Original Article

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Assessment of Urinary Albumin-Creatinine Ratio and Estimated Glomerular Filtration Rate in Non-Diabetic Hypertensive Patients Attending a TertiaryCare Hospital in Chattogram, Bangladesh

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Abstract

Background: Hypertension is a major risk factor for Chronic Kidney Disease (CKD) often causing subclinical renal damage long before overt symptoms appear. Microalbuminuria, as reflected by the Urinary Albumin-to-Creatinine Ratio (UACR) and a decline in estimated Glomerular Filtration Rate (eGFR) are key indicators of early renal impairment. Evaluating these parameters in non-diabetic hypertensive individuals can aid in timely identification of renal involvement. This study aimed to assess the UACR and eGFR in non-diabetic hypertensive patients compared to normotensive individuals attending a tertiary care hospital in Chattogram, Bangladesh.

Materials and methodos: This hospital-based cross-sectional comparative study was conducted at Chittagong Medical College Hospital during the period from January to December 2023. A total of 136 participants were enrolled using purposive sampling and divided into two equal groups: hypertensive (n=68) and normotensive (n=68). Key variables were measured include UACR, eGFR, systolic and diastolic blood pressures, Body Mass Index (BMI) serum creatinine and lipid profile.

Results: The prevalence of microalbuminuria was significantly higher in the hypertensive group compared to the normotensive group (33.8% vs. 7.4%, p<0.001). Hypertensive participants had higher mean age, BMI, systolic and diastolic blood pressures compared to normotensive counterparts (p<0.001 for all). The mean eGFR was significantly lower in hypertensive individuals (85.64 ± 14.15 vs. 94.28 ± 14.09 mL/min/1.73 m², p=0.001) while the mean UACR was significantly higher (49.15 ± 10.56 vs. 10.08 ± 1.16 mg/g, p<0.001). Multivariate linear regression identified SBP (β=0.619, p<0.001) as the only independent predictor of UACR levels. No variable independently predicted eGFR in the regression model. A graded relationship was observed between hypertension severity and microalbuminuria, with the highest prevalence noted in stage 2 hypertension (50%).

Conclusion: Microalbuminuria is significantly more prevalent among hypertensive patients and is strongly associated with early organ damage. Early detection through routine screening in hypertensive individuals is recommended to prevent long-term complications.

Key words: Estimated Glomerular Filtration Rate (eGFR); Hypertension; Non-diabetic; Urinary Albumin-to-Creatinine Ratio (UACR).

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Introduction

Hypertension is a leading global public health concern and a major contributor to morbidity and mortality, primarily through its association with cardiovascular and renal diseases. According to the World Health Organization, over 1.28 billion adults aged 30–79 years globally have hypertension, with a significant proportion residing in low- and middle-income countries such as Bangladesh.[1,2] Long-standing hypertension can lead to functional and structural changes in the kidneys, often culminating in CKD if not identified and managed early.[3]

Microalbuminuria, defined as the urinary excretion of 30–300 mg of albumin per gram of creatinine, is one of the earliest detectable markers of renal damage. It reflects increased glomerular permeability, often due to endothelial dysfunction, and is now recognized not only as a marker of renal injury but also as an independent predictor of cardiovascular morbidity and mortality.[4] Increased urinary albumin excretion is observed more frequently in hypertensive patients than in normotensive individuals, even in the absence of overt diabetes or kidney disease.[5] Another critical indicator of renal function is the eGFR, which provides a measure of how efficiently the kidneys are filtering waste products. Hypertension is associated with a progressive decline in eGFR, particularly in those with coexisting risk factors or subclinical renal damage.[6]

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Despite the clinical importance of UACR and eGFR, routine assessment in hypertensive patients-especially those without diabetes-is often underutilized in many healthcare settings, including Bangladesh.[7] Early identification of microalbuminuria and reduced eGFR may facilitate timely interventions to slow the progression of renal and cardiovascular complications.[8]

The present study aimed to evaluate the UACR and eGFR among non-diabetic hypertensive patients attending a tertiary care hospital in Chattogram, Bangladesh. By comparing these parameters with normotensive controls, the study seeks to explore the extent of early renal involvement in hypertensive patients, thereby highlighting the importance of early screening and monitoring in routine clinical practice.

Materials and methods

A cross-sectional comparative study was conducted from January 2023 to December 2023 in the Department of Medicine, Chittagong Medical College Hospital and the Department of Biochemistry, Chittagong Medical College, Chattogram, Bangladesh. Ethical approval was obtained from the Ethical Review Committee of Chittagong Medical College (Memo No: 59.27.0000.013.19.PG.009.2023/978). Permission was also granted by the concerned departments. Written informed consent was obtained from all participants after the aims and procedures of the study were clearly explained. A pre-designed and pre-tested structural questionnaire was used for collection of data.

A total of 136 adult participants aged 25–70 years were enrolled using a non-probability purposive sampling technique and categorized into two groups: Group A included 68 non-diabetic hypertensive patients, while Group B included 68 normotensive, non-diabetic individuals as controls. Patients with diabetes mellitus, CKD, nephrotic syndrome, urinary tract infection, heart failure, pregnancy, acute infections, or those receiving steroid therapy were excluded.

Participants were recruited from the Outpatient Department (OPD) of the Department of Medicine during routine clinical visits. Each participant was instructed to report to the Department of Biochemistry between 8:00 and 9:00 AM following an overnight fast (8–12 hours). After taking a short history, anthropometric measurements were recorded. Following aseptic precautions, 6 mL of fasting venous blood was collected from each participant with minimal tourniquet use. Blood was allowed to clot and centrifuged at 4000 rpm for 10 minutes. The serum was used to measure fasting blood glucose, serum creatinine and lipid profile. A midstream spot urine sample was collected to estimate UACR and perform Routine Urine Examination (R/E) to rule out urinary tract infection. Urinary albumin and creatinine were estimated using Cobas C311 systems by immunoturbidimetric and Jaffe kinetic methods, respectively. UACR was expressed as mg/g of creatinine. Serum creatinine was measured using the Humalyzer 2000 semi-automated analyzer by the modified Jaffe method. Fasting blood glucose was measured by using highly accurate enzymatic method (Glucose oxidase method) using large automated biochemical analyzer (Beckman). Lipid profile were estimated using enzymatic assays on automated clinical chemistry analyzer (Biochem FC-200). The eGFR was calculated using the Cockcroft-Gault equation, with sex-adjusted values.[9]

Hypertension was defined based on either a systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg on two or more occasions, or the current use of antihypertensive medications. Classification followed the ISH 2020 guidelines, where blood pressure was categorized into normal, high normal and Grade 1 or 2 hypertension depending on systolic and diastolic values.[10] Urinary Albumin-Creatinine Ratio (UACR) was used to assess early renal involvement. According to NKF/KDOQI guidelines, UACR values <30 mg/g were considered normoalbuminuria, values between 30–299 mg/g indicated microalbuminuria and values ≥300 mg/g were categorized as macroalbuminuria.[11]

Strict safety precautions were followed throughout the sample collection and laboratory procedures. Data were collected using a pre-tested structured questionnaire and recorded on a standardized case record form. Data were entered and analyzed using IBM SPSS version 26.0. Variables were

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summarized using means ± SD or SEM. Between-group comparisons were performed using the independent samples t-test and Chi-square test where appropriate. Multivariate linear regression analysis was performed to identify the independent associations of UACR and eGFR with clinical and biochemical variables such as age, BMI, systolic and diastolic blood pressure, fasting blood sugar and lipid profile. Variables with a p-value <0.05 were considered statistically significant.

Results

Hypertensive participants were significantly older and had higher BMI compared to normotensive individuals. Systolic and diastolic blood pressures were also significantly higher in the hypertensive group. A higher proportion of males was observed in the hypertensive group compared to the normotensive group without any statistical significance (Table I).

Table I Demographic and clinical characteristics between hypertensive and normotensive participants

Characteristics	Study Groups		p value
	Hypertensive (n=68)	Normotensive (n=68)	
Age, Years	51.63 ± 7.95	46.0 ± 8.81	<0.001*
Gender			
Male	49 (72.0)	42 (61.7)	0.202†
Female	19 (28.0)	26 (38.3)	
BMI, kg/m^2	25.58 ± 3.97	21.62 ± 2.59	<0.001*
SBP, mmHg	153.51 ± 17.66	126.69 ± 5.63	<0.001*
DBP, mmHg	93.71 ± 9.27	81.69 ± 2.38	<0.001*

Data were expressed as mean ±SD or frequency (%). BMI: Body Mass Index, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure. *Independent sample t test, †Chi-square test.

Hypertensive participants had significantly higher mean UACR (49.15 ± 10.56 mg/g vs. 10.08 ± 1.16 mg/g, p < 0.001) serum creatinine (1.03 ± 0.02 mg/dl vs. 0.85 ± 0.13 mg/dl, p = 0.001) total cholesterol (p = 0.022) triglycerides (p = 0.008), and LDL-C (p = 0.007) compared to normotensive individuals. Conversely, eGFR was significantly lower in the hypertensive group (74.92 ± 1.75 vs. 93.51 ± 2.68 ml/min/1.73m², p < 0.001). No significant differences were observed in fasting blood sugar (p = 0.480) or HDL-C levels (p = 0.270) (Table II).

Table II Biochemical parameters between hypertensive and normotensive participants

Variables	Study Groups		p value*
	Hypertensive (n=68)	Normotensive (n=68)	
UACR, mg/g	49.15 ± 10.56	10 .08 ± 1.16	<0.001
FBS, mmol/L	5.59 ± 0.07	5.64 ± 0.05	0.480
S Creatinine, mg/dl	1.03 ± 0.02	0.85 ± 0.13	0.001
eGFR(cal) ml/min/1.73m^2	74.92 ± 1.75	93.51 ± 2.68	< 0.001
TC, mg/dl	202.25 ± 5.86	186.07 ± 3.79	0.022
TG, mg/dl	258.81 ± 19.23	185.12 ± 9.62	0.008
HDL-C, mg/dl	44.63 ± 1.17	42.87 ± 1.09	0.270
LDL-C, mg/dl	133.69 ± 4.06	120.10 ± 2.90	0.007

Data were expressed as mean ± Standard Error of Mean (SEM) *Independent sample t test, UACR: Urinary Albumin Creatinine Ratio, FBS: Fasting Blood Sugar, eGFR: Estimated Glomerular Filtration Rate, HDL-C: High Density Lipoprotein Cholesterol, LDL-C: Low Density Lipoprotein Cholesterol, TG: Triglyceride, TC: Total Cholesterol.

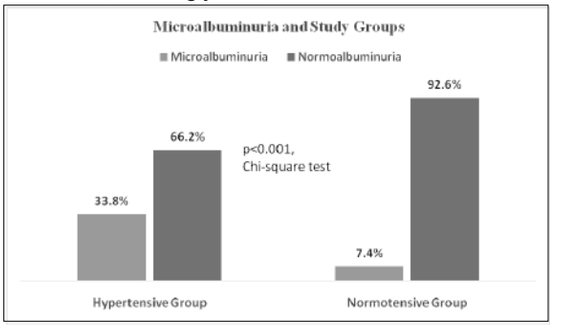

Figure 1 Microalbuminuria in hypertensive and normotensive Groups

Among the 136 study participants, micro-albuminuria was present in 33.8% (23 out of 68) of the non-diabetic hypertensive patients (Group A) whereas only 7.4% (5 out of 68) of the normotensive individuals (Group B) exhibited microalbuminuria. This difference was statistically significant (p < 0.001) indicating a strong association between hypertension and the presence of micro-albuminuria (Figure 1).

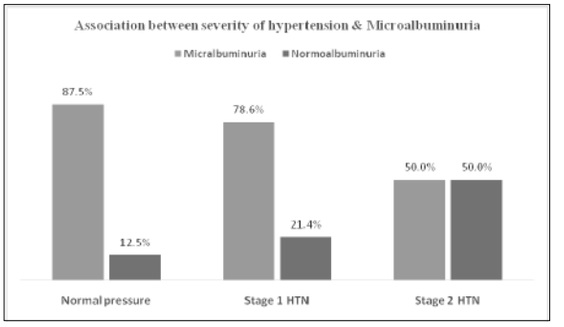

Figure 2 Association between severity of hypertension and microalbuminuria in Hypertensive Group

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Figure 2 showed a clear association between the severity of hypertension and the presence of microalbuminuria. Among patients with normal blood pressure, only 12.5% had microalbuminuria, whereas its prevalence increased to 21.4% in those with stage 1 hypertension and significantly to 50% in stage 2 hypertensive patients.

The multivariate linear regression analysis in Table III revealed that among the variables analyzed, only SBP showed a significant independent association with UACR) with a strong positive standardized beta coefficient (β= 0.619, $p < 0.001$). This indicates that higher SBP was significantly associated with elevated UACR levels. Other variables such as age, BMI, DBP, FBS, total cholesterol, triglycerides, HDL-C and LDL-C did not show statistically significant independent associations with UACR ($p > 0.05$). These findings suggest that systolic blood pressure is the primary predictor of increased UACR, reflecting early renal involvement in hypertensive patients.

Tabel III Multivariate linear regression analysis for the independent relation of UACR levels with other variables (n=138)

Variables	Standardized Coefficients Beta	p value	95.0% Confidence Interval for B Lower Bound	Upper Bound
Age, Years	0.142	0.059	-0.026	1.419
BMI, kg/m^2	0.052	0.538	-1.242	2.370
SBP, mmHg	0.619	<0.001	0.932	1.926
DBP, mmHg	-0.161	0.095	-1.680	.137
FBS, mmol/L	0.059	0.396	-6.508	16.340
TC, mg/dl	0.033	0.718	-0.153	.222
TG, mg/dl	0.116	0.135	-0.012	.089
HDL, mg/dl	0.031	0.677	-0.536	.823
LDL, mg/dl	-0.093	0.327	-0.405	.136

Dependent Variable: UACR.

The multivariate linear regression analysis for eGFR showed that none of the examined variables-including age, BMI, systolic and diastolic blood pressure, fasting blood sugar, total cholesterol, triglycerides, HDL-C and LDL-C—had a statistically significant independent association with eGFR levels (all $p > 0.05$). Although BMI demonstrated a positive trend (β = 0.179, p = 0.097) this did not reach statistical significance. These results suggest that within this study population, eGFR was not independently influenced by the analyzed demographic or biochemical variables.

Tabel IV Multivariate linear regression analysis for the independent relation of eGFR levels with other variables (n=138)

Variables	Standardized Coefficients Beta	p value	95.0% Confidence Interval for B Lower Bound	Upper Bound
Age, Years	-0.136	0.156	-0.880	0.143
BMI, kg/m^2	0.179	0.097	-0.198	2.359
SBP, mmHg	-0.150	0.281	-0.544	0.159
DBP, mmHg	-0.077	0.529	-0.848	0.438
FBS, mmol/L	0.033	0.711	-6.570	9.604
TC, mg/dl	0.077	0.509	-0.088	0.177
TG, mg/dl	-0.047	0.630	-0.045	0.027
HDL, mg/dl	0.068	0.478	-0.308	0.654
LDL, mg/dl	-0.042	0.728	-0.225	0.158

Dependent Variable: eGFR.

Discussion

This study evaluated renal function markers-UACR and eGFR-among non-diabetic hypertensive patients compared to normotensive controls. The findings revealed a significantly higher prevalence of microalbuminuria (33.8%) in hypertensive patients compared to normotensive individuals (7.4%) suggesting early renal involvement in the hypertensive population. This aligns with findings from Dhaka, Bangladesh, where a prevalence of approximately 10% was reported among non-diabetic hypertensive individuals, underscoring that microalbuminuria is not uncommon even in South Asian populations when adequately screened.[7,12] Globally, comparable rates around 30% have been documented in both Nepal and India, affirming the external validity of our observations.[13,14] Poudel et al. and Salim and Christopher, also demonstrated a significant association between microalbuminuria and hypertension.[13,14] Furthermore, Stamm et al. reported a significantly higher frequency of target organ damage in patients with microalbuminuria (76.2%) compared to those without (43.9%) with statistical significance ($p = 0.006$) reinforcing the clinical relevance of early renal markers in hypertensive populations.[15] In aggregates, results indicating that hypertension contributes to glomerular damage, leading to increased urinary albumin excretion even in the absence of diabetes. Hypertensive patients also exhibited significantly higher mean UACR levels and lower eGFR values than their normotensive counterparts, reinforcing the concept that hypertension is an independent

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risk factor for renal impairment. Similar differences in mean were found by Poudelet al.[12] and Vijay Kumar. et al.[16] Among the various clinical and biochemical variables assessed, SBP emerged as the strongest independent predictor of elevated UACR in our regression model (β= 0.619, $p < 0.001$). This is consistent with larger international studies showing that elevated SBP and declining eGFR are key risk factors for microalbuminuria even when renal function appears preserved.[17,18] In particular, the LIFE and MAGIC studies have demonstrated that microalbuminuria strongly predicts progression to overt kidney disease and cardiovascular outcomes in non-diabetic hypertensive cohorts.[18] This finding underscores the crucial role of blood pressure control in preventing hypertensive nephropathy.

Although eGFR was significantly lower in hypertensive patients than controls, it was not independently associated with any examined variable in multivariate analysis. Similar findings were reported in long-term cohort studies evaluating early-stage essential hypertension, where progression in GFR and albuminuria followed a parabolic trajectory and dynamic changes in eGFR did not consistently correlate with microalbuminuria unless stratified over longer durations.[19] This suggests that UACR elevations may precede significant declines in eGFR and highlights the sensitivity of albuminuria as an early marker of hypertensive renal injury in non-diabetic populations.

Additionally, the strong dose–response relationship between hypertension severity and microalbuminuria prevalence in our study (12.5% in patients with normal blood pressure, 21.4% in stage 1 and 50% in stage 2 hypertension),mirrors previous findings that demonstrated progressive albuminuria with worsening hypertension and higher odds of target-organ damage, including left ventricular hypertrophy, ischemic heart disease and retinopathy.[20] These data reinforce the hypothesis that microalbuminuria reflects early vascular and glomerular dysfunction, occurring before overt renal function impairment is detectable. This dose-response relationship highlights the progressive nature of hypertensive kidney damage and supports the need for early detection and intervention.

Limitations

This study was limited by its cross-sectional design, which prevented the establishment of causal relationships between hypertension and renal function markers. It was conducted in a single tertiary hospital, which may have restricted the generalizability of the findings. A relatively small sample size was used and longitudinal data were not collected, so the progression of renal impairment over time could not be assessed. Additionally, certain confounding factors, such as dietary habits, physical activity and genetic predispositions, were not evaluated.

Conclusions

This study demonstrated that non-diabetic hypertensive patients had significantly higher levels of microalbuminuria and reduced eGFR compared to normotensive individuals, indicating early renal involvement. SBP was found to be independently associated with elevated UACR levels, and the prevalence of microalbuminuria increased with the severity of hypertension. These findings suggest that microalbuminuria may serve as an early, non-invasive marker of hypertensive kidney damage, even before eGFR declines.

Recommendations

It was recommended that routine screening of Urinary Albumin-Creatinine Ratio (UACR) be implemented in hypertensive patients to detect early renal involvement. Emphasis was placed on the strict control of systolic blood pressure to prevent hypertensive kidney damage. It was further suggested that future studies be conducted using larger, multi-center cohorts and longitudinal designs to validate the present findings. Public health strategies promoting awareness of kidney health in hypertensive individuals were also encouraged.

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Contribution of authors

RB-Acquisition of data, data analysis, interpretation of data, drafting & final approval.
NT-Conception, design, interpretation of data, critical revision & final approval.
MMH-Acquisition of data, interpretation of data, critical revision & final approval.
SP-Data analysis, drafting & final approval.

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Disclosure

The authors declared no conflicts of interest.

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