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Correlation of systolic blood pressure and pulse pressure with albuminuria in patients of hypertension without diabetes

Sanjeev Thind*
Doctor of Medicine, Resident
Hind Institute of Medical Sciences
225001, Safedabad, Barabanki, Uttar Pradesh, India
https://orcid.org/0000-0002-7528-1973

Chandra Shekhar Tiwari
Doctor of Medicine, Associate Professor
Hind Institute of Medical Sciences
225001, Safedabad, Barabanki, Uttar Pradesh, India
https://orcid.org/0009-0001-6201-0007

Neeraj Tripathi
Doctor of Medicine, Professor
Hind Institute of Medical Sciences
225001, Safedabad, Barabanki, Uttar Pradesh, India
https://orcid.org/0000-0002-0565-6602

Abstract. Hypertension and microalbuminuria (MAU) are independent yet interconnected markers of cardiovascular and renal dysfunction. While MAU is an early indicator of renal impairment, its relationship with blood pressure (BP) components, particularly systolic blood pressure (SBP) and pulse pressure (PP), remains underexplored in non-diabetic hypertensive populations. The purpose of this study was to investigate the independent association between these BP components and MAU, identify which parameter exhibits a stronger correlation, and enhance the understanding of early renal dysfunction in hypertension management. For this analytical cross-sectional study was conducted at Hind Institute of Medical Sciences, Safedabad, Barabanki, Uttar Pradesh, over 12 months, involving 120 hypertensive patients attending outpatient and inpatient departments. Patients with comorbidities like diabetes mellitus, renal failure, or overt proteinuria were excluded. BP was measured in two separate readings using a standard sphygmomanometer, and microalbuminuria was assessed via immunoturbidometric assay. The urine albumin-to-creatinine ratio (ACR) served as the primary indicator of MAU. SBP and PP demonstrated significant positive correlations with MAU ($\rho = 0.25$, $p = 0.032$; $\rho = 0.30$, $p = 0.015$, respectively), while diastolic BP (DBP) showed a negative yet non-significant association ($\rho = -0.20$, $p = 0.065$). Among anthropometric parameters, body mass index (BMI) and waist-hip ratio exhibited no significant differences between groups. Urine ACR was markedly higher in the MAU group (182.5 ± 156.5 mg/L) compared to the non-MAU group (17.6 ± 7.1 mg/L; $p < 0.0001$). Lipid profiles, fasting blood glucose, and renal function markers like serum creatinine and blood urea nitrogen were comparable between groups, highlighting BP components as primary predictors of albuminuria. The strong positive correlations between SBP, PP, and MAU highlighted the need for precise BP management in non-diabetic hypertensive patients. Regular monitoring and treatment to optimise SBP and PP levels could mitigate renal damage and reduce cardiovascular risks

Keywords: renal dysfunction; cardiovascular disease; urine albumin-to-creatinine ratio; nephropathy

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*Corresponding author

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INTRODUCTION

Hypertension is a major public health concern and a leading cause of cardiovascular and renal complications. It is estimated to affect over 1.28 billion adults worldwide and remains a key risk factor for end-organ damage, particularly involving the heart, brain, and kidneys [1]. The relationship between blood pressure components and early markers of kidney dysfunction, such as microalbuminuria (MAU), has gained significant attention. Identifying hypertensive patients with early renal function changes is critical for preventing the progression of chronic kidney disease (CKD). Systolic blood pressure (SBP) and pulse pressure (PP) have been suggested as independent predictors of renal damage, yet their correlation with albuminuria remains underexplored in non-diabetic hypertensive patients.

W. Zhang *et al*. [2] reported that intensive control of both blood glucose and SBP (<130/80 mmHg) significantly reduced the prevalence of albuminuria in patients with coexisting diabetes and hypertension, suggesting the role of SBP in renal protection. Similarly, S. Coşkun *et al*. [3] found that in patients with isolated systolic and diastolic hypertension, SBP and left atrial diameter were independent predictors of microalbuminuria, although PP was not significant in multivariate analysis. Conversely, L. Liu *et al*. [4] conducted a large community-based study in prediabetic individuals and demonstrated a significant positive association between PP and albuminuria. The highest PP quartile showed a more than twofold increase in the odds of albuminuria compared to the lowest quartile, independent of antihypertensive treatment and other covariates. In another study, J. Sołtysiak *et al*. [5] found that nocturnal diastolic BP, diastolic load, and non-dipping BP status were significantly associated with increased urinary albumin excretion in adolescents with type 1 diabetes, pointing to the importance of circadian BP rhythm and vascular stiffness.

D. Suzuki *et al*. [6], in their analysis of over 4,000 participants in the J-HOP study, found a significant association between average home SBP and urinary albumin-to-creatinine ratio (UACR), regardless of diabetes status. However, day-to-day BP variability metrics were inconsistently associated with albuminuria, with only average real variability (ARV) of morning SBP showing a significant correlation in diabetic patients. B.A. Durak *et al*. [7] further supported this link in a study utilising ambulatory BP monitoring (ABPM). They observed that non-dipper and reverse-dipper BP profiles, along with elevated 24-hour and nocturnal SBP and DBP, were strongly associated with proteinuria. This suggests that altered BP patterns and nocturnal hypertension may accelerate renal microvascular injury in hypertensive individuals. Moreover, in the context of secondary and resistant hypertension, G.P. Rossi *et al*. [8] and G. Lamirault *et al*. [9] emphasised the need for early identification and targeted treatment of high-risk hypertensive profiles to prevent renal sequelae such as albuminuria. These reviews highlighted that resistant and secondary hypertension were often underdiagnosed yet accounted for a significant portion of patients with progressive renal damage.

Despite these findings, several gaps remain in the literature. While the association between diabetes and albuminuria has been extensively studied, the specific correlation between different blood pressure components and MAU in non-diabetic hypertensive patients has not been thoroughly examined. Moreover, existing studies often focus on broad hypertensive populations without distinguishing the independent effects of SBP and PP. The relative contribution of these parameters to renal dysfunction remains unclear, necessitating further investigation. The objective of the present study was to assess the independent associations between microalbuminuria and blood pressure components, specifically SBP and PP, after adjusting for potential confounders.

MATERIALS AND METHODS

The present study was conducted at the Department of Medicine, Hind Institute of Medical Sciences, Safedabad, Barabanki, Uttar Pradesh, over a period of 12 months, from May, 2023 to May, 2024. It was designed as an analytical cross-sectional study to evaluate the correlation between SBP, PP, and albuminuria in hypertensive patients. The study included patients attending both the outpatient and inpatient departments. Ethical clearance was obtained from the Institutional Ethics Committee (HIMS/IHEC/MD/MS/0011/2023), and written informed consent was obtained from all participants. The study adhered to the ethical principles outlined in the Declaration of Helsinki [10].

A total of 120 patients diagnosed with hypertension or undergoing treatment for hypertension, defined as having SBP >140 mmHg and DBP >90 mmHg, were enrolled in the study. To maintain the specificity of the study, patients with confounding conditions such as overt proteinuria, congestive cardiac failure, renal failure, diabetes mellitus, urinary tract infections, obstructive uropathy, nephrolithiasis, or pregnancy were excluded. The total study population consisted of 120 hypertensive patients, with 57 participants in GROUP-NA and 63 in GROUP-MA. The mean age of the total cohort was 66.07 ± 13.25 years, with the majority of patients falling within the 60-69 age group (62 out of 120), followed by 50-59 years (30 patients), and 70-79 years (28 patients). The gender distribution was relatively balanced, with 63 males and 57 females.

Blood pressure measurements were performed using a standard sphygmomanometer. Participants were advised to avoid smoking, tea, and coffee for at least 30 minutes prior to measurement to prevent acute fluctuations. Two BP readings were taken 10 minutes apart in a seated position, and the higher value was recorded for analysis. Body mass index (BMI) was calculated using the standard formula: weight (kg)/height (m^2), with both parameters measured using calibrated instruments. A detailed cardiovascular examination was conducted, including palpation of peripheral pulses – specifically carotid and femoral arteries – to assess early signs of atherosclerosis. Fundoscopic examination was performed to evaluate hypertensive retinopathy.

Comprehensive laboratory investigations were conducted to assess renal and metabolic parameters. Routine urine analysis included albumin, sugar, and sediment evaluation. Fasting and postprandial blood glucose levels, serum urea, and creatinine tests were conducted to rule out underlying metabolic or renal dysfunction. Lipid profiles, including total cholesterol, low-density lipoprotein (LDL),

high-density lipoprotein (HDL), and triglycerides, were measured using an enzymatic colorimetric method to examine their potential association with microalbuminuria. Blood urea nitrogen (BUN) was quantified using a kinetic UV assay to evaluate renal function.

Urine albumin levels were determined using an immunoturbidometric assay performed on early morning urine samples. The urine albumin-to-creatinine ratio (UACR) was calculated from spot urine samples, with laboratory analysis completed on the same morning, to ensure consistency and accuracy. All collected data were systematically recorded in Microsoft Excel and analysed using SPSS version 26 (SPSS Inc., Chicago, IL, USA). Continuous variables, such as SBP, PP, BMI, lipid levels, UACR, and BUN, were presented as mean ± standard deviation (SD). Categorical variables, including gender, smoking status, and hypertensive retinopathy, were expressed as frequency distributions. To compare group means for normally distributed continuous variables, the Student's t-test was utilised, while categorical variables were analysed using the Chi-square test. The relationship between blood pressure components (SBP and PP) and albuminuria was assessed using Spearman's correlation coefficient. Statistical significance was set at a p-value <0.05, with values <0.001 considered highly significant.

RESULTS AND DISCUSSION

The age distribution between GROUP-NA and GROUP-MA did not show any statistically significant differences, as indicated by the p-values of 0.2739 for the categorical age ranges (50-59, 60-69, 70-79) and 0.1262 for the mean age comparison. While GROUP-NA had a greater proportion of younger participants in the 50-59 age range (25 vs 5), GROUP-MA had a higher representation in the older age brackets, specifically 60-69 years (40 vs 22) and 70-79 years (18 vs 10). The mean age in GROUP-MA was slightly higher (68.2 ± 8.9 years) than in GROUP-NA (65.5 ± 10.3 years). However, this difference was not statistically significant, suggesting that age alone was not a confounding factor in determining differences in clinical outcomes between the two groups. Gender distribution was also comparable between the groups, with GROUP-NA consisting of 28 males and 29 females, while GROUP-MA had 35 males and 28 fe males. The p-value for gender distribution was 0.7047, further confirming no significant difference in gender proportions between the groups. This suggests that any observed variations in clinical parameters or outcomes are unlikely to be attributed to differences in age or gender, allowing for a more focused assessment of hypertension-related factors and albuminuria levels in both cohorts (Table 1).

Table 1. Demographical profile of the enrolled patients among the groups

Variable	GROUP-NA (n = 57)	GROUP-MA (n = 63)	TOTAL (n = 120)	p-value
Age (years)				
50-59	25	5	30	X = 2.590 p = 0.2739
60-69	22	40	62	
70-79	10	18	28	
Mean ± SD	65.5 ± 10.3	68.2 ± 8.9	66.07 ± 13.25	t = 1.540 p = 0.1262
Gender				X = 0.4966 p = 0.7047
Male	28	35	63	
Female	29	28	57	

Source: compiled by the authors

Blood pressure parameters showed that systolic blood pressure (SBP) was significantly higher in GROUP-MA (149.6 ± 18.4 mmHg) compared to GROUP-NA (142.9 ± 17.2 mmHg), with a t-value of 2.528 and a p-value of 0.0057. This indicates that individuals in GROUP-MA had a greater elevation in SBP, which is a well-established risk factor for cardiovascular events and renal impairment. The higher SBP in GROUP-MA suggests a potential association with the presence of microalbuminuria, as increased systolic pressure has been linked to endothelial dysfunction and glomerular damage, which contribute to albuminuria. Diastolic blood pressure (DBP) was slightly higher in GROUP-MA (86.3 ± 11.7 mmHg) compared to GROUP-NA (85.5 ± 10.1 mmHg), but this difference was not statistically significant (t = 0.3989, p = 0.6907). The lack of a significant difference in DBP suggests that elevated DBP alone may not be a strong predictor of microalbuminuria in this study population. However, the combination of high SBP and widened pulse pressure (PP) is likely to contribute more to vascular damage and renal complications than DBP alone.

Pulse pressure (PP), which represents the difference between SBP and DBP, was also significantly higher in GROUP-MA (62.3 ± 16.2 mmHg) compared to GROUP-NA (56.1 ± 14.6 mmHg), with a t-value of 1.684 and a p-value of 0.0154. This finding suggests that widened PP, often indicative of arterial stiffness and reduced vascular compliance, may play a critical role in the pathogenesis of microalbuminuria. Elevated PP has been associated with increased cardiovascular morbidity and mortality, and renal impairment due to increased glomerular pressure fluctuations. Overall, these findings highlight that GROUP-MA, which had significantly higher SBP and PP, may be at an elevated risk for microvascular and renal complications compared to GROUP-NA. This underscores the importance of early detection and management of elevated SBP and PP in hypertensive patients to prevent the progression of albuminuria and its associated complications (Fig. 1).

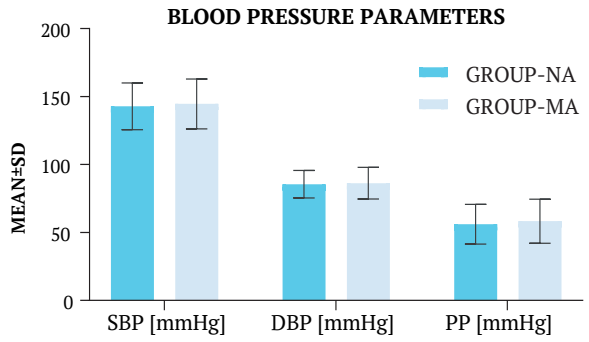

Figure 1. Graphical representation of blood pressure parameters
Source: compiled by the authors

Anthropometric measurements showed no significant differences, with the BMI averaging 25.1 ± 3.7 kg/m² for GROUP-NA and 25.4 ± 4 kg/m² for GROUP-MA (p = 0.6715). The waist-hip (W-H) ratio was identical for both groups at 0.92 ± 0.07 for GROUP-NA and 0.92 ± 0.06 for GROUP-MA (p > 0.9999). Additionally, the mean duration until hypertension diagnosis was slightly longer in GROUP-MA (9.65 ± 3.88 years) compared to GROUP-NA (8.63 ± 2.71 years), showing non-significant differences (t = 1.653, p = 0.1010). The use of antihypertensive medications was comparable between groups, with 36 participants in GROUP-NA and 41 in GROUP-MA using Amlodipine (5 mg), 42 in GROUP-NA and 56 in GROUP-MA using Losartan (50 mg), and 19 in GROUP-NA and 14 in GROUP-MA using Hydrochlorothiazide (25 mg). The Chi-square test for medication distribution yielded a non-significant difference (p = 0.3414)

A number of lifestyle characteristics, including smoking, alcohol intake, and levels of physical exercise, were likewise comparable amongst the groups. Smoking was reported by 18 participants in GROUP-NA and 21 in GROUP-MA, while 39 and 42 participants in GROUP-NA and GROUP-MA, respectively, were non-smokers (p = 0.2049). Alcohol consumption was reported by 13 participants in GROUP-NA and 17 in GROUP-MA, while 44 and 46 participants, respectively, did not consume alcohol (p = 0.5977). Regular physical activity was noted in 28 participants in GROUP-NA and 36 in GROUP-MA, while 29 and 37 participants in GROUP-NA and GROUP-MA, respectively, were sedentary (p = 0.9826).

Fasting blood glucose (FBG) levels were not significantly different between GROUP-NA (5.2 ± 1.1 mmol/L) and GROUP-MA (5.6 ± 1.8 mmol/L, p = 0.1497). Urine ACR was significantly higher in GROUP-MA (182.5 ± 156.5 mg/L) compared to GROUP-NA (17.6 ± 7.1 mg/L, p < 0.0001). Total protein levels were similar between GROUP-NA (71.52 ± 5.85 mg/L) and GROUP-MA (72.09 ± 5.67 mg/L, p = 0.5890), as were albumin levels (45.51 ± 3.76 mg/L vs 45.37 ± 3.78 mg/L, p = 0.8394). Creatinine levels were 76 ± 24.8 µmol/L for GROUP-NA and 79.9 ± 40.7 µmol/L for GROUP-MA (p = 0.5327), and blood urea nitrogen (BUN) levels were 5.31 ± 1.69 mmol/L for GROUP-NA and 5.51 ± 2.07 mmol/L for GROUP-MA (p = 0.5657), with no significant differences (Table 2). Lipid profile markers such TC, TG, LDL-C, and HDL-C were likewise similar between groups (all p-values > 0.05) (Fig. 2).

Table 2. Comparison of the metabolic parameters in enrolled patients among the groups

METABOLIC PARAMETERS	GROUP-NA (n = 57)	GROUP-MA (n = 63)	TOTAL (n = 120)	p-value
FBG [mmol/L]	5.2 ± 1.1	5.6 ± 1.8	5.4 ± 1.5	t = 1.450 p = 0.1497
Urine ACR [mg/gm]	17.6 ± 7.1	182.5 ± 156.5	99.7 ± 135.7	t = 7.944 p < 0.0001
Total protein [mg/L]	71.52 ± 5.85	72.09 ± 5.67	71.8 ± 5.77	t = 0.5417 p = 5890
Albumin [mg/L]	45.51 ± 3.76	45.37 ± 3.78	45.44 ± 3.77	t = 0.2031 p = 0.8394
Cr [µmol/L]	76 ± 24.8	79.9 ± 40.7	77.5 ± 33.1	t = 0.6258 p = 0.5327
BUN [mmol/L]	5.31 ± 1.69	5.51 ± 2.07	5.41 ± 1.89	t = 0.5761 p = 0.5657

Source: compiled by the authors

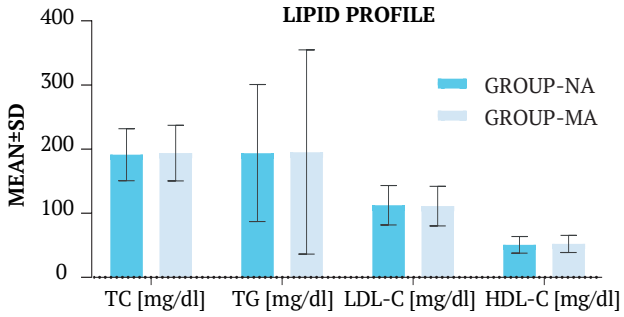

Figure 2. Graphical representation of lipid profile in enrolled patients
Source: compiled by the authors

Correlational analysis revealed that SBP had a significant positive correlation with albuminuria (ρ = 0.25), as did PP (ρ = 0.30), indicating that increased systolic pressure and widened pulse pressure are associated with greater urinary albumin excretion. This reinforces the idea that SBP and PP contribute to endothelial dysfunction and heightened glomerular permeability, which can lead to renal damage in hypertensive patients. Conversely, DBP exhibited a negative but non-significant correlation (ρ = -0.20), suggesting that DBP alone may not have a substantial direct impact on albuminuria. In addition, total protein (ρ = 0.35, p = 0.009) and albumin (ρ = 0.28) showed significant positive correlations with albuminuria, indicating that variations in plasma protein levels could influence albumin excretion

in hypertensive patients. This aligns with the hypothesis that systemic inflammatory responses and alterations in endothelial function may be linked to renal impairment.

Other metabolic parameters, including uric acid (ρ = -0.12) and fasting blood glucose (FBG) (ρ = 0.08), did not exhibit significant correlations with albuminuria, suggesting that these factors may not independently contribute to increased urinary albumin excretion in the studied cohort. Similarly, creatinine (ρ = -0.05) and blood urea nitrogen (BUN) (ρ = 0.20) showed non-significant correlations, indicating that kidney function, as measured by these markers, was not directly associated with the degree of albuminuria in this population. Ultimately, the most significant associations were noted for SBP, PP, total protein, and albumin, highlighting the importance of these parameters in predicting early renal impairment in hypertensive patients. These findings support the need for targeted blood pressure management strategies focusing on reducing SBP and PP to mitigate the risk of albuminuria and subsequent renal complications (Table 3).

Table 3. Correlation analysis of the enrolled patients' parameters with the albuminuria

Parameters vs Albuminuria correlation analysis	Spearman's Rho	p-value
SBP vs Albuminuria	0.25	0.032
DBP vs Albuminuria	-0.20	0.065
PP vs Albuminuria	0.30	0.015
Uric acid vs Albuminuria	-0.12	0.252
FBG vs Albuminuria	0.08	0.421
Total protein vs Albuminuria	0.05	0.529
Albumin vs Albuminuria	0.28	0.026*
Cr vs Albuminuria	-0.05	0.654
BUN vs Albuminuria	0.20	0.071

Source: compiled by the authors

CVD risk is increased by arterial hypertension, which is characterised by high systolic and/or diastolic blood pressure. A drop in blood pressure reduced cardiovascular events. MAU is a sensitive and specific diagnostic for early renal impairment. MAU is strongly linked to diabetes or metabolic syndrome. There is insufficient evidence linking MAU and blood pressure components. After controlling for possible variables, the present study investigated whether pulse pressure (PP) and SBP/DBP were independently linked with MAU. While analysing the correlation between SBP and microalbuminuria, the present study found that SBP has a positive correlation with albuminuria (ρ = 0.25, p = 0.032), indicating a significant relationship. DBP shows a negative but non-significant correlation (ρ = -0.20, p = 0.065). Pulse pressure (PP) is positively correlated with albuminuria (ρ = 0.30, p = 0.015), indicating significance. Uric acid has a negative and non-significant correlation (ρ = -0.12, p = 0.252). FBG also shows a non-significant correlation (ρ = 0.08, p = 0.421). Total albumin has a significant positive correlation with albuminuria (ρ = 0.28, p = 0.026). Creatinine (Cr) and blood urea nitrogen (BUN) have non-significant correlations with albuminuria (ρ = -0.05, p = 0.654 and ρ = 0.20, p = 0.071, respectively). Significant correlations are noted with SBP, PP, total protein, and albumin. J. Song *et al*. [11] found inconsistent findings on fasting blood glucose (FBG) and albuminuria. S. Yimthiang *et al*. [12] found a 5-fold increase in albuminuria risk as blood glucose levels approached 180 mg/dL. P.K. Chandie Shaw *et al*. [13] found no significant correlation between FBG and albuminuria in South Asian type 2 diabetics. B. Lei *et al*. [14] found no indication that lowering blood glucose levels could prevent albuminuria. There is also controversy about whether rigorous glycaemic therapy improves albuminuria. In another trial, S.G. Coca *et al*. [15] found that intense blood glucose management reduced microalbuminuria and macroalbuminuria. Intensive glycaemic management did not significantly slow clinical albuminuria progression. Conventional and aggressive glycaemic therapy had no statistically significant longitudinal effect on clinical albuminuria progression.

According to K.M. Ahmed Aziz [16], spot urine protein excretion is 67.5 mg/dL at 150 mmHg SBP. If SBP is 110 mmHg, spot urine protein is 12 mg/dL. A MAP of 110 yields 63 mg/dL spot urine protein. If MAP is 120, spot urine protein excretion is 81.6 mg/dL. According to the data, pulse pressure can estimate spot urine protein. Thus, spot urine protein is 20 mg/dL if PP is 40, which is normal. Spot urine protein excretion is 82 mg/dL at PP 80. The present study's mathematical calculations and connections between blood pressure and renal protein excretion are noteworthy. These mathematical formulae estimate kidney protein excretion under specified blood pressure values to monitor proteinuria and diabetic kidney disease. Long-term HTN patients had a greater rate of microalbuminuria than recently diagnosed patients, according to K.R. Bhusal *et al*. [17]. Patients without controlled blood pressure showed a greater rate of microalbuminuria.

In the current study, fasting blood glucose (FBG) levels were comparable between GROUP-NA (5.2 ± 1.1 mmol/L) and GROUP-MA (5.6 ± 1.8 mmol/L), with a p-value of 0.1497, indicating no significant difference. Urine ACR, however, was significantly elevated in GROUP-MA (182.5 ± 156.5 mg/L) compared to GROUP-NA (17.6 ± 7.1 mg/L), with a p-value of <0.0001. Total protein (71.52 ± 5.85 mg/L vs 72.09 ± 5.67 mg/L, p = 0.5890), albumin (45.51 ± 3.76 mg/L vs 45.37 ± 3.78 mg/L, p = 0.8394), creatinine (76 ± 24.8 µmol/L vs 79.9 ± 40.7 µmol/L, p = 0.5327), and blood urea nitrogen (5.31 ± 1.69 mmol/L vs 5.51 ± 2.07 mmol/L, p = 0.5657) levels showed statistically non-significant differences.

While previous studies, such as by K. Thieme & M. Oliveira-Souza [18], found that SBP and PP were independently associated with MAU after adjusting for confounders, the current study did not identify such a relationship. An increase in SBP typically elevates glomerular pressure and

filtration rate (GFR), potentially causing proteinuria due to trans-membrane protein leakage, as seen in animal models with angiotensin II infusion. E. Lee *et al*. [19] reported higher GFR in MAU patients compared to normoalbuminuric individuals. Unlike earlier research, this study focused on newly diagnosed, treatment-naïve hypertensive patients, avoiding medication-related confounding factors. Findings suggest that PP is a meaningful biomarker for assessing the renal impact of elevated blood pressure, with SBP and PP showing comparable sensitivity and specificity for predicting MAU based on ROC analysis.

In this study, SBP demonstrated a significant positive correlation with albuminuria ($\rho = 0.25$,), while diastolic blood pressure (DBP) had a negative but non-significant correlation ($\rho = -0.20$). Pulse pressure (PP) showed a significant positive correlation ($\rho = 0.30$). Uric acid and fasting blood glucose (FBG) exhibited non-significant correlations with albuminuria ($\rho = -0.12$, and $\rho = 0.08$ respectively). Significant positive correlations were observed for total protein ($\rho = 0.35$, $p = 0.009$) and albumin ($\rho = 0.28$). Creatinine (Cr) and blood urea nitrogen (BUN) showed non-significant correlations ($\rho = -0.05$, and $\rho = 0.20$, respectively). Overall, significant associations were found between albuminuria and SBP, PP, total protein, and albumin.

According to N. Xie *et al*. [20], patients with microalbuminuria (MAU) exhibited greater SBP and PP than those with normo-albuminuria ($p < 0.05$), whereas DBP was identical. Significantly higher serum levels of FBG, total protein, Cr, BUN, and HDL-C were seen in the MAU group ($p < 0.05$). Urine ACR was significantly greater in the MAU group (182.5 ± 156.5 mg vs 17.6 ± 7.1 mg, $p < 0.001$). In this study, systolic blood pressure (SBP) averages were similar between GROUP-NA (142.9 ± 17.2 mmHg) and GROUP-MA (144.6 ± 18.4 mmHg), with no significant difference ($p = 0.6032$). Diastolic blood pressure (DBP) was also comparable, with GROUP-NA at 85.5 ± 10.1 mmHg and GROUP-MA at 86.3 ± 11.7 mmHg ($p = 0.6907$). Pulse pressure (PP) showed no significant difference either, with GROUP-NA at 56.1 ± 14.6 mmHg and GROUP-MA at 58.3 ± 16.2 mmHg ($p = 0.4379$). Therefore, blood pressure indicators were not significantly different across groups.

B.J. Kim *et al*. [21] found that even in prehypertension, SBP of 120-129 mmHg or DBP of 80-84 mmHg posed a lower risk than higher thresholds. Y.H. Jung *et al*. [22] also noted a higher incidence of albuminuria in patients with SBP below 150 mmHg compared to those above, suggesting that elevated SBP affects albuminuria, particularly in patients not on medication. The researchers concluded that less stringent controls (SBP <150 mmHg, DBP <90 mmHg) are appropriate for those over 60 years old, balancing the risk of cardiovascular disease and albuminuria. Conversely, S. Bangalore *et al*. [23] highlighted the benefits of stricter blood pressure control, noting fewer cardiovascular events and strokes in adults with SBP <140 mmHg compared to those with SBP <150 mmHg. In adults without diabetes, stricter control (SBP <120 mmHg) reduced total mortality and vascular disease compared to less strict targets (SBP <140 mmHg).

The discrepancy between the two studies can be interpreted in light of their differing patient populations. The present study evaluated hypertensive individuals without confirmed ischaemic heart disease, in whom the influence of smoking and other lifestyle factors on renal microvascular injury may be less pronounced or more multifactorial. In contrast, A. Basu & J.S. Jhala [24] highlighted the exacerbating effect of smoking on endothelial dysfunction in patients with established cardiovascular disease, which may amplify the risk and severity of microalbuminuria. Therefore, while lifestyle factors did not significantly influence microalbuminuria status in the present hypertensive cohort, they appear to play a more critical role in populations with advanced atherosclerotic disease. A. Mimarm [25] made similar observations. However, the present study did not show any significant difference.

According to A. Basu & J.S. Jhala [24], BMI was categorised as >25 or <25, with 26 cases having BMI >25. Among these, microalbuminuria was observed in 21 cases (80.76%), while 5 (19.24%) did not have it. This aligns with the Gubbio population study by M. Cirillo *et al*. [26], which also reported an association between high BMI and microalbuminuria. The study further noted that among controls with BMI >25, 5 out of 10 (50%) had microalbuminuria, showing a significant association. In the study group of 36 individuals with BMI >25, 26 (72.22%) were cases, and 10 (27.77%) were controls. Microalbuminuria was observed in 26 participants in this group, with 21 being cases and 5 controls, suggesting a higher prevalence of microalbuminuria among cases with BMI >25. However, non-significant differences, indicating that microalbuminuria could be an independent risk factor for ischaemic heart disease (IHD). The chance of microalbuminuria positivity was higher among cases (26.58%) compared to controls (0.65%) despite similar BMI values, emphasising the potential role of microalbuminuria in IHD risk. In contrast, the current study found no significant difference in BMI between GROUP-NA (25.1 ± 3.7 kg/m^2) and GROUP-MA (25.4 ± 4 kg/m^2, $p = 0.6715$). Similarly, the waist-hip (W-H) ratio was identical between the groups (0.92 ± 0.07 for GROUP-NA and 0.92 ± 0.06 for GROUP-MA, $p > 0.9999$). These findings indicate that anthropometric measurements (BMI and W-H ratio) showed non-significant differences.

In this study, lipid profile parameters show non-significant differences between GROUP-NA and GROUP-MA. Total cholesterol (TC) levels were 193 ± 40.6 mg/dL for GROUP-NA and 195.5 ± 43.3 mg/dL for GROUP-MA ($p = 0.7455$). Triglycerides (TG) levels were 195.1 ± 607.8 mg/dL for GROUP-NA and 196.8 ± 160.7 mg/dL for GROUP-MA ($p = 0.9464$). LDL-C levels were 113.2 ± 31 mg/dL for GROUP-NA and 111.9 ± 31.2 mg/dL for GROUP-MA ($p = 0.8193$). High-density lipoprotein cholesterol (HDL-C) levels were 51.1 ± 13.1 mg/dL for GROUP-NA and 52.5 ± 13.5 mg/dL for GROUP-MA ($p = 0.5662$). These findings indicate non-significant differences in lipid profiles between the two groups. J.S. Jensen *et al*. [27] also reported a significant association between lipid abnormalities and microalbuminuria, with cases showing higher TC levels (188.54 ± 29.25 mg/dL) compared to controls (174 ± 17.90 mg/dL). However, no significant variations in TC, TG, LDL-C, or HDL-C levels were found between the two groups in this investigation, demonstrating diversity between populations.

This study highlighted the significant correlation between systolic blood pressure (SBP), pulse pressure (PP), and albuminuria in hypertensive patients without diabetes, suggesting that elevated SBP and widened PP were key contributors to early renal impairment. The findings

emphasised that SBP and PP, rather than diastolic blood pressure (DBP), are stronger predictors of microalbuminuria, likely due to their role in endothelial dysfunction and glomerular pressure fluctuations. Additionally, the significant correlation between albuminuria and total protein levels suggests potential systemic inflammatory or vascular mechanisms contributing to renal damage. These results underscore the importance of early and aggressive blood pressure management, particularly targeting SBP and PP, to mitigate the risk of albuminuria and subsequent renal complications.

CONCLUSIONS

The findings of this study reinforce the crucial role of blood pressure regulation in preventing renal and cardiovascular complications in hypertensive patients without diabetes. The significant positive correlation between systolic blood pressure (SBP), pulse pressure (PP), and albuminuria suggests that elevated SBP and widened PP contribute to microvascular damage and increased glomerular permeability, leading to early renal impairment. Clinically, this underscores the necessity of stringent blood pressure control strategies, including lifestyle modifications, regular monitoring, and the use of appropriate antihypertensive therapies to maintain optimal SBP and PP levels. Effective blood pressure management not only reduces the risk of albuminuria and slows the progression of chronic kidney disease but also mitigates the risk of cardiovascular events such as stroke and heart failure, given the well-established link between hypertension and vascular dysfunction. Future research should aim to validate and expand upon the observed statistically significant correlations between albuminuria and systolic blood pressure ($\rho = 0.25$, $p = 0.032$), pulse pressure ($\rho = 0.30$, $p = 0.015$), and serum albumin ($\rho = 0.28$, $p = 0.026$), to better elucidate their predictive value for early renal impairment in hypertensive patients without diabetes.

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CONFLICT OF INTEREST

None.

REFERENCES

[1] Kidney Disease: Improving Global Outcomes (KDIGO) Blood Pressure Work Group. KDIGO 2021 clinical practice guideline for the management of blood pressure in chronic kidney disease. Kidney Int. 2021;99(3):1–87. DOI: 10.1016/j.kint.2020.11.003

[2] Zhang W, Liu CY, Ji LN, Wang JG. Blood pressure and glucose control and the prevalence of albuminuria and left ventricular hypertrophy in patients with hypertension and diabetes. J Clin Hypertens. 2020;22(2):212–20. DOI: 10.1111/jch.13793

[3] Coşkun Ş, Cordan J, Mehmetoğlu E, Sağ S, Yeşilbursa D, Serdar OA, et al. Association between microalbuminuria and pulse pressure among patients with isolated systolic and diastolic hypertension. J Updates Cardiovasc Med. 2021;9(1):39–48. DOI: 10.32596/ejcm.galenos.2020-10-056

[4] Liu L, Wu X, Tang Q, Miao Y, Bai X, Li J, et al. Positive association of pulse pressure with presence of albuminuria in Chinese adults with prediabetes: A community-based study. Metab Syndr Relat Disord. 2024;22(4):302–14. DOI: 10.1089/met.2023.0177

[5] Sołtysiak J, Skowrońska B, Maćkowiak-Lewandowicz K, Blumczyński A, Elżbieta K, Ostalska-Nowicka D, et al. Ambulatory blood pressure parameters and their association with albuminuria in adolescents with type 1 diabetes mellitus. Pediatr Nephrol. 2024;39(10):3037–47. DOI: 10.1007/s00467-024-06416-3

[6] Suzuki D, Hoshide S, Kario K. Associations between day-by-day home blood pressure variability and renal function and albuminuria in patients with and without diabetes. Am J Hypertens. 2020;33(9):860–8. DOI: 10.1093/ajh/hpaa091

[7] Durak BA, Durak Mİ, Özbakkaloğlu A. The relationship between proteinuria and ambulatory blood pressure in hypertensive patients. Cardiovasc Surg Int. 2025;12(1):28–35. DOI: 10.5606/e-cvsi.2025.1775

[8] Rossi GP, Bisogni V, Rossitto G, Maiolino G, Cesari M, Zhu R, et al. Practice recommendations for diagnosis and treatment of the most common forms of secondary hypertension. High Blood Press Cardiovasc Prev. 2020;27(6):547–60. DOI: 10.1007/s40292-020-00415-9

[9] Lamirault G, Artifoni M, Daniel M, Barber-Chamoux N. Resistant hypertension: Novel insights. Curr Hypertens Rev. 2020;16(1):61–72. DOI: 10.2174/1573402115666191011111402

[10] The World Medical Association. Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects [Internet]. [cited 22024 November 28]. Available from: https://surl.lu/xnqdiv

[11] Song J, Wang P, Li H. U-shaped relationship between fasting blood glucose and urinary albumin-to-creatinine ratio in the general United States population. Front Endocrinol. 2024;15:1334949. DOI: 10.3389/fendo.2024.1334949

[12] Yimthiang S, Pouyfung P, Khamphaya T, Kuraeiad S, Wongrith P, Vesey DA, et al. Effects of environmental exposure to cadmium and lead on the risks of diabetes and kidney dysfunction. Int J Environ Res Public Health. 2022;19(4):2259. DOI: 10.3390/ijerph19042259

[13] Chandie Shaw PK, Berger SP, Mallat M, Frölich M, Dekker FW, Rabelink TJ. Central obesity is an independent risk factor for albuminuria in nondiabetic South Asian subjects. Diabetes Care. 2007;30(7):1840–4. DOI: 10.2337/dc07-0028

[14] Lei B, Nakano D, Fan YY, Kitada K, Hitomi H, Kobori H, et al. Add-on aliskiren elicits stronger renoprotection than high-dose valsartan in type 2 diabetic KKAy mice that do not respond to low-dose valsartan. J Pharmacol Sci. 2012;119(2):131–8. DOI: 10.1254/jphs.12031fp

[15] Coca SG, Ismail-Beigi F, Haq N, Krumholz HM, Parikh CR. Role of intensive glucose control in development of renal end points in type 2 diabetes mellitus: Systematic review and meta-analysis intensive glucose control in type 2 diabetes. Arch Internal Med. 2012;172(10):761–9. DOI: 10.1001/archinternmed.2011.2230
[16] Ahmed Aziz KM. Association of high levels of spot urine protein with high blood pressure, mean arterial pressure and pulse pressure with the development of diabetic chronic kidney dysfunction or failure among diabetic patients. Statistical regression modeling to predict diabetic proteinuria. Curr Diabetes Rev. 2019;15(6):486–96. DOI: 10.2174/1573399814666180924114041
[17] Bhusal KR, Devkota S, Pathak S, Khanal P, Khanal U, Thapalia P, et al. Prevalence of microalbuminuria in non-diabetic hypertensive patients and its correlation with changes in left ventricular and left atrial characteristics. J Nepal Health Res Counc. 2023;20(4):838–41. DOI: 10.33314/jnhrc.v20i4.3786
[18] Thieme K, Oliveira-Souza M. Renal hemodynamic and morphological changes after 7 and 28 days of leptin treatment: The participation of angiotensin II via the AT_1 receptor. PLoS One. 2015;10(3):e0122265. DOI: 10.1371/journal.pone.0122265
[19] Lee E, Oh HJ, Park JT, Han SH, Ryu DR, Kang SW, et al. The incidence of cardiovascular events is comparable between normoalbuminuric and albuminuric diabetic patients with chronic kidney disease. Medicine. 2016;95(15):e3175. DOI: 10.1097/MD.0000000000003175
[20] Xie N, Li X, Zhong Q, Zhou D, Cai A, Zhang Y, et al. Association of systolic blood pressure and pulse pressure with microalbuminuria in treatment-naïve hypertensive patients. Arch Med Sci. 2019;15(4):832–6. DOI: 10.5114/aoms.2018.77727
[21] Kim BJ, Lee HJ, Sung KC, Kim BS, Kang JH, Lee MH, et al. Comparison of microalbuminuria in 2 blood pressure categories of pre hypertensive subjects. Circ J. 2007;71(8): 1283–7. DOI: 10.1253/circj.71.1283
[22] Jung YH, Cho AR, Chung TH, Lee YJ. Association between systolic blood pressure and albuminuria in elderly people without type 2 diabetes or chronic kidney disease. Korean J Fam Pract. 2016;6(5):509–13. DOI: 10.21215/kjfp.2016.6.5.509
[23] Bangalore S, Gong Y, Cooper-DeHoff RM, Pepine CJ, Messerli FH. 2014 Eighth Joint National Committee panel recommendation for blood pressure targets revisited: Results from the INVEST study. J Am Coll Cardiol (JACC). 2014;64(8):784–93.
[24] Basu A, Jhala JS. Association of microalbuminuria in non-diabetic and non-hypertensive patients with myocardial infarction. Int J Adv Med. 2015;2(3):196–200. DOI: 10.18203/2349-3933.ijam20150003
[25] Mimarm A. Microalbuminuria in essential hypertension. Clin Exp Hypertens. 1997;19(5–6):753–67. DOI: 10.3109/10641969709083184
[26] Cirillo M, Senigalliesi L, Laurenzi M, Alfieri R, Stamler J, Stamler R, et al. Microalbuminuria in nondiabetic adults. Relation of blood pressure, body mass index, plasma cholesterol levels, and smoking: The Gubbio population study. Arch Intern Med. 1998;158(17):1933–9. DOI: 10.1001/archinte.158.17.1933
[27] Jensen JS, Borch-Johnsen K, Jensen G, Feldt-Rasmussen B. Atherosclerotic risk factors are increased in clinically healthy subjects with microalbuminuria. Atherosclerosis. 1995;112(2):245–52. DOI: 10.1016/0021-9150(94)05420-n

Кореляція систолічного артеріального та пульсового тиску з альбумінурією у пацієнтів з артеріальною гіпертензією без діабету

Санджив Тхінд

Доктор медицини, ординатор
Хіндський інститут медичних наук
225001, м. Сафедабад, Барабанкі, Уттар-Прадеш, Індія
https://orcid.org/0000-0002-7528-1973

Чандра Шекхар Тіварі

Доктор медицини, доцент
Хіндський інститут медичних наук
225001, м. Сафедабад, Барабанкі, Уттар-Прадеш, Індія
https://orcid.org/0009-0001-6201-0007

Ніраж Тріпаті

Доктор медицини, професор
Хіндський інститут медичних наук
225001, м. Сафедабад, Барабанкі, Уттар-Прадеш, Індія
https://orcid.org/0000-0002-0565-6602

Анотація. Артеріальна гіпертензія та мікроальбумінурія (МАУ) є незалежними, але взаємопов'язаними маркерами серцево-судинної та ниркової дисфункції. Хоча МАУ є раннім індикатором ураження нирок, її зв'язок з компонентами артеріального тиску (АТ), зокрема систолічним артеріальним тиском (САТ) та пульсовим тиском (ПТ), залишається недостатньо вивченим у популяції гіпертензивних пацієнтів без діабету. Метою даного дослідження було встановити незалежний зв'язок між цими компонентами АТ і МАУ, визначити, який з параметрів має сильнішу кореляцію, та поглибити розуміння раннього ниркового ураження в контексті ведення пацієнтів з артеріальною гіпертензією. Це аналітичне поперечне дослідження проводилося в Хіндському інституті медичних наук, Сафедабад, Барабанкі, Уттар-Прадеш, протягом 12 місяців і включало 120 пацієнтів з гіпертензією, які звернулися до амбулаторного або стаціонарного відділень. Пацієнтів із супутніми захворюваннями, такими як цукровий діабет, ниркова недостатність або явна протеїнурія, було виключено. Артеріальний тиск вимірювався двічі за допомогою стандартного сфігмоманометра, а мікроальбумінурія визначалась імунотурбідиметричним методом. Основним показником МАУ був співвідношення альбумін/креатинін у сечі (ACR). САТ та ПТ продемонстрували значущу позитивну кореляцію з МАУ ($\rho = 0,25$, $p = 0,032$; $\rho = 0,30$, $p = 0,015$ відповідно), тоді як діастолічний тиск (ДАТ) мав негативний, але незначущий зв'язок ($\rho = -0,20$, $p = 0,065$). Серед антропометричних параметрів індекс маси тіла (ІМТ) та співвідношення талія/стегна не виявили значущих відмінностей між групами. Середній ACR був суттєво вищим у групі з МАУ (182,5 ± 156,5 мг/л), ніж у групі без МАУ (17,6 ± 7,1 мг/л; $p < 0,0001$). Показники ліпідного профілю, глюкози натще та функції нирок (сироватковий креатинін, сечовина) були подібними в обох групах, що підкреслило важливість компонентів АТ як основних предикторів альбумінурії. Сильна позитивна кореляція між САТ, ПТ і МАУ підкреслила необхідність точного контролю АТ у гіпертензивних пацієнтів без діабету. Регулярний моніторинг і терапія, спрямована на оптимізацію рівнів САТ і ПТ, можуть допомогти зменшити ниркові ушкодження та знизити серцево-судинні ризики

Ключові слова: ниркова дисфункція; серцево-судинні захворювання; співвідношення альбумін/креатинін у сечі; нефропатія