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Original Research Article

Study on the Effects of Common Salt on Blood Pressure in Normal and Hypertensive Subjects in North Bihar

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Abstract:

Background: The growing population is making hypertension a significant issue. In order to shed light on a single aspect of this enormous issue and provide a straightforward preventive measure, we shall now investigate the effects of regular salt on hypertension. Aim of this study to observe the effects of common salt on blood pressure in normal and hypertensive subjects.

Methods: The study was done in the Physiology department, Darbhanga Medical College, Laheriasarai, Bihar, but involving persons attending various out patients department of the Hospital. Paper approval from the institutional ethical committee was obtained before starting the actual study. The timeline of the study was between June 2022 and December 2022. Test subjects were adults and recently diagnosed as hypertensive. Controls were healthy adults and normotensive, but were similar to controls in all other respects. BP (systolic & diastolic) was measured for all and pulse pressure and mean arterial blood pressure were calculated. Serum sodium, potassium and chloride levels were estimated and the urine also was examined for specific gravity, sodium and chloride. Salt intake of all subjected was enquired about and they were divided into four categories according to salt intake, viz. low salt in taker, optimum salt intaker, high and very high salt intaker. Their BP and other parameters were calculated and compared. Results were statistically analyzed using the SPSS software.

Results: The mean systolic blood pressure (SBP)± standard deviation (SD) in mm of Hg in the normal or control population was 117.5±16.7 compared with that in the hypertensive or test population being 167.6±31.6. The mean diastolic blood pressure (DBP) ± SD in mm of Hg, obtained is 76.7±9.5 as compared to the test in control population which is 102.3±14.8. The mean pulse pressure (PP) ± SD in mmHg in the control population is 40.5 ±6.7 and that in the hypertensive population is 64.7±18.In our study, MABP was 90.2±11.5 in normal population, whereas it was 123.5±19.6 in the hypertensive population. The mean serum sodium of the control population was 138.7±19.7 mmol/L; on the other hand, the same in our hypertensive population was 144.2±20.1.In control population, the serum chloride levels were 101.4±13.0 mmol/L, whereas the same in hypertensive population was 112.4±15.4. The latter was distinctly higher, though was not statistically significant. The urinary specific gravity in normal, healthy person is 1.015± 0.051 (SD). In case of hypertensives, the same is 1.021±0.052.The average PPs in mm of Hg (SBP-DBP) were 40.7±5.4 in low salt takers, 42.2±10.7 in optimum salt takers, and 61.0±16.3 in high salt takers and 77.2±18.3 in very high salt takers. Again, the average MABP (DBP+1/3 PP) in mm of Hg in different types of salt takers, 92.6±10.5 in optimum salt takers, 119.3±8.4 in high salt takers and 135.8±7.4 in very salt takers.

Conclusion: In conclusion it can be said that dietary salt has a reflection on blood pressure. Salt ingested quantity is directly proportional to blood pressure, whether it is SBP, DBP, PP or MABP. Further studies on a much bigger scale and in a more elaborate way are required to establish the validity of this issue. But, right now it is time enough to start salt restriction in general population, particularly hypertensive in a massive way.

Keywords: Systolic blood pressure, diastolic blood pressure, hypertensive population, optimal salt.

Introduction

Hypertension is one of the major killers and most prevalent chronic and major public health challenge to population in socio-economic and epidemiological transition. As a matter of fact hypertension kills the patients prematurely resulting into the impairment of the life expectancy, and hence it is a disastrous disease of the world. It is the commonest cardiovascular disorder, posing to become a serious

public health problem in the developing countries like India and also all over the world. Sir George Peckering formulated a concept that blood pressure, in a population, is distributed continuously as a bell-shaped curve with no real separation between normotension and hypertension.[1]

Even though our understanding of the pathophysiology of an elevated arterial pressure has increased in recent times, yet in 90 to 95% of cases the aetiology (and thus potentially the prevention or cure) is still largely unknown.[2]

In most populations the mean blood pressure level in males and females rises progressively with advancing age. This gradual increase in blood pressure is not a benign process and there is an increasing morbidity and mortality as the pressure rises, even at levels which most clinicians would accept as normal. There is no clear dividing line in this population between normal and abnormal, and most workers would agree that what we call essential hypertension represents the upper end of a frequency distribution curve. Nevertheless, there are communities in whom blood pressure does not rise with age and in whom the problem of essential hypertension and its complications appears to be virtually non-existent.

The relation of salt intake to hypertension is a controversial subject, and much circumstantial evidence suggests that there is a positive correlation between the level of salt intake and the prevalence of hypertension in a community.[3]

In primitive societies where salt intake is very low, raised blood pressure is rarely seen, and blood pressure does not rise with advancing age. [4]

The hypothesis that excessive salt ingestion may be an important factor in the development of hypertension is based on much indirect evidence but little direct proof. [5] Excessive salt intake is probably a major cause of the epidemic of hypertension in civilized countries and a reduction in salt intake may help to combat the epidemic.[6]

Hypertension was induced in rats by mixing sodium chloride with food and allowing ad libitum consumption of food as well as water.[7] When daily dietary ingestion of sodium chloride is below 60 mmol per day approximately as in an acculturated person, the prevalence of hypertension is negligible. Few comparative studies have been conducted on the short and long term effects, that altering dietary sodium intake has, on the blood pressure in both normotensive and hypertensive subjects.[9]

Deficient sodium excretion must be an essential factor in all types of persistent hypertension. [10]

It this is so, a reduction in the dietary sodium intake should cause a greater fall in blood pressure in patients with hypertension than in normotensive subjects, unless irreversible changes have occurred in the patients with hypertension. [11] There is only a small change in blood pressure with alteration of dietary sodium intake in normotensive subjects. [12]

Hypertension is becoming sizable problem due to population explosion day by day. We will hereby study the effects of common salt on hypertension just to reveal one facet of this huge problem and to get a simple preventive measure.

Material and Methods

This institutional and community-based observational and cross sectional study was done in the Physiology department of Darbhanga Medical College, Laheriasarai, Bihar from June 2022 to December 2022 involving persons attending various out patients departments of the hospital. Proper approval from institutional ethical committee was obtained before starting the actual study. Total 100 patients divided in two groups: 50 adult subjects of 20 yrs. of age or above but below 60yrs in case and 50 apparently healthy normal subjects between 20 & 60 yrs. of age were be taken as control group. Subjects were randomly selected for study population and similar in both case and control group, except regarding blood pressure.

Inclusion criteria:

1. Adult subjects between 40 yrs. And 60 yrs. of age. In the controls, the selected age group was the same.
2. Patients without receiving antihypertensive drugs. Newly diagnosed hypertensive as test subjects, duration of hypertension being less than a month and not yet on drugs.
3. Supine diastolic pressure must be 90 mm of Hg or more, even after 10 min rest.
4. Patients not taking sodium depleting drugs like common diuretics or other relevant drugs which might impair study results.

Exclusion criteria:

1. Evidence of ischemic heart disease, cerebro vascular disease, renal failure, diabetes mellitus, acromegaly, Cushing syndrome, pheochromocytoma etc.
2. Patients having H/O taking antidepressant, Analgesic, oral contraceptives, sedatives and those who are drug abusers.

Subjects were selected from Medicine and other OPD and control subjects from staff and students mainly, provided they fulfilled the inclusion and exclusion criteria.

Methods

Normal and hypertensive subjects were studied with high salt intake (350 mmol/day) for fifteen days and with low salt intake 910 mmol/day) for

fifteen days. The higher salt diet was achieved by adding common salt to the normal food; lower salt diet was achieved by not adding any amount of salt to the food materials.

Boiled rice and fruits are the best source for providing low salt, and avoided any type of food materials that supplied high salt. (e.g, pickles, salted nuts, chines food etc). All the subjects on normal diet were admitted in the ward two days before beginning of the trial. Blood pressure, pulse and weight of the subjects were recorded on two consecutive days. On the same days, serum sodium was also estimated and simultaneously 24 hours urinary sodium excretion was measured from collected urine for 24 hours in a large container. All the subjects were instructed to take common salt strictly prescribed to them and advised to do their normal work as before.

All the subjects were admitted again on the 13th day, and blood pressure, pulse rate, weight, serum sodium and 24 hour urinary sodium excretion ere measured on the 14th and 15th day. Then they were instructed to take boiled rice and fruits or foods completely devoid of salt and advised to do their usual work as before and the patients are discharged. And again they were admitted on 13th day and blood pressure, pulse rate; weight, serum sodium and 24 hour urinary sodium excretion were measured on the 14th and 15th day. The average readings were considered for calculation.

Biochemical methods:

By six multichannel Autoanalyser of Technician Ltd. The fasting blood sugar, blood urea, serum creatinine and uric acid, cholesterol and serum triglycerides were measured.

Serum sodium and 24 hour urinary sodium excretion were measured by an Autoanalyser flame photometer.

Results

Table 1: Comparative study of SBP between normal and hypertensive populations

Populations	DBP± SD (mmHg)
Normal	**117.5±16.7**
Hypertensive	**167.6±31.6**

Table 2: Comparative study of DBP between normal and hypertensive populations

Populations	DBP± SD (mmHg)
Normal	**76.7±9.5**
Hypertensive	**102.3±14.8**

Table 3: Comparative study of PP between normal and hypertensive populations

Populations	PP±SD (mmHg)
Normal	**40.5±6.7**
Hypertensive	**64.7±18.0**

Table 4: Comparative study of MABP between normal and hypertensive populations

Populations	MABP±SD (mmHg)
Normal	**90.2±11.5**
Hypertensive	**123.5±19.6**

Table 5: Comparative study of serum Na^+ between normal and hypertensive populations

Populations	Serum Na^+ ± SD (mmHg)
Normal	**138.7±19.7**
Hypertensive	**144.2±20.1**

Table 6: Comparative study of serum Cl^- between normal and hypertensive populations

Populations	Na^+±SD (mmol/L)
Normal	**101.4±13.0**
Hypertensive	**112.4±15.4**

Table 7: Comparative study of urine Na^+ between normal and hypertensive populations

Populations	Mean Na^+±SD (mmol/L)
Normal	**151.4±29.7**
Hypertensive	**233±39.8**

Table 8: Comparative study of urine Cl^- between normal and hypertensive populations

Populations	Mean Cl^-±SD (mmol/L)
Normal	**104.7±15.0**
Hypertensive	**128.5±19.1**

Table 9: Comparative study of urine Specific gravity between normal and hypertensive populations

Populations	Urine Specific gravity
Normal	**1015±51**
Hypertensive	**1021±52**

Table 10: Comparative study of no of high salt takers between normal and hypertensive populations

Populations	High salt takers (%ge)
Normal	**6.67**
Hypertensive	**90**

Table 11: Comparative study of average pulse pressure (PP) among different salt consuming populations

Populations	Average PP (mmHg)
Low salt takers	**40.7±5.4**
Optimum salt takers	**42.2±10.7**
High salt takers	**61.0±16.3**
Very high salt takers	**77.2±18.3**

Table 12: Comparative study of average mean arterial blood pressure (MABP) among different salt consuming populations

Populations	Average MABP (mmHg)
Low salt takers	**92.4±10.8**
Optimum salt takers	**92.6±10.5**
High salt takers	**119.3±8.4**
Very high salt takers	**135.8±7.4**

Discussion

In the present study we compared the same parameters in two groups of populations who were age and sex matched and were otherwise healthy and similar in all respects except that one group was not usually normotensive and the people belonging to the other group were essentially hypertensive who were recently diagnosed as suffering from essential hypertension and were yet to be put on drugs. The parameters examined were a part from anthropometric measurements, a subject recall and scoring of their dietary salt intake, plasma electrolytes, particularly sodium and 24 hour urinary sodium excretion.

The mean systolic blood pressure (SBP)± standard deviation (SD) in mm of Hg in the normal or control population was 117.5±16.7 compared with that in the hypertensive or test population being 167.6±31.6. According to American Heart Association's (www.heart.org) online posting on "understanding blood pressure readings" (Oct, 2016), the SBP in a normal person should be 120 mmHg or lower than that (vide Table 1). An SBP is called low only when it is accompanied by symptoms of dehydration or syncope or at least some significant dizziness; otherwise, no lower limit of SBP has been prescribed.[13]

In our study, the mean diastolic blood pressure (DBP) ± SD in mm of Hg, obtained is 76.7±9.5 as compared to the test in control population which is 102.3±14.8. According to Mayo clinic website on normal blood pressure when the DBP is 80-89, it is stage 1 and ≥ 90, it is stage 2 hypertension. Only when the DBP is <80, can it be called normal DBP. Our study shows that our control population is truly normotensive, and our test population reflects stage 2 hypertension.[14]

It may be worth mentioning here that both SBP and DBP reflect stage 2 hypertension in our test population and nicely tally with both AHA and Mayo clinic criteria.

Our study shows the mean pulse pressure (PP) ± SD in mmHg in the control population is 40.5 ±6.7 and that in the hypertensive population is 64.7±18. A study by Benetos et al [15] shows that normal mean PP is <45 mmHg, and it is clinically highly important in that it is the single independent major predictor of cardiac risk, and the greater the PP, the larger is the risk. Neither SBP nor DBP is a better predictor than PP of an impending myocardial infarction.

In the same above study [15] MABP was called normal when it was <107 and high when it was >107. In our study, it was 90.2±11.5 in normal population, whereas it was 123.5±19.6 in the hypertensive population. The above study concludes that MABP is much less important in predicting cardiac disorder or death as compared, to PP. However, unlike PP, it (MABP) is a powerful predictor of cerebrovascular events (CVA).

In the study of our assignment, the mean serum sodium of the control population was 138.7±19.7 mmol/L; on the other hand, the same in our hypertensive population was 144.2±20.1.

In Medline plus Medical Encyclopedia [16] the normal range for blood sodium levels is 135 to 145 mmol/L. Our value grossly represents that figure,

although it is also stated that normal value ranges may vary slightly among different laboratories. Some labs also use different measurement technique which is a cause of difference in results.[17]

It is told that chloride ion has also some role in maintaining blood pressure; in fact, it has a hidden hand in hypertension.[18] In the present study, in control population, the serum chloride levels was 101.4±13.0 mmol/L, whereas the same in hypertensive population was 112.4±15.4. The latter was distinctly higher, though was not statistically significant.

Early studies in salt-sensitive rats (Dah 1) showed that hypertension occurred within several weeks when animals were fed on a high NaCl diet, but not when the animals were fed on identical Na+ load provided as NaHCO3 or other non-chloride salts of Na+.[19,20,21]

Curiously, a diet containing a combination of sodium chloride and sodium bromide induces hypertension more readily than other non-chloride sodium salts in rats, suggesting that the role of Cl- in the effect of NaCl on blood pressure may be related to some property common to halides.[22]

The earliest clinical study in 1929 showed that Cl- was the main blood pressure increasing component with the observation that sodium carbonate did not have the same pressor effects as sodium chloride in hypertensive individuals.[23] Kurtz et al[24] demonstrated that the rise in blood pressure in response to a high sodium diet (high salt diet, 240 mmol sodium chloride per day; 5.52 gm Na+) was abolished upon substituting an equimolar amount of sodium citrate. Luft et al [25] showed opposite effect of NaCl and NaHCO3 on blood pressure and calcium excretion with NaHCO3 reducing blood pressure but increasing calcium excretion. Shoreetg al[26] reported that NaCl intake induced a greater rise in blood pressure than sodium phosphate intake. Quollman showed that both Na+ and Cl- were required to increase BP in humans.[27]

A urine specific gravity test performed by a simple urinometer is part of all urinalysis. In our study, we also performed this urine specific gravity measurement. This test is an inexpensive easy to perform and painless way to know how well the kidneys are diluting the urine. This test compares the density of urine to the density of distilled water. Urine specific gravity is increased in: dehydration, heart failure, shock, diabetes mellitus any type of nephropathy particularly nephrotic syndrome and chronic renal failure. It is also increased in urinary tract infection (UTI) and hypernatroemia. Specific gravity of urine is grossly decreased in diabetes insipidus, over drinking of water and hyponatroemia.

Other than urinometer, a refrartometer may also be used to know the specific gravity of urine. A dip test is much simpler but an inaccurate method.

In our study, we have wanted to see if hypertension can have any effect on urinary specific gravity. The normal urine specific gravity in a healthy adult in 1.010 to 1.020 more the specific gravity, more in the urinary concentration.

In the study done by us, the urinary specific gravity in normal, healthy person is 1.015± 0.051 (SD). In case of hypertensives, the same is 1.021±0.052. That means, in hypertensives, the urinary specific gravity is a bit higher than that in the normal control population.

Rapoport et al [28] also found urinary specific gravity higher in essential hypertension. He attributed this to increased renal concentrating capacity in this disease. Rowat et al [29] in a pilot study also found increased urine specific gravity in acute stroke in which blood pressure is almost invariably raised.

Again, Astrung et al [30] in a Bulgarian journal also reported in his study on 58 patients with arterial hypertension, that the urine specific gravity and osmolarity are significantly raised in hypertensive patients compared to normal normotensive healthy adult controls. They suggested that urine specific gravity rather than osmolarity can be used to assess renal concentrating power in everyday clinical practices due to its convenience.

We also did studies on salt intake of patients and their relationship with blood pressure. We divided the population according to salt intake patterns (as mentioned in the methodology chapter) into four categories: those who took less than average salt in food (i.e., most people would like to add salt to the food while eating) are called “low salt takers” those who took average salt in food were designated as “optimum salt takers” those who always took a little bit of added salt to optimally salty food were called “high salt takers” and there who take extra salt and lib while eating were named “very high salt takers”.

It was found in our study that in the hypertensive population (diagnosed first and not advised on diet) 90% were high salt takers, compared to the control i.e., normotensive population where only 6.67% were high salt takers.

The average PPs in mm of Hg (SBP-DBP) were 40.7±5.4 in low salt takers, 42.2±10.7 in optimum salt takers, and 61.0±16.3 in high salt takers and 77.2±18.3 in very high salt takers. Again, the average MABP (DBP+1/3 PP) in mm of Hg in different types of salt takers, 92.6±10.5 in optimum salt takers, 119.3±8.4 in high salt takers and 135.8±7.4 in very salt takers. So, no difference in MABP was found between low and optimum salt takers; but it

progressively and significantly became higher in high and very salt takers respectively. The same was noted in case of PPs also.

No comparable studies were found by through literature survey on issue like ours viz, PP and MABP in different categories of salt takers. However, in a study from Belgium by Staessen et al similar results were observed.[31] But, more than sodium intake effect, in their study it was found that increased potassium intake reduces blood pressure. According to the salt-genetic hypothesis an excessive salt intake in genetically susceptible individuals may lead to an increase in blood pressure and eventually to the development of hypertension. A positive relationship between salt-intake and blood pressure has been demonstrated in rats.[32]

In humans, the salt-genetic hypothesis has also received much attention and has been discussed by Freis [33], Simpsm [34], Swales [35] and Amery et al[36]. Isolated societies have been described [37-39] where the dietary sodium intake is low, and where blood pressure does not increase with advancing age. It is not clear whether these observations can be extrapolated in Western societies. Also, practically no studies have been made in Asian region, let alone in India.

This is why we have ventured to perform this study on salt intake and blood pressure in an eastern Indian population. Food habits may be different in relation not only to salt intake, but also, for example to potassium intake, protein intake and calorie consumption. Also, genetic differences have to be considered. Staessen et al in his article [28] concluded that to them it was more relevant to study the relationship between salt intake and blood pressure within Western industrialized society. In their study, they investigated this relationship in a sample of a Belgian community and compared their results to those obtained in Scotland as reported by Beevere et al.[40] Interestingly in our Indian study also, we found similar results were found like those in Western industrialized country studies, that is, increased salt intake leads to more preponderance of increased blood pressure. It is important to note that there are at least three other variables which might affect blood pressure measurement, and these are age, body weight and pulse rate. Staessen et al adjusted these three variables while taking into account their subject's blood pressures. However, because of technical reasons the same could not be done in our studies; yet, we got similar results.

The global burden of essential hypertension was in 2000 around 972 million and is predicted to be in 2025, around 1.56 billion. [41]This tremendous state of affairs places a huge number of people suffering from essential hypertension at a higher risk for cardiovascular diseases, renal failure and ocular problems and so on. The Dietary Approaches to stop Hypertension (DASH) trial has demonstrated that a change in diet rather than traditional American or European diet definitely decreases blood pressure and chances of hypertension related morbidity. Clinical trials have shown that reducing the sodium chloride content of typical diets lowers blood pressure [42-44] and guidelines recommended reducing the daily dietary sodium intake to 100 mmol (equivalent to 2.3 gm of sodium or 5.8 gm of sodium chloride) or lees.[45]

Our studies prove, along with so many other similar studies in the USA and Europe, that high dietary salt intake does lead to hypertension and worsens an existing hypertensive state. In short our results provide support for a more aggressive target for reduced salt intake for the prevention and treatment of elevated blood pressure.[42]

Conclusion

In conclusion it can be said that dietary salt has a reflection on blood pressure. Salt ingested quantity is directly proportional to blood pressure, whether it is SBP, DBP, PP or MABP. Since, prolonged blood pressure in the higher stratum has many untoward effects or various systems of the body, particularly the CVS, it has to be consistently kept under control. Now that salt restriction is such an easy and simple thing, and it imparts a profound impact on both prevention and treatment of hypertension, there is no reason why this should not be practiced by all. A lot of papers have been published in the Western world, but not all of them had similar results.

Some researchers found no effect of excess sodium on blood pressure, but that potassium causes diminution of blood pressure has been observed. Again, some authorities have found deleterious effects of sodium chloride on blood pressure, but not of other sodium salts. Effects of salt on blood pressure, as is most physiological parameters, are also dependent on the genetic set up of the community.

Therefore, as Westerners concentrated on studies on Western industrialized society, there is logic on Asia/Indian counter parts to work on this region of the globe. But, unfortunately, very little work has been done on this issue in Indian subcontinent. Further studies on a much bigger scale and in a more elaborate way are required to establish the validity of this issue. But, right now it is time enough to start salt restriction in general population, particularly hypertensive in a massive way.

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