A SECOND LOOK ON DIALYSIS PRACTICES: THE EFFECTS OF LOWER SODIUM DIALYSATE ON BLOOD PRESSURE IN INTRADIALYTIC HYPERTENSION PATIENTS IN INDIA

Srinivasa Sanjay Surya Gorla[1], B. Karthikeyan[2], Ramkumar M[3]*

[1] Srinivasa Sanjay Surya Gorla, Department of General Medicine, Saveetha Medical College and Hospital, Thandalam, Kancheepuram District, Tamil Nadu, India.
[2] B. Karthikeyan, Senior Consultant Nephrologist, Kauvery Hospital, Radial Road, Kovilambakkam, Chennai, Tamil Nadu, India.
[3] Ramkumar M, Associate Professor, Department of General Medicine, Saveetha Medical College and Hospital, Thandalam, Kancheepuram District, Tamil Nadu, India.
*CORRESPONDING AUTHOR**
Ramkumar M, Associate Professor, Department of General Medicine, Saveetha Medical College and Hospital, Thandalam, Kancheepuram District, Tamil Nadu, India - 602105
ORCID : 0000-0001-8419-1235 | Email ID: docrk83@yahoo.co.in

Keywords:

intraadialytic hypertension, dialysate sodium, ambulatory blood pressure, hemodialysis.

Abstract

Background: Introduction intradialytic hypertention (IDHTN) is a crital complication in patients on hemodialysis and is a we-establition cardiovascular morbidity and mortality event. Dialsate sodium concertation is a crucial determinant of blood pressure, but the optimal dialysate sodium concertation in the prevention of IDHTN continues to be known.
Aim: This study assesses the differences with respect to the pattern of ambulator blood pressure (ABP) in patients with isometric dialysate HTN (IDHTN) when dialysate sodium concertration is reduced.
Methodology: This was an 18-month prospective observational study done at a tertiary care hospital in Chennai enrolling 47 hemodialysis patients with IDHTN. There were two phases of dialysis (Phase 1 140 mEq/L dialysate sodium; Phase 2 138 mEq/L); ABP was measured over 24 hours during the interdialytic period following each phase.
Results: With 138 mEq/L dialysate, there was a significant decrease in systolic blood pressure (SBP) from 148.85 ± 11.67 mmHg at hour two to 142.60 ± 12.88 mmHg at the end of dialysis ($p < 0.05$). There were also reductions in diastolic blood pressure (DBP)but these did not achieve statistical significance. Phase 2 also evidenced lower mean SBP and DBP in a 24-hour ABP analysis. Hypotensive events remained the same, but muscle cramps and shivering events were less frequent.
Conclusion: In patients with IDHTN, dropping dialysate sodium to 138 mEq/L persistently reduces blood pressure and the potential for non-drug intervention like this is now high. Nonetheless, there is a prospect of hypotension if the sodium has not been closely monitored, and so individual sodium profiling is key.

1. INTRODUCTION

Nearly 85% of hemodialysis patients with end-stage kidney disease (ESKD) have hypertension, which dramatically raises their risk of cardiovascular disease (1). If left untreated, chronic kidney disease (CKD) can proceed irreversibly and result in end-stage renal disease (ESRD) (2,3). Between 1990 and 2017, the

mortality rate from CKD and ESRD increased by 41.5%, and the prevalence of CKD is estimated to be between 11.7% and 15.1% worldwide (4).

CKD and ESRD are significant worldwide health issues because of their high prevalence, mortality, and expense. The care of end-stage renal disease (ESRD) requires renal replacement therapies (RRT), among which hemodialysis (HD), peritoneal dialysis (PD), and kidney transplantation are the most commonly utilized modality (2). Nearly 63% of patients were on HD in the 2017 US Renal Data System (USRDS), which showed a 2.6% rise in ESRD incidence compared to 2016 (5).

CKD and ESRD have grown to be significant worldwide public health issues because of their high prevalence, mortality rates, and expensive medical expenses. Hemodialysis (HD), peritoneal dialysis (PD), and kidney transplantation are examples of renal replacement treatments (RRT) used to treat end-stage renal disease (ESRD). The most popular of these is hemodialysis, which is still the main course of treatment for most ESRD patients (2). Nearly 63% of ESRD patients in the United States are receiving hemodialysis, according to the 2017 United States Renal Data System (USRDS) report, which shows a 2.6% rise in ESRD patients from 2016 (5).

Blood is circulated through an external dialysis machine during hemodialysis, which exchanges metabolic waste with a dialysate solution that is intended to replicate the electrolyte composition of the body. This procedure preserves fluid, electrolyte, and acid-base balance while eliminating waste materials by ultrafiltration, diffusion, adsorption, and convection. The patient is then given back the cleaned blood (6). During dialysis, the ultrafiltration procedure lowers blood pressure initially. However, because of fluid retention and neurohormonal activation, blood pressure gradually rises during the interdialytic period. These variations raise the risk of cardiovascular disease and emphasize how important it is to effectively regulate blood pressure in this population (1, 7).

Intradialytic hypertension (IDHTN) is one of the most prevalent hemodialysis complications. It frequently affects older patients or those with particular clinical features, such as lower dry weight, decreased interdialytic weight gain, lower serum creatinine and albumin levels, or those on multiple antihypertensive medications (8).

IDHTN has a complicated etiology that incorporates several variables. These include increased sympathetic nervous system activity, renin-angiotensin system activation, increased endothelin release (a powerful vasoconstrictor), intradialytic sodium gain that causes fluid retention and elevated blood pressure, volume overload that can worsen hypertension, and electrolyte imbalances, especially those involving potassium and calcium. In addition to causing blood pressure swings, stopping antihypertensive drugs during dialysis can make managing IDHTN more difficult (9).

When compared to intradialytic hypotension (IDH), IDHTN is generally overlooked, and its prevalence and therapeutic needs are not as commonly highlighted. Although some research has indicated that IDHTN might be related with a higher risk of death, the literature currently lacks a thorough synthesis of evidence regarding the magnitude of this association and the negative consequences linked to IDHTN (10).

Sodium and Fluid Overload

The kidneys, along with the epidermis and gastrointestinal system, play a crucial role in maintaining salt balance. In healthy individuals, sodium homeostasis is maintained with just 50 mmol/day of dietary salt (sodium chloride) (11). However, kidney disease disrupts this balance, leading to fluid and sodium retention, which increases blood pressure (BP). For those with reduced renal function, strict dietary salt control is essential.

In chronic kidney disease (CKD), particularly in dialysis patients, extracellular fluid volume (ECV) and salt balance are closely linked. Dialysis removes sodium mainly through ultrafiltration, while intake comes from diet, medications, and diffusion. Residual kidney function and minor losses through the skin and gastrointestinal tract also contribute.

Excess sodium intake increases thirst, leading to higher ECV and water intake, triggering pressure natriuresis to restore balance (12). In patients with impaired renal function, reduced sodium excretion leads

to sodium sensitivity and elevated BP. Consequently, fluid volume is primarily influenced by salt intake rather than water, making volume overload a major cause of hypertension. In end-stage kidney disease (ESKD), hypertension is largely driven by ECV expansion and volume overload (13).

Dialysis patients often experience hypertension and volume overload, contributing to complications like myocardial infarction, heart failure, ischemic heart disease, left ventricular hypertrophy, stroke, sudden cardiac death, and increased cardiovascular and overall mortality (14–16). Despite this, many dialysis patients receive multiple antihypertensive drugs with suboptimal BP control.

Dialysate composition, particularly sodium concentration, significantly affects hemodynamic stability during dialysis. Dialysate sodium influences serum sodium levels and osmolarity, impacting fluid shifts and vascular tone. Careful assessment of dialysate sodium's role in intradialytic hypertension (IDH) is crucial (17). Evidence suggests that lower dialysate sodium levels can effectively manage IDH (18).

This study aims to evaluate the impact of lower dialysate sodium concentrations on IDH in Indian hemodialysis patients at a tertiary care center. Lowering dialysate sodium is expected to enhance cardiovascular stability, reduce IDH incidence and severity, and improve BP control. Findings will provide insights into optimizing dialysis protocols, reducing cardiovascular risks, and tailoring treatment strategies based on regional patient characteristics.

2. METHODOLOGY

2.1 Study Design

Over the course of 18 months, Saveetha Medical College and Hospital in Chennai, Tamil Nadu, India, hosted this prospective observational study. A two-sided t-test was used to determine the sample size in order to compare the means of the two groups. With parameters established at a minimum detectable effect size of 10 mmHg, a power of 80%, and a significance threshold of 0.05, a power analysis was conducted to guarantee a sufficient sample size. These estimates indicated that a crossover cohort needed a minimum of 42 participants. 47 participants were registered in order to allow for any dropouts. Regarding per-protocol analysis, the study's findings were examined for each patient who adhered strictly to the procedure. Under approval number SMC/IEC/2020/12/044, ethical clearance was acquired by the Institutional Ethical Committee—Institutional Review Board (IEC-IRB). Prior to enrollment, all individuals provided written informed consent.

2.2. Study Population and Selection Criteria

Based on eligibility requirements, convenience sampling was used to choose participants from the dialysis unit population. During the selecting procedure, no randomization was used. Prior to enrollment, informed consent was obtained.

Adults between the ages of 18 and 80 receiving continuous hemodialysis for a minimum of three months met the inclusion criteria. Intradialytic hypertension, which is defined as an increase in systolic blood pressure of ≥10 mmHg from pre- to post-hemodialysis in at least four of the six most recent dialysis sessions, was a need for participation. Participants also needed to be in clinically stable condition and free of acute medical events, such as infections or recent hospitalizations, and have stable electrolyte values, with serum sodium levels between 138 and 142 mEq/L.

Patients who had fewer than three months of hemodialysis experience, acute kidney injury (AKI) requiring dialysis, or frequent intradialytic hypotension needing fluid resuscitation were excluded. Individuals with a history of dialysis disequilibrium syndrome, seizures, or cerebrovascular accidents were not included. Those with heart issues, including reduced ejection fraction (≤40%), severe valve disease, arrhythmias, or any condition that affects the control of blood pressure, were not eligible. Women who were nursing or pregnant were also not included. All subjects followed established dialysis procedures, including maintaining constant dialysate salt concentrations, to reduce confounding factors. Medication schedules and hydration

levels were tracked and recorded. The final analysis did not include participants who significantly deviated from these parameters during the trial.

2.3. Rationale for selecting 138 Meq/L sodium dialysate:

During dialysis, salt is eliminated by diffusion and ultrafiltration. Many variables affect how sodium diffuses across the membranes. Therefore, it is concluded that the sodium diffusion process only takes place when the sodium dialysate concentration is at least 2 mEq/L lower than the sodium plasma concentration (19).

Numerous investigations are being conducted on the choice of dialysate sodium content. Hemodialysis hemodynamic stability was achieved by dialysate glucose concentrations exceeding 1800 mg/dL and dialysate sodium concentrations ranging from an average of 126 to 130 mEq/L. The ongoing development of dialysis technology made it possible for treatment to last less time. However, because of the higher ultrafiltration rates, hemodynamic instability predominated. Although the increased sodium dialysate concentration improved hemodynamics, side effects such as intradialytic hypertension, hypotension, and interdialytic weight gain were still debatable. This led the researchers to investigate lower concentrations of sodium dialysate. Numerous investigations were conducted using decreased sodium dialysate at concentrations between 136 and 138 mEq/L. In our investigation on the South Indian population, we chose 138 mEq/L to examine the intradialytic blood pressure (20) based on collective evidence.

2.4. Methods and Evaluations of Study

All recruited subjects underwent baseline laboratory evaluations. During four-hour sessions, hemodialysis was carried out utilizing multi-use Elisio M dialyzers and Nipro dialysis machines. Standard amounts of sodium, potassium, calcium, magnesium, chloride, and bicarbonate were included in the dialysate, along with either glucose or dextrose. To avoid confusing dietary influences on blood pressure, participants throughout the trial adhered to a salt-restricted diet.

As seen in Figure 1, the study was split into two stages. Participants in Phase 1 received eight dialysis sessions with a sodium concentration of 140 mEq/L in the dialysate. They finished eight dialysis sessions in Phase 2 with a sodium dialysate concentration of 138 mEq/L. Following the seventh and eighth dialysis sessions in each phase, ambulatory blood pressure (ABP) monitoring was carried out during the interdialytic time. The Microlife WatchBP 03, a clinically approved oscillometric device that complies with European Society of Hypertension (ESH) guidelines, was used to monitor ABP for a whole day (21). The cuff was placed two to three centimeters above the elbow on the arm that was not infected. Throughout the day and night, blood pressure readings were taken every 30 minutes.

Blood pressure readings were routinely recorded and examined before and after dialysis. Any intradialytic side effects, including shivering, cramping in the muscles, hypotension, or hyperglycemia, were noted and treated as necessary. To assess the effect of dialysate sodium decrease on ambulatory blood pressure in individuals with intradialytic hypertension, data from the two periods were compared.

2.5. Research Findings

Assessing changes in 24-hour ambulatory blood pressure in patients with intradialytic hypertension after dialysate sodium was reduced from 140 mEq/L to 138 mEq/L was the study's main goal. The incidence of problems during each phase, intradialytic blood pressure at the second and fourth hours, and pre- and post-dialysis blood pressure comparisons were examples of secondary outcomes.

2.6. Statistical Analysis

Descriptive statistics were used to summarize baseline laboratory and demographic data. The chi-square test was used to assess categorical variables. The Kolmogorov-Smirnov test was used to evaluate the normality of the data distribution. Blood pressure readings before and after dialysis were analyzed using Repeated Measures Analysis of Variance (RMANOVA), and pairwise comparisons were performed using post hoc

Bonferroni testing. Statistical significance was defined as a p-value of less than 0.05. For statistical studies, SPSS version 22.0 was used.

3. RESULTS

3.1 Baseline Laboratory and Demographic Characteristics

Of the 89 patients who were screened, 47 (ten females and 37 males) satisfied the requirements for inclusion. Hemodialysis gender inequality is frequently observed and is driven by sociocultural variables, education, health insurance access, and vascular access inequalities (males have greater AVF rates) (22). This discrepancy could affect the results of the study.

Baseline characteristics are shown in Table 1. Dialysis lasted 28.7 ± 19.2 months, and the mean age was 52.61 ± 11.10 years. Alcohol use and hypertension were common comorbidities (78.7%). The two main causes of CKD were diabetes mellitus and hypertension. Three individuals tested positive for HBV and three for HCV, according to the serological study.

Calcium channel blockers were the most commonly utilized antihypertensive medication (97.8%) in the majority of hypertension patients. Thirteen patients with a mean urine output of 0.34 ± 0.187 L/24 hours were taking loop diuretics.

3.2 Ambulatory Blood Pressure During Dialysis

Dialysis-related ambulatory blood pressure readings with two dialysate sodium concentrations (140 mEq/L and 138 mEq/L) are described in Table 2. SBP dramatically decreased in the 138 mEq/L group from 148.85 ± 11.67 mmHg at the second hour to 147.94 ± 15.68 mmHg at the fourth hour and 142.60 ± 12.88 mmHg at the end of dialysis ($p < 0.05$).

The 138 mEq/L group had reduced DBP at the 4-hour mark (83.89 ± 3.6 mmHg) and after dialysis (84.92 ± 2.90 mmHg). However, because of a lower frequency of hypotension, DBP decreases at the second and fourth hours were not statistically significant. Profiles of SBP and DBP are shown in Figures 2 and 3.

3.3 24-Hour Ambulatory Blood Pressure Comparison

During the seventh and eighth dialysis sessions, 24-hour ambulatory blood pressure monitoring is shown in Table 3. In comparison to phase 1, the 138 mEq/L group's 24-hour average SBP (164.13 ± 18.57 mmHg) and DBP (92.63 ± 6.43 mmHg) demonstrated statistically significant decreases ($p < 0.05$).

The average SBP and DBP dips over the night were 8.72 ± 4.23% and 8.61 ± 3.74%, respectively. Despite increased nocturnal dipping in phase 2, the changes were not statistically significant.

3.4 Complications Associated with Dialysis in Patients with Intradialytic Hypertension

Complications from dialysis, such as shivering, muscle cramps, hypotension, and hyperglycemia, are compiled in Table 4. Shivering and cramping in the muscles were significantly reduced ($p < 0.05$) in phase 2. Although the 138 mEq/L group showed decreases in both hypotension and hyperglycemia, they did not become statistically significant.

4. DISCUSSION

This prospective observational study examined blood pressure (BP) in patients with intradialytic hypertension (IDH) over 24 hours, comparing standard sodium dialysate (140 mEq/L) with reduced sodium dialysate (138 mEq/L). Results showed improved ambulatory BP regulation with lower sodium dialysate, though the mean BP reduction of 3.27/-1.28 mmHg was not statistically significant.

IDH is defined as a systolic BP increase of over 10 mmHg from pre- to post-dialysis in at least four of six consecutive sessions (23) and is linked to factors like sympathetic activation, endothelial dysfunction, and extracellular volume overload (24). Sodium plays a key role in vascular refilling and plasma tonicity, and research suggests that reducing dialysate sodium lowers IDH risk. A randomized crossover study with 29 patients found that a 137 mEq/L dialysate reduced 48-hour ambulatory BP by -5.3/-2.6 mmHg compared to

140 mEq/L (25), while another study showed that increasing dialysate sodium from 137 to 141 mEq/L led to gradual BP elevation (26). Similar trials confirmed that low sodium dialysate reduces BP, though short-term variability remained unchanged (27). A study with 11 patients also found that hyponatremic dialysate reduced 24-hour mean BP (28).

Our findings align with previous research, suggesting low sodium dialysate as a potential BP management strategy. We also examined dialysis-related complications. Hypotension, influenced by dialysate composition, ultrafiltration rate, and patient characteristics, showed a non-significant decrease in our study, despite the common association of low sodium dialysate with increased hypotension (29,30).

A notable finding was the reduction in shivering episodes, possibly linked to improved hemodynamic stability, though mainly affected by dialysate temperature (31). Dialysis-induced glucose fluctuations in diabetic patients can lead to hypoglycemia followed by glucagon-induced hyperglycemia (32), but our study found no significant effect of low sodium dialysate on hyperglycemia.

Muscle cramps, often caused by osmotic shifts and electrolyte imbalances (33), may worsen with aggressive ultrafiltration due to high plasma sodium, potassium, calcium, and magnesium levels. However, sodium profiling and hypotension prevention strategies can help reduce cramping (34). Patients on reduced sodium dialysate in our study experienced fewer cramps, likely due to better fluid balance.

Overall, low sodium dialysate presents a non-pharmacological approach to BP control in IDH, but intradialytic hypotension remains a concern due to its association with increased mortality (35). Interdialytic weight control, symptom monitoring, individualized dialysis protocols, and personalized sodium profiling may optimize BP management while minimizing hypotension risks.

Limitations include reliance on clinical symptoms for volume status assessment, introducing variability, and the use of convenience sampling, which may cause selection bias. Including intradialytic weight gain data could have strengthened our conclusions. Further studies with larger cohorts and detailed hydration assessments are needed.

5. CONCLUSION

This study offers a workable non-pharmacological method for blood pressure control in hemodialysis patients by showing that lowering sodium dialysate concentration significantly lowers intradialytic hypertension within 24 hours. Lower sodium dialysate does, however, carry a higher risk of hypotension, which should be carefully considered. A weakness of this study is the absence of interdialytic weight growth measurements, endocrine assessments, and a thorough volume status assessment. Deeper insights into adjusting dialysate sodium concentrations for better patient outcomes may be obtained from future studies that address these parameters with customized dialysis prescriptions.

Conflicts of interest

The authors declare no competing interest in the work reported in this paper.

Acknowledgment

The authors would like to acknowledge ScienceBy and DhiShi Scientific Private Limited for their editorial service while preparing this manuscript.

REFERENCES:

1. Sarafidis PA, Persu A, Agarwal R, Burnier M, de Leeuw P, Ferro CJ, et al. Hypertension in dialysis patients: a consensus document by the European Renal and Cardiovascular Medicine (EURECA-m) working group of the European Renal Association-European Dialysis and Transplant Association (ERA-EDTA) and the Hypertension and the Kidney working group of the European Society of Hypertension (ESH). Nephrol Dial Transplant. 2017 Apr 1;32(4):620–40.
2. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013 Jul 20;382(9888):260–72.

3. Lv JC, Zhang LX. Prevalence and Disease Burden of Chronic Kidney Disease. Adv Exp Med Biol. 2019;1165:3–15.
4. GBD Chronic Kidney Disease Collaboration. Global, regional, and national burden of chronic kidney disease, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020 Feb 29;395(10225):709–33.
5. Saran R, Robinson B, Abbott KC, Agodoa LYC, Bhave N, Bragg-Gresham J, et al. US Renal Data System 2017 Annual Data Report: Epidemiology of Kidney Disease in the United States. Am J Kidney Dis. 2018 Mar;71(3 Suppl 1):A7.
6. Saha M, Allon M. Diagnosis, Treatment, and Prevention of Hemodialysis Emergencies. Clin J Am Soc Nephrol. 2017 Feb 7;12(2):357–69.
7. Karpetas A, Loutradis C, Bikos A, Tzanis G, Koutroumpas G, Lazaridis AA, et al. Blood pressure variability is increasing from the first to the second day of the interdialytic interval in hemodialysis patients. J Hypertens. 2017 Dec;35(12):2517–26.
8. Faye M, Lemrabott AT, Kane Y, Cissé MM. Intradialytic Hypertension: Prevalence and Associated Factors in Chronic Hemodialysis Patients in Senegal. 2018 [cited 2025 Jan 25]; Available from: http://rivieresdusud.uasz.sn/xmlui/handle/123456789/1869
9. Georgianos PI, Sarafidis PA, Zoccali C. Intradialysis Hypertension in End-Stage Renal Disease Patients: Clinical Epidemiology, Pathogenesis, and Treatment. Hypertension [Internet]. 2015 Sep [cited 2024 Dec 30];66(3):456–63. Available from: https://www.ahajournals.org/doi/10.1161/HYPERTENSIONAHA.115.05858
10. Nongnuch A, Campbell N, Stern E, El-Kateb S, Fuentes L, Davenport A. Increased postdialysis systolic blood pressure is associated with extracellular overhydration in hemodialysis outpatients. Kidney Int. 2015 Feb;87(2):452–7.
11. Cumming A PW. Davidson's principles and practice of medicine. 19th ed. Edinburgh, New York: Churchill Livingstone; 2002, pg 1296.
12. Guyton AC. Kidneys and fluids in pressure regulation. Small volume but large pressure changes. Hypertension. 1992 Jan;19(1 Suppl):I2-8.
13. Vertes V, Cangiano JL, Berman LB, Gould A. Hypertension in end-stage renal disease. N Engl J Med. 1969 May 1;280(18):978–81.
14. Paoletti E, Specchia C, Di Maio G, Bellino D, Damasio B, Cassottana P, et al. The worsening of left ventricular hypertrophy is the strongest predictor of sudden cardiac death in haemodialysis patients: a 10 year survey. Nephrol Dial Transplant. 2004 Jul;19(7):1829–34.
15. Tozawa M, Iseki K, Iseki C, Takishita S. Pulse pressure and risk of total mortality and cardiovascular events in patients on chronic hemodialysis. Kidney International [Internet]. 2002 Feb 1 [cited 2025 Jan 25];61(2):717–26. Available from: https://www.sciencedirect.com/science/article/pii/S0085253815482638
16. Iseki K, Kinjo K, Kimura Y, Osawa A, Fukiyama K. Evidence for high risk of cerebral hemorrhage in chronic dialysis patients. Kidney Int. 1993 Nov;44(5):1086–90.
17. Movilli E, Camerini C, Gaggia P, Zubani R, Feller P, Poiatti P, et al. Role of dialysis sodium gradient on intradialytic hypertension: an observational study. Am J Nephrol. 2013;38(5):413–9.
18. Munoz Mendoza J, Sun S, Chertow GM, Moran J, Doss S, Schiller B. Dialysate sodium and sodium gradient in maintenance hemodialysis: a neglected sodium restriction approach? Nephrol Dial Transplant. 2011 Apr;26(4):1281–7.
19. Kooman JP, van der Sande F, Leunissen K, Locatelli F. Sodium balance in hemodialysis therapy. Semin Dial. 2003;16(5):351–5.
20. Flythe JE, Mc Causland FR. Dialysate Sodium: Rationale for Evolution over Time. Semin Dial [Internet]. 2017 Mar [cited 2025 Jan 5];30(2):99–111. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5334180/

21. Ragazzo F, Saladini F, Palatini P. Validation of the Microlife WatchBP O3 device for clinic, home, and ambulatory blood pressure measurement, according to the International Protocol. Blood Press Monit. 2010 Feb;15(1):59–62.
22. Shankar M, Satheesh G, A. K, C. G. S, Reddy GG. Gender disparity in maintenance hemodialysis units in South India: a cross-sectional observational study. Front Nephrol [Internet]. 2024 Mar 25 [cited 2025 Jan 6];4. Available from: https://www.frontiersin.org/journals/nephrology/articles/10.3389/fneph.2024.1322003/full
23. Flythe JE, Chang TI, Gallagher MP, Lindley E, Madero M, Sarafidis PA, et al. Blood pressure and volume management in dialysis: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int. 2020 May;97(5):861–76.
24. Theofilis P, Vordoni A, Kalaitzidis RG. Epidemiology, pathophysiology, and clinical perspectives of intradialytic hypertension. American Journal of Nephrology [Internet]. 2023 [cited 2024 Dec 30];54(5–6):200–7. Available from: https://karger.com/ajn/article-abstract/54/5-6/200/843595
25. Iatridi F, Malandris K, Ekart R, Xagas E, Karpetas A, Theodorakopoulou MP, et al. Low dialysate sodium and 48-h ambulatory blood pressure in patients with intradialytic hypertension: a randomized crossover study. Nephrology Dialysis Transplantation [Internet]. 2024 [cited 2024 Dec 30];gfae104. Available from: https://academic.oup.com/ndt/advance-article-abstract/doi/10.1093/ndt/gfae104/7665745
26. Bratsiakou A, Iatridi F, Theodorakopoulou M, Sarafidis P, Goumenos DS, Papachristou E, et al. The effect of different dialysate sodium concentrations on ambulatory blood pressure in hemodialysis patients: a prospective interventional study. Clinical Kidney Journal [Internet]. 2024 Jul 1 [cited 2024 Dec 30];17(8):sfae041. Available from: https://doi.org/10.1093/ckj/sfae041
27. Iatridi F, Ekart R, Xagas E, Karkamani E, Karpetas A, Theodorakopoulou MP, et al. Dialysate sodium and short-term blood pressure variability in patients with intradialytic hypertension: a randomized crossover study. Journal of Human Hypertension [Internet]. 2024 [cited 2024 Dec 30];38(11):750–7. Available from: https://www.nature.com/articles/s41371-024-00947-w
28. Robberechts T, Allamani M, Galloo X, Wissing KM, Niepen PVD. Individualized Isonatremic and Hyponatremic Dialysate Improves Blood Pressure in Patients with Intradialytic Hypertension: A Prospective Cross-Over Study with 24-h Ambulatory Blood Pressure Monitoring. Open Journal of Nephrology [Internet]. 2020 Apr 2 [cited 2025 Jan 2];10(2):144–57. Available from: https://www.scirp.org/journal/paperinformation?paperid=101063
29. Hamrahian SM, Vilayet S, Herberth J, Fülöp T. Prevention of Intradialytic Hypotension in Hemodialysis Patients: Current Challenges and Future Prospects. Int J Nephrol Renovasc Dis. 2023;16:173–81.
30. Davenport A. Why is intradialytic hypotension the commonest complication of outpatient dialysis treatments? Kidney International Reports [Internet]. 2023 [cited 2024 Dec 30];8(3):405–18. Available from: https://www.sciencedirect.com/science/article/pii/S2468024922018563
31. Tsujimoto Y, Tsujimoto H, Nakata Y, Kataoka Y, Kimachi M, Shimizu S, et al. Dialysate temperature reduction for intradialytic hypotension for people with chronic kidney disease requiring haemodialysis. Cochrane Database of Systematic Reviews [Internet]. 2019 [cited 2024 Dec 30];(7). Available from: https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD012598.pub2/abstract
32. Nakao T, Inaba M, Abe M, Kaizu K, Shima K, Babazono T, et al. Best practice for diabetic patients on hemodialysis 2012. Therapeutic Apheresis and Dialysis [Internet]. 2015 [cited 2024 Dec 30];19:40–66. Available from: https://www.ishen365.com/aetherupload/display/file/201709/0a300d2548c926ec053d1dfccef8ca42.pdf
33. Dousdampanis P, Trigka K, Ntouvas I, Assimakopoulos SF, Musso CG, Papadoulas S. Cramps during Hemodialysis: Are They Always Innocent? Saudi Journal of Kidney Diseases and

Transplantation [Internet]. 2019 Aug [cited 2025 Jan 3];30(4):985. Available from: https://journals.lww.com/sjkd/fulltext/2019/30040/Cramps_during_Hemodialysis__Are_They_Always.32.aspx
34. Ulu S, Ahsen A. Muscle cramps during hemodialysis: what can we do? new approachs for treatment and preventing. European Journal of General Medicine [Internet]. 2015 [cited 2024 Dec 30];12(3):277–81. Available from: https://www.ejgm.uk/download/muscle-cramps-during-hemodialysis-what-can-we-do-new-approachs-for-treatment-and-preventing-7266.pdf
35. Marshall MR, Wang MY, Vandal AC, Dunlop JL. Low dialysate sodium levels for chronic haemodialysis. Cochrane Database of Systematic Reviews [Internet]. 2024 [cited 2024 Dec 31];(11). Available from: https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD011204.pub3/abstract

TABLES

Table 1: Baseline demographic and laboratory details of the study population

Parameter	Value (N=47)
Age (years)	52.61 ± 11.10
Gender (n, %)	
Male Female	37 (78.7) 10 (21.3)
CKD duration in months	32.24 ± 23.79
CKD vintage (months)	28.70 ± 19.10
Comorbidities (n, %)	
Diabetes mellitus Hypertension Cardiovascular disease Smoking Alcohol	24 (51.1) 37 (78.7) 12 (25.5) 23 (48.9) 37 (78.7)
CKD Etiology	
Diabetes mellitus (DM) Hypertension (HTN) NSAIDS Nephrolithiasis Unknown DM, HTN HTN, NSAIDS DM, NSAIDS	5 (10.6) 10 (21.2) 3 (6.3) 1 (2.1) 4 (8.5) 15 (31.9) 6 (12.7) 3 (6.3)

Laboratory Values	
Hemoglobin (g/dL) Serum urea (mg/dL) Serum creatinine (mg/dL) Serum sodium (mEq/L) Serum potassium (mEq/L) Serum calcium (mg/dL) Serum phosphate (mg/dL) Serum bicarbonate (mEq/L) Serum chloride (mEq/L) Serum albumin (g/dL)	8.80 ± 1.19 97.23 ± 25.82 9.98 ± 3.30 134.56 ± 1.31 5.42 ± 0.78 8.08 ± 1.04 6.12 ± 1.61 19.63 ± 3.20 102.56 ± 3.86 4.05 ± 0.3
Serology test (n, %)	
HBV positive HCV positive	3 (5.4) 3 (5.4)
Anti-hypertensive drugs per day (n, %)	
Monotherapy Dual Therapy Triple Therapy Quadruple Therapy	28 (59.6) 10 (21.3) 7 (14.9) 2 (4.3)
Type of drugs (n, %)	
CCB Diuretics β blockers ARBS	46 (97.8) 13 (27.6) 3 (6.38) 2 (4.2)

Data is represented as n (%) or mean ± standard deviation. CKD: chronic kidney disease; DM: diabetes mellitus; HTN: hypertension; NSAIDS: nonsteroidal anti-inflammatory drugs; HBV: hepatitis B virus; HCV: hepatitis C virus; CCB: calcium channel blockers; ACEIs: angiotensin receptor blockers.

Table 2: Ambulatory blood pressure in individuals treated with dialysate sodium 140 mEq/L and 138 mEq/dL during dialysis at different time points.

Parameters	Phase 1 (Dialysate sodium 140 mEq/L) (n=47)		Phase 2 (Dialysate sodium 138 mEq/L) (n=47)		P value	
BP	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Pre-Dialysis	146.15 ± 9.73	83.66 ± 4.68	145.87 ± 8.86	83.29 ± 3.02	0.886	0.657
Post-dialysis	161.68 ± 9.61	87.83 ± 5.19	142.60 ± 12.88	84.92 ± 2.90	0.000*	0.000*
2nd-hour	162.94 ± 17.96	86.26 ± 7.42	148.85 ± 11.67	85.14 ± 4.18	0.000*	0.379
4th-hour	163.36 ± 20.79	87.28 ± 6.53	147.94 ± 15.68	83.89 ± 3.60	0.000*	0.003

Data are represented as mean ± standard deviation. BP: Blood Pressure. * Statistically significant with a p-value <0.001.

Table 3: Ambulatory blood pressure levels between dialysate sodium 140 mEq/L and dialysate sodium 138 mEq/L treatment in intradialytic patients at 24-hour intervals.

Parameters	Phase 1 (Dialysate sodium 140 mEq/L) (n=47)		Phase 2 (Dialysate sodium 138 mEq/L) (n=47)		P value	
BP	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
24-hour interval	175.0 ± 18.50	102.46 ± 8.03	164.13 ± 18.57	92.63 ± 6.43	0.006	0.000*
Night time Dip %	9.37 ± 4.73	8.77 ± 4.94	8.72 ± 4.23	8.61 ± 3.74	0.486	0.855

Data are represented as mean ± SD. * statistically significant with a p-value <0.001.

Table 4: Complications in intradialytic patients treated with dialysate sodium 140 mEq/L and 138 mEq/L

Complications	Dialysate sodium 140 mEq/L	Dialysate sodium 138 mEq/L	χ^2 value	P value
Hypotension	4 (8.5)	2 (4.3)	0.712	0.399
Hypoglycemia	7 (14.9)	3 (6.4)	1.790	0.181

Muscle cramp	9 (19.1)	1 (2.1)	7.162	0.007*
Shivering	10 (21.3)	0	11.190	0.001*

Data is represented as n(%). *statistically significant with p-value <0.001