

# Assessment of Electrolyte Imbalance among Hypotensive Patients: A Hospital Based Study

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## ABSTRACT

**Background:** Epidemiologic, experimental, and clinical studies suggest that sodium and potassium are important regulators of blood pressure. Surveys conducted in widely divergent geographic locations indicate significant prevalence of hypotension in populations ingesting diets altered electrolyte levels. Hence; we planned the present study to assess electrolyte imbalance among hypotensive patients.

**Materials & Methods:** We planned the present study to assess electrolyte imbalance among hypotensive patients. A total of 20 hypotensive patients and 20 normal controls (Blood pressure within physiologic range) were included in the present study. All the subjects were called up in the early morning and serum blood samples were obtained. These samples were subjected to analysis on Autoanalyzer for estimation of serum sodium and potassium levels. All the results were analyzed by SPSS software.

**Results:** In the present study, we analyzed a total of 40 subjects. Out these 40, 20 subjects were hypotensive, while the remaining 20 were normal controls. Mean sodium levels in

the hypotensive and normal control group was found to be 130.8 and 138.1 mEq/L respectively. Mean potassium levels in the hypotensive group and the normal control group was found to be 3.2 and 3.8 mmol/L respectively.

**Conclusion:** Correlation does exist between the serum electrolyte levels and blood pressure.

**Key words:** Electrolyte, Hypotension, Potassium, Sodium.

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## INTRODUCTION

Hypotension can be defined as any blood pressure (BP) that is below the normal expected for an individual in a given environment. There is no single numerical cutoff universally accepted as representing hypotension. For example, while <90 mmHg may be considered hypotensive for someone with "normal BP" of 120 mmHg, many healthy young adults will have a resting BP at or potentially below this level and will not be considered hypotensive. As a result, it is difficult to estimate the prevalence.<sup>1-3</sup> Background history and accompanying clinical findings should be considered, bearing in mind that many common illnesses present atypically in older adults, without characteristic symptoms such as pain. Epidemiologic, experimental, and clinical studies suggest that sodium and potassium are important regulators of blood pressure. Surveys conducted in widely divergent geographic locations indicate significant prevalence of hypotension in populations ingesting diets with altered electrolyte levels.<sup>4-6</sup> Amelioration of hypokalemia lowers blood pressure in mineralocorticoid-induced hypertension in rats and in essential hypertensive patients receiving thiazide diuretics.<sup>7</sup> Hence; we planned the present study to assess electrolyte imbalance among hypotensive patients.

## MATERIALS & METHODS

We planned the present study in the department of general medicine of R.B.M. Hospital, Bharatpur, Rajasthan, and it included assessment electrolyte imbalance among hypotensive patients. A written consent was obtained after explaining in detail the entire research protocol.

A total of 20 hypotensive patients and 20 normal controls (Blood pressure within physiologic range) were included in the present study. Patients were defined as hypotensive when systolic blood pressure was less than 90 millimeters of mercury (mm Hg) or diastolic was less than 60 mm Hg.<sup>6-8</sup> Patients with any other cardiac pathology, patients with history of any other systemic illness, any known drug allergy, and patients on any form of drug therapy which alters the renal excretion of electrolytes were excluded from study.

All the subjects were called up in the early morning and serum blood samples were obtained. These samples were subjected to analysis on Autoanalyzer for estimation of serum sodium and potassium levels. All the results were analyzed by SPSS software. Chi-square test was used for assessment of level of significance. P-value of less than 0.05 was taken as significant.

Table 1: Clinical and demographic details of the patients

Parameter	Hypotensive group	Normal control group
Mean age (years)	45.5	46.1
Males	12	11
Females	8	9
Mean systolic blood pressure (mm of Hg)	84	118
Mean diastolic blood pressure (mm of Hg)	58	82

Graph 1: Clinical and demographic details of the patients

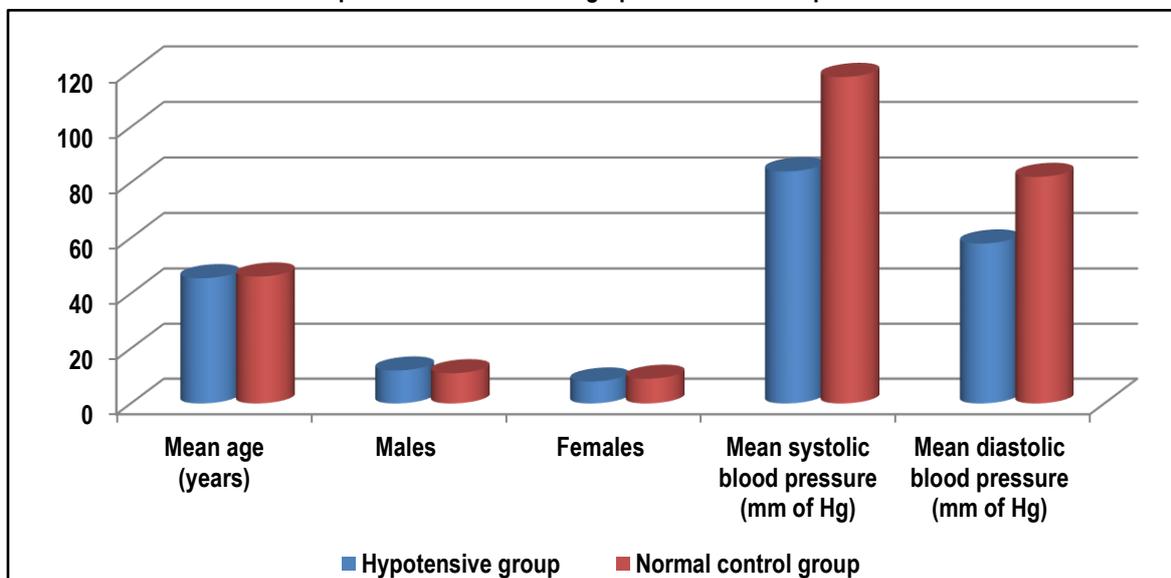


Table 2: Comparison of mean sodium and potassium levels in b/w hypotensive patients and normal controls

Parameter	Hypotensive patients	Normal controls	P- value
Mean sodium levels (mEq/L)	130.8	138.1	0.06
Mean potassium levels (mmol/L)	3.2	3.8	0.08

## RESULTS

In the present study, we analyzed a total of 40 subjects. Out these 40, 20 subjects were hypotensive, while the remaining 20 were normal controls. Mean age of the subjects of hypotensive group was 45.5 years while mean age of the normal control group was 46.1 years. There were 12 males and 8 females in the hypotensive group, while there were 11 males and 9 females in the normal control group. Mean sodium levels in the hypotensive and normal control group was found to be 130.8 and 138.1 mEq/L respectively. Mean potassium levels in the hypotensive group and the normal control group was found to be 3.2 and 3.8 mmol/L respectively. We observed non-significant alteration in the mean serum electrolyte levels in between the two study groups.

## DISCUSSION

Hypotension, defined as systolic blood pressure less than 90 mm Hg, is recognized as a sign of hemorrhagic shock and is a validated prognostic indicator. A fall in arterial blood pressure results in failure to perfuse the systemic capillary networks. Compensatory reflexes usually prevent this situation and awareness of these mechanisms helps in understanding the causes and management of hypotension. Intrinsic, local tissue factors regulate blood flow through certain vascular beds ('autoregulation'), e.g. brain, but superimposed upon these are

factors controlling the systemic arterial pressure. Blood pressure is the product of cardiac output, peripheral vascular resistance and blood volume. Cardiac output depends mainly upon left ventricular end diastolic volume, myocardial contractility and heart rate. Peripheral vascular resistance is largely determined by the degree of vascular smooth muscle contraction, the blood viscosity and length of vessels being less important.<sup>7-9</sup> Hence; we planned the present study to assess electrolyte disturbance in hypotensive patients.

In the present study, we didn't observe any significant alteration in the mean serum electrolyte levels and normal controls. Krishna GG observed that in normotensive subjects ingesting normal amounts of sodium, short-term potassium depletion increases the mean arterial pressure from 90.9 +/- 2.2 mm Hg to 95.0 +/- 2.2 mm Hg. Furthermore, acute sodium loading increases blood pressure in potassium-depleted subjects but it had no effect in subjects ingesting normal amounts of potassium. Preliminary studies indicate that short-term potassium depletion also elevates blood pressure in hypertensive patients. Potassium supplementation lowers blood pressure in hypertensive patients ingesting normal amounts of sodium. Blacks appear to be more sensitive to the hypotensive effects of potassium. The mechanism of potassium-induced changes in blood pressure is not well understood. Potassium depletion consistently induces sodium

retention. The hypertensive effects of potassium depletion and hypotensive effects of potassium supplementation are not observed when sodium intake is kept low. Direct vasoconstrictive effects of hypokalemia may contribute to the pressor effect of potassium depletion.<sup>10</sup>

Thunhorst RL et al examined the effects of hypotension and fluid depletion on water and sodium ingestion in rats in response to intracerebroventricular infusions of ANG II. Hypotension was produced by intravenous infusion of the vasodilator drug minoxidil (25 microg x kg(-1) x min(-1)) concurrently with the angiotensin-converting enzyme inhibitor captopril (0.33 mg/min) to prevent endogenous ANG II formation. Hypotension increased water intake in response to intracerebroventricular ANG II (30 ng/h) but not intake of 0.3 M NaCl solution and caused significant urinary retention of water and sodium. Acute fluid depletion was produced by subcutaneous injections of furosemide (10 mg/kg body wt) either alone or with captopril (100 mg/kg body wt sc) before intracerebroventricular ANG II (15 or 30 ng/h) administration. Fluid depletion increased water intake in response to the highest dose of intracerebroventricular ANG II but did not affect saline intake. In the presence of captopril, fluid depletion increased intakes of both water and saline in response to both doses of intracerebroventricular ANG II. Because captopril administration causes hypotension in fluid-depleted animals, the results of the two experiments suggested that hypotension in fluid-replete animals preferentially increases water intake in response to intracerebroventricular ANG II and in fluid-depleted animals increases both salt and water intake in response to intracerebroventricular ANG II.<sup>11</sup>

Tabuchi Y et al investigated the hypotensive mechanism of potassium supplementation, blood pressure responses and hormonal changes were measured during KCl supplementation in salt-loaded patients with essential hypertension. Ten patients with essential hypertension were placed on low sodium intake, high sodium intake, and high sodium intake with KCl supplementation. Blood pressure increased during NaCl loading and decreased during KCl supplementation. The levels of urinary sodium and fractional sodium excretion increased after KCl supplementation. Reduction in blood pressure after KCl supplementation was correlated with an increase in blood pressure after NaCl supplementation, i.e., potassium sensitivity and sodium sensitivity were correlated. Plasma PGE2 and norepinephrine increased after KCl loading. Increases in plasma PGE2 were correlated with a reduction of blood pressure by KCl. These results indicate that natriuresis and hypotensive action of KCl are associated with increased PGE2 production, and the sympathetic nerve activity suggests a compensatory increase for the decrease in blood pressure.<sup>12</sup>

## CONCLUSION

Under the light of above mentioned data, the authors conclude that correlation do exist between the serum electrolyte levels and blood pressure. However, further studies are recommended.

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