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Effect of Salt on Blood Pressure

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Abstract

The prevalence of Hypertension has been on a steady rise. Various lifestyle factors have been postulated to be a reason for the same. Dietary habits have been shown to effect the levels of blood pressure. The oral intake of salt has been over the years shown to have a direct effect on the blood pressure levels. This paper aims at analyzing various studies conducted over the years to understand the extent of the effect of salt. In the studies, a reduction in the salt intake resulted in the lowering of urinary excretion of sodium, as well as a reduction in the blood pressure levels. This is an important finding, as lifestyle changes including a healthier diet may help in controlling Hypertension.

INTRODUCTION

The chemical name for salt is Sodium Chloride. The words salt and sodium are not exactly the same, yet these words are often used synonymously. For example, the Nutrition Facts label uses "sodium," whereas the front of the package may say "low salt."[1] Ninety percent of the sodium consumed is in the form of salt through the diet.[2]

Physiology of Sodium regulation and its effect on Blood Volume

Kidneys play a major role in regulating the fluid and electrolyte balance within the body. They are responsible for maintaining the Osmolarity (the amount of solute per unit volume) of body fluids. The Osmolarity of the body plasma is achieved by balancing the intake and the excretion of Sodium and water. Sodium is the major solute present in the extra-cellular fluid and so, it effectively determines the Osmolarity of the extra-cellular fluids.

The Anti- Diuretic Hormone (ADH) increases the water resorption from the kidneys and thus lowers the Osmolarity (reduces sodium concentration). To prevent Hypo-osmolarity, the Aldosterone mechanism comes into play by reabsorbing sodium in the distal tubules of the kidneys. Its secretion is controlled by the following mechanisms-

1. The aldosterone secretion is inhibited when the osmolarity increases above normal. The adrenal cortex directly senses plasma osmolarity. The lack of aldosterone causes less sodium to be reabsorbed in the distal tubule. In this scenario, ADH secretion increases to conserve water, thus complementing the effect of low aldosterone levels to decrease the osmolarity of bodily fluids. The net effect on urine excretion is a decrease in the amount of urine excreted, with an increase in the osmolarity of the urine.

2. The kidneys sense low blood pressure (which results in lower filtration rates and lower flow through the tubule). This triggers a complex response to raise blood pressure and conserve volume. Specialized cells (juxtaglomerular cells) in the afferent and efferent arterioles produce Renin, a peptide hormone that initiates a hormonal cascade that ultimately produces Angiotensin II. Angiotensin II stimulates the adrenal cortex to produce Aldosterone.

Urinary Salt Excretion

Under normal activity conditions and sweating, humans excrete almost 90% of dietary sodium through urine. Therefore, 24 hrs urinary sodium levels provide an indication to the daily intake of sodium. These levels vary among individuals and depend on the salt intake as well. Thus, a single collection may not reliably reflect the habitual intake and repeated estimations of the same are suggested.

The Intersalt study[9] published in 1988 established a statistically significant positive correlation between the 24hr Urinary Sodium Excretion and the prevalence of Hypertension. It was observed in this study that higher Blood Pressure values were seen in populations where the sodium excretion was higher. This indicated that the oral intake of sodium influenced the Blood Pressure values and a direct proportional relation is established between the two. Additionally it was noted that the populations with a higher Urinary Sodium Excretion and increasing age had higher Blood Pressure values. In a similar study conducted by Law and Co-workers, similar findings were observed and a positive relation was established by the study between mean blood pressure and sodium intake[14].

The American Heart Association in its 2013 guidelines has propagated that maintaining a healthy diet and an active lifestyle contributes to a great extent towards reducing the risk for CVD in general public. Diet and lifestyle are modifiable risk factors responsible for development of CVD of which Hypertension is of major concern from public health point of view. Positive interventions in these factors are considered to be mainstay of the treatment protocol in spite of major advances in clinical medicine.[4]

A substantial body of evidence strongly supports the concept that multiple dietary factors affect BP.[6] Dietary modifications that lower BP are reduced salt intake, caloric deficit to induce weight loss, moderation of alcohol consumption (among those who drink), increased potassium intake, and consumption of an overall healthy diet, based on the DASH (Dietary Approaches to Stop Hypertension) diet.[7] The latter is a carbohydrate-rich diet that emphasizes fruits, vegetables, and low-fat dairy products; includes whole grains, poultry, fish, and nuts; and is reduced in fats, red meat, sweets, and sugar-containing beverages. Replacement of some carbohydrates with either protein from plant sources or with monounsaturated fat can further lower BP.[8]

Dietary Approach to Stop Hypertension (DASH) is a diet which is rich in vegetables and fruits and low in fats and high fat dairy products. It has been widely advised and propagated in Hypertensive patients. In United States, a multicentre trial was conducted to assess the effect of dietary sodium intake, in conjunction with DASH diet on Blood pressure levels in persons with and without Hypertension.[15] The participants were given a control and a DASH diet randomly. In both the groups, the participants were given foods with high, intermediate and low sodium levels for 30 consecutive days, randomly. A reduction in systolic blood pressure was noted on reducing the sodium intake from the high to the intermediate level during the control diet and by 1.3 mm Hg during the DASH diet. Similarly systolic blood pressure reduction of 4.6 mm Hg during control diet and 1.7 mm Hg during DASH diet was seen when the sodium intake was reduced from the intermediate to the low level. This reduction was additional to the former reduction from high to intermediate sodium intake levels. These effects were observed in participants of African-American origin and other races, with or without Hypertension and in both males and females. It was observed that the DASH diet was associated with lower blood pressure levels at the each of the three sodium intake levels (low, intermediate and high) with a greater difference at the higher sodium levels than the lower sodium levels. The participants in the DASH diet consuming low sodium as compared to the control diet participants had a mean systolic blood pressure that was 7.1 mm Hg lower in the group without hypertension, and 11.5 mm Hg lower in the group with hypertension.

In a study where experimental diets of 1200-1600mmol/day of sodium was given to 14 normotensive volunteers, a significant rise in the blood pressure was seen in all participants within three days.[16] Studies involving increasing the salt intake on a long term basis cannot be conducted due to ethical concerns. A 20-month study was carried out on chimpanzees that were given salt supplements and their systolic blood pressures measured. They were previously on a salt free diet. It was noted that an average rise of 33 mm Hg of systolic blood pressure was seen in them over the 20-month study period. There was no change in the chimpanzees in the control group living in the same conditions (on salt free diet). The blood pressures returned to baseline within six months in all chimpanzees when they were put on a salt free diet. [17]

Salt reduction and blood pressure

In view of the above observations, researches have been continually trying to assess the efficacy of reducing the dietary salt intake in reducing blood pressure levels[6-12] through various systemic reviews and meta-

analysis. [18,19] The meta-analysis of randomized controlled trials by He and Mac Gregor concluded that a reduction in the salt intake for four weeks or more achieved a reduction of 5 mm Hg of systolic blood pressure in people classified as hypertensive.[22]

In another meta-analysis by Law *et al*[20] it was observed that a reduced sodium excretion significantly reduced the systolic blood pressure levels in both normotensive and hypertensive individuals.

Dietary Guidelines for Sodium

The 2010 Dietary Guidelines for Americans recommends limiting sodium to less than 2,300 milligrams (mg) per day. For individuals the age of 51 years and above, especially of the African- American origin or those people who already have Hypertension, Diabetes or Chronic Kidney Disease should limit their Sodium intake to 1,500 mg per day[3].Individuals who are 51 years and older and those of any age, including children, who are African American or have high Blood Pressure, Diabetes, or Chronic Kidney Disease should limit intake to 1,500 mg of sodium per day as per the guidelines.[3]

Strategies to reduce sodium intake include:

- Preferring foods which have been processed without salt and foods labeled as 'no added salt' and avoiding all 'high salt' processed foods.
- A 'low salt' food label means that the food item contains no more than 120 mg of sodium per 100 g
- Reduced salt products have a higher salt content than the low salt containing products. These can be opted for if the latter is not available.
- Avoiding adding salt during cooking and at the table.
- Low sodium salts usually have high Potassium levels and these should be avoided in patients with renal dysfunction or in those who are taking potassium-sparing diuretics.
- Dosages of antihypertensive medications and of lithium carbonate may need to be reduced in patients who adopt a low salt diet.
- The combination of diuretic treatment and low dietary salt intake may result in unacceptable volume depletion and hyponatraemia.
- Dietary salt restriction is inappropriate in patients with salt-wasting forms of renal and gastrointestinal disease.

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