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Original Article

Comparison of Urinary Sodium and Potassium Excretion between Hypertensive and Normotensive Individuals

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Key Words : *Hypertension, Sodium, Potassium, Bangladeshi people.*

Abstract:

Background: *Sodium and potassium intake is known to be a modifiable determinant of hypertension. Also, daily intake of these electrolytes is subject to much geographical and ethnic variation. The impact of sodium and potassium intake on blood pressure in Bangladeshi subjects in incompletely known. Our objective was to determine the relationship between urinary sodium and potassium excretion, and blood pressure in Bangladeshi subjects.*

Methods: *Patients attending the Outpatient Department of the Department of Cardiology, SSMC & MH, who fulfilled the inclusion and exclusion criteria, were taken as study sample. Hypertensive individuals were considered as group 1 and apparently healthy individuals who did not have hypertension were considered as group 2 for the study. Estimates of 24-hour sodium and potassium excretion were made from a single spot urine specimen and were used as surrogates for intake. We assessed the relationship between sodium and potassium excretion and blood pressure.*

Results: *The spot urinary sodium excretion was significantly higher in hypertensive group than in normotensive group (98.03 ± 71.13 mmol/L vs. 77.11 ± 41.71 mmol/L) (p=0.011). On the other hand, the spot urinary potassium excretion was lower in hypertensive group than normotensive group (33.95 ± 41.03 mmol/L vs. 39.43 ± 31.47 mmol/L), but the differences did not reach statistical significance (p=0.679). The estimated 24-hour urinary sodium excretion was higher in hypertensive group than in normotensive group (8.84 ± 3.57 gm vs. 7.67 ± 3.16 gm) and the difference was statistically significant (p = 0.015). Estimated 24-hour urinary potassium excretion was lower in hypertensive group (4.15 ± 1.37 gm) than in normotensive group (4.48 ± 1.56 gm), but the difference between 2 values did not reach statistical significance (p=0.114).*

Conclusion: *Compared to the normotensive counterparts, sodium intake is significantly higher and potassium intake is lower in hypertensive Bangladeshi subjects.*

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Introduction:

Hypertension affects 1 billion people and is a leading cause of death, stroke, myocardial infarction, congestive heart failure and chronic renal impairment.[1] It is also identified as a global disease burden and is ranked third as a cause of disability-adjusted life-years (DALYs).[2] According to World Health Organization (WHO) about 17 million deaths occur worldwide due to cardiovascular diseases (CVDs), of which hypertension alone accounts for 9.4 million deaths and 80% of CVD-related deaths occurred in the developing countries.[3] Essential hypertension comprises more than 90% of hypertension.[4]

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Although hypertension is more prevalent in developed countries like USA,[5] its prevalence is increasing in the low and middle-income countries.[6] Bangladesh is passing through epidemiological transition from communicable diseases to noncommunicable diseases (NCDs) and currently has a double burden of disease.[7] The prevalence of hypertension in Bangladesh is about 20-25% in adults.[8] Hypertension is a multifactorial disease. Sodium intake is reported to be a modifiable determinant of hypertension.[9] Most of the global population consumes between 3.0 and 6.0 g of sodium per day (7.5 to 15.0 g of salt per day).[10] In 2010, the estimated mean level of global sodium consumption was 3.95 g per day and regional mean levels ranged from 2.18 to 5.51 g per day.[11] Guidelines on CVD prevention recommend a maximum sodium intake of 1.5 to 2.4 g per day, but achieving this target will require a substantial change in diet for most people.[12] In addition to a primary increase in cardiac function propelled by overactive sympathetic nervous system, primary retention of salt and water by kidney contribute to hypertension.[4] Positive correlation exists between high sodium intake and increase in blood pressure.[13] Previous studies showed that blood pressure is directly related to sodium intake and inversely and independently related to potassium intake.[14] Higher sodium intake was associated with a higher risk of death and cardiovascular events than lower intake, and higher potassium excretion was associated with a lower risk of death and cardiovascular events.[15] Recent population-based studies have shown a positive association between salt excretion and blood pressure; however, some studies have negated this association.[16]

Also, daily intake of these electrolytes is subject to much geographical and ethnic variation. The impact of sodium and potassium intake on blood pressure in Bangladeshi subjects in incompletely known.

Methods:

This cross-sectional study was conducted in the Department of Cardiology, Sir Salimullah Medical College & Mitford Hospital (SSMC & MH), Dhaka, Bangladesh over a period of one year from April 2017 to March 2018, extended up to the March 2019. The study included 200 subjects out of which 100 were hypertensive with blood pressure ≥140/90 mm of Hg (group 1) while resting 100 were normotensive with blood pressure <140/90 mm of Hg (group 2). Group 1 patients were enrolled from the Outpatient Department of the Department of Cardiology, SSMC & MH, while group 2 subjects were the age- and sex-matched normotensive subjects accompanying the patients. For the study, convenient sampling was used. Patients with intake of non-steroidal anti-inflammatory agents, diuretics, steroids, hormonal contraceptives, acute and chronic kidney disease, congestive cardiac failure, malignant hypertension, chronic liver diseases, suspected secondary hypertension were excluded from the study. All statistical analysis was performed using the statistical package for social science (SPSS) program, version 22 for Windows.

After enrollment, meticulous history was taken and detailed clinical examination was done. The findings, including were recorded in pre-designed structured proforma. Current medications were interrogated and recorded. All participants were subjected to thorough physical examination which included pulse and at least 3 blood pressure measurements with same standard sphygmomanometer and Littman's stethoscope in sitting position in both arms. Laboratory investigations were done including fasting blood sugar, 2-hour post prandial blood sugar, serum creatinine, spot urinary electrolytes, urinary creatinine, and routine urine analysis. Estimation of 24-h urinary sodium and potassium excretion was done by using Tanaka method.[17] Spot and estimated 24-hour sodium and potassium excretion were obtained and compared between two groups by using appropriate statistical methods.

Results:

The results of the present study is depicted below:

Table-I

Distribution of study subjects by age (N=200).

Age (in years)	Group 1 (n=100) No. (%)	Group 2 (n=100) No. (%)	p value
<30	11 (11.0%)	13 (13.0%)	
31-50	43 (43.0%)	46 (46.0%)	
51-70	46 (46.0%)	41 (41.0%)	
Mean ± SD	49.11±12.2	49.73±12.6	0.724^{ns}

Here, *ns* means not significant. Data were expressed as mean ± SD. Unpaired Student's t-test was performed to compare between two groups. Figure in the parenthesis indicate percentage.

The table-I shows that, majority of the group 1 (hypertensive) and group 2 (normotensive) belonged to 31 to 70 years age group. No statistically significant difference was found between the groups (p>0.05).

Table-II

Distribution of study subjects by sex (N=200).

Sex	Group 1 (n=100) No. (%)	Group 2 (n=100) No. (%)	p value
Male	69 (69.0%)	71 (71.0%)	0.623^{ns}
Female	31 (31.0%)	29 (29%)	

Here, *ns* means not significant. Data were expressed as frequency and percentage. Chi-square test was performed to see the association between two groups.

Table-II shows that, above 2/3rd of the participants in both groups were male. However, no statistically significant differences exist between the groups (p> 0.05)

Table-III

Distribution of study subjects by risk factors (N=200).

Risk factors	Group 1 (n=100) No. (%)	Group 2 (n=100) No. (%)	p value
Diabetes mellitus	21 (21.0%)	24 (24.0%)	0.514^{ns}
Smoking	45 (45.0%)	42 (42.0%)	0.631^{ns}
Family history of IHD	5 (5.0%)	7 (7.0%)	0.320^{ns}

Here, *ns* means not significant. Data were expressed as frequency and percentage. Chi-square test was performed to see the association between two groups.

Table-III shows that, smoking was the commonest risk factor in both groups, while family history of IHD was the least common. However, no statistically significant differences were observed in respect of risk factor distribution between 2 groups (p>0.05).

Table-IV

Distribution of study subjects by body-mass index (BMI) (N=200).

BMI (Kg/m^2)	Group 1 (n=100) No. (%)	Group 2 (n=100) No. (%)	p value
Underweight (<18.5)	2 (2.0%)	4 (4.0%)	
Normal (18.5-24.9)	55 (55.0%)	61 (61.0%)	
Overweight (25.0-29.9)	34 (34.0%)	31 (31.0%)	
Obese (>30.0)	8 (8.0%)	4 (4.0%)	
Mean ± SD	24.4±4.92	23.8±3.11	0.314^{ns}

Here, *ns* means not significant. Data were expressed as mean ± SD. Unpaired Student's t-test was performed to compare between two groups.

Table-IV shows that, majority of the cases and controls had normal BMI. 42% of group 1 and 35% of group 2 had BMI above normal limit. However no statistically significant differences were observed in respect of distribution of BMI between 2 groups (p > 0.05).

Table-V

Distribution of mean blood pressure in two groups (N=200).

Blood pressure	Group 1 Mean±SD (n=100)	Group 2 Mean±SD (n=100)
SBP (mm Hg)	149.47±12.23	120.93±11.07
DBP (mm Hg)	92.63±13.87	76.60±7.35

SBP means systolic blood pressure, DBP means diastolic blood pressure. Data were expressed as mean ± SD.

Table-V shows that mean systolic blood pressure in group 1 and group 2 were 149.47±12.23 mm Hg and 120.93±11.07 mm Hg respectively while mean diastolic blood pressure were 92.63±13.87 and 76.60±7.35 mm Hg respectively.

Table-VI

Comparison of glycemic status and serum creatinine between two groups (N=200).

Variables	Group 1 (n=100) Mean±SD	Group 2 (n=100) Mean±SD	p value
FBS (mmol/L)	5.76±1.36	6.25±3.15	0.244^{ns}
2HPPBS (mmol/L)	7.91±2.83	8.41±3.68	0.532^{ns}
Serum creatinine (mg/dl)	1.02±0.23	1.07±0.24	0.629^{ns}

Here, *ns* means not significant. FBS means Fasting blood glucose. 2HPPBS means 2-hour post prandial blood sugar. Data were expressed as mean ± SD. Unpaired Student's t-test was performed to compare between two groups.

Table-VI shows that, fasting blood sugar, 2 hours post-prandial blood sugar and serum creatinine had higher values in group 2 in comparison to group 1. However, the differences were not statistically significant (p > 0.05).

Table-VII

Distribution of study subjects by spot urinary sodium and spot urinary potassium (N=200).

Variables	Group 1 (n=100) Mean±SD	Group 2 (n=100) Mean±SD	p value
Spot urinary sodium (mmol/L)	98.03±71.13	77.11±41.71	0.011*
Spot urinary potassium (mmol/L)	33.95±53.03	39.43±31.47	0.375 *ns*

Here * means significant. *ns* means not significant. Data were expressed as mean ± SD. Unpaired Student's t-test was performed to compare between two groups,

Table-VII shows that mean spot urinary sodium excretion was significantly higher in group 1 than in group 2 (p < 0.05). On the other hand, the mean spot urinary potassium is lower in group 1 in comparison to the group 2. However the differences were not statistically significant (p > 0.05).

Table-VIII

Comparison of estimated 24-hour urinary sodium and estimated 24-hour urinary potassium between two groups (N=200).

Variables	Group 1 (n=100) Mean±SD	Group 2 (n=100) Mean±SD	Total (n=1) Mean±SD	p value
Estimated Na24h (gm/24-hour)	8.84±3.57	7.67±3.16	8.25±3.37	0.015*
Estimated K24h (gm/24-hour)	4.15±1.37	4.48±1.56	4.32±1.47	0.114 *ns*

Here * means significant. *ns* means not significant. Na24h means 24-hour urinary sodium. K24h means 24-hour urinary potassium. Data were expressed as mean ± SD. Unpaired Student's t-test was performed to compare between two groups.

Table-VIII shows that mean estimated 24-hour urinary sodium excretion was significantly higher in group 1 than in group 2 (p <0.05). On the other hand, mean estimated 24-hour urinary potassium excretion was lower in group 1 than in group 2, but the differences were not statistically significant (p >0.05).

Discussion:

The baseline characteristics were similar between the 2 groups. In the present study, almost 2/3rds of the subjects were male in both hypertensive and control groups (69% and 71% respectively). It may be due to the fact that in our social background, males tend to seek medical attention more than the females. Smoking was the commonest risk factor in both groups (45% and 42% respectively). Similar male predominance was also seen in the study done by Mente et al.[15] Majority of the cases (55%) and controls (61%) had normal BMI. This finding mimics that of the study done in China, where 57.4% participants had normal BMI.[18]

In the present study, the spot urinary sodium excretion was significantly higher in hypertensive group than in normotensive group (98.03 ± 71.13 mmol/L vs. 77.11 ± 41.71 mmol/L) (p=0.011). On the other hand, spot urinary potassium excretion was lower in hypertensive group than normotensive group (33.95 ± 53.03 mmol/L vs. 39.43 ± 31.47 mmol/L), but the differences did not reach statistical significance (p=0.114). The spot urinary sodium and potassium excretion was used to estimate 24-hour urinary sodium and potassium excretion. Estimated 24-hour urinary sodium and potassium excretion are the indicators of daily intake of sodium and potassium respectively. In this study estimated 24-hour urinary sodium excretion was higher in hypertensive group than in normotensive group (8.84 ± 3.57 gm vs. 7.67 ± 3.16 gm) and the difference was statistically significant (p = 0.015). A multicenter study, involving 18 countries of 5 continents showed that high urinary sodium excretion is associated with high blood pressure.[15] Urinary sodium excretion in the hypertensive participants were higher than normotensive group in a study done in China (11.62 ± 3.01gm/day vs. 10.52 ± 2.42gm/day).[18] Earlier study also had similar finding that high 24-hour urinary sodium excretion was associated with high blood pressure.[19]

In this study, the mean estimated 24-hour urinary sodium excretion including both hypertensive and normotensive subjects was 8.25±3.37 gm/day. The amount was lower than that of the previous study

done in Bangladesh which was 21 gm/day.[20] Lower values of 24-hour urinary sodium excretion may be due to increasing awareness among people about harmful effects of high salt intake. Still the amount is high may be because of the habit of overall excess dietary salt intake in different ways like use of extra salt in cooking, consumption of salty food and adding raw salt in meal. In the multicenter study by Mente et al.[15] the mean urinary sodium excretion was 4.93±1.73 g/day which is much lower than the present study. This lower value of 24-hour urinary sodium excretion may be due to overall low dietary intake of sodium in other parts of the world. On the other hand, the amount of 24-hour urinary sodium excretion was almost similar to study done in Kashmir which was 7.6 gm/day.[19] It may be because of the presumed similarity in dietary habits of 2 regions of South Asia. Kashmiri people also have the peculiar habit of excessive salt intake in the form of salted tea.[21] The mean 24-hour urinary sodium excretion was 11.62 ± 3.01 gm in Chinese people which is higher than that in the present study.[18] Estimated 24-hour urinary sodium excretion also represents the daily sodium intake which is 8.25±3.37 gm/day in this study. But the amount is much more than that in WHO guidelines and Dietary Guidelines for Americans, 2020-2025 recommendation for sodium intake which is <2 gm/day and <2.3gm/day, respectively in adults.[22,23]

Estimated 24-hour urinary potassium excretion was lower in hypertensive group (4.15 ± 1.37 gm) than in normotensive group (4.48 ± 1.56 gm), but the difference between 2 values did not reach statistical significance (p=0.114). Jan et al.[19] did not find any association between 24-hour urinary potassium excretion and blood pressure, though Mente et al.[15] reported that potassium excretion was inversely associated with blood pressure (p<0.001).

Estimated 24-hour urinary potassium excretion also represents the daily potassium intake which was 4.32 ± 1.46 gm/day in the present study. In the study by Mente et al.[15] the mean urinary potassium excretion was 2.12 ± 0.60 gm/day which is much lower than the present study. In the present study, the overall urinary potassium excretion was actually lower than the recommendation for potassium intake by Dietary guidelines for Americans 2005 which is >4.7 gm/day.[24] This finding may in part be explained by the low dietary intake of potassium rich foods e.g., fruits. In the Bangladesh NCD Risk Factor Survey 2010, consumption of inadequate fruit and/or vegetables (<5 servings/day) was found in 95.7% people (average 1.8 days/week).[25]

The study has got some limitations. The sample size is small. Convenient sampling method was applied in the study. Also this was a single-centre study, compromising generalizability. Despite these limitations, the study may stimulate future research in this field in Bangladesh.

Conclusion:

Mean 24-hour urinary sodium excretion is significantly higher in adult hypertensive subjects than that in adult normotensive subjects. Mean 24-hour urinary potassium excretion is lower in hypertensive than that in normotensive adult; however, the difference is not statistically significant.

Conflict of Interest - None.

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