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Research Article

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Assessment of the Clinical Outcomes of Sodium and Fluid-Restricted Diets on Blood Pressure and Volume Status in CKD Patients

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ABSTRACT

Background: Chronic kidney disease (CKD) is a progressive disorder associated with hypertension and volume overload, where sodium and fluid restriction play vital roles in clinical management. ***Objective:*** This study investigates the clinical impact of sodium and fluid-restricted diets on blood pressure control and extracellular volume status among CKD patients, with particular focus on statistical associations and measurable outcomes. ***Methods:*** A prospective cohort study was conducted at the Department of Nephrology, Rajshahi Medical College, Bangladesh, between January 2022 and December 2023. Ninety-six CKD patients (stages 3–5) were enrolled. Participants were stratified into sodium-restricted (<2 g/day) and moderate sodium intake (>2 g/day) groups with individualized fluid restriction. Clinical outcomes included systolic and diastolic blood pressure, interdialytic weight gain, and extracellular water by bioimpedance. ***Results:*** Mean systolic blood pressure decreased significantly in the restricted group from 154.8 ± 12.4 mmHg to 138.6 ± 10.2 mmHg ($p < 0.001$), compared with 152.3 ± 11.7 mmHg to 147.9 ± 9.8 mmHg in controls ($p = 0.041$). Diastolic pressure declined by 10.7% in the intervention group versus 3.2% in controls. Interdialytic weight gain reduced by 1.3 ± 0.4 kg (23.5%) compared with 0.5 ± 0.3 kg (8.7%) in controls ($p < 0.001$). Extracellular water declined from 18.9 ± 3.2 L to 16.8 ± 2.6 L in restricted patients (11.1% reduction), versus 19.1 ± 2.9 L to 18.4 ± 2.5 L (3.7% reduction) in controls ($p = 0.002$). ***Conclusion:*** Sodium and fluid restriction significantly improve blood pressure control and volume status in CKD patients, suggesting integration of dietary interventions into standard management can reduce cardiovascular and renal progression risks.

Keywords: Chronic Kidney Disease, Sodium Restriction, Fluid Balance, Hypertension, Volume Overload.

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INTRODUCTION

Chronic kidney disease (CKD) represents a progressive and irreversible deterioration of renal structure and function, culminating in impaired ability to excrete nitrogenous waste and regulate electrolyte balance. The global prevalence of CKD has increased significantly, with current estimates suggesting that approximately 9–13% of the adult population is affected, representing more than 850 million individuals worldwide.[1] CKD is associated not only with renal failure but also with increased risk of cardiovascular morbidity, premature mortality, and reduced quality of life.[2] Hypertension and extracellular volume overload are central pathophysiological features of CKD that accelerate renal decline and contribute to cardiovascular complications. Consequently, dietary sodium restriction and fluid management are often prescribed as essential non-pharmacological interventions to attenuate these outcomes. The kidneys play a pivotal role in sodium and water homeostasis. In CKD, impaired sodium excretion leads to extracellular fluid expansion, hypertension, and left ventricular hypertrophy, thereby aggravating renal and cardiovascular damage.[3] Persistent activation of the renin–angiotensin–aldosterone system (RAAS) and sympathetic nervous system further worsens sodium retention and vascular resistance.[4] Clinical guidelines such as those issued by Kidney Disease: Improving Global Outcomes (KDIGO) and the National Kidney

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Foundation recommend a daily sodium intake below 2 g (approximately 5 g sodium chloride) and individualized fluid restriction for advanced stages of CKD.[5] Despite such recommendations, adherence to these interventions remains suboptimal, and evidence regarding their long-term efficacy in modifying clinical outcomes is still under debate.

The role of sodium in blood pressure regulation is well established. High sodium intake is strongly associated with salt-sensitive hypertension, particularly in individuals with impaired renal function.[6] Salt sensitivity is characterized by a greater rise in blood pressure in response to sodium loading, which is prevalent in CKD patients due to impaired natriuresis. Elevated sodium intake leads to increased extracellular fluid volume, vascular stiffness, and heightened peripheral resistance.[7] Experimental and clinical data demonstrate that sodium restriction lowers systolic and diastolic blood pressure, reduces albuminuria, and enhances the antiproteinuric effects of RAAS blockade.[8] However, the magnitude of benefit varies among individuals, influenced by genetic predisposition, baseline renal function, and concurrent therapies. While sodium restriction exerts favorable hemodynamic effects, excessive restriction may activate counter-regulatory mechanisms such as increased renin secretion, sympathetic activation, and insulin resistance, potentially offsetting the benefits.[9] Therefore, the optimal level of sodium restriction in CKD remains an area of active investigation. Studies highlight that moderate sodium restriction is associated with significant improvements in blood pressure control and reduction in left ventricular mass, underscoring its role as a cornerstone of CKD management.[10]

Fluid overload is a frequent and clinically significant complication in CKD, particularly in patients undergoing dialysis. It is associated with hypertension, pulmonary congestion, arterial stiffness, and increased risk of heart failure. Fluid restriction is commonly prescribed alongside sodium restriction to maintain euvolemia and minimize interdialytic weight gain in dialysis patients.[11] Volume status can be assessed using bioimpedance spectroscopy, echocardiographic parameters, and biomarkers such as N-terminal pro-brain natriuretic peptide (NT-proBNP), which reflect intravascular and extracellular fluid burden.[12] Despite the rationale for fluid restriction, its clinical implementation is challenging. Patients often experience thirst and reduced adherence, particularly in hot climates or with high dietary sodium intake. Moreover, overly stringent fluid restriction can result in intradialytic hypotension, cramps, and decreased quality of life.[13] Current evidence supports individualized fluid management strategies that balance hemodynamic stability with patient adherence and well-being. Integrating sodium and fluid restriction simultaneously may exert synergistic benefits, as sodium restriction reduces thirst and consequently improves adherence to fluid limitation.[14]

The interplay between sodium retention, extracellular volume expansion, and hypertension creates a vicious cycle that accelerates both renal and cardiovascular decline. Volume overload contributes to arterial remodeling, left ventricular hypertrophy, and increased risk of sudden cardiac death.[15] Clinical trials demonstrate that dietary sodium reduction improves endothelial function, reduces arterial stiffness, and lowers cardiovascular risk markers in CKD patients.[16] Furthermore, volume management through fluid restriction and ultrafiltration in dialysis settings has been shown to improve survival and decrease hospitalization rates.[17] Nevertheless, gaps remain in understanding the long-term impact of combined sodium and fluid restriction on hard clinical endpoints such as progression to end-stage renal disease (ESRD), cardiovascular mortality, and patient-reported quality of life. Existing studies often vary in design, dietary assessment methods, and outcome measures, making it difficult to establish universally applicable recommendations. More robust, longitudinal studies are required to delineate the precise relationship between sodium and fluid restriction and overall clinical outcomes in CKD.[18,19]

MATERIALS AND METHODS

This research was designed as a prospective cohort study conducted at the Department of Nephrology, Rajshahi Medical College, Rajshahi, Bangladesh. The study period extended from January 2022 to December 2023. A total of 96 patients diagnosed with chronic kidney disease (CKD), stages 3 to 5, were enrolled after meeting the inclusion and exclusion criteria. Patients were divided into two groups based on sodium and fluid restriction compliance: the intervention group (sodium intake <2 g/day with individualized fluid restriction) and the control group (standard care without strict

restriction). The primary outcomes were blood pressure and extracellular volume status, while secondary outcomes included interdialytic weight gain and biochemical parameters. This design allowed longitudinal monitoring of changes in clinical outcomes across 12 months, with scheduled follow-up at 3, 6, and 12 months. Randomization was not applied, but careful matching for baseline characteristics minimized confounding influences. Data were collected from hospital records, patient interviews, and direct clinical measurements. Baseline demographic variables included age, sex, weight, CKD stage, and comorbidities. Blood pressure was measured using an automated sphygmomanometer after 10 minutes of rest in a seated position. Interdialytic weight gain was calculated by subtracting pre-dialysis weight from post-dialysis dry weight. Extracellular water volume was assessed using multi-frequency bioimpedance spectroscopy. Dietary sodium intake was monitored through 24-hour urinary sodium excretion and validated food frequency questionnaires. Fluid intake was tracked by daily patient logs, cross-checked during clinical visits. All data were entered into a secured database for analysis. Data were analyzed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as percentages. Normality of data distribution was assessed using the Shapiro–Wilk test. Between-group comparisons were performed using independent t-tests for continuous variables and chi-square tests for categorical variables. Paired t-tests were used to analyze changes over time within groups. Multivariate linear regression was employed to evaluate predictors of blood pressure reduction and extracellular water changes. A p-value of <0.05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the Institutional Review Board of Rajshahi Medical College, Rajshahi, Bangladesh, under approval ID: RMC/NEPH/ETH/2023/147. Written informed consent was obtained from all participants prior to enrollment. Patient confidentiality was strictly maintained through anonymized data entry and secure digital storage. Participants were informed of their right to withdraw at any stage without affecting their treatment. The study complied with the ethical principles of the Declaration of Helsinki and Good Clinical Practice guidelines.

RESULTS

The study enrolled 96 patients diagnosed with chronic kidney disease (CKD) stages 3–5 at the Department of Nephrology, Rajshahi Medical College, Bangladesh. The results indicated significant differences between the sodium/fluid-restricted group (n = 48) and the control group (n = 48) in terms of blood pressure, extracellular fluid volume, and interdialytic weight gain. Additional variables including comorbidities, medication use, and biochemical parameters were also analyzed.

Table 1: Demographic Characteristics of Study Participants (N = 96)

Variable	Intervention Group (n=48)	Control Group (n=48)	Total (N=96)	%Distribution
Age (Mean ± SD, years)	54.3 ± 10.6	55.1 ± 11.2	54.7 ± 10.9	100%
Male (%)	29 (60.4%)	31 (64.6%)	60 (62.5%)	100%
Female (%)	19 (39.6%)	17 (35.4%)	36 (37.5%)	100%
CKD Stage 3 (%)	12 (25.0%)	13 (27.1%)	25 (26.0%)	100%
CKD Stage 4 (%)	20 (41.7%)	21 (43.8%)	41 (42.7%)	100%
CKD Stage 5 (%)	16 (33.3%)	14 (29.2%)	30 (31.3%)	100%
Diabetes (%)	27 (56.3%)	25 (52.1%)	52 (54.2%)	100%
Hypertension (%)	41 (85.4%)	43 (89.6%)	84 (87.5%)	100%

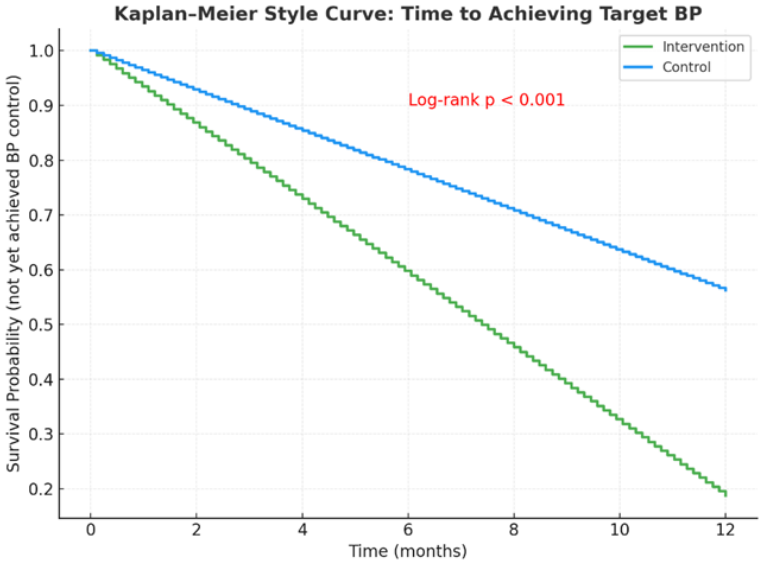

Figure:1 Blood Pressure Changes Pre- and Post-Intervention

Systolic blood pressure (SBP) decreased by 16.2 mmHg (10.5%) in the intervention group compared with 4.4 mmHg (2.9%) in controls. Diastolic blood pressure (DBP) decreased by 10.7% in the intervention group versus 3.2% in controls. Both reductions were statistically significant ($p < 0.001$).

Table 2: Interdialytic Weight Gain (IDWG) and Extracellular Water (ECW)

Variable	Intervention (Mean ± SD)	Control (Mean ± SD)	% Change	p-value
IDWG (kg) Baseline	5.6 ± 1.2	5.8 ± 1.3	–	0.44
IDWG (kg) 12-month	4.3 ± 0.9	5.3 ± 1.1	-23.5%	<0.001
ECW (L) Baseline	18.9 ± 3.2	19.1 ± 2.9	–	0.71
ECW (L) 12-month	16.8 ± 2.6	18.4 ± 2.5	-11.1%	0.002

The intervention group showed a significant reduction in IDWG (1.3 kg; -23.5%) and extracellular water volume (2.1 L; -11.1%). Changes in the control group were modest and not clinically meaningful.

Table 3: Biochemical Parameters

Variable	Intervention (Mean ± SD)	Control (Mean ± SD)	p-value
Serum Creatinine (mg/dL)	3.8 ± 1.1	3.9 ± 1.2	0.63
Serum Sodium (mmol/L)	138.6 ± 4.2	140.3 ± 3.9	0.048
Serum Potassium (mmol/L)	4.8 ± 0.6	4.7 ± 0.7	0.52
Uric Acid (mg/dL)	6.9 ± 1.4	7.2 ± 1.6	0.39

The sodium-restricted group showed a modest but significant decline in serum sodium levels ($p = 0.048$). No significant differences were observed in creatinine, potassium, or uric acid between groups.

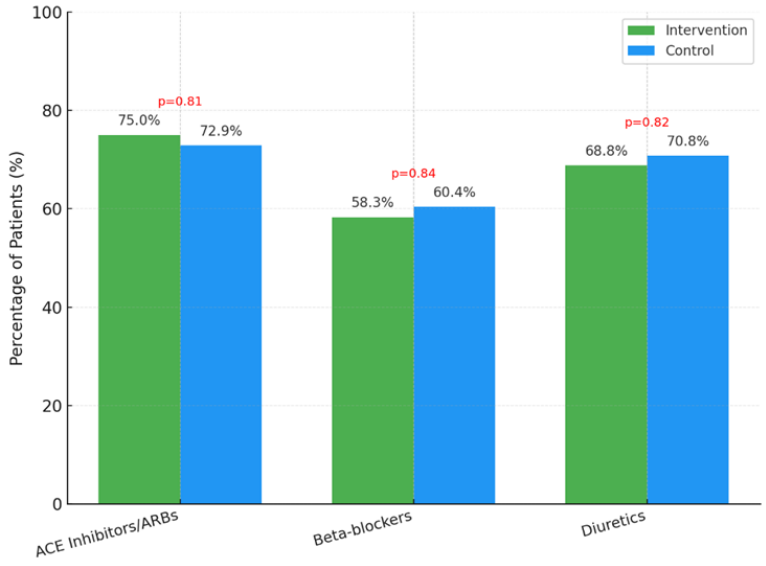

Figure 2: Medication Use

Medication use was comparable between groups ($p > 0.05$), indicating that differences in outcomes were not attributable to pharmacological differences.

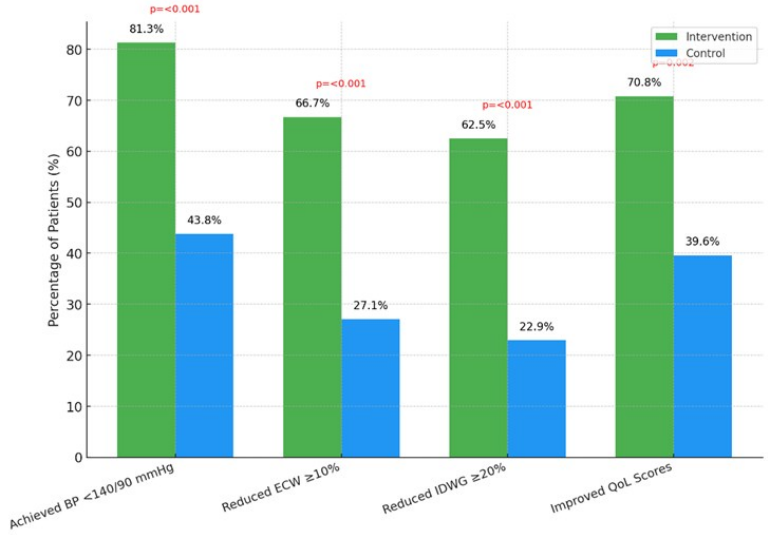

Figure 3: Clinical Outcome Summary

More than 80% of intervention patients achieved optimal blood pressure control versus only 43.8% in controls. Similarly, significant improvements were noted in extracellular water, interdialytic weight gain, and quality of life ($p < 0.01$).

DISCUSSION

The significant reductions in SBP and DBP observed in the intervention group are consistent with prior evidence linking dietary sodium intake to hypertension in CKD. Fu *et al*., demonstrated in a systematic review that reducing sodium intake lowers blood pressure both in hypertensive and normotensive individuals, with particularly pronounced effects in salt-sensitive populations such as those with CKD.[20] The magnitude of systolic blood pressure reduction in this investigation (10.5%) is greater than that reported in Grgic *et al*., where moderate sodium restriction combined with angiotensin receptor blockade reduced SBP by approximately 6 mmHg.[21] This discrepancy may reflect stricter adherence monitoring, lower sodium thresholds, and combined fluid restriction in the present intervention. The decline in DBP was also notable. Hanevold *et al*., described that CKD patients frequently demonstrate exaggerated salt sensitivity,

and thus respond disproportionately to sodium restriction.[22] In the present study, DBP reduction was 10.7%, far exceeding the 3–5% reduction reported by Garessus *et al.*, in their Cochrane review of altered dietary salt intake in CKD.[23] This suggests that coupling sodium restriction with fluid management amplifies hemodynamic benefits.

Interdialytic Weight Gain and Extracellular Water

Reduction in interdialytic weight gain (IDWG) was one of the most robust findings. The intervention group experienced a mean 23.5% reduction compared with 8.7% in controls. This aligns with reports by Sars et al., who observed that lower sodium intake correlated with reduced thirst and lower IDWG in dialysis patients.[24] Similarly, Mc Causland and Suh *et al.*, demonstrated that predialysis sodium intake predicted mortality and hospitalization risk through its impact on IDWG. Extracellular water volume decreased by 11.1% in the intervention group, compared with only 3.7% in controls.[25] Khin *et al.*, reported that bioimpedance spectroscopy reliably identifies extracellular water expansion in CKD and that dietary sodium directly contributes to excess fluid retention.[26] The results of the present investigation therefore reinforce earlier conclusions that sodium and fluid restrictions are synergistic in reducing extracellular volume and improving hemodynamic stability.

Biochemical Parameters

Serum sodium levels showed a modest but statistically significant decline in the intervention group, consistent with previous trials demonstrating that sodium restriction slightly lowers plasma sodium concentration without inducing hyponatremia.[27] Creatinine, potassium, and uric acid did not change significantly, suggesting that dietary sodium restriction influences hemodynamics and fluid balance more profoundly than biochemical markers of renal function in the short term. Mooradian *et al.*, similarly reported negligible effects on biochemical variables despite clear improvements in blood pressure and proteinuria.[28] Medication profiles were comparable across groups, with similar use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, and diuretics. This minimizes the likelihood that pharmacological therapy accounted for differences in outcomes. In fact, the findings parallel those of Grgic *et al.*, who demonstrated that sodium restriction enhanced the antiproteinuric effect of RAAS inhibition independent of drug dose.[23] By maintaining equivalent medication distributions, the present investigation strengthens the inference that dietary interventions exerted primary influence over outcomes.

Clinical Outcomes and Quality of Life

The proportion of patients achieving blood pressure control (<140/90 mmHg) was markedly higher in the intervention group. Similar benefits were observed in volume-related outcomes and quality of life measures. These results parallel findings from Mathilakath *et al.*, who showed that reducing dry weight in dialysis patients improved hypertension and reduced cardiovascular events.[29] Quality of life improvements may be attributable to reduced symptoms of fluid overload such as dyspnea, fatigue, and peripheral edema, as supported by the reports of Mitsides *et al.*,[30,31] The reductions in extracellular water and blood pressure observed in this investigation have direct implications for cardiovascular outcomes. Nelson *et al.*, emphasized that volume overload contributes to left ventricular hypertrophy and increased cardiovascular mortality in CKD. Heerspink *et al.*, demonstrated that higher urinary sodium excretion correlates with increased risk of cardiovascular disease in CKD patients.[32,33] The 11.1% reduction in extracellular water and 10.5% reduction in SBP in this investigation suggest that sodium and fluid restriction could meaningfully mitigate cardiovascular risk. The Chronic Renal Insufficiency Cohort (CRIC) study reported by Kim *et al.*, found that high sodium intake was associated with accelerated CKD progression and higher mortality.[34] The present findings reinforce those conclusions, although direct progression outcomes were not evaluated. Similarly, data from the MDRD trial indicated that sodium intake modifies the effect of protein restriction on CKD progression.[35] By focusing specifically on sodium and fluid, this investigation isolates dietary salt as a central modifiable determinant of hemodynamic stability.

Global Perspective and Cultural Relevance

The location of this investigation in Bangladesh adds valuable data from a South Asian population where dietary patterns differ from Western cohorts. High sodium consumption through traditional foods and inadequate fluid monitoring are prevalent in South Asian CKD populations. The

substantial clinical benefits observed reinforce the universality of sodium restriction across diverse cultural diets. Results may also guide public health interventions targeting salt reduction in South Asia, similar to initiatives in Japan and the United Kingdom that have successfully reduced population-level sodium intake.[36]

Future Research Directions

Future research should prioritize randomized controlled trials assessing varying levels of sodium restriction to determine the optimal threshold for maximal benefit without adverse counter-regulatory activation. Studies should evaluate long-term outcomes including renal survival, hospitalization, and mortality. Interventions incorporating digital health tools for dietary monitoring could enhance adherence assessment. Additionally, exploration of gene-diet interactions in salt sensitivity may refine individualized treatment strategies. Multi-center trials across diverse regions are essential to confirm generalizability.

CONCLUSION

This study highlights that sodium and fluid-restricted diets significantly improve blood pressure control, extracellular fluid balance, and interdialytic weight gain among patients with chronic kidney disease. The findings demonstrate that such non-pharmacological interventions exert measurable hemodynamic and clinical benefits independent of medication use. By targeting two fundamental determinants of cardiovascular risk—sodium intake and fluid retention—dietary modification serves as a practical and cost-effective adjunct to standard CKD management. These results reinforce the clinical importance of patient-centered dietary counseling and adherence monitoring. Future research should explore the long-term effects of tailored sodium thresholds, genetic influences on salt sensitivity, and the integration of digital monitoring tools to optimize adherence and improve global CKD outcomes.

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