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Original Research Article

Microalbuminuria in Essential Hypertension and its Correlation with Left Ventricular Mass Index: A Cross-Sectional Study from a Tertiary Care Centre in South India

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Abstract

Background: Hypertension remains among the commonest diagnoses in Indian inpatient and outpatient practice and carries substantial morbidity and mortality through target organ damage. Left ventricular hypertrophy is the anatomical hallmark of hypertensive heart disease, while microalbuminuria is regarded as a marker of generalised endothelial dysfunction. Data correlating the two in Indian hypertensive cohorts remain limited.

Objectives: To estimate microalbuminuria in patients with essential hypertension, and to correlate microalbuminuria with left ventricular mass index in the same population.

Methods: A hospital-based cross-sectional study was conducted at hospitals attached to Bangalore Medical College and Research Institute between February 2021 and August 2022. Sixty adults aged above 18 years with essential hypertension defined by JNC 8 criteria were enrolled. Patients with diabetes mellitus, renal failure, obstructive uropathy, urinary tract infection, fever, overt proteinuria, valvular, congenital or coronary artery disease were excluded. Microalbuminuria was measured on a 24-hour urine collection and defined as 30 to 299 mg per 24 hours. Left ventricular mass was derived echocardiographically using the Devereux formula and indexed to body surface area. Analysis was performed using IBM SPSS version 26.

Results: Microalbuminuria was present in 51 of 60 patients (85.0%). Left ventricular mass index was normal in 31 (51.7%), mildly enlarged in 13 (21.7%), moderately enlarged in 7 (11.7%) and severely enlarged in 9 (15.0%). Every patient with any degree of left ventricular hypertrophy was microalbuminuric (p=0.019). Mean left ventricular mass index was 117.29±35.94 g/m² in microalbuminuric patients versus 71.18±5.51 g/m² in normoalbuminuric patients (p=0.001). Microalbuminuria correlated strongly with left ventricular mass (r=0.858, p=0.001) and with left ventricular mass index (r=0.732, p=0.001), and modestly with age (r=0.485, p=0.047).

Conclusion: Microalbuminuria is highly prevalent in essential hypertension and correlates strongly and positively with left ventricular mass index. It merits use as an inexpensive early screening marker for hypertensive cardiac target organ damage.

Keywords: Microalbuminuria; Essential Hypertension; Left Ventricular Mass Index; Left Ventricular Hypertrophy; Target Organ Damage; Endothelial Dysfunction.

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Introduction

Hypertension is among the most consequential non-communicable disease challenges of the present era, both because of its sheer prevalence and because of the silent, cumulative injury it inflicts on the heart, brain, kidneys and vasculature long before symptoms appear. The World Health Organization estimates that approximately 1.13 billion people worldwide live with raised blood pressure, and that roughly two-thirds of these individuals reside in low- and middle-income countries where detection and control rates remain poor.[1] Pooled analyses of population-based measurement studies encompassing more than nineteen million participants have demonstrated that while mean blood pressure has fallen in high-income regions over recent decades, the absolute number of adults with raised blood pressure has risen substantially, driven largely by demographic growth and by rising burdens in South Asia and sub-Saharan Africa.[2] This epidemiological

transition places countries such as India at the centre of the global hypertension burden.

Indian data reflect this pattern with considerable regional heterogeneity. A systematic review and meta-analysis of Indian studies reported an overall hypertension prevalence of approximately 30% among adults, with regional estimates ranging from 14.5% in rural north India to 31.7% in rural east India, and from 28.8% to 35.8% across urban regions.[3] More concerning than prevalence alone are the cascading deficits in awareness, treatment and control. In the same analysis, only about a quarter of rural Indians with hypertension were aware of their diagnosis, a similar proportion were receiving treatment, and roughly one in ten had blood pressure controlled to target; urban figures, while better, remained inadequate.[3] The practical consequence is that a large fraction of Indian hypertensive patients accumulate years of untreated or inadequately treated pressure load, and present to medical attention only when target organ damage is already established. Globally, hypertension has been estimated to affect more than a quarter of the adult population, with projections indicating further increase, and it consistently ranks among the leading attributable risk factors for death and disability.[4] The corollary is equally well demonstrated: meta-analyses of randomised trials show that every 10 mmHg reduction in systolic blood pressure produces significant proportional reductions in major cardiovascular events, coronary heart disease, stroke, heart failure and all-cause mortality.[5] Effective identification and risk stratification of hypertensive individuals therefore has direct and measurable clinical yield.

Within the spectrum of hypertensive target organ damage, left ventricular hypertrophy occupies a special position. It is the anatomical hallmark of hypertensive heart disease and represents the myocardial structural adaptation to chronically elevated afterload. Initially compensatory, this remodelling becomes maladaptive: myocyte hypertrophy outstrips capillary supply, interstitial fibrosis accumulates, coronary flow reserve declines, and diastolic relaxation is impaired. Clinically, left ventricular hypertrophy in hypertensive patients is an independent risk factor for ventricular arrhythmias, atrial fibrillation, heart failure and sudden cardiac death, and it carries prognostic weight over and above the blood pressure level that produced it. Accurate quantification of left ventricular mass is therefore central to cardiovascular risk assessment in hypertension.

Echocardiography remains the practical clinical standard for this purpose. The formula validated by Devereux and Reichek against post-mortem anatomical measurement derives left ventricular mass from interventricular septal thickness, left ventricular internal dimension and posterior wall thickness in diastole, and demonstrated close correlation with true anatomic mass across a wide range of values.[6] Because absolute mass scales with body size, it is conventionally indexed, most commonly to body surface area, to yield the left ventricular mass index. Contemporary chamber quantification recommendations from the American Society of Echocardiography and the European Association of Cardiovascular Imaging retain this approach and provide sex-specific partition values that permit grading of hypertrophy as mild, moderate or severe.[7] Echocardiographic assessment is considerably more sensitive than electrocardiography for the detection of hypertrophy, though it requires equipment and operator expertise that are not universally available at the primary and secondary care levels where most hypertensive patients in India are first encountered. This accessibility gap creates a clear clinical need for a simpler surrogate marker of early cardiac target organ damage.

Microalbuminuria has emerged as a leading candidate for that role. Defined conventionally as urinary albumin excretion between 30 and 300 mg per 24 hours, a range below the detection threshold of routine dipstick testing, microalbuminuria is understood not merely as an index of renal injury but as a window onto systemic vascular biology. The prevailing interpretation, consistent with the Steno hypothesis, holds that increased transglomerular albumin leak reflects a generalised increase in vascular permeability arising from endothelial dysfunction. Endothelial dysfunction in turn is characterised by reduced nitric oxide bioavailability, enhanced vasoconstrictor and pro-inflammatory signalling, and structural and functional abnormalities of the microvasculature. These changes raise peripheral vascular resistance, increase arterial stiffness and thereby elevate cardiac afterload, providing a coherent mechanistic pathway by which the same process that produces albumin leak in the glomerulus also drives hypertrophic remodelling of the left ventricle.

Reported prevalence of microalbuminuria in essential hypertension varies widely across the literature, from below 5% to nearly half of study populations, and this variability is itself informative. The MAGIC Study, which evaluated 787 untreated essential hypertensive patients using albumin-to-creatinine ratios on three non-consecutive first morning urine samples, reported a prevalence of only 6.7%, and found albuminuric patients to be characterised by higher blood pressure, higher body mass index, higher uric acid and an adverse lipid profile, as well as by greater frequency of electrocardiographic abnormalities and retinal vascular change.[8] Much higher figures

are reported where populations are older, hypertension is more severe or longer standing, or where measurement is performed on timed collections rather than spot samples. The determinants of this heterogeneity include age, sex, ethnicity, treatment status, duration and severity of hypertension, the assay employed, and the number of samples used to establish persistence. Any interpretation of prevalence figures must therefore be anchored to the methodology and case mix of the study reporting them.

The association between microalbuminuria and structural cardiac damage has been demonstrated in carefully matched comparisons. In a study of essential hypertensive patients matched for sex, age, duration of hypertension and body mass index, microalbuminuric patients showed significantly greater left ventricular mass index than normoalbuminuric hypertensive patients and normotensive controls, at 167±7, 139±9 and 118±5 g/m² respectively, together with greater carotid intima-media thickness and higher intrarenal vascular resistance.[9] This constellation supports the interpretation of microalbuminuria as a marker of diffuse rather than organ-specific damage.

The prognostic significance of albuminuria extends beyond structural correlates to hard clinical outcomes. In the PREVEND cohort drawn from a general population, urinary albumin excretion predicted all-cause mortality, with excess risk attributable predominantly to cardiovascular death, independent of conventional cardiovascular risk factors, and with the relationship evident even at albumin excretion levels then considered normal.[10] Among high-risk individuals enrolled in the HOPE trial, albuminuria was associated with increased risk of major cardiovascular events, death and hospitalisation for heart failure in both diabetic and non-diabetic participants, again in graded fashion.[11] Within hypertensive populations specifically, the LIFE study demonstrated that microalbuminuria in patients with electrocardiographic left ventricular hypertrophy clustered with other markers of cardiovascular risk and organ damage.[12]

Indian evidence, while growing, remains comparatively sparse and heterogeneous. A study of 150 non-diabetic hypertensive patients from Kerala reported microalbuminuria in 26.67% and found that echocardiographically proven left ventricular hypertrophy was markedly more frequent in microalbuminuric patients, with an odds ratio of 9.42, alongside similarly elevated odds for stroke and hypertensive retinopathy.[13]

Work from sub-Saharan Africa in newly diagnosed, treatment-naive hypertensive patients has reported microalbuminuria in approximately 40% of subjects, with concomitant left ventricular hypertrophy in a substantial proportion.[14] Nonetheless, most available Indian studies employ spot albumin-to-creatinine ratios rather than timed collections, and few report quantitative correlation coefficients between albumin excretion and left ventricular mass index in patients who have not yet developed clinically overt hypertensive heart disease.

It is against this background that the present study was designed. If microalbuminuria, measurable on a simple timed urine collection at minimal cost, tracks reliably with left ventricular mass index, then it offers a pragmatic screening instrument for the very setting in which echocardiography is least available and hypertensive target organ damage is most likely to go undetected. The present cross-sectional study was undertaken in patients with essential hypertension diagnosed by JNC 8 criteria[15] and without clinically manifest hypertensive heart disease, to quantify the burden of microalbuminuria and to define its relationship with left ventricular mass index in a South Indian tertiary care population.

Aims and Objectives

1. To estimate microalbuminuria in patients with essential hypertension.
2. To correlate microalbuminuria with left ventricular mass index in patients with essential hypertension.

Materials and Methods

Study Design and Setting: This was a hospital-based, observational, cross-sectional study conducted in the Department of General Medicine at hospitals attached to Bangalore Medical College and Research Institute, Bengaluru, Karnataka, India. Data were collected over an eighteen-month period from February 2021 to August 2022. Both outpatients attending the medicine outpatient department and patients admitted to the inpatient medical wards were eligible for enrolment.

Ethical Considerations: Approval and clearance were obtained from the Institutional Ethics Committee prior to commencement of the study. Written informed consent was obtained from every participant after explaining the nature, purpose and procedures of the study in a language the participant understood. Patients who declined consent were not enrolled. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Sample Size Calculation: The sample size was derived from the reported prevalence of microalbuminuria among newly diagnosed hypertensive patients in a previously published cross-sectional study.[14] The formula $n = Z^2pq/d^2$ was applied, where Z was the standard normal deviate for a 95% confidence interval (1.96), p was

the anticipated prevalence of microalbuminuria, q was 100 minus p, and d was the absolute precision set at 13%. Substitution yielded n = 54.32. An additional 10% was added to account for attrition, giving 59.75. The sample size was accordingly rounded to 60 subjects.

Inclusion Criteria: Patients were included if they were willing to provide written informed consent, were aged more than 18 years, and had hypertension diagnosed according to JNC 8 guidelines.[15]

Exclusion Criteria: Patients were excluded if they were unwilling to give informed consent, or if they had any of the following: renal failure or obstructive uropathy; urinary tract infection or febrile illness at the time of assessment; diabetes mellitus; overt proteinuria; valvular heart disease; congenital heart disease; or coronary artery disease. These exclusions were applied to isolate the effect of essential hypertension itself on albumin excretion and left ventricular geometry, and to remove alternative causes of albuminuria and of ventricular hypertrophy or dilatation.

Data Collection and Clinical Assessment: A structured case record proforma was used to record demographic details, duration of hypertension in years, current and past antihypertensive therapy, and relevant clinical history. Height in centimetres and weight in kilograms were measured using standard calibrated instruments, and body mass index was calculated as weight in kilograms divided by the square of height in metres. Blood pressure was recorded using a mercury sphygmomanometer with the patient seated and rested, and the mean of readings was used for analysis. Body surface area was calculated as the square root of the product of height in centimetres and weight in kilograms divided by 3600.

Estimation of Microalbuminuria: Microalbuminuria was estimated on a 24-hour urine collection. Participants were instructed in the correct technique for timed collection and were provided with appropriate containers.

Microalbuminuria was defined as urinary albumin excretion in the range of 30 to 299 mg per 24 hours. Values below 30 mg per 24 hours were classified as microalbuminuria absent, and patients with overt proteinuria were excluded at the screening stage as described above. Relevant baseline blood investigations, including renal function tests, were performed on all participants.

Echocardiographic Assessment: Two-dimensional and M-mode transthoracic echocardiography was performed on all participants. Interventricular septal thickness in diastole, left ventricular internal dimension in diastole and posterior wall thickness in diastole were measured. Left ventricular mass was calculated using the Devereux formula:[6]

$$\text{LV mass} = 0.80 \times 1.04 \times [(\text{IVSTd} + \text{LVIDd} + \text{PWTd})^3 - (\text{LVIDd})^3] + 0.6$$

where IVSTd is interventricular septal thickness in diastole, LVIDd is left ventricular internal dimension in diastole and PWTd is posterior wall thickness in diastole. Left ventricular mass index was then derived by dividing left ventricular mass by body surface area. Left ventricular mass index was categorised into four groups, namely normal, mildly enlarged, moderately enlarged and severely enlarged, using standard sex-specific partition values.[7]

Statistical Analysis: Data from the case record proforma were entered into a Microsoft Excel spreadsheet (version 2021) and analysed using IBM SPSS Statistics version 26. Normality of continuous data was assessed using the Kolmogorov–Smirnov test. Categorical variables were expressed as frequencies and percentages, and continuous variables as mean with standard deviation for normally distributed data, or as median with interquartile range for non-normally distributed data. Associations between categorical variables were tested using the Chi-square test, with Fisher's exact test applied where expected cell counts were small. Differences in means for normally distributed continuous variables were tested using the independent samples t-test for two groups and analysis of variance for more than two groups; the Mann–Whitney U test was applied for non-normally distributed continuous data. The relationship between 24-hour urinary albumin excretion and continuous variables including age, duration of hypertension, body mass index, left ventricular mass, left ventricular mass index and body surface area was assessed using the Pearson correlation coefficient. A p-value below 0.05 was considered statistically significant for all comparisons.

Results

Sixty subjects fulfilling the inclusion and exclusion criteria were enrolled and completed all study assessments.

Distribution of Left Ventricular Mass Index and Demographic Profile: Left ventricular mass index was normal in 31 subjects (51.7%), mildly enlarged in 13 (21.7%), moderately enlarged in 7 (11.7%) and severely enlarged in 9 (15.0%). Thus 29 of 60 subjects (48.3%) demonstrated some degree of left ventricular hypertrophy. The overall mean age of the cohort was 55±13 years, with the largest single stratum being the 50 to 59 year group (17 subjects, 28.3%). The distribution of left ventricular mass index categories across age strata is presented in Table 1.

Table 1: Distribution of left ventricular mass index categories across age groups (n=60)

Age (years)	Normal n (%)	Mildly enlarged n (%)	Moderately enlarged n (%)	Severely enlarged n (%)	Total n (%)
30–39	4 (12.9)	2 (15.4)	2 (28.6)	3 (33.3)	11 (18.3)
40–49	3 (9.7)	2 (15.4)	3 (42.9)	2 (22.2)	10 (16.7)
50–59	8 (25.8)	6 (46.2)	0 (0.0)	3 (33.3)	17 (28.3)
60–69	10 (32.3)	2 (15.4)	2 (28.6)	0 (0.0)	14 (23.3)
70–80	6 (19.4)	1 (7.7)	0 (0.0)	1 (11.1)	8 (13.3)
Total	31 (100.0)	13 (100.0)	7 (100.0)	9 (100.0)	60 (100.0)

Percentages are column percentages within each left ventricular mass index category.

The mean age did not differ significantly across left ventricular mass index categories. Subjects in the normal category had a mean age of 58±12 years, those with mild enlargement 53±12 years, those with moderate enlargement 49±12 years and those with severe enlargement 49±15 years, with p=0.736. Of the total cohort, 39 subjects (65.0%) were male and 21 (35.0%) were female. Males predominated within every abnormal left ventricular mass index category, accounting for 76.9% of the mildly enlarged, 85.7% of the moderately enlarged and 77.8% of the severely enlarged groups, compared with 51.6% of the normal category; this gradient did not reach statistical significance (p=0.156). Similarly, the distribution of microalbuminuria between the sexes was not significantly different, with 16 of 21 women (76.2%) and 35 of 39 men (89.7%) being microalbuminuric (p=0.161).

Prevalence and Distribution of Microalbuminuria: Microalbuminuria was detected in 51 of 60 subjects, giving an overall prevalence of 85.0%, while 9 subjects (15.0%) were normoalbuminuric. The distribution of microalbuminuria across age strata is shown in Table 2. Microalbuminuric subjects were concentrated in the middle and later decades, with 16 of 51 (31.4%) in the 50 to 59 year band and 13 of 51 (25.5%) in the 60 to 69 year band. Mean age was 56±19 years in normoalbuminuric subjects and 54±12 years in microalbuminuric subjects, a difference that was not statistically significant (p=0.983).

Table 2: Distribution of microalbuminuria across age groups (n=60)

Age (years)	Microalbuminuria absent n (%)	Microalbuminuria present n (%)	Total n (%)
30–39	3 (33.3)	8 (15.7)	11 (18.3)
40–49	1 (11.1)	9 (17.6)	10 (16.7)
50–59	1 (11.1)	16 (31.4)	17 (28.3)
60–69	1 (11.1)	13 (25.5)	14 (23.3)
70–80	3 (33.3)	5 (9.8)	8 (13.3)
Total	9 (100.0)	51 (100.0)	60 (100.0)

Percentages are column percentages within each microalbuminuria category.

Relationship Between Microalbuminuria and Left Ventricular Mass Index Category: The central finding of the study is presented in Table 3. Among the 31 subjects with a normal left ventricular mass index, 9 (29.0%) were normoalbuminuric and 22 (71.0%) were microalbuminuric. In striking contrast, every one of the 29 subjects with any degree of left ventricular hypertrophy, whether mild, moderate or severe, was microalbuminuric.

All 9 normoalbuminuric subjects in the cohort therefore fell within the normal left ventricular mass index category. This association was statistically significant (p=0.019).

Table 3: Microalbuminuria status by left ventricular mass index category (n=60)

Left ventricular mass index category	Microalbuminuria absent n (%)	Microalbuminuria present n (%)	Total n	p-value
Normal	9 (29.0)	22 (71.0)	31	
Mildly enlarged	0 (0.0)	13 (100.0)	13	
Moderately enlarged	0 (0.0)	7 (100.0)	7	0.019
Severely enlarged	0 (0.0)	9 (100.0)	9	
Total	9 (15.0)	51 (85.0)	60	

Percentages are row percentages within each left ventricular mass index category. Chi-square test.

A graded relationship was likewise evident when albumin excretion was analysed as a continuous variable across left ventricular mass index categories. Mean 24-hour urinary albumin excretion rose progressively from 45±21 mg in the normal category, to 100±17 mg in the mildly enlarged category, 103±6 mg in the moderately enlarged category and 121±38 mg in the severely enlarged category, against an overall cohort mean of 75±39 mg per 24 hours (p=0.001). Mean left ventricular mass showed a parallel gradient, rising from 145.35±28.26 g in the normal category to 168.90±29.97 g, 186.33±36.84 g and 215.67±32.63 g in the mildly, moderately and severely enlarged categories respectively (p=0.001).

Comparison of Clinical and Echocardiographic Variables by Microalbuminuria Status: Table 4 summarises the comparison of continuous clinical and echocardiographic variables between subjects with and without microalbuminuria. Both left ventricular mass and left ventricular mass index were markedly higher in the microalbuminuric group. Mean left ventricular mass was 201.62±58.60 g in microalbuminuric subjects versus 128.27±16.89 g in normoalbuminuric subjects (p=0.001), and mean left ventricular mass index was 117.29±35.94 g/m² versus 71.18±5.51 g/m² respectively (p=0.001).

Systolic and diastolic blood pressures were both significantly higher among microalbuminuric subjects, at 139.0±14.7 mmHg and 90.1±12.3 mmHg respectively, compared with 120.0±7.1 mmHg and 76.7±5.0 mmHg in normoalbuminuric subjects (p=0.001 for both).

By contrast, age, duration of hypertension, body mass index and body surface area did not differ significantly between the two groups. Mean duration of hypertension was numerically longer in microalbuminuric subjects at 7.4±5.5 years compared with 4.5±3.3 years, but this difference did not reach significance (p=0.752). Mean body mass index was marginally lower in microalbuminuric subjects at 23.37±2.87 kg/m² versus 24.50±2.94 kg/m² (p=0.534), and mean body surface area was 1.739±0.181 m² versus 1.784±0.160 m² (p=0.073).

Table 4: Comparison of clinical and echocardiographic variables by microalbuminuria status (n=60)

Variable	Microalbuminuria absent (n=9) Mean±SD	Microalbuminuria present (n=51) Mean±SD	p-value
Age (years)	56±19	54±12	0.983
Duration of hypertension (years)	4.5±3.3	7.4±5.5	0.752
Body mass index (kg/m²)	24.50±2.94	23.37±2.87	0.534
Body surface area (m²)	1.784±0.160	1.739±0.181	0.073
Systolic blood pressure (mmHg)	120.0±7.1	139.0±14.7	0.001
Diastolic blood pressure (mmHg)	76.7±5.0	90.1±12.3	0.001
Left ventricular mass (g)	128.27±16.89	201.62±58.60	0.001
Left ventricular mass index (g/m²)	71.18±5.51	117.29±35.94	0.001

SD, standard deviation. Independent samples t-test.

Clinical Variables Across Left Ventricular Mass Index Categories: Table 5 presents the distribution of continuous clinical variables across the four left ventricular mass index categories. A clear and statistically significant blood pressure gradient was observed. Mean systolic blood pressure rose from 124.8±7.7 mmHg in the normal category through 141.4±6.9 mmHg and 150.0±10.0 mmHg to 156.7±14.1 mmHg in the severely enlarged category and mean diastolic blood pressure rose correspondingly from 78.6±7.0 mmHg to 104.4±7.3 mmHg (p=0.001 for both). Twenty-four-hour urinary albumin excretion showed a parallel and highly significant stepwise increase across categories (p=0.001). Neither duration of hypertension (p=0.525), body mass index (p=0.281) nor body surface area (p=0.098) differed significantly across categories.

Table 5: Clinical, biochemical and echocardiographic variables across left ventricular mass index categories (n=60)

Variable	Normal (n=31)	Mildly enlarged (n=13)	Moderately enlarged (n=7)	Severely enlarged (n=9)	Total (n=60)	p-value
Age (years)	58±12	53±12	49±12	49±15	55±13	0.736
Duration of hypertension (years)	6.8±5.3	9.5±6.9	5.7±3.6	5.0±2.7	7.0±5.3	0.525
Body mass index (kg/m²)	23.64±3.22	23.81±2.66	23.17±2.68	23.06±2.43	23.54±2.88	0.281
Body surface area (m²)	1.712±0.199	1.795±0.158	1.790±0.142	1.757±0.145	1.746±0.178	0.098
Systolic BP (mmHg)	124.8±7.7	141.4±6.9	150.0±10.0	156.7±14.1	136.1±15.4	0.001
Diastolic BP (mmHg)	78.6±7.0	93.8±6.5	98.6±6.9	104.4±7.3	88.1±12.4	0.001
Left ventricular mass (g)	145.35±28.26	168.90±29.97	186.33±36.84	215.67±32.63	—	0.001
Microalbuminuria (mg/24 h)	45±21	100±17	103±6	121±38	75±39	0.001

Values expressed as mean±standard deviation. BP, blood pressure. Analysis of variance.

Antihypertensive Treatment Status: Fifty-eight of the 60 subjects (96.7%) were receiving antihypertensive therapy, most commonly calcium channel blockers or agents acting on the renin angiotensin system. All subjects in the normal, mildly enlarged and moderately enlarged left ventricular mass index categories were on treatment, whereas 2 of the 9 subjects (22.2%) in the severely enlarged category were untreated, and this difference reached statistical significance (p=0.008). Treatment status did not differ significantly between microalbuminuric and normoalbuminuric subjects (p=0.546). These findings are summarised in Table 6.

Table 6: Antihypertensive treatment status by left ventricular mass index category and by microalbuminuria status (n=60)

Group	On treatment n (%)	Not on treatment n (%)	p-value
LVMI normal (n=31)	31 (100.0)	0 (0.0)	
LVMI mildly enlarged (n=13)	13 (100.0)	0 (0.0)	0.008
LVMI moderately enlarged (n=7)	7 (100.0)	0 (0.0)	
LVMI severely enlarged (n=9)	7 (77.8)	2 (22.2)	
Microalbuminuria absent (n=9)	9 (100.0)	0 (0.0)	0.546
Microalbuminuria present (n=51)	49 (96.1)	2 (3.9)	

LVMI, left ventricular mass index. Chi-square test.

Correlation Analysis: Pearson correlation coefficients between 24-hour urinary albumin excretion and the studied continuous variables are presented in Table 7. The strongest associations were with the two cardiac structural parameters. Left ventricular mass correlated with 24-hour albumin excretion at r=0.858 (p=0.001), and left ventricular mass index at r=0.732 (p=0.001), both representing strong positive relationships.

A moderate positive correlation was observed with age (r=0.485, p=0.047). Duration of hypertension (r=0.059, p=0.652), body mass index (r=−0.040, p=0.791) and body surface area (r=0.083, p=0.528) showed no significant correlation with albumin excretion.

Table 7: Pearson correlation of 24-hour urinary albumin excretion with clinical and echocardiographic variables (n=60)

Variable correlated with microalbuminuria (mg/24 h)	Pearson correlation coefficient (r)	p-value
Age (years)	0.485	0.047
Duration of hypertension (years)	0.059	0.652
Body mass index (kg/m²)	−0.040	0.791
Left ventricular mass (g)	0.858	0.001
Left ventricular mass index (g/m²)	0.732	0.001
Body surface area (m²)	0.083	0.528

Statistically significant at p<0.05.

Discussion

The principal findings of this cross-sectional study of 60 patients with essential hypertension are threefold. First, microalbuminuria was present in 85.0% of the cohort. Second, every patient with any grade of left ventricular hypertrophy was microalbuminuric, so that normoalbuminuria was confined entirely to patients with a normal left ventricular mass index. Third, 24-hour urinary albumin excretion correlated strongly and positively with both left ventricular mass (r=0.858) and left ventricular mass index (r=0.732), with mean albumin excretion rising in stepwise fashion across the grades of hypertrophy from 45 mg to 121 mg per 24 hours.

The prevalence of microalbuminuria observed here is substantially higher than several widely cited figures and warrants careful interpretation rather than uncritical acceptance. The MAGIC Study reported microalbuminuria in only 6.7% of 787 untreated essential hypertensive patients, using albumin-to-creatinine ratios measured on three non-consecutive first morning urine specimens.[8] Indian data occupy an intermediate position: a Kerala series of 150 non-diabetic hypertensive patients reported a prevalence of 26.67%,[13] an eastern Indian tertiary hospital study of 100 hypertensive patients reported 36%,[16] and a study from Agra of 125 essential hypertensive patients meeting JNC 8 criteria reported 40.8%.[17] A prospective series of 50 treatment-naive Indian hypertensive patients likewise examined the correlation of microalbuminuria with left ventricular hypertrophy and carotid intima-media thickness.[18] African data from newly diagnosed, treatment-naive patients report approximately 39.5%.[14] Population-based work has demonstrated how strongly prevalence estimates depend on the diagnostic criteria applied, with the reported figure shifting several-fold according to whether spot, overnight or timed collections and which cut-offs are used.[19]

Three methodological features plausibly account for the higher prevalence in the present cohort. The use of a quantitative 24-hour timed collection rather than a spot albumin-to-creatinine ratio removes the dilutional and diurnal variability of untimed samples and is likely to detect abnormal excretion that spot testing misses. The setting was a tertiary referral centre drawing both outpatients and inpatients, so the case mix was weighted toward established rather than incident hypertension, with a mean duration of seven years and 96.7% of subjects already on antihypertensive therapy. Finally, mean systolic blood pressure in the cohort was 136.1 mmHg despite treatment, indicating incomplete control and a persistent haemodynamic load on the glomerular capillary bed. Prevalence figures across the literature are known to range from below 5% to nearly half of study populations for precisely these reasons, and the present figure should be read as characteristic of a treated but imperfectly controlled tertiary care population rather than as a community estimate.

The relationship between microalbuminuria and left ventricular hypertrophy in this study is both categorical and quantitative, and it is here that the findings align most closely with the published literature. The observation that all 29 patients with left ventricular hypertrophy were microalbuminuric mirrors the direction, though not the absolute magnitude, of the Agra series, in which 72% of patients with left ventricular hypertrophy were microalbuminuric and albumin excretion was significantly related to both left ventricular mass index and diastolic blood pressure.[17] The Kerala series quantified the same association as an odds ratio of 9.42 for echocardiographically proven left ventricular hypertrophy in microalbuminuric patients.[13] A South Indian observational study from Kochi demonstrated that microalbuminuria was associated with concentric left ventricular hypertrophy (p=0.003) and, on multivariate regression, emerged as an independent risk factor for concentric hypertrophy alongside serum creatinine and diastolic blood pressure (p=0.016).[20] Taken together with the present data, these studies converge on the same conclusion from different Indian populations using different assay methods.

The magnitude of the difference in left ventricular mass index between albuminuric and non-albuminuric patients in the present study, 117.29±35.94 g/m² versus 71.18±5.51 g/m², is larger than that reported in carefully matched European comparisons, where microalbuminuric hypertensive patients showed a left ventricular mass index of 167±7 g/m² against 139±9 g/m² in normoalbuminuric hypertensive patients and 118±5 g/m² in normotensive controls.[9] The absolute values differ because that study selected patients after pharmacological washout and matched groups for age, sex, duration of hypertension and body mass index, whereas the present cohort was unselected and predominantly treated. The direction and the significance of the difference are nonetheless identical, and the very tight standard deviation in the normoalbuminuric group here (5.51 g/m²) reflects the fact that all nine such patients clustered within the normal range of ventricular mass.

The correlation coefficient of 0.732 between albumin excretion and left ventricular mass index is stronger than most previously reported values, and this deserves comment rather than celebration. Two features of the design plausibly inflate it. Albumin excretion was quantified continuously on a timed collection rather than dichotomised, and the cohort

spanned a wide range of ventricular mass, from clearly normal to severely hypertrophied, which mechanically widens the observed correlation relative to studies restricted to mild hypertension. The finding should therefore be presented as internally consistent and biologically plausible rather than as evidence of a stronger underlying relationship than that established in larger series.

The blood pressure gradient across left ventricular mass index categories was steep and highly significant, with mean systolic pressure rising from 124.8 mmHg in the normal category to 156.7 mmHg in the severely enlarged category and diastolic pressure from 78.6 mmHg to 104.4 mmHg ($p=0.001$ for both). Microalbuminuric patients likewise had significantly higher systolic and diastolic pressures than normoalbuminuric patients. This is consistent with the interpretation that haemodynamic load is the shared driver of both glomerular albumin leak and ventricular remodelling, and with the observation from the Agra cohort that diastolic pressure in particular tracked with albumin excretion.[17] It also cautions against treating microalbuminuria as an entirely blood pressure independent marker in a cross-sectional design of this size, since the two are not statistically separable here.

Several associations reported in larger studies were not reproduced. Duration of hypertension, body mass index and body surface area showed no significant relationship with albumin excretion, whereas the Kerala series found microalbuminuria significantly more frequent with longer duration and greater severity of hypertension ($p<0.001$ for each) and with higher body mass index ($p<0.04$),[13] and the MAGIC Study identified body mass index and uric acid among the clinical correlates of albuminuria.[8] Two explanations are likely. A sample of 60 with only nine normoalbuminuric subjects has limited power to detect modest differences, and the highly unequal group sizes render the comparisons involving the normoalbuminuric group statistically fragile. In addition, the narrow body mass index distribution in this cohort, with an overall mean of 23.54±2.88 kg/m² and little representation at the obese end of the range, leaves insufficient variance for an adiposity signal to emerge. The moderate correlation with age ($r=0.485$, $p=0.047$) despite the absence of a significant difference in mean age between albuminuric and non-albuminuric groups is consistent with a graded rather than threshold effect of age on albumin excretion.

The mechanistic account that best fits these observations is that of microalbuminuria as a renal manifestation of generalised endothelial dysfunction. Increased transglomerular passage of albumin reflects loss of the charge and size selectivity of the glomerular barrier under the combined influence of raised intraglomerular pressure, angiotensin II mediated efferent arteriolar constriction, and endothelial injury with reduced nitric oxide bioavailability. The same endothelial and microvascular abnormalities raise systemic vascular resistance and arterial stiffness, increasing afterload and driving concentric hypertrophic remodelling of the left ventricle, with activation of the renin angiotensin aldosterone system and profibrotic signalling contributing to interstitial fibrosis. On this account, microalbuminuria and left ventricular hypertrophy are parallel expressions of a single vascular pathology rather than one causing the other, which is consistent with the cross-sectional association demonstrated here and with the inability of this design to establish temporal precedence.

The clinical relevance of these findings rests on the established prognostic weight of albuminuria. Urinary albumin excretion predicts all-cause and cardiovascular mortality in general populations independently of conventional risk factors, and does so at levels of excretion previously regarded as normal.[10] Among high-risk individuals, albuminuria predicts major cardiovascular events, death and hospitalisation for heart failure in both diabetic and non-diabetic patients.[11] In hypertensive patients with electrocardiographic left ventricular hypertrophy, microalbuminuria clusters with other markers of cardiovascular risk and organ damage.[12] If a quantitative urine test identifies the same patients whom echocardiography would classify as hypertrophied, then in settings where echocardiography is unavailable, microalbuminuria offers a defensible triage instrument for identifying which hypertensive patients most urgently require intensified blood pressure control, preferential use of renin angiotensin system blockade, and onward referral for cardiac imaging.

Limitations: Several limitations temper these conclusions. The cross-sectional design permits no inference about causality or temporal sequence between albumin excretion and ventricular remodelling. The sample of 60 was calculated for prevalence estimation rather than for correlation or subgroup analysis, and the resulting group of only nine normoalbuminuric subjects makes several comparisons underpowered and their p-values unstable. Microalbuminuria was established on a single 24-hour collection rather than the two or three separate collections generally recommended to confirm persistence, so some proportion of the observed positivity may represent transient albuminuria. The study was conducted at a single tertiary centre, and 96.7% of subjects were already receiving antihypertensive therapy, including agents that modify albumin excretion, which limits generalisability to treatment-naive and community populations. No normotensive control group was

included. Ambulatory blood pressure monitoring was not performed, so the contribution of nocturnal non-dipping and true pressure load could not be assessed. Finally, no multivariable adjustment was undertaken, so the observed correlations cannot be regarded as independent of blood pressure or age.

Recommendations: Prospective longitudinal studies with larger samples, treatment-naive participants, repeated timed urine collections and multivariable adjustment for blood pressure, age and renal function are required to determine whether microalbuminuria precedes, accompanies or follows the development of left ventricular hypertrophy in Indian hypertensive populations, and whether regression of albuminuria on therapy is accompanied by regression of ventricular mass.

Conclusion

In this cross-sectional study of 60 patients with essential hypertension, microalbuminuria was present in 85.0% of subjects and left ventricular hypertrophy of some grade in 48.3%. The two findings were closely linked: every patient with an elevated left ventricular mass index, whether mildly, moderately or severely elevated, was microalbuminuric, and normoalbuminuria was found exclusively among patients with normal ventricular mass (p=0.019). Mean 24-hour urinary albumin excretion increased in stepwise fashion across the grades of hypertrophy, from 45 mg in patients with normal mass to 121 mg in those with severe enlargement (p=0.001), and mean left ventricular mass index was substantially higher in microalbuminuric than in normoalbuminuric patients (117.29±35.94 versus 71.18±5.51 g/m², p=0.001).

Quantitatively, 24-hour albumin excretion correlated strongly and positively with left ventricular mass (r=0.858, p=0.001) and with left ventricular mass index (r=0.732, p=0.001), and moderately with age (r=0.485, p=0.047), while showing no significant correlation with duration of hypertension, body mass index or body surface area. Both systolic and diastolic blood pressures were significantly higher in microalbuminuric patients and rose progressively across the grades of hypertrophy, indicating that haemodynamic load is a shared determinant of both abnormalities.

These findings support the interpretation of microalbuminuria as an early and readily measurable marker of hypertensive target organ damage, reflecting a generalised endothelial and microvascular disturbance of which left ventricular hypertrophy is the cardiac expression. Given that echocardiography is not universally accessible at the primary and secondary care levels at which most Indian hypertensive patients are managed, quantitative estimation of urinary albumin excretion offers a practical and inexpensive means of identifying those patients who require intensified antihypertensive therapy and onward cardiac evaluation. More extensive screening for microalbuminuria in hypertensive subjects is therefore recommended to permit better stratification of absolute cardiovascular risk. The correlation demonstrated here warrants confirmation in larger prospective studies with treatment-naive participants and multivariable adjustment before microalbuminuria can be adopted as a stand-alone surrogate for left ventricular mass.

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