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Association Between Microalbuminuria and Left Ventricular Hypertrophy in Hypertensive Patients: A Marker of Target Organ Damage

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About Article

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ABSTRACT

Microalbuminuria, an indicator of endothelial dysfunction and organ damage, is often linked to left ventricular hypertrophy (LVH) in individuals with hypertension. Identifying microalbuminuria early can offer critical insights into assessing cardiovascular risks. This research aimed to explore the relationship between microalbuminuria and LVH in hypertensive patients and to evaluate the clinical relevance of microalbuminuria as an indicator of organ damage. This cross-sectional study involved 150 hypertensive patients, divided into two groups: 75 with LVH (Group 1) and 75 without LVH (Group 2). LVH was diagnosed using echocardiography, specifically the left ventricular mass index (LVMI). Microalbuminuria was measured via the albumin-to-creatinine ratio (ACR) in a single urine sample, with a cutoff of 30–300 µg/mg. Data on blood pressure, hypertension duration, and body mass index (BMI) were collected. Statistical analyses included t-tests and chi-square tests. Microalbuminuria was significantly more common in Group 1 than in Group 2 (60% vs. 20%; $p < 0.001$). The average ACR was also higher in Group 1 (65.4 ± 20.7 µg/mg vs. 22.5 ± 10.2 µg/mg; $p < 0.001$). Patients with LVH had a longer history of hypertension, higher BMI, and increased blood pressure compared to those without LVH ($p < 0.05$). Microalbuminuria is closely associated with LVH in hypertensive patients and can serve as a non-invasive marker for organ damage. Regular screening for microalbuminuria in hypertensive patients can enhance risk assessment and inform targeted treatments.

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1. INTRODUCTION

Microalbuminuria, characterized by the presence of small amounts of albumin in urine, is an early sign of endothelial dysfunction and kidney damage. It is particularly relevant in patients with left ventricular hypertrophy (LVH), a condition marked by thickening of the left ventricular wall, often due to chronic hypertension. The presence of both microalbuminuria and LVH is associated with an increased risk of cardiovascular complications.

The LIFE study examined this relationship in hypertensive patients with LVH, finding that microalbuminuria was present in 23% of participants and independently linked to persistent LVH, even after adjusting for factors like age, blood pressure, and diabetes (Dahlöf *et al.*, 2002). This suggests that microalbuminuria and LVH may represent parallel processes of organ damage in hypertension.

A case-control study at Al-Yarmouk Teaching Hospital in Baghdad also found that hypertensive patients with LVH had higher ACR levels compared to those without LVH (70.5 ± 4.6 mg/mg vs. 30.3 ± 16.6 mg/mg, $p < 0.05$) (Al-Mudhaffer *et al.*, 2019). These findings highlight the importance of detecting microalbuminuria in patients with LVH, as it may indicate underlying organ damage and increased cardiovascular risk.

Early detection of microalbuminuria through routine urine testing allows for more aggressive management of hypertension and associated risks. Incorporating microalbuminuria screening into clinical practice can improve risk stratification and guide interventions to enhance cardiovascular outcomes (Weir, 2007). This research aimed to explore the relationship between microalbuminuria and LVH in hypertensive patients and to evaluate the clinical relevance of microalbuminuria as an indicator of organ damage.

2. LITERATURE REVIEW

The relationship between microalbuminuria and LVH has been extensively studied. The LIFE trial (Dahlöf *et al.*, 2002) demonstrated that microalbuminuria is independently associated with persistent LVH in hypertensive patients, even after adjusting for confounding factors such as age, blood pressure, and diabetes. This suggests that microalbuminuria and LVH may represent parallel processes of target organ damage in hypertension, driven by common pathophysiological mechanisms such as endothelial dysfunction, inflammation, and oxidative stress.

Further supporting this association, Al-Mudhaffer *et al.* (2019) found that hypertensive patients with LVH had significantly higher albumin-to-creatinine ratios (ACR) compared to those without LVH, highlighting the role of microalbuminuria as a non-invasive marker of cardiovascular risk. Similarly, Mogensen (1984) established that microalbuminuria predicts clinical proteinuria and early mortality in diabetic patients, underscoring its prognostic value in cardiovascular and renal outcomes.

Recent studies have further expanded our understanding of this relationship. A 2021 meta-analysis by Ribó-Coll *et al.* (2021) confirmed that microalbuminuria is strongly associated with LVH in hypertensive patients, with a pooled odds ratio of 2.45 (95% CI: 1.98–3.03). This study also highlighted the role of inflammation and oxidative stress as key mediators in the progression of both microalbuminuria and LVH. Additionally, Loncaric *et al.* (2020) demonstrated that microalbuminuria is an independent predictor of LVH progression in patients with resistant hypertension, emphasizing the need for aggressive management in this high-risk population.

Echocardiographic studies have also contributed to understanding the structural changes in the heart associated with hypertension. Devereux and Reichek (1977) validated the use of echocardiography for measuring left ventricular mass, a key parameter in diagnosing LVH. Ganau *et al.* (1992) further elucidated the patterns of LVH and geometric remodeling in essential hypertension, emphasizing the role of chronic pressure overload in driving these changes. More recently, Lang *et al.* (2015) updated the guidelines for cardiac chamber quantification by echocardiography, providing standardized methods for assessing LVH and its progression.

The clinical implications of these findings are significant. Microalbuminuria not only reflects renal and endothelial dysfunction but also serves as a surrogate marker for LVH and cardiovascular risk. Routine screening for microalbuminuria in hypertensive patients, particularly those with LVH, can facilitate early intervention and aggressive management of blood pressure and other risk factors. ACE inhibitors and ARBs, which have been shown to reduce both microalbuminuria and LVH, are particularly effective in this context (Schmieder *et al.*, 1999; Parving *et al.*, 2001). A 2022 study by Kumar *et al.* (2022) further supported the use of ARBs in reducing microalbuminuria and LVH in hypertensive patients with diabetes, demonstrating significant improvements in both renal and cardiac outcomes.

In conclusion, the strong association between microalbuminuria and LVH in hypertensive patients underscores the importance of early detection and management of both conditions. Microalbuminuria serves as a valuable marker for identifying patients at higher cardiovascular risk, and its incorporation into routine clinical practice can improve risk stratification and guide targeted interventions to reduce morbidity and mortality. Future research should focus on longitudinal studies to further elucidate the underlying mechanisms and optimize treatment strategies.

3. METHODOLOGY

This cross-sectional study aimed to assess the role of microalbuminuria in hypertensive patients with and without LVH. A total of 150 participants were enrolled, including 75 with LVH (Group 1) and 75 without LVH (Group 2). The study was conducted at Rizgary Teaching Hospital from November 2023 to November 2024.

3.1. Inclusion and Exclusion Criteria

Participants were aged 30–70 years with a diagnosis of essential hypertension for at least one year, defined as blood pressure ≥140/90 mmHg or current use of antihypertensive medication (AHA, 2017). LVH was diagnosed using echocardiography, with LVMI thresholds of >115 g/m² for men and >95 g/m² for women (Lang *et al.*, 2015).

Exclusion criteria included secondary hypertension, diabetes, chronic kidney disease (eGFR <60 mL/min/1.73 m²), urinary

tract infections, pregnancy, or any condition affecting urinary albumin levels.

Demographic and clinical data, including age, sex, BMI, hypertension duration, and antihypertensive medications, were collected. Blood pressure was measured using a calibrated sphygmomanometer.

Morning urine samples were collected to measure ACR. Microalbuminuria was defined as an ACR of 30–300 µg/mg (NKF, 2002).

Transthoracic echocardiography was performed by a certified cardiologist. LVMI was calculated using Devereux's formula, and LVH was confirmed based on predefined thresholds (Devereux *et al.*, 1977).

3.2. Statistical Analysis

Continuous variables were expressed as mean ± SD, and categorical variables as frequencies and percentages. Independent samples t-tests and chi-square tests were used for comparisons. Logistic regression was performed to assess the association between microalbuminuria and LVH, adjusting for confounders. A p-value ≤0.05 was considered significant.

The study was approved by the ethics committee of Hawler Medical University, and informed consent was obtained from all participants.

4. RESULTS AND DISCUSSION

4.1. Results

The study included 150 hypertensive patients, with 75 in Group 1 (LVH) and 75 in Group 2 (no LVH). Baseline characteristics are summarized in Table 1.

The mean age was similar between groups (58.2 ± 8.4 years in Group 1 vs. 56.7 ± 9.1 years in Group 2; p = 0.342). There was no significant difference in sex distribution (p = 0.631). However, Group 1 had a higher BMI (29.8 ± 3.5 kg/m² vs. 28.2 ± 3.9 kg/m²; p = 0.028) and longer hypertension duration (10.2 ± 3.7 years vs. 8.7 ± 3.2 years; p = 0.012). Blood pressure was also higher in Group 1 (systolic: 152.3 ± 10.5 mmHg vs. 144.8 ± 8.9 mmHg; diastolic: 92.4 ± 6.8 mmHg vs. 88.2 ± 6.1 mmHg; p < 0.001).

Table 1. Baseline characteristics of the study participants.

Characteristics	Group I: hypertensive patients with LVH (n=75)	Group II: hypertensive patients without LVH (n=75)	P-value
Age(years)	58.2 ±8.4	57.6± 9.1	0.342
Male (%)	48(64%)	45(60%)	0.631
Female (%)	27(36%)	30(40%)	0.631
Body mass index (BMI) kg/m²	29.8±3.5	28.2±3.9	0.028
Duration of hypertension (years)	10.2±3.7	8.7±3.2	0.012
Systolic BP (mmhg)	152.3±10.5	144.8±8.9	<0.001
Diastolic BP (mmhg)	92.4±6.8	88.2±6.1	<0.001

Table 2 shows that microalbuminuria was more prevalent in Group 1 (60% vs. 20%; p < 0.001), and the mean ACR was higher (65.4 ± 20.7 µg/mg vs. 22.5 ± 10.2 µg/mg; p < 0.001). Echocardiography confirmed LVH in all Group 1 patients, with a higher mean LVMI (134.7 ± 18.5 g/m² vs. 87.6 ± 12.3 g/m²; p < 0.001).

Table 2. Microalbuminuria and Echocardiographic findings of the study population.

Parameters	Group I: hypertensive patients with LVH (n=75)	Group II: hypertensive patients without LVH (n=75)	P-value
Microalbuminuria (%)	45(60%)	15(20%)	<0.001
Mean ACR (µg/mg)	65.4±20.7	22.5±10.2	<0.001
LVM index (LVMI, g/m²)	134.7±18.5	87.6±12.3	<0.001
LVH (%)	100%	0%	-

4.2. Discussion

This study found a strong association between microalbuminuria and LVH in hypertensive patients. Microalbuminuria was significantly more common in patients with LVH, who also had higher ACR levels, longer hypertension duration, and elevated blood pressure. These findings align with previous research, including the LIFE study, which identified microalbuminuria as an independent predictor of LVH (Dahlöf *et al.*, 2002).

The higher ACR levels in LVH patients are consistent with findings from Al-Yarmouk Teaching Hospital (Al-Mudhaffer *et al.*, 2019). These results underscore the value of microalbuminuria as a non-invasive marker for cardiovascular damage in hypertensive patients.

The longer hypertension duration and higher blood pressure in LVH patients are consistent with the role of chronic hypertension in LVH development (Ganau *et al.*, 1992). Elevated blood pressure exacerbates endothelial dysfunction, contributing to microalbuminuria (Mogensen, 1984).

4.3. Clinical implications

The coexistence of microalbuminuria and LVH is clinically significant, as both are linked to increased cardiovascular risks, including heart attack, stroke, and heart failure (Weir, 2007). Early detection of microalbuminuria in hypertensive patients with LVH allows for more aggressive management of blood pressure and risk factors, potentially reducing morbidity and mortality.

Routine screening for microalbuminuria in hypertensive patients, especially those with LVH, can provide valuable insights into organ damage and guide treatment. ACE inhibitors and ARBs have been shown to reduce both microalbuminuria and LVH, suggesting that addressing these factors simultaneously may improve outcomes (Schmieder *et al.*, 1999; Parving *et al.*, 2001).

5. CONCLUSIONS

This study demonstrates a strong association between microalbuminuria and LVH in hypertensive patients. Microalbuminuria reflects subclinical organ damage and serves as a valuable marker for identifying patients at higher cardiovascular risk. Routine screening for microalbuminuria in hypertensive patients, particularly those with LVH, is recommended to improve risk stratification and guide targeted interventions.

LIMITATIONS AND RECOMMENDATIONS

This study has limitations, including its cross-sectional design, which prevents causal inferences. The sample size, while adequate, limits generalizability. Future studies with larger cohorts and longitudinal designs are needed to confirm these findings and explore underlying mechanisms.

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