

EFFECTS OF STANDARD AND INDIVIDUALIZED DIALYSATE SODIUM IN CHRONIC HEMODIALYSIS PATIENTS UPON ECHOCARDIOGRAPHY PARAMETERS

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ABSTRACT

Aims: This study assessed the effects of individualized dialysis sodium prescription on changes of echocardiography in hemodialysis (HD) patients.

Methods: 77 chronic hemodialysis patients were analysed. In the first phase all patients underwent dialysis with standard dialysate sodium of 138 mmol/L followed by the second phase where dialysis was performed with individualized dialysate sodium concentration according to average pre HD serum sodium concentration. After the first phase, the subjects were divided into 3 groups: normotensive, hypertensive and hypotensive based on the average pre-HD systolic BP during the first phase. In all patients echocardiography was performed at the end of the first and second phase.

Results: Patients had no statistical significant change in blood pressure compared with standard dialysate sodium, only statistical significant change in interdialytic weight gain (IDWG). By dividing the patients, sodium individualization resulted in significantly lower blood pressure and IDWG ($p=0.018$) in hypertensive patients, whereas normotensive patients showed only significant decrease in IDWG ($p=0.004$). Hypertensive patients had significant highest sodium gradient compared to other patients ($p<0.05$), followed by significant increase of 0,6% IDWG confirmed with univariate regression analysis. In all patients, echocardiography analysis showed an increase of 2.04 mm of LVDD by increasing the sodium gradient for 1 mmol/L and significantly increased LVM of 35.69 gr by 1 kg increase in IDWG.

Conclusions: A reduction of the dialysate sodium concentration based on the pre HD serum sodium level of the patient, reduced the SBP, DBP and IDWG and decreased the volume overload upon the heart and consequently heart hypertrophy assessed by echocardiography.

Keywords: dialysate sodium, interdialytic weight gain, left ventricular hypertrophy

INTRODUCTION

Individual hemodialysis patients have a stable concentration of pre-dialysis serum sodium over time, suggesting the existence of an individual sodium set point, with small variations of only 1-2%. Sodium balance during dialysis depends on sodium diffusion and convection. Convection is generally higher compared to diffusion. [1] The amount of sodium removed by convection largely depends on the magnitude of the ultrafiltration, and this is more or less equal to the product of the ultrafiltration volume and the plasma sodium concentration. The mechanisms of sodium removal by diffusion are more complicated. Sodium diffusion between blood and dialysate depends on its concentration in plasma water and dialysate. Only chemically active sodium is able to travel through the dialysis membrane by diffusion, and it is the difference between the activity of sodium in the blood and the activity of sodium in the dialysate that allows diffusion through the dialysis membrane. [2, 3, 4] Sodium influx can be expected in dialysis with supraphysiological dialysis sodium concentrations and in patients with low plasma sodium levels. However, there are still no clear data on sodium balance during dialysis. [5, 6]

Chronic hemodialysis patients have much higher cardiovascular morbidity and mortality compared to the general population. [7, 8] This increased mortality and morbidity can only be partially explained by traditional cardiovascular factors and pre-existing cardiac disease before starting treatment by hemodialysis. [7] Constantly increasing blood pressure and volume overload as a result of positive water and salt balance are considered to be the main risk factors for worsening of these conditions. The current literature recognizes that left ventricular hypertrophy is a major factor which contributes to a high rate of premature cardiovascular mortality in patients on dialysis. The mortality rate in the dialysis population is several times higher in relation to the general population, thus it has become a priority to find a way to slow down left ventricular hypertrophy in dialysis patients. Reduced exposure to sodium by reducing sodium dialysis may be a promising method. Therefore, additional investigations are needed in order to show wheth-

er there is an association between dialysate and serum sodium at morbidity and mortality rates.

This study assessed the effects of individualized dialysate sodium prescription on changes of echocardiography in hemodialysis patients.

METHODS

77 anuric, non-diabetic patients with end stage renal failure on chronic hemodialysis were studied (36 females, 41 males) with a mean age 65.90 ± 11.47 (35-86) years. The mean time on HD was 76.42 ± 67.71 (18-373) months. In the first phase, all patients underwent dialysis with a standard dialysate sodium of 138 mmol/L (12 weeks), followed by the second phase (which lasted one year), where in all patients dialysis was performed with an individualized dialysate sodium concentration, according to average pre HD serum sodium concentration measured over the course of one year. Laboratory analysis were taken in the middle of the week. Blood pressure levels were recorded every hour in each hemodialysis sessions in all patients during the study. According to the average of all measurements of blood pressure levels (before and after hemodialysis), after the first phase, patients were divided into 3 groups: normotensive, hypotensive, and hypertensive. Hypertension was defined as blood pressure greater than 140/90 mmHg, i.e. systolic blood pressure before hemodialysis greater than 140 mmHg or after hemodialysis greater than 130 mmHg or systolic blood pressure greater than 10 mmHg pre-post-dialysis in four of six consecutive hemodialysis. Intradialysis hypotension was defined as a blood pressure less than 110/60 mmHg or a drop in blood pressure greater than 20 mmHg during a hemodialysis session, and at least one drop in systolic blood pressure greater than 20 mmHg during a dialysis session associated with the presence of symptoms (headache, cramps, nausea, vomiting, collapse) or intervention (Trendelenburg position, saline, ultrafiltration reduction). We measured the dry weight of all patients and interdialytic weight gain (difference between body weight before and after the dialysis session). Echocardiography was performed at the end of the first and second phases. Left atrium (LA), left ventricle systolic diameter (LVSD), left ventricle diastolic diameter (LVDD), interventricular septum, left

ventricle mass (LVM), left ventricle mass index (LVMI), relative wall thickness (RWT) and ejection fraction (EF) were measured.

Statistical analysis was made using SPSS 17.0 and statistically significant value was considered when $p < 0.05$.

RESULTS

According to the study protocol, for 12 weeks all patients were dialyzed continuously with a standard dialysate sodium. The pre-dialysis plasma sodium concentration was 136.18 ± 1.39 mmol/L (range 132.53 to 140 mmol/L). The number of patients with pre-dialysis plasma sodium values less than 138 mmol/L was 69 (89.61%). Their distribution is shown on Chart 1.

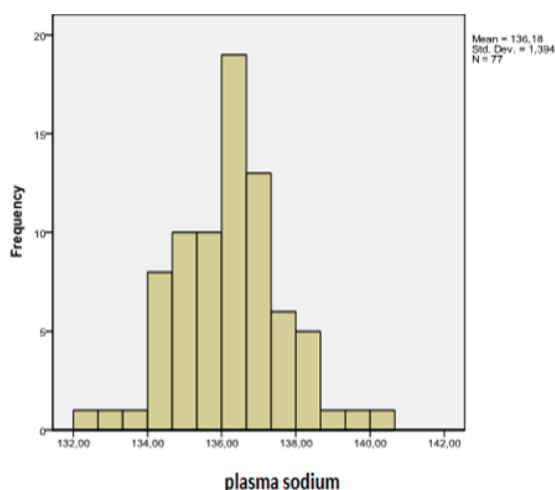


Chart 1. Histogram showing the distribution of pre-dialysis plasma sodium concentration in 77 subjects

When comparing the effects on blood pressure and IDWG using standard 138 mmol/L dialysis sodium compared with pre-hemodialysis plasma sodium, a statistically significant difference was found only in the pulse at the end of hemodialysis, but not in the blood pressure. We confirm statistically significant decrease in IDWG by using individualized dialysate sodium, as shown on table 1.

However, when patients were divided into 3 groups according to blood pressure (hypertensive, normotensive, and hypotensive), hypertensive patients were shown to have a significant

reduction in SBP before HD (148.42 ± 2.50 versus 151.21 ± 2.70 mm Hg; $p < 0.001$), DBP before HD (84.55 ± 9.92 versus 87.40 ± 10.61 mmHg; $p = 0.002$), SBP after HD (133.31 ± 11.71 versus 135.47 ± 12.42 , $p < 0.001$) and DBP after HD (76.30 ± 9.32 versus 79.14 ± 9.75 , $p = 0.004$) in individualized dialysate sodium, according to the pre-hemodialysis plasma sodium concentration compared to standard dialysis sodium. Normotensive patients did not show a statistically significant change in SBP and DBP before and after HD ($p > 0.05$). A statistically significant drop in heart rate was found in normotensive patients after HD (77.40 ± 9.96 versus 78.95 ± 12.30 , $p = 0.013$), but not in hypertensive patients. During standard sodium dialysis, both normotensive and hypertensive patients had a similar IDWG (2.15 ± 0.82 kg and 2.14 ± 0.75 kg, respectively). This was significantly reduced by individualization of dialysis sodium by pre-hemodialysis plasma sodium concentrations (both 1.88 ± 0.61 kg; $p < 0.001$ and 1.89 ± 0.58 kg; $p = 0.020$, respectively). Hypotensive patients had a statistically significant decrease in SBP before HD (80.13 ± 12.14 versus 77.80 ± 12.24 , $p = 0.002$) and DBP after HD (53.17 ± 13.78 versus 45.13 ± 10.93 , $p = 0.006$). Hypotensive patients have an increase in IDWG, but this is statistically insignificant (2.53 ± 0.57 versus 2.64 ± 0.84 , $p = 0.676$). A statistically significant decrease in heart rate post dialysis was noticed in normotensive and hypotensive patients. (Table 2).

Plasma sodium in all 3 groups of patients was close to 136 mmol/L, with a positive sodium gradient in all groups dialyzed with standard 138 mmol/L dialysis sodium. Hypertensive patients had the highest positive sodium gradient compared with normotensive and hypotensive patients. When patients were placed on sodium dialysis, according to pre-hemodialysis plasma sodium concentrations, there was no sodium gradient (zero-gradient eunatremic hemodialysis was performed).

The correlation between IDWG and sodium gradient in standard dialysis sodium did not show any statistical significance ($r = 0.004$; $p = 0.975$). After determining the IDWG as a percentage of the “ideal body weight”, however, a statistically significant correlation was shown ($r = 0.266$, $p = 0.019$), i.e. the two parameters were positively correlated, determinate by the increasing sodium gradient IDWG (Chart 2). However, no significant correlation was found between so-

Table 1. The difference in analyzed variables using standard dialysis sodium (DNa.138 mmol/L) compared to individual dialysis sodium according to the pre-hemodialysis plasma sodium concentration

Variable	Standard DNa (mmol/L)	Individualized DNa(mmol/L)	p-value
Pre HD SBP (mmHg)	128.28±18.22	127.74±17.54	0.098
Pre HD DBP (mmHg)	79.23±12.68	78.21±12.81	0.063
Pre HD heart rate (beats/min)	74.83±8.77	74.42±8.40	0.549
Post HD SBP (mmHg)	119.80±17.52	119.18±17.53	0.059
Post HD DBP (mmHg)	73.85±10.56	72.70±11.39	0.055
Post HD heart rate (beats/min)	79.75±11.65	78.04±9.26	<0.001*
IDWG(kg)	2.17±0.79	1.93+/-0.64	<0.001**

SBP = systolic blood pressure; DBP= diastolic blood pressure; IDWG = interdialytic weight gain; DNa=dialysate sodium

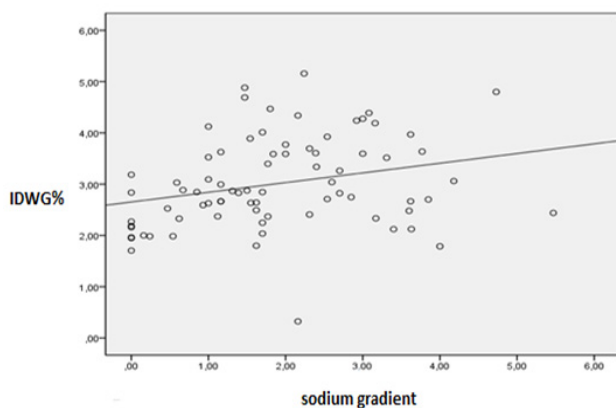
Table 2. Comparison of variables between standard dialysate sodium and individualized dialysatesodium according to pre-hemodialysis serum sodium concentrations in the three patient groups (normotensive, hypertensive, and hypotensive)

variable	Normotensive patients N=58		Hypertensive patients N=14		Hypotensive patients N=5	
Dialysate sodium (mmol/L)	sNa	iNa	sNa	iNa	sNa	iNa
Pre HD SBP (mmHg)	126.92±9.71	127.05±8.61	151.21±2.70	148.42±2.50*	80.13±12.14	77.80±12.24
Pre HD DBP(mmHg)	79.22±10.73	79.15±10.35	87.40±10.61	84.55±9.92*	56.58±13.14	49.51±9.34
Pre HD heart rate	74.42±9.42	73.09±8.42	76.54±6.54	79.15±6.72	74.73±6.76	76.60±8.59
Post HD SBP(mmHg)	119.95±10.72	120.03±9.79	135.47±12.42	133.31±11.71*	74.18±15.14	69.69±16.09
Post HD DBP(mmHg)	74.35±8.30	74.20±8.53	79.14±9.75	76.30±9.32*	53.17±13.78	45.13±10.93*
Post HD heart rate	78.95±12.30	77.40±9.96**	82.19±9.89	80.61±6.34	80.25±7.64	78.35±7.39*
IDWG(kg)	2.15±0.82	1.88±0.61**	2.14±0.75	1.89±0.58*	2.53±0.57	2.64±0.84
Serum sodium (mmol/L)	136.77±1.47		136.36±0.24		136.66±1.50	
Sodium gradient	1.21±1.49	//	1.63±0.80	//	1.30±0.81	//

SBP = systolic blood pressure; DBP= diastolic blood pressure; IDWG = interdialytic weight gain

dium gradient and blood pressure (SBP and DBP before and after HD) when analyzing patients as a single group.

Chart 2. Correlation between sodium gradient and IDWG as a percentage of “ideal body weight” (IDWG/BW%) in 77 subjects

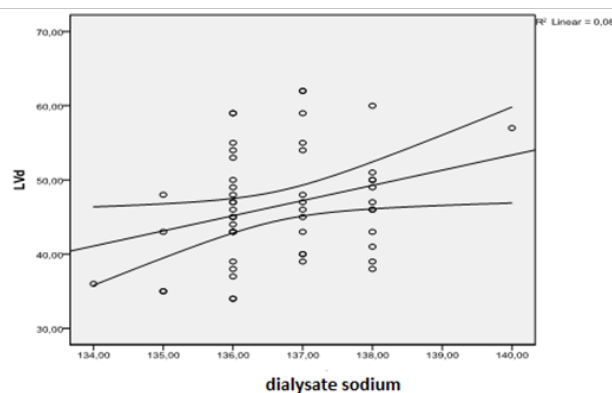


By using the linear regression method, we quantified the correlation between the sodium gradient and the IDWG as a percentage of the “ideal weight”. The results showed that by increasing the sodium gradient by 1 mmol/L, IDWG increased by an average of 0.189% (Beta 0.189). An R^2 value of 0.071 shows that 7.1% of the changes in IDWG, as a percentage of “ideal body weight”, can be explained by a change in the sodium gradient. Thus, linear regression confirmed the predictive value of the sodium gradient (Table 3)

In the second phase, we performed echocardiography in all 77 patients. In 6 patients the E/A ratio could not be determined due to observed cardiac arrhythmia (atrial fibrillation = AFF). In 9.8% of the analyzed patients, the E/A ratio was below 1, i.e. they had left ventricular diastolic dysfunction. Statistical analysis showed a statistically significant decrease in left ventricular mass and left ventricular mass index, but no statistically significant change in relative wall thickness (RWT) when patients were dialyzed with sodium according to pre HD plasma sodium concentration. (Table 4).

A statistically significant association of individualized dialysate sodium with LV diameter in diastole was confirmed ($r=0.298$, $p=0.03$), and both parameters were in positive association (Chart 3).

Chart 3. Correlation between individualized dialysate sodium and left ventricular dimension in diastole.



Quantifying this association with linear regression analysis, it was confirmed that increasing the dialysate sodium by 1 mmol/L, the size of the left ventricle in diastole increases by 2.04 mm, i.e. in 8.9% of dialysis patients, the change in the size of the left ventricular diastole can be explained by a change in dialysis sodium (Table 5).

No statistically significant association was found between individualized dialysate sodium and left ventricular systole diameter and right ventricular diameter. Also, the statistical analysis did not show any significant association, when using individualized dialysate sodium, with the marker for left ventricular diastolic dysfunction (E/A ratio) and with the more sensitive marker for diastolic left ventricular dysfunction (E/E ratio) ($r=0.172$, $p=0.227$; $r=-0.47$, $p=0.744$, respectively). Analysis of the association of IDWG and left ventricular mass, as well as IDWG and left ventricular mass index, showed a statistically significant positive association between the analyzed parameters ($r=0.346$, $p=0.011$, $r=0.314$, $p=0.022$, respectively), i.e. increasing IDWG increases left ventricular mass and left ventricular mass index (Charts 4 and 5). Logistic regression analysis confirmed that the increase in IDWG by 1 kg increases the left ventricular mass by 35 g, i.e. the increase in left ventricular mass in 12% of dialysis patients is due to increased IDWG (Table 6).

Table 3. Overview of the impact of sodium gradient on IDWG as a percentage of “ideal body weight” using linear regression analysis

Variable	Unstand. coefficient		Stand coefficient	T	p-value
	B	Std error	Beta		
IDWG(%)	2.653	0.179		14.861	0.000
Sodium gradient	0.189	0.079	0.266	2.394	0.019

IDWG = interdialytic weight gain

Table 4. Comparison of echocardiographic parameters with standard dialysate sodium and dialysate sodium according to pre HD serum sodium concentration.

Variable	Standard DNa	Individualized DNa	p- value
LVM(g)	236.26±72.58	189.79±63.06	<0.001*
LVMI (g/m ²)	140.52±42.23	112.47±35.10	<0.001*
RWT	0.45±0.08	0.44±0.09	0.181
LVd	52.86±6.45	48.33±6.55	<0.001*
LVs	34.19±6.21	33.38±5.65	0.466
LA	41.38±6.59	40.76±7.54	0.582
IVS	13.62±1.35	12.29±1.41	<0.001*

LVM= left ventricular mass; LVMI=left ventricular mass index; RWT=relative wall thickness; LVd=left ventricular diastole; LVs= left ventricle systole; LA= left atrium; IVS= interventricular septum; DNa=dialysate sodium

Table 5. Demonstration of the strength of correlation between left ventricular diameter and individualized dialysate sodium using logistic regression analysis.

variable	Unstand.coefficient		Stand coefficient	T	Sig.
	B	Std. Error	Beta		
LVd	-232.834	125.87		-1.858	0.069
DNa	2.044	0.917	0.298	2.230	0.030

LVd=left ventricular diastole; DNa=dialysate sodium

Table 6. Overview of the logistic linear regression analysis on IDWG impact upon left ventricular mass

Variable	Unstand. coefficient		Stand coefficient	t	p
	B	Std. Error	Beta		
LVM(g)	122.620	26.778		4.579	0.000
IDWG (kg)	35.698	13.546	0.346	2.635	0.011

LVM= left ventricular mass; IDWG – interdialytic weight gain

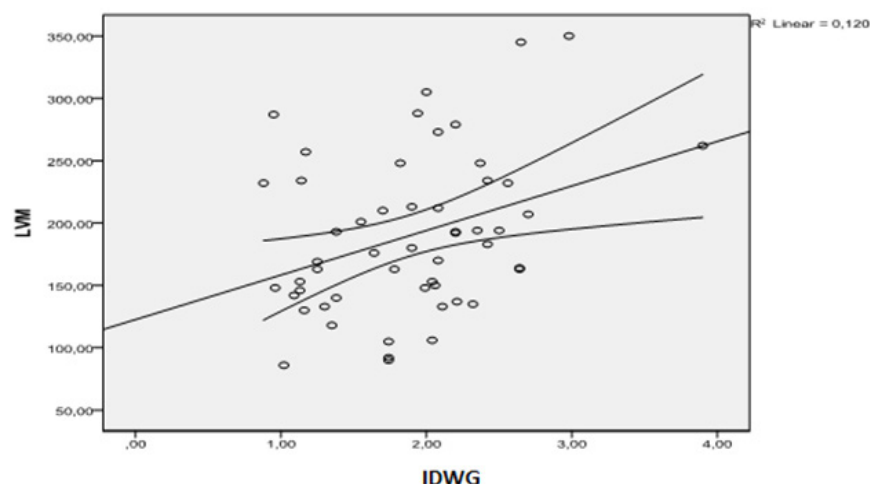


Chart 4. Overview of the correlation between IDWG and left ventricular mass in patients on individualized dialysate sodium.

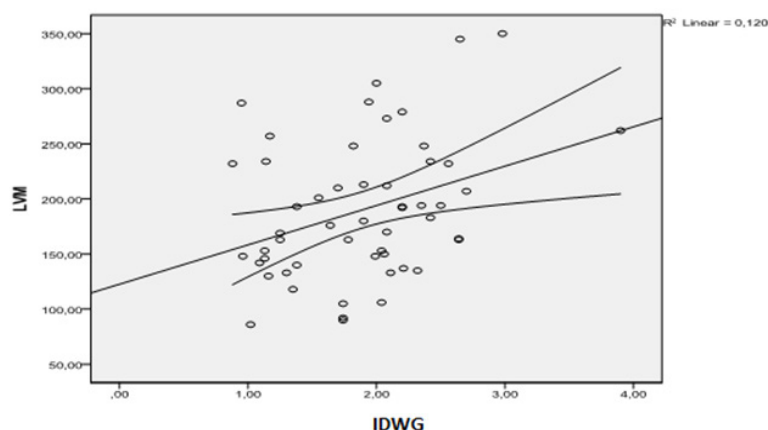


Chart 5. Overview of the linear association between IDWG and left ventricular mass index.

DISCUSSION

Our research was of a retrospective-prospective character, conducted in two phases. In the first phase, we analyzed the effects of standard dialysate sodium on blood pressure and IDWG, and in the second phase we analyzed the influence of individualized dialysate sodium according to pre HD serum sodium concentration. Analysis the effect of individualized dialysate sodium, according to the pre-hemodialysis plasma sodium concentration, did not confirm any statistically significant difference in blood pressure and IDWG compared to standard dialysate sodium (DNa 138 mmol/L) when all patients

were analyzed as one group. The same conclusion was published by De Paula et al. [9] We did not find a statistically significant association between the sodium gradient and IDWG. When we, however, determined IDWG as a percentage of “ideal body weight”, a statistically significant association was confirmed ($p=0.019$). The same association was confirmed in De Paula's study. [9] Quantifying the association with linear logistic regression, it was confirmed that with an increase of sodium gradient by 1 mmol/L, the average IDWG increases by 0.189%. In our study, the sodium gradient was significantly higher in hypertensive patients when compared to normotensive and hypotensive patients, therefore suggesting lower pre-hemodialysis plasma sodium concentrations in patients with poorly controlled

blood pressure and higher blood pressure. This resulted in thirst and volume load, i.e. higher IDWG ($p = 0.017$). This conclusion is also confirmed by a previously proven association between sodium gradient and IDWG in hypertensive patients. This was statistically much stronger compared to the previously proven association of both parameters in all patients, as a group. Via linear regression analysis, Keen and Gotch and Mendoza et al. showed a statistically significant association between sodium gradient size, interdialysis weight gain, and blood pressure in a smaller sample of hemodialysis patients. [10, 11] However, Hecking et al. reported that higher sodium dialysis was associated with greater interdialysis weight gain, but not with a higher risk of hospitalization and mortality. In fact, patients dialyzed with higher dialysate sodium had a significantly lower risk of hospitalization, even in centers where all or almost all patients were dialyzed with the same dialysate sodium, showing a significantly lower risk of mortality. [12] The analysis of the association of the individualized dialysate sodium, according to the pre-hemodialysis plasma sodium concentration with IDWG, did not confirm a statistically significant association in our study, which confirms that during the individualization of the dialyzed sodium HD, eunatremic hemodialysis is performed. Even higher dialysate sodium values do not lead to higher IDWG if they are adjusted to pre-hemodialysis plasma sodium concentrations ($p > 0.05$), as confirmed in our study. But research by Jung et al. has shown that increasing dialysate sodium based on plasma sodium concentrations increases IDWG and thirst in patients who have previously been dialyzed with a negative sodium gradient. [13]

Individualization of dialysate sodium to pre-hemodialysis plasma sodium concentration is a simple complementary strategy that limits sodium in dialysis, thus possibly helping reduce intradialytic weight gain in some patients. In our study, we did not find a direct correlation between the sodium gradient and blood pressure, correlation which was confirmed by other researchers. [14] We confirmed that, individualization of dialysate sodium to pre-hemodialysis plasma sodium concentration was well tolerated by patients, most likely due to lower IDWG and lower ultrafiltration rate, with almost no side effects (only a few cases of hypotension and muscle cramps). It is clear that the tendency to conduct eunatremic hemodialysis increases the risk of intradialysis

hypotension. Therefore, we believe that the individualization of dialysate sodium, according to the pre-hemodialysis plasma sodium concentration, mainly affects IDWG and leads to better blood pressure control in patients with poorly regulated blood pressure (volume-dependent hypertension), and our study group of patients is generally asymptomatic. On the other hand, this is not the case with hemodynamically stable patients, where individualization of dialysate sodium has no effect on blood pressure but only on IDWG, as confirmed in our study. Namely, normotensive patients did not have a statistically significant change in systolic and diastolic blood pressure pre- and post-hemodialysis, but they did have a statistically significant decrease in IDWG compared to standard dialysis sodium, which was confirmed ($p < 0.001$). Therefore, we believe that prescribing dialysate sodium according to pre-hemodialysis plasma sodium concentration is a safe method. Beside a significant decrease of blood pressure, we confirmed a statistically significant decrease of heart rate in normotensive and hypotensive patients, which can be explained by autonomic neuropathy, with decreased sympathetic activity, resulting in decreased heart rate.

The main concern about the individualization of dialysate sodium, according to pre-hemodialysis plasma sodium concentration, is the risk of hyponatremia and plasma hypoosmolality, as well as complications associated with both conditions, as there is no diffusion by sodium during dialysis. However, reduced IDWG leads to lower UF and, consequently, less sodium loss by convection, so we expect plasma sodium to remain unchanged (eliminating only sodium introduced during the interdialysis period).

The current literature recognizes that left ventricular hypertrophy (LVH) is a major factor contributing to the high rate of premature cardiovascular (CV) mortality in dialysis patients. Left ventricular hypertrophy plays a key role in sudden cardiac death in dialysis patients and is a strong independent risk factor of mortality. [15, 16] Therefore, it has become a priority to find a way to slow left ventricular hypertrophy in dialysis patients. Reduced sodium exposure by reducing dialysis sodium can be a promising method.

There is evidence to suggest that the reduction of LVM can be achieved by aggressive control of blood pressure and ultrafiltration

during HD. [17] The most effective intervention involves the use of frequent and long-lasting HD. Our study confirmed a statistically significant decrease in left ventricular mass and left ventricular mass index after individualization of dialysate sodium compared to standard dialysate sodium ($p < 0.001$). Although a statistically significant decrease in LVM was confirmed, the interventricular septum and left ventricular diastolic diameter, in contrast to, relative wall thickness, the left ventricular systolic diameter, and left atrium did not show significant change. In their study, Sayarlioglu et al. reported statistical reductions in LV systolic diameter, tricuspid regurgitation, inferior vena cava diameter, and pulmonary arterial pressure after dialysate sodium reduction, [18] whereas the study of Aybal et al. showed no changes in LVM after dialysate sodium reduction, but endothelial dysfunction was significantly reduced; this was confirmed by other studies. [19]

Our study confirmed a statistically significant positive association of IDWG and LV mass, as well as between IDWG and LV mass index ($r=0.346$, $p=0.011$; $r=0.314$, $p=0.022$). Linear regression analysis confirmed that the increase in IDWG per 1 kg increases the left ventricular mass by 35 g. LV hypertrophy is usually persistent and progressive, despite antihypertensive therapy in dialysis patients. A significantly more acceptable alternative may be to reduce sodium exposure by reducing dialysate sodium. Salt loading through increased dietary intake or diffusion during dialysis has been shown to increase blood pressure and IDWG. Elevated plasma sodium may induce hypertension, independent of increasing ECV, through mechanisms that mainly involve vascular endothelial stiffness. [20, 21] In our study, the positive linear association between individualized dialysate sodium and left ventricular diastole diameter was shown to be statistically significant.

In the study, we confirmed that there is no association between dialysis sodium and IDWG (even at different values of individualized dialysis sodium; $p > 0.05$), and we believe that IDWG in individual dialysis sodium is mainly due to interdialysis dietary salt intake. This finding confirms, that in addition to volume load, other factors (sodium by itself, endothelial dysfunction, decreased synthesis and nitric oxide release) are involved in the development of diastolic dysfunction and left ventricular hypertrophy. We consid-

er the cardioprotective form of hemodialysis to be frequent and long-lasting HD, with a lower dialysis sodium of 138 mmol/L. An important feature of this model is the improved intradialysis hemodynamic stability. Slow elimination of fluids and substances minimizes the risk of intradialysis hypotension and the development of disequilibrium. Ironically, this approach takes us back to the beginnings of dialysis: long dialysis with hypotonic dialysis used in the 1970s.

It may be considered that the duration of the individualized and standard sodium dialysis phase (1 year vs. 12 weeks) was a limitation of this study, although numerous studies have shown that serum sodium is stable over years, with small variations of 1-2 %, and patients in almost all dialysis centers are dialyzed with a standard dialysis sodium of 138 mmol/L. Patients are dialyzed with standard dialysis solution (Na 138 mmol/L, CH₃COO 3.0 mmol/L, Cl 110 mmol/L, Mg 1.0 mmol/L, K 2.0 mmol/L) in our institution as well, and in the first phase they continued to be dialyzed with the same dialysate sodium as before, therefore no change has been undertaken to the patients during the first phase, and, hence, no echocardiography changes were expected. Echocardiography was therefore performed after completion of the first phase, 12 weeks later, i.e. before the individualized phase, and again, after completion of the individualized phase in order to assess the possible impact of the intervention upon the echocardiography parameters.

CONCLUSION

It can be concluded that dialysate sodium is a fundamental component of hemodialysis which can be easily modified. However, research is still needed to prove the safety of using low dialysis sodium. The prescription of individualized sodium dialysate, according to the pre-hemodialysis plasma sodium concentration leads to: better control of interdialysis weight gain, reduction of subjective thirst, hemodynamic stability (maintenance of blood pressure in normotensive and hypotensive patients, control of blood pressure in hypertensive patients with uncontrolled blood pressure), reduced use of antihypertensive drugs, and reduced frequency of intradialysis complications and discomfort. The most important influencing factor on individualized dialysate sodium

was the improvement of cardiovascular parameters: reduction of left ventricular hypertrophy, improvement of left ventricular systolic and diastolic dysfunction as a result of decreased volume overload assessed by echocardiography.

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Резиме**ЕФЕКТИТЕ ОД СТАНДАРДНИОТ И ОД ИНДИВИДУАЛИЗИРАНИОТ
ДИЈАЛИЗАТЕН НАТРИУМ ВРЗ ЕХОКАРДИОГРАФСКИТЕ ПАРАМЕТРИ
КАЈ ПАЦИЕНТИ НА ХРОНИЧНА ХЕМОДИЈАЛИЗА****Наташа Ефтимовска-Отовиќ¹, Оливера Стојчева-Танева², Наташа Петковиќ³**¹ Градска општа болница 8 Септември, Одделение за нефрологија, 1000 Скопје, РС Македонија² Приватен центар за дијализа Дијаверум, 1000 Скопје, РС Македонија³ Градска општа болница, Оддел за кардиологија, 1000 Скопје, РС Македонија.

Цели: Студија ги процени ефектите од индивидуализираниот дијализатен натриум врз ехокардиографските промени кај пациентите на хемодијализа (ХД).

Методи: Анализирани се 77 пациенти на хронична хемодијализа. Во првата фаза сите пациенти беа дијализирани со стандарден дијализатен натриум од 138 mmol/L, проследено со втората фаза, каде што дијализите беа водени со индивидуализиран дијализатен натриум според просечната концентрација на натриум во серумот пред хемодијализата. По првата фаза испитаниците беа поделени во 3 групи според средниот пред-ХД систолен крвен притисок во текот на првата фаза на: нормотензивни, хипертензивни и хипотензивни. Кај сите пациенти е направена ехокардиографија на крајот од првата и од втората фаза.

Резултати: Пациентите немаа статистичка значајна промена во крвниот притисок во споредба со стандардниот дијализатен натриум, само статистичка значајна промена во интердијализното зголемување на телесната тежина (ИДДТ). Со поделбата на пациентите индивидуализацијата на натриумот резултираше во значително понизок крвен притисок и ИДДТ ($p = 0,018$) кај хипертензивните пациенти, додека нормотензивните пациенти покажаа само значително намалување на ИДДТ ($p = 0,004$). Хипертензивните пациенти имаа значајно најголем натриум градиент во споредба со другите пациенти ($p < 0,05$), проследено со значително зголемување на ИДДТ од 0,6 %, потврдено со униваријатна регресивна анализа. Кај сите пациенти ехокардиографската анализа покажа зголемување од 2,04 mm на ЛВДД со зголемување на градиентот на натриум за 1 mmol/L и значително зголемување на ЛВМ од 35,69 g за 1 kg зголемување на ИДДТ.

Заклучоци: Намалувањето на концентрацијата на дијализатниот натриум врз основа на нивото на натриум во серумот пред ХД на пациентот ги намалува СКП, ДКП и ИДДТ и го намалува волуменското преоптоварување на срцето и, следствено, срцевата хипертрофија проценета со ехокардиографија.

Клучни зборови: дијализатен натриум, интердијализно добивање во тежина, лево вентрикуларна хипертрофија