Dialysate sodium and sodium gradient in maintenance hemodialysis: a neglected sodium restriction approach?

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Abstract

Background. A higher sodium gradient (dialysate sodium minus pre-dialysis plasma sodium) during hemodialysis (HD) has been associated with sodium loading; however, its role is not well studied. We hypothesized that a sodium dialysate prescription resulting in a higher sodium gradient is associated with increases in interdialytic weight gain (IDWG), blood pressure (BP) and thirst.

Methods. We conducted a cross-sectional study on 1084 clinically stable patients on HD. A descriptive analysis of the sodium prescription was performed and clinical associations with sodium gradient were analyzed.

Results. The dialysate sodium prescription varied widely across dialysis facilities, ranging from 136 to 149 mEq/L, with a median of 140 mEq/L. The mean pre-HD plasma sodium was 136.7 ± 2.9 mEq/L, resulting in the majority of subjects (n = 904, 83%) being dialyzed against a positive sodium gradient, while the mean sodium gradient was 4.6 ± 4.4 mEq/L. After HD, the plasma sodium increased in nearly all patients (91%), reaching a mean post-HD plasma sodium of 141.3 ± 2.5 mEq/L. We found a direct correlation between IDWG and sodium gradient (r = 0.21, P < 0.0001). After adjustment for confounders and clustering by facilities, the sodium gradient was independently associated with IDWG (70 g/mEq/L, P < 0.0001). There were no significant associations among sodium gradient and BP, whether measured as pre-HD systolic (r = −0.02), diastolic (r = −0.06) or mean arterial pressure (r = −0.04). Post-HD thirst was directly correlated with sodium gradient (r = 0.11, P = 0.02).

Conclusion. Sodium gradient is associated with statistically significant and clinically meaningful differences in IDWG in stable patients on HD.

Keywords: dialysate sodium; hemodialysis; hypertension; interdialytic weight gain; sodium gradient

Introduction

Patients with end-stage renal disease (ESRD) on hemodialysis (HD) experience exceptionally high rates of cardiovascular mortality and morbidity [1]. Important risk factors include hypertension (HTN) [2,3] and fluid retention,
measured as interdialytic weight gain (IDWG) [4,5]. The normal renal regulatory mechanisms to control extracellular fluid volume and osmolality are no longer effective in patients with ESRD. In the absence of kidney function and urine output, the burden of maintaining volume homeostasis, electrolyte and acid–base balance falls solely on the HD procedure [6,7]. A large percentage of patients on HD require multiple antihypertensive agents, yet fail to achieve satisfactory control of HTN. The degree to which the dialysate prescription, and in particular the dialysate sodium concentration, influences blood pressure (BP) and IDWG via changes in sodium flux, plasma volume or other parameters is not well understood.

Sodium is removed during HD by both diffusion and convection (ultrafiltration). The contribution of diffusion and the direction of sodium transfer depend on the difference between dialysate and plasma sodium concentrations, the so called ‘sodium gradient’ [7, 8]. If the sodium gradient is positive, the plasma sodium concentration after HD will generally increase [9], potentially resulting in higher IDWG and BP by stimulating thirst [10]. These hypothesized relationships have been studied in only a small number of patients [6, 11].

This study aimed to describe the distribution of prescribed dialysate sodium concentrations in a medium-sized dialysis provider (Satellite Healthcare, Inc.) and to investigate the relationships among dialysate sodium, sodium gradient, IDWG, BP and thirst. We hypothesized that higher sodium gradients would be directly correlated with IDWG, mean arterial pressure, systolic BP and thirst.

Materials and methods

We conducted a cross-sectional study with data obtained from 1084 HD patients during the routine monthly blood draw in September 2009 in all 26 satellite healthcare dialysis facilities (21 located in California and 5 in Texas). We included clinically stable adult prevalent HD patients with minimal residual kidney function (urine output<100 mL/day) undergoing conventional thrice weekly in-center HD for at least 3 months. Patients were excluded if they had any of the following criteria: hospitalization within the preceding month, more than one missed treatment in the preceding 2 weeks, change in dialysate sodium prescription within the preceding 3 months, spKt/V<1.2, pre-HD plasma glucose > 300 mg/dL, HD session length shortened by >25% and plasma sodium not measured on the study day.

The majority of subjects were dialyzed with Fresenius F-180 or F-200 dialyzers, using primarily Fresenius 2008K or 2008H and Gambro Phoenix delivery systems. Automated BP machines (M-200-NIBP Colin, Press-Mates BP-8800C Colin or HD-BPM) and Tronix 6102, 6702 scales were used as per routine care in the facilities.

On the study day, 62% of patients undergoing conventional thrice weekly HD in participating dialysis facilities and 59% of the actual study population had a pre-HD BP ≥140/90 mmHg. All patients received dietary advice to limit salt intake to no >5 g/day as per standard of care. Following an initial assessment of diet, patients were evaluated monthly or more frequently if the IDWG was >4% of the dry weight. The dialysate sodium and dry weight were prescribed by the patient's treating nephrologist based upon clinical evaluation. The study was approved by the Satellite Health-care Research Committee.

We used the electronic medical record to collect data on patient demographics, BP (pre- and intradialytic and post-HD), adverse events (including headache, cramps, nausea and vomiting) and interventions (Trendelenburg position, saline use, reduction in ultrafiltration rate) for intradialytic hypotension, dry weight, dialysate sodium concentration and pre- and postdialysis weight.

Blood samples were collected prior to the first treatment of the week according to the standard protocol for monthly laboratory monitoring and processed at Satellite Laboratories (Redwood City, CA). Laboratory tests included albumin and pre- and post-HD plasma sodium (measured by direct ion-selective electrode) and blood urea nitrogen (BUN). Pre- and post-HD BUN were used for formal urea kinetic modeling, which included an estimated total body water and normalized protein equivalent of nitrogen appearance.

We assessed thirst in subjects from the 10 facilities with the highest variability in dialysate sodium prescription (>10% of subjects with higher or lower than 140 mEq/L) in order to capture a wide range of sodium gradients. All patients from the sampled facilities were asked to indicate their degree of thirst immediately before and after HD using a visual analogue scale (with 0 indicating no thirst and 10 very thirsty) in response to the question, ‘How thirsty are you now?’ Similar tools have been used in healthy individuals [12] and in HD patients [13].

Operational definitions

We estimated IDWG as the difference between pre-HD weight minus the post-HD weight reached after the previous HD session, calculated as the average of the preceding six available intervals, excluding the interdialytic periods of >3 days. The BP was estimated as the average sitting pre- or post-HD BPs from the last six HD sessions. We defined intradialytic hypotension as at least one episode of a fall in systolic BP >20 mmHg associated with symptoms or with any intervention during the last treatment. We calculated the mean plasma sodium by averaging the last three available monthly pre-HD plasma sodium concentrations over the preceding 6 months. If the patient was prescribed sodium modeling, we calculated the sodium gradient as the difference between the dialysate sodium time-averaged concentration (TACNa) [14] and the pre-HD plasma sodium.

Statistical analysis

Continuous variables were summarized as mean ± SD or median with interquartile range (IQR). Categorical variables were expressed as proportions. Differences in pre-HD plasma sodium among dialysate sodium groups were compared by one-way analysis of variance. Correlations were assessed with the Pearson product moment or Spearman rank coefficient. We used multivariable regression analysis to estimate the effect of sodium gradient on IDWG, while adjusting for age, sex, race, dialysis vintage (time since initiation of dialysis) and dry weight. We tested for the presence of interactions between sodium gradient and the other covariates (age, sex, race and dialysis vintage); no significant interactions were detected. The generalized estimated equations approach was used to account for clustering of patients at the facility level. Although alternate correlation structures were explored, the final estimates assumed an exchangeable correlation structure, meaning that pairs of patients from a given facility share the same correlation. We considered two-tailed P-values <0.05 as statistically significant. Statistical analyses were performed using SAS EG version 4.2 (SAS Institute, Cary, NC).

### Table 1. Clinical characteristics of patient population grouped by dialysate sodium concentrations

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Dialysate sodium concentration, mEq/L</th>
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<tbody>
<tr>
<td></td>
<td>140</td>
</tr>
<tr>
<td>Patients, %</td>
<td>560 (52)</td>
</tr>
<tr>
<td>Age, years</td>
<td>65 (53–76)</td>
</tr>
<tr>
<td>Male, %</td>
<td>290 (52)</td>
</tr>
<tr>
<td>Black, %</td>
<td>48 (8.6)</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>322 (58)</td>
</tr>
<tr>
<td>Dry body weight, kg</td>
<td>71.0 ± 14.8</td>
</tr>
<tr>
<td>Body water volume, L</td>
<td>32.1 ± 6.6</td>
</tr>
<tr>
<td>Vintage, months</td>
<td>45 (24–77)</td>
</tr>
<tr>
<td>Sodium modeling, %</td>
<td>20 (3.6)</td>
</tr>
<tr>
<td>Pre-HD plasma sodium, mEq/L</td>
<td>136 ± 2.9</td>
</tr>
<tr>
<td>Albumin, g/dL</td>
<td>3.8 ± 0.3</td>
</tr>
<tr>
<td>spKt/V</td>
<td>1.7 ± 0.3</td>
</tr>
<tr>
<td>nPNA</td>
<td>1.0 ± 0.1</td>
</tr>
</tbody>
</table>

*Values are expressed as mean ± SD, numbers (%) or median (25th–75th) to describe selected characteristics at baseline.*
Results

Of the 1397 adult prevalent HD patients with minimal residual kidney function undergoing thrice weekly in-center HD, 313 subjects (22%) were excluded according to one or more of the criteria outlined above, resulting in a final analytic sample of 1084 subjects.

Dialysate sodium prescription

The median dialysate sodium prescription was 140 mEq/L, ranging from 136 to 149 mEq/L. More than half of the patients (n=560, 52%) were dialyzed with a standard dialysate sodium of 140 mEq/L. Nearly one-third (n=332, 31%) were dialyzed with sodium modeling: 158 (47.6%) with a linear and 174 (52.4%) with a step protocol.

The patient characteristics are shown in Table 1, stratified by dialysate sodium prescription (140, <140 and >140 mEq/L). The patients treated with dialysate sodium concentrations <140 mEq/L were younger, heavier and more likely to be black, while patients with a dialysate sodium prescription >140 mEq/L had a longer dialysis vintage and a high proportion (77%) were prescribed sodium modeling.

The dialysate sodium prescription practices varied across facilities (Figure 1). Almost all the subjects (97%, n=123) prescribed a dialysate sodium <140 mEq/L were from the five Texas facilities, reflecting the practice of the treating nephrology group. A dialysate sodium prescription >140 mEq/L was used for all patients in one facility and was also used in ≥50% of the patients in another seven facilities. Sodium modeling was prescribed in nearly all fa-
cilities (24 of 26) with varying proportions: in six facilities at least 50% of patients were on sodium modeling and in one facility it was the exclusive prescription.

**Plasma sodium and sodium gradient**

The mean pre-HD plasma sodium concentration was 136.7 ± 2.9 mEq/L. The majority (n = 904, 83%) of patients had pre-HD plasma sodium concentrations <140 mEq/L, the most common dialysate sodium prescription (Figure 2). However, the pre-HD plasma sodium concentration was not significantly different among the patients dialyzed with different dialysate sodium concentrations (P = 0.12; Table 1).

After dialysis, the mean plasma sodium concentration increased to 141.3 ± 2.5 mEq/L. The vast majority (91%) of patients experienced an increase in plasma sodium concentration (post- versus pre-HD). The mean sodium gradient was 4.6 ± 4.4 mEq/L, with a wide range from −7 to 24 mEq/L (Figure 3). Only a small number of patients (n = 180, 17%) had a sodium gradient ≤ 0 mEq/L, and, of these, 124 were dialyzed against a negative gradient.

**Sodium gradient and IDWG**

The mean IDWG was 2.8 ± 1.1 kg, with a range from −0.7 to 7.2 kg. There was a direct correlation between IDWG and sodium gradient (r = 0.21, P < 0.0001; Figure 4). When IDWG was indexed to the estimated dry weight (i.e. estimated as percent IDWG), the correlation with sodium gradient was stronger (r = 0.30, P < 0.0001). Both

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**Fig. 3.** Frequency distribution of sodium gradient in 1084 HD patients.

**Fig. 4.** Correlation between sodium gradient and IDWG (r = 0.21, P < 0.0001).
Sodium gradient in HD

**Discussion**

Recommending dietary sodium restriction is a part of routine HD-related care for the management of both HTN and fluid overload. However, higher dialysate sodium prescriptions have been widely adopted in recent years in an attempt to decrease hemodynamic instability in patients undergoing HD [15]. This trend was evidenced in our population, despite variation in prescription practice at the facility level, where high dialysate sodium concentrations and positive sodium gradients were the rule.

We found a direct correlation between sodium gradient and IDWG (and percent IDWG), as previously reported in a smaller sample of 58 HD patients by Keen and Gotch [6]. The effect of sodium gradient on IDWG remained significant after taking into consideration other factors that may influence IDWG. The association between higher sodium gradients and higher IDWG is likely attributable to the increased post-HD plasma sodium [9,11,16,17].

Excessive fluid intake manifesting as IDWG can contribute to left ventricular hypertrophy and cardiovascular events. Kalantar-Zadeh et al. recently found IDWG ≥4.0 kg to be associated with a 25% increased risk of cardiovascular death compared to IDWG of 1.5–2.0 kg [4]. Although our current approach with dietary salt restriction may decrease left ventricular hypertrophy [18], BP [19–21] and IDWG [19,20], the benefits may be muted due to poor adherence. Individualizing the dialysate sodium prescription by matching dialysate sodium to the patient's pre-HD sodium [10,15] is a simple complementary strategy to restrict sodium in HD that may help reduce IDWG in some patients. We did not find a direct correlation between sodium gradient and pre-HD BP. However, any correlation may well be masked by the use of antihypertensive agents—and indeed, one would expect antihypertensive therapy to be maximized in those patients who are most prone to HTN with high IDWG. In a recent analysis of a large cohort of prevalent HD patients, a 1% increase in IDWG was only associated with a 1 mmHg increase in systolic BP [22]. Also, our cross-sectional design may have prevented us from detecting changes in BP that occur weeks to months after reducing dry weight—the ‘lag phenomenon’ [23,24].

The prescription of lower dialysate sodium concentrations in an attempt to reach eunatremia raises the concern of provoking intradialytic hypotension. However, as in previous studies [6, 25], we found no increased frequency of intradialytic hypotension in patients with negative or no sodium gradient and post-HD mean arterial pressure (r = 0.09, P = 0.005) and post-HD diastolic BP (r = 0.11) (P = 0.004). We found no increased frequency of intradialytic hypotension in patients with sodium gradients of zero or less compared to patients with positive sodium gradients (22 versus 24%, P = 0.52).

**Table 2.** Increase in IDWG/1 mEq/L increase in sodium gradient adjusted for clustering of patients at the facility level

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate</th>
<th>95% CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted model</td>
<td>0.05</td>
<td>0.04–0.06</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Multivariate adjusted model a</td>
<td>0.06</td>
<td>0.05–0.07</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Plus facility clustering</td>
<td>0.07</td>
<td>0.06–0.08</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*a*Adjusted by age, male, black, dry weight and vintage.

correlations remained similar in magnitude and statistically significant (i.e. non-zero) when excluding subjects on sodium modeling. After adjustment for age, sex, race, dialysis vintage, dry weight and accounting for clustering at the facility level, a 1 mEq/L increase in the sodium gradient was associated with a 70 g increase in IDWG (P < 0.0001). This was roughly equivalent to a 350 g difference in IDWG for a 5 mEq/L difference in sodium gradient (Table 2, Figure 4). Of note, the group of patients with a sodium gradient ≤0 mEq/L had an IDWG lower than subjects with positive sodium gradient (2.5 versus 2.9 kg, P < 0.001).

**Sodium gradient and BP**

We found no significant associations between sodium gradient and BP, whether the latter was measured as pre-HD systolic (r = −0.02), diastolic (r = −0.06) or mean arterial pressure (r = −0.04) (P > 0.05 for all comparisons). We found weak inverse correlations between sodium gradient and post-HD mean arterial pressure (r = −0.09, P = 0.005) and post-HD diastolic BP (r = −0.11) (P = 0.004).

We found no increased frequency of intradialytic hypotension in patients with sodium gradients of zero or less compared to patients with positive sodium gradients (22 versus 24%, P = 0.52).

**Sodium gradient and thirst**

We noted a surprisingly low degree of self-reported thirst measured by a visual analogue scale (from 0 to 10). The median thirst score was 2 pre-HD (IQR 1–5) and 2 post-HD (IQR 0–5). Post-HD thirst was directly, albeit weakly, correlated with sodium gradient (r = 0.11, P = 0.02), but there was no correlation between sodium gradient and pre-HD thirst (r = −0.04, P = 0.37).

Increasing post-HD plasma sodium [9,11,16,17]. Excessive fluid intake manifesting as IDWG can contribute to left ventricular hypertrophy and cardiovascular events. Kalantar-Zadeh et al. recently found IDWG ≥4.0 kg to be associated with a 25% increased risk of cardiovascular death compared to IDWG of 1.5–2.0 kg [4]. Although our current approach with dietary salt restriction may decrease left ventricular hypertrophy [18], BP [19–21] and IDWG [19,20], the benefits may be muted due to poor adherence. Individualizing the dialysate sodium prescription by matching dialysate sodium to the patient's pre-HD sodium [10,15] is a simple complementary strategy to restrict sodium in HD that may help reduce IDWG in some patients. We did not find a direct correlation between sodium gradient and pre-HD BP. However, any correlation may well be masked by the use of antihypertensive agents—and indeed, one would expect antihypertensive therapy to be maximized in those patients who are most prone to HTN with high IDWG. In a recent analysis of a large cohort of prevalent HD patients, a 1% increase in IDWG was only associated with a 1 mmHg increase in systolic BP [22]. Also, our cross-sectional design may have prevented us from detecting changes in BP that occur weeks to months after reducing dry weight—the ‘lag phenomenon’ [23,24].

The prescription of lower dialysate sodium concentrations in an attempt to reach eunatremia raises the concern of provoking intradialytic hypotension. However, as in previous studies [6, 25], we found no increased frequency of intradialytic hypotension in patients with negative or no sodium gradient or less (in theory the highest risk group) compared to patients with positive sodium gradients. Indeed, two other studies have reported a reduction in the frequency of intradialytic hypotension after decreasing the dialysate sodium [11, 16]. Additionally, biofeedback systems that modulate blood volume contraction by adjusting the ultrafiltration and dialysate conductivity have been shown to decrease intradialytic hypotension without salt loading [26].

As reported previously, the sodium gradient was associated with post-HD thirst [11,16]. The low degree of thirst scores reported here may be related to the timing of the self-assessment. It has been previously shown that thirst is more commonly experienced 4–6 h after the end of
HD [27], rather than immediately before and after HD as in our study. In addition, our visual analogue instrument may have had limited discriminatory power.

Some authors have suggested the concept of a unique pre-HD plasma sodium, the so-called sodium ‘set-point’ for each patient [6,10,15,17]. Supporting this hypothesis, recently, Peixoto et al. [28] observed stable long-term pre-HD sodium concentrations in a veterans population predominantly using a single dialysate sodium prescription. In our larger and more diverse patient population, we found no differences in the pre-HD plasma sodium concentrations among patients grouped by dialysate sodium prescription ranges, but we need prospective studies to confirm this observation. Whether lower pre-HD plasma sodium is the cause or the consequence of excess fluid intake remains unanswered. Lindley [29] postulated that there are two groups of patients on HD: one group with normal plasma sodium drinking water in response to sodium-induced osmometric thirst and another group with low plasma sodium ingesting fluid due to non-salt-related reasons, such as xerostomia, comfort or social drinking.

To the best of our knowledge, this is the largest reported study of clinically stable adult patients on HD with minimal residual kidney function dialyzed under current techniques with different dialysate sodium concentrations and a broad range of sodium gradients, demonstrating an association of sodium gradient and IDWG.

There are several important limitations to this study. The cross-sectional design precludes the establishment of a causal relationship between sodium gradient and IDWG. Sodium balance was not calculated by including fluid and salt intake, and actual dialysate and ultrafiltrate sodium were not measured. Thirst and intradialytic hypotension data were obtained in a subset of patients; comorbidities and antihypertensive agent use were not captured. Finally, the absence of an association between sodium gradient and BP could reflect the limited precision and lack of uniformity with which BP was measured, as well as the effect of prescribed antihypertensive medication. In future studies, more comprehensive determinations of BP, including ambulatory BP monitoring, could be employed [30]. Moreover, interventional studies would help to determine whether changes in sodium gradient and IDWG induced by lower dialysate sodium prescriptions would yield lower BPs over time.

In conclusion, the sodium gradient is associated with statistically significant and clinically meaningful differences in IDWG in stable patients on HD and possibly with post-HD thirst. Prospective studies altering dialysate sodium prescription to reduce the sodium gradient with or without matching the dialysate sodium to the patient’s pre-HD plasma sodium are needed to determine a causal relationship and estimate clinical benefit.

Conflict of interest statement. My statement (on behalf of all the authors) is as follows: we have had no involvements that might raise the question of bias in the work reported or in the conclusions, implications or opinions stated.

J.M.M. MD: ‘The results presented in this paper have not been published previously in whole or part, except in abstract form’.

(See related article by Lomonte et al. Do not forget to individualize dialysate sodium prescription. Nephrol Dial Transplant 2011; 26: 1126–1128)

References


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**Comparison of 4- and 8-h dialysis sessions in thrice-weekly in-centre haemodialysis**

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**Abstract**

**Background.** Longer dialysis sessions may improve outcome in haemodialysis (HD) patients. We compared the clinical and laboratory outcomes of 8- and 4-h thrice-weekly HD.

**Methods.** Two-hundred and forty-seven HD patients who agreed to participate in a thrice-weekly 8-h in-centre nocturnal HD (NHD) treatment and 247 age-, sex-, diabetes status- and HD duration-matched control cases to 4-h conventional HD (CHD) were enrolled in this prospective controlled study. Echocardiography and psychometric measurements were performed at baseline and at the 12th month. The primary outcome was 1-year overall mortality.

**Results.** Overall mortality rates were 1.77 (NHD) and 6.23 (CHD) per 100 patient-years (P = 0.01) during a mean 11.3 ± 4.7 months of follow-up. NHD treatment was associated with a 72% risk reduction for overall mortality compared to the CHD treatment (hazard ratio = 0.28, 95% confidence interval 0.09–0.85, P = 0.02). Hospitalization rate was lower in the NHD arm. Post-HD body weight and serum albumin levels increased in the NHD group. Use of antihypertensive medications and erythropoietin declined in the NHD group. In the NHD group, left atrium and left ventricular end-diastolic diameters decreased and left ventricular mass index regressed. Both use of phosphate binders and serum phosphate level decreased in the NHD group. Cognitive functions improved in the NHD group, and quality of life scores deteriorated in the CHD group.

**Conclusions.** Eight-hour thrice-weekly in-centre NHD provides morbidity and possibly mortality benefits compared to conventional 4-h HD.

**Keywords:** in-centre haemodialysis; nocturnal; outcomes; survival

**Introduction**

The mortality rate of patients treated with haemodialysis (HD) remains unacceptably high despite several improvements in dialysis technology and general medical care [1]. The low level of quality of life (QOL) is also an important concern in this population.