

BEYOND SODIUM: INFLAMMATION AS THE STRONGEST PREDICTOR OF MORTALITY AMONG NON-DIABETIC HEMODIALYSIS PATIENTS

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ABSTRACT

Introduction: Standard dialysate sodium concentration (sDNa) may not adequately reflect individual sodium requirements in each hemodialysis patient, potentially contributing to interdialytic weight gain (IDWG), hypertension and inflammation. The aim of this study was to compare clinical and biochemical outcomes between individualized and standard dialysate sodium prescriptions and to assess factors associated with mortality.

Materials and Methods: This was a prospective interventional study conducted in two phases. In the first phase, patients were treated with standard hemodialysis (HD) using a fixed dialysate sodium concentration of 138 mmol/L. In the second phase, dialysate sodium was individualized based on each patient's pre-dialysis serum sodium concentration. Each patient served as their own control. Outcomes included IDWG, blood pressure, thirst score, dialysis adequacy (Kt/V, URR), nutritional markers, C-reactive protein (CRP), electrolytes, and mortality.

Results: Individualized dialysate sodium prescription was associated with a significant reduction in IDWG (1.93 ± 0.64 vs 2.17 ± 0.79 kg; $p = 0.001$) and improved dialysis adequacy (Kt/V: 1.50 ± 0.24 vs 1.36 ± 0.22 ; $p < 0.001$). Survivors had higher serum albumin levels and lower CRP values compared to non-survivors. Serum and dialysate sodium concentrations were not independently associated with mortality. In logistic regression analysis, CRP >10 mg/L showed the strongest observed association with mortality (OR 10.278; 95% CI 1.709–61.826; $p = 0.011$).

Conclusion: Individualized dialysate sodium prescription may improve fluid control and dialysis adequacy in selected patients. Inflammation was significantly associated with mortality in this cohort; however, results should be interpreted with caution due to the limited number of events.

Keywords: hemodialysis, dialysis solutions, sodium gradient, inflammation, mortality

INTRODUCTION

In the early development of dialysis, the system lacked the ability to regulate the hydrostatic ultrafiltration. Salt and water that have accumulated during the interdialytic period were removed primarily through diffusion and osmosis using dextrose-based hyponatremic dialysate,

rather than through ultrafiltration [1, 2]. Advances in dialysis technology later enabled the implementation of controlled ultrafiltration via regulation of hydrostatic transmembrane pressure. However, the rapid reduction in serum urea or osmolality occasionally resulted in dialysis dis-

equilibrium syndrome – a potentially fatal complication. High-sodium dialysis helped mitigate this risk, and dialysate sodium concentrations gradually increased from 125 mmol/L to 130–135 mmol/L, eventually reaching today's levels of 140–145 mmol/L [3]. Patients with end-stage renal disease (ESRD), particularly those who are oligoanuric, are highly dependent on dialysis to manage fluid volume, electrolyte concentrations, and plasma osmolality. To date, the optimal dialysate sodium concentration remains uncertain. Most dialysis centers use a dialysate sodium concentration of 138 mmol/L or higher to maintain hemodynamic stability and avoid imbalances. Individual HD patients tend to maintain a stable pre-dialysis serum sodium level over time, suggesting a personalized sodium “set point.” A fixed dialysate sodium concentration may therefore expose many patients to chronic positive sodium balance. Several studies have confirmed this, showing pre-dialysis serum sodium variation within just 1–2%. Sodium balance during HD is influenced by both diffusion (defined by the sodium gradient between dialysate and serum sodium) and convection (which depends on the ultrafiltration rate) [4]. Mechanical ultrafiltration produces a hypotonic ultrafiltrate and disrupts salt-water homeostasis, often leading to salt loading proportional to weight loss during dialysis. This creates a vicious cycle of hypertension, excessive thirst, and increased interdialytic weight gain (IDWG), requiring further ultrafiltration and worsening sodium overload. Volume overload and sodium imbalance may also contribute to chronic inflammation and cardiovascular injury in hemodialysis patients. To achieve isonatremic HD, dialysate sodium activity should ideally be lower than that of plasma water [5]. Few prospective studies have simultaneously evaluated individualized dialysate sodium prescription, dialysis adequacy, nutritional status, inflammation, and mortality predictors in non-diabetic hemodialysis patients. The aim of this study was to compare the clinical and biochemical outcomes between individualized and standard DNa prescriptions, and to assess the predictors of mortality in maintenance HD patients.

MATERIALS AND METHODS

The study was a prospective interventional study conducted in two phases. During the

first 12 months, the standard dialysate sodium concentration phase (sDNa phase), 77 non-diabetic patients underwent maintenance hemodialysis (HD) using a fixed standard dialysate sodium concentration (sDNa) of 138 mmol/L. During the subsequent 12 months, the individualized dialysate sodium concentration phase (iDNa phase), the dialysate sodium concentration was individualized according to each patient's pre-hemodialysis serum sodium concentration. The within-subject design reduced the interindividual variability because each patient served as their own control. Exclusion criteria included malignancy, preserved residual renal function (urine output >500 mL/day) and diabetes mellitus due to the potential influence of poor glycemic control on serum sodium levels. All patients provided written informed consent prior to study participation. Patients were advised to restrict dietary salt intake to <5 g/day. Patients with interdialytic weight gain (IDWG) >5% of dry weight were reevaluated. All patients received high-flux bicarbonate hemodialysis using polyethersulfone dialyzers sterilized by gamma irradiation. Treatments were performed three times per week for 4.5 hours per session. The acid component of the dialysis solution (Niprosol BC 1.50) contained the following electrolyte composition: K^+ 2.0 mmol/L, Ca^{2+} 1.5 mmol/L, Mg^{2+} 1.0 mmol/L, HCO_3^- 35.0 mmol/L, Cl^- 110.04 mmol/L, and CH_3COO^- 3.0 mmol/L. Throughout the study period, dialysis session duration, dialysis modality, dialyzer type, and bicarbonate-based dialysis prescription remained unchanged. Patients continued their standard antihypertensive and erythropoiesis-stimulating agent (ESA) therapy according to routine clinical practice. No major changes in dietary counseling protocols occurred during follow-up. However, detailed longitudinal data regarding antihypertensive therapy, ESA dose adjustments, ultrafiltration rates, and other medications were not systematically recorded, which represents a limitation of the study. Seasonal bias was partially minimized by the longitudinal within-subject design, as each patient served as their own control during both study phases. The intervention consisted of individualization of dialysate sodium concentration according to each patient's average pre-hemodialysis serum sodium concentration (“sodium set point”), calculated from the repeated measurements obtained during the preceding 6–12 months. The mean pre-dialysis

serum sodium concentration during this period was used as the reference value for each individual patient. Dry weight and IDWG (defined as the difference between post-dialysis body weight and pre-next-session body weight) were recorded. Each patient completed the Dietary Thirst Inventory (DTI) questionnaire [6]. Laboratory tests were performed pre- and post-dialysis during the midweek session and included complete blood count, urea, creatinine, electrolytes (sodium, potassium, total calcium, phosphorus), parathyroid hormone (PTH), C-reactive protein (CRP) and albumin. Patients with inadequate dialysis adequacy ($Kt/V < 1.2$) were clinically reevaluated according to routine dialysis unit protocols, including reassessment of vascular access, dialysis duration, and blood flow parameters. Laboratory analyses were performed at the central laboratory of the General City Hospital “8th September”, Skopje. Serum sodium concentration was measured using the direct ion-selective electrode method on the SmartLyte Electrolyte Analyzer (Diamond Diagnostics). Dialysis adequacy was assessed using the urea kinetic model by applying the second-generation logarithmic Daugirdas formula for Kt/V and the urea reduction ratio (URR) [7,8]. Protein catabolic rate (PCR) was calculated using standard urea kinetic modeling equations based on urea generation and removal during a single dialysis session. Statistical analysis was performed using SPSS version 17.0. Continuous variables were presented as mean $\pm$ standard deviation, while categorical variables were expressed as frequencies and percentages. The Shapiro–Wilk test was used to assess the normality of distribution. The differences between independent groups were

analyzed using the independent t-test or Mann–Whitney U test, as appropriate. Within-subject comparisons were performed using the paired t-test or Wilcoxon signed-rank test. Chi-square test or Fisher’s exact test was used for categorical variables. Logistic regression analysis was performed to evaluate factors associated with mortality. Due to the limited number of mortality events, only univariate logistic regression analysis was performed to reduce the risk of overfitting. Regression results should be interpreted cautiously because the events-per-variable principle was not fully met, increasing the possibility of model overfitting. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 77 patients were included in the analysis, comprising 41 males (53.2%) and 36 females (46.8%), with a mean age of 59.62 ± 12.66 years and a dialysis vintage of 96.76 ± 91.69 months. Baseline demographic and clinical characteristics of the study population are presented in Table 1.

During the standard dialysate sodium phase (sDNA phase), the mean pre-hemodialysis serum sodium concentration was 136.18 ± 1.39 mmol/L (range 132.53–140 mmol/L). A total of 69 patients (89.6%) had pre-dialysis serum sodium concentrations below 138 mmol/L. The distribution of serum sodium concentrations is shown in Figure 1, indicating the presence of a positive sodium gradient during the standard sodium phase. The mean post-hemodialysis serum sodium concentration was 138.9

Table 1. Baseline demographic and clinical characteristics of the study population ($n=77$)

Parameter	Mean $\pm$ SD / n (%)
Age (years)	59.62 ± 12.66
Dialysis vintage (months)	96.76 ± 91.69
Sex (male/female)	41 (53.2%) / 36 (46.8%)
BMI (kg/m^2)	25.96 ± 5.32
Cause of kidney disease	
– Alport syndrome	2 (2.6%)
– Adult polycystic kidney disease	5 (6.5%)
– Glomerulonephritis	22 (28.6%)
– Nephroarteriosclerosis	34 (44.2%)
– Obstructive nephropathy	5 (6.5%)
– Interstitial pyelonephritis	4 (5.2%)
– Unknown etiology	7 (9.1%)
Comorbidities (cardiovascular disease: present/absent)	27 (35.1%) / 50 (64.9%)
Vascular access (AVF/catheter)	69 (89.6%) / 8 (10.4%)

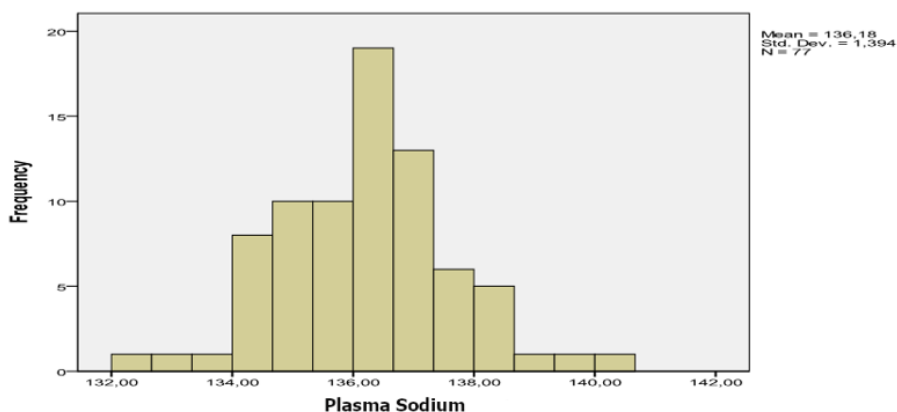


Figure 1. Distribution of serum sodium concentration among hemodialysis patients

± 2.9 mmol/L during the sDNA phase compared with 136.54 ± 1.59 mmol/L during the individualized dialysate sodium phase (iDNA phase), although the difference did not reach statistical significance ($p = 0.068$).

Comparison of the clinical outcomes between the two study phases demonstrated a statistically significant reduction in IDWG during the iDNA phase (2.17 ± 0.79 vs. 1.93 ± 0.64 kg, $p = 0.001$). No statistically significant differences were observed in systolic or diastolic blood pressure, or thirst score between the two phases (Table 2).

Dialysis adequacy improved significantly during the iDNA phase, reflected by higher Kt/V and URR values. Although mean Kt/V values remained within clinically acceptable ranges in both phases, the proportion of patients with inadequate dialysis adequacy ($Kt/V < 1.2$) decreased significantly during the individualized sodium phase, supporting the clinical relevance of this intervention (Table 3).

A comparison of biochemical parameters between the two study phases is presented in Table 4. Serum urea, creatinine and hemoglobin levels were significantly higher during the iDNA

Table 2. Comparison of clinical parameters using standard (sDNA) and individualized dialysate sodium (iDNA)

Parameters	sDNA phase	iDNA phase	p-value
IDWG (kg)	2.17 ± 0.79	1.93 ± 0.64	0.001*
Pre HD systolic BP (mm Hg)	128.28 ± 18.22	127.74 ± 17.54	0.098
Post HD systolic BP (mm Hg)	119.80 ± 17.52	119.18 ± 17.53	0.059
Pre HD diastolic BP (mm Hg)	79.23 ± 12.68	78.21 ± 12.81	0.063
Post HD diastolic BP (mm Hg)	73.85 ± 10.56	72.70 ± 11.39	0.055
DTI	11.42 ± 3.80	10.74 ± 3.40	0.099
Post HD serum sodium (mmol/L)	138.9 ± 2.9	136.54 ± 1.59	0.068

DTI= Dialysis Thirst Inventory; IDWG= interdialytic weight gain

Table 3. Comparison of dialysis adequacy and nutrition parameters between sDNA phase and iDNA phase

Parameters	sDNA phase	iDNA phase	p-value
Kt/V	1.36 ± 0.22	1.50 ± 0.24	0.000*
Kt/V < 1.2, n(%)	20 (26%)	9 (11.7%)	0.031*
URR (%)	67.00 ± 6.23	70.80 ± 5.24	0.000*
BMI (kg/m ²)	23.96 ± 3.82	24.12 ± 4.07	0.312
PCR (g/kg/day)	0.83 ± 0.021	0.90 ± 0.01	0.001*
Serum albumin (g/L)	34.46 ± 2.53	38.28 ± 3.80	0.000*

BMI- body mass index; URR-urea reduction rate; PCR-protein catabolic rate

phase. Serum potassium levels were significantly lower during the iDNa phase, while no statistically significant differences were observed for serum sodium, calcium, phosphorus, PTH or CRP levels.

During the study period, seven hospitalizations occurred, including four due to cardiovascular events, one vascular access malfunction, one bronchopneumonia, and one parathyroidectomy. Seven deaths were recorded, six due to cardiovascular causes and one due to polytrauma.

The differences in the clinical and laboratory parameters between surviving and deceased patients are presented in Table 5. Deceased patients had significantly lower Kt/V, URR, serum potassium, and albumin levels, as well as signifi-

cantly higher serum phosphorus and CRP levels. Although serum sodium differed between survivors and deceased patients in univariate comparison, it was not significantly associated with mortality in regression analysis. CRP values remained relatively low in the overall cohort, although substantially higher levels were observed among deceased patients.

Logistic regression analysis identified Kt/V <1.2, URR <65% and CRP >10 mg/L as factors significantly associated with mortality. CRP >10 mg/L showed the strongest association with mortality in logistic regression analysis (Table 6). However, because only seven mortality events occurred during the follow-up, these findings should be interpreted cautiously due to the limited events-per-variable ratio and the potential risk of model overfitting.

Table 4. Biochemical parameters during sDNa phase and iDNa phase

Hemoglobin (g/L)	114.62±10.34	120.32±10.14	0.001*
Serum sodium (mmol/L)	136.18±1.39	136.96±1.29	0.07
Serum potassium (mmol/L)	5.62±0.60	5.15±0.94	0.001*
Serum calcium (mmol/L)	2.31±0.14	2.33±0.14	0.262
Serum phosphate (mmol/L)	1.48±0.35	1.43±0.35	0.156
PTH (pg/mL)	405.49±421.39	432.48±463.68	0.234
CRP (mg/L)	3.65±1.94	3.91±2.08	0.424

PTH-parathyroid hormone; CRP-C reactive protein

Table 5. Comparison of clinical and laboratory parameters between surviving and deceased patients

Variable	Survivors (N=70)	Deceased (N=7)	p-value
Age (years)	55.00 ± 10.47	63.00 ± 9.20	0.12
HD session duration (min)	258.60 ± 14.89	254.08 ± 14.04	0.644
Kt/V	1.54 ± 0.23	1.29 ± 0.12	0.005*
URR (%)	71.55 ± 5.06	65.90 ± 3.69	0.007*
PCR (g/kg/day)	0.91 ± 0.12	0.82 ± 0.13	0.207
Hemoglobin (g/L)	121.31 ± 9.38	113.86 ± 13.26	0.121
Serum urea (mmol/L)	20.91 ± 2.87	18.47 ± 3.72	0.115
Serum creatinine (μmol/L)	767.61 ± 132.89	673.63 ± 118.82	0.128
Serum sodium (mmol/L)	136.85 ± 1.33	137.71 ± 0.77	0.018*
Serum potassium (mmol/L)	5.21 ± 0.96	4.82 ± 0.70	0.036*
Serum calcium (mmol/L)	2.33 ± 0.13	2.35 ± 0.20	0.90
Serum phosphorus (mmol/L)	1.18 ± 0.33	1.73 ± 0.35	0.024*
Serum albumin (g/L)	39.18 ± 1.98	32.40 ± 7.04	0.003*
CRP (mg/L)	6.09 ± 5.73	21.50 ± 17.18	0.004*
Ferritin (μg/L)	265.81 ± 178.20	206.24 ± 141.46	0.478
PTH (pg/mL)	421.65 ± 461.98	503.70 ± 505.80	0.636
Dialysate sodium (mmol/L)	136.63 ± 1.12	136.85 ± 0.89	0.552
post-HDserum sodium (mmol/L)	134.77 ± 2.20	135.00 ± 1.15	0.712

HD – hemodialysis; URR-urea reduction rate; PCR-protein catabolic rate

Table 6. *Univariate binary logistic regression analysis of predictors associated with mortality in hemodialysis patients treated with individualized dialysate sodium*

Variable	B coefficient	OR	95% CI for OR	p-value
Kt/V <1.2	2.174	8.800	(1.008-76.863)	0.049*
URR < 65%	2.392	10.933	(1.878-63.644)	0.008*
CRP>10 mg/L	2.330	10.278	(1.709-61.826)	0.011*

URR-urea reduction rate; CRP-C reactive protein

DISCUSSION

In this prospective single-center interventional study, individualized dialysate sodium prescription based on patients' pre-hemodialysis serum sodium "set points" was associated with lower interdialytic weight gain, improved dialysis adequacy (Kt/V and URR) and better nutritional parameters, including higher serum albumin and protein catabolic rate. Although the mean Kt/V values remained within the clinically acceptable ranges during both study phases, the proportion of patients with inadequate dialysis adequacy (Kt/V <1.2) decreased significantly during the individualized sodium phase, suggesting clinically meaningful improvement beyond statistical significance.

The high prevalence (89.6%) of pre-HD serum sodium concentrations below the standard dialysate sodium concentration (138 mmol/L) supports the presence of a positive sodium gradient during the sDNA phase, potentially contributing to sodium loading, thirst, and fluid retention. These findings are consistent with the concept of an individualized sodium "set point" previously described in hemodialysis patients with minimal intra-individual sodium variation over time [9, 10]. By minimizing the sodium gradient, individualized sodium prescription may reduce net sodium transfer during dialysis and improve fluid balance. The observed post-dialysis reduction in serum sodium and potassium may reflect mobilization of non-osmotic sodium stores and shifts in transcellular ion gradients mediated by Na⁺/K⁺-ATPase activity, consistent with findings reported by Conte et al. [11].

Our findings are in agreement with previous studies demonstrating that sodium loading during hemodialysis contributes to increased interdialytic weight gain and volume overload [12,13]. In the present study, individualized sodium prescription significantly reduced IDWG,

while blood pressure and thirst scores showed non-significant trends toward improvement. Longer follow-up periods and larger patient cohorts may be required to demonstrate statistically significant hemodynamic effects.

Serum potassium levels decreased significantly during the individualized sodium phase, possibly reflecting improved extracellular volume control and transcellular ion redistribution. In contrast, calcium, phosphate and PTH levels did not differ significantly between study phases, suggesting limited short-term effects of sodium individualization on mineral metabolism. The observed reduction in potassium levels is consistent with previous reports suggesting altered potassium rebound under lower dialysate sodium conditions [14, 15].

Unlike the DOPPS study, which reported increased mortality among patients with lower serum sodium concentrations [16], serum sodium and dialysate sodium concentrations were not independently associated with mortality in our cohort. This discrepancy may reflect differences in patient selection, extracellular volume status, sodium handling, and study size, as previously discussed by Causland et al. [17].

Elevated CRP demonstrated the strongest observed association with mortality in the logistic regression analysis, emphasizing the potential importance of systemic inflammation in hemodialysis outcomes, consistent with previous reports [18]. The relatively low mean CRP values observed in both study phases may partly reflect the exclusion of diabetic patients and the absence of active infectious episodes during routine laboratory assessment. This may partly explain the modest inflammatory profile observed despite the presence of cardiovascular comorbidity. Survivors demonstrated better dialysis adequacy, higher serum albumin and potassium levels and lower phosphorus and CRP levels, supporting previously established associations between inflammation, nutritional status, dialysis adequacy

and survival in hemodialysis patients [18–20]. Improved albumin levels may reflect better nutritional status, reduced inflammatory burden, or altered extracellular volume status during individualized sodium prescription.

However, mortality findings should be interpreted cautiously because only seven deaths occurred during the follow-up. The limited number of events reduces the robustness of regression analysis and increases the possibility of model overfitting. In this cohort, elevated CRP was associated with mortality and showed the strongest observed association in regression analysis; however, larger multicenter studies are required to confirm its independent prognostic significance.

The study has several limitations. It was conducted at a single center with a relatively small sample size and excluded diabetic patients, which may limit generalizability, as diabetic nephropathy represents a substantial proportion of the global hemodialysis population. In addition, detailed longitudinal data regarding antihypertensive therapy, erythropoiesis-stimulating agent dose adjustments, ultrafiltration rates, and other medications were not systematically recorded and may represent potential confounding factors. The relatively wide confidence intervals observed in regression analysis likely reflect the limited number of mortality events. Furthermore, the study evaluated mortality as a binary outcome without formal survival analysis. The absence of Kaplan–Meier survival analysis and Cox proportional hazards modeling further limits the interpretation of long-term prognostic associations. Because prognostic significance in hemodialysis populations strongly depends on the follow-up duration, longer-term studies with survival analyses over periods of at least five years are needed.

Despite these limitations, the present study suggests that individualized dialysate sodium prescription may represent a useful strategy for improving fluid management and dialysis adequacy in selected non-diabetic hemodialysis patients. Further prospective multicenter studies are needed to better define the relationship between sodium management, inflammation and long-term cardiovascular and survival outcomes.

CONCLUSION

Individualized dialysate sodium prescription based on patients' pre-dialysis sodium "set points" was associated with lower interdialytic weight gain, improved dialysis adequacy and better nutritional parameters without adverse effects on electrolyte balance. Although serum and dialysate sodium concentrations were not independently associated with mortality, elevated CRP levels were significantly associated with mortality risk, supporting the potential role of systemic inflammation in hemodialysis outcomes. Larger multicenter studies with longer follow-up are needed to confirm these findings and further evaluate the long-term clinical impact of individualized sodium prescription in hemodialysis patients.

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Резиме

НАДВОР ОД УЛОГАТА НА НАТРИУМОТ: ВОСПАЛЕНИЕТО Е НАЈСИЛНИОТ ПРЕДИКТОР НА СМРТНОСТ КАЈ ПАЦИЕНТИТЕ НА ХЕМОДИЈАЛИЗА БЕЗ ДИЈАБЕТЕС

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Вовед: Стандардната концентрација на дијализатниот натриум (сДНа) можеби не ги одразува индивидуалните потреби за натриум кај секој хемодијализен пациент, што потенцијално може да придонесе за интердијализно зголемување на телесната тежина (ИДЗТ), хипертензија и воспаление. Целта на оваа студија беше да се споредат клиничките и биохемиските исходи меѓу индивидуализирани и стандардни прескрипции на дијализатен натриум и да се проценат факторите поврзани со морталитетот.

Материјали и методи: Студијата беше проспективна, интервенциска, спроведена во две фази. Во првата фаза пациентите беа третирани со стандардна хемодијализа (ХД) со фиксна концентрација на дијализатен натриум од 138 mmol/L. Во втората фаза дијализатниот натриум беше индивидуализиран врз основа на преддијализната серумска концентрација на натриум кај секој пациент. Анализирани беа ИДЗТ, крвниот притисок, индексот на жед, адекватноста на дијализа (Kt/V, URR- стапка на редукција на уреа), нутритивните маркери, CRP (Ц реактивен протеин), електролитите и морталитетот.

Резултати: Индивидуализираната прескрипција на дијализатен натриум беше поврзана со значајно намалување на ИДЗТ ($1,93 \pm 0,64$ vs $2,17 \pm 0,79$ kg; $p = 0,001$) и подобрување на дијализна адекватност (Kt/V: $1,5 \pm 0,24$ vs $1,36 \pm 0,22$; $p < 0,001$). Пациентите што преживеаја имаа повисоки вредности на албумин и пониски вредности на CRP. Серумскиот и дијализатниот натриум не беа независно поврзани со морталитетот. Во логистичка регресија CRP > 10 mg/L покажа најсилна асоцијација со морталитет (OR 10,278; 95 % CI 1,709–61,826; $p = 0,011$).

Заклучок: Индивидуализираната прескрипција на дијализатен натриум може да ја подобри контролата на течностите и дијализната адекватност кај селектирани пациенти. Воспалението беше значајно асоцирано со морталитет во оваа кохорта, иако резултатите треба да се интерпретираат внимателно поради ограничениот број настани.

Клучни зборови: хемодијализа, дијализни раствори, натриум градиент, воспаление, морталитет