has been previously reported by Liao et al. in the Atherosclerosis Risk in Communities (ARIC) study, where elevated blood pressure within the prehypertensive range was independently associated with worse diastolic function indices. (15)

The clustering of metabolic risk factors in our prehypertensive subjects—higher BMI, waist circumference, fasting glucose, dyslipidemia (elevated LDL-C, low HDL-C, elevated triglycerides), and a higher prevalence of metabolic syndrome—parallels observations from national and international population-based studies. (16, 17) The Framingham Heart Study demonstrated that prehypertensive individuals had a significantly higher prevalence of metabolic syndrome and insulin resistance, each independently contributing to LV structural changes. (18)

The independent association of elevated LDL-C and low HDL-C with abnormal LV geometry in our logistic regression model is noteworthy. Dyslipidemia promotes LV fibrosis and impairs diastolic compliance through direct lipotoxic mechanisms, inflammation-mediated pathways, and augmentation of neurohormonal activation. (19) The role of hs-CRP and inflammatory pathways in mediating cardiac remodelling in prehypertension warrants further study.

The strong positive correlation between LVMI and SBP (r=0.61) and BMI (r=0.54) in our study is consistent with the well-established mechanistic link between volume-pressure overload and cardiac structural adaptation. (20) Notably, even within the

prehypertensive BP range (SBP 120–139 mmHg), the linear relationship with LVMI was maintained, suggesting a continuous rather than threshold-based cardiovascular risk.

Our findings build upon the landmark Verdecchia et al. study, which first demonstrated that LV geometric patterns carry independent prognostic value beyond BP levels, and support the recommendation for echocardiographic screening in high-risk prehypertensive individuals—particularly those with additional metabolic risk factors. (21)

From a clinical standpoint, these results argue for proactive cardiovascular risk assessment in prehypertensive individuals beyond simple blood pressure monitoring. The 2017 ACC/AHA hypertension guidelines reclassified prehypertension into 'elevated BP' and 'Stage 1 hypertension,' reflecting the recognition of progressive risk at blood pressures previously considered borderline. (22) Lifestyle interventions—including weight reduction, dietary sodium restriction, physical activity, and lipid-lowering therapy—have demonstrated regression of LVH and improvement in diastolic function even without pharmacotherapy. (23)

Strengths and Limitations

Strengths of this study include its case-control design with well-matched controls, comprehensive cardiovascular risk factor assessment, standardized BP measurement across three visits, and echocardiographic evaluation by a single cardiologist to minimize inter-observer variability. Limitations include the relatively small sample size (n=100) limiting generalizability, hospital-based recruitment introducing potential selection bias, cross-sectional echocardiographic assessment precluding longitudinal conclusions, and the inability to measure tissue Doppler imaging-based diastolic indices. The study did not assess physical activity levels objectively, and a 24-hour ambulatory blood pressure monitor was not employed. (24, 25)

5 | CONCLUSION

Prehypertension is associated with a significantly higher burden of cardiovascular risk factors—including obesity, dyslipidemia, impaired fasting glucose, and metabolic syndrome—and is accompanied by adverse left ventricular geometric remodelling even before the threshold of clinical hypertension is reached. Concentric remodelling and concentric hypertrophy are the predominant LV geometric patterns, and early diastolic dysfunction is already evident in prehypertensive subjects. These subclinical cardiac changes are independently associated with elevated blood pressure, BMI, and dyslipidemia. Our findings advocate for early echocardiographic evaluation and aggressive multifactorial risk reduction strategies in all prehypertensive individuals to prevent or delay the progression of adverse cardiac remodelling and its consequent cardiovascular sequelae.

Future large-scale longitudinal studies examining the reversibility of LV geometric changes with lifestyle modifications and targeted pharmacotherapy in prehypertension are necessary to formulate evidence-based clinical guidelines.

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