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ORIGINAL RESEARCH

Study of prevalance of microalbunuria in pt with essential hypertension and its correlation with target organ damage

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ABSTRACT

Background: Microalbuminuria (MA) is an early marker of endothelial dysfunction and renal impairment in patients with essential hypertension. Its presence has been linked to an increased risk of target organ damage, including cardiovascular and renal complications. This study aims to assess the prevalence of microalbuminuria in patients with essential hypertension and evaluate its correlation with target organ damage.**Materials and Methods:** A cross-sectional study was conducted on 200 hypertensive patients aged 35–70 years. Urinary albumin excretion was measured using the albumin-to-creatinine ratio (ACR) in a spot urine sample. Microalbuminuria was defined as an ACR between 30–300 mg/g. Target organ damage was assessed through echocardiography (left ventricular hypertrophy), renal function tests (serum creatinine, eGFR), and fundoscopic examination (hypertensive retinopathy). Statistical analysis was performed using Pearson's correlation and regression analysis to evaluate the association between microalbuminuria and organ damage.**Results:** Microalbuminuria was detected in 45% of hypertensive patients. A significant correlation was found between microalbuminuria and left ventricular hypertrophy ($p<0.01$), decreased eGFR ($p<0.05$), and hypertensive retinopathy ($p<0.05$). Patients with microalbuminuria had a higher mean systolic blood pressure (154.6 ± 10.2 mmHg) compared to those without (142.3 ± 8.5 mmHg). The regression model indicated that microalbuminuria is an independent predictor of target organ damage (OR = 2.5, 95% CI: 1.8–3.7).**Conclusion:** Microalbuminuria is prevalent among hypertensive patients and is strongly associated with early signs of target organ damage. Routine screening for microalbuminuria in essential hypertension may help in early risk stratification and prevention of complications.

Keywords: Microalbuminuria, Essential Hypertension, Target Organ Damage, Left Ventricular Hypertrophy, Renal Dysfunction, Hypertensive Retinopathy.

INTRODUCTION

Hypertension is a major global health concern and a leading cause of morbidity and mortality worldwide (1). It significantly contributes to cardiovascular diseases, renal dysfunction, and cerebrovascular complications. One of the earliest markers of hypertensive damage to the vascular system is microalbuminuria (MA), which reflects endothelial dysfunction and increased vascular permeability (2). The presence of MA in hypertensive individuals has been linked to an elevated risk of cardiovascular events, chronic kidney disease (CKD), and overall mortality (3).

Microalbuminuria is defined as urinary albumin excretion ranging between 30 and 300 mg/day and serves as a key indicator of subclinical organ damage in patients with essential hypertension (4). Several studies have demonstrated that MA is associated with left ventricular hypertrophy (LVH), a decline in estimated glomerular filtration rate (eGFR), and hypertensive retinopathy, all of which are indicative of target organ damage (5,6). Furthermore, MA has been recognized as an independent predictor of future cardiovascular events, emphasizing the importance of early detection and management in hypertensive patients (7).

Despite its clinical significance, microalbuminuria is often underdiagnosed in routine hypertensive assessments. The early detection of MA can provide valuable prognostic information and aid in timely interventions to prevent progression to severe organ damage (8). Therefore, this study aims to evaluate the prevalence of microalbuminuria in patients with essential hypertension and assess its correlation with target organ damage, including cardiac, renal, and ocular complications.

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MATERIALS AND METHODS

Study Design and Population

This cross-sectional study was conducted in a tertiary care hospital over a period of six months. A total of 200 patients diagnosed with essential hypertension, aged between 35 and 70 years, were enrolled. Patients with secondary hypertension, chronic kidney disease (CKD), diabetes mellitus, or any known cardiovascular disease were excluded to minimize confounding factors.

Data Collection and Clinical Assessment

Each participant underwent a detailed medical history evaluation and clinical examination. Blood pressure (BP) was measured using a standard sphygmomanometer, with the average of three readings taken at five-minute intervals recorded. Hypertension was defined as a systolic BP ≥140 mmHg and/or a diastolic BP ≥90 mmHg, in accordance with the American Heart Association guidelines.

Assessment of Microalbuminuria

Urine samples were collected from each participant for albumin-to-creatinine ratio (ACR) analysis. Microalbuminuria was defined as an ACR ranging from 30 to 300 mg/g in a spot urine sample. Samples were processed using an automated immunoturbidimetric assay.

Evaluation of Target Organ Damage

Target organ damage was assessed using the following parameters:

- **Cardiac Assessment**: Left ventricular hypertrophy (LVH) was evaluated using transthoracic echocardiography, based on left ventricular mass index (LVMI) values.
- **Renal Function Assessment**: Serum creatinine and estimated glomerular filtration rate (eGFR) were measured using the CKD-EPI formula. A reduced eGFR (<60 mL/min/1.73 m²) was considered an indicator of renal impairment.
- **Ophthalmic Evaluation**: Hypertensive retinopathy was assessed through fundoscopic examination, graded according to the Keith-Wagener-Barker classification.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were represented as frequencies and percentages. The chi-square test was used to compare categorical variables, and Pearson's correlation coefficient was applied to assess the relationship between microalbuminuria and target organ damage. Multivariate logistic regression was performed to determine independent predictors of organ damage, with statistical significance set at $p<0.05$.

RESULTS

Prevalence of Microalbuminuria in Hypertensive Patients

Among the 200 participants, microalbuminuria was detected in 90 patients (45%), while 110 patients (55%) had normoalbuminuria. The mean age of participants was 52.4 ± 8.6 years, with a male-to-female ratio of 1.3:1. The average systolic and diastolic blood pressures in patients with microalbuminuria were significantly higher compared to those without microalbuminuria ($p<0.05$) (Table 1).

Association Between Microalbuminuria and Target Organ Damage

The prevalence of left ventricular hypertrophy (LVH), reduced estimated glomerular filtration rate (eGFR), and hypertensive retinopathy was significantly higher in patients with microalbuminuria compared to those with normoalbuminuria. LVH was present in 60% of patients with microalbuminuria, whereas only 25% of normoalbuminuric patients exhibited LVH ($p=0.002$). Similarly, reduced eGFR was noted in 40% of patients with microalbuminuria compared to 15% in the normoalbuminuric group ($p=0.01$) (Table 2).

Correlation Between Microalbuminuria and Clinical Parameters

A statistically significant positive correlation was found between microalbuminuria and systolic blood pressure ($r=0.42$, $p<0.01$), diastolic blood pressure ($r=0.35$, $p=0.03$), and left ventricular mass index ($r=0.47$, $p<0.01$). Multivariate logistic regression analysis showed that microalbuminuria was an independent predictor of LVH (OR=2.5, 95% CI: 1.8–3.7, $p<0.01$) and reduced eGFR (OR=2.1, 95% CI: 1.5–3.2, $p=0.02$) (Table 3).

Table 1: Baseline Characteristics of Study Participants

Parameter	Microalbuminuria (n=90)	Normoalbuminuria (n=110)	p-value
Age (years)	53.1 ± 7.8	51.8 ± 9.2	0.24
Male (%)	58 (64.4)	65 (59.1)	0.48
Systolic BP (mmHg)	154.6 ± 10.2	142.3 ± 8.5	0.001
Diastolic BP (mmHg)	94.8 ± 6.1	88.5 ± 5.9	0.02
BMI (kg/m²)	27.5 ± 3.2	26.8 ± 3.0	0.15

(*BP: Blood Pressure; BMI: Body Mass Index*)

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Table 2: Prevalence of Target Organ Damage in Study Participants

Parameter	Microalbuminuria (n=90)	Normoalbuminuria (n=110)	p-value
Left Ventricular Hypertrophy (%)	54 (60)	28 (25)	0.002
Reduced eGFR (%)	36 (40)	17 (15)	0.01
Hypertensive Retinopathy (%)	42 (47)	22 (20)	0.008

(*eGFR: Estimated Glomerular Filtration Rate*)

Table 3: Correlation of Microalbuminuria with Clinical Parameters

Parameter	Correlation Coefficient (r)	p-value
Systolic BP	0.42	<0.01
Diastolic BP	0.35	0.03
Left Ventricular Mass Index	0.47	<0.01

(*BP: Blood Pressure*)

These findings suggest that microalbuminuria is strongly associated with increased blood pressure, left ventricular hypertrophy, and impaired renal function, reinforcing its role as a marker of target organ damage in hypertensive individuals (Table 3).

DISCUSSION

Microalbuminuria (MA) is widely recognized as an early indicator of endothelial dysfunction and an independent predictor of cardiovascular and renal complications in patients with essential hypertension (1). In this study, we observed that 45% of hypertensive patients exhibited microalbuminuria, consistent with previous reports indicating its high prevalence in hypertensive populations (2,3). This finding underscores the importance of routine screening for MA to identify patients at higher risk of target organ damage.

The present study demonstrated a significant association between microalbuminuria and left ventricular hypertrophy (LVH), with 60% of MA-positive patients exhibiting LVH compared to 25% in the normoalbuminuric group. Several studies have reported a similar correlation, attributing this relationship to the increased cardiac workload and altered hemodynamics caused by endothelial dysfunction in hypertensive patients (4,5). The underlying mechanism involves chronic pressure overload, leading to structural cardiac remodeling and increased left ventricular mass (6). Furthermore, microalbuminuria has been shown to reflect systemic vascular injury, which contributes to myocardial hypertrophy and fibrosis (7).

Renal impairment, as indicated by reduced estimated glomerular filtration rate (eGFR), was significantly more common in patients with microalbuminuria, with 40% of affected individuals showing reduced eGFR compared to 15% in the normoalbuminuric group. This finding aligns with previous research that highlights the role of MA as an early marker of nephropathy in hypertensive patients (8,9). Persistent albuminuria indicates glomerular endothelial dysfunction and increased permeability, which can ultimately lead to progressive renal damage (10). Early detection of MA can therefore serve as a crucial step in preventing the transition to chronic kidney disease (11).

Hypertensive retinopathy was another important finding in this study, with a higher prevalence among patients with microalbuminuria. This supports previous studies suggesting that retinal microvascular changes parallel systemic endothelial dysfunction seen in microalbuminuric individuals (12,13). The Keith-Wagener-Barker classification has been widely used to assess retinal changes in hypertension, and our results reaffirm its utility in detecting early microvascular damage in high-risk individuals (14).

Our study further demonstrated a significant correlation between microalbuminuria and elevated systolic and diastolic blood pressure, as well as left ventricular mass index. This is in agreement with prior studies emphasizing that the severity of hypertension is a key determinant of microvascular injury and subsequent organ damage (15,16). The strong correlation between microalbuminuria and blood pressure suggests that stricter antihypertensive control, particularly through renin-angiotensin-aldosterone system (RAAS) inhibitors, may play a vital role in reducing both albuminuria and associated cardiovascular risks (17,18).

The clinical implications of our findings highlight the need for incorporating microalbuminuria screening as a routine part of hypertension management. Given that MA is a reversible marker in early disease stages, interventions such as blood pressure optimization, renoprotective therapies, and lifestyle modifications may significantly reduce its progression to overt nephropathy and cardiovascular complications (19,20).

CONCLUSION

Despite these findings, the study has certain limitations. Being a cross-sectional study, it does not establish a causal relationship between microalbuminuria and target organ damage. Additionally, confounding factors such as dietary protein intake and undiagnosed diabetes may have influenced urinary albumin excretion. Future longitudinal studies are needed to further validate these associations and evaluate the impact of targeted interventions on microalbuminuria and organ damage progression.

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