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Original Article

Renal Function Status Among Patients of Essential Hypertension

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Abstract:

Background: Essential hypertension is typically described as elevated blood pressure without an identifiable underlying cause. However, there are important clinical, pathologic, and hemodynamic features that characterize this entity. Hypertension and renal impairment share a two-way cause and effect relationship, with each one being a common etiology of the other. Early diagnosis and proper management of hypertension reduced the burden of renal insufficiency.

Objectives: To evaluate correlation of renal function status with blood pressure of subjects having essential hypertension.

Materials and Method: This cross-sectional analytical study was carried out in the Department of Physiology, Sylhet M.A.G Osmani Medical College in collaboration with OPD of department of medicine, SOMCH, Sylhet, during the period between July 2020 to June 2021. Total 130 subjects were selected for study and allocated into two groups, 65 in each. Group A, patients with essential hypertension (as case) and group-B, age-sex matched normotensive subjects (as control) were taken. Exclusion criteria was pregnancy, any acute illness, chronic disease(heart disease, renal disease)Diabetes mellitus &other endocrine & metabolic disease, Drugs OCP containning oestrogens, steroids. Study subjects were selected by convenient sampling technique and ethical approval was obtained from the ethical review committee. Comprehensive socio-demographic information was obtained from the participants using a structured questionnaire. Clinical examination and relevant investigation were done. A venous blood sample was obtained from each patient for routine hematology, biochemical testing. The laboratory report was analyzed using standard methods. eGFR was calculated by Modification of diet in renal disease (MDRD) formula. Renal dysfunction was considered the presence of eGFR <60 ml/min/1.73 m^2 and normal eGFR is >60ml/min/1.73sqm. Statistical analysis and data processing were performed utilizing computer program SPSS and Microsoft excel.

Result: In this study age ranging from 18 to 50 years. It was observed that majority, e.g., 83(63.8%) patients belonged to age 41-50 years. Out of 130 cases 82(63.0%) cases were male and 48(36.9%) were female. Male and female ratio was 1.7:1. The mean serum creatinine (mg/dl) was 1.98 ± 0.43 in group-A; whereas 0.96 ± 0.43 in group-B. The mean serum creatinine was significantly higher in group-A than that of group-B (t=-10.659; p<0.001). On evaluation of eGER, maximum patients in group-A (e.g., 41.5%) had 60-89 ml/min/1.73 m^2, whereas in group-B 45(69.2%) patients had >90 ml/min/1.73 m^2. The mean e-GFR was significantly reduced in group-A than that of group-B (p<0.001). The occurrence of renal dysfunction was 29.2% in group-A or patients with essential hypertension, whereas this occurance was 4.60% in group-B or normal healthy subject. Positive significant correlation (r=0.637; p=0.001) between the serum creatinine, proteinuria and blood pressure. It was evident from this study, a negative significant correlation (r=-0.176; p=0.001) between the GFR and blood pressure. This indicates that as blood pressure increases, there is a corresponding decline in kidney function, reflected by a reduction in the glomerular filtration rate.

Conclusion: Hypertension contributes to a higher likelihood of developing renal dysfunction. This study revealed a 29.2% prevalence of CKD in hypertensive patients. Hypertension and CKD are closely interlinked pathophysiologic states, such that sustained hypertension can lead to worsening kidney function and progressive decline in kidney function can conversely lead to worsening blood pressure (BP) control. Therefore, proper evaluation and appropriate management of HTN is pivotal for prevention of complication.

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Introduction:

Hypertension is a global burden of non-communicable disease that significantly increases the risk of myocardial infarction, heart

failure, kidney failure, retinopathy and stroke. It ranks among the leading causes of early mortality across the globe. Of the estimated 1.13 billion people who have hypertension, fewer than one in five has it under control.[1]

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Different risk factors predisposing the hypertension, such as diabetes mellitus, dyslipidaemia, smoking, obesity, alcohol consumption, physical inactivity and unhealthy diet, may explain some of the regional heterogeneity in hypertension prevalence.[2] The main contributors to the high and rising prevalence of hypertension in less affluent countries are unhealthy diets especially excess sodium and also insufficient potassium , physical inactivity, and the consumption of alcohol (WHO, 2020).[1] In Bangladesh, approximately 20% of adult and 40–65% of elderly people suffer from HTN.[3] The prevalence of hypertension among adults was higher in lower middle-income countries (LMICs; 31.5%, 1.04 billion people) than in high-income countries (HICs; 28.5%, 349 million people). Despite the increasing prevalence, the proportions of hypertension awareness, treatment and BP control are low, particularly in lower middle-income countries, and few comprehensive assessments of the economic impact of hypertension exist.[2]

Hypertension plays a crucial role in the development of CKD by adversely affecting the blood vessels in the kidneys. Long-term, uncontrolled HTN raises pressure inside the glomeruli, impairing their filtering function. Epidemiological data among 365 outpatients with hypertension revealed, 110 (30.2%) had serum creatinine >140 μmol/L (1.6 mg/dL), 48 had serum creatinine >400 μmol/L (>4.5 mg/dL), and 96 (25.5%) had proteinuria.[5] In an autopsy study, it was showen that hypertension was an important cause of end-stage renal failure in Ghana, accounting for 33 out 78 (42%) cases.[6]

Hypertension is a closely interlinked pathophysiologic states, such that sustained hypertension can lead to worsening kidney function and progressive decline in kidney function can conversely lead to worsening blood pressure (BP) control. The pathophysiology of renal dysfunction in hypertension is a sequelae of multiple factors, including reduced nephron mass, increased sodium retention and extracellular volume expansion, sympathetic nervous system overactivity, activation of hormones including the renin-angiotensin-aldosterone system, and endothelial dysfunction.[4] Extensive experimental studies highlight proteinuria as a key factor in the progression of kidney disease toward end-stage renal failure. It contributes through several mechanisms, such as stimulating tubular chemokine production and activating the complement system. Moreover, proteinuria is a well-established indicator of chronic kidney disease progression risk. Uncontrolled hypertension leads to proteinuria through two primary mechanisms: first, by compromising the structural integrity of the glomerular capillaries, and second, by allowing excessive proteins to pass through due to heightened permeability of the capillary wall, followed by inadequate reabsorption by proximal tubular epithelial cells. Injury to the glomeruli results in elevated protein leakage into the urine, leading to the presence of abnormal protein levels, such as microalbuminuria or proteinuria. Microalbuminuria, characterized by low levels of albumin in the urine, is often an early indicator of chronic kidney disease. As the disease advances, proteinuria measured via the

protein-to-creatinine ratio becomes more prominent and is linked to worse clinical outcomes, and is associated with a poor prognosis and increased mortality & morbidity.[7] The proposed study is to evaluate correlation of renal function status with blood pressure of subjects having essential hypertension.

Materials and Methods:

This analytical study, employing a cross-sectional design, was carried out within the Department of Physiology, Sylhet M.A.G Osmani Medical College in collaboration with OPD of department of medicine, SOMCH, Sylhet. This study was carried out during the period from July 2020 to June 2021. Patients with essential hypertension attending outpatient department (OPD) of Medicine, Sylhet MAG Osmani Medical College Hospital, fulfilling the inclusion criteria within the given period was considered as study population.

This research was conducted between July 2020 and June 2021. Individuals diagnosed with essential hypertension who visited the Medicine outpatient department (OPD), fulfilling the inclusion criteria were included. Those who fulfilled the inclusion criteria were enrolled in the study. A total of 130 subjects were selected for study and allocated into two groups, 65 in each Group A consisted of individuals diagnosed with essential hypertension (considered as cases), while Group B included age and sex matched normotensive participants (serving as controls). A detailed history was taken regarding disease and clinical examination was done accordingly. Diagnosis of essential hypertension was made on the basis of patient's medical record, statement of the witness, characteristic features of manifestation, clinical examination and available medical records. After fulfilling the inclusion and exclusion criteria, patients were enrolled with unique ID. Patients were approached to take participation in the study and risk, benefits, freedom from participation were briefed. Informed written consent was obtained accordingly. A structured questionnaire containing items to elicit socio-demographic information (e.g., age, gender, resident, occupation, monthly income, level of education etc.) and clinical variables (e.g., blood pressure, serum creatinine, eGFR, spot urine ACR) were evaluated.

Height and weight were measured using a Portstad portable stadiometer (HM200P) and an Osaka digital weighing scale, respectively. Body weight was measured in light clothing and without shoes. Weight was recorded in kilogram and height recorded in meter.

Blood pressure was measured by using a sphygmomanometer (ALP K2 TOKYO JAPAN), BP was measured twice at 15 minutes intervals. Patient arm was placed on the table and the middle of the cuff was placed on the left upper arm of seated subjects. Patient sit quietly for five minutes before a reading was taken. With all aseptic precaution about 5 ml of venous blood was drawn from anticubital vein by using a disposable syringe then blood transferred to a clean test-tube. Blood samples was kept in room temperature for one hour. After clot retraction centrifugation of blood was done at 3000 rpm for 30 mins. Afterward, serum removed by disposable pasture pipettes and transferred into air tight tube.

Serum creatinine was measured by automated biochemistry analyzar, vitros 350 machine with calibration. Unit of s.creatinine mg/dl. eGFR was calculated by Modification of diet in renal disease (MDRD) equation. Renal dysfunction was considered the presence of eGFR <60 ml/min/1.73 m^2 and normal eGFR is >60ml/min/1.73sqm (AtsuhiroKanno, et al, 2012; Anand et al, 2014). Spot urine sample was collected for detection of ACR [Urinary albumin (mg/dl)/ urine creatinine (g/dl)]. All these investigations were carried out in SOMC college pathology laboratory during office hours with prior permission of concerned authorities & kind cooperation of Department of Pathology, SOMC. All investigations was done from researcher's own budget. Data was recorded in pre-tested structured data sheet and analyzed in SPSS 22.0. Categorical variables were summarized as percentages. Quantitative variables were summarized as mean standard deviation. Test for association between groups and categorical variables were performed using chi square test. For quantitative variable means was compared by student t test. $P<0.05$ was accepted as statistical significant.

Results:

It was observed that majority, e.g., 83(63.8%) patients belonged to age 41-50 years, followed by 28 patients belonged to age 31-40 years.

Participants in Group A and Group B had mean ages of 42.1 ± 9.4 years and 42.5 ± 9.8 years, respectively (Table I). Out of 130 cases 82 (63.0%) cases were male and 48 (36.9%) were female. Male and female ratio was 1.7:1 (Fig 1). The clinical profile of the participants among the groups. Group A had a mean BMI of 26.04 ± 2.92 kg/m² (range: 24.69–30.00), while Group B had a mean BMI of 21.42 ± 3.24 kg/m² (range: 19.43–24.88) (Table II). The mean serum creatinine (mg/dl) was 1.98 ± 0.43 in group-A; whereas 0.96 ± 0.43 in group-B. The mean serum creatinine was significantly higher in group-A than that of group-B (t=-10.659; p<0.001). The ACR was <30 mg/g in 35 (53.8%) patients in group-A and 54(83.0%) patients in group-B, 30–300 mg/g in 18 (27.6%) patients in group-A and 8 (12.3%) patients in group-B. ACR >300 mg/g was 12 (18.4%) patients in group-A, 3 (4.7%) in group-B and the difference was significant between group p=0.045). The mean e-GFR was significantly declined in group-A than that of group-B (p<0.001). On evaluation of eGER, maximum patients in group-A (e.g., 41.5%) had 60-89 ml/min/1.73 m^2, whereas in group-B 45(69.2%) patients had >90 ml/min/1.73 m^2. The mean e-GFR was significantly reduced in group-A than that of group-B (p<0.001) (Table III and IV). Among hypertensive participants (Group A), renal dysfunction was present in 29.2%, whereas this prevalence was 4.60% in group-B or normal healthy subjects (Fig. 2). A scatter plot illustrates a significant positive correlation (r = 0.267; p = 0.001) between serum creatinine and arterial blood pressure (Fig. 3). Correlation between ACR and blood pressure level(Fig 4). It was evident from this study, a negative significant correlation (r=-0.176; p=0.001) between the eGFR and blood pressure (Fig. 5).

Table- I: Age distribution of the participants (n=130)

Age (years)	Number of patients		Total (%)	P value
	Group A (n = 65) No. (%)	*Group B (n = 65) No. (%)*		
18-30	10(15.3%)	9(13.8%)	19(14.7%)	
31-40	13(20.0%)	15(23.0%)	28(21.5%)	0.306*
41-50	42(64.6%)	41(63.0%)	83(63.8%)	
Mean ± S.D.	*42.1±9.4*	*42.5±9.8*		0.866**

*Fisher's Exact test and **Unpaired t test were used to see the level of significance.

Figure- 1: Gender distribution of study subject (n=130

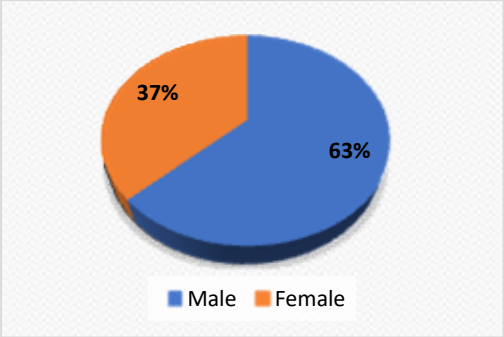


Table- II Baseline clinical profile of the participants (n=130)

Variables	Frequency & Percentage		p-value
	Group A (n = 65) No. (%)	*Group B (n = 65) No. (%)*	
Body mass index (BMI)	26.04 ± 2.92	21.42 ± 3.24	p<0.001*
Systolic BP (mm of Hg)	145.68 ± 13.07	119.76 ± 12.75	p<0.001*
Diastolic BP (mm of Hg)	82.06 ± 8.43	78.96 ± 8.15	0.0637*

* Unpaired t test was used to see the level of significance.

Table- III Distribution of the participants by laboratory parameters (n=130)

Biochemical parameters	Frequency and Percentage		p-value
	Group A (n = 65) No. (%)	*Group B (n = 65) No. (%)*	
Serum creatinine (mg/dl)	1.98 ± 0.43	0.96 ± 0.43	p=0.001*
ACR (mg/g)			0.045**
<30 mg/g	35 (53.8%)	54 (83.0%)	
30–300 mg/g	18 (27.6%)	8 (12.3%)	
>300 mg/g	12 (18.4%)	3 (4.7%)	
eGFR	64.20 ± 11.97	91.16 ± 10.54	<0.0001*

* Unpaired t test and **Chi-Square test were used to analyse the data.

Table- IV Comparison of eGFR in between groups (n=130)

eGFR (ml/min/1.73 m^2)	Frequency and Percentage		p-value
	Group A (n = 65) No. (%)	*Group B (n = 65) No. (%)*	
>90 ml/min/1.73 m^2	19 (29.2%)	45 (69.2%)	p=0.001*
60-89 ml/min/1.73 m^2	27 (41.5%)	17 (26.1%)	
30-59 ml/min/1.73 m^2	19 (29.2%)	3 (4.6%)	
15-29 ml/min/1.73 m^2	0	0	
<15 ml/min/1.73 m^2	0	0	

* Unpaired t test and **Chi-Square test were used to analyse the data.

Figure- 2: Frequency and pattern of renal dysfunction in between groups (n=130)

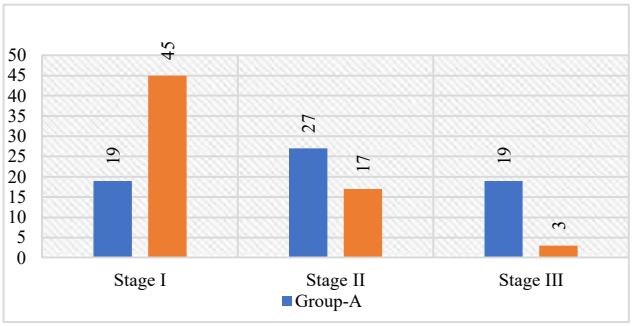
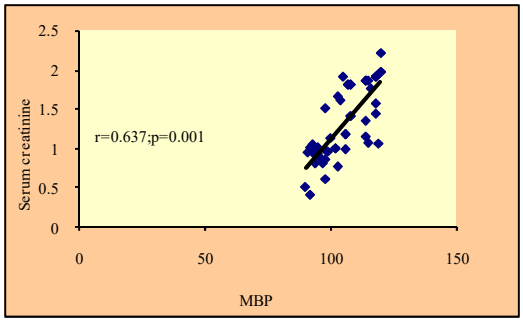


Figure in the parentheses indicate corresponding percentage;

Figure- 3: Correlation between the serum creatinine and blood pressure of patients with essential hypertension (n=65)



Scatter diagram showing positive significant correlation (r=0.637; p=0.001) between the serum creatinine and blood pressure.

Figure- 4: Correlation between ACR and blood pressure of patients with essential hypertension (n=65)

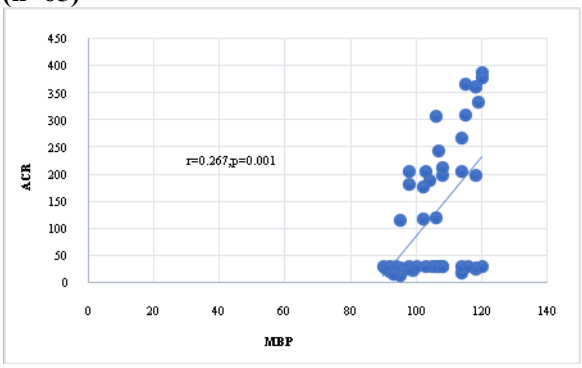
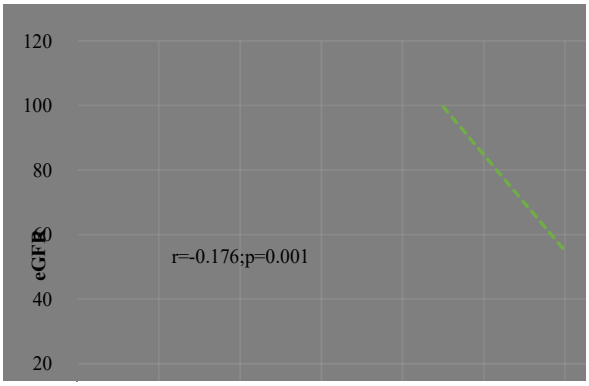


Figure- 5: Correlation between eGFR and blood pressure of patients with essential hypertension (n=65)



Discussion:

In this study age ranging from 18 to 50 years. It was observed that majority, e.g., 83(63.8%) patients belonged to age 41-50 years, followed by 28 patients belonged to age 31-40 years. The mean age was found *42.1±9.4* years in Group-A and *42.5±9.8* years in Group-B. Out of 130 cases 82(63.0%) cases were male and 48(36.9%) were female. Male and female ratio was 1.7:1. There was no significant difference between two groups in respect of demographic characteristics, which reflects the homogeneity of the groups.

Findings consistent with result of previous study. Bahrey et al.[8] conducted a study which revealed that age of the patients ranges from 30 to 87 years with the median age of 54.14 years. Out of the total patients half, 290 (50.2%), were females.[8] In another study, mean age was 59 years (range 19–90 years) and 560 (78.7%) of the patients were female.[9]

On evaluation of clinical profile, mean BMI of the group-A was 26.04 ± 2.92 (range 19.43-24.88) Kg/M^2; whereas the mean BMI of the group-B subject was 21.42 ± 3.24 (range 24.69-30.0) Kg/M^2. The mean BMI of group-A was significantly higher than that of group-B ($p<0.001$). The Systolic BP (mm of Hg) was 145.68 ± 13.07 in group-A and 119.76 ± 12.75 in group-B; SBP differ significantly between the groups (t=-1.641; p=0.001). Comparison of Diastolic BP (mm of Hg) was non-significant between study groups was shown in table.

Zhou et al.[10] demonstrated that mean BMI is significantly higher in hypertensive population and obesity considered as predisposing factor for HTN. Men are known to have a higher blood pressure than women[10] but this relationship could vary by age and geography. According to NCD-RisC data, in 2015, body mass index proportionally related to age-standardized mean SBP.

In a comparative study between hypertensive (cases) and non hypertensive patients (controls) shows that mean systolic blood pressure among cases and controls were 135.55 and 117.97 mmHg, respectively, while mean diastolic blood pressure were 83.47 and 75.62 mmHg, respectively. Mean BMI of study population was 25.17 kg/m2 with 95% CI range 24.73; 25.61. Females had higher BMI both among cases and controls across all age groups.[11]

In this study the mean serum creatinine (mg/dl) was 1.98 ± 0.43 in group-A; whereas 0.96 ± 0.43 in group-B. The mean serum creatinine was significantly higher in group-A than that of group-B (t=-10.659; $p<0.001$). On evaluation of eGER, maximum patients in group-A (e.g., 41.5%) had 60-89 ml/min/1.73 m^2, whereas in group-B 45(69.2%) patients had >90 ml/min/1.73 m^2. The mean e-GFR was significantly reduced in group-A than that of group-B ($p<0.001$). Present study shows that prevalence of renal dysfunction was 29.2% in group-A or patients with essential hypertension, whereas this prevalence was 4.60% in group-B or normal healthy subjects.

Ku et al. reported that renal dysfunction is associated with hypertensions. The prevalence ranges from 60% to 90% depending on the stage of CKD and its cause.[4] The relationship between HTN and CKD is cyclic in nature. Uncontrolled hypertension increases the risk of chronic kidney disease, accelerates its progression, and ranks as the second most common cause of end-stage renal disease[7] In a study, of the total 578 hypertensive patients the prevalence of chronic kidney disease was found to be (22.1%).[8] Epidemiological study in Bangladesh reported that overall, there were 79 (22%) participants with albuminuria, 65 (83%) of whom had microalbuminuria. Applying an albuminuria threshold of ≥ 3.4 mg/mmol creatinine, 56 participants were identified with albuminuria, while 72 (20%) were classified as having chronic kidney disease. Prevalence of CKD was evident in 26%.[12]

Long-term, uncontrolled, high BP leads to high intraglomerular pressure, impairing glomerular filtration.[4] In a study among 365 outpatients with hypertension, 110 (30.2%) had serum creatinine >140 µmol/L (1.6 mg/dL), 48 had serum creatinine >400 µmol/L (>4.5 mg/dL), and 96 (25.5%) had proteinuria.[5] A study conducted in Burkina Faso reported that chronic renal failure was present in 117 of 317 (44%) hospitalized hypertensive patients.6 Additionally, an autopsy study in Ghana found that hypertension was a significant contributor to end-stage renal failure, accounting for 33 out of 78 cases (42%).[6]

Previous study noted that overall prevalence of CKD was 46.9%; 19.1% had CKD stages 1–2

and 27.8% had CKD stages 3–5. No significant age difference was observed between patients with and without chronic kidney disease (p = 0.12). Proteinuria was present in 28.9% of the study population (95% CI: 25.6–32.4%). Among participants, 14.7% had a prior diagnosis of diabetes mellitus, with a CKD prevalence of 55% (95% CI: 42.4–62.2), which was not significantly different from the 46% prevalence (95% CI: 41.9–50.0) observed in non-diabetic individuals (p = 0.133). Overall, CKD affected 46.9% of hypertensive patients in this cohort.[9] The associated factors of CKD were age with greater than 60 years (this might be due to the nature as the age increase, the function of the kidney decrease), overweight and obese, uncontrolled hypertension, dyslipidemia and diabetic mellitus.[8]

Present study noted that a positive significant correlation (r=0.637; p=0.001) between the serum creatinine, proteinuria and blood pressure. It was evident from this study, a negative significant correlation (r=-0.176; p=0.001) between the eGFR and blood pressure. It means that increasing the blood pressure, progressively declining the renal function or reduction of glomerular filtration rate.

A notable finding from the previous study was that 28.9% of the participants exhibited proteinuria. Interestingly, its severity appeared greater in stages CKD 1, 2, and 4 compared to stages 3 and 5, though the reason for this pattern remained unclear. The underlying causes of proteinuria were not specifically investigated in that study; however, potential explanations include hypertensive renal injury, diabetic nephropathy, or undiagnosed glomerulonephritis. It is well established that effective blood pressure control and the use of angiotensin-converting enzyme inhibitors or receptor blockers can slow the progression of kidney disease.[9]

Hypertension and CKD are closely interlinked pathophysiologic states, such that sustained hypertension can lead to worsening kidney function and progressive decline in kidney function can conversely lead to worsening blood pressure (BP) control. The pathophysiology of renal dysfunction in hypertension is complex and is a sequelae of multiple factors.[4] Injury to the glomerular structures results in excessive protein passing through the filtration barrier, leading to elevated levels of protein in the urine, commonly referred to as microalbuminuria or proteinuria. Microalbuminuria defined by the presence of small quantities of albumin in the urine often serves as an early indicator of chronic kidney disease. As the condition advances, proteinuria (measured by the protein-to-creatinine ratio) becomes more pronounced and is linked to poorer clinical outcomes, including higher rates of morbidity and mortality.[7]

From the previous study, it was found that approximately one in five patients' 128 (22.1%) with 95% CI (18.5–25.4) were had an evidence of chronic kidney disease using the calculated e-GFR.[8] This result was lower than the study done in developing country. This discrepancy may be due the difference in study design, study population, and age of the participants. On the other hand, study in developing country shows higher prevalence than that of the study conducted in US (6%).[13] The variation may be due to standard living condition of the countries. Our study also higher than with study conducted in Ethiopia that was (12.2%).[14] The difference may be due to the study design and difference in study population.

Conclusion:

The current study found that 29.2% of individuals diagnosed with hypertension were affected by chronic kidney disease. There was a significant association between serum creatinine and albuminuria with blood pressure. The major associated factors were male gender, urban population, uncontrolled hypertension and overweight/obese. Addressing these risk factors is essential in controlling the epidemic of hypertension, which in turn is driving the other major NCDs as well. Lifestyle changes are the cornerstone in determining how the epidemic of hypertension will move now on. Bangladesh has achieved a remarkable progress in terms of human development and health of the people. Bangladesh is therefore an ideal place to have interventions related to lifestyle modifications which have proved beyond doubt to help control the burden of NCDs, hypertension being one of the most important ones. Therefore, the state should immediately start implementing the core

strategies under the national framework to control hypertension. This requires adequate facilities to diagnose and treat hypertension at the earliest.

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