JMSCR Vol||05||Issue||01||Page 15809-15825||January 2017

www.jmscr.igmpublication.org
Impact Factor 5.84
Index Copernicus Value: 83.27
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v5i1.93

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

A Study Onserum Sodium and Potassium Level in Essential Hypertension

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Abstract

Background: *Hypertension remains the most common readily identifiable and reversible risk factor for myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection and peripheral arterial disease. Because of the escalating obesity and population aging, the global burden of hypertension is rising and it is estimated to affect 1.5 billion persons – ie, one third of the world's population-by the year 2025. Currently, high blood pressure (BP) causes about 54% of stroke and 47% of ischemic heart disease worldwide.*

Half of this disease burden is in people with hypertension; the other half is in people with lesser degrees of high BP (prehypertension). Thus, high BP remains the leading cause of death worldwide and one of the world's great public health problems.

In 90% to 95% of hypertensive patients, a single reversible cause of the elevated BP cannot be identified, hence the term essential hypertension. In the remaining 5% to10%- cases denoted secondary or identifiable hypertension- a more discrete mechanism can be identified.

Materials And Methods: *The study was conducted in medicine OPD and wards of Kanyakumari Government Medical College, Kanyakumari. It is an Analytical type of study. The study period was from January2016-December 2016. A total of 100cases (50 cases and 50 controls) were studied. The present project was submitted in Institutional Ethical Committee and its approval was obtained. The participants were taken up after getting a written informed consent from them.*

Inclusion Criteria:

1. *Patients with essential hypertension.*
2. *Age above 30 yrs.*
3. *Both sexes were included.*

Exclusion Criteria:

1. *Patients below 30 years.*
2. *Patients with diabetes mellitus.*
3. *Patients with renal failure / active urinary sediment / significant proteinuria.*
4. *Pregnancy*

5. *Patients whose BP shows significant disparity between right and left arm or between upper limbs and lower limbs*
6. *Patients with bruit in renal arteries*
7. *Patients with peripheral vascular diseases*
8. *Patients admitted with features of malignant hypertension, hypertensive encephalopathy, flash pulmonary edema and other hypertensive emergencies.*
9. *Patients with acute diarrhoeal disease.*
10. *Patients on NSAIDS, anti-hypertensives, diuretics, oral contraceptives, beta blockers or agonists.*

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. Patient's hip and waist circumferences were measured. 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.

Observation: *The total number of subjects included in this study was 100. Among these 100 subjects, 50 were cases (hypertensive) and 50 were controls (normotensive).*

Results: *Serum sodium was significantly more among hypertensives and it was independent of associated risk factors and gender& also correlated positively with the level of blood pressure. Serum potassium was significantly less among hypertensives and it correlated negatively with blood pressure.*

Introduction

Hypertension currently is defined as a usual BP of 140/90mm Hg or higher, for which the benefits of drug treatment have been definitively established in randomized placebo-controlled trials.

JNC 8 Classification of Blood Pressure for Adults ≥ 18 years

SI. No	AGE&CO-MORBIDITY	Systolic BP		Diastolic BP
1.general Population with no DM or CKD	AGE< 60	<140	And	<90
	AGE>/=60	<150	And	<90
2.ALL AGES	WITH DM; NO CKD	<140	And	<90
3.all ages and races	with CKD present with or without DM	>140	and	<90

Guidelines for measuring blood pressure:

I Conditions For the Patient

A. Posture:

1. 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, diabetic or receiving anti – hypertensive theraphy, check for postural changes by taking readings immediately and 2 minutes after the patient stands.

B. Circumstances:

1. No caffeine for preceding hour
2. No smoking for preceding 15 minutes.
3. No exogenous adrenergic stimulants like phenylephrine in nasal decongestants or eye drops for papillary dilation.
4. A quite, warm setting.
5. 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. If not, place the bladder over the brachial artery; if bladder is small spuriously high readings may result.

III Technique

A. Number of readings

1. 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.
2. For diagnosis, obtain at least three sets of readings a week apart.
3. Initially, take pressure in both arms, if pressure differs, use arm with higher pressure.
4. If arm pressure is elevated, take pressure inone leg, particularly inpatients below age 30.

B. Performance

1. Inflate the bladder quickly to a pressure 20mm Hg above the systolic, as recognized by the disappearance of the radial pulse.
2. Deflate the bladder 3 mm Hg every second.
3. Record the Korotkoff phase V (disappearance) except in children, in whom use of phase IV (muffling) is advocated.
4. 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, light 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 urea was done by using Diacety1 monoxime (DAM) technique. Serum creatinine was estimated using COBAS auto analyser. Serum sodium and potassium was estimated using Flame emission photometric method.

Essential Hypertension

Hypertension was defined in accordance to the JNC- 8 REPORT AS SYSTOLIC BLOOD PRESSURE 140MM 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.

Sodium and Potassium Normal Values

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

Diabetes Mellitus

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

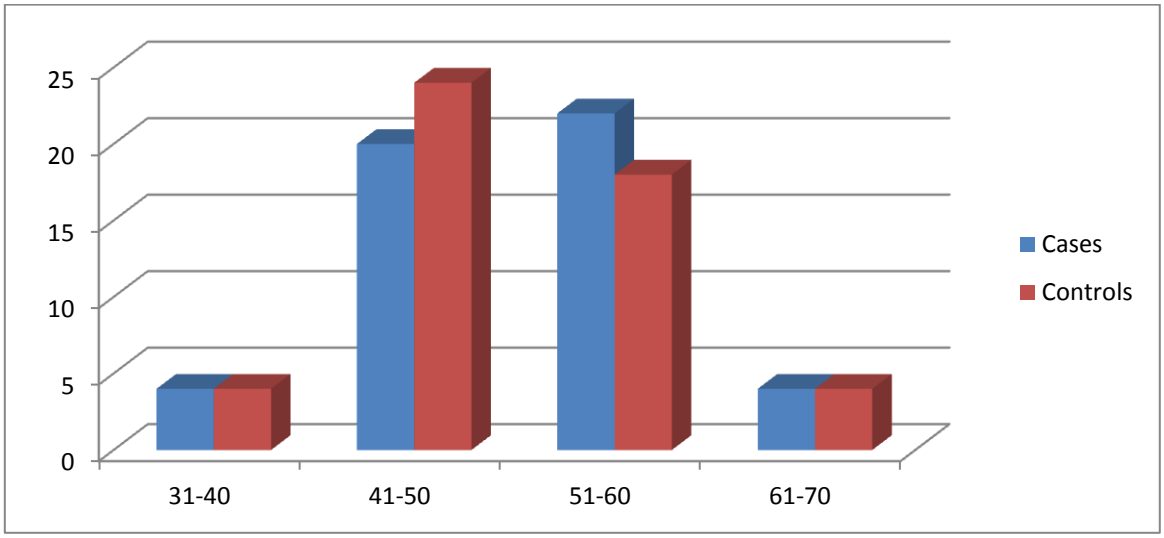
Analysis of cases and controls with respect to age

The age of the subjects in the study group ranged from thirty to sixty five years. The mean and standard deviation for the age of the cases and controls were 51.6 +- 6.82 and 50.8 =- 6.59 years respectively. The study group and the control group did not differ from each other statistically with reference to age. The distribution of the cases and controls in relation to age is provided in the Table – IV and fig – 1 given below

Age distribution of cases and controls: (Table IV)

Age group	Cases		Controls	
	No.	%	No	%
31-40	4	8	4	8
41-50	20	40	24	48
51-60	22	44	18	36
61-70	4	8	4	8
Mean	51.6		50.8	
SD	6.82		6.59	

Age distribution of cases and controls (FIG – 1)



Majority of the patients in both the study and control group lie between 41 and 60 years[7]. There was no significant difference in the age composition of those with and without hypertension in this study. Almost same age group of patients was selected in both groups.

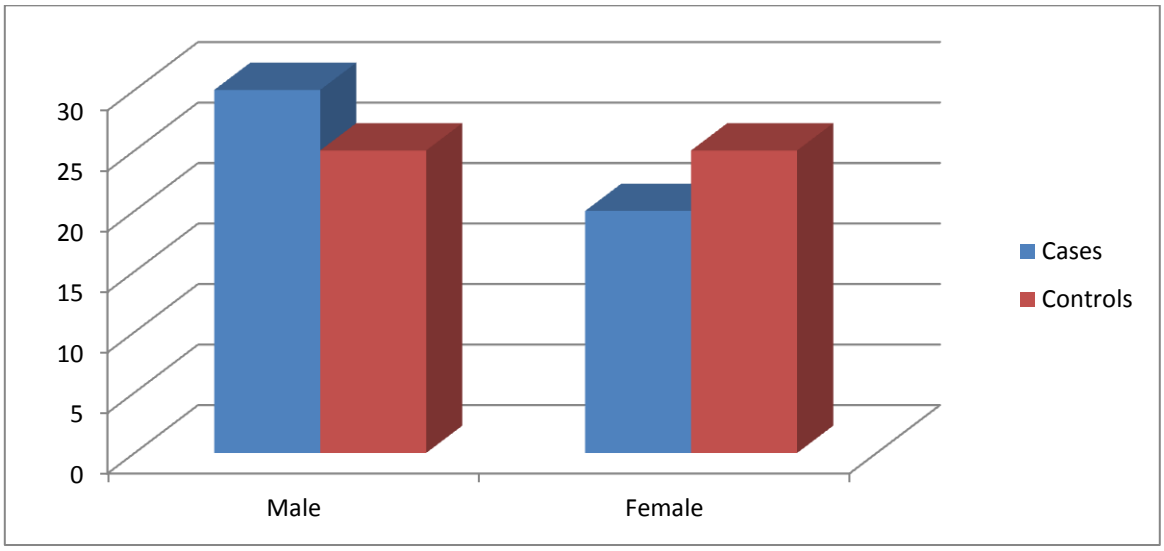
Gender wise distribution of cases and controls

Among the 70 cases studied, there were 38 males and 32 females. Among the 30 controls, there were 20 males and 10 females. The mean age distribution for the males in the case and control groups was 51.4 +- 7.2 years and 52.1 =- 6.7 years respectively. The mean age distribution for the females in tehc case and control groups was 51.8 +- 6.39 years and 49.4 +- 6.33 years respectively. The details are given in the Table –V provided below and shown in fig – 2

Gender wise distribution of cases and controls : (Table V)

sex	Cases		Controls	
	No	%	No	%
Male	30	60	25	50
Female	20	40	25	50
Total	50	100	50	100

Gender wise distribution of cases and controls (Fig-2)



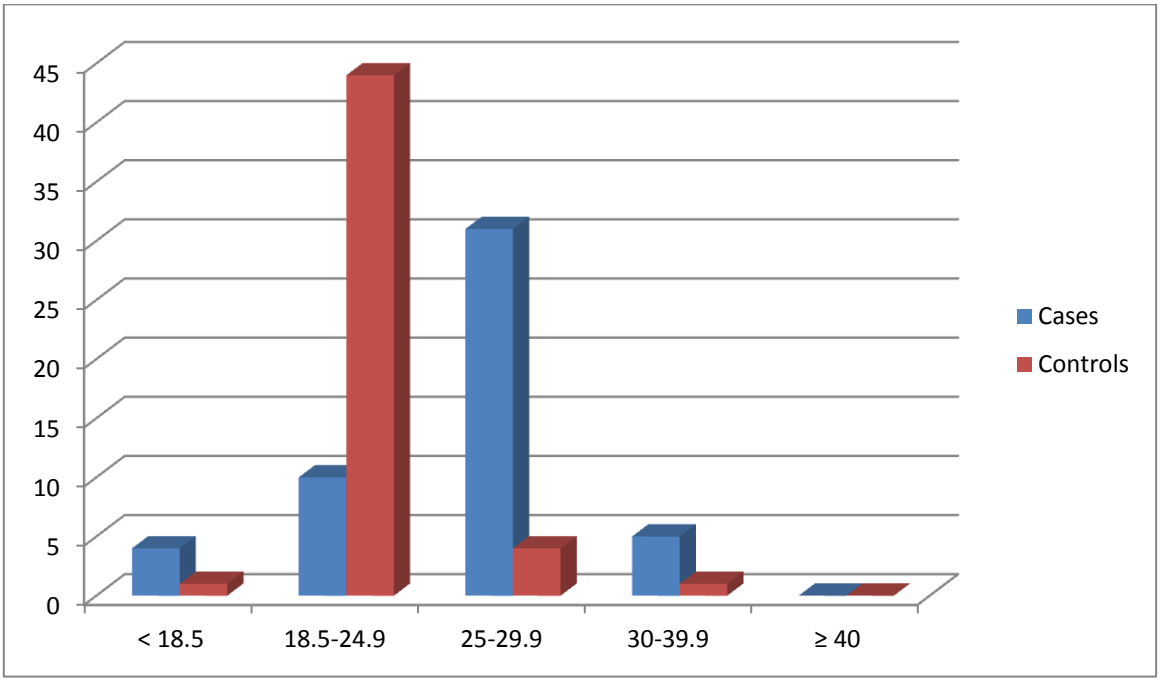
Distribution of cases anc controls with respect to BMI

62% of the cases were overweight and 10% cases were obese, whereas only 8% of controls were overweight and 2% cases were obese. Details were shown in table VI and FIG – 3 below.

Distribution of cases and controls with respect to BMI (Table VI)

BMI	Cases		Controls	
	No	%	No	%
Underweight < 18.5	4	8	1	2
Healthy weight 18.5-24.9	10	20	44	88
Over weight 25-29.9	31	62	4	8
Obesity 30-39.9	5	10	1	2
Extreme obesity ≥ 40	0	0	0	0
Total	50	100	50	100

Distribution of cases and controls with respect to BMI (FIG -3)



BMI among cases and controls

The mean body mass index in the case group is 26.1 +- 3.64 and in the control group is 23 +- 2. The details are given in the Table - VII given below (fig -4).

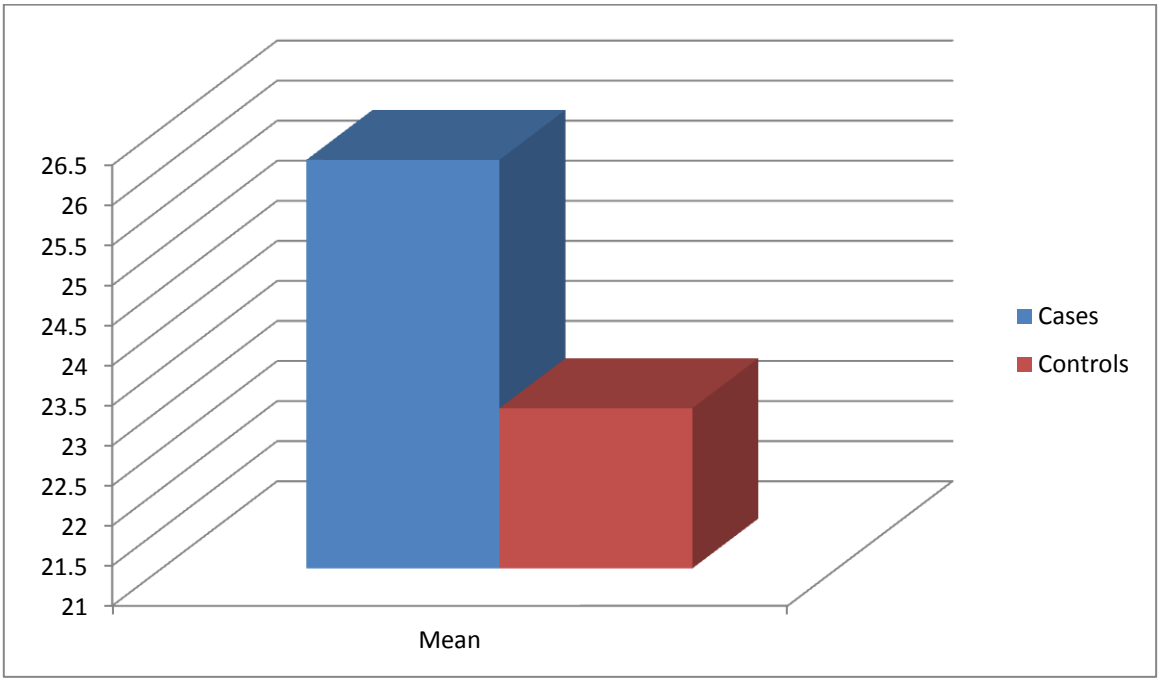
BMI among cases and controls (Table VII)

BMI	Cases	Controls
Mean	26.1	23
SD	3.64	2

P value < 0.0001

This shows that the difference in Body Mass Index between cases and controls was statistically significant.

BMI among cases and controls L (FIG -4)



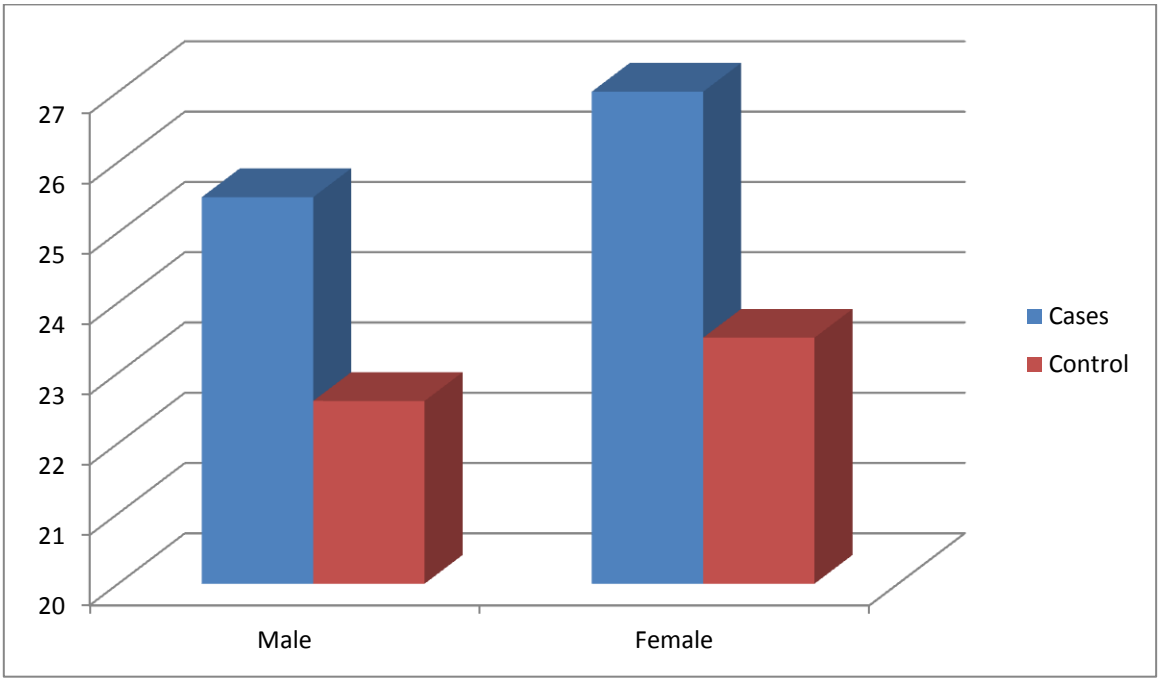
BMI of cases and controls with respect to gender

The mean BMI of cases and controls according to gender is given in the Table – VIII below (fig-5)

BMI of cases and controls with respect to gender: (Table VIII)

Group	Male		Female	
	Mean BMI	SD	Mean BMI	SD
Cases	25.5	4.03	27	2.8
Controls	22.6	2.29	23.5	1.57

BMI of cases and controls with respect to gender: (FIG – 5)



Body mass index was independent of gender and electrolyte status.

Analysis of presenting symptoms

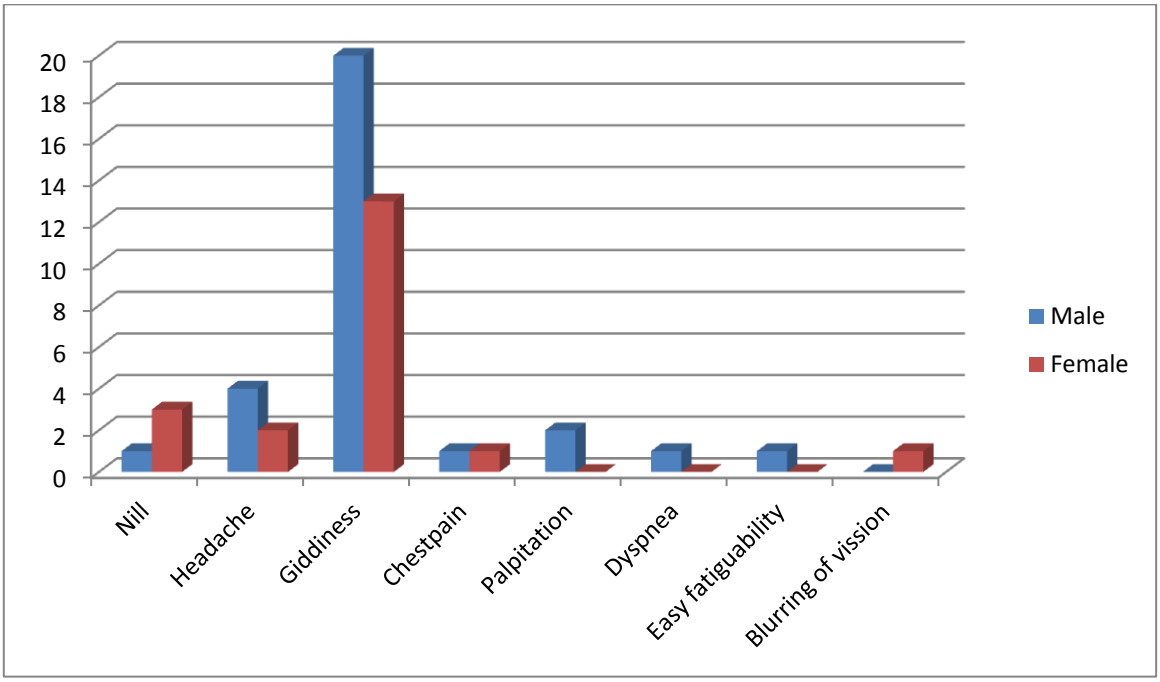
The most common presenting symptom among cases is giddiness. Other symptoms were in the order of headache, palpitation, dyspnea, chest pain, easy fatiguability[4].

The details of the presenting symptoms are given in the Table-X given below (fig-7)

Analysis of presenting symptoms: (Table-X)

Symptoms	Male		Female	
	No	%	No	%
No symptoms	1	2	3	6
Headache	4	8	2	4
Giddiness	20	40	13	26
Chest pain	1	2	1	2
Palpitation	2	4	0	0
Dyspnea	1	2	0	0
Easy fatiguabiliity	1	2	0	0
Blurring of vision	0	0	1	2

Analysis of presenting symptoms: (fig-7)



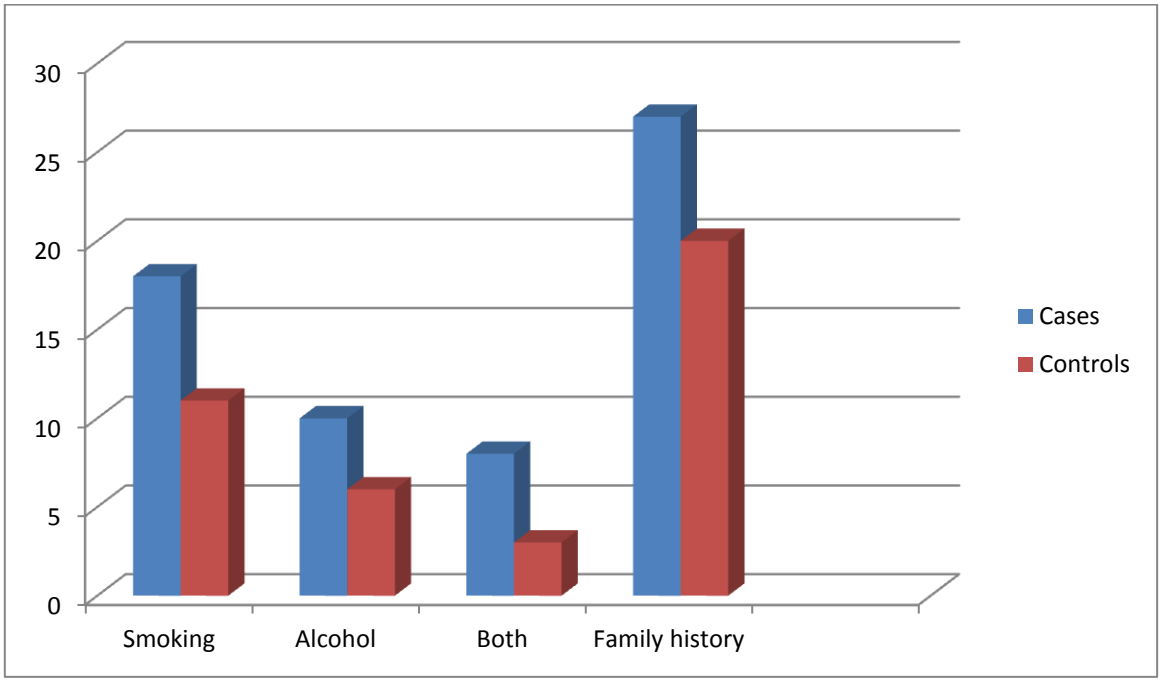
Risk Factor Analysis

Analysis of other risk factors like smoking, alcoholism and family history were done among hypertensive. Their details are furnished in the Table-XI and figure-8 below.

Risk factor analysis: (Table - XI)

	Smoking	Alcohol	Both	Positive family history
Cases	18	10	8	27
Controls	11	6	3	20

Risk factor analysis (Fig - 8)



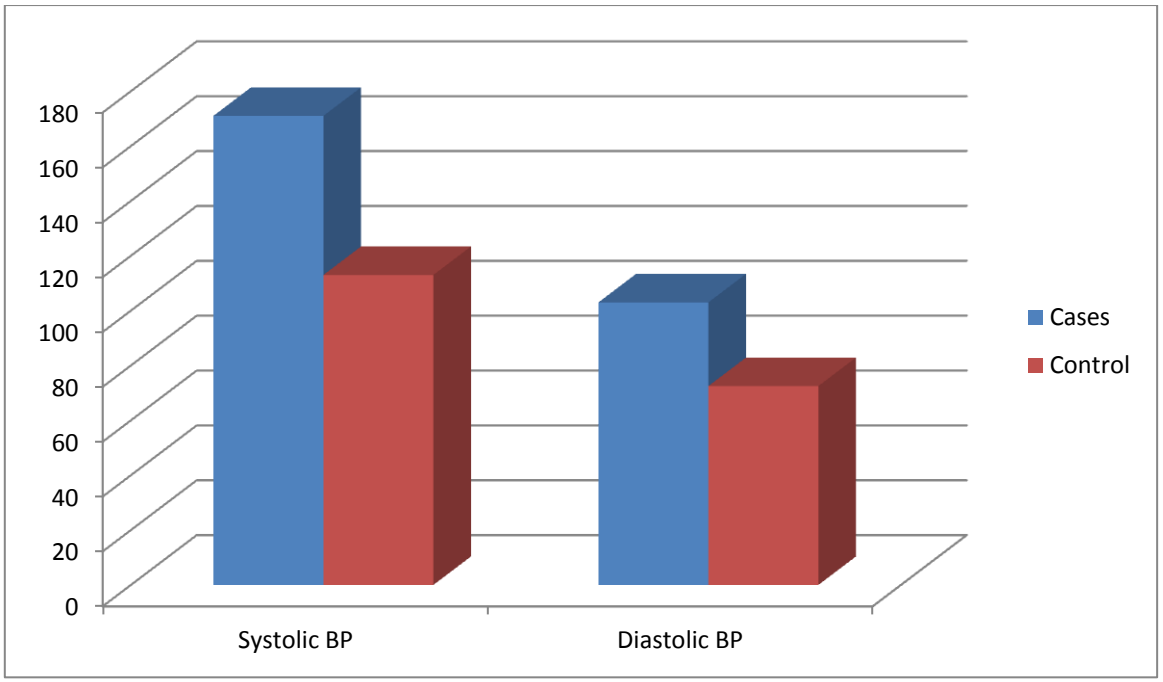
Distribution of systolic and diastolic blood pressures

The details of the blood pressure distribution are given in the table –XII given below (fig-9).

Distribution of systolic and diastolic blood pressures: (Table XII)

Blood pressure	Cases		Controls	
	Mean	SD	Mean	SD
Systolic	171	14.7	113	5.94
Diastolic	103	7.89	72.5	4.08

Distribution of systolic and diastolic blood pressures (fig -9)



The mean systolic blood pressure for the cases was 171 ± 14.7 mm Hg. Similarly the mean diastolic blood pressure for the cases was 103 ± 7.89 mm Hg. Since the systolic and diastolic blood pressure was elevated in cases and it was due to the nature of the disease taken into study, the statistical analysis was not done.

Serum sodium levels in cases and controls

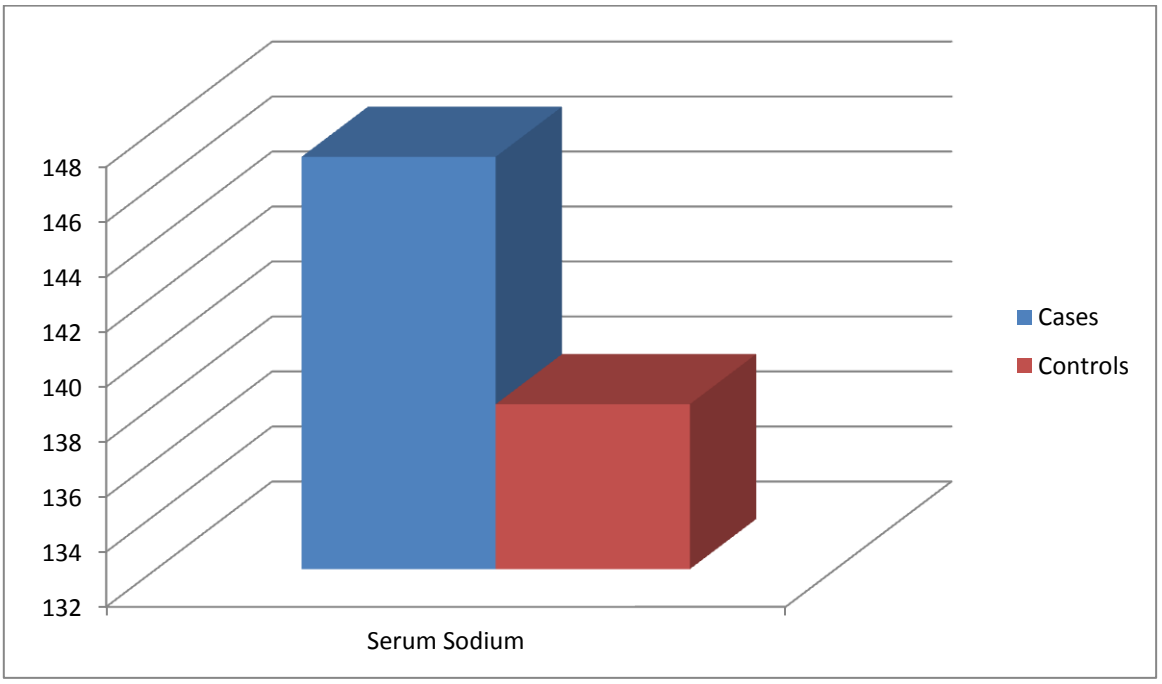
Serum sodium in the study population varied from 139 to 153 mmol / L and in the control from 136 to 144 mmol /L. The mean and standard deviation of serum sodium among cases was 147 ± 3.19 mmol / L while in the control group it was 138 ± 1.8mmol / L respectively. This table clearly shows that the serum sodium level was significantly more among hypertensive population studied.

The details are shown in the table – XIII given below (fig-10).

Serum sodium levels in cases and controls: (Table XIII)

Serum sodium	Cases		Controls		P value
	Mean	SD	Mean	SD	
	147	3.19	138	1.8	<0.0001

Serum Sodium levels in cases and controls (FIG-10)



Serum sodium in relation to gender

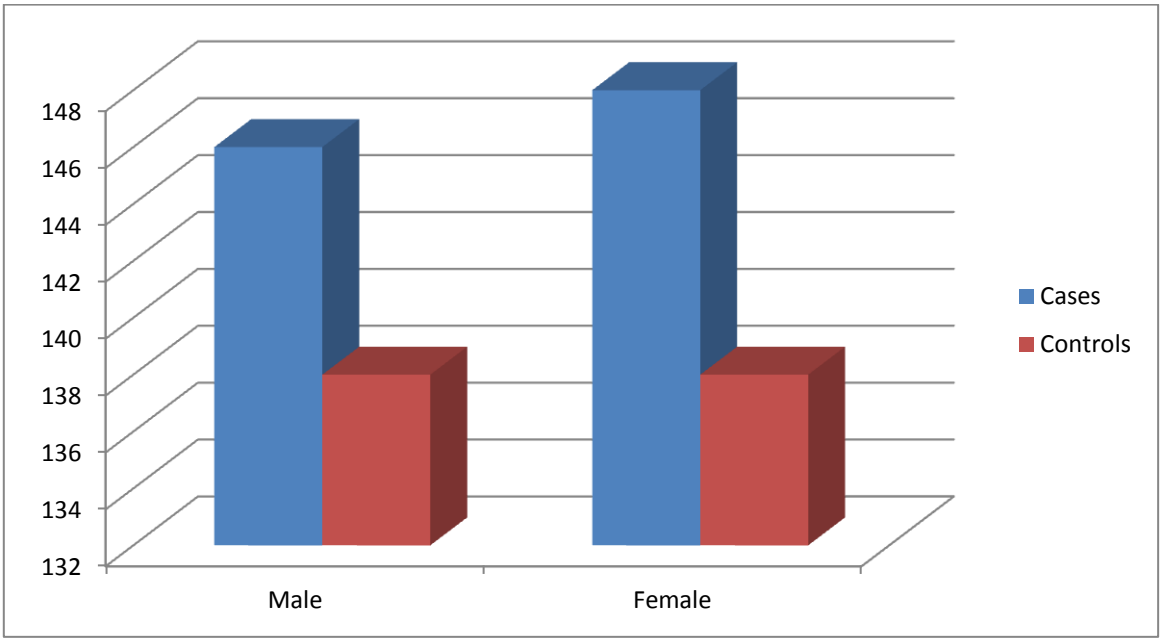
The mean value of serum sodium was 146 ± 2.81mmo / L in males and 148 ± 4.2 mmo/ L in females among cases. The mean value of serum sodium was 138 ± 3.1 mmol / L in females among controls.

This is shown in Table- XIV and (fig -11) given below.

Serum sodium in relation to gender (Table XIV)

Sex	Case	Control
Male	146	138
Female	148	138

Serum sodium in relation to gender (fig - 11)



Serum potassium level in case and controls

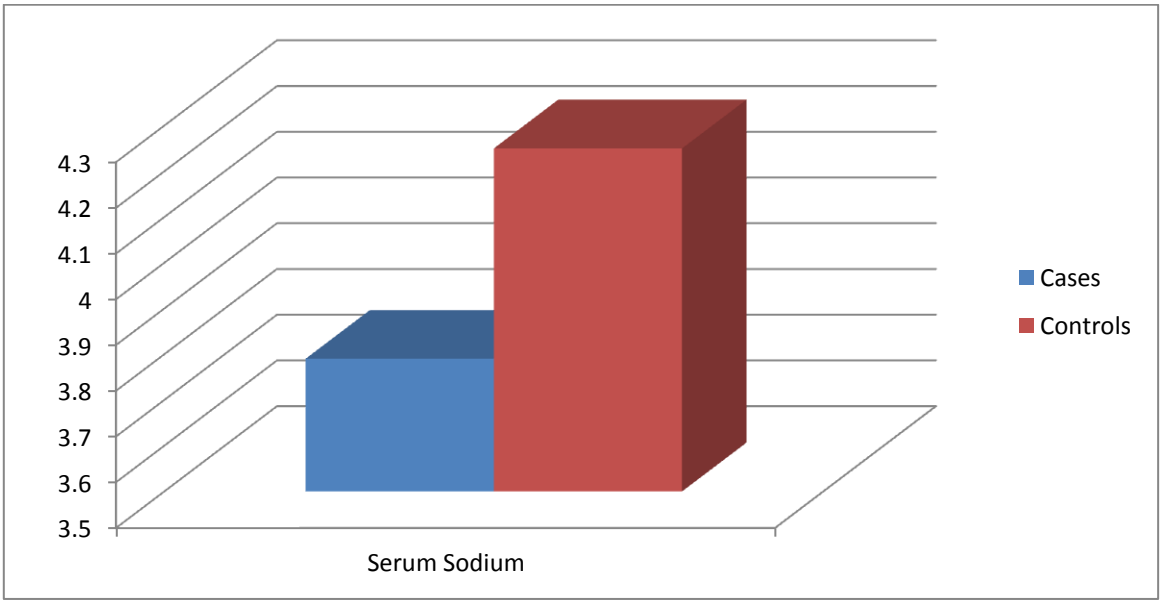
Serum potassium in the study population varied from 3.3 to 4.5 mmol / L and in the control from 3.8 to 4.6 mmol / L. The mean and standard deviation of serum potassium among cases was 3.79 ± 0.179 mmol / l while in the control group it was 4.25 ± 0.223 mmol / l respectively. This table clearly shows that the serum potassium level was significantly lower among the hypertensive populated studied.

The details are given in Table –XV given below (fig -12).

Serum sodium in relation to gender (Table XV)

Serum Sodium	Cases		Controls		P value
	Mean	SD	Mean	SD	
	3.79	0.179	4.25	0.223	<0.0001

Serum sodium in relation to gender (FIG -12)



Serum potassium values in relation to gender

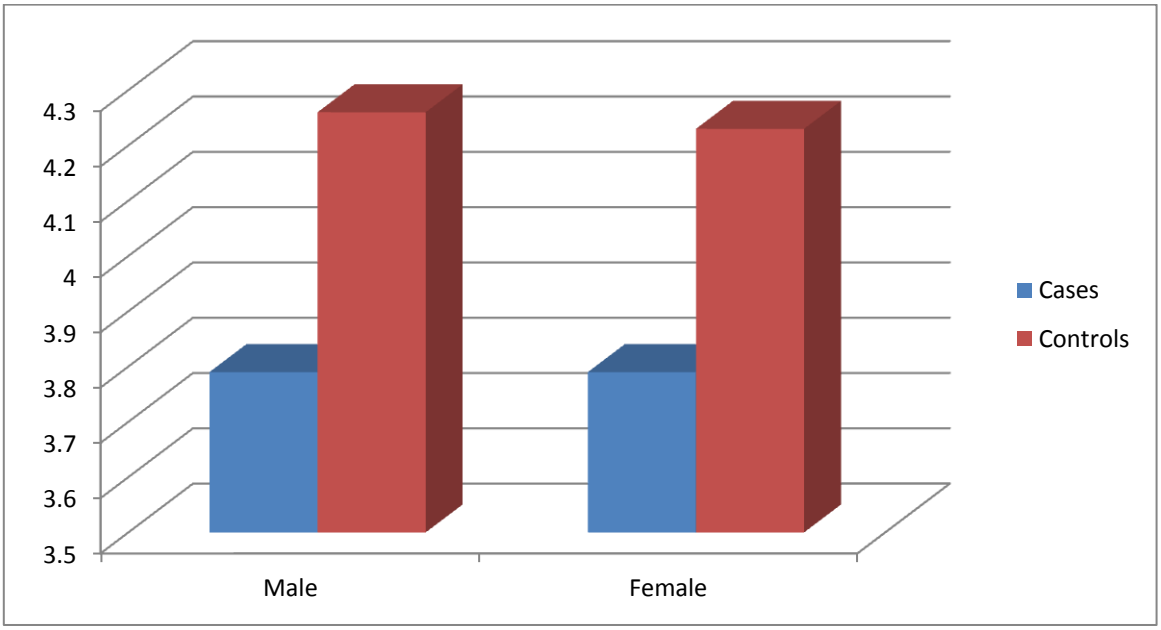
The mean value of serum potassium was 3.79 ± 0.182 mmol / L in males and 3.79 ± 0.224 mmol/ L in females among cases. The mean value of serum potassium was 4.26 ± 0.231 mmol / L in males and 4.23 ± 0.219 mmol / L in females among controls.

This is shown in Table – XVI given below (fig - 13).

Serum potassium values in relation to gender (Table XVI)

Sex	Cases	Controls
Male	3.79 ± 0.182	4.26 ± 0.231
Female	3.79 ± 0.224	4.23 ± 0.219

Serum potassium values in relation to gender (fig - 13)



Urinary analysis, Blood glucose, blood urea and serum creatinine were well within acceptable limits and did not differ from healthy control.

Electrocardiogram revealed left ventricular hypertrophy in about 24 percent of study group, left atrial enlargement in 22% and left anterior hemiblock in 2%.

Chest X-ray showed cardiomegaly in about 42 percent of cases.

Ocular fundus examination revealed hypertensive retinopathy in about 30 percent of the study group.

Discussion

Hypertension affects approximately 25% of the adult population worldwide, and its prevalence is predicted to increase by 60% by 2025, when a total of 1.56 billion people may be affected[1,5]. It is the major risk factor for cardiovascular disease and is responsible for most deaths worldwide. Primary hypertension, also known as essential or idiopathic hypertension, accounts for as many as 95% of all cases of hypertension. Primary hypertension results from the interplay of internal derangements (primarily in the kidney) and the external environment. Sodium, the main extracellular cation, has long been considered the pivotal environmental factor in the disorder.

Sodium in hypertension

Since sodium is largely restricted to the extra cellular compartment, total body sodium content is a reflection of the extra cellular fluid volume. Sodium is actively pumped out of cells by the Na+ k+ ATPase pump. As a result, 85 to 90% of all sodium is extra cellular, and the extra fluid volume is a reflection of total body sodium

content. Changes in sodium concentration generally reflect disturbed water homeostasis, whereas alterations in sodium content are manifest as extra cellular fluid volume contraction or expansion and imply abnormal sodium balance[10].

Tubule sodium reabsorption is the major regulatory mechanism controlling sodium excretion. Almost two-thirds of filtered sodium is reabsorbed in the proximal convoluted tubule. Further reabsorption (25 to 30%) occurs in the thick ascending limb of the loop of Henle via the apical Na+-k+- 2cl— co transporter. Distal convoluted tubule reabsorption of sodium (5%) is mediated by the thiazide-sensitive Na+-cl—co transporter. Final sodium reabsorption being reasonably equivalent to the amount ingested per day.

The renal sympathetic nervous system directly stimulates sodium reabsorption and renin release from the juxtaglomerular apparatus. Several studies have linked nervous system hyperactivity with greater than normal increases in blood pressure in response to a given sodium load.

Most authorities believe that the mechanism by which the kidney causes hypertension is impairment in the excretion of sodium this impairment may be related to genetic changes in various sodium exchangers in the proximal and distal tubules that result in altered responses to stimulation by the sympathetic nervous system and the renin angiotensin aldosterone system.

An increase in cytosolic free sodium concentration in cells of hypertensive patients compared with age and sex matched normotensive controls have been documented.

This results from altered activity of the Na+ /H+ antiporter and the Na + /Li+ counter transporter. This increase in intra cellular sodium is highly correlated with the presence of an elevated diastolic blood pressure.

Most patients with essential hypertension have a defect in the pressure natriuresis curve, in which higher systemic pressures are required to excrete a sodium load [2].

Another mechanism for decreased sodium excretion in patients with essential hypertension is an enhancement of tubule glomerular feedback.

Alterations in intrarenal vasoactive mediators may be involved in the impairment of sodium excretion in patients with essential hypertension. There may be low levels of renal vasodilators, such as Prostaglandins, dopamine, and nitric oxide as well as elevated levels of renal vasoconstrictors such as angiotensin II and adenosine and increased activity of the renal sympathetic nervous system.

Alterations in the levels of these agents could contribute to net sodium reabsorption because of their direct effects on tubular sodium transport.

Potassium and Hypertension

Potassium is the major intracellular cation. The normal plasma potassium concentration is 3.5 to 5mmol / L, whereas that inside the cells is about 150mmol / L. therefore, the amount of potassium in the extra cellular fluid is less than 2% of the total body potassium content[14].

The intake of individuals on an average western diet is 40 to 120mmol/d 90% of which is absorbed by the gastrointestinal tract. Immediately following a meal, most of the absorbed potassium enters cells as a result of the initial elevation in the plasma potassium concentration and facilitated by the insulin release and basal catecholamine levels.

Renal excretion is the major route of elimination of dietary and other sources of excess potassium. 90% of the filtered potassium is reabsorbed by the proximal convoluted tubule and the loop of Henle. Proximally, Potassium is reabsorbed passively with sodium and water, whereas the luminal Na+ - K+ -2Cl – co transporter medicates potassium uptake in the thick ascending limb of the loop of Henle. Therefore, potassium delivery to the distal nephron approximates dietary intake. The cell responsible for potassium secretion in the late distal convoluted tubule and cortical collecting duct is the principal cell.

Consequences of hypertension

Heart

Heart disease is the most common cause of death in hypertensive patients[6]. Hypertensive heart disease is the result of structural and functional adaptations leading to

1. Left ventricular hypertrophy.
2. CHF
    a. Diastolic dysfunction.
    b. systolic dysfunction.
3. Abnormalities of blood flow due to atherosclerotic coronary artery disease.
4. Micro vascular disease
5. Cardiac arrhythmias.

Individuals with left ventricular hypertrophy are at increased risk for CHD, stroke, CHF, and sudden death. Aggressive control of hypertension can regress or reverse left ventricular hypertrophy and reduce the risk of cardiovascular disease.

CHF may be related to systolic dysfunction, diastolic dysfunction, or a combination of the two. Abnormalities of diastolic function that range from asymptomatic heart disease to overt heart failure are common in hypertensive patients. Patients with diastolic heart failure have a preserved ejection fraction, which is a measure of systolic function. Approximately one-third of patients with CHF have normal systolic function but abnormal diastolic function. Diastolic dysfunction is an early consequence of hypertension-related heart disease and is exacerbated by left ventricular hypertrophy and ischemia.

Brain

Stroke is the second most frequent cause of death in the World; it accounts for 5 million deaths each year, with an additional 15 million persons having nonfatal strokes. Elevated blood pressure is the strongest risk factor for stroke. Approximately 85% of strokes are due to infarction, and the remainder are due to either intracerebral or subarachnoid haemorrhage.

The incidence of stroke rises progressively with increasing blood pressure levels, particularly systolic blood pressure in individuals >65 years. Treatment of hypertension convincingly decreases the incidence of both ischemic and hemorrhagic strokes.

Hypertension also is associated with impaired cognition in an aging population, and longitudinal studies support an association between midlife hypertension and late-life cognitive decline. Hypertension-related cognitive impairment and dementia may be consequence of a single infarct due to occlusion of a “strategic” larger vessel or multiple lacunar infarcts due to occlusive small vessel disease resulting in subcortical white matter ischemia.

Cerebral blood flow remains unchanged over a wide range of arterial pressures (mean arterial pressure of 50-150 mmHg) through a process termed auto regulation of blood flow. In patients with the clinical syndrome of malignant hypertension, encephalopathy is related to failure of auto regulation of cerebral blood flow at the upper pressure limit, resulting in vasodilation and hyper perfusion. Signs and symptoms of hypertensive encephalopathy may include severe headache, nausea and vomiting (often of a projectile nature), focal neurologic signs, and alterations in mental status. Untreated, hypertensive encephalopathy may progress to stupor, coma, seizures, and death within hours. It is important to distinguish hypertensive encephalopathy from other neurologic syndromes that may be associated with hypertension, e.g., cerebral ischemia, hemorrhagic or thrombotic stroke, seizure disorder, mass lesions, pseudotumorcerebri, delirium tremens, meningitis, acute intermittent porphyria, traumatic or chemical injury to the brain, and uremic encephalopathy.

Kidney

The kidney is both a target and a cause of hypertension. Primary renal disease is the most common etiology of secondary hypertension.

Mechanisms of kidney-related hypertension include a diminished capacity to excrete sodium, excessive renin secretion in relation to volume status, and sympathetic nervous system over activity.

Conversely, hypertension is a risk factor for renal injury and end-stage renal disease. The increased risk associated with high blood pressure is graded, continuous, and present throughout the distribution of blood pressure above optimal pressure. Renal risk appears to be more closely related to systolic than to diastolic blood pressure, and black men are at greater risk than white men for developing ESRD at every level of blood pressure. Proteinuria is a reliable marker of the severity of chronic kidney disease and is a predictor of its progression.

Patients with high urine protein excretion (>3 g/24h) have a more rapid rate of progression than do those with lower protein excretion rates.

Atherosclerotic, hypertension-related vascular lesions in the kidney primarily affect pre glomerular arterioles, resulting in ischemic changes in the glomeruli and post glomerular structures. Glomerular injury also may be a consequence of direct damage to the glomerular capillaries due to glomerular hyper perfusion. Studies of hypertension-related renal damage, primarily in experimental animals suggest that loss of auto regulation of renal blood flow at the afferent arteriole results in transmission of elevated pressures to an unprotected glomerulus with ensuing hyper filtration, hypertrophy, and eventual focal segmental glomerular sclerosis.

Clinically, micro albuminuria (a random urine albumin/creatinine ratio>300 mg/g) or micro albuminuria (a random urine albumin/creatinine ratio 30 300 mg/g) are early markers of renal injury. These are also risk factors for renal disease progression and cardiovascular disease.

Peripheral Arteries

In addition to contributing to the pathogenesis of hypertension, blood vessels may be a target organ for atherosclerotic disease secondary to long-standing elevated blood pressure. Hypertensive patients with arterial disease of the lower extremities are at increased risk for future cardiovascular disease. Although patients with stenotic lesions of the lower extremities may be asymptomatic, intermittent claudication is the classic symptom of PAD. This is characterized by aching pain in the calves or buttocks while walking that is relieved by rest. The ankle-brachial index is a useful approach for evaluating PAD and is defined as the ratio of noninvasively assessed ankle to brachial (arm) systolic blood pressure. An ankle-brachial index <0.90 is considered diagnostic of PAD and is associated with >50% stenosis in at least one major lower limb vessel. Several studies suggest that an ankle-brachial index <0.80 is associated with elevated blood pressure, particularly systolic blood pressure.

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 essential 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 essential hypertension.

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 the, 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)[8,9]. Such an amount of salt consumption present in our 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 147 ± 3.19 while in the control group it was 138 ± 1.38 respectively. Our study was supported by Jan et al (2006), 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 was 140 ± 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.

A study was carried out by lever et al arterial pressure and body content of electrolytes in 91 patients with essential hypertension and 121 normal controls.

Plasma and exchangeable sodium was found to be positively correlated with arterial pressure in the patients and plasma, exchangeable, and total body potassium correlated inversely with arterial pressure in the patients, the correlations being closest in young patients[3]. Three hypotheses were proposed to explain the mechanisms relating electrolytes and arterial pressure in essential hypertension.

Three hypotheses were proposed to explain the mechanisms relating electrolytes and arterial pressure in essential hypertension- namely.

1. Cell- salt hypothesis
2. Dietary salt hypothesis
3. Kidney –salt hypothesis

It was concluded that two mechanisms probably operate in essential hypertension.

In the early stages of the disease blood pressure is raised by an abnormal process related more closely to potassium than to sodium. A renal lesion develops later, possibly as a consequence of the hypertension. This lesion is characterised by resetting of pressure natriuresis and is manifest by an abnormal relation between body sodium and arterial pressure and by susceptibility to increased dietary sodium intake.

In another study conducted by Nanjiet al[12], it was shown that a positive correlation exists between serum sodium and hypertension.

A study was conducted among Japanese people by komiya et al. They studied 3222 normal Japanese subjects (610 in Kashiwa City Hospital and 2612 in Shinshu University hospital) 741 Japanese patients with essential hypertension (256 in Kashiwa City Hospital and 485 in Shinshu University Hospital) to determine the possible roles of sodium, renal function, and plasma aldosterone concentration (PAC) on blood pressure elevation. They found that the peak of the serum sodium distribution curve was approximately 2mmol / L higher in the hypertensive group as compared with that in the control group. The prevalence of higher serum sodium concentration (< or = 147mmol/ l) was also significantly higher in the hypertensive group.

In another study conducted by Bulpitt[13], 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 measured. It was found that plasma sodium and potassium concentrations measured. It was found that plasma sodium was positively related to that of blood pressure and an increase in serum sodium of 1mmol/ L was associated with an increase of 1 mm of Hg in both men and women.

In a study carried out at the University of Tokyo, they measured plasma electrolytes in 82 essential hypertensive patients to examine the relation between blood pressure and plasma electrolytes. Significant negative correlations were observed between plasma potassium concentration and 24-h systolic blood pressure (r=-0.336) and diastolic pressure (r=-0.298) in their patients.

Plasma potassium concentration inversely correlated also with both daytime and night time systolic and diastolic blood pressure. There was no relation between office blood pressure and plasma potassium concentration. These findings indicate that in essential hypertensives plasma potassium concentration is inversely related to ambulatory blood pressure including daytime and night time blood pressure and suggest that potassium may be a factor determining the whole day blood pressure[11]. 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.

BMI and Hypertension:

In our study the mean BMI among the study group was 26.1 ± 3.64 and among the control group was 23 ±2. The 'p' value was 0.0001. This shows that overweight and obesity also plays a role in the development of essential hypertension.

This was supported by a study conducted by Stamler[15]. They showed that the hypertension is about six times more common in obese than it is in lean subjects. The present study concurs with above observation. However body mass index was not related to electrolyte levels.

Similarly 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. This study showed a positive correlation between BMI and blood pressure which supported out study.

In INTERSALT, the relationship between body mass index (kg/m2) and blood pressure was studied in 10,079 men and women aged 20-59, sampled from 52 centres around the world, based on a standardized protocol with central training of observers, a central laboratory and extensive quality control. Body mass index-blood pressure relationships were first studied in men and women with each centre, and results of these regression analyses were then pooled for all 52 centres. With adjustment for age, alcohol intake, smoking, and sodium and potassium excretion, body mass index was positively associated with systolic blood pressure among men in 51 of 52 centres and among women in 47, significantly so in 24 and 27, respectively. Body mass index was positively associated with diastolic blood pressure in 51 and 49 centres in men and women, respectively, significantly so in 33 and 31. Overall, a 10 kg difference in body weight was associated on average with a 3.0 mmHg difference in systolic and a 2.2 mmHg difference in diastolic pressure[16]. In further analyses across centres, median body mass index was related significantly to median systolic blood pressure, median diastolic pressure and the prevalence of hypertension in both men and women body mass index was related to the slopes of systolic and diastolic blood pressure with age in women, but not in men.

Limitations

1. Only serum sodium and potassium were done.
2. 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.
3. Body water content was not assessed which may alter the sodium and potassium levels.
4. Tissue sodium and potassium was not measured nor correlated with serum sodium and potassium levels.
5. Hormones related to sodium and potassium handling in kidney was not estimated.
6. The salt intake of the patients could not be assessed quantitatively and qualitatively because of social constraints.

Conclusion

Serum sodium was significantly more among hypertensives 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 hypertensives 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 level (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 essential hypertension.

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