Research Article

A Study on Serum Sodium and Potassium Levels in Newly Diagnosed Primary Hypertension

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Abstract: Primary hypertension comprises of more than 90% of hypertension. Many studies have shown that a positive correlation between serum sodium and blood pressure and a negative correlation between blood pressure and serum potassium. The objective is to study the serum levels of sodium and potassium and correlate them with blood pressure. The study was conducted in medical wards at Mamata general hospital during October 2012 – October 2013 and sample of 100 (50 cases +50 controls) patients with primary hypertension and age more than 20 years were included and investigations like serum electrolytes, fasting blood sugars were sent. Serum sodium was higher and serum potassium was lower in hypertensive group respectively than in the control group and mean and standard deviation were 146.9±4.58 and 3.77± 0.36 respectively in hypertensive group. Serum sodium was significantly more among hypertensive group and correlated positively with blood pressure unlike serum potassium which was lower and correlated negatively with blood pressure.

Keywords: Primary hypertension, Serum sodium, Serum potassium, BMI.

INTRODUCTION
Hypertension is one of the most common worldwide diseases afflicting humans & 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. Primary hypertension comprises more than 90% of hypertension [1]. The extent and tempo of investigation of the elevated pressure are determined by the clinical situation. Hypertension also presents as an incidental finding in other clinical situations and as a result is often sub optimally managed or even ignored. Even though substantial progress has been made in the awareness, treatment, and prevention of CVD in the last decade, hypertension is often underestimated and undiagnosed [2].

Hypertension is an emerging health problem in India. When majority of people come to know that they have hypertension they have already advanced into a stage with target organ damage a fatal stroke or myocardial infarction or irreversible renal failure [3]. Our distant ancestors consumed a low-sodium, high-potassium diet [4] and accordingly your kidneys are adapted to conserve sodium and excrete potassium [5].

In a country like India, people will have a diet rich in sodium and poor in potassium. We have known for over 2000 years that an acute high dietary sodium intake in the form of a salty [6] meal, results in a temporary increase in blood pressure and is associated with several other important diseases [7]. In developed countries, contemporary diets are high in sodium, primarily resulting from the salt added to manufactured foods and low in good sources of potassium such as vegetables and fruit.

Many studies have shown that a positive correlation exists between serum sodium and blood pressure and a negative correlation exists between serum potassium and blood pressure. They have shown that a decreased intake of sodium and increased potassium intake or both together may be effective in prevention or even treatment of hypertension. Independent reports on serum sodium and potassium among Indian hypertensive population were lacking and hence the present study was conducted.

MATERIALS AND METHODS
Period of the Study: One year

Sample size: 100 (50 cases + 50 controls)

Setting: The work is carried out in the medical wards of Mamata General Hospital, Khammam.
Ethical committee approval: The present study was approved by ethical committee.

Inclusion Criteria
- Patients with primary hypertension
- Patients above 20 years
- Both males and females

Exclusion Criteria
- Patients below 20 years
- Patients with renal failure
- Pregnancy
- Patients with secondary hypertension
- Patients on non-steroidal anti-inflammatory agents, antihypertensive agents
- Females on oral contraceptive medication
- Patients with diabetes mellitus
- Patients with acute diarrheal diseases

Investigations
- Serum sodium and potassium estimation
- Fasting blood sugar
- Urine albumin, sugar, microscopy
- A twelve lead electrocardiogram
- Chest X-ray
- Patient’s height and weight
- The body mass index
- Blood urea & serum creatinine
- Fundoscopy

Consent
The study group thus identified by the above criteria (inclusion and exclusion criteria) was first instructed about the nature of the study. Willing participants were taken up after getting a written informed consent from them.

Study Subjects and Controls
Fifty newly diagnosed primary hypertensive patients attending the medicine OPD or admitted to the medical wards of Mamata General Hospital for the period of one year from October 2012 to October 2013 formed the study group. Fifty healthy people were kept as controls. This control group comprised of normotensive individuals who were attendants of patients with primary hypertension living in the same environment other than their own siblings.

Details of the Study Subjects
All the patients were subjected to detailed history taking, careful physical examination and biochemical analysis to exclude secondary hypertension. Patient’s height and weight were measured. The body mass index was calculated using the formula weight / height [2].

All the peripheral pulses were checked with special attention to carotid and the femoral to detect evidence for early atherosclerosis. An ocular fundus examination was done to detect hypertensive retinopathy.

Patients were informed to refrain from smoking or drinking tea or coffee for at least thirty minutes before measuring blood pressure. Then blood pressure was measured using the following guidelines.

Guidelines for Measuring Blood Pressure
I. Conditions for the Patient
A. Posture
Sitting postures are usually adequate for routine follow-up. Patient should sit quietly with back supported for five minutes and arm supported at the level of heart.2. For patients who are over 65 at first visit measure BP at 1 and 3 min after assumption of the standing position in which orthostatic hypotension may be frequent or suspected.

B. Circumstances
- No caffeine for preceding hour
- No smoking for preceding 15 minutes.
- No exogenous adrenergic stimulants like phenylephrine in nasal decongestants or eye drops for papillary dilation
- A quite, warm setting
- Home readings taken under various circumstances and 24 hour ambulatory recordings may be preferable

II. Equipment
A. Cuff Size
The bladder should encircle and cover 2/3rds of the arm length. A standard bladder (12–13 cm wide and 35 cm long) is used, but have a larger and a smaller bladder available for large (arm circumference >32 cm) and thin arms, respectively. If not, place the bladder over the brachial artery; if bladder is small spuriously high readings may result.

B. Manometer
Aneroid gauges should be calibrated every six months against the mercury manometer.

III. Technique
A. Number of readings
- On each occasion, take at least two readings, separated by as much time as practical. If readings vary by more than 5 mm Hg, take additional readings until two are close.
- For diagnosis, obtain at least three sets of readings a week apart.
- Initially, take pressure in both arms, if pressure differs, use arm with higher pressure.
- If arm pressure is elevated, take pressure in one leg, particularly in patients below age
B. Performance

- Inflate the bladder quickly to a pressure 20 mm Hg above the systolic, as recognized by the disappearance of the radial pulse.
- Deflate the bladder 3 mm Hg every second.
- Use phase I and V (disappearance) Korotkoff sounds to identify systolic and diastolic, respectively.
- If Korotkoff sounds are weak, have the patients raise the arm, open and close the hand 5 to 10 times, after which the bladder should be inflated quickly.

C. Recording

Note the pressure, patient position, the arm, cuff size (e.g., 140/90, seated, right arm, large adult cuff).

Urine albumin, sugar, microscopy and pH were done for all the subjects. A twelve lead electrocardiogram and chest X-ray were also taken. Overnight (12 hour) fasting blood sugar and blood urea were estimated.

Serum creatinine was estimated using Modified Jaffe’s kinetic method. Serum sodium and potassium was estimated using Flame emission photometric method.

Definitions Used in the Present Study

Primary Hypertension

Hypertension was defined in accordance to the JNC-VII report as systolic blood pressure 140 mm of Hg and above and or diastolic blood pressure 90 mm of Hg and above. The diagnosis that the hypertension is essential and not secondary was made on the overall clinical impression only. Laboratory investigations to rule out secondary causes were not done in each case.

Sodium and Potassium Normal Values

The normal range for serum sodium was from 135 to 150 mmol / L. The normal range for serum potassium was from 3.5 to 5 mmol / L.

Obesity

According to the proposed classification of weight by BMI in adult Asians, the patients with a BMI ≤ 18.5 were classified as underweight, 18.5 – 22.9 were classified as normal, ≥ 23 were classified as overweight and ≥ 25 were classified as obese.

Diabetes Mellitus

Patients with fasting plasma glucose ≥ 126 mg / dl or two hour plasma glucose ≥ 200 mg / dl or with symptoms of diabetes plus random blood glucose ≥ 200 mg / dl were considered to be diabetic.

Left ventricular hypertrophy

Based on the electrocardiographic findings, which satisfy either Sokolow-Lyton criteria or Cornell voltage criteria [8, 9]

Statistical Analysis:

The collected data was entered in Microsoft excel spread sheet and analyzed statistically. Student’s t values were applied for significance. Significance was considered if the ‘p’ value was below 0.05.

RESULTS AND DISCUSSION

Analysis of cases and controls with respect to Body Mass Index (BMI)

36% of cases were obese while in the control group obesity was noticed in 2%. Details are given in following table.

Table 1: Distribution of cases and controls with respect to Body Mass Index (BMI)

<table>
<thead>
<tr>
<th>BMI</th>
<th>Cases</th>
<th></th>
<th>Controls</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Underweight &lt;18.5</td>
<td>5</td>
<td>10</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Normal weight 18.5-22.9</td>
<td>16</td>
<td>32</td>
<td>34</td>
<td>68</td>
</tr>
<tr>
<td>Overweight 23-24.9</td>
<td>11</td>
<td>22</td>
<td>9</td>
<td>18</td>
</tr>
<tr>
<td>Obese ≥25</td>
<td>18</td>
<td>36</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>100</td>
<td>50</td>
<td>100</td>
</tr>
</tbody>
</table>

Distribution of BMI between cases and controls

The mean body mass index in the case group is 23.73 ± 3.28 and in the control group is 21.36 ± 2.12. This shows that the difference in Body Mass Index between cases and controls was statistically significant.

Table 2: Distribution of BMI between cases and controls

<table>
<thead>
<tr>
<th>BMI</th>
<th>Cases</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>23.73</td>
<td>21.36</td>
</tr>
<tr>
<td>S.D</td>
<td>3.28</td>
<td>2.12</td>
</tr>
</tbody>
</table>

'p' value = 0.00004
Distribution of BMI with respect to grade of hypertension

No significant difference was found for BMI between grade I and grade II hypertension case

Table 3: Distribution of BMI with respect to grade of hypertension

<table>
<thead>
<tr>
<th>BMI</th>
<th>Grade I Hypertension</th>
<th>Grade II Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;18.5</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>18.5-22.9</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>23-24.9</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>&gt;25</td>
<td>4</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>34</td>
</tr>
</tbody>
</table>

'p' value = 0.06

Distribution of cases with respect to grade of hypertension

16 cases were of grade I hypertension and 34 cases were of grade II hypertension.

Table 4: Distribution of cases with respect to grade of hypertension

<table>
<thead>
<tr>
<th>Hypertension</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1</td>
<td>16</td>
<td>32</td>
</tr>
<tr>
<td>Grade 2</td>
<td>34</td>
<td>68</td>
</tr>
</tbody>
</table>

Serum sodium levels in grade II hypertension in males was 143.05 ± 6.53 and in females was 141.87 ± 6.85. Though sodium values are high in males compared to females but it is not statistically significant ('p' value = 0.61).

Table 5: Serum sodium with respect to gender in grade I and grade II hypertension

| Grade of Hypertension | Males | | | Females | | |
|-----------------------|-------|---|---|---------|---|
|                       | Mean  | S.D. | Mean | S.D. | |
| Grade I               | 141.36| 5.00 | 144.6 | 7.76 | |
| Grade II              | 143.05| 6.53 | 141.87| 6.85 | |

Serum potassium levels in grade II hypertension in males was 3.78 ± 0.37 and in females was 3.81 ± 0.41. Not statistically significant ('p' value = 0.82).

Table 6: Serum potassium with respect to gender in grade I and grade II hypertension

| Grade of Hypertension | Males | | | Females | | |
|-----------------------|-------|---|---|---------|---|
|                       | Mean  | S.D. | Mean | S.D. | |
| Grade I               | 141.36| 5.00 | 144.6 | 7.76 | |
| Grade II              | 143.05| 6.53 | 141.87| 6.85 | |

Serum sodium in grade I hypertension is 142.4±5.93 and in grade II hypertension is 142.5±6.61. It is not statistically significant ('p' value = 0.94). Mean serum potassium in grade I hypertension is 3.72±0.32 and in grade II hypertension is 3.80±0.38 and is not statistically significant ('p' value = 0.49).

Table 7: Serum sodium and potassium levels in grade I and grade II hypertension

<table>
<thead>
<tr>
<th>Grade of Hypertension</th>
<th>Serum Sodium</th>
<th>Serum Potassium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>142.4</td>
<td>3.72</td>
</tr>
<tr>
<td>Grade II</td>
<td>142.5</td>
<td>3.80</td>
</tr>
</tbody>
</table>
DISCUSSION

Hypertension is one of the leading causes of death and disability among adults all over the world. Hypertension the most common form of cardiovascular disease is present in nearly 25% of adults and increases in prevalence with age. It remains the major risk factor for coronary, cerebral and peripheral vascular disease. Primary hypertension comprises more than 90% of hypertension [1].

Patients were studied on the basis of clinical parameters and simple biochemical investigations. Serum sodium and potassium was done for all the patients.

Serum sodium among Hypertensives

In our part of the country, there is excessive intake of dietary salt. But in spite of that not everyone has primary hypertension. The rarity of hypertension among those consuming large amount of salt may probably be related to chronic adaptation of body system towards renal clearance of sodium. However this aspect of chronic adaptation of sodium handling by kidneys requires further molecular studies. So in addition to the hereditary predisposition and high sodium intake and lower potassium intake, the renal handling of these cations also play an important role in the pathogenesis of primary hypertension [10-12].

Salt intake was more in the tropical countries by and large in order to overcome sodium loss through sweating. In modern days the consumption of salt is more than earlier days in view of various food preparations or a combination of them, as man is tuned more to taste of the food. Combination of food materials requires additional salt. As a result, people consume more than actually required (2 vs. 8-10 g / day / person). Such an amount of salt consumption contributes for the development of hypertension in a genetically susceptible population.

In our study the mean serum sodium was estimated in the control and study groups.

Results were compared with other studies.

Serum sodium was higher in the hypertensive group than the control group even though both were within the normal range. The mean and standard deviation of serum sodium among cases was 146.96±4.58 while in control group it was 139.7±4.30 respectively. Our study was supported by Jan et al., Srinagar, Kashmir. In his study, one hundred thirty five hypertensive patients and equal number of age and sex matched healthy controls were taken for the study. Serum sodium in the hypertensive group was140 ± 2.90 while in the control group it was found to be 138.5 ± 1.12. Serum sodium was higher in the hypertensive group than the control group and considered to be a factor responsible for the causation or perpetuation of blood pressure [13].

In the Dietary Approaches to Stop Hypertension (DASH) trial, 412 patients with mild-moderate hypertension (range 120–159/80–95 mm Hg) were randomized to the DASH diet (which is rich in vegetables, fruits, and low-fat dairy products) or control diet [14]. Each group received increasing levels of dietary sodium (50, 100, 150 mmol/24-hr) for 30 consecutive days in a cross-over design. As compared with the control diet during high dietary sodium intake, the DASH diet and low dietary sodium intake lowered systolic BP by 11.5 mm Hg in participants with hypertension (12.6 mm Hg for blacks; 9.5 mm Hg for others).

Kawasaki et al, found the effect of very low sodium (10 mEq/day) or a high (200 mEq/day) dietary sodium intake on blood pressure in patients with essential hypertension. They found that half of the patients fed with high dietary sodium intake displayed a rise in blood pressure >10% [15].

Serum potassium among Hypertensive

In our study serum potassium was estimated in control and study groups and compared between them. Serum potassium was found to be lower in the hypertensive group when compared with the control group even though both were within the normal range. The mean serum potassium in the study group was 3.77 ± 0.36. The mean potassium in the control group was 4.32 ± 0.32.

A study was carried out by Bulpitt et al., among 2328 men and 1496 women between the ages of 35 and 64 years and were screened for hypertension and their plasma sodium and potassium concentrations measured. Those on antihypertensive or diuretic treatment were excluded from further analysis. After adjusting for age, body mass index and other variables, plasma potassium was negatively associated with both systolic and diastolic pressure in men and women. A decrease in plasma potassium of 1 mmol/l was associated with an increase in systolic pressure in women of 7 mmHg (p less than 0.001) and diastolic pressure of 4 mmHg (p less than 0.001). In men the corresponding increases were 4 mmHg (p less than 0.01) and 2 mmHg (p less than 0.05) [16].

BMI and Hypertension

In our study the mean BMI among the study group was 23.73 ± 3.28 and among the control group was 21.36 ± 2.12. The ‘p’ value was .00004. This shows that overweight and obesity also plays a role in the development of essential hypertension.

A study conducted by Huang stated that even a small amount of weight gain is associated with a marked increase in the incidence of hypertension [17]. This study showed a positive correlation between BMI and blood pressure which supported our study.
CONCLUSION
- Serum sodium was significantly more among hypertensive and it was independent of associated risk factors and gender
- Serum sodium level was also correlated positively with the level of blood pressure.
- Serum potassium was significantly less among hypertensive and it correlated negatively with blood pressure.
- Serum sodium and potassium were independent of body mass index.
- In view of the significant changes in simple electrolyte levels (sodium and potassium) among hypertensive population, community must be motivated to reduce their intake of common salt and encouraged to consume potassium rich nutrients – diets as a form of primary prevention for primary hypertension.

Limitations
- Only serum sodium and potassium were done.
- Twenty four hours urinary sodium and potassium and arterial blood gas analysis were not done due to technical and financial limitations. Renal handling of sodium and potassium was not attempted as it was beyond the scope of the present study.
- Body water content was not assessed which may alter the sodium and potassium levels
- Tissue sodium and potassium was not measured nor correlated with serum sodium and potassium level.
- Hormones related to sodium and potassium handling in kidney was not estimated.
- The salt intake of the patients could not be assessed quantitatively and qualitatively because of social constraints.

REFERENCES