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Hyperuricemia and Microalbuminuria are Independently Associated with Hypertension

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Abstract

Background: *High uric acid (UA) levels have been traditionally considered a risk factor for gouty arthritis and kidney stones. However, it has emerged recently that elevated serum UA level is a risk factor for hypertension, chronic kidney disease, and coronary artery disease. Microalbuminuria is associated with an increased risk of hypertension, renal impairment, and cardiovascular morbidity. Evidence suggests untreated essential hypertension increases serum uric acid and urinary albumin excretion.*

Aim of the study: The study aims to observe the association of serum uric acid level and microalbuminuria with hypertension.

Methods: This cross-sectional study was conducted at the Nephrology Department of Dhaka Medical College & Hospital, Dhaka, from March 2021 to August 2022 over 18 months. One hundred sixteen male and female patients were included in this study; all were dipstick-negative for proteinuria. Among them 58 patients with essential hypertension, and the remaining 58 were normotensive subjects (according to JNC VIII). After approval from the Research Review Committee (RRC) of Nephrology and Ethical Review Committee (ERC) of DMC, written informed consent was obtained from all study subjects. Patient's data on baseline characteristics (age, gender, blood pressure) and complete clinical and laboratory data were recorded in a predesigned data collection sheet. The uricase method measured serum uric acid, and microalbuminuria was calculated using the immune-turbidimetric method. After data collection, data analysis was done with SPSS version 26.

Result: It was observed that more than one-third (37.9%) of patients belonging to age were 41-50 years in the hypertensive group and 44.8% in the normotensive group. The mean age was 44.79±9.13 years in hypertensive and 41.07±9.87 years in normotensive. Almost two-thirds (65.5%) of patients were male in the hypertensive group and 33(56.9%) in the normotensive group. The two groups' age differences were statistically significant ($p<0.05$). In male patients, it was observed that more than one-third (34.2%) of patients had hyperuricemia in hypertensive, which was significantly ($p<0.05$) associated between the two groups. Hyperuricemia had a 5.2 times risk of developing hypertension (OR=5.2; 95% CI=1.18-26.11). In female patients, it was observed that three (15.0%) patients had hyperuricemia in hypertensive and 1(4.0%) in normotensive, which was not significantly ($p>0.05$) associated between the two groups. The mean of S. Uric acid was 5.3±1.3 in patients with stage I hypertension and 6.4±1.2 in patients with stage II hypertension. The differences in S. Uric acid were statistically significant ($p<0.05$) between stage I and II hypertension. Almost two-thirds (60.3%) of patients had microalbuminuria in the

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hypertensive group, which was statistically significant ($p<0.05$) between the two groups. Microalbuminuria had 16.13 times the risk of developing hypertension (OR=16.13; 95%CI=5.15-54.08). The differences in microalbuminuria were statistically significant ($p<0.05$) between the two stages of hypertension.

Conclusion: This study demonstrates a significant solid association between uric acid level and microalbuminuria with hypertension. There was an association between serum uric acid and microalbuminuria in hypertensive patients with systolic diastolic blood pressure and MAP. There was also a significant association between S. uric acid and microalbuminuria in hypertensive patients.

Keywords: Hyperuricemia; Microalbuminuria; Hypertension

Introduction

Hypertension is a significant public health issue in Bangladesh and one of the most prominent killer diseases in the world. Moreover, it is responsible for 1 in every eight deaths [1]. The prevalence of hypertension grows with aging. In more than 95% of cases, no specific underlying cause is found. Patients are said to have essential hypertension [2]. Commonly found secondary causes of hypertension include renal diseases, endocrine diseases, pregnancy, obesity, and drugs. Essential (primary) hypertension (HTN) is a multifactorial disease, and it causes secondary changes in blood vessels, the heart, and kidneys [3]. Long-term sequelae from HTN are atherosclerotic vascular disease, cardiac hypertrophy and failure, and renal failure [4]. Evidence suggests untreated essential hypertension increases serum uric acid and urinary albumin excretion [5]. In the human body, uric acid (Urate) is synthesized in the liver from purine compounds provided by the diet or the endogenous pathway of purine synthesis de novo. Some uric acid is also produced in peripheral tissues, especially the intestine and kidney. Uric acid produced in the liver is released into the circulation in its soluble form (monosodium urate), readily filtered by the glomerulus. The proximal tubular cells of the kidney reabsorb most of the uric acid, resulting in a normal fractional excretion of approximately 10% [6]. *Hyperuricemia* is a metabolic derangement that may develop in CKD [7]. Hyperuricemia may develop due to either production excretion, on, or both. However, in most cases, it occurs due to under-excretion [8]. In normal conditions, the kidney excretes about two-thirds of the daily uric acid produced by the body. Renal handling of uric acid involves four subsequent steps: Glomerular filtration, tubular secretion, reabsorption, and post-secretory reabsorption. Defects in the above steps may raise serum uric acid levels [9]. It has emerged recently that elevated serum UA level is a risk factor for hypertension [10], chronic kidney disease [11], and coronary heart disease [12].On the other hand, kidney dysfunction can increase serum urate levels due to glomerular damage, reducing serum uric acid excretion [13]. The increase in serum uric acid in hypertension is due to a decrease in renal blood flow accompanying the hypertensive state, and low renal blood flow increases urate reabsorption. Evidence also suggested that levels of uric acid correlate with the duration and severity of HTN. Stage II HTN has higher serum uric acid than stage 1 [1]. The risk of albuminuria significantly increased in patients with hyperuricemia. Microalbuminuria develops from progressive, subclinical, structural, and functional changes within the kidney and represents a sensitive marker of early renal disease, which is often found in patients with essential hypertension [14]. An increased pressure load from an elevated aortic pulse pressure, an increased peripheral resistance, and an augmented volume load from increased flow pulsation could alter the renal hemodynamics and consequently damage the renal microvasculature. Therefore, the albumin is filtered to an extent that exceeds the retrieval capacity of the proximal tubules.Moreover, the elevated angiotensin II and transforming growth factor-beta-1 in hypertension could disturb the lysosomal degradation pathway and consequently lead to albuminuria. In addition to being an early sign of kidney damage, microalbuminuria is a marker of the inflammatory process. It is often found in patients with essential hypertension or glucose intolerance. Microalbuminuria is associated with an increased risk of hypertension [15], cardiovascular morbidity [16], and renal impairment [17]. With this background, this study will explore the correlation between serum uric acid and microalbuminuria in patients with essential hypertension. This study will simplify the importance of the correlation between microalbuminuria hyperuricemia and essential hypertension.

Methodology and Materials

This cross-sectional study was conducted at the Department of Nephrology, Dhaka Medical College (DMC), from February 2021 to August 2022. A total of 116 participants were enrolled, consisting of 58 patients with essential hypertension and 58 healthy normotensive adults. Participants were approached and informed about the study's aims, objectives, and procedures. After obtaining informed written consent, clinical history was taken, and physical examinations were conducted according to standard protocols. The study received formal ethical approval from DMC's Ethical Review Committee (ERC). Patient selection was carried out based on predefined inclusion and exclusion criteria.

Inclusion criteria:

- Patients with esse ntial hypertension.
- Healthy age-matched normotensive individual.

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Exclusion criteria:

- Patients with diabetes mellitus, a history of gout, obesity (BMI >30 kg/m²), a history of alcohol abuse, or a history of using drugs known to cause hyperuricemia (e.g., thiazide diuretics, anticancer therapy).
- Patients with a history of leukemia, polycythemia, lymphoma, or any neoplastic disease.
- Patients under 18 years of age with chronic kidney disease (CKD), glomerulonephritis (GN), or hypothyroidism.

All subjects were dipstick negative for proteinuria. In some cases, the diagnosis of essential hypertension was based on excluding secondary causes by history, clinical examination, and investigation. BP measurement was done in resting condition, sitting in a position with arms resting on the arm of the chair. Thick clothing was removed, and the brachial artery was palpated. Then, the cuff was wrapped 2.5 cm above the antecubital fossa, and placing the diaphragm of the stethoscope over the brachial artery crossing the cubital fossa inflation of the cuff was done and reached above 10 mm of HG from the obliteration of the brachial artery pulsation. Then, the cuff was deflated 2 to 3 mm of HG/sec, and systolic and diastolic BP were measured on appearing and disappearing sounds. In the case of high BP, a second measurement was taken after 5 minutes of rest. In case of borderline results or suspected white coat, hypertension was confirmed by daytime ambulatory and home-based BP measurement and categorized as normotensive hypertensive. All the subjects in the hypertensive group and normotensive group enrolled for investigation of serum uric acid and microalbuminuria.

Microalbuminuria is typically defined as a 24-hour urinary albumin excretion rate (UAE) of 30-300 mg (20–200 µg/min) [14]. Hyperuricemia is serum uric acid over 7mg/dl for males and serum uric acid over 6mg/dl for females [18]. BMI defines a person as underweight, average weight, overweight, or obese instead of traditional height vs. weight charts. However, individual variations do exist. WHO recommends that ≥30.0 is obese, 25-29.9 is overweight, 18.5-24.9 is normal, and <18.5 is underweight [19].

In this study, 116 samples were collected. After all aseptic precautions, 3 ml venous blood was collected in a tube to measure serum uric acid. All laboratory investigations were carried out in the laboratory of the Department of Biochemistry, Bangabandhu Sheikh Mujib Medical University, Dhaka. All the collected blood samples were sent to the biochemistry department of BSMMU within 30 minutes and kept at an average temperature for 30 minutes.

For the measurement of serum uric acid, 11 microlitre serum was taken from a tube and centrifuge for 10-12 minutes, then a bar code was attached and ran into a multichannel biochemical analyzer (Atellica, Siemens Germany). The system automatically dispenses reagent N-ethyl-N-sulfopropyl-3-methyl-aniline into the sample and incubates the mixture at 37 degrees Celsius. The result was obtained after 7 minutes. Serum uric acid was measured using the uricase method.

For microalbuminuria, 24-hour urine was collected in an amber-colored (protect light sensitivity) container of about 3L capacity, containing toluene from 7 am to 7 am. Instruction was given not to urinate directly into the 24-hour collection container. Each time of urination, it is collected in a clean container and carefully poured into the 24-hour collection container.

Data collection and analysis:

All the patients were enrolled using a purposive sampling technique. They explained the type and nature of the study and ensured that there was no potential risk of this study, and no experimental drug was used. Data regarding sociodemographic characteristics, clinical parameters, and ultimately, the results of laboratory investigation and the patient‘s current medical document, along with these other variables, was recorded. Every individual questionnaire was preserved with proper identification of the patient, maintaining confidentiality. Collected data were compiled and edited. The data were then processed with the help of the software Statistical Package for Social Sciences (SPSS, version 26, Chicago, IL). Categorical data were presented as frequency with percentage and numerical data as mean with standard deviation or median with range. The statistical tests for different variables were performed using the Chi-square, Unpaired t-test, and Pearson correlation tests. P value<0.05 was considered statistically significant.

Result

One-third (37.9%) of patients belonged to the hypertensive group at 41-50 years of age and 26(44.8%) to the normotensive group. The two groups' age differences were statistically significant (p<0.05). Almost two-thirds (65.5%) of patients were male and one-third were female in the hypertensive group with no significant differences (Table 1). The hypertensive group had a significantly higher mean systolic blood pressure (154.91±10.07 mmHg) than the normotensive group (122.93±7.95 mmHg). Similarly, the mean diastolic blood pressure was notably higher in the hypertensive group (93.19±7.24 mmHg) than in the normotensive group (76.21±6.16 mmHg). The mean arterial pressure (MAP) further highlighted significant differences, with the hypertensive group averaging 112.96±5.14 mmHg compared to 91.78±6.19 mmHg in the normotensive group (Table 2). It was observed that the mean S. uric acid was 6.71±1.10 mg/dl in hypertensive and 5.35±1.31 mg/dl in normotensive. At the same time, urinary micro-albumin was 124.41±76 mg in hypertensive and 29.78±15.79 mg in normotensive. The difference between s.uric acid level and

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microalbuminuria between hypertensive and normotensive was significant (Table 3). Table 4 shows that 13(34.2%) of patients had Hyperuricemia in the hypertensive group and 3(9.1%) in the normotensive group, which was significant ($p<0.05$) between the two groups. Hyperuricemia had a 5.2 times risk of developing hypertension (OR=5.2; 95% CI=1.18-26.11). Male's mean S. Uric acid was 6.86±1.22 mg/dl in hypertensive and 5.34±1.37 mg/dl in normotensive (Table 4). In the hypertensive group, 15% had Hyperuricemia, compared to 4% in the normotensive group. The OR for Hyperuricemia in hypertensive females was 4.24, with significant association. Additionally, the majority of both hypertensive (85%) and normotensive (96%) females had normal uric acid levels (Table 5). The mean serum Uric Acid was 5.3±1.3 mg/dl in stage I and 6.4±1.2 mg/dl in stage II. There was a statistically significant difference in serum uric acid levels between Stage I and Stage II hypertension (Table 6). Among hypertensive individuals, 35(60.3%) exhibited microalbuminuria (30-300 mg/day), while only 5(8.6%) of normotensive individuals showed similar levels. The OR for microalbuminuria among hypertensive participants was 16.13. In contrast, 23(39.7%) of hypertensive participants and 53(91.4%) of normotensive participants had normal microalbumin levels (<30 mg/day) (Table 7). Table 8 reports that there was a statistically significant difference ($p<0.05$) in mean microalbuminuria levels between Stage I (51.79±44.62 mg/day) and Stage II (187.5±62.56 mg/day) hypertension. Furthermore, Hyperuricemia had 3.94 times the risk of developing microalbuminuria (OR=3.94; 95% CI=0.85-20.53) in hypertensive patients (Table 9).

Table 1: Demographic characteristics of study population (N=116).

Characteristics	Hypertensive (N=58)		Normotensive (N=58)		p-value
	(n)	(%)	(n)	(%)	
Age(years)					
≤30	5	8.62	5	8.62	a0.037s
31-40	13	22.41	20	34.48	
41-50	22	37.93	26	44.83	
51-60	18	31.03	6	10.34	
>60	0	0	1	1.72	
Sex					
Male	38	65.52	33	56.9	b0.340ns
Female	20	34.48	25	43.1	

Table 2: Distribution of the study population according to blood pressure (N=116).

Blood pressure (mmHg)	Hypertensive (N=58)	Normotensive (N=58)	p-value
Systolic BP			
Mean ± SD	154.91±10.07	122.93±7.95	0.001[s]
Diastolic BP			
Mean ± SD	93.19±7.24	76.21±6.16	0.001[s]
MAP			
Mean ± SD	112.96±5.14	91.78±6.19	0.001[s]

Table 3: Mean distribution of S. Uric acid and Urinary microalbumin in the study population (N=116).

Variable	Hypertensive (n=58)	Normotensive (n=58)	p-value
	Mean±SD	Mean±SD	
S. Uric acid (mg/dl)	6.71±1.10	5.35±1.31	0.001s
Microalbuminuria	124.41±76	29.78±15.79	

Table 4: Distribution of the study population according to Serum Uric Acid in males (N=71).

Variable	Hypertensive (N=38)		Normotensive (N=33)		OR (95% CI)	p-value
	(n)	(%)	(n)	(%)		
Hyperuricemia	13	34.2	3	9.1	5.2 (1.18-26.11)	a0.012s
Normal uric acid	25	65.8	30	90.9		

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Table 5: Distribution of the study population according to S. Uric acid in females (N=45).

S. Uric acid (mg/dl)	Hypertensive (N=20)		Normotensive (N=25)		OR (95% CI)	p-value
Female	(n)	(%)	(n)	(%)		
Hyperuricemia	3	15	1	4	4.24(0.34-100)	0.223ns
Normal	17	85	24	96		

Table 6: Comparison of Serum Uric Acid with stages of hypertension (N=58).

Variable	Male (N=38)	Female (N=20)	Total (N=58)
	Mean ± SD	Mean ± SD	Mean ± SD
Stage I (n=42)	5.34±1.37	5.31±1.3	5.3±1.3
Stage II (n=16)	6.6±1.27	6.17±1.22	6.4±1.2
p-value	0.001s	0.001s	0.001s

Table 7: Distribution of the study population according to microalbuminuria (N=116).

Variable	Hypertensive (N=58)		Normotensive (N=58)		OR (95% CI)	p-value
	(n)	(%)	(n)	(%)		
30-300 mg/day (Microalbuminuria)	35	60.3	5	8.6	16.13 (5.15-54.08)	0.001s
<30 mg/day (Normal)	23	39.7	53	91.4		

Table 8: Comparison of microalbuminuria with stages of hypertension (N=58).

Variable	Mean ± SD	p-value
Stage I (N=42)	51.79±44.62	0.001s
Stage II (N=16)	187.5±62.56	

Table 9: Association of hyperuricemia and microalbuminuria in hypertensive patients(N=58)`

Variable	30-300 mg/day (Microalbuminuria) (n=35)		<30 mg/day (Normoalbuminuria) (n=23)		OR (95% CI)
	n	%	n	%	
Hyperuricemia	13	37.1	3	13	3.94(0.85-20.53)
Normal	22	62.9	20	87	

Discussion

This cross-sectional study aimed to detect levels of microalbuminuria and to study the relationship between serum uric acid levels and microalbuminuria with hypertension. In this present study, it was observed that 37.9% of patients were 41-50 years old in the hypertensive group and 44.8% in the normotensive group. The mean age was 44.79±9.13 years in hypertensive and 41.07±9.87 years in normotensive. The mean age was statistically significant ($p<0.05$) between the two groups. Similarly, the Aggarwal et al. study found that the mean age was 42.50±10.89 in hypertensive patients and 41.9±9.55 in the control group [20]. Ofori and Odia also made similar observations [21]. Dar et al. [1] study observed that the mean age in cases was 51 years, and 49 was in the control group, which is higher than the present study [1]. Other observations regarding the higher mean age and age range also revealed the same results [10,22-24]. The above authors' higher and lesser mean age and age range may be due to geographical variations, racial and ethnic differences, and genetic causes that may significantly influence their study subjects. According to our study, 65.5% of patients were male in the hypertensive group and 56.9% in the normotensive group, indicating that males were predominant in this study. However, the differences were not statistically significant ($p>0.05$) between the two groups, which is consistent with Dar et al. [1]. However, female predominant is observed by Belo et al. and Ofori and Odia studies, possibly due to racial or ethnic differences [21,25]. Our study found that the mean Systolic BP was 154.91 ± 10.07 mmHg in the hypertensive group and 122.93 ± 7.95 mmHg in the normotensive group. The mean Diastolic BP was 93.19 ± 7.24 mmHg and 76.21 ± 6.16 mmHg in hypertensive and normotensive groups. The Mean Arterial Pressure (MAP) was 112±5.14 in the hypertensive group and 91.78±6.19 in the normotensive group. The mean blood pressure differences were significantly ($p<0.05$) elevated in the hypertensive group. The study of Dar et al. demonstrated that overall mean systolic BP mmHg and mean diastolic BP among hypertensive and normotensive groups closely resembled the present study [1]. Similarly, average systolic blood pressure and diastolic blood pressure were significantly ($p<0.05$) higher in the hypertensive group

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were also observed by Aggarwal et al., Ofori and Odia, and Poudel et al. [20,21,23]. In male subjects in this study, it was observed that 34.2% had hyperuricemia in hypertensive and 9.1% in normotensive, which was significantly associated with hypertension. Hyperuricemia had 5.2 times significantly ($p<0.05$) increased risk of developing hypertension with 95% CI=1.18-26.11. The mean Serum uric acid of males was significantly ($p<0.05$) higher in hypertensive patients compared to normotensive subjects. In female subjects, 15.0% and 4.0% had hyperuricemia in hypertensive and normotensive subjects, respectively, which was not significantly associated between the two groups. The mean serum uric acid was also significantly higher in hypertensive subjects compared to normotensive subjects. Dar et al. found similar results to the present study [1]. The study by AL-Sharifia et al. found that the mean serum uric acid value was higher than the present study [26]. This difference is mainly because most patients in their study were non-vegetarian in dietary habits. In contrast, the current study states that a mixed population comprises vegetarians and non-vegetarians by diet. Similar observations regarding the serum uric acid level were significantly ($p<0.05$) higher in hypertensive compared to normotensive subjects were also observed by Aggarwal et al., Ofori and Odia, Meena et al. and Poudel et al. [20,21,23,27]. This current study observed that the mean S. Uric acid was 5.34±1.37 mg/dl in stage I and 6.6±1.27 mg/dl in stage II in male patients. Similarly, in female patients, the mean S. Uric acid was 5.31±1.3 mg/dl and 6.17±1.22 mg/dl in stage I in stage II, respectively. The mean differences of S. Uric acid were significantly ($p<0.05$) higher in stage II in both male and female gender. Similar results in the present study for both males and females were found by Dar et al. [1]. The patients with higher values of uric acid levels with stage II than stage I were also observed by Tykarski et al. [28]. There was a significant positive correlation between S. uric acid and systolic blood pressure ($r=0.448$; $p=0.001$) of hypertensive patients, but no significant positive correlation was observed with diastolic blood pressure ($r=0.170$; $p=0.201$) in our study. Ofori and Odia's study showed a significant positive correlation between serum uric acid and SBP and DBP, respectively, supporting the present study [21]. The observation of our research revealed a positive significant Pearson's correlation ($r=0.367$; $p=0.005$) between MAP and S. uric acid of hypertensive patients. According to microalbuminuria, 60.3% of patients had microalbuminuria in hypertensive and 8.6% in normotensive, which was significantly ($p<0.05$) associated with hypertension. Microalbuminuria had 16.13 times significantly ($p<0.05$) increased risk of developing hypertension with 95%CI=5.15-54.08. The mean microalbuminuria was significantly ($p<0.05$) higher in hypertensive than normotensive subjects. According to Aggarwal et al. [20], 47.0% had microalbuminuria in essential hypertensive subjects. In contrast, none of the normotensive subjects had microalbuminuria, which may point toward the subclinical and subtle changes happening in the glomeruli of these patients [20]. Another study found microalbuminuria to be 26.67% in Indian hypertensive patients [29]. The overall prevalence of microalbuminuria in more than 20,000 individuals from 26 countries was 58.0% in a global study by Bohm et al. [30]. In another study, microalbuminuria was present in 54.1% of the hypertensive cases, and none had microalbuminuria in normotensive subjects [21]. Belo et al. [25] mentioned that microalbuminuria might represent a marker of severity of vascular involvement in hypertensive patients [25]. Poudel et al. found that excess body weight is associated with microalbuminuria and albuminuria independently of other risk factors such as hypertension and diabetes [23]. Their report suggested that microalbuminuria may be due to different causes other than hypertension and diabetes [31]. Regarding the comparison between stage with urinary microalbumin of the hypertensive population in this current study, it was observed that the mean urinary microalbumin was 51.79±44.62 mg in stage I and 187.5±62.56 mg in stage II. The mean microalbuminuria was significantly ($p<0.05$) elevated in stage II compared to stage I. Similarly, an association was also observed between S. uric acid and microalbuminuria in hypertensive patients. Hyperuricemia had a 3.94-fold increased risk of developing microalbuminuria in hypertensive patients. Ofori and Odia mentioned in their study that serum uric acid correlated positively with microalbuminuria [21]. Lee et al. demonstrated that serum uric acid level is associated with an increased risk for microalbuminuria in subjects with prehypertension [32].

Limitations of the study:

The study population was selected from a single hospital in Dhaka city, which limits the ability to generalize the results to the broader country. This research involved a limited population, so the findings may only apply to some populations with different cultural and dietary backgrounds. Additionally, the study was conducted over a very short period, preventing a longitudinal examination of the impact of lowering serum uric acid levels on hypertension. The small sample size further constrained the study, indicating the need for future research with a larger sample size to validate the findings.

Conclusion and Recommendations

Serum uric acid and microalbuminuria were significantly elevated in hypertensive as compared to normotensive individuals in both males and females. Hyperuricemia and microalbuminuria significantly increase the risk of developing hypertension. Early diagnosis of hyperuricemia and microalbuminuria prevents early renal damage in a hypertensive population.

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