Original Article

Microalbuminuria as an indicator for LVH severity in patients with primary hypertension

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Abstract

Background: Hypertension is among the most complex global public health concerns. Hypertension and kidney diseases share a strong correlation. The existence of microalbuminuria signifies early renal impairment. Left ventricular hypertrophy (LVH) is one of the early indicators of target organ damage in hypertension individuals. **Objective:** To evaluate the connection between microalbuminuria and left ventricular mass index (LV mass index), as well as systolic and diastolic blood pressure, in people with primary hypertension. **Methods:** This cross-sectional analytical study was conducted for about 2 years and included 125 essential hypertensive patients who met JNC-8 criteria for ambulatory hypertension. Urine albumin excretion was estimated by the method of immunoturbidimetry. 2D echocardiography was performed to determine the LV mass index. Statistical analysis by standard methods to measure proportions and Chi-square test for analyzing association between variables was used. **Results:** Out of the 125 patients, 50 (40.0%) patients had LVH and 51 (40.8%) patients had microalbuminuria. Patients with LVH exhibited a prevalence of microalbuminuria at 72%. Microalbuminuria was significantly related with diastolic blood pressure and LV mass index. **Conclusion:** This study shows that microalbuminuria is very common in essential hypertension with LVH. There is a significant association of microalbuminuria with LV mass index and diastolic blood pressure in patients of primary hypertension.

Keywords: Left ventricular mass index, LVH, microalbuminuria

Introduction

Hypertension is estimated to affect more than a billion people worldwide.[1] According to the latest figures from WHO, it is the foremost cause of death globally, with an estimated 13% of fatalities attributed to it.[2] It is a disorder of circulatory regulation. Persistent high blood pressure leads to kidney failure, heart failure, and stroke and accelerates atherosclerosis. Heart disease, stroke, and kidney failure are three main manifestations in 50%, 33%, and 10% to 15% of people, respectively, if untreated.[3] The presence of hypertension significantly contributes to heart diseases. According to epidemiological studies, more than 200 million people in India alone have primary hypertension, and this number appears to be increasing.[4] 24% of all coronary artery disease (CAD) deaths and about 57% of all cerebrovascular accident (CVA) deaths in India are due to hypertension.[5] Hypertension is the most common and modifiable risk factor of heart diseases and kidney diseases. It accounts for the highest number of deaths and disabilities globally.[6] Hypertension elevates the heart's workload, inducing structural and functional aberrations. These modifications encompass hypertrophy of the left ventricle, culminating in elevated left ventricular mass and potentially leading to cardiac failure.

Microalbuminuria is defined by 24-hour urinary albumin levels between 30 and 299 mcg and denotes renal and cardiovascular damage. It frequently occurs in people with primary hypertension.[7] The microalbuminuria prevalence in primary hypertension has

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been the subject of numerous studies, but the exact data are still unknown. Even mildly elevated albumin levels, well within the range of microalbuminuria, increase cardiovascular event risks such as ischemic heart disease, cerebrovascular accident, heart failure, and peripheral vascular resistance.[8] Monitoring of urinary albumin excretion (UAE) is the simplest way of estimating risk of cardiovascular maladies.[9] This research sought to elucidate the association of microalbuminuria with left ventricular mass index (LV mass index) and with systolic and diastolic blood pressure (BP).

Material and Methods

This cross-sectional analytical study was conducted for about 2 years, and 125 patients diagnosed with primary hypertension, as per JNC-8 criteria, were taken in the study. Exclusion criteria for the study included patients with diabetes mellitus, pyrexia (current or within the past month), serum creatinine >1.5 mg/dl, kidney disease, obesity, and cardiovascular and cerebrovascular accidents within the past 6 months. All patients provided a detailed history, and a clinical examination was carried out. A detailed questionnaire was used to exclude secondary hypertension. Standard blood tests such as electrocardiogram, blood count, blood glucose, kidney function tests, liver function tests, and 2D echocardiography were then performed in all study participants. To calculate the LV mass, the formula = $0.8\{1.04[([LVEDD + IVSd + PWd]^3 - LVEDD^3)]\} +0.6$ was used. After adjustment for surface area of body, the binary prognostic value of left ventricular hypertroph (LVH) (LV mass index exceeding 115 g/m2 in men or 95 g/m2 in women) is determined.[10]

Statistical analysis

SPSS was employed for statistical evaluation. Data were visually depicted using mean (standard deviation), frequency count, and proportion (%). Independent t-tests were implemented to contrast discrete variables between groups, whereas Chi-square tests were utilized for categorical variables.

Results

The frequencies of ages ≤35, 36–45, 46–55, and 56–65 years were 0.00%, 4.00%, 10.00%, and 86.00% in the group with LVH and 1.33%, 8.00%, 14.67%, and 76.00% in the group without LVH, respectively. The average age of individuals with LVH was 58.90 years, with an SD of 4.77 years. In contrast, the average age of those without LVH was 57.85 years, with an SD of 7.53 years. The percentages of male and female sex were 62.00% and 38% in the group with LVH and 57.33% and 42.67% in the group without LVH, respectively. The percentage of familial hypertension was 36.00% in the presence of LVH and 16.0% in the absence of LVH. The percentage of smoking was 36.00% in the presence of LVH and 37.33% in the absence of LVH. On the basis of familial H/O hypertension, both groups differed significantly. Mean height (cm), weight (kg), and BMI (kg/m2) were not significantly associated in the presence of LVH and in the absence of LVH [Table 1].

The mean systolic and diastolic blood pressures (BPs) (mmHg) were 174.12 ± 8.99 and 105.68 ± 6.70 mmHg in the group with LVH present and 169.39 ± 13.62 and 102.56 ± 6.70 mmHg in the group without LVH, respectively. The mean systolic BP and diastolic BP were significantly elevated in the presence of LVH [Table 2]. Mean Hb, ESR, RBS, S urea, S creatinine, SGOT, and SGPT did not differ significantly between LVH present and LVH absent [Table 2].

The percentage of absence and presence of microalbuminuria was 28.00% and 72.00% in the LVH-present group and 80.00% and 20.00% in the LVH-absent group, respectively. On the basis of presence and absence of microalbuminuria, both groups were significantly different [Table 3]. The mean urine

Table 1: Baseline characteristics of the patients in between LVH-present and LVH-absent groups

	Age (years)	LVH Present (*n*=50)		LVH Absent (*n*=75)		Total	Chi Sq.	*P*
		n	%	*n*	%			
Age (years)	≤35	0	0.00	1	1.33	1	2.30	0.512
	36-45	2	4.00	6	8.00	8		
	46-55	5	10.00	11	14.67	16		
	56-65	43	86.00	57	76.00	100		
	Mean±SD	58.90±4.77		57.85±7.53		-	0.87	0.385
Gender	Male	31	62.00	43	57.33	74	0.12	0.738
	Female	19	38.00	32	42.67	51		
Family H/O Hypertension	Yes	18	36.00	12	16.00	30	5.53	0.019*
	No	32	64.00	63	84.00	95		
Smoking	Yes	18	36.00	28	37.33	46	0.02	0.880
	No	32	64.00	47	62.67	79		
Height (cm)		157.98	4.06	158.28	4.62		0.373	0.710
Weight (kg)		58.78	5.82	58.65	4.85		0.132	0.895
BMI (kg/m^2)		23.51	1.67	23.39	1.28		0.458	0.648

*=Significant (*P*<0.05)

Table 2: Association of mean systolic and diastolic BP and biochemical parameters in between LVH-present and LVH-absent groups

	LVH Present (*n*=50)		LVH Absent (*n*=75)		*t*	*P*
	Mean	±SD	Mean	±SD		
Systolic BP	174.12	8.99	169.39	13.62	2.16	0.033*
Diastolic BP	105.68	6.70	102.56	6.70	2.56	0.012*
Hb	10.28	1.21	10.31	1.14	-0.122	0.903
ESR	16.52	2.19	16.60	2.19	-0.200	0.842
RBS	124.98	19.09	128.27	20.81	-0.894	0.373
S. Urea	33.42	4.90	33.33	4.96	0.10	0.924
S. Creatinine	1.10	0.28	1.09	0.25	0.29	0.770
S. Cholesterol	156.20	25.12	161.07	22.44	-1.13	0.260
HDL	42.06	8.98	43.24	8.95	-0.72	0.472
LDL	132.92	17.92	132.79	14.61	0.05	0.964

*=Significant (*P*<0.05)

albumin-creatinine ratio was significantly higher in the group with LVH (122.92 ± 89.54) compared with the group without LVH (26.48 ± 25.65).

The mean body surface area, LVEDD, IVSd, and PWd were 1.60 ± 0.10, 3.88 ± 0.44, 1.46 ± 0.20, and 1.46 ± 0.20 in the LVH-present group and 1.61 ± 0.09, 3.89 ± 0.39, 1.08 ± 0.20, and 1.08 ± 0.20 in the LVH-absent group, respectively. Individuals with LVH exhibited a significantly higher mean interventricular septal thickness (IVSd) and posterior wall thickness (PWd) compared to those without LVH. However, there was no significant difference in mean body surface area or left ventricular end-diastolic diameter (LVEDD) between the two groups [Table 4].

The mean left ventricular mass was significantly greater in the LVH-present group (213.02 ± 20.70 and 132.90 ± 12.04, respectively) compared to the LVH-absent group (135.29 ± 27.86 and 84.38 ± 17.36, respectively) [Table 5].

Microalbuminuria was significantly positively correlated with DBP, LV mass, and LV mass index in patients overall and DBP in the group with LVH. In addition, microalbuminuria was significantly positively correlated with DBP and LV mass in the group without LVH [Table 6].

Table 3: Association frequencies of presence and absence of microalbuminuria between LVH-present and LVH-absent groups

Microalbuminuria	LVH Present (*n*=50)		LVH Absent (*n*=75)		Total	Chi Sq.	*P*
	n	%	*n*	%			
Absent	14	28.00	60	80.00	74	31.47	<0.001*
Present	36	72.00	15	20.00	51		

*=Significant (*P*<0.05)

Table 4: Relationship between mean urine albumin creatinine ratio, body surface area, LVEDD, IVSd, and PWd in the group with LVH present and LVH-absent group

	LVH Present (*n*=50)		LVH Absent (*n*=75)		*t*	*P*
	Mean	±SD	Mean	±SD		
Urine albumin creatinine ratio	122.92	89.54	26.48	25.65	8.82	<0.001*
Body Surface Area	1.60	0.10	1.61	0.09	-0.02	0.614
LVEDD	3.88	0.44	3.89	0.39	-0.08	0.937
IVSd	1.46	0.20	1.08	0.20	10.46	<0.001*
PWd	1.46	0.20	1.08	0.20	10.46	<0.001*

*=Significant (*P*<0.05)

Table 5: Association of mean LV mass and LV mass index between LVH-present and LVH-absent groups

	LVH Present (*n*=50)		LVH Absent (*n*=75)		*t*	*P*
	Mean	±SD	Mean	±SD		
LV mass	213.02	20.70	135.29	27.86	16.86	<0.001*
LV Mass Index	132.90	12.04	84.38	17.36	17.19	<0.001*

*=Significant (*P*<0.05)

Discussion

This cross-sectional analytical investigation aims to evaluate the relationship between microalbuminuria and concentric LVH in a group of individuals diagnosed with primary hypertension. The results of our investigation indicated a strong association between the presence of microalbuminuria and the thickening of the left ventricle wall (concentric LVH) in our patient population. Approximately 41% of our patients had microalbuminuria, with over 70% of those individuals exhibiting concentric LVH. Patients with microalbuminuria exhibited a significantly higher left ventricular mass index (LVMI) compared to those without. This association was statistically significant (Pearson correlation = 0.459, *P* value < 0.001). The underlying mechanisms for this correlation are likely multifaceted, with previous studies proposing various hypotheses.[11] Alterations in hemodynamic and elevated BP may contribute to initial renal injury (manifesting as microalbuminuria) in primary hypertension. These same factors may also induce changes in LV architecture, leading to concentric LVH. Both microalbuminuria and concentric LVH may therefore be end-stage manifestations of a common underlying mechanism. Other proposed mechanisms include microalbuminuria as a marker of vascular endothelial dysfunction, facilitating greater penetration of atherogenic lipoproteins into arterial walls. Microalbuminuria has also been implicated in impaired large artery hemodynamics in essential hypertension. Both the LVMI and urine albumin excretion are significant predictors of cardiovascular events. Multiple cross-sectional investigations[11,12] have found a linear connection between urine albumin excretion and left ventricular mass. In our study, individuals with LVH had substantially larger mean left ventricular mass and LVMI compared to those without LVH. Patients with LVH within the study group exhibited significantly increased left ventricular mass and mass index. The LIFE study, focusing on losartan intervention in hypertensive patients, revealed a concurrent pattern between changes in urinary albumin excretion and left ventricular mass over time.[13] The LVH group demonstrated significantly higher mean systolic BP (174.12 ± 8.99 mmHg) and diastolic BP (105.68 ± 6.70 mmHg) compared to the non-LVH group (169.39 ± 13.62 mmHg and 102.56 ± 6.70 mmHg, respectively). No significant correlation was found between LVH and the mean values of hemoglobin, ESR, RBS, serum urea, serum creatinine, SGOT, SGPT, serum cholesterol, HDL, and LDL. However, the mean urine albumin-to-creatinine ratio was significantly higher in patients with LVH, implicating that microalbuminuria may serve as a simple indicator of LVH in hypertensive patients. A study conducted in the southern part of India found that microalbuminuria serves as a reliable indicator for forecasting the likelihood of developing concentric LVH in patients of primary hypertension.[14] Consistent with this, our study demonstrated a strong positive association between microalbuminuria and DBP, LV mass, and LV mass index in patients of primary hypertension.

Table 6: Pearson correlation of microalbuminuria with systolic BP, diastolic BP, LV mass, and LV MASS INDEX in all patients, LVH-present and LVH-absent groups

	Overall		LVH Present		LVH Absent	
	Pearson Correlation	*P*	Pearson Correlation	*P*	Pearson Correlation	*P*
Systolic BP	0.149	0.098	-0.082	0.573	0.131	0.262
Diastolic BP	0.381	<0.001*	0.480	<0.001*	0.263	0.022*
LV mass	0.540	<0.001*	0.256	0.073	0.231	0.046*
LV mass index	0.459	<0.001*	-0.064	0.660	0.162	0.164

*=Significant (P<0.05)

Conclusions

Our study revealed a strong association between microalbuminuria and concentric LVH in the study population. Patients with LVH exhibited a significantly higher incidence of microalbuminuria. These findings suggest that microalbuminuria could be a valuable, readily available, and noninvasive tool for predicting LVH. These observations indicate a potential connection between microalbuminuria and LV hypertrophy in hypertension, suggesting a shared underlying pathological mechanism. A positive correlation was observed between microalbuminuria and LVMI as well as diastolic BP in this population. The established correlations necessitate further research to validate the link between microalbuminuria and LVH.

Ethics approval and Consent to participate

The present study was performed in the department of Medicine at S.N. Medical College, Agra, India. The study was performed in line with the principles of declaration of Helsinki. Approval was granted by the Ethics Committee (SNMC/IEC/Thesis/2023/174). Written informed consent was taken before enrolment.

Authors contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Dr. Govind Tripathi, Dr. Akhilesh Kumar Singh, Dr. Shobhit Shah, Dr. Akshyaya Pradhan. The first draft of manuscript was written by Dr. Shobhit Shah and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Conflicts of interest

There are no conflicts of interest.

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