"ASSOCIATION BETWEEN 24 HOUR URINARY SODIUM AND POTASSIUM EXCRETION AND BLOOD PRESSURE IN NEWLY DIAGNOSED HYPERTENSIVE SUBJECTS, A ONE YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE HOSPITAL."

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LIST OF ABBREVIATIONS

- BP: Blood Pressure
- DBP: Diastolic Blood Pressure
- SBP: Systolic Blood Pressure
- Na: Sodium
- K: Potassium
- HTN: Hypertension
- DM: Diabetes Mellitus
- IHD: Ischemic Heart Disease
- WHO: World Health Organization
- SPSS: Statistical Package for the Social Sciences

ABSTRACT

Introduction: Hypertension is a leading global health concern, significantly contributing to cardiovascular and renal diseases. Among various factors, dietary sodium and potassium intake play a crucial role in blood pressure regulation. Excess sodium intake is associated with increased blood pressure, while potassium has an antihypertensive effect. This study aims to examine the correlation between 24-hour urinary sodium and potassium excretion in newly diagnosed hypertensive patients.

Methods: A cross-sectional hospital-based study was conducted on 97 newly diagnosed hypertensive individuals at KAHER's Jawaharlal Nehru Medical College, Belagavi. Sodium and potassium excretion levels were assessed using 24-hour urine samples. Blood pressure measurements and demographic data were recorded. Statistical analysis was performed using IBM SPSS Version 20.0 to evaluate correlations.

Results: The mean age of participants was 58.47 years, with a male predominance (70.1%). The mean systolic and diastolic blood pressure were 142.95 mmHg and 86.7 mmHg, respectively. 40.21% of subjects had increased sodium levels, and 48.45% had decreased potassium levels. A significant negative correlation was observed between 24-hour urinary potassium excretion and diastolic blood pressure ($p=0.0023$), whereas sodium excretion showed a weak correlation with systolic blood pressure ($p=0.6723$).

Conclusion: The findings highlight the importance of sodium and potassium intake in blood pressure regulation. A higher potassium excretion was associated with lower diastolic blood pressure, while sodium excretion did not show a significant correlation with DBP. The study underscores the need for dietary interventions to increase potassium intake while moderating sodium consumption to mitigate hypertension risk.

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INTRODUCTION

Hypertension, a leading global health concern, affects over 1 billion individuals worldwide and is responsible for a significant proportion of death associated with cardiovascular as well as renal diseases.[1] Hypertension is an important contributor to severe health issues, like stroke, heart attack, heart failure, and chronic kidney disease. Often referred to as the “silent killer,” it is characterized by a lack of symptoms in its initial stages. Although there have been improvements in medical treatments, the rate of hypertension is still increasing especially in low and middle income nations, in which urban growth and lifestyle modifications have led to a higher prevalence of this condition.[2]

Among the many factors influencing blood pressure, dietary intake, particularly sodium and potassium consumption, plays a pivotal role. Sodium is critical for maintaining extracellular fluid volume, and cellular function.[3] However, increased sodium intake is strongly associated with increased blood pressure, primarily due to its effects on fluid retention, vascular stiffness, and endothelial dysfunction. Potassium, another vital electrolyte, exhibits antihypertensive properties by promoting sodium excretion, reducing vascular resistance, and improving endothelial health.[4] The ratio of sodium to potassium serves as a more effective indicator of variations in blood pressure and cardiovascular health than looking at the levels of each electrolyte separately.[5]

Numerous epidemiological and interventional studies have already examined the connection between sodium and potassium intake and hypertension. Research, including the "International Study on Salt and Blood Pressure (INTERSALT)," has demonstrated a strong correlation between increased sodium intake and raised blood-pressure. Furthermore, these studies indicate that increased potassium intake is

associated with decreased blood pressure levels. Despite these findings, the interplay between these electrolytes is complex and influenced by factors that include genetics, dietary habits, physical activity and underlying comorbidities.[6]

India, with its diverse dietary patterns and high prevalence of hypertension, presents a unique challenge in addressing this health issue.[7] The average sodium intake among Indians far exceeds the recommended daily limits, while potassium intake remains significantly below optimal levels. This dietary imbalance, coupled with a lack of awareness and insufficient dietary interventions, has contributed to the escalating rates of hypertension in the population. Accurate estimate of dietary sodium and potassium consumption is crucial in understanding their role in the pathogenesis of hypertension and designing effective interventions.[8]

The assessment of sodium and potassium intake through a 24 hour urine collection is widely considered the most accurate method. This approach is preferred over dietary recall techniques, which often involve errors due to memory lapses and inaccuracies. In contrast, analyzing urine over a full day provides a trustworthy measure of the intake levels of these essential electrolytes.[9] This approach improves the comprehension of the connection between electrolytes and blood pressure, leading to more focused strategies for managing hypertension.

This study focuses on newly diagnosed hypertensive patients, a group that is particularly critical for early intervention and long-term risk reduction. By examining the relationship between 24 hour urinary sodium and potassium excretion and blood pressure in this study population, the study aims to provide insights into the dietary as well as physiological factors influencing hypertension. Such findings can contribute to development of population-specific dietary recommendations along with public health policies to mitigate the burden of hypertension and its complications.

AIMS AND OBJECTIVES

Objective

- To study the correlation of 24 hour urinary sodium and potassium excretion in newly diagnosed hypertensive subjects.

REVIEW OF LITERATURE

Increased blood pressure, also known as hypertension, is a very serious health problem that affects millions globally. The World Health Organization reports that around 1.13 billion people experience this condition, which increases the likelihood of developing cardiovascular diseases, experiencing strokes, and facing kidney failure.[10] Hypertension rates vary across different geographical areas and population groups, shaped by factors like age, nutrition, lifestyle choices, and genetic predispositions. Recognizing the global effects of hypertension is vital for creating successful prevention and treatment approaches.[2]

Sodium and potassium are crucial for maintaining healthy blood pressure levels. A diet high in sodium can lead to raised blood pressure, whereas consuming enough potassium may help alleviate this issue. To effectively prevent and manage hypertension, it's beneficial to lower sodium intake and eat more foods rich in potassium, like fruits and vegetables. Providing education on nutritional choices is important for fostering healthier habits and reducing the occurrence of high blood pressure.[11]

24-hour urinary excretion studies are valuable tools in hypertension research, providing insights into individual sodium and potassium balance. These studies measure the excretion levels of these electrolytes, enabling researchers and healthcare professionals to assess dietary intake and understand their relationship with blood pressure regulation. By examining urinary excretion patterns, clinicians can tailor dietary recommendations and treatment plans to better manage hypertension, ultimately improving patient outcomes.[12]

Pathophysiology of Sodium and Potassium in Hypertension

Initial research into how potassium affects blood pressure—specifically in terms of deficiency and supplementation—largely ignored the detrimental impacts that a lack of potassium could have on blood vessels. These studies primarily focused on young rats and often involved low concentrations of both sodium and chloride. When potassium intake is restricted, cells experience a deficit in potassium, prompting them to retain sodium to maintain volume and tonicity. This condition results in a contraction of both intracellular and extracellular compartments, ultimately leading to reduced blood pressure.[13]

Recent studies on rats have demonstrated that a lack of potassium mainly increases blood pressure when sodium chloride consumption is high, such as when approximately 4.5 grams of sodium chloride is consumed for every 100 grams of food. Observational studies revealed that a negative correlation between the potassium consumption and blood pressure, and a lower occurrence of hypertension and reduced stroke risk associated with higher potassium intake.[14] In their study, researchers noted that when potassium excretion decreased by 50 mmol per day, the systolic blood pressure rose by 3.4 mm Hg, while the diastolic blood pressure increased by 1.9 mm Hg. They also discovered a notable inverse relationship between the urinary potassium to sodium ratio and blood pressure, which showed a stronger statistical connection than the levels of sodium or potassium excretion on their own.[15]

Studies indicate that raising potassium levels in hypertensive rats consuming high-sodium diets can markedly reduce blood pressure, lower the likelihood of stroke and associated deaths, and avert issues like cardiac hypertrophy, damage to

mesenteric blood vessels, and kidney harm.[16,17] Studies indicate that these positive outcomes can happen without relying solely on dietary changes to lower blood pressure. The rice-fruit diet, designed by Kempner in the 1940s, was characterized by very low sodium content and high potassium levels, making it a popular option for treating hypertension and congestive heart failure. Many later investigations have validated the positive role of potassium in managing blood pressure.[18]

A comprehensive review of 33 randomized trials found that supplementing potassium, typically with a dosage of around 60 mmol per day, significantly lowers the systolic blood pressure by roughly 4.4 mm Hg and the diastolic blood pressure by about 2.5 mm Hg in individuals diagnosed with hypertension. For those with normal blood pressure, the reductions were smaller but still noteworthy. The results suggested that even people who do not have a potassium deficiency benefited from this supplementation, with more substantial effects observed in individuals with higher sodium excretion and predominantly Black participants. Additionally, increasing the intake of potassium in the diet can lessen dependence on hypertension medications, as many participants reported needing less or no medication after increasing their potassium consumption. Overall, these findings emphasize the critical role potassium plays in controlling blood pressure and enhancing cardiovascular health.[19]

The process of sodium reabsorption in the renal tubules is intensified in cases of primary hypertension. This increase is largely attributed to the activation of various sodium transporters present at the luminal membrane and the sodium pump present on the basolateral membrane. A significant player in this process is the sodium hydrogen exchanger type 3 which is mainly located in the proximal tubule and the thick ascending limb of the loop of Henle, which are areas responsible for most sodium reabsorption. Research indicates that the activity of the exchanger is heightened in

models of hypertension observed in rats.[20] A shortage of potassium can lead to an increased function of this exchanger, which is driven by factors such as acidosis within the cells and activation of the sympathetic nervous system, along with the renin-angiotensin system. In contrast, consuming more potassium in your diet may help alleviate these effects.

In primary hypertension, excessive production of aldosterone greatly enhances the activity of the sodium chloride cotransporter in the distal tubule and the epithelial sodium channel in the collecting duct. This process leads to sodium retention and potassium loss. Increased sodium intake further elevates the potassium excretion by enhancing the delivery of sodium to the distal parts of nephron. Additionally, a naturally occurring "digitalis-like factor," similar to ouabain is released from the adrenal glands and the brain in response to increased sodium levels. This factor is found in elevated concentrations in approximately 40% of untreated hypertensive patients and correlates with higher blood pressure levels. While short-term potassium depletion inhibits the sodium pump, long-term effects actually stimulate this pump, promoting sodium retention. Research suggests that inherited genetic factors affecting sodium transport and vascular regulation are likely contributors to primary hypertension. However, the exact genetic relationships are complex and influenced by environmental factors.[21]

The retention of sodium, driven by a substance similar to digitalis, combined with low levels of potassium (hypokalemia), disrupts the operation of the sodium pump present in the smooth muscle cells of blood vessels. This disturbance then causes an increase in the sodium concentration inside the cells while potassium levels decrease. The higher sodium concentration triggers the sodium-calcium exchanger type 1, allowing more of calcium to enter the cells. Moreover, reduced potassium

levels negatively affect the potassium channels present in the cell membrane, resulting in depolarization, or a shift in the membrane potential toward a more positive value. This depolarization further lowers the overall membrane potential due to the sodium pump's electrogenic properties.[22]

Membrane depolarization increases in intracellular calcium levels in vascular smooth muscle cells, by activating the voltage-gated calcium channels and the sodium-calcium exchanger. The increase in calcium concentration within the cytosol prompts the contraction of these muscle cells. Studies have pointed out that certain substances, like rostafuroxin, which is a compound that inhibits the effects of digitalis like factors on sodium pumps—show potential as effective antihypertensive agents. It is crucial to maintain a proper balance of sodium and potassium for healthy endothelial function. Excess sodium can decrease the production of nitric oxide, which serves as a vasodilator. On the other hand, a diet that is rich in potassium or raised potassium levels can promote vasodilation through endothelial hyperpolarization. This implies that consuming adequate potassium may lower cardiovascular risk by reducing arterial stiffness and supporting the overall health of arteries.[22]

Epidemiological Trends and Global Insights

Reddin et al. analyzed 104 studies that covered data from 52 countries and included more than 1.6 million participants. These studies represented at least one nation from 17 out of 21 regions identified in the global burden of disease. However, there were no suitable studies available for Andean Latin America, Central Asia, Central Sub-Saharan Africa and Oceania.[23]

The findings on potassium intake revealed a global average of 2.25 grams per day, which corresponds to approximately 57.6 millimoles. This average varied by gender, with men reporting a higher intake of 2.40 grams per day compared to 2.09 grams per day for women. Notably, there were significant regional disparities in potassium consumption. East Asia had the lowest average intake at 1.89 grams, while Eastern Europe reported the highest at 3.53 grams per day.[23]

In the regions that are included in the study, about 35% of the study population consumed more than 2.5 grams of potassium daily. Additionally, approximately 14% of participants had an intake exceeding 3.5 grams, which aligns with the World Health Organization's recommended level. However, six regions showed mean intakes lower than those identified as effective in the SaSS intervention trial, indicating the need for further examination of dietary potassium consumption in these areas.[23]

Among the individual countries, Lithuania stood out with the highest potassium intake, whereas Taiwan recorded the lowest. Moreover, four countries—Estonia, Latvia, Russia, and Lithuania—reported average intakes greater than the recommended 3.5 grams. These findings highlight the variations in potassium intake across different countries and regions, underscoring the importance of addressing nutritional disparities globally.[23]

The INTERSALT Study is a large-scale global research initiative that includes a significant number of 10,079 people aged 200-9 years. The objective of this study was to investigate both within the population and cross population hypotheses regarding the effects of 24 hour sodium excretion on blood pressure. The results demonstrated a positive and independent association between 24 hour sodium

excretion and SBP. After considering potential underreporting, the study found that an increase in sodium intake is linked to an increase in SBP and DBP estimated at about 3-6 mm Hg for SBP and 0-3 mm Hg for DBP. This relationship was consistent across different genders and age groups, involving 8,344 participants who did not have hypertension. When examining cross-population hypotheses with 52 sample groups, researchers identified significant independent correlations between median 24 hour urinary sodium excretion and median values for SBP and DBP, as well as the rates of hypertension prevalence. Specifically, a rise in median sodium intake of 100 mmol per day was associated with a rise in SBP and DBP equivalent to an additional 30 years of age; for instance, a 55 year old showed increases of 10 to 11 mm Hg in SBP and 6 mm Hg in DBP compared to a 25-year-old. [(24)]

Ndanuko et al. pointed out that high blood pressure is an important risk factor for cardiovascular diseases, stroke, and kidney issues. To manage blood pressure effectively, many lifestyle changes are advised, including weight reduction, increased physical activity, and maintaining a balanced diet. Although analyzing individual nutrients can be beneficial, it's crucial to recognize that foods are often part of larger dietary habits, which may result in nutrient interactions. Research shows that adhering to healthy dietary patterns—like the DASH (Dietary Approaches to Stop Hypertension) diet which can lead to significant reductions in systolic blood pressure. These diets are rich in fruits, vegetables, whole grains, and dairy, while minimizing the intake of meat, sugary foods, and alcohol. Additionally, making lifestyle changes such as engaging in regular physical activity and achieving weight loss can further aid in lowering blood pressure when combined with dietary modifications. Ndanuko et al. emphasized the need for more research to understand the effects of dietary patterns on

blood pressure across different cultural settings that were not covered in this review.[25]

Gay et al. conducted a systematic review and meta-analysis that examined the favorable effects of different dietary patterns on managing blood pressure (BP). However, the precise effectiveness of these diets remains uncertain. The review comprised of 24 trials involving a total of 23,858 participants with data being analyzed using random effects meta-analysis models. The findings revealed an overall reduction in systolic blood pressure of −3.07 mm Hg and a decrease in diastolic blood pressure of −1.81 mm Hg resulting from dietary changes. Among the diets studied, the DASH diet showed the most pronounced effect, causing a decrease of −7.62 mm Hg in systolic BP and −4.22 mm Hg in diastolic BP. Other dietary approaches, including low-sodium, low-sodium high-potassium, low-sodium low-calorie, and low-calorie diets, also yielded significant reductions in both systolic and diastolic BP. In contrast, the Mediterranean diet was associated with a notable drop in diastolic BP but did not exhibit a significant impact on systolic BP. The subgroup analyses indicated that the success of these dietary interventions varied depending on factors such as the length of the study, the size of the participant group, and demographic characteristics. In summary, this review's findings suggest that dietary changes can lead to significant, though variable, reductions in BP, with some dietary strategies being more effective than others in specific contexts. These outcomes carry critical implications for clinical practice and public health initiatives.[26]

Impact of Sodium and Potassium on Blood Pressure

Naser et al. explored the connection between sodium consumption and SBP by analyzing 24-hour urine samples from 2,143 individuals, resulting in a total of 10,031

blood pressure measurements. Their approach involved self-reported urine collections, a creatinine index of ≥0.7, and sex-specific mean creatinine excretion rate (mCER) ranges. They employed multilevel linear models and restricted cubic spline (RCS) plots to assess how a 100 mmol raise in sodium excretion in the 24 hour urine impacted mean SBP .The RCS plots showed an initial steep positive correlation between sodium intake and SBP, which became less pronounced for some methods, while the Kawasaki methods demonstrated a plateau. The study found the following SBP changes for each 100 mmol increase in urinary sodium: 0.64 mmHg for self-reported collections, 0.68 mmHg for the creatinine index ≥0.7, 0.87 mmHg for mCER within 25% of Kawasaki predictions, 0.98 mmHg for mCER within 15%, and 1.96 mmHg for sex-specific mCER ranges. The results indicated that the criteria for determining urine collection completeness could lead to varying findings in studies on 24-hour urine sodium and health outcomes.[27]

Kay-Tee et al. found that both SBP, DBP levels increased as the ratio of urinary sodium to creatinine rose, based on measurements from random urine samples. They noted significant variances of 7.2 mm Hg for SBP and 3.0 mm Hg for DBP when they compared the highest and lowest quintiles of sodium levels. This association persisted regardless of several factors including age, body mass index (BMI), urinary potassium to creatinine ratio, smoking habits, as well as among different genders and hypertension histories.

Importantly, the percentage of individuals with SBP of 160 mm Hg or higher dropped noticeably, from 12% in the highest sodium quintile to 6% in the lowest. The odds ratios for men and women present in the upper quintile compared to those present in the lower quintile were found to be 2.48 and 2.67, respectively. The estimated sodium intake was around 80 mmol/day in the lowest quintile and 220

mmol/day in the highest. These results indicate a significant link between urinary sodium concentrations, dietary sodium intake and blood pressure, emphasizing the necessity for reduced sodium intake among the general population..[28]

Lemogoum et al. conducted a study with 150 individuals from the Pygmy community and 150 individuals from the Bantu population. The average ages were 38 ± 12 years for the Pygmies and 33 ± 11 years for the Bantus ($p < 0.0001$). The results revealed significant differences in height, weight: Pygmies averaged 1.54 ± 0.09 m in height and 54.4 ± 9.2 kg in weight, while Bantus averaged height of 1.72 ± 0.12 m and weighed 77.2 ± 14.8 kg. The study also found that the prevalence of hypertension, standardized by age, was lower among Pygmies, at 3.3%, compared to 28% among Bantus. Additionally, when adjusting for age, SBP and DBP measurements were considerably lower in Pygmies. The average SBP for Pygmies was 107 ± 12 mmHg, compared to 119 ± 17 mmHg for Bantus, and the diastolic readings were 71 ± 11 mmHg for Pygmies versus 78 ± 13 mmHg for Bantus (all $p < 0.0001$). While blood pressure tends to increase with age, this increase was less pronounced among Pygmies (all $p < 0.01$). The study also reported that urine sodium excretion was significantly decreased in Pygmies, at 46.9 ± 32.4 mmol/l, compared to 121.5 ± 61.0 mmol/l in Bantus. Furthermore, a positive correlation between blood pressure and urine sodium excretion was found. Urinary potassium levels were similar in both groups and showed no significant association with blood pressure. In the whole population studied, and specifically among Bantus, higher urinary sodium excretion was linked to an increased likelihood of hypertension. A logistic regression analysis identified urine sodium excretion, Bantu ethnicity and age as key independent predictors of hypertension. The odds ratios (OR) and 95% confidence intervals (CI) were as

follows: 1.012 (1.005–1.018) for urinary sodium excretion, 11.408 (3.599–36.165) for Bantu ethnicity, and 1.095 (1.057–1.135) for age (all $p < 0.0001$).[29]

Ziaei et al. performed a systematic review and analysis aimed at understanding the inconsistent relationship that is present between urine potassium levels, blood pressure (BP) and hypertension risk. Their study incorporated twelve research articles involving a total of 16,174 participants, resulting in the extraction of 21 effect sizes. The analysis revealed that individuals with normal blood pressure had an average potassium excretion rate that was 3.46 mmol per 24 hours higher than those with hypertension. However, an increase in potassium excretion did not appear to impact the likelihood of developing hypertension. In general, there was found to be no significant correlation between blood pressure and urinary potassium levels. Notably, a subgroup analysis showed a positive correlation between these variables in children. To summarize, the study concluded that the measurement of 24-hour urinary potassium output is not associated with blood pressure or hypertension risk. Additionally, it was observed that those with normal blood pressure tended to have higher average potassium levels. The authors suggested that future investigations should focus on the influence of various dietary potassium sources on blood pressure.[30]

The influence of dietary methods aimed at lowering hypertension risk is a significant concern in public health. Studies indicate that decreasing sodium intake while increasing potassium consumption can effectively help control high blood pressure. Ongoing research is investigating how adjustments in these dietary elements impact the RA system and endothelial function. However, a thorough systematic review comparing the sodium-to-potassium ratio with the separate consumption of sodium- potassium concerning blood pressure and related factors in humans is

lacking. The analyses suggest that, among the reviewed trials, the sodium-potassium ratio correlates more strongly with BP results than the individual intakes of sodium/ potassium in adults who are hypertensive. Observational studies further reinforce the idea that the sodium-to-potassium ratio serves as a more effective measure for blood pressure management and the likelihood of developing hypertension. Still, it is uncertain whether these findings extend to normotensive individuals and children, or to other pertinent measurements such as the renin-angiotensin system and arterial stiffness, indicating a need for further research in these fields.[31]

Studies have demonstrated that the urine sodium-to-potassium (Na/K) ratio has a stronger association with high blood pressure and cardiovascular disease risk compared to assessing sodium or potassium levels in isolation. This consensus statement advises calculating the average Na/K ratio from casual urine samples taken at various times throughout at least four days each week. This method helps provide more accurate personal estimates, considering the considerable fluctuations in these measurements from day to day and even within the same day. These recommendations are aimed at healthy individuals and may not be appropriate for those with specific medical conditions, as there is insufficient evidence to support their application in such cases. Moreover, the usefulness of the urine Na/K ratio for preventing or managing hypertension is still uncertain and requires additional investigation.[32]

Comparative Regional and Population-Specific Analyses

Dietary intake of sodium and potassium varies widely across regions, largely influenced by cultural practices and lifestyle changes. Countries such as China and

India tend to have high sodium consumption alongside low potassium intake, while the United States reflects similar trends with notable regional differences.[33]

According to Liu et al. (2023), China has an exceptionally high mean sodium intake, estimated at about 4,121 mg per day, surpassing WHO recommendations. Moreover, northern regions report even higher values, averaging 4,388 mg, compared to the southern areas, which average 3,998 mg.[34] In India, high sodium levels are similarly linked to the prevalence of processed foods and traditional cooking methods. In USA, the average sodium intake stands at around 3,400 mg per day, primarily due to processed foods.[35]

China's potassium intake is relatively low, with averages of 1,534 mg per day, which falls short of 60% of the WHO's recommended levels.[34] India also displays low potassium consumption, contributing to the health risks associated with hypertension.[35] In contrast, the average potassium intake in the US is approximately 3,500 mg per day, which is closer to the recommended levels, although it still varies by region.[35]

The N/K ratio is notably high in all regions, with China indicating a concerning ratio of 3.68, presenting significant cardiovascular risks. [35,36]

Nonetheless, emerging studies suggest that dietary habits are shifting in urban areas toward healthier options, which may help improve potassium intake and reduce sodium levels over time. This trend underscores the necessity for ongoing public health initiatives aimed at addressing these dietary imbalances on a global scale.

Hypertension is a complex health condition shaped by a combination of genetic and environmental influences. Emerging research sheds light on how these

factors interact, especially in various populations. Gaining a deeper understanding of these relationships is important for creating effective strategies for prevention/ treatment.

Genetics plays a significant role in the development of hypertension, with studies estimating that hereditary factors account for approximately 30-60% of variations in blood pressure.[37] Certain genetic markers, particularly those involved in the RAAS and genes that regulate sodium processing, have been related with a heightened risk of hypertension, particularly among individuals of West African descent.[38] Research involving African-Brazilian groups has highlighted 14 candidate genes linked to essential hypertension, demonstrating the impact of genetic ancestry on this condition.[37]

Aside from genetics, environmental influences are crucial in the emergence of hypertension. Factors such as dietary choices, obesity, and overall lifestyle significantly contribute to this condition. A diet high in sodium combined with a lack of exercise can intensify genetic tendencies.[38] The HUNT study revealed that elements such as cardiorespiratory fitness and dietary habits interact with individual genetic risk scores, meaning those predisposed to hypertension may experience greater effects from environmental factors related to lifestyle.[39]

There is proof that gene-environment interactions play a pivotal role in determining hypertension risk. For example, certain genes have been found to influence how alcohol consumption affects blood pressure levels.[40] Additionally, an exposome-wide association study discovered various environmental exposures associated with hypertension, underscoring the importance of evaluating both genetic and environmental factors in assessing risk.[41]

While genetic factors are indeed significant, the ability to modify environmental influences offers potential intervention opportunities. Recognizing this dual impact is crucial for designing targeted prevention and management approaches for hypertension. focus on genetics and environment can enhance hypertension management strategies.

Mechanisms Underlying Sodium and Potassium Excretion

Potassium excretion is managed through a variety of intricate mechanisms located in different parts of the nephron. The majority of potassium that is filtered is reabsorbed mainly through passive mechanisms in the proximal tubule. This process predominantly occurs through the paracellular pathway, supported by solvent drag and fluctuations in transepithelial voltage. Typically, fewer than 10% of the filtered potassium passes on to the distal nephron.[42]

In the thick ascending limb of the loop of Henle, potassium is reabsorbed through both paracellular and transcellular mechanisms. The Na^+-K^+-ATPase pump located on the basolateral side generates a positive charge within the lumen, which aids in the functioning of the apical Na^+-K^+-$2Cl^-$ (NKCC2) cotransporter. This cotransporter facilitates the simultaneous reabsorption of one potassium ion, one sodium ion, and two chloride ions without altering the overall charge balance. For NKCC2 to function effectively, potassium must be recycled from the cell back to the lumen, and this process is supported by the renal outer medullary K^+ (ROMK) channel. Additionally, the ROMK channel's activity helps maintain a positive lumen environment, which promotes passive reabsorption of potassium via the paracellular route.[42]

In the early part of the distal convoluted tubule (DCT), the reabsorption of sodium takes place via the thiazide-sensitive Na^+-Cl^- cotransporter (NCC2) located on the apical membrane. This reabsorption is facilitated by the low intracellular sodium levels, which are kept low by the activity of the Na^+-K^+-ATPase on the basolateral side. Throughout all sections of the DCT, the ROMK channel is consistently present. Beginning in the DCT2 segment, the amiloride-sensitive epithelial Na^+ channel (ENaC) starts to be expressed and remains active in the connecting tubule and the cortical collecting duct, where it helps manage potassium excretion through the ROMK channel. Aldosterone significantly influences potassium secretion by various mechanisms, chiefly by boosting the function of the basolateral Na^+-K^+-ATPase and increasing the availability of sodium and potassium channels. Furthermore, potassium reabsorption occurs through the action of the H^+-K^+-ATPase, which facilitates the release of one hydrogen ion and the uptake of one potassium ion through ATP hydrolysis.[42]

Aaron et al. indicated that consuming a high amount of salt not only leads to elevated blood pressure but also causes a range of health problems, such as endothelial dysfunction, alterations in cardiovascular structure and function, albuminuria, the advancement of kidney disease, and an increase in cardiovascular-related health issues and mortality within the general population. On the other hand, increasing dietary potassium can mitigate these adverse effects and is significantly associated with lower incidences of stroke and a decreased risk of cardiovascular diseases. Certain populations, particularly those who are overweight or obese and older individuals, tend to be more sensitive to the effects of decreased salt intake and may derive the greatest benefits from such dietary modifications. It is advised to adopt a dietary approach that includes moderate salt reduction and higher potassium intake

as an effective means to prevent and manage hypertension, thereby reducing the rates of cardiovascular morbidity and mortality.[43]

To summarize, existing evidence suggests that reducing sodium intake on a national level is vital, alongside increasing potassium consumption as recommended in current health guidelines. This strategy is crucial for public health, as it seeks to avert kidney disease, strokes, and cardiovascular issues.

There is a significant need for more long-term studies to investigate the enduring effects of sodium and potassium balance. Moreover, there is a lack of research regarding how variations in the sodium-to-potassium ratio affect different demographic groups. Additionally, information is limited on the molecular and genetic factors that contribute to individual variations in susceptibility to hypertension.

Relevance to Indian Context

Mathur et al. point out that a diet high in sodium is a significant factor contributing to the loss of -DALYs related to cardiovascular diseases. This research examines the awareness, habits, and factors that affect dietary salt intake among adults aged 18 to 69, based on the findings of the National NCD Monitoring Survey conducted in India. The study involved a randomly selected sample of 3,000 adults drawn from 150 Primary Sampling Units that are representative of the population. The data collection process focused on evaluating individuals' awareness and behaviors concerning their salt intake, as well as estimating dietary salt consumption through urinary sodium levels in spot urine samples. The analysis explored various sociodemographic, behavioral, and metabolic factors influencing salt intake. Findings showed that less than one-third of adults across all genders and age categories, in both

rural and urban environments, acknowledged that high daily salt consumption could adversely affect their health. The average daily intake of salt was determined to be 8.0 grams, with men consuming an average of 8.9 grams per day and women averaging 7.1 grams. Importantly, men, individuals living in rural areas, and those categorized as overweight or obese had notably higher salt intakes. Additionally, there was a general lack of knowledge about the negative health impacts of high salt intake, and efforts to reduce salt consumption were found to be limited. The average daily salt intake was significantly above the WHO recommendation of 5 grams per day. This highlights an urgent need for effective measures to lower dietary salt consumption among the population in India.[44]

Bhattacharya et al. point out the creation of various strategies to lower salt consumption in order to address hypertension, but many of these methods do not consider the significant variation in individuals' salt sensitivity. This paper examines high salt intake as a modifiable risk factor for hypertension within the Indian context and stresses the necessity for updated thresholds. Studies indicate that average salt intake among the Indian population varies from 5.22 to 42.30 grams per day. In addition, a connection has been established between daily salt intake of 5 grams or more and the development of hypertension. Although key stakeholders recognize the importance of reducing salt consumption to prevent hypertension and associated CVDs in India, various challenges impede the successful implementation of these strategies. These obstacles include enduring social and cultural beliefs, a chaotic food retail environment, and insufficient food regulation. Certain multinational food companies have started investigating this issue, highlighting the importance of further understanding the contextual barriers and facilitators to formulate effective strategies. These strategies should aim at raising consumer awareness, encouraging the food

industry to reduce salt, and establishing more user-friendly food labeling practices across the nation. In summary, Bhattacharya et al. contend that salt intake in India remains elevated, with evaluations primarily based on subjective approaches. Currently, dietary salt intake guidelines are standardized nationwide, neglecting the varied dietary habits and environmental factors. It is critical to tackle these issues through population-level, evidence-based research..[45]

The existing literature on hypertension emphasizes the significant relationship between dietary intake of sodium and potassium and their influence on blood pressure regulation. Hypertension is a major global health concern, affecting more than a billion people and leading to serious cardiovascular and kidney-related issues. Research consistently indicates that high sodium consumption is a major factor in increased blood pressure, while sufficient potassium intake can help mitigate this effect, leading to healthier blood pressure levels.

Important studies, such as the INTERSALT Study, provide compelling evidence linking sodium consumption to blood pressure levels across varied populations. This research demonstrates that even small increases in sodium intake can considerably raise both systolic and diastolic blood pressures, highlighting the necessity for effective dietary changes. Additionally, the established inverse relationship between potassium intake and high blood pressure suggests that boosting consumption of potassium-rich foods might be an effective approach to managing hypertension.

The findings also indicate variations in dietary patterns across different regions, with numerous populations consuming sodium well beyond recommended levels while not meeting the guidelines for potassium intake. This discrepancy is

especially noticeable in countries like India, where cultural eating habits are linked to increasing hypertension rates. Monitoring 24-hour urinary sodium and potassium excretion is a crucial method for accurately assessing an individual's dietary intake and shaping public health initiatives.

In conclusion, the literature strongly supports public health campaigns aimed at decreasing sodium consumption and increasing potassium intake as critical aspects of managing hypertension. Future studies should further investigate the intricate interactions among these electrolytes, genetic influences, and lifestyle factors to create customized dietary guidelines. By tackling these nutritional imbalances and raising public awareness, it is feasible to reduce the escalating challenge of hypertension and improve cardiovascular health outcomes worldwide.

MATERIALS AND METHODS

Source of Data

The study utilized data from newly diagnosed hypertensive patients admitted to the KLE Dr Prabhakar Kore Hospital, Belagavi Primary data were collected through patient interviews, clinical evaluations, and 24-hour urinary sodium and potassium analyses. Secondary data, such as medical records, were also reviewed to confirm hypertension diagnosis and ensure data reliability.

Study Design

A cross-sectional, hospital-based study design was employed to evaluate the relationship between 24-hour urinary sodium and potassium excretion and blood pressure in newly diagnosed hypertensive individuals.

Study Period

April 1 2023 to March 31 2024 over period of one year

Sample Size

Formula used for sample size calculation

Sample size at 95% confidence interval

At 18% tolerable error

N = [Z(1- a/2)2 X (SD)2 / (18% of SD)2

N = 97

Sampling technique

Purposive sampling was used to recruit newly diagnosed hypertensive patients. Inclusion criteria ensured a homogenous population relevant to the study objective while maintaining diversity in demographic factors such as age, gender, and dietary patterns..

Inclusion Criteria and Exclusion criteria

Inclusion Criteria:

- Adults aged ≥18 years with a confirmed diagnosis of hypertension.
- Willingness to provide informed consent and participate in 24-hour urine collection.
- No prior use of antihypertensive medications.

Exclusion Criteria:

- Patients with secondary hypertension or chronic illnesses that could influence sodium or potassium metabolism (e.g., renal disorders).
- Recent hospitalization or acute illnesses.
- Pregnancy or lactation.

Data collection procedure

Participants were instructed on proper 24-hour urine collection methods to ensure accuracy in measuring sodium and potassium excretion. Blood pressure readings were obtained using a calibrated sphygmomanometer, with measurements

taken in a seated position after a 5-minute rest. The BP cuff was placed over the right arm on bare skin at the level of the chest. Patient was asked to place both feet flat on the ground, and the blood pressure readings were recorded. Demographic and clinical data, including dietary history, were collected through interviews using structured questionnaires.

Urine samples were analyzed in a certified laboratory to quantify sodium and potassium concentrations. The sodium-to-potassium ratio was calculated to assess its relationship with systolic and diastolic blood pressure.

Data processing and analysis

The data was first collected using a custom proforma tailored to the requirements of this study. Subsequently, it was imported into Microsoft Excel for further analysis. The statistical calculations, including the p-value, were performed using IBM SPSS Version 20.0. Descriptive statistics were reported as both numerical values and percentages. To compare proportions, the Z test for two sample proportions was utilized. The relationship between two non-parametric variables was evaluated using the Pearson Chi-square test. Additionally, to compare the means of more than two groups, the One-Way ANOVA test was employed. A p-value of less than 0.05 was deemed statistically significant

RESULTS

Data contains measurement on 97 subjects.

Figure 1: Distribution of subjects according to sex.

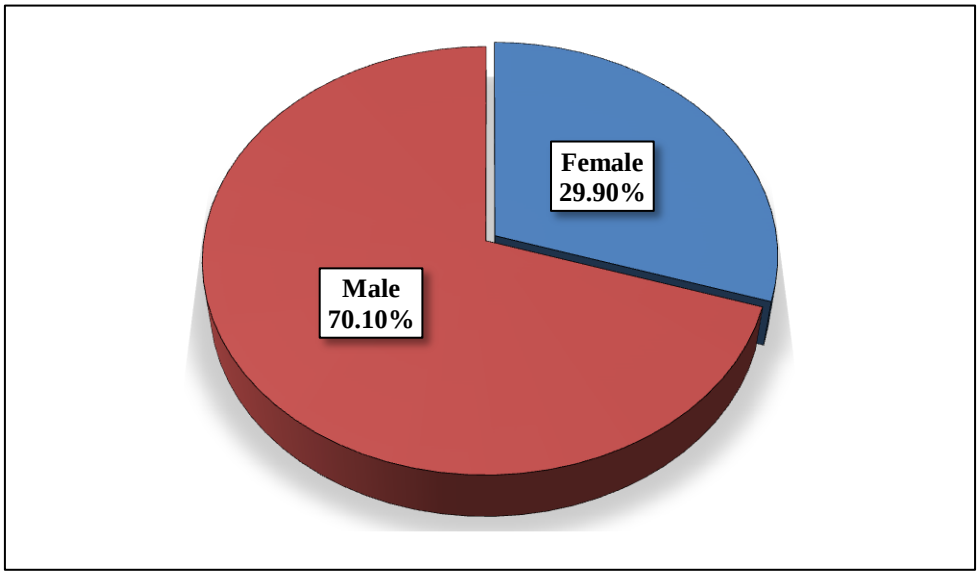

Table 1: Distribution of subjects according to demographic details.

Variables	Sub Category	Number of subjects (%)
Age (years)	Mean ± SD	58.47 ± 14.6
	Median (Min, Max)	60 (24, 98)
Sex	Female	29 (29.9%)
	Male	68 (70.1%)

The mean age of the subjects is 58.47 years, with a standard deviation of 14.6. The median age is 60 years with age ranging from 24 to 98 years. Regarding sex distribution, the majority of subjects are male (70.1%), with females accounting for 29.9% of the sample.

Table 2: Distribution of subjects according to BP.

Variables	Mean ± SD	Median (Min, Max)
SBP	142.95 ± 6.55	140 (130, 160)
DBP	86.7 ± 5.72	90 (80, 100)

The mean systolic blood pressure (SBP) was 142.95 ± 6.55 mmHg. The median SBP was 140 mmHg, with values ranging from 130 to 160 mmHg. Similarly, the mean diastolic blood pressure (DBP) was 86.7 ± 5.72 mmHg. The median DBP was 90 mmHg, with a range between 80 and 100 mmHg. These results suggest a population with predominantly higher-than-normal BP values, indicative of prehypertension or hypertension in the majority of participants.

Figure 2: Distribution of subjects according to 24-hour sodium.

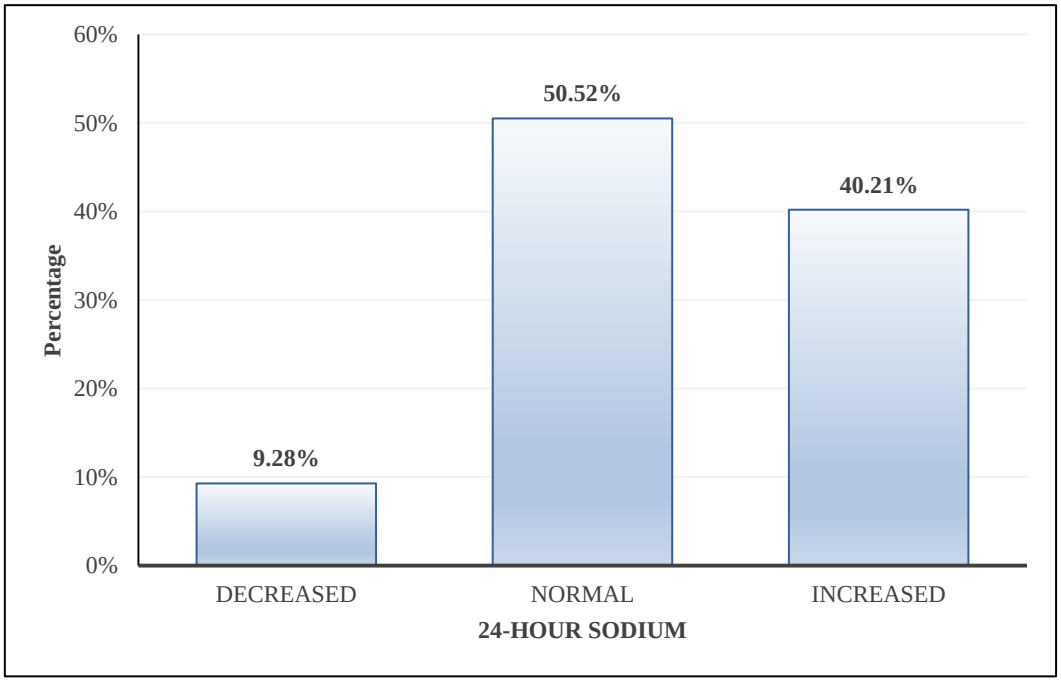

Table 3: Distribution of subjects according to 24-hour sodium.

24-hour Sodium	Number of subjects (%)
Decreased	9 (9.28%)
Normal	49 (50.52%)
Increased	39 (40.21%)
Mean ± SD Median (Min, Max)	544.43 ± 785.31 154.9 (1.9, 3546)

Out of the total subjects, 9 (9.28%) had decreased sodium levels, 49 (50.52%) had normal sodium levels, and 39 (40.21%) had increased sodium levels. The mean sodium level was 544.43 ± 785.31, with a median of 154.9, ranging from a minimum of 1.9 to a maximum of 3546.

Figure 3: Distribution of subjects according to 24-hour potassium.

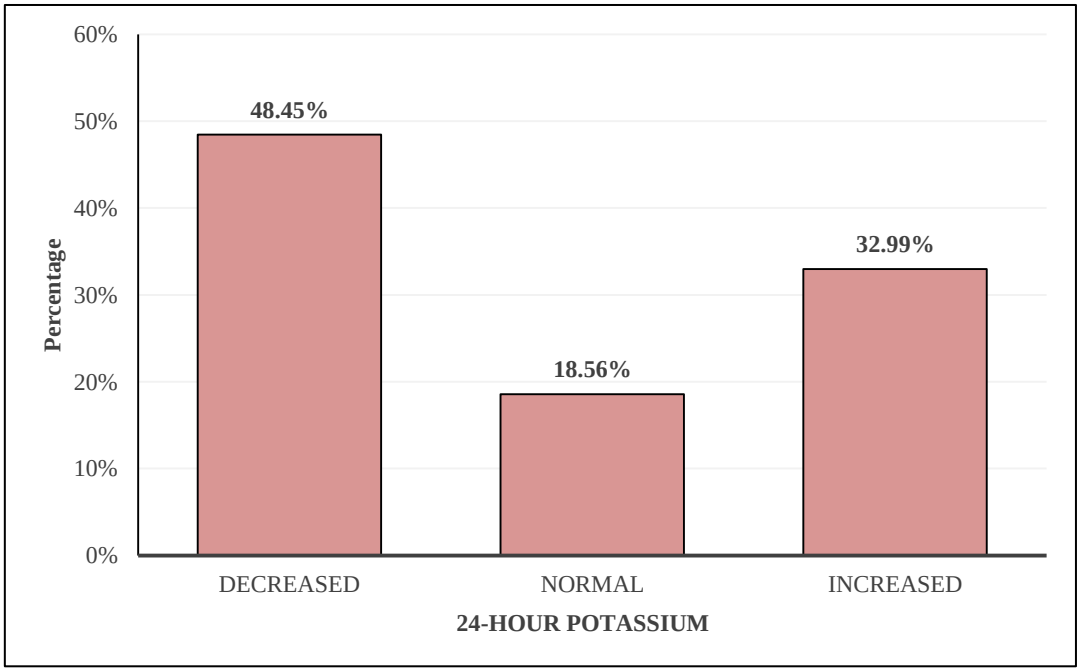

Table 4: Distribution of subjects according to 24-hour potassium.

24-hour Potassium	Number of subjects (%)
Decreased	47 (48.45%)
Normal	18 (18.56%)
Increased	32 (32.99%)
Mean ± SD Median (Min, Max)	113.88 ± 177.79 42.96 (2.1, 794)

Among the subjects, 47 (48.45%) had decreased potassium levels, 18 (18.56%) had normal potassium levels, and 32 (32.99%) had increased potassium levels. The mean potassium level was 113.88 ± 177.79, with a median of 42.96, and the levels ranged from a minimum of 2.1 to a maximum of 794.

Figure 4: Distribution of subjects according to habits.

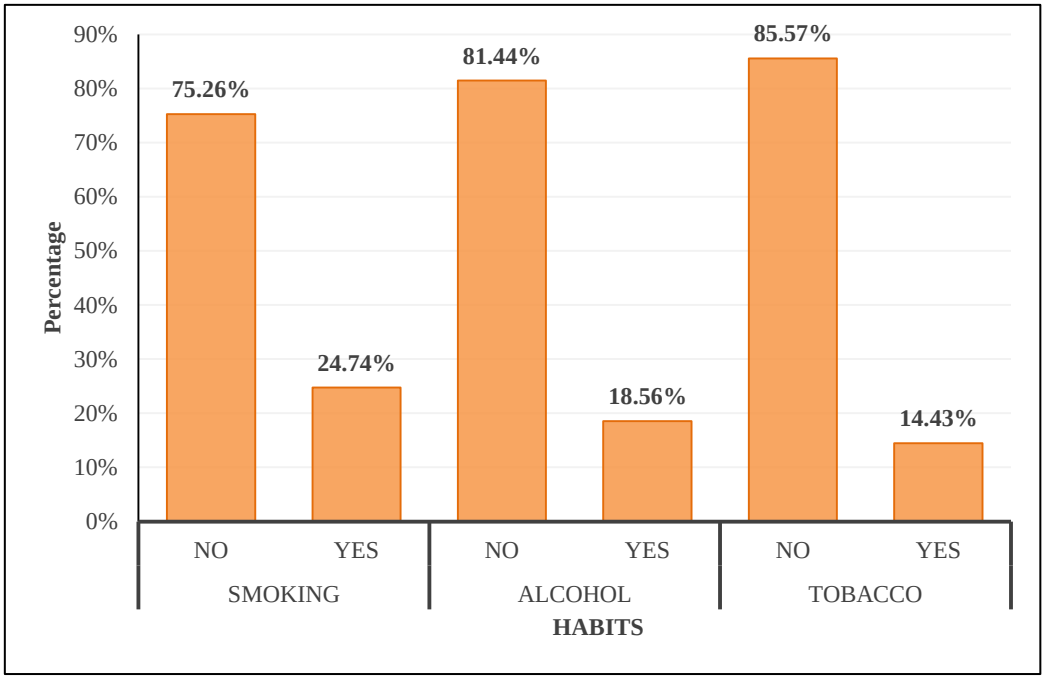

Table 5: Distribution of subjects according to habits.

Variables	Sub Category	Number of subjects (%)
Smoking	No	73 (75.26%)
	Yes	24 (24.74%)
Alcohol	No	79 (81.44%)
	Yes	18 (18.56%)
Tobacco	No	83 (85.57%)
	Yes	14 (14.43%)

A majority of participants, 73 (75.26%), reported not smoking, while 24 (24.74%) were smokers. Similarly, 79 (81.44%) did not consume alcohol, whereas 18 (18.56%) reported alcohol consumption. Regarding tobacco use, 83 (85.57%) subjects did not use tobacco, while 14 (14.43%) were tobacco users.

Figure 5: Distribution of subjects according to medical history.

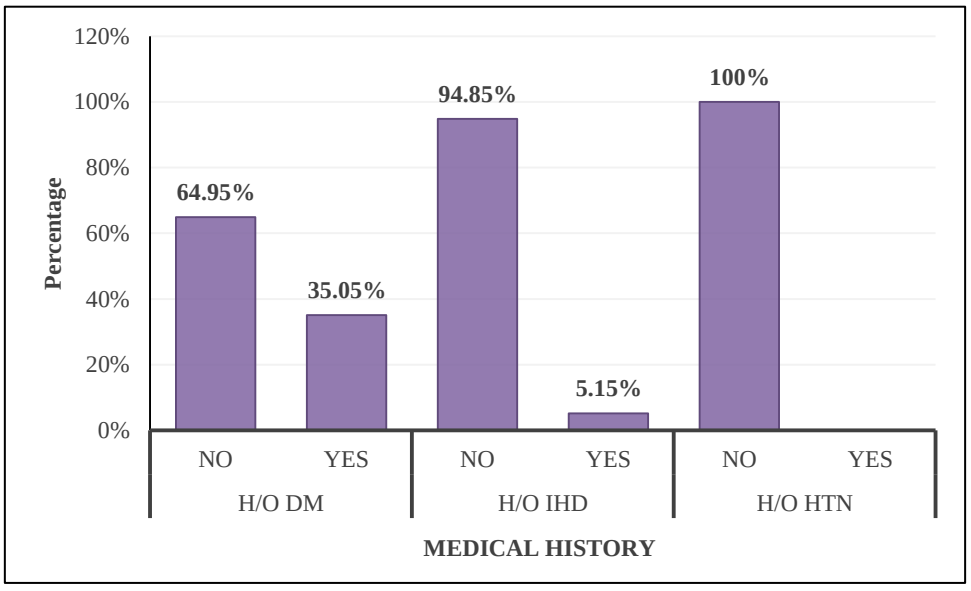

Table 6: Distribution of subjects according to medical history.

Variables	Sub Category	Number of subjects (%)
H/O DM	No	63 (64.95%)
	Yes	34 (35.05%)
H/O IHD	No	92 (94.85%)
	Yes	5 (5.15%)
H/O HTN	No	97 (100%)

A total of 63 (64.95%) participants had no history of diabetes mellitus (DM), while 34 (35.05%) reported a history of DM. Regarding ischemic heart disease (IHD) 92 (94.85%) subjects had no history of IHD, whereas 5 (5.15%) reported a history of the condition. Interestingly, none of the participants had a prior history of hypertension (HTN), with 97 (100%) subjects reporting no such history.

Figure 6: Scatter plot of 24-hour sodium with diastolic blood pressure.

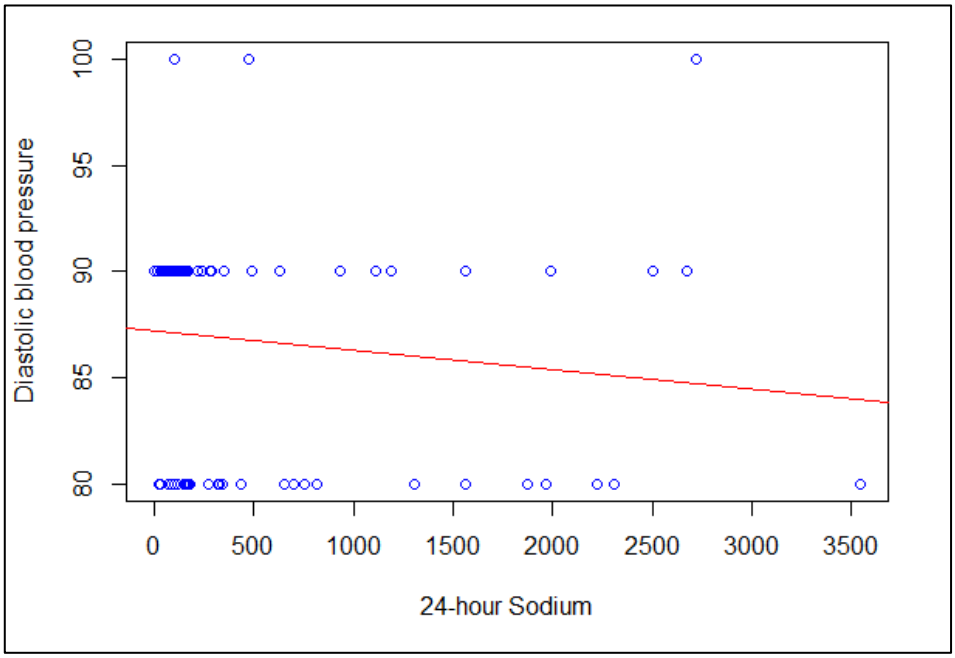

Figure 7: Scatter plot of 24-hour potassium with diastolic blood pressure.

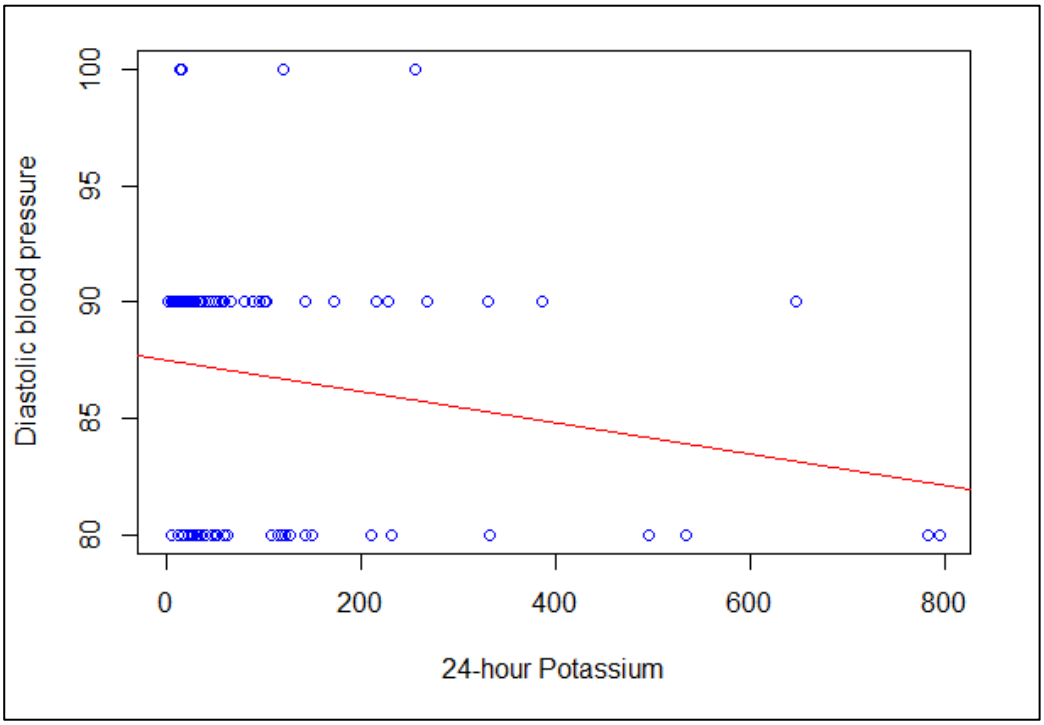

Figure 8: Scatter plot of 24-hour sodium with systolic blood pressure.

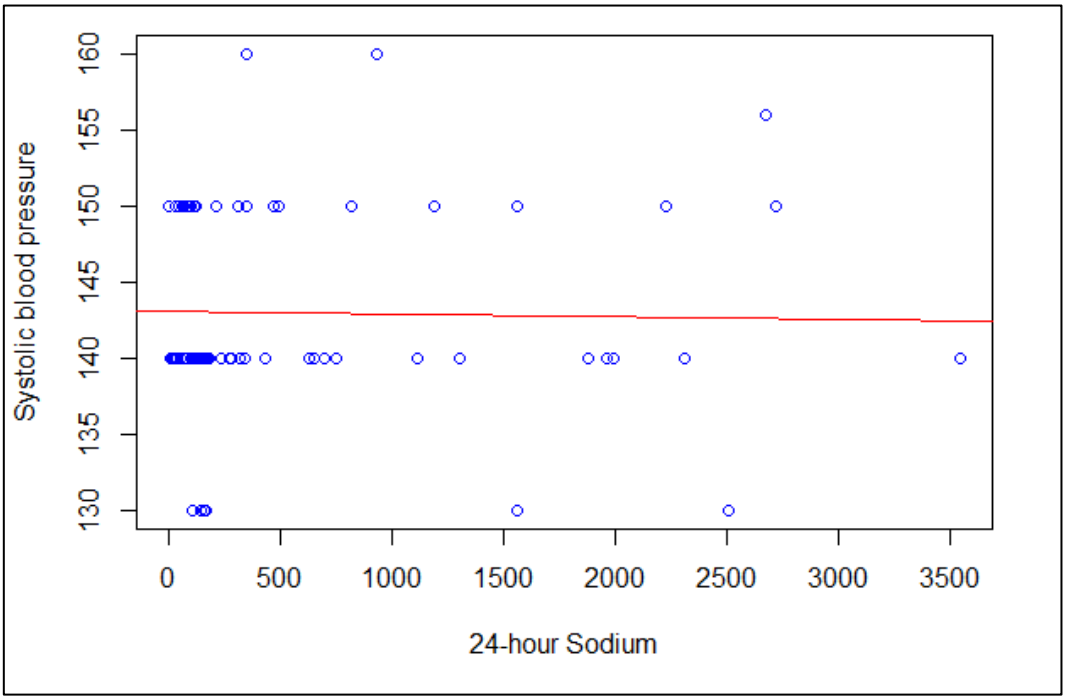

Figure 9: Scatter plot of 24-hour potassium with systolic blood pressure.

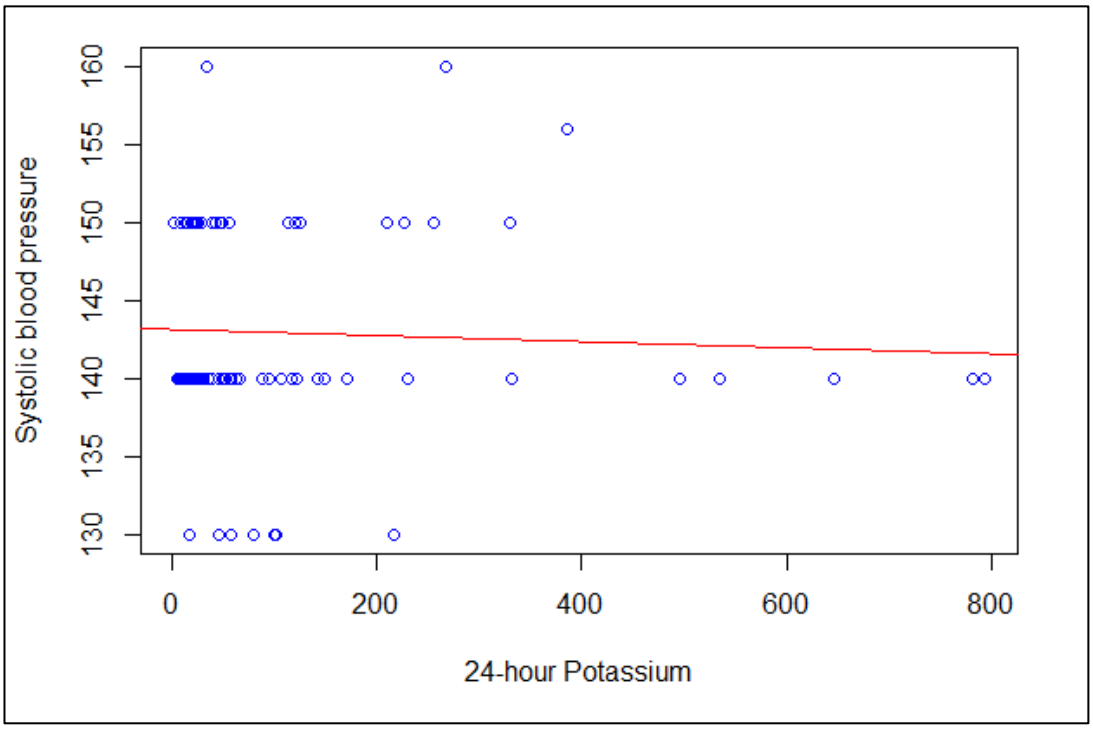

Table 7: Correlation of 24-hour sodium and potassium with BP.

Variables	Correlation coefficient	p-value [SP]
24-hour sodium & SBP	-0.0435	0.6723
24-hour sodium & DBP	-0.09	0.38
24-hour potassium & SBP	-0.0945	0.3573
24-hour potassium & DBP	-0.3054	**0.0023***

*Abbreviation: SP – Spearman's rank correlation test, * indicates statistical significance.*

The correlation between 24-hour sodium and systolic blood pressure (SBP) was weak and not statistically significant (Correlation coefficient = -0.0435, p-value = 0.6723). There was a non-significant negative correlation between 24-hour sodium and diastolic blood pressure (DBP) (Correlation coefficient = -0.09, p-value = 0.38). For 24-hour potassium, the correlation with SBP was also weak and not statistically significant (Correlation coefficient = -0.0945, p-value = 0.3573), but a significant negative correlation was observed with DBP (Correlation coefficient = -0.3054, p-value = 0.0023), indicating that higher potassium excretion may be associated with lower DBP.

Table 8: Comparison of different variables over 24-hour sodium.

Variables	Sub Category	24-hour sodium			p-value
		Decreased	Normal	Increased	
Age (years)	Mean ± SD Median (Min, Max)	69 ± 17.73 63 (40, 98)	57.43 ± 14.36 58 (27, 90)	57.36 ± 13.53 60 (24, 83)	0.0748[A]
Sex	Female	2 (22.22%)	20 (40.82%)	7 (17.95%)	0.0670[MC]
	Male	7 (77.78%)	29 (59.18%)	32 (82.05%)	
SBP	Mean ± SD Median (Min, Max)	143.33 ± 5 140 (140, 150)	142.45 ± 6.3 140 (130, 150)	143.49 ± 7.22 140 (130, 160)	0.8748[K]
DBP	Mean ± SD Median (Min, Max)	85.56 ± 5.27 90 (80, 90)	88.16 ± 5.27 90 (80, 100)	85.13 ± 6.01 80 (80, 100)	0.38 [K]
Smoking	No	7 (77.78%)	40 (81.63%)	26 (66.67%)	0.2849[MC]
	Yes	2 (22.22%)	9 (18.37%)	13 (33.33%)	
Alcohol	No	9 (100%)	38 (77.55%)	32 (82.05%)	0.3093[MC]
	Yes	0	11 (22.45%)	7 (17.95%)	
Tobacco	No	7 (77.78%)	40 (81.63%)	36 (92.31%)	0.3308[MC]
	Yes	2 (22.22%)	9 (18.37%)	3 (7.69%)	
H/O DM	No	6 (66.67%)	31 (63.27%)	26 (66.67%)	0.9550[MC]
	Yes	3 (33.33%)	18 (36.73%)	13 (33.33%)	
H/O IHD	No	8 (88.89%)	48 (97.96%)	36 (92.31%)	0.2814[MC]
	Yes	1 (11.11%)	1 (2.04%)	3 (7.69%)	
H/O HTN	No	9 (100%)	49 (100%)	39 (100%)	-

*Abbreviation: A – One way ANOVA, K – Kruskal Wallis test, MC – Chi square test with Monte Carlo simulation, * indicates statistical significance*

There is no significant difference in the distribution of age, sex, SBP, smoking, alcohol use, tobacco use, or the history of diabetes, ischemic heart disease, and hypertension across the 24-hour sodium groups.

Table 9: Comparison of different variables over 24-hour potassium.

Variables	Sub Category	24-hour potassium			p-value
		Decreased	Normal	Increased	
Age (years)	Mean ± SD Median (Min, Max)	58.87 ± 16.41 60 (27, 98)	60.33 ± 11.31 62 (40, 79)	56.84 ± 13.62 59.5 (24, 83)	0.7000[A]
Sex	Female	16 (34.04%)	7 (38.89%)	6 (18.75%)	0.2257[C]
	Male	31 (65.96%)	11 (61.11%)	26 (81.25%)	
SBP	Mean ± SD Median (Min, Max)	144.04 ± 5.77 140 (130, 160)	140.56 ± 7.25 140 (130, 150)	142.69 ± 7.01 140 (130, 160)	0.1967[K]
DBP	Mean ± SD Median (Min, Max)	88.09 ± 5.37 90 (80, 100)	85.56 ± 5.11 90 (80, 90)	85.31 ± 6.21 80 (80, 100)	0.0541[K]
Smoking	No	37 (78.72%)	13 (72.22%)	23 (71.88%)	0.7851[MC]
	Yes	10 (21.28%)	5 (27.78%)	9 (28.13%)	
Alcohol	No	40 (85.11%)	13 (72.22%)	26 (81.25%)	0.5177[MC]
	Yes	7 (14.89%)	5 (27.78%)	6 (18.75%)	
Tobacco	No	39 (82.98%)	16 (88.89%)	28 (87.5%)	0.8066[MC]
	Yes	8 (17.02%)	2 (11.11%)	4 (12.5%)	
H/O DM	No	29 (61.7%)	13 (72.22%)	21 (65.63%)	0.7253[C]
	Yes	18 (38.3%)	5 (27.78%)	11 (34.38%)	
H/O IHD	No	44 (93.62%)	18 (100%)	30 (93.75%)	0.7031[MC]
	Yes	3 (6.38%)	0	2 (6.25%)	
H/O HTN	No	47 (100%)	18 (100%)	32 (100%)	-

Abbreviation: A – One way ANOVA, K – Kruskal Wallis test, C – Chi square test, MC – Chi square test with Monte Carlo simulation.

None of the variables show any significant differences between the potassium groups.

DISCUSSION

Hypertension, commonly called "silent killer," is a critical health issue impacting more than a billion people globally. It significantly contributes to the incidence of cardiovascular diseases, strokes, chronic kidney conditions, and other severe health issues.[46] Despite progress in medical interventions, hypertension rates are still on the rise, especially in low and middle income nations in which the urban lifestyles and dietary habits have led to increased prevalence.[47] Among the various elements that affect blood pressure the intake of sodium and potassium through diet is crucial. This research sought to examine the relationship between 24 hour urine excretion of sodium and potassium and blood pressure levels in patients newly diagnosed with hypertension, aiming to offer valuable insights into the dietary and physiological factors that affect this condition.[48]

Our study revealed several critical findings regarding the relationship between urine sodium and potassium excretion and blood pressure in newly diagnosed hypertensive patients. The mean age of the study participants was 58.47 years with a majority being male (70.1%). The mean systolic blood pressure (SBP) was 142.95 mmHg, and the mean diastolic blood pressure (DBP) was 86.7 mmHg, indicating that the majority of participants had elevated blood pressure levels. The 24 hour urinary sodium and potassium excretion levels varied significantly among the participants, with 40.21% having increased sodium levels and 48.45% having decreased potassium levels.

The study found the correlation between urine sodium excretion and systolic blood pressure (SBP) was weak and not statistically significant (correlation coefficient = -0.0435, p-value = 0.6723). The INTERSALT study for instance,

demonstrated a strong positive correlation between sodium intake and blood pressure, with even small increases in sodium intake leading to significant rises in both SBP and DBP.[24]

The correlation between urine sodium excretion and diastolic blood pressure (DBP) was weak and not statistically significant (correlation coefficient = -0.09, p-value = 0.38). Indicating that sodium excretion does not show a clear relationship with DBP in this study population. These findings contrast with some studies that have reported significant associations between sodium intake and blood pressure. The variability in sodium handling among individuals could be a contributing factor to this result. Firstly, the participants were newly diagnosed hypertensive patients, and their sodium intake might not have been consistently high before diagnosis. Secondly, the body's ability to excrete sodium varies among individuals, and some participants might have had efficient sodium excretion mechanisms, leading to lower blood pressure despite higher sodium intake.[49]

The study also found a significant negative correlation between 24 hour urine potassium excretion and diastolic blood pressure (DBP), with a correlation coefficient of -0.3054 (p-value = 0.0023). Indicating that higher potassium excretion is associated with lower DBP. However, the correlation between potassium excretion and systolic blood pressure (SBP) was weak and not statistically significant (correlation coefficient = -0.0945, p-value = 0.3573). These findings coinside with previous research that has demonstrated the antihypertensive effects of potassium. Potassium promotes sodium excretion, reduces vascular resistance, and improves endothelial function, all of which contribute to reduced blood pressure.[50,51]

The inverse relationship present between potassium excretion and DBP underscores the importance of adequate potassium intake in managing hypertension.[52] The DASH (Dietary Approaches to Stop Hypertension) diet which is rich in fruits, vegetables, and low fat dairy products, has shown to significantly reduce blood pressure, partly due to its high potassium content.[53] The findings in this study suggest that increasing potassium intake, either through dietary changes or supplementation, could be an effective strategy for managing hypertension, particularly in populations with low potassium intake.

The sodium to potassium ratio has been proposed as a more effective indicator of blood pressure variations than the individual levels of sodium or potassium.[54] This study did not explicitly analyze the sodium to potassium ratio, but the findings regarding sodium as well as potassium excretion provide valuable insights. A high sodium-to-potassium ratio, often resulting from excessive sodium intake along with insufficient potassium intake, is linked to increased blood pressure and cardiovascular risk.[55] Therefore, public health interventions aimed at decreasing sodium intake and increasing potassium intake could help mitigate the burden of hypertension.

The findings of this study are consistent with several large-scale epidemiological studies that have explored the relationship between sodium, potassium, and blood pressure. The INTERSALT study, which included over 10,000 participants from 52 countries, demonstrated a positive correlation between sodium intake and blood pressure. The study found that a 100 mmol raise in sodium intake is associated with a 3-6 mmHg increase in SBP and a 0-3 mmHg increase in DBP.[24] Similarly, the DASH diet trials have shown that dietary patterns rich in potassium can significantly reduce blood pressure, with reductions of up to 7.62 mmHg in SBP and 4.22 mmHg in DBP.[53]

However, some studies have reported inconsistent findings regarding the relationship that is between the potassium excretion and blood pressure. For instance, a systematic review by Ziaei et al. (2020) found no significant correlation between 24 hour urine potassium excretion and blood pressure and hypertension risk. The discrepancies between studies could be due to variations in study design, population characteristics, and methods of assessing potassium intake and excretion.[30] A study by Mente et al. revealed that each 1-gram increase in sodium excretion was linked to rises of 2.11 mm Hg in systolic and 0.78 mm Hg in diastolic blood pressure. Higher sodium intake resulted in greater blood pressure increases: 2.58 mm Hg for over 5 grams, 1.74 mm Hg for 3 to 5 grams, and 0.74 mm Hg for less than 3 grams per day ($P<0.001$). Individuals with hypertension experienced a stronger reaction, with a 2.49 mm Hg increase per gram, compared to 1.30 mm Hg for those without. Age also played a role, with increases of 2.97 mm Hg for those over 55 years, 2.43 mm Hg for 45-55 years, and 1.96 mm Hg for those under 45 ($P<0.001$). Additionally, potassium excretion showed an inverse relationship with systolic blood pressure, particularly in hypertensive individuals and as age increased ($P<0.001$).[56]

The pathophysiological mechanisms underlying the relationship between sodium and potassium and blood pressure are complex and involve multiple systems, including the renin-angiotensin-aldosterone system (RAAS), endothelial function and vascular smooth muscle tone.[57] Sodium retention leads to increased extracellular fluid volume, which increases blood pressure by increasing cardiac output and peripheral resistance. Potassium, on the other hand, counteracts the effects of sodium by promoting natriuresis (sodium excretion) and reducing vascular resistance.[57]

The study's findings are particularly relevant in the context of global and regional variations in sodium and potassium intake. In many low- and middle-income countries, including India, sodium intake far exceeds the recommended levels, while potassium intake remains insufficient.[58] This dietary imbalance contributes to the high prevalence of hypertension in these regions. For example, the average sodium intake in India is approximately 8.0 grams per day, significantly higher than the WHO recommendation of 5 grams per day. In contrast, potassium intake in India is well below the recommended levels, exacerbating the risk of hypertension and cardiovascular diseases.[59]

India's diverse dietary patterns, characterized by high consumption of processed foods and traditional cooking methods, contribute to the high sodium intake observed in this study.[60] The lack of awareness about the health risks associated with high salt consumption further exacerbates the problem. Public health interventions aimed at reducing sodium intake and increasing potassium intake are urgently needed to address the growing burden of hypertension in India. These interventions should include educational campaigns, food labeling regulations, and policies to reduce the sodium content in processed foods.[61]

Globally, there are significant disparities in sodium and potassium intake, with some regions, such as East Asia, reporting particularly low potassium intake. The global average potassium intake is approximately 2.25 grams per day, well below the WHO recommendation of 3.5 grams per day. In contrast, regions such as Eastern Europe report higher potassium intake, which may contribute to lower hypertension rates in these areas.[62] These regional variations highlight the need for targeted public health interventions to address dietary imbalances and reduce hypertension risk.

At the policy level there is a need for comprehensive strategies, to reduce sodium intake and raise potassium intake at the population level. These strategies should include regulations to limit sodium content in processed foods mandatory food labeling to indicate sodium and potassium content, and public health campaigns to promote healthier dietary choices. Research has demonstrated that a high sodium to potassium ratio is a stronger predictor of cardiovascular risk than sodium or potassium intake alone. In India, where traditional diets are high in sodium and low in potassium therefore public health efforts should focus on achieving a balanced sodium-to-potassium ratio. Policymakers should consider initiatives such as sodium reduction strategies and potassium fortification programs to mitigate hypertension risk at the population level.

SUMMARY

This study examined the relationship between 24-hour urinary sodium and potassium excretion and blood pressure in newly diagnosed hypertensive patients.

40.21% of participants had high sodium excretion. However, the correlation between sodium excretion and both SBP and DBP was weak and not statistically significant, contrasting with studies like INTERSALT, which showed a strong link between sodium intake and blood pressure.

48.45% had low potassium excretion. A significant negative correlation was found between potassium excretion and DBP (correlation coefficient = -0.3054, p-value = 0.0023), but the correlation with SBP was weak. These findings support the role of potassium in lowering blood pressure.

While this study did not explicitly analyse the sodium-to-potassium ratio, previous research suggests that a high ratio is a stronger predictor of hypertension than sodium or potassium levels alone.

High sodium intake and low potassium intake are prevalent in many regions, including India, where average sodium intake exceeds WHO recommendations. Public health interventions should focus on reducing sodium intake, increasing potassium intake, and improving awareness through food labelling, regulations, and dietary modifications.

These findings reinforce the importance of dietary modifications, particularly increasing potassium intake, as a strategy to manage and prevent hypertension.

STRENGTHS

1. Using 24-hour urinary sodium and potassium excretion, which is a gold standard method for accurate dietary intake assessment.
2. Focusing on newly diagnosed hypertensive patients to eliminate the influence of long-term antihypertensive medications.
3. Assessment of sodium and potassium separately highlights the importance of both electrolytes in blood pressure regulation.

LIMITATIONS

1. Cross-sectional design: Limits the ability to establish causality between sodium, potassium, and blood pressure.
2. Study population: Consisted solely of newly diagnosed hypertensive patients, which may restrict the generalizability of the findings to other populations.
3. Patients were admitted and started on low salt diet, which may have influenced the 24 hour urinary sodium excretion.

CONCLUSION

This research provides insights into the impact of dietary sodium and potassium on blood pressure regulation. The significant negative correlation between potassium excretion and diastolic blood pressure suggests that higher potassium intake may contribute to better hypertension management. On the other hand, sodium excretion did not exhibit a strong correlation with systolic blood pressure, which could be influenced by individual variations in sodium metabolism and dietary habits.

Further research is necessary to explore the long term effects of dietary changes on blood pressure, particularly the sodium to potassium ratio as a more precise indicator of cardiovascular health. Additionally, investigating genetic and lifestyle factors that influence sodium and potassium metabolism could lead to more personalized dietary recommendations.

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ANNEXURES

ANNEXURE – I - INFORMED CONSENT FORM

"ASSOCIATION BETWEEN 24 HOUR URINARY SODIUM AND POTASSIUM EXCRETION AND BLOOD PRESSURE IN NEWLY DIAGNOSED HYPERTENSIVE SUBJECTS, A ONE YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE HOSPITAL."

Name of Student/Principal Investigator: _____

Name of Guide/Co Investigators: _____

Introduction:

Hypertension affects 1 billion people and is considered to be a leading cause of death, stroke, myocardial infarction, congestive heart failure, and chronic renal impairment.

Despite most populations exhibiting high sodium intake, only a few develop hypertension, which demonstrates an individual susceptibility taking into consideration- sodium intake, genetic background and environmental factors.

Diet, environmental factors, lifestyle, physical inactivity and genetics have been shown to contribute to the risk of non communicable diseases. Control of these primary risk factors could reduce the incidence of some non communicable diseases by up to 80%.

This study aims to correlate the urinary excretion of sodium and potassium in newly diagnosed hypertensive subjects.

Patients will be diagnosed with hypertension according to CDC guidelines. Patient

will then be explained about bottle collection of urine for 24 hours, after which 2 samples for urinary sodium and urinary potassium will be obtained. The results will then be analysed.

Explanation of procedure:

Patients will be diagnosed with hypertension according to CDC guidelines : Don't eat or drink anything 30 minutes before you take your blood pressure.

Sit in a comfortable chair with your back supported for at least 5 minutes before your reading.

Put both feet flat on the ground and keep your legs uncrossed.

Rest your arm with the cuff on a table at chest height.

Make sure the blood pressure cuff is snug but not too tight. The cuff should be against your bare skin, not over clothing.

Once diagnosed with hypertension (according to CDC criteria) patient will then be explained about bottle collection of urine for 24 hours. The results will be obtained after a day and are then analysed.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator**

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions:

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**ASSOCIATION BETWEEN 24 HOUR URINARY SODIUM AND POTASSIUM EXCRETION AND BLOOD PRESSURE IN NEWLY DIAGNOSED HYPERTENSIVE SUBJECTS, A ONE YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE HOSPITAL**." My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE – II

PROFORMA

CASE NO	
NAME	
IP NO	
AGE	**YEARS**
SEX	**MALE FEMALE**
ADDRESS	
OCCUPATION	

Complaints at presentation	
Past history	
Family history	
Personal history	
Treatment history	

Vitals :

Temperature	
Pulse	
Respiratory rate	
Blood pressure	

PHYSICAL EXAMINATION:

	Yes	**No**
Pallor		
Icterus		
Lymphadenopathy		
Cyanosis		
Clubbing		
Edema		

SYSTEMIC EXAMINATION:

C.V.S	
R.S.	
C.N.S	
PER ABDOMEN	

INVESTIGATIONS:

24 hour urinary sodium	
24 hour urinary potassium	

ANNEXURE: III- MASTER CAHRT

SR NO	NAME	IP NO	AGE	SEX	1ST BP	2ND BP	3RD BP	24 HOUR SODIUM	24 HOUR POTASSIUM	SMOKING	ALCOHOL	TOBACCO	H/O DM	H/O IHD	H/O HTN
1	RAMGODA	10074854	45	MALE	150/90	140/90	156/90	2673	386.1	NO	YES	NO	NO	NO	NO
2	RAJENDRA	10074592	44	MALE	140/90	140/90	150/90	347.6	56.98	YES	NO	NO	YES	NO	NO
3	SHIVAPPA	10074623	58	MALE	140/90	150/80	140/80	1965	149.2	NO	YES	NO	NO	NO	NO
4	APPAYYA	10072230	75	MALE	170/100	160/100	160/90	352.4	34.66	YES	NO	NO	YES	NO	NO
5	APPASAHEB	10073882	60	MALE	150/90	140/90	140/80	24.65	12.31	NO	NO	NO	NO	NO	NO
6	GURUNATH	10067694	63	MALE	150/90	150/80	140/80	2308	495	NO	NO	NO	NO	NO	NO
7	LAXMAN	10069124	69	MALE	150/90	140/90	140/80	3546	533.8	YES	NO	NO	NO	NO	NO
8	SIDRAI	10075027	83	MALE	170/100	150/100	150/100	2720	255	YES	NO	NO	YES	NO	NO
9	LATHA	10075179	38	FEMALE	150/90	150/80	140/80	180	143	NO	NO	NO	NO	NO	NO
10	CHANDRASHEKHAR	10071693	62	MALE	170/110	170/90	160/90	930	268	NO	YES	NO	NO	NO	NO
11	SHAILAJA	10076396	63	MALE	150/90	140/90	140/80	272.25	51.87	NO	NO	NO	NO	NO	NO
12	KEMPANNA	10074226	62	MALE	150/100	150/90	140/90	236.5	11.03	YES	NO	NO	YES	YES	NO
13	VITTAL	10080928	56	MALE	160/100	160/90	150/90	85.79	25.26	YES	YES	NO	NO	NO	NO
14	NILAVVA	10076316	84	FEMALE	150/90	150/80	140/80	150.6	21.9	NO	NO	NO	YES	NO	NO
15	RUDRAPPA	10080092	61	MALE	150/90	150/80	150/90	69.4	15.09	YES	NO	NO	YES	NO	NO
16	NIRMALA	10081230	58	FEMALE	150/90	140/90	140/90	55.7	53.7	NO	NO	NO	NO	NO	NO
17	RATNA	10079441	60	MALE	150/90	150/90	150/90	33.84	26.59	NO	NO	NO	YES	NO	NO
18	ESHAWANT	10078930	61	MALE	150/90	140/90	140/90	161.1	14	NO	NO	NO	YES	NO	NO
19	RAJSHEKHAR	100803488	89	MALE	140/90	130/90	140/90	39.9	15.02	NO	NO	YES	NO	NO	NO
20	CHANDRASHEKHAR	10080985	61	MALE	160/100	150/90	140/80	167.57	46.24	YES	YES	NO	YES	NO	NO
21	VIVEK	10081078	27	MALE	140/90	146/92	140/90	75.12	8.26	NO	NO	NO	NO	NO	NO
22	PRAKASH	10080931	51	MALE	150/90	150/90	140/90	285.5	95.88	YES	NO	YES	NO	NO	NO
23	RUDRAVVA	10081173	62	FEMALE	140/90	150/90	150/90	103.2	13.27	NO	NO	NO	YES	NO	NO
24	VINODINI	10081049	74	FEMALE	160/90	150/90	150/90	92.17	24.86	NO	NO	NO	NO	NO	NO
25	SHIVANGOUDA	10079729	62	MALE	140/90	140/90	130/90	158.8	17.3	YES	NO	YES	NO	NO	NO
26	VEERAPPA	10080104	76	MALE	170/100	160/90	150/90	1.9	2.1	YES	NO	NO	YES	YES	NO
27	RAMESH	10080392	52	MALE	160/100	150/90	150/100	101.4	14.8	YES	NO	NO	NO	NO	NO
28	BALAPPA	10079101	52	MALE	140/90	150/90	140/90	82.8	29.8	NO	NO	YES	YES	NO	NO
29	RUDRAVVA	10081173	62	FEMALE	150/90	150/90	150/100	103.2	13.27	NO	NO	NO	YES	NO	NO
30	SHIVANAND	10080964	47	MALE	140/90	130/90	140/80	342	35.15	NO	YES	NO	NO	NO	NO
31	MARUTI	10079623	52	MALE	150/100	140/90	140/80	323.55	121.58	NO	NO	NO	NO	NO	NO
32	VENKATESH	10080809	63	MALE	140/90	140/80	140/90	13.1	6.42	YES	NO	NO	NO	NO	NO
33	POOJA	10081543	58	FEMALE	150/90	140/90	140/90	53.27	5	NO	NO	NO	YES	NO	NO
34	EXPRANCE	10080522	52	FEMALE	140/90	140/80	130/90	168.8	58.4	NO	NO	NO	NO	NO	NO
35	SHANKAR	10074232	73	MALE	140/90	140/80	130/90	2505	99.6	YES	NO	NO	YES	YES	NO
36	NILAVVA	10076316	84	FEMALE	140/100	140/90	140/90	150.6	21.9	NO	NO	NO	YES	NO	NO
37	DASTAGIR	10072680	59	MALE	160/100	160/100	150/80	1560	210	NO	NO	NO	YES	NO	NO
38	BHAVAKANNA	10075369	40	MALE	150/90	150/80	150/90	217.8	18.7	NO	NO	NO	NO	NO	NO
39	RAVI	10074295	60	MALE	150/90	150/80	140/90	1989	646	NO	NO	NO	NO	NO	NO
40	KUTUBUDDIN	10075483	41	MALE	150/90	150/90	140/90	278	20.83	YES	NO	NO	NO	NO	NO
41	ABDUL	10075709	50	MALE	160/100	150/100	150/90	490.5	330	NO	YES	NO	NO	NO	NO
42	SHIVANAND	10074952	52	MALE	150/90	150/90	140/80	116.2	26.3	NO	NO	NO	NO	NO	NO
43	KAVITA	10070983	54	FEMALE	150/90	140/90	140/80	1875	781.5	NO	NO	NO	NO	NO	NO
44	SHIVANAND	10074294	34	MALE	150/90	150/80	140/90	76.5	172	NO	NO	NO	NO	NO	NO
45	SHOBHA	10078998	67	FEMALE	140/90	140/90	150/90	98.4	42.96	NO	NO	NO	YES	NO	NO

46	SHAILAJA	10076396	63	FEMALE	150/80	140/80	140/80	272.25	51.87	NO	NO	NO	YES	NO	NO
47	HANAMANT	10081276	56	MALE	160/100	150/90	140/90	42.7	10.05	NO	YES	NO	NO	NO	NO
48	MAHADEVI	10078054	52	FEMALE	140/90	150/100	140/80	118	30.71	NO	NO	NO	YES	NO	NO
49	BHARAMHGOUDA	10081234	90	MALE	150/90	140/90	140/80	143	22.22	NO	NO	YES	YES	NO	NO
50	NAGAPPA	10081383	72	MALE	150/100	140/80	140/80	111.72	26.78	NO	NO	NO	NO	NO	NO
51	LAXMI	10080746	42	FEMALE	150/100	150/90	140/90	137.76	22.1	NO	NO	NO	NO	NO	NO
52	TANAJI	10074659	45	MALE	140/90	140/90	140/80	650	230	NO	NO	NO	YES	NO	NO
53	NAGAPPA	10074577	67	MALE	140/90	140/90	140/80	697.5	62.3	NO	YES	NO	NO	NO	NO
54	BALAPPA	10074330	67	MALE	170/100	160/100	150/90	1188	227.7	NO	NO	NO	NO	NO	NO
55	SHIVABABU	10074581	31	MALE	140/90	140/80	130/90	1560	216	NO	NO	NO	NO	NO	NO
56	MALLIKARJUN	10080418	48	MALE	140/90	140/80	130/90	148.32	80	YES	NO	NO	NO	NO	NO
57	VIJENDRA	10080778	54	MALE	150/90	140/90	150/80	70.83	114.1	YES	YES	YES	NO	NO	NO
58	MARUTI	10081142	98	MALE	150/90	150/90	140/90	29.9	6.96	NO	NO	NO	NO	NO	NO
59	SIDAPPA	10072126	70	MALE	140/90	140/80	140/80	112	39.7	NO	YES	NO	NO	NO	NO
60	SUBHADRA	10080614	78	FEMALE	160/100	160/90	150/90	122.15	20.79	NO	NO	NO	NO	NO	NO
61	MALLIKARJUN	10081820	79	MALE	170/100	150/90	140/90	172.44	50.12	NO	YES	NO	NO	NO	NO
62	SANTOSH	10082030	36	MALE	140/90	150/100	140/90	79.4	8.15	NO	YES	YES	NO	NO	NO
63	SUNITA	10080825	40	FEMALE	160/100	150/100	150/90	116	8.24	NO	NO	NO	NO	NO	NO
64	VIRUPAXI	10081155	60	MALE	140/90	140/80	130/90	110.2	45.7	NO	YES	NO	NO	NO	NO
65	KIRAN	10081229	35	MALE	150/100	150/90	140/90	122	23.7	NO	NO	NO	NO	NO	NO
66	KAREPPA	10081240	69	MALE	160/100	150/80	140/80	102	117	YES	NO	YES	YES	NO	NO
67	BASAVARAJ	10081570	70	MALE	140/90	140/80	150/90	47.1	8.1	NO	YES	NO	NO	NO	NO
68	ROOPA	10081574	43	FEMALE	140/90	150/90	140/80	163	50.76	NO	NO	NO	NO	NO	NO
69	APPAYAPPA	10081468	76	MALE	150/100	150/90	140/90	631	143	YES	NO	YES	NO	NO	NO
70	GOPAL	10074550	66	MALE	140/90	140/90	140/80	1307	794	NO	NO	NO	YES	NO	NO
71	PARVATI	10079899	70	FEMALE	160/90	150/90	150/100	472	120	NO	NO	NO	YES	NO	NO
72	VIDYA	10081129	36	FEMALE	140/90	150/90	150/80	819	21.9	NO	NO	NO	NO	NO	NO
73	SUVIDHA	10081227	24	FEMALE	140/90	150/90	150/80	2227	126	NO	NO	NO	NO	NO	NO
74	HETAL	10081504	77	FEMALE	150/100	140/80	140/90	106	8	NO	NO	NO	NO	NO	NO
75	TULASABAI	10079836	62	FEMALE	160/90	150/90	150/90	64.53	38.9	NO	NO	YES	NO	YES	NO
76	RAMU	10077688	74	MALE	150/90	150/90	150/80	314	47.25	YES	NO	YES	NO	NO	NO
77	IRAPPA	10077633	45	MALE	150/90	140/90	140/90	128.2	20.3	NO	NO	NO	NO	NO	NO
78	PREMA	10074299	58	FEMALE	140/90	140/80	140/80	32.2	5.19	NO	NO	NO	YES	NO	NO
79	SADANAND	10072937	60	MALE	160/100	160/90	140/80	750	332	YES	YES	NO	NO	NO	NO
80	GURUNATH	10082252	59	MALE	140/90	140/80	140/80	80.7	17.7	YES	NO	YES	YES	NO	NO
81	MARUTI	10079551	77	MALE	150/90	150/80	140/80	18.85	57.98	NO	NO	YES	NO	NO	NO
82	PRASANNA	10081803	41	MALE	130/90	140/80	150/90	122	28.8	NO	YES	NO	NO	NO	NO
83	SHALAN	10082304	40	FEMALE	150/90	150/80	150/80	29.9	49.8	NO	NO	NO	NO	NO	NO
84	SUVIDHA	10081237	44	FEMALE	140/90	150/90	150/80	2227	126	NO	NO	NO	NO	NO	NO
85	VIRUPAXI	10081234	60	MALE	140/90	140/80	130/90	110.2	45.7	NO	YES	NO	NO	NO	NO
86	SHOBHA	10079998	67	FEMALE	140/90	140/90	150/90	98.4	42.96	NO	NO	NO	YES	NO	NO
87	RAVI	10076543	60	MALE	150/90	150/80	140/90	1989	646	NO	NO	NO	NO	NO	NO
88	SHANKAR	10068763	73	MALE	140/90	140/80	130/90	2505	102	YES	NO	NO	YES	YES	NO
89	RUDRAVVA	10098765	62	FEMALE	150/90	150/90	150/100	103.2	13.27	NO	NO	NO	YES	NO	NO
90	APPASAHEB	19987654	60	MALE	150/90	140/90	140/80	432.2	107	NO	NO	NO	NO	NO	NO
91	BHAVAKANNA	10074567	40	MALE	150/90	150/80	150/90	217.8	18.7	NO	NO	NO	NO	NO	NO
92	GOPAL	10065432	66	MALE	140/90	140/90	140/80	1307	794	NO	NO	NO	YES	NO	NO
93	POOJA	10081678	58	FEMALE	150/90	140/90	140/90	154.9	88	NO	NO	NO	YES	NO	NO
94	TANAJI	10074677	45	MALE	140/90	140/90	140/80	650	230	NO	NO	NO	YES	NO	NO
95	VENKATESH	10080902	63	MALE	140/90	140/80	140/90	1113	66.42	YES	NO	NO	NO	NO	NO
96	VIDYA	10089089	36	FEMALE	140/90	150/90	150/80	819	21.9	NO	NO	NO	NO	NO	NO
97	BALAPPA	10087625	52	MALE	140/90	150/90	140/90	82.8	29.8	NO	NO	YES	YES	NO	NO