

RESEARCH ARTICLE

A STUDY ON SERUM SODIUM AND POTASSIUM IN NEWLYDIAGNOSED PRIMARY HYPERTENSION

Dr. Mohammad Salman¹ and Dr. Sanjay H. Kalbande²

- 1. 3rd Year Post-Graduate, Department of General Medicine, Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar.
- 2. Professor and HOD of Department of General Medicine, Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar.

Manuscript Info

Manuscript History Received: 30 November 2022 Final Accepted: 31 December 2022 Published: January 2023

Abstract

Background Of Study: Hypertension is one of the world's leading causes of death and disability among adults. It is the major risk factor responsible for coronary, cerebral and peripheral vascular disease. Primary hypertension comprises more than 90% of hypertension.

Objective: To study the levels of serum sodium and potassium in newly diagnosed primary hypertension patients. To correlate the sodium and potassium levels with the blood pressure.

Method: The work was carried out in the outpatient Medicine department of Chalmeda Anand Rao institute of medical sciences.Cross-sectional Study (Analytical Study)

Sample size: 50 cases (35 cases and 15 controls)

Results: Serum sodium was higher in the hypertensive group than in the control group even though both were within the normal range. The mean and standard deviation of serum sodium among cases was 147.94 \pm 2.88 meq/L while in the control group, it was 138.86 \pm 3.44 meq/L respectively. 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 3.55 \pm 0.29 meq/L. The mean potassium in the control group was 4.67 \pm 0.22 meq/L.

Conclusion: Serum sodium was significantly more among the hypertensive population and it was independent of gender. Serum potassium was significantly less among the hypertensive population and it correlated negatively with the level of blood pressure. Serum sodium level also correlated positively with the level of blood pressure.

.....

Copy Right, IJAR, 2023,. All rights reserved.

Introduction:-

Hypertension is one of the world's leading causes of death and disability among adults. It is the major risk factor responsible for coronary, cerebral and peripheral vascular disease. Primary hypertension comprises more than 90% of hypertension (1). When the majority of people are aware of their hypertension status, they have already advanced into a stage with target end organ damage – a fatal stroke or myocardial infarction or irreversible renal failure In addition to a primary increase in cardiac function as a result of the overactive sympathetic nervous system, primary retention of salt and

Corresponding Author:- Dr. Mohammad Salman

Address:- 3rd Year Post-Graduate, Department of General Medicine, Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar.

water by the kidney, other factors that contribute to hypertension are hereditary predisposition and high sodium and low potassium intake and excretion. Decreased intake of sodium and an increased intake of potassium or both together may be effective in the prevention or even in the treatment of hypertension. Recent studies among Indians have shown an increasing prevalence of hypertension in both urban and rural areas (2,3).

Mechanisms Of Primary Hypertension:

1. Non-renal factors, 2. Renal factors

Non-Renal Factors

A. Genetic Predisposition:

Harrap suggested that the average population blood pressure is determined by theenvironment but the blood pressure rank within the distribution is decided largely by genes (4). If genetic markers of a predisposing factor for the development of hypertension are found, then specific environmental manipulations could be directed toward the susceptible individuals or subjects (5). Genetic abnormalities may be monogenic in some rare forms of hypertension like glucocorticoid remediable aldosteronism, Liddle's syndrome, and syndrome of apparent mineralocorticoid excess (6).

B. Fetal Environment:

Low birth weight as a consequence of fetal under-nutrition is followed by an increased incidence of high blood pressure later in life with an overall estimate that a 1 kg lower birth weight is associated with a 2 to 4 mm Hg higher systolic blood pressure in adulthood (7). Brenner and Chertow hypothesized that a decreased number of nephrons from the intrauterine growth retardation could very well serve as a permanent irreparable defect that results in hypertension (8).

C. Vascular Remodeling:

Several factors increase peripheral resistance by both functional contraction and vascular remodelling and hypertrophy. Multiple vaso-active substances act as pressure-growth promoters which results in both vascular contraction and hypertrophy simultaneously, but the perpetuation of hypertension involves hypertrophy. Lever and Harrap (9) postulated that primary hypertension has two mechanisms similar to secondary hypertension – (a) a growth-promoting process in children, and (b) a self-perpetuating mechanism in adults.

D. Neurohumoral Causes Of Primary Hypertension:

A large number of circulatory hormones and locally acting substances may be responsible and involved in the development of hypertension which causes hypertension by factors such as vascular hypertrophy, capillary rarefaction and impaired microvascular dilation (10)

Sympathetic Nervous Hyperactivity:

Young hypertensives tends to have a higher level of circulating catecholamines, augmented sympathetic nerve traffic in muscles, faster heart rate and heightened vascular reactivity to α adrenergic agonists (11).Renin–Angiotensin System: Both as a direct pressor and as a growth promoter, therein–angiotensin mechanism may be also involved in the pathogenesis of hypertension. All functions of renin are mediated through the synthesis of angiotensin II. This system is the primary stimulus responsible for the secretion of aldosterone and hence mediates mineralocorticoid responses to varying sodium intake and volume overload. When sodium intake is reduced or effective plasma volume shrinks, the increase in renin–angiotensin II stimulates aldosterone secretion, which in turn is responsible for a portion of the enhanced renal retention of sodium and water. When large populations of hypertensives patients are surveyed, only about 30 per cent have low plasma renin activity levels, whereas 50 per cent have normal levels and the remaining 20 per cent have high levels (12).

Hyperinsulinemia / Insulin resistance:

An association between hypertension and hyperinsulinemia has been recognized for many years, particularly with accompanying obesity but also in about 20 per cent of non-obese hypertensive patients (13).

Endothelial Cell dysfunction:

The endothelium is now known to be the source of multiple relaxing and contracting substances, of which nitric oxide is an important vasodilator (14). The impairment of normal vasodilation in insulin resistance syndrome has been shown to involve failure to synthesize the normal endothelium-derived relaxing factor (NO).

Minerals:

Excess of lead and changing ratios among dietary sodium, potassium, calcium and magnesium have also been postulated in the pathogenesis of primary hypertension (15).

Renal Retention Of Excess Dietary Sodium:

A considerable amount of circumstantial evidence supports the role of sodium in the genesis of hypertension. To induce hypertension, some of the excess sodium must be retained by the kidneys. Such retention could arise in several ways.

Nephron heterogeneity is described as the presence of a subpopulation of nephrons that is ischemic either from afferent arteriolar vasoconstriction or from an intrinsic narrowing of the lumen. Renin secretion from this subgroup of nephrons is tonically elevated. This increased renin secretion then interferes with the compensatory capacity of intermingled normal nephrons to adaptively excrete sodium and consequently, overall blood pressure homeostasis (16). A decrease in the filtration surface by a congenital or acquired deficiency in nephron number or function. A resetting of the normal pressure – natriuresis relationship – the Guyton hypothesis (17). An acquired inhibitor of the sodium pump or other abnormalities in sodiumtransport (18). Defensive responsiveness to atrial natriuretic hormones (19).

Association Of Hypertension With Other Conditions:

- 1. Physical inactivity
- 2. alcohol
- 3. smoking
- 4. **Haematological findings**: Higher hematocrits are found in hypertensive persons and are associated with abnormal left ventricular filling on echocardiography (20).
- 5. **hyperuricemia**: Hyperuricemia is present in 25 per cent of individuals with untreated hypertension, and more than 75% of patients with malignant hypertension which are about five times the frequency found in normotensive persons
- 6. with sleep apnoea
- 7. **Hypercholesterolemia**: Hypercholesterolemia frequently coexists with hypertension, at least in part because it impairs endothelium–dependent vasodilation. Lipid-lowering therapy restores the bioavailability of nitric oxide, reduces arterial stiffness, and lowers blood pressure

Aims & Objectives:-

To study the levels of serum sodium and potassium in newly diagnosed primary hypertension patients. To correlate the sodium and potassium levels with the blood pressure.

Materials And Methods:-

The work was carried out in the outpatient Medicine department of Chalmeda Anand Rao institute of medical sciences. Sample size: 50 cases (35 cases and 15 controls)

Inclusion Criteria:

- 1. Patients with newly diagnosed primary hypertension.
- 2. Patients above 20 years.
- 3. Both males and females.

Exclusion Criteria:

- 1. Patients below 20 years.
- 2. Patients with diabetes mellitus.
- 3. Patients with renal failure.
- 4. Pregnancy.
- 5. Females on oral contraceptive pills.
- 6. Patients with secondary hypertension.

Results:-

The majority of the patients in both the study and control group lie between 31 and 70 years. The mean age group in both the case and control groups are 56.2 ± 11.6 and 49.6 ± 10.8 respectively. There was no significant difference in the age composition of those with and without hypertension in this study with a 'p-value of 0.066. Since alcoholism and smoking were noticed among men only in this part of the country, statistical analysis was not attempted for these risk factors.

Blood pressure distribution among cases:

Distribution of systolic and diastolic blood pressure

	Cases	Controls
	Mean + SD	Mean + SD
Blood Pressure		
Systolic	161.25 ± 18.99	110.53 ± 5.97
Diastolic	91.17 ± 12.79	71.06 ± 5.28

Serum sodium in relation to gender:

The mean value of serum sodium was $148 \pm 3.65 \text{ meq} / \text{L}$ in males and $147.88 \pm 2.22 \text{ meq} / \text{L}$ in females among cases. The mean value of serum sodium was $137.6 \pm 1.67 \text{ meq} / \text{L}$ in males and $139.5 \pm 3.97 \text{ meq} / \text{L}$ in females among controls.

Distribution of cases and controls in relation to serum potassium:

Serum potassium in the study population varied from 3.1 to 5.2 mmol / L and in the control from 3.8 to 4.8 mmol / L. The mean and standard deviation of serum potassium among cases was $4.03 \pm 0.49 \text{ mmol}$ / L while in the control group, it was $4.29 \pm 0.33 \text{ mmol}$ / L respectively. This table clearly shows that the serum potassium level was significantly lower among the hypertensive population studied.

Serum potassium level in cases and controls

	Cases		Controls	Controls		Statistical
					value	Test
Serum	Mean	SD	Mean	SD		TWO way
	3.55	0.29	4.67	0.22		
Potassium					< 0.0001	ANOVA

Data are expressed as mean and standard deviation. The table above clearly shows that the serum potassium levels were significantly lower among the hypertensive population with a 'p-value of <0.0001.

Serum potassium in relation to gender:

The mean value of serum potassium was $3.62 \pm 0.32 \text{ meq} / \text{L}$ in males and $3.48 \pm 0.26 \text{ meq} / \text{L}$ in females among cases. The mean value of serum potassium was $4.68 \pm 0.16 \text{ meq} / \text{L}$ in males and $4.67 \pm 0.25 \text{ mmol} / \text{L}$ in females among controls.

Distribution of cases and controls in relation to serum sodium :

Serum sodium in the study population varied from 138 to 152 meq / L and in the control from 136 to 150 meq / L. The mean and standard deviation of serum sodium among cases was $147.94 \pm 2.88 \text{ meq}$ / L, while in the control group, it was $138.86 \pm 3.44 \text{ meq}$ / L respectively. This table clearly shows that the serum sodium level was significantly more among the hypertensive population studied.

Serum Sodium levels in cases and controls

	Cases	Cases			Р
					value
Serum Sodium	Mean	SD	Mean	SD	
	147.94	2.88	138.86	3.44	
					< 0.0001

Data are expressed as mean and standard deviation. The table above clearly shows that the serum sodium levels were significantly more among hypertensives witha 'p-value of <0.0001.

Discussion:-

Serum sodium among Hypertensives:

In our part of the country, there is an excessive intake of dietary salt. But despite that, not everyone has primary hypertension. The rarity of hypertension amongthose consuming a large amount of salt may probably be related to chronic adaptation of **b**ody 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 plays an important role in the pathogenesis of essential hypertension. 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 in the control group even though both were within the normal range. The mean and standard deviation of serum sodium among cases was 147.94 ± 2.88 meq/L while in the control group, it was 138.86 ± 3.44 meq/L respectively. A study was carried out by Lever et al on arterial pressure and body content of electrolytes in 91 patients with essential hypertension and 121 normal controls. In another study conducted by Williams et al, they studied the relationship of body sodium, chlorine and potassium in 30 patients with essential hypertension. They found that a positive correlation exists between serum sodium and blood pressure in this study group. In another study conducted by Bulpitt, two thousand, three hundred and twenty-eight men and 1496 women between the ages of 35 and 64 years were screened for hypertension and their plasma sodium and potassium concentrations were measured. It was found that plasma sodium was positively related to that blood pressure and an increase in serum sodium of 1 mmol/L was associated with an increase of 1 mm of Hg in both men and women

Serum potassium among Hypertensives:

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 $3.55 \pm 0.29 \text{ meq/L}$. The mean potassium in the control group was $4.67 \pm 0.22 \text{ meq/L}$.

Similarly, a study was conducted at the National Institute of Public Health and Environmental Protection, Bilthoven, The Netherlands. The relationships between the serum cations sodium, potassium, calcium and magnesium and blood pressure were investigated in a population-based sample of 182 Dutch persons aged 20-59 years. In the combined analysis, a weak inverse relationship was found between serum potassium and diastolic blood pressure; this relationship was also found in women. In another study carried out in Karachi, Pakistan, thirty hypertensive diabetic patients and an equal number of age and sex-matched controls were taken for the study. The mean serum potassium was 4.59 mmol / 1 among the study group and 5.03 mmol / 1 among the control group. To investigate the role of potassium on blood pressure Luft et al, conducted a study among 431 normotensive and 478 hypertensive subjects. They observed an inverse relationship between serum potassium and blood pressure supporting our study (21)

Conclusion:-

Primary hypertension is the major risk factor for coronary, cerebral and renal vascular diseases. The aetiology of primary hypertension is not known. Many theories were postulated. The present study attempts to focus on the serum sodium and potassium level among isolated newly diagnosed primary hypertensives who were free from any other illnesses or under any medication and to correlate the electrolyte status with the blood pressure. Serum sodium and potassium levels were estimated (using a flame photometer) in thirty-five hypertensives (m=17, f=18; mean age 56.5 \pm 12.2) and fifteen healthy controls (m=5 f=10; mean age 49.6 \pm 10.8). Body mass index was significantly more in those with hypertension. However, it was independent of gender. Mean serum sodium level was elevated significantly ('p-value = <0.0001) among hypertensives whereas serum potassium level was significantly lower among them when compared to healthy controls ('p-value = <0.0001). The blood pressure also correlated positively with serum sodium and body mass index whereas negatively correlated with serum potassium. Changing lifestyles have modified food habits, making people consume food rich in sodium but low in potassium. As a result genetically susceptible populations when exposed to high sodium content coupled with low potassium in their diet, hypertension becomes overt. The possible mechanisms were discussed.

Conflict Of Interest:

None.

References:-

1.Berglund G, Anderson O, Wellebonsa L. Prevalence of primary and secondary hypertension studies in a random population sample. Br. Med Jr 1976; 2: 554.

2. Gupta R. Trends in hypertension epidemiology in India. J Human Hypertension 2004; 18: 73-78.

3.Gupta R, Al-Udat NA, Gupta UP. Hypertension epidemiology in India: Meta- analysis and fifty year prevalence rates and blood pressure trends. J Human Hypertens1996; 10: 465-472.

4.Harrap SP: Hypertension: Genes versus environment. Lancet 1994; 344: 169.

5. Pratt RE, Dzau VJ: Genetics and hypertension concepts, potential and opportunities. Hypertension. 1993; 33: 238.

6. Lifton RP, Gnaravi AG, Geller DS: Molecular mechanisms of human hypertension(review). Cell 104: sys, 2001.

7.Law CM, Sheil AW, Newsone LA. Fetal, infant, and childhood growth and adult blood pressure. A longitudinal study from birth to 22 years of age. Circulation 2002; 105: 1088.

8. Brenner BM, Nertow CM: Congenital oligonephropathy. An inborn cause of adult hypertension and progressive renal injury? Curr Opin Nephrol Hypertens 1993; 2: 094.

9. Lever AF, Harrap SB: Essential hypertension: A disorder of growth with origins inchildhood? J Hypertens 1992; 10: 101.

10.Pries AR, Seromb TW, Gaentgens P: Structural auto regulation of terminal vascular beds: Vascular adaptation and development of hypertension. Hypertension 1999; 33: 153.

11.Esler M, Rumantir M, Lambert G et al: The sympathetic neurobiology of essential hypertension. Am J Hypertens 2001; 14 (suppl): 139s.

12.Brunner HR, Sealy JE, Laragh JH: Renin subgroups in essential hypertension.

Circ Res 1973; 32 (suppl): 99.

13.Liese AD, Mayer-Davis EJ, Haffner SM: Development of multiple metabolic syndrome: An epidemiologic prospective. Epidemiol Rev. 1998; 20: 157.

14.Consentino F, Luschor TF: Effects of blood pressure and glucose on endothelialfunction. Curr Hypertens Rep 2001; 3: 79.

15.Ascherio A, Henekens C, Willet WC. Prospective study of nutritional factors, blood pressure and hypertension among US women. Hypertension 1998; 27: 1065.

16.Sealey JE, Blumenfeld JR, Bell GM. On the renal basis for essential hypertension:

Nephron heterogeneity with discordant rennin secretion and sodium excretion causing a hypertensive vasoconstriction – volume relationship. J Hypertens 1988; 6: 763.

17.Guyton AC: Kidneys and fluids in pressure regulation. Small volume but large pressure changes. Hypertension. 1992: 19 (suppl): 2.

18. Aperia A: regulation of sodium / potassium ATPase activity. Curr Hypertens Resp2001; 3: 165.

19. Richards AM: The atrial natriuretic peptides and hypertension. J Inter Med. 1998; 235: 1284.

20.Schunkert H, Koenig W, Brockel U. Hematocrit profoundly affects left ventricular diastolic filling as assessed by Doppler echocardiography. J. Hypertens 2000; 18:1483.

21.Weinberger MH, Fireberg NS, Grim CE. Effects of volume expansion and concentration on potassium homeostasis in normal and hypertensive humans. J Am Coll Nutr. 1985; 5(4): 357-69.