Sodium and Potassium Excretion in Normotensive and Hypertensive Population in Kashmir

RA Jan*, S Shah*, SM Saleem**, A Waheed***, S Mufti+, MA Lone++, M Ashraf*

Abstract

One hundred thirty five hypertensive patients and equal number of age and sex matched healthy controls were taken up for studying the relationship of 24 hour urinary sodium and potassium excretion, sodium-potassium molar ratio and body mass index (BMI) with blood pressure in normotensive and hypertensive population in Kashmir. There was statistically significant elevated 24 hour urinary sodium excretion (p < .001), increased Na⁺ - K⁺ molar ratio, significantly higher BMI in hypertensive population as compared to controls whereas there was a lower 24 hour urinary excretion of potassium (p > .20) in patients with hypertension. Thus sodium and potassium excretion, Na⁺ - K⁺ molar ratio and body mass index has direct bearing in perpetuation or causation of hypertension in Kashmir which may be related to intake of salt tea.

INTRODUCTION

Hypertension is one of the leading causes of death and disability among adults all over the world. It remains the major risk factor for coronary, cerebral and peripheral vascular disease. Essential hypertension comprises more than 90% of hypertension.

In addition to a primary increase in cardiac function propelled by overactive sympathetic nervous system, primary retention of salt and water by kidney, other factors contributing to hypertension are hereditary predisposition and high sodium and low potassium intake and excretion. Positive correlation exists with high sodium intake and increase in blood pressure.

Recent population based studies have shown a positive association between salt excretion and blood pressure, however some studies have negated this association. A high potassium diet has been claimed to give protection to subjects on high sodium diet, but in most population studies no such association has been found. A positive correlation has also been found between urinary sodium/potassium molar ratio and blood pressure by some studies but denied by others. Decrease in urinary Na/K⁺ molar ratio was strong predictor of lowering blood pressure, so decreased Na intake and increased potassium intake or both together may be effective in prevention or even treatment of hypertension.

In large Intersalt study Potassium intake as judged by 24 hour urinary potassium excretion was found to be important independent determinant of population blood pressure. Blood pressure was directly related to sodium intake and inversely and independently related to potassium intake. Recently additive effect of increasing potassium and reducing sodium intake in controlling blood pressure has been found.

Prevalence of hypertension in Kashmir is 20% and this population has a peculiar habit of high salt intake in the form of salt tea.

Present study was aimed at studying relationship of sodium and potassium excretion, sodium potassium molar ratio and body mass index with blood pressure in normotensive and hypertensive people in Kashmir.

MATERIAL AND METHODS

Study was conducted in Sher-i-Kashmir Institute of Medical Sciences which is the premier institute of the state. One hundred and thirty five patients as per criteria of JNC VI report and equal number of normal age and sex matched controls were taken up for study.

Study group (Hypertensive group)

Inclusion criteria

Hypertension as per criteria of JNC VI report i.e. blood pressure 140/90 mmHg least at three different occasions after refraining from antihypertensives and diuretics for at least 3 weeks before the study and refraining from eating, smoking or indulging in any stressful activity 30 minutes before recordings.

Exclusion criteria

1. Patients of secondary hypertension.

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2. Patients on non-steroidal antiinflammatory agents, antihypertensives, diuretics.
3. Patients with congestive cardiac failure.
4. Patients with malignant hypertension.
5. Females on oral contraceptive medication.

Control group
1. Same age and sex
2. BP lower than the specified for hypertensive group
   Both the groups maintained their normal salt intake.

Values obtained in the control group were compared with hypertensive group.

A detailed history of all subjects especially family history of hypertension, cardiovascular disease, renal disease, diabetes mellitus, duration of hypertension, levels of elevated blood pressure, results and side effects of antihypertensive therapy, history of weight gain, sodium intake, alcohol use and symptoms suggestive of secondary hypertension. History of other cardiovascular risk factors (including obesity, smoking, hyperlipidemia, diabetes) and all prescribed and over the counter medications.

All subjects were subjected to thorough physical examination which included pulse and at least three blood pressure measurements with same standard mercury sphygmomanometer and Littman’s stethoscope in both supine and standing in both the arms, examination of extremities for oedema, diminished and absent peripheral arterial pulsations, measurement of body mass index (BMI) calculated by weight/height$^2$ (kg/m$^2$), optic fundoscopic examination for any hypertensive changes, neck for distended veins, thyromegaly, cardiovascular, respiratory, abdominal and CNS examination. Various investigations done in all subjects were:

1. CBC
2. Routine urine analysis
3. Serum urea, creatinine, sugar, cholesterol, calcium, phosphorous, uric acid on Hitachi 704 analyser with commercially available kits from Boehringer Kinetic Ltd. Germany.
4. Serum sodium and potassium levels.
5. 24 hr urinary excretion of protein, creatinine, sodium and potassium and the Na$^+/K^+$ molar ratio was calculated.
6. Chest skiagram
7. Electrocardiogram

Electrolyte estimation in both serum and urine were done on sodium potassium analyser KNa2 (Radiometer). Before analysing urine sample because of wide variation of pH, ionic strength and electrolyte concentration samples were diluted with pH and ionic strength solution in the ratio of one part urine to two parts diluent.

24 hr urinary creatinine was measured by Jaffes Method on Hitachi 704 Multichannel analyser to R/o any error in collection of samples.

All values were obtained from Todd-Sanford for adults.

Normal values

<table>
<thead>
<tr>
<th>Serum (mmol/dl)</th>
<th>Urine mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium Na+</td>
<td>136-142</td>
</tr>
<tr>
<td>Potassium K+</td>
<td>4.0 - 5.6</td>
</tr>
</tbody>
</table>

OBSERVATIONS/RESULTS

The age of hypertensive group ranged from 20-65 years with (mean 40.02 ± 9.99 yrs), whereas in control group in ranged from 20-60 years with (mean 38.69 ± 8.82 yrs). 62 (45.93%) were males and 73 (54.07%) females in hypertensive group and 53 (51.96%) were males and 49 (48.04%) females in control group.

Serum sodium, potassium, calcium, phosphorous, cholesterol levels were significantly higher (p < .001) in hypertensives as compared to controls, whereas there was no statistically significant difference in serum urea, creatinine, glucose and uric acid levels between two groups (p > .05) (Table 1).

The average 24 hr urinary sodium excretion in hypertensive group was higher as compared to controls which was statistically significant (p < .001). There was no statistically significant different in average 24 hr urinary potassium excretion between two groups (p > 0.20). Mean Na$^+/K^+$ molar ratio was higher in hypertensive group as compared to controls (Table 2).

Both in hypertensives and controls average 24 hr sodium excretion was significantly high in subjects with high BMI as compared to those with normal BMI (p < 0.005 and p < 0.025 respectively). Whereas there was no statistically significant different (p > 0.50) in average 24 hr potassium excretion in both groups between subjects with normal and high body mass index (Table 3).

The average 24 hr Na$^+$ and K$^+$ excretion was higher in hypertensives with duration of hypertension < 1 yr as compared to those with duration of hypertension > 1 yr which was statistically significant (p < 0.05 and < 0.001 respectively). Molar ratio was lower in patients with duration of hypertension < 1 yr as compared to those with > 1 yr duration.

Mean 24 hr sodium excretion was higher in subjects with severe hypertension in comparison to patients with mild to moderate hypertension which was statistically significant (p < 0.05). Whereas the average 24 hr K$^+$ excretion was higher among severe hypertensive patients in comparison to mild to moderate hypertensive group which was statistically insignificant (p > 0.50). Molar ratio was higher in severe hypertension as compared to mild to moderate hypertension (i.e. 8.83 : 1 v/s 7.64 : 1 respectively).
**Table 1**: Comparison of serum chemistry between hypertensives and normals

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Hypertensives Mean ± SD</th>
<th>Controls Mean ± SD</th>
<th>t</th>
<th>p</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na+</td>
<td>140.4 ± 2.90</td>
<td>138.5 ± 1.12</td>
<td>7.04</td>
<td>&lt; .001</td>
<td>HS</td>
</tr>
<tr>
<td>K</td>
<td>3.97 ± 0.45</td>
<td>3.89 ± 0.18</td>
<td>2.00</td>
<td>&lt; .05</td>
<td>S</td>
</tr>
<tr>
<td>Ca</td>
<td>9.4 ± 0.42</td>
<td>8.9 ± 0.33</td>
<td>10.00</td>
<td>&lt; .001</td>
<td>HS</td>
</tr>
<tr>
<td>Phos</td>
<td>3.46 ± 0.29</td>
<td>3.30 ± 0.25</td>
<td>4.00</td>
<td>&lt; .001</td>
<td>HS</td>
</tr>
<tr>
<td>Chol</td>
<td>195 ± 6.53</td>
<td>29.82 ± 5.28</td>
<td>0.97</td>
<td>&gt; .20</td>
<td>NS</td>
</tr>
<tr>
<td>Creat</td>
<td>0.98 ± 0.29</td>
<td>0.97 ± 0.37</td>
<td>0.25</td>
<td>&gt; .50</td>
<td>NS</td>
</tr>
<tr>
<td>Urea</td>
<td>98.57 ± 9.88</td>
<td>96.32 ± 8.97</td>
<td>1.84</td>
<td>&gt; .05</td>
<td>NS</td>
</tr>
<tr>
<td>Glucose</td>
<td>5.52 ± 1.11</td>
<td>5.68 ± 1.16</td>
<td>1.07</td>
<td>&gt; .20</td>
<td>NS</td>
</tr>
</tbody>
</table>

S = Significant; HS = Highly significant

**Table 2**: Comparison of 24 hrs urinary Na, K and their molar ratio between hypertensives and normals

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Hypertensives Mean ± SD</th>
<th>Normals Mean ± SD</th>
<th>t</th>
<th>p</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>424 ± 150.50</td>
<td>337 ± 121.50</td>
<td>4.92</td>
<td>&lt; .001</td>
<td>HS</td>
</tr>
<tr>
<td>K</td>
<td>53.4 ± 38.86</td>
<td>48.6 ± 30.10</td>
<td>1.07</td>
<td>&gt; .20</td>
<td>NS</td>
</tr>
<tr>
<td>Mean</td>
<td>7.9:1</td>
<td>6.9:1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

S = Significant; HS = Highly significant

**Table 3**: Relationship of body mass index with 24 hr urinary Na+ and K+ excretion and their molar ratios among hypertensives and normals

<table>
<thead>
<tr>
<th>Group</th>
<th>Body mass index</th>
<th>t</th>
<th>p</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Sodium (Na+)</td>
<td>392.85 ± 188.02</td>
<td>2.83</td>
<td>&lt; .005</td>
</tr>
<tr>
<td></td>
<td>Potassium (K+)</td>
<td>52.59 ± 26.22</td>
<td>.49</td>
<td>&gt; .50</td>
</tr>
<tr>
<td></td>
<td>Molar ratio</td>
<td>7.47:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>Sodium (Na+)</td>
<td>497.98 ± 201.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Potassium (K+)</td>
<td>55.32 ± 30.85</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Molar ratio</td>
<td>9.001:1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

HS = Highly significant; S = Significant; NS = Not significant

**Table 4**: Relationship of duration of hypertension with 24 hours urinary Na+, K+ and their molar ratio

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Duration of hypertension</th>
<th>t</th>
<th>p</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 yr</td>
<td>Sodium (Na+)</td>
<td>463.12 ± 182.57</td>
<td>2.09</td>
<td>&lt; .05</td>
</tr>
<tr>
<td>&gt; 1 yr</td>
<td>Potassium (K+)</td>
<td>49.12 ± 22.18</td>
<td>.44</td>
<td>&gt; .50</td>
</tr>
<tr>
<td></td>
<td>Molar ratio</td>
<td>6.36:1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

S = Significant; HS = Highly significant

**DISCUSSION**

Essential hypertension comprises 90% of all hypertensive patients. Various factors have been responsible for perpetuation or causation of hypertension. In addition to hereditary predisposition, high sodium intake and lower potassium intake and excretion may also contribute to the development of hypertension which has been proved. Excretion of sodium and potassium depend on their intake. Positive correlation between salt excretion and blood pressure has been proved by some and disproved by other recent studies.
In the present study there were more females in hypertensive group whereas more males in the control group probably because more males volunteered for control group. Sr. calcium levels were significantly higher (p < .001) which could have contributed to hypertension in these patients because of its vasoconstrictor effect as hypertension disappears when hypercalcemia is treated as in hyperparathyroidism. Although some epidemiological studies suggest that high calcium lowers blood pressure as is effect of calcium channel blockers. Phosphorus levels were significantly higher (p < .001) in hypertensive group, significance of which is not clear and needs further studies. Although it could be related to high calcium levels in our patients. Sr. Cholesterol beyond doubt has been proved independent risk factor for hypertension which has been proved by our study as well.

Average 24 hr urinary sodium excretion was significantly (p < .001) higher in hypertensive group although high in both groups probably because of very high salt intake (average 27 g/day) consistent with a studies done in Japan (average 11-27 g/day). Excessive 24 hr urinary sodium excretion has been seen in other studies as well. Statistically significant increased 24 hr excretion of sodium in hypertensives in our study was possibly responsible in causation of hypertension which was in agreement to various other studies. Sensitivity to hypertensive effect of sodium may be genetically determined which may be also operative in our cases as well. It is also supported by fact that hypertension decreased by low salt intake and hypertension is absent or rare in population with low salt intake as is the antihypertensive effect of thiazides due to loss of sodium and extra cellular fluid. In both hypertensives and normotensives, average 24 hour urinary excretion of potassium was lower because of low intake of potassium in this part of world which might also contribute to hypertension as has also been seen by Menealy and Belarbee in 1976. Low potassium intake leading to increase blood pressure and vice versa is confirmed by various other studies independent of sodium excretion. Recently there was evidence of additive effect of high sodium intake and low potassium intake in causation of hypertension which may be operative in our hypertensive group as well. Na+/K+ molar ratio was higher in hypertensive group as compared to controls which is consistent with various other studies. It is because of excessive excretion of sodium in hypertensive group as compared to controls.

Our study observed significant impact of BMI on 24 hr Na+ excretion in both groups with increased sodium excretion with high BMI while there was no association of BMI with K+ excretion in either group. Na+/K+ molar ratios were also higher in people with increased BMI in both groups. Intersalt study has shown significant and independent relationship of BMI with hypertension in individual subjects. Relationship of BMI with Na+ output and hypertension as observed in our study has been proved by Sampson et al in 1978. BMI showed significant influence on Na+ excretion in hypertension patients in our study so while estimation BMI should always be considered. Similar observations has been made in entire population by Preteman 1979. 24 hr Na+ and K+ excretion increased upto 40 yrs of age beyond which it was only K+ which increased further with molar ratios of 6.52:1 were seen upto 40 yrs as compared to 10.85 : 1 beyond 40 yrs. These observations explain that age modifies a number of factors i.e., GFR, renal hemodynamics and responsiveness of renin angiotensin aldosterone system confirmed by Norma K Hollenberg as well. Age influences the capacity of kidneys to conserve sodium, so age related changes must be considered while estimations.

Both Na+ and K+ excretions were significantly elevated in hypertensive males with molar ratio of 7.12:1 and 8.9:1 among females while it was only potassium elevated in normotensives with molar ratio of 6.27:1 among males and 7.87:1 among females. These observations are in contrast to observations from Tuo Milehto et al 1980. Simpson et al in 1978 found that mean 24 hour sodium excretion at all ages was significantly higher in males as compared to females which goes nearer to our observations. We also observed that 24 hr Na+/K+ excretion was significantly elevated among those whose hypertension is of less than 1 yr duration with molar ratio of 8.85 : 1. Possibly because of initially high sodium intake might contribute to raised blood pressure which is subsequently sustained by other irreversible factors on long term basis. Needs further studies as no such studies are available in the literature.

Thus we conclude that

1. Markedly increased 24 hour sodium excretion and low potassium excretion in hypertensive group indicates a very high intake of sodium and low intake of dietary potassium both playing significant individual as well as additive role in causation or perpetuation of hypertension.
2. Age, sex and body mass index, duration of hypertension has direct bearing on the sodium excretion and blood pressure.
3. Serum Na+, calcium, phosphorus and cholesterol levels were higher in hypertensive group and can be additional factors responsible for causation or perpetuation of blood pressure.

REFERENCES


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