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Original Research Article

MICROALBUMINURIA AND LEFT VENTRICULAR HYPERTROPHY IN NEWLY DIAGNOSED HYPERTENSIVE PATIENTS: A CROSS-SECTIONAL STUDY OF EARLY CARDIOVASCULAR–RENAL INTERACTION

Satyabrata Sahoo[1], Tushar Kantee Behera[2], Sunita Sethy[3], Aviram Panda[4]

[1-3]*Assistant Professor, Department of General Medicine, S.C.B., Medical College & Hospital, Cuttack, Odisha, India.*
[4]*Senior Resident, Department of Medicine, Fakir Mohan Medical College and Hospital, Balasore, India.*

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Corresponding Author:
Dr. Satyabrata Sahoo,
Assistant Professor, Department of General Medicine, S.C.B., Medical College & Hospital, Cuttack, Odisha, India.
Email: dr.satya143@gmail.com

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ABSTRACT

Background: Hypertension is a major contributor to cardiovascular and renal morbidity, with target organ damage often present at diagnosis. Microalbuminuria (MAU) and left ventricular hypertrophy (LVH) are early markers of renal and cardiac involvement, respectively, reflecting systemic vascular injury. This study aimed to evaluate the correlation between MAU and LVH in newly diagnosed hypertensive patients.

Materials and Methods: This cross-sectional observational study included 100 newly diagnosed hypertensive patients aged ≥18 years at a tertiary care hospital. Clinical evaluation, blood pressure measurement, and anthropometric assessment were performed. LVH was assessed using electrocardiography and echocardiography, with left ventricular mass index calculated using the Devereux formula. MAU was determined using the urine albumin-to-creatinine ratio (ACR). Statistical analysis included descriptive statistics and Chi-square/Fisher's exact tests, with $p<0.05$ considered significant.

Results: The mean age was 57.72 ± 9.14 years, with a predominance of males (64%). LVH and MAU were present in 52% and 36% of participants, respectively. A strong association was observed between MAU and LVH ($\chi^2=22.125$, $p<0.00001$), with 83.3% of MAU-positive individuals exhibiting LVH. Both MAU and LVH were significantly associated with advancing age and higher stages of hypertension. Pulse pressure was significantly associated with LVH ($p=0.0026$), while BMI was significantly associated with MAU ($p=0.009$).

Conclusions: MAU and LVH are highly prevalent and strongly interrelated in newly diagnosed hypertensive patients, reflecting early target organ damage. Their combined assessment may improve risk stratification and guide early therapeutic interventions.

Keywords: Hypertension; Microalbuminuria; Left ventricular hypertrophy; Target organ damage; Pulse pressure; Cardiovascular risk.

INTRODUCTION

Hypertension remains one of the leading global contributors to cardiovascular morbidity and mortality, accounting for a substantial burden of ischemic heart disease, stroke, and chronic kidney disease worldwide.[1,2] In low- and middle-income countries such as India, the prevalence of hypertension has risen steadily over the past decades, with a large proportion of cases remaining undiagnosed or inadequately controlled.[3,6] Importantly, hypertension is not merely a disorder of elevated blood pressure but a systemic vascular disease that leads to progressive target organ damage, particularly affecting the heart and kidneys.[12,13]

Left ventricular hypertrophy (LVH) is a well-established manifestation of cardiac target organ damage in hypertension and is associated with increased risk of adverse cardiovascular outcomes, including heart failure, arrhythmias, and sudden cardiac death.[12] LVH develops as an adaptive response to chronic pressure overload, resulting in structural and functional alterations in the myocardium. Parallel to cardiac involvement, microalbuminuria (MAU) reflects early renal injury and generalized endothelial dysfunction, serving as a sensitive marker of systemic vascular damage.[9] Increasing evidence suggests that MAU is not merely a renal parameter but an indicator of widespread microvascular pathology and heightened cardiovascular risk.[11]

The coexistence of LVH and MAU in hypertensive patients has been increasingly recognized as a marker of advanced target organ damage, reflecting shared underlying mechanisms such as endothelial dysfunction, oxidative stress, and activation of the renin–angiotensin–aldosterone system.[9,11] Previous studies have demonstrated a significant association between microalbuminuria and increased left ventricular mass, suggesting that these conditions may represent parallel manifestations of a common pathophysiological process.[10,11] Furthermore, arterial stiffness, as reflected by increased pulse pressure, has been implicated in the development of LVH by increasing left ventricular afterload, while metabolic factors such as elevated body mass index contribute to renal microvascular injury and albuminuria.[5,15]

Despite growing evidence from international studies, data exploring the relationship between MAU and LVH in newly diagnosed hypertensive patients, particularly in the Indian population, remain limited. Most available studies are either conducted in long-standing hypertensive cohorts or do not adequately account for early-stage disease, where identification of subclinical organ damage could have significant prognostic implications.[7,8] Early detection of both cardiac and renal involvement at the time of diagnosis is crucial, as it may facilitate timely intervention and prevent progression to overt cardiovascular and renal disease.

Given this background, the present study was undertaken to evaluate the correlation between microalbuminuria and left ventricular hypertrophy in newly diagnosed hypertensive patients attending a tertiary care hospital. By examining the association between these two markers of target organ damage, along with relevant clinical parameters such as age, body mass index, pulse pressure, and hypertension stage, this study aims to provide insights into the early interplay between cardiac and renal involvement in hypertension. Understanding this relationship may have important implications for risk stratification and integrated management strategies in hypertensive populations.

MATERIALS AND METHODS

This was a single-center, cross-sectional observational study conducted at SCB Medical College and Hospital, a tertiary care teaching institution in Cuttack, India. The study was carried out over a period of 15 months from April 2023 to June 2024. The study involved collaboration between the Departments of General Medicine and Cardiology.

The study protocol was approved by the Institutional Ethics Committee of SCB Medical College. Written informed consent was obtained from all participants prior to enrolment, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

A total of 100 newly diagnosed hypertensive patients aged ≥18 years were included in the study. Participants were recruited using a convenient sampling technique from patients attending the outpatient and inpatient services.

Inclusion Criteria: Newly diagnosed hypertension (blood pressure ≥140/90 mmHg) and age ≥18 years were included in the study.

Exclusion Criteria: Secondary hypertension; Diabetes mellitus; Known renal parenchymal disease unrelated to hypertension; Urinary tract infection; Heart failure; and Significant aortic valvular heart disease individuals were excluded from the study.

All participants underwent a detailed clinical assessment, including demographic profiling, anthropometric measurements, and comprehensive physical examination. Body mass index (BMI) was calculated using standard formulae.

Blood pressure was measured using a calibrated mercury sphygmomanometer with an appropriately sized cuff. Measurements were taken in the sitting position after adequate rest, with the back supported. Two readings were recorded on separate occasions 24 hours apart, and the average value was considered for analysis. Systolic and diastolic blood pressures were determined using Korotkoff sounds (Phase V for diastolic pressure), in accordance with established international guidelines. Hypertension was defined as blood pressure ≥140/90 mmHg.

A standard 12-lead electrocardiogram (ECG) was performed for all participants. Left ventricular hypertrophy was assessed using established voltage criteria, including the Sokolow–Lyon index (S in V1 + R in V5/V6 ≥35 mm or R in aVL ≥11 mm) and Cornell voltage criteria (S in V3 + R in aVL >28 mm in men and >20 mm in women).

Two-dimensional guided M-mode echocardiography was performed by an experienced cardiologist who was blinded to the clinical status of participants. Imaging was conducted using standard parasternal and apical views with the patient in the left lateral decubitus position. Measurements included left ventricular internal diameter in diastole (LVIDd),

interventricular septal thickness in diastole (IVSd), and posterior wall thickness in diastole (PWTd).

Left ventricular mass (LVM) was calculated using the Devereux formula:
$LVM\ (g) = 0.8 \times 1.04\ [(IVSd + LVIDd + PWTd)^3 - (LVIDd)^3] + 0.6$.

Left ventricular mass index (LVMI) was obtained by normalizing LVM to body surface area (1.73 m^2).

Relative wall thickness (RWT) was calculated as:
$RWT = (IVSd + PWTd) / LVIDd$

LVH was defined based on standard echocardiographic criteria.

Assessment of Microalbuminuria: Microalbuminuria was assessed using a morning spot urine sample. Urinary albumin-to-creatinine ratio (ACR) was measured using the turbidimetric sulfosalicylic acid method after excluding overt proteinuria and urinary sediment abnormalities. An ACR of 30–300 µg/mg was considered indicative of microalbuminuria.

Statistical analysis:

Data were analyzed using SPSS version 16 and Microsoft Excel 2021. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Comparisons between categorical variables were performed using the Chi-square test or Fisher's exact test as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1: Baseline Demographic and Clinical Characteristics (n = 100)

Variable	Category	n (%) / Mean ± SD	Range / Median
Age (years)	18–40	4 (4%)	
	41–50	15 (15%)	
	51–60	44 (44%)	
	>60	37 (37%)	
	Overall	57.72 ± 9.14	37–90 / 56
Sex	Male	64 (64%)	
	Female	36 (36%)	
BMI (kg/m²)	18.5–23	42 (42%)	
	>23	58 (58%)	
	Overall	24.27 ± 2.28	20.32–30 / 23.95
Hypertension Stage	Stage 1	67 (67%)	
	Stage 2	33 (33%)	
Pulse Pressure (mmHg)	≤40	6 (6%)	
	41–50	22 (22%)	
	51–60	49 (49%)	
	>60	23 (23%)	
Cardiac Geometry	Normal	48 (48%)	
	LVH	52 (52%)	
Microalbuminuria	Present	36 (36%)	
	Absent	64 (64%)	

Table 1 shows the baseline demographic and clinical characteristics. The study population (n=100) was predominantly older, with 81% aged ≥51 years and a mean age of 57.72 ± 9.14 years. Males constituted 64% of participants. A majority (58%) had BMI >23 kg/m², with an overall mean BMI of 24.27 ± 2.28 kg/m². Stage 1 hypertension was more common (67%) than Stage 2 (33%). Pulse pressure was most frequently in the 51–60 mmHg range (49%). Left ventricular hypertrophy (LVH) was present in 52% of subjects, while microalbuminuria (MAU) was observed in 36%. Overall, the cohort reflects an older, predominantly male hypertensive population with substantial cardiovascular and renal involvement.

Table 2: Comparison of Pulse Pressure and BMI Across Clinical Groups

Variable	Group	n	Pulse Pressure (Mean ± SD)	BMI (Mean ± SD)
MAU Status	Positive	36	60.44 ± 6.96	24.86 ± 2.25
	Negative	64	51.22 ± 6.28	23.90 ± 2.55
Cardiac Geometry	Normal	48	52.00 ± 6.06	23.99 ± 2.18
	LVH	52	56.69 ± 8.65	24.49 ± 2.38

Table 2 shows the comparison of pulse pressure and BMI across clinical groups. Comparison of clinical parameters revealed that participants with microalbuminuria had higher mean pulse pressure (60.44 ± 6.96 mmHg) compared to those without MAU (51.22 ± 6.28 mmHg). Similarly, BMI was slightly elevated in the MAU-positive group (24.86 ± 2.25 kg/m² vs 23.90 ± 2.55 kg/m²). Patients with LVH also demonstrated higher pulse pressure (56.69 ± 8.65 mmHg) compared to those with normal cardiac geometry (52.00 ± 6.06 mmHg), along with marginally higher BMI. These findings suggest that increased pulse pressure and BMI may be associated with target organ damage, particularly LVH and microalbuminuria, in hypertensive individuals.

Table 3: Key Associations between Clinical Variables

Association	Key Findings	Chi-square (χ^2)	p-value
Age vs MAU	MAU increases with age (69.4% in >60 yrs)	23.85	0.000027
Age vs LVH	LVH highest in >60 yrs (81.1%)	22.896	0.000042
HTN Stage vs LVH	LVH predominant in Stage 2 (90.9%)	29.87	<0.00001
HTN Stage vs MAU	MAU higher in Stage 2 (81.8%)	44.878	<0.00001
BMI vs LVH	No significant association	2.425	0.119
BMI vs MAU	Higher MAU in BMI >23 (46.6%)	6.67	0.009
Pulse Pressure vs LVH	Higher PP associated with LVH	14.211	0.0026
Pulse Pressure vs MAU	No association	0.885	0.82

Table 3 shows key associations between clinical variables. Significant associations were observed between multiple clinical variables. Advancing age showed strong correlation with both MAU (χ^2=23.85, p<0.001) and LVH (χ^2=22.896, p<0.001). Hypertension severity was a major determinant, with Stage 2 HTN strongly associated with LVH (χ^2=29.87, p<0.00001) and MAU (χ^2=44.878, p<0.00001). BMI was significantly associated with MAU (p=0.009) but not LVH (p=0.119). Pulse pressure showed a significant association with LVH (p=0.0026), but not with MAU (p=0.82). These findings indicate differential influences of hemodynamic and metabolic factors on cardiac and renal target organ damage.

Table 4: Interrelationship between LVH and MAU

Group	Normal n (%)	LVH n (%)	Total	Chi-square (χ^2)	p-value
MAU Negative	42 (65.6%)	22 (34.4%)	64		
MAU Positive	6 (16.7%)	30 (83.3%)	36	22.125	0.000003
Stage 1 HTN - MAU Negative	42 (72.4%)	16 (27.6%)	58		
Stage 1 HTN - MAU Positive	3 (33.3%)	6 (66.7%)	9	5.396	0.020
Stage 2 HTN - MAU Negative	0 (0%)	6 (100%)	6		
Stage 2 HTN - MAU Positive	3 (11.1%)	24 (88.9%)	27	0.733	0.391

Table 4 shows the interrelationship between LVH and microalbuminuria. A strong association was observed between microalbuminuria and left ventricular hypertrophy (χ^2=22.125, p<0.00001). Among MAU-positive individuals, 83.3% had LVH compared to 34.4% in MAU-negative subjects. In Stage 1 hypertension, MAU was significantly associated with LVH (p=0.020), indicating early coexistence of cardiac and renal damage. However, in Stage 2 hypertension, the association was not significant (p=0.391), likely due to the uniformly high prevalence of LVH. These findings suggest that MAU may serve as an early marker of cardiac structural changes, particularly in less advanced stages of hypertension.

DISCUSSION

The present study evaluated the interrelationship between microalbuminuria (MAU), left ventricular hypertrophy (LVH), pulse pressure, and metabolic parameters in hypertensive individuals. The high prevalence of LVH (52%) and MAU (36%) observed reflects substantial target organ damage in this cohort, consistent with the concept of hypertension as a systemic vascular disorder.[1,2]

The predominance of older individuals (mean age 57.72 years) aligns with prior Indian and international studies demonstrating that advancing age is a major determinant of both MAU and LVH.[3,4] The present study showed a strong association between age and MAU as well as LVH (p<0.001), supporting evidence that cumulative vascular injury, endothelial dysfunction, and arterial stiffness increase with age.[5] A multicentric Indian study reported similar findings, where both albuminuria and cardiac hypertrophy increased significantly beyond 50 years of age.[6]

The prevalence of MAU (36%) in this study is comparable to hospital-based Indian studies reporting rates between 30–40%,[7] though higher than community-based estimates (~20–25%).[8] This difference likely reflects inclusion of patients with more advanced hypertension. Microalbuminuria is increasingly recognized as a marker of generalized endothelial dysfunction rather than isolated renal involvement.[9]

A key finding of the present study is the strong association between MAU and LVH (p<0.00001), with 83.3% of MAU-positive individuals demonstrating LVH. Similar associations have been reported in both Indian and international studies.[10,11] A South Indian observational study showed that MAU independently predicts LVH, even after adjusting for confounders.[10] Likewise, a large cohort analysis demonstrated that albuminuria correlates with increased left ventricular mass index, suggesting shared pathophysiological pathways.[11] These findings reinforce the concept that MAU and LVH represent parallel manifestations of hypertension-mediated target organ damage.

Hypertension severity emerged as a major determinant of both cardiac and renal damage. In the present study, Stage 2 hypertension was strongly associated with LVH (90.9%) and MAU (81.8%), consistent with previous reports.[12] Elevated blood pressure increases afterload and promotes myocardial hypertrophy while simultaneously inducing glomerular hypertension and albumin leakage.[13] The absence of significant association

between MAU and LVH in Stage 2 hypertension in this study may be explained by a ceiling effect, where LVH prevalence is already high.

Pulse pressure, a surrogate marker of arterial stiffness, was significantly associated with LVH (p=0.0026), consistent with earlier studies demonstrating its role in increasing left ventricular workload.[14] However, no association was found between pulse pressure and MAU, suggesting that renal microvascular injury may be influenced more by endothelial dysfunction and metabolic factors than by large artery stiffness alone.[5]

BMI showed a significant association with MAU (p=0.009) but not with LVH. This finding is supported by recent studies indicating that obesity contributes to microalbuminuria through mechanisms such as insulin resistance, inflammation, and glomerular hyperfiltration.[15] However, LVH appears to be more strongly driven by hemodynamic load rather than metabolic factors alone.

The higher mean pulse pressure and BMI observed in MAU-positive and LVH groups further highlight the interplay between hemodynamic stress and metabolic risk factors. Contemporary literature suggests that combined exposure to increased arterial stiffness and metabolic dysregulation accelerates vascular aging and organ damage.[2,9]

Pathophysiologically, the coexistence of MAU and LVH can be explained by shared mechanisms including endothelial dysfunction, oxidative stress, and activation of the renin–angiotensin–aldosterone system. Microalbuminuria reflects systemic vascular permeability, while LVH represents myocardial adaptation to chronic pressure overload. Their coexistence indicates widespread vascular injury and increased cardiovascular risk.[11]

The present study has several limitations. First, its cross-sectional design precludes causal inference between MAU, LVH, and associated factors. Second, the relatively small sample size (n=100) limits generalizability. Third, potential confounders such as duration of hypertension, medication use (e.g., ACE inhibitors/ARBs), lipid profile, and glycemic status were not comprehensively analyzed. Fourth, single-time measurement of microalbuminuria may not accurately reflect persistent albumin excretion. Additionally, echocardiographic assessment of LVH was not supplemented with advanced imaging modalities, which may provide more precise quantification.

Future studies should adopt longitudinal designs to establish causal relationships between MAU and LVH progression. Large multicentric studies across diverse Indian populations are needed to improve external validity. Incorporation of biomarkers of oxidative stress, inflammation, and endothelial dysfunction would enhance mechanistic understanding. Repeated measurements of microalbuminuria and ambulatory blood pressure monitoring could provide more accurate assessment. Furthermore, interventional studies evaluating the impact of targeted therapies (e.g., RAAS inhibitors, SGLT2 inhibitors) on simultaneous regression of MAU and LVH would be valuable. Integration of cardiac and renal screening in routine hypertension management protocols is strongly recommended for early detection of subclinical organ damage.

CONCLUSION

This study demonstrates a high burden of hypertension-mediated target organ damage, with substantial prevalence of left ventricular hypertrophy and microalbuminuria. Advancing age and higher hypertension stages were the strongest determinants of both cardiac and renal involvement, while pulse pressure showed a significant association with LVH, reflecting the role of arterial stiffness. BMI was independently associated with microalbuminuria, highlighting the contribution of metabolic factors. The strong relationship between LVH and microalbuminuria underscores their shared pathophysiological basis and utility as complementary markers of systemic vascular injury. These findings support integrated cardiovascular and renal risk assessment for early detection and improved management of hypertensive patients.

Conflict of interest: There is no conflict of interest among the present study authors.

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