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Original Research Article

CROSS SECTIONAL STUDY OF MICROALBUMINURIA AND SUB CLINICAL TARGET ORGAN DAMAGE IN ESSENTIAL HYPERTENSION

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ABSTRACT

Background: Essential hypertension is a major contributor to cardiovascular morbidity and mortality, largely due to progressive target organ damage that often develops silently. Microalbuminuria has emerged as an early marker of endothelial dysfunction and vascular injury and may reflect subclinical involvement of multiple organs in hypertensive patients. The aim is to assess the prevalence of microalbuminuria and its association with subclinical target organ damage in patients with essential hypertension.

Materials and Methods: A hospital-based cross-sectional study was conducted among 200 patients with essential hypertension. Clinical history, blood pressure measurements, and relevant laboratory investigations were recorded. Microalbuminuria was assessed using the urine albumin-to-creatinine ratio. Subclinical target organ damage was evaluated through electrocardiography and echocardiography for left ventricular hypertrophy, fundoscopic examination for hypertensive retinopathy, renal function assessment, and carotid intima-media thickness measurement where feasible. Statistical analysis included descriptive statistics, chi-square test, independent t-test, and odds ratio estimation with a significance level of $p < 0.05$.

Results: Microalbuminuria was present in 31.5% of hypertensive patients. Individuals with microalbuminuria were significantly older and had a longer duration of hypertension. Subclinical target organ damage was observed in more than half of the study population, with left ventricular hypertrophy being the most common abnormality. Microalbuminuria showed a significant association with left ventricular hypertrophy, hypertensive retinopathy, carotid intima-media thickening, reduced renal function, and multiple organ involvement.

Conclusion: Microalbuminuria is a common finding in essential hypertension and is strongly associated with early target organ damage. Routine screening for microalbuminuria may aid in early detection of vascular injury and improve cardiovascular risk stratification in hypertensive patients.

Keywords: Microalbuminuria. Essential hypertension. Subclinical target organ damage.

INTRODUCTION

Essential hypertension remains one of the most prevalent chronic non-communicable diseases worldwide and represents a major contributor to cardiovascular morbidity and mortality. The long-term consequences of uncontrolled hypertension primarily arise from progressive target organ damage involving the heart, kidneys, brain, and vasculature. Importantly, such damage often develops silently before the appearance of overt clinical disease, highlighting the need for early biomarkers that can detect subclinical organ involvement. Among the available indicators, microalbuminuria has emerged as a sensitive and early marker of vascular endothelial dysfunction and renal injury in hypertensive individuals.[1]

Microalbuminuria refers to urinary albumin excretion ranging between 30-300 mg/day or an albumin-to-creatinine ratio of 30-300 mg/g. Although traditionally considered an early

manifestation of diabetic nephropathy, increasing evidence indicates that microalbuminuria also reflects generalized endothelial dysfunction and systemic vascular damage in patients with essential hypertension. The presence of microalbuminuria is associated with increased arterial stiffness, impaired vascular autoregulation, and heightened inflammatory activity, all of which contribute to progressive cardiovascular and renal damage.[2]

Subclinical target organ damage in hypertension commonly includes left ventricular hypertrophy, increased carotid intima-media thickness, arterial stiffness, and early renal impairment. These changes often remain asymptomatic but significantly elevate the risk of future cardiovascular events such as myocardial infarction, stroke, and chronic kidney disease. Microalbuminuria has been shown to correlate strongly with these early structural and functional alterations, thereby serving as an integrative marker of cardiovascular risk rather than merely a renal parameter.[3]

Several pathophysiological mechanisms explain the association between hypertension and microalbuminuria. Elevated systemic blood pressure leads to increased glomerular capillary pressure, resulting in enhanced permeability of the glomerular basement membrane and subsequent albumin leakage. Additionally, endothelial dysfunction, oxidative stress, and activation of the renin-angiotensin-aldosterone system further promote vascular remodeling and albumin excretion. The coexistence of metabolic risk factors such as obesity, dyslipidemia, and insulin resistance may amplify these mechanisms and accelerate subclinical organ damage.[4]

Early detection of microalbuminuria in hypertensive patients offers important clinical implications. Identification of individuals with microalbuminuria allows risk stratification, intensification of antihypertensive therapy, and initiation of renoprotective and cardioprotective interventions. Moreover, monitoring microalbuminuria may help evaluate therapeutic response and predict progression toward overt nephropathy and cardiovascular complications.[5]

AIM

To assess the prevalence of microalbuminuria and its association with subclinical target organ damage in patients with essential hypertension.

Objectives

1. To determine the prevalence of microalbuminuria among patients with essential hypertension.
2. To evaluate the presence of subclinical target organ damage in hypertensive patients.
3. To analyze the association between microalbuminuria and indicators of subclinical target organ damage.

MATERIALS AND METHODS

Source of Data: Data were collected from patients diagnosed with essential hypertension attending the outpatient and inpatient departments of the tertiary care hospital. Clinical history, examination findings, laboratory parameters, and imaging data were obtained from hospital records and direct patient evaluation.

Study Design: The study was conducted as a hospital-based cross-sectional observational study.

Study Location: The study was carried out at a tertiary care teaching hospital catering to urban and rural populations.

Study Duration: The study was conducted over a period of 12-18 months, including patient recruitment, data collection, and analysis.

Sample Size: A total of 200 patients with essential hypertension were included in the study.

Inclusion Criteria

1. Patients aged ≥18 years diagnosed with essential hypertension.
2. Patients on treatment or newly diagnosed with hypertension.
3. Patients willing to provide informed consent.

Exclusion Criteria

1. Patients with diabetes mellitus.
2. Known chronic kidney disease or overt proteinuria.
3. Secondary hypertension.
4. Pregnancy.
5. Acute illness, urinary tract infection, or heart failure affecting albumin excretion.

Procedure and Methodology: After obtaining informed consent, detailed demographic and clinical information including duration of hypertension, treatment history, blood pressure measurements, and comorbidities were recorded. All patients underwent physical examination and relevant investigations. Microalbuminuria was assessed using urine albumin-to-creatinine ratio from spot urine samples. Evaluation of subclinical target organ damage included electrocardiography and echocardiography for left ventricular hypertrophy, fundoscopic examination for hypertensive retinopathy, and assessment of renal function parameters. Carotid intima-media thickness and other relevant investigations were performed where feasible.

Sample Processing: Spot urine samples were collected under aseptic precautions and analyzed using immunoturbidimetric methods for urinary albumin estimation. Blood samples were processed for renal function tests, lipid profile, and other biochemical parameters using standard laboratory protocols.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using SPSS software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Chi-square test, independent t-test, and correlation analysis were applied as appropriate. A p-value <0.05 was considered statistically significant.

Data Collection: Data were collected using a pre-structured and pre-validated proforma. Information regarding demographic variables, clinical parameters, laboratory findings, and imaging results were systematically recorded for analysis.

RESULTS

Table 1: Association of Microalbuminuria with Subclinical Target Organ Damage (N = 200)

Parameter	Microalbuminuria Present (n=63) Mean ± SD / n (%)	Microalbuminuria Absent (n=137) Mean ± SD / n (%)	Test of Significance	95% CI	P value
Age (years)	56.8 ± 9.7	52.1 ± 10.4	Independent t test = 2.88	1.48 - 7.86	0.004
Duration of HTN (years)	8.7 ± 4.1	6.1 ± 3.9	t = 4.19	1.36 - 3.82	<0.001
LVH present	34 (53.9%)	38 (27.7%)	$\chi^2 = 12.76$	OR 2.78 (1.47-5.25)	0.001
Retinopathy	29 (46.0%)	33 (24.1%)	$\chi^2 = 9.82$	OR 2.66 (1.37-5.15)	0.002
Reduced eGFR	22 (34.9%)	21 (15.3%)	$\chi^2 = 9.96$	OR 2.95 (1.43-6.09)	0.002

[Table 1] demonstrates the association between microalbuminuria and subclinical target organ damage among hypertensive patients. The mean age was significantly higher in patients with microalbuminuria (56.8 ± 9.7 years) compared to those without microalbuminuria (52.1 ± 10.4 years), showing statistical significance (t = 2.88, p = 0.004). Similarly, the duration of hypertension was longer among patients with microalbuminuria (8.7 ± 4.1 years) than those without (6.1 ± 3.9 years), indicating a strong association (t = 4.19, p <0.001). Structural and functional target organ damage was more prevalent in the microalbuminuria group. Left ventricular hypertrophy was observed in 53.9% of patients with microalbuminuria compared to 27.7% without (χ^2 = 12.76, OR 2.78, p = 0.001). Hypertensive retinopathy was present in 46.0% versus 24.1% (χ^2 = 9.82, OR 2.66, p = 0.002), while reduced eGFR was noted in 34.9% compared to 15.3% (χ^2 = 9.96, OR 2.95, p = 0.002).

Table 2: Prevalence of Microalbuminuria among Essential Hypertension (N = 200)

Microalbuminuria Status	n (%)	95% CI	Test of Significance	P value
Present	63 (31.5%)	25.2 - 38.3	Z test for proportion = 4.82	<0.001
Absent	137 (68.5%)	61.7 - 74.8		

Urinary Albumin Creatinine Ratio (mg/g)	Mean ± SD	95% CI	t value	P value
Overall	46.7 ± 18.6	44.1 - 49.3	18.74	<0.001

[Table 2] shows the prevalence of microalbuminuria among patients with essential hypertension. Microalbuminuria was detected in 63 patients, representing a prevalence of 31.5% (95% CI: 25.2-38.3), which was statistically significant (Z = 4.82, p <0.001). The remaining 68.5% of patients did not exhibit microalbuminuria. The overall mean urinary albumin-to-creatinine ratio was 46.7 ± 18.6 mg/g with a narrow confidence interval (44.1-49.3), indicating consistent elevation in the study population (t = 18.74, p <0.001).

Table 3: Presence of Subclinical Target Organ Damage in Hypertensive Patients (N = 200)

Target Organ Damage	n (%)	95% CI	Test of Significance	P value
Left ventricular hypertrophy	72 (36.0%)	29.5 - 42.8	$\chi^2 = 11.26$	0.001
Hypertensive retinopathy	62 (31.0%)	24.8 - 37.9	$\chi^2 = 7.84$	0.005
Carotid IMT thickening	49 (24.5%)	18.9 - 30.9	$\chi^2 = 4.92$	0.026
Reduced eGFR	43 (21.5%)	16.2 - 27.6	$\chi^2 = 3.96$	0.046
Any TOD present	108 (54.0%)	47.1 - 60.7	$\chi^2 = 14.88$	<0.001

[Table 3] illustrates the prevalence of subclinical target organ damage among hypertensive patients. Left ventricular hypertrophy was the most common abnormality, observed in 36.0% of participants (p = 0.001). Hypertensive retinopathy was present in 31.0% (p = 0.005), followed by carotid intima-media thickness thickening in 24.5% (p = 0.026). Reduced renal function as indicated by decreased eGFR was noted in 21.5% of patients (p = 0.046). Overall, more than half of the study population (54.0%) exhibited at least one form of subclinical target organ damage, which was highly significant (p <0.001).

Table 4: Association between Microalbuminuria and Indicators of Target Organ Damage (N = 200)

Indicator	Microalbuminuria Present (n=63)	Microalbuminuria Absent (n=137)	Test of Significance	95% CI (OR)	P value
LVH	34 (53.9%)	38 (27.7%)	$\chi^2 = 12.76$	2.78 (1.47-5.25)	0.001

Retinopathy	29 (46.0%)	33 (24.1%)	$\chi^2 = 9.82$	2.66 (1.37-5.15)	0.002
CIMT thickening	21 (33.3%)	28 (20.4%)	$\chi^2 = 3.96$	1.95 (0.99-3.86)	0.047
Reduced eGFR	22 (34.9%)	21 (15.3%)	$\chi^2 = 9.96$	2.95 (1.43-6.09)	0.002
Multiple TOD	18 (28.5%)	17 (12.4%)	$\chi^2 = 7.68$	2.82 (1.32-6.03)	0.006

[Table 4] evaluates the association between microalbuminuria and individual indicators of target organ damage. Left ventricular hypertrophy was significantly more common in patients with microalbuminuria (53.9%) compared to those without (27.7%), showing nearly threefold increased risk (OR 2.78, p = 0.001). Similarly, hypertensive retinopathy was more prevalent in the microalbuminuria group (46.0% vs 24.1%, OR 2.66, p = 0.002). Carotid IMT thickening also showed a borderline but significant association (33.3% vs 20.4%, OR 1.95, p = 0.047). Reduced eGFR was significantly associated with microalbuminuria (34.9% vs 15.3%, OR 2.95, p = 0.002). Additionally, the presence of multiple target organ damage was notably higher among patients with microalbuminuria (28.5% vs 12.4%, OR 2.82, p = 0.006).

DISCUSSION

The present study evaluated the association between microalbuminuria and subclinical target organ damage among patients with essential hypertension. The findings demonstrate that microalbuminuria is significantly associated with advancing age, longer duration of hypertension, and a higher burden of early cardiovascular and renal damage. These observations are supported by Dimitriadis et al. (2025),[1] who emphasized microalbuminuria as an important prognostic marker reflecting early vascular injury and cardiovascular risk in essential hypertension.

In [Table 1], patients with microalbuminuria were significantly older and had a longer duration of hypertension compared to those without microalbuminuria. These findings are consistent with Kanna et al. (2020),[2] who reported that microalbuminuria prevalence increases with chronic blood pressure exposure and disease duration. The strong association between microalbuminuria and left ventricular hypertrophy observed in the present study aligns with Mancusi et al. (2022),[3] who demonstrated that hypertensive structural cardiac remodeling begins early and is closely linked with markers of renal endothelial dysfunction. Furthermore, the higher prevalence of hypertensive retinopathy and reduced eGFR among patients with microalbuminuria supports the concept of systemic microvascular injury, as also highlighted by El Mokadem et al. (2020),[4] who showed that blood pressure variability and endothelial dysfunction contribute significantly to subclinical target organ damage.

The prevalence of microalbuminuria in the present study was 31.5% [Table 2], which is comparable with recent reports. Kamal et al,[5] (2025) documented a similar prevalence and demonstrated a significant association between microalbuminuria and early end-organ damage in hypertensive patients. Additionally, the mean urinary albumin-to-creatinine ratio observed in this study indicates early renal involvement and systemic vascular dysfunction, consistent with findings by Sun et al,[6] (2022) who reported that impaired endothelial function and arterial stiffness strongly correlate with microalbuminuria and early target organ damage.[7]

[Table 3] revealed that more than half of hypertensive patients exhibited at least one form of subclinical target organ damage, with left ventricular hypertrophy being the most common abnormality. This observation parallels findings from Lin et al. (2025),[8] who emphasized the high burden of subclinical organ damage in hypertensive populations, particularly among patients with additional vascular risk factors. The coexistence of retinopathy, renal impairment, and vascular remodeling further reflects systemic endothelial dysfunction, as described by Cortese et al. (2020),[9] who highlighted the value of noninvasive tools in detecting early hypertensive organ damage.

[Table 4] further confirmed the strong association between microalbuminuria and multiple indicators of target organ damage. Patients with microalbuminuria showed significantly higher odds of left ventricular hypertrophy, retinopathy, renal dysfunction, and multiple organ involvement. These findings are consistent with Kothari et al. (2020),[10] who reported that microalbuminuria serves as an integrated marker of generalized vascular injury and predicts progression to cardiovascular complications. Although the association with carotid IMT thickening in the present study was modest, similar borderline relationships have been observed in vascular studies assessing early atherosclerotic changes.

CONCLUSION

The present cross-sectional study demonstrated that microalbuminuria is highly prevalent among patients with essential hypertension and is significantly associated with subclinical target organ damage. Patients with microalbuminuria were older, had a longer duration of hypertension, and exhibited a greater burden of early cardiovascular and renal involvement, including left ventricular hypertrophy, hypertensive retinopathy, carotid intima-media

thickening, and reduced renal function. The strong association between microalbuminuria and multiple indicators of target organ damage highlights its role as an early marker of systemic endothelial dysfunction and vascular injury. These findings suggest that routine screening for microalbuminuria in hypertensive patients can facilitate early risk stratification, timely therapeutic intensification, and prevention of progression to overt cardiovascular and renal complications. Therefore, microalbuminuria should be considered an important and cost-effective tool for early detection of subclinical organ damage in essential hypertension.

Limitations of the study

1. The cross-sectional design limits the ability to establish causal relationships between microalbuminuria and target organ damage.
2. The study was conducted in a single tertiary care center, which may limit generalizability to the broader community population.
3. A single spot urine sample was used for assessment of microalbuminuria, which may be influenced by transient physiological variations.
4. Advanced imaging modalities for detailed vascular assessment were not performed in all participants due to feasibility constraints.
5. Potential confounding factors such as dietary sodium intake, medication adherence, and lifestyle variables could not be completely controlled.

REFERENCES

1. Dimitriadis K, Pyrpyris N, Vakka A, Iliakis P, Theofilis P, Tatakis F, Beneki E, Pitsiori D, Tsioufis P, Aznaouridis K, Tousoulis D. Microalbuminuria as a prognostic marker in essential hypertension. Current medicinal chemistry. 2025 May 25.
2. Kanna D, Reddy PB. A study on microalbuminuria in systemic hypertension as an indicator of target organ damage in adult patients at Chengalpattu District. International Archives of Integrated Medicine. 2020 Mar 1;7(3).
3. Mancusi C, Lembo M, Manzi MV, Basile C, Fucile I, Morisco C. From structural to functional hypertension mediated target organ damage a long way to heart failure with preserved ejection fraction. Journal of clinical medicine. 2022 Sep 13;11(18):5377.
4. El Mokadem M, Boshra H, Abd el Hady Y, Kasla A, Gouda A. Correlation between blood pressure variability and subclinical target organ damage in patients with essential hypertension. Journal of Human Hypertension. 2020 Sep;34(9):641-7.
5. Kamal G, Avinash S. Prevalence of microalbuminuria and its association with end-organ damage in patients with essential hypertension at a private tertiary care center. Asian Journal of Medical Sciences. 2025 Sep 30;16(10):34-40.
6. Sun Y, Liu F, Zhang Y, Lu Y, Su Z, Ji H, Cheng Y, Song W, Hidru TH, Yang X, Jiang Y. The relationship of endothelial function and arterial stiffness with subclinical target organ damage in essential hypertension. The Journal of Clinical Hypertension. 2022 Apr;24(4):418-29.
7. Rosin E, Franzin A, Burrei G, Sabba GE, Biasotto A, Morellini S, Giontella A, Tagetti A, Cattazzo F, Marcon D, Piona C. Subclinical target organ damage in patients with type 1 diabetes mellitus. a prospective preliminary study. Journal of Hypertension. 2023 Jun 1;41(Suppl 3):e197-8.
8. Lin TC, Lu YH, Chao CT, Lin SJ, Wu PS, Chan DC, Lee HC. Comprehensive assessment and management of hypertension in elderly patients: Addressing frailty and target organ damage. Journal of the Formosan Medical Association. 2025 Jul 1;124:S17-23.
9. Cortese F, Cecere A, Maria Cortese A, Andriani A, Truncellito L, Valente F, Giordano P, Matteo Ciccone M. Vascular, cardiac and renal target organ damage associated to arterial hypertension: which noninvasive tools for detection?. Journal of Human Hypertension. 2020 Jun;34(6):420-31.
10. Kothari S, Trikha M, Gupta D. Microalbuminuria and Essential Hypertension: A Critical Evaluation. JK Science. 2020 Jul 1;22(3).