Renal Replacement Therapy

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# Prediction of end-dialysis serum sodium concentration in severely hyponatremic kidney failure patients undergoing conventional hemodialysis using sodium kinetic equation

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## Abstract

**Background and Objectives** Conventional hemodialysis (HD) for kidney failure patients with severe hyponatremia may be complicated by rapid correction of hyponatremia, which increases the risk of osmotic demyelination syndrome. A simple sodium kinetic equation was effective in prediction of end-dialysis serum $Na^+$ in severely hyponatremic kidney failure patient treated with continuous venovenous hemofiltration, but was not tested in conventional HD. The aim of this study was to assess the validity of this equation when used in conventional HD.

**Methods** Twenty conventional HD sessions were delivered to 12 kidney failure patients with severe hyponatremia (serum $Na^+$ < 120 mEq/L). The target change in serum $Na^+$ was 4 mEq/L. The $D_{Na}$.t/V that obtained this change was predetermined according to the sodium kinetic equation and monitored in real time by online clearance monitoring software embedded in dialysis machine. The dialysis session was terminated once the target $D_{Na}$.t/V was achieved.

**Results** The mean observed and predicted serum $Na^+$ were 119.80 ± 3.42 mEq/L and 119.45 ± 3.12 mEq/L, respectively. Bland–Altman plot analysis revealed a mean difference ± SD of 0.33 ± 1.26 mEq/L, and 95% limits of agreement of − 2.13 to 2.83. The imprecision in prediction of end-dialysis serum $Na^+$ was 2.52 mEq/L. The small difference and clinically insignificant 95% limits of agreement indicate a good agreement between the observed and predicted serum $Na^+$.

**Conclusion** The sodium kinetic equation was effective in prediction of end-dialysis serum $Na^+$ in kidney failure patients with severe hyponatremia.

**Keywords** Severe hyponatremia, Conventional hemodialysis, Sodium kinetic equation, Online clearance monitoring

## Introduction

Hyponatremia is not uncommon in patients with kidney failure. The prevalence and incidence of hyponatremia are higher in CKD patients than in non-CKD patients [1, 2]. In kidney failure patients with severe hyponatremia, conventional intermittent hemodialysis (IHD) may result in rapid correction with a risk of developing ODS [3]. This is due to the inability to reduce the dialysate $Na^+$ to less than 130 mEq/L and the use of a relatively high blood flow rate. Despite this risk, ODS occurs less frequently in patients with kidney failure who receive renal replacement therapy (RRT) due to the protective effect of azotemia [4].

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Complex sodium kinetic models [5] have been developed for many years and are quite accurate in determining the end-dialysis serum $Na^+$ or the dialysate $Na^+$ that achieves a predetermined end-dialysis serum $Na^+$. They are used in maintenance hemodialysis (MHD) to provide a neutral sodium balance, but have not previously been studied in severe hyponatremia correction in kidney failure patients who require RRT. Yessayan et al. [6] proposed a sodium kinetic equation for estimating end-dialysis serum $Na^+$ levels during RRT. The reliability of this equation was examined in severe hyponatremia correction during continuous venovenous hemofiltration but not in conventional IHD. By using the kinetic equation and OCM, it is possible to estimate the dialysis time point at which a predetermined intradialytic safe change of serum $Na^+$ is reached. Subsequently, we can stop the dialysis session at this time in order to avoid rapid correction. The aim of this study is to assess the validity of this sodium kinetic equation in predicting end-dialysis serum $Na^+$ in severely hyponatremic kidney failure patients treated with conventional IHD.

## Materials and methods

### Sodium kinetic equation

The steps of the equation development have been described in previous publications [7, 8] and can be summarized as follows:

(1) Urea and sodium have similar dialyzer transfer characteristics as both are small solutes, are non-protein bound, and have comparable effective blood water flow. Subsequently, the principles of urea clearance can be applied to $Na^+$ dialysance.

(2) The sodium concentration adjustment ratio (NaAR) is the reduction fraction of the difference between predialysis serum $Na^+$ and fresh dialysate $Na^+$. It is equivalent to the urea reduction ratio (URR) and is calculated as follows:

$$\mathrm{NaAR} = \frac{\Delta \mathrm{Na}}{\nabla \mathrm{Na}} = \frac{\mathrm{Na}_{(t)} - \mathrm{Na}_{(0)}}{\mathrm{Na}_{(D)} - \mathrm{Na}_{(0)}} \quad (1)$$

where ΔNa is the difference between end-dialysis [$Na_{(t)}$] and predialysis [$Na_{(0)}$] serum $Na^+$, ∇Na is the difference between fresh dialysate [$Na_{(D)}$] and predialysis [$Na_{(0)}$] serum $Na^+$.

(3) The NaAR is also a function of sodium dialysance, dialysis duration, and sodium distribution volume. Similar to URR, it is calculated as follows:

$$\mathrm{NaAR} = 1 - \mathrm{e}^{-\frac{D_{\mathrm{Na}} \cdot t}{V}} \quad (2)$$

where $D_{Na}$ is the sodium dialysance, which is the volume of blood water that equilibrates with the dialysate $Na^+$ per unit time and is equivalent to urea clearance ($K_{urea}$), $V$ is the sodium distribution volume and is equivalent to TBW, and t is the session duration.

(4) Combining Eqs. 1 and 2 yields

$$1 - \mathrm{e}^{-\frac{D_{\mathrm{Na}} \cdot t}{V}} = \frac{\mathrm{Na}_{(t)} - \mathrm{Na}_{(0)}}{\mathrm{Na}_{(D)} - \mathrm{Na}_{(0)}} \quad (3)$$

(5) By rearrangement of Eq. 3, the $D_{Na}.t/V$ that is required to achieve a desired end-dialysis serum $Na^+$ can be calculated as follows:

$$\frac{D_{\mathrm{Na}} \cdot t}{V} = -\ln\left(-\frac{\mathrm{Na}_{(t)} - \mathrm{Na}_{(0)}}{\mathrm{Na}_{(D)} - \mathrm{Na}_{(0)}} + 1\right) \quad (4)$$

where ln is the natural log.

(6) For mathematical simplicity:

i. The measured serum $Na^+$ was not corrected for plasma water volume or the Donnan effect (the correction factor for these effects cancels each other out).

ii. The sodium kinetic equation assumed that the $Na^+$ is distributed in a single pool throughout the body and did not account for changes in TBW, i.e., single-pool fixed-volume. We considered a fixed sodium distribution volume as we did not anticipate significant fluid loss through ultrafiltration due to the short dialysis session or fluid gain as fluids were not administered during HD.

### Monitoring of $D_{Na}.t/V$ during the dialysis session

(1) The dialysate and plasma water $Na^+$ concentration and conductivity are strongly correlated, and effective sodium dialysance can be considered equivalent to conductivity (ionic) dialysance [9]. Replacing sodium dialysance with ionic dialysance ($D_{cn}$) in Eq. 4 yields

$$\frac{D_{\mathrm{cn}} \cdot t}{V} = -\ln\left(-\frac{\mathrm{Na}_{(t)} - \mathrm{Na}_{(0)}}{\mathrm{Na}_{(D)} - \mathrm{Na}_{(0)}} + 1\right) \quad (5)$$

(2) Ionic dialysance can be measured periodically in real time during HD without blood sampling by OCM software embedded in dialysis machines. By this technique, the dialysate conductivity at the dialyzer inlet is briefly altered from baseline, which leads to a change of the dialysate conductivity at the outlet. Ionic dialysance is then calculated using conductivity values at the inlet and outlet at two differ-

ent points [10]. The value of $D_{cn}.t/V$ is then determined using the mean of conductivity dialysance ($D_{cn}$), session length ($t$), and TBW ($V$) and is displayed on the dialysis machine screen.

### Patients

Twelve patients were prospectively enrolled in this study. Inclusion criteria were adults aged ≥ 18 years with severe hyponatremia and kidney failure who required RRT. The cause of kidney failure was either acute or chronic kidney disease, and severe hyponatremia was defined as serum $Na^+$ < 120 mEq/L. Exclusion criteria were serum $Na^+$ ≤ 105 mEq/L; alcoholism; advanced chronic liver disease; serum potassium levels < 3.5 mEq/L or ≥ 6.5 mEq/L; fingerstick capillary blood glucose in diabetic patients prior to HD session > 200 mg/dl; uremic encephalopathy; uremic pericarditis; clinical hypovolemia or hypervolemia with unknown dry weight; and symptoms of severe hyponatremia, e.g., coma or seizure.

### Methods

One hour before the start of HD, a blood sample was sent for predialysis serum $Na^+$ measurement. $D_{Na}.t/V$ that achieves the desired end-dialysis serum $Na^+$ is calculated by Eq. 4. The dialysate $Na^+$ was 130 mEq/L, and the desired change in serum $Na^+$ was 4 mEq/L. The latter value was chosen because the safe rate of hyponatremia correction in a brief HD session is unknown and because there may be a further increase in serum $Na^+$ after the session, so that the overall daily change does not exceed the recommended value of 8 mEq/L.

TBW was calculated according to Watson's equation using the dry weight for all patients, and edema fluid was then added: for men: $2.447 - 0.09156 \times$ (age, yr) $+ 0.1074 \times$ (height, cm) $+ 0.3362 \times$ (weight, kg) and for women: $-2.097 + 0.1069 \times$ (height, cm) $+ 0.2466 \times$ (weight, kg).

Conventional IHD was delivered using a Fresenius 4008 s machine equipped with OCM, a low-flux polysulfone dialyzer with a surface area of 1.3 $m^2$ (Hemoflow F6HPS, Fresenius Medical Care), and bicarbonate-buffered dialysate. A central venous catheter was used as a vascular access. The blood flow rate was set to 200 ml/min, and the dialysate flow rate was set to 300 ml/min. The dialysate $Na^+$ concentration was set at 130 mEq/L, and the dialysate temperature was set at 37.0 °C. Anticoagulation was achieved by administering unfractionated heparin in a bolus dose of 1000 IU at the beginning of the session and then a continuous infusion at a rate of 500 IU hourly. The dialysis time was initially set at two hours, and the ultrafiltration goal for patients with edema was individualized.

The OCM software was activated at the start of the HD. Target $D_{Na}.t/V$ and TBW were entered, and the machine was set to measure ionic dialysance every 25 min. The dialysis nurse was instructed to closely monitor $D_{cn}.t/V$ and to terminate the session once it reached the target value, even if it was achieved before the 2 h session duration.

For patients who had received more than one HD session and whose serum $Na^+$ remained < 120 mEq/L before each session, the HD prescription was the same as the first, and TBW was recalculated before each session if the change in body weight was greater than one kg. When the end-dialysis serum $Na^+$ level reached 120 mEq/L, patients were released from the study protocol. Dialysis was then prescribed, if required, in accordance with routine center protocol, with the exception of setting dialysate $Na^+$ at 130 mEq/L.

At the end of the HD session, the blood flow rate was reduced to 50 ml/min for 10 s, the ultrafiltration was switched off, and a blood sample was taken for measurement of end-dialysis serum $Na^+$. Pre- and end-dialysis serum $Na^+$ levels were measured by the indirect ion-selective electrode (ISE) method. Intravenous fluids and eating were not allowed during HD.

### Statistical analysis

Statistical analysis was done using SPSS 26.0 software. Numerical data were presented as mean ± standard deviation (SD). Categorical data were presented as numbers and percentages. Agreement between observed and predicted end-dialysis serum $Na^+$ was assessed by a Bland–Altman plot.

## Results

Twenty HD sessions were delivered to 12 patients. Before serum $Na^+$ reached 120 mEq/L, five patients received one HD session, six patients received two sessions, and one patient received three sessions. The mean age of the patients was 55.45 ± 11.91 years, and seven of them were men. Primary renal disease was chronic kidney disease stage 5 (CKD5) in seven patients who were maintained on conservative treatment before dialysis, while the remaining had acute kidney disease (AKD). Causes of CKD were diabetic kidney disease ($n = 2$), hypertensive nephrosclerosis ($n = 2$), ADPKD in ($n = 1$), amyloidosis ($n = 1$) and chronic glomerulopathy ($n = 1$), and unknown ($n = 1$). Causes of AKD were NSAID-induced AIN ($n = 1$), ATN ($n = 1$), lupus nephritis ($n = 1$), and obstructive uropathy ($n = 1$). Four patients were clinically euvolemic, while the remaining eight patients were hypervolemic, with an estimated edema fluid of 2–4 kg. The mean estimated TBW was 37.23 ± 3.49 L. The patients' characteristics are summarized in Table 1.

**Table 1** Patients' characteristics ($n = 12$)

| Parameter | Mean ± SD | Range |
|---|---|---|
| No of dialysis sessions | 20 | |
| Age, years | 54.58 ± 11.75 | 36–74 |
| *Sex, n (%)* | | |
| Males | 7 (58.33) | |
| Females | 5(41.66) | |
| *Primary renal disease, n (%)* | | |
| AKD | 4 (33.33) | |
| CKD | 8 (66.66) | |
| Weight, kg | 67.53 ± 7.24 | 55–83 |
| Height, cm | 170.33 ± 6.31 | 159–181 |
| Body water volume by Watson's equation, L | 35.82 ± 4.00 | 29–42 |
| Edema fluid, L | 2.44 ± 0.77 | 2–4 |
| Total body water, L | 37.23 ± 3.49 | 32–43 |
| BUN, mg/dl | 84.57 ± 25.85 | 59–131 |
| Creatinine, mg/dl | 9.41 ± 2.05 | 6–12 |
| Predialysis $Na^+$, mEq/L | 115.45 ± 3.12 | 109–119 |
| Predicted $Na^+$, mEq/L | 119.45 ± 3.12 | 113–123 |
| Observed $Na^+$, mEq/L | 119.80 ± 3.42 | 114–125 |
| Target $D_{Na}.t/V$ | 0.34 ± 0.07 | 0.21–0.45 |
| Time to target $D_{Na}.t/V$, min | 86.46 ± 22.82 | 57–132 |
| Observed $Na^+$—predialysis $Na^+$, mEq/L | 4.35 ± 1.26 | 2–6 |
| Observed $Na^+$—predicted $Na^+$, mEq/L | 0.33 ± 1.26 | −2 to 2 |

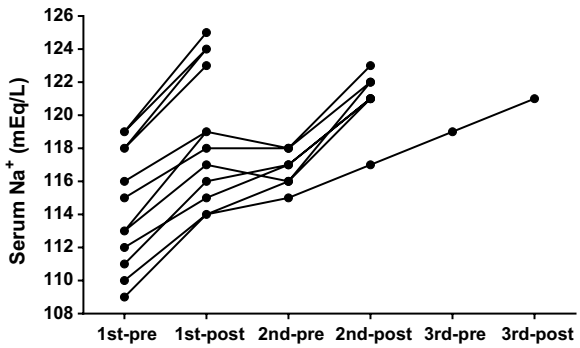


**Fig. 1** Changes in serum $Na^+$ during HD

### Intradialytic change of serum $Na^+$

The mean predialysis serum $Na^+$ and predicted end-dialysis serum $Na^+$ were 115.45 ± 3.12 mEq/L and 119.45 ± 3.12 mEq/L, respectively. The $D_{Na}.t/V$ target for achieving the predicted serum $Na^+$ was 0.34 ± 0.07. This target was reached after a mean HD session duration of 86.46 ± 22.82 min. In two dialysis sessions, the time was extended by 10 min after the initially prescribed 2 h to reach the target $D_{Na}.t/V$. The mean observed serum $Na^+$ was 119.80 ± 3.42 mEq/L. The changes in serum $Na^+$ during HD are shown in Fig. 1.

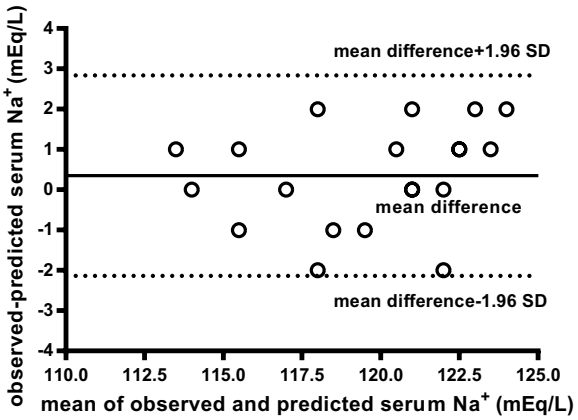


**Fig. 2** Bland–Altman plot. The *x*-axis represents the mean of the observed and predicted serum $Na^+$, and the *y*-axis represents the difference between them. The solid line represents the mean difference (bias) between the two values, and the dashed lines represent 95% limits of agreement. Note that, the values of four HD sessions are superimposed at 2 points

### Equation validation

A scatter diagram of the observed and predicted serum $Na^+$ is shown in Fig. 2. Data are presented using a Bland–Altman plot. The mean value of the observed and predicted serum $Na^+$ was plotted on the *x*-axis, and the difference between them was plotted on the *y*-axis. The bias (mean difference) ± SD was 0.35 ± 1.26 mEq/L, and the 95% confidence interval (limits of agreement) was −2.13 to 2.83 mEq/L. The imprecision of the equation in the prediction of end-dialysis serum $Na^+$ was obtained by doubling the SD, which gives a value of 2.52 mEq/L. The predicted serum $Na^+$ was identical to the observed values in five sessions, higher in 10 sessions, and lower in five sessions.

## Discussion

This study was conducted to assess the validity of prediction of end-dialysis serum $Na^+$ levels during conventional IHD in severely hyponatremic kidney failure patients using a sodium kinetic equation with substitution of sodium dialysance with ionic dialysance measured by OCM software embedded in dialysis machines. In the 20 HD sessions, the predicted serum $Na^+$ levels were within 2 mEq/L of the observed values. The sodium kinetic equation can be considered clinically quite effective, as the mean difference between observed and predicted serum $Na^+$ was small, and the 95% limits of agreement were narrow and clinically insignificant.

In patients with kidney failure and severe hyponatremia, renal replacement therapy may result in rapid correction of hyponatremia, which may be complicated by ODS. If those patients are treated by CRRT, lowering

dialysate and replacement fluid $Na^+$ to safe levels by replacing or adding sterile water to fluid bags may be an option to avoid rapid correction [6, 11, 12]. CRRT is not widely available, especially in developing countries, mainly due to high costs, and patients with kidney failure and severe hyponatremia in these countries are usually treated with IHD.

The lowest possible dialysate $Na^+$ concentration during conventional IHD is 130 mEq/L, and if patients were treated with this dialysis modality, rapid correction of severe hyponatremia may ensue. Prescribing a low-efficiency HD by using a small dialyzer surface area, low blood and dialysate flow rates, the lowest dialysate $Na^+$ available, a short session time, and the infusion of dextrose 5% in the venous limb of the dialysis circuit may reduce this risk, but the change in serum $Na^+$ is still unpredictable, and frequent measurement of serum $Na^+$ throughout the session is needed [13]. Wendland and Kaplan [14] described a method for prediction of end-dialysis serum $Na^+$ in conventional IHD without a sodium kinetic model. The authors achieved a desired change in serum $Na^+$ ($\Delta Na$) of 6 mEq/L in a patient with a TBW of 25 l, who had severe hyponatremia (serum $Na^+$ of 113 mEq/L) and AKI using a particular dialysis prescription. They used the dialysis machine in pediatric mode, setting the blood flow to 50 ml/ min, the dialysate flow to 800 ml/ min, and the dialysate $Na^+$ to 130 mEq/L, which produced a diffusive sodium gradient ($\nabla Na$) of 17 mEq/L. The required sodium mass that achieved the desired $\Delta Na$ was 150 mEq (6 mEq × 25 kg). Complete equalization of the blood with dialysate $Na^+$ occurred as a result of the low prescribed blood flow, and a cumulative blood flow of roughly 9 L (150/17) was required to transfer the estimated sodium mass. The predicted session length was 180 min (9000/50).

Yessayan et al. [6] introduced the sodium kinetic equation investigated in our study and successfully used it to predict end-dialysis serum $Na^+$ during predilution CVVHF for severely hyponatremic (serum $Na^+$ 98 mEq/L) kidney failure patient. They estimated the replacement fluid (RF) $Na^+$ (a substitute for dialysate $Na^+$ in Eq. 4) that was required to achieve the desired increase in serum $Na^+$ every 24 h. The estimated RF $Na^+$ was then prepared by exchanging sterile water for an equal volume of the standard RF bags. The results demonstrated that the predicted serum $Na^+$ for each 24-h period was approximately equal to the measured value. Other investigators used the equation in correction of severe hypernatremia (serum $Na^+$ 177 mEq/L) in a patient with AKI using postdilution CVVH. They estimated the RF $Na^+$ that was required to obtain a desirable decline in serum $Na^+$ and reported a good agreement between the predicted and observed end-dialysis serum $Na^+$ levels [15]. Hamdi et al. [7] induced hypernatremia during online CVVHD using a Fresenius 2008 K/K2 machine equipped with OCM to treat cerebral edema in patients with acute liver failure and AKI. The authors used the dialysis machine in pediatric mode and prescribed a blood flow of 6o ml/ min and a dialysate flow of 400 ml/ min. The sodium dialysance (about 3 L/h) was equal to the blood water volume due to complete equalization of the blood with dialysate $Na^+$. The sodium kinetic equation was used to model the time required to reach the target serum $Na^+$. They reported estimated serum $Na^+$ levels within 2 mEq/L of the target values.

This study is the first work (to the best of the author's knowledge) that assessed the validity of the tested sodium kinetic equation in the prediction of end-dialysis serum $Na^+$ in severely hyponatremic kidney failure patients undergoing conventional IHD rather than CRRT. The results showed an imprecision of 2.64 mEq/L. Complex sodium kinetic models were developed many years ago and validated by determining the dialysate $Na^+$ that was needed to reach a predetermined end-dialysis serum $Na^+$ in chronic HD patients and then calculating the difference between the predicted and observed serum $Na^+$. These kinetic models are used to achieve a neutral sodium balance in MHD, but their validity in severe hyponatremia correction during conventional IHD was not assessed. The first sodium kinetic model was developed by Gotch et al. [16], who measured dialysate and plasma water $Na^+$ using flame photometry, and the imprecision of this model was ± 2.9 mEq/L. Di Filippo et al. [17] modified the original sodium kinetic model and reassessed its validity by measuring the dialysate and plasma water $Na^+$ using direct potentiometry instead of flame photometry, reporting a decrease in model imprecision to less than 0.84 mEq/L. Both of these kinetic models were not clinically applicable as they required the measurement of initial plasma $Na^+$ and sodium dialysance in real time, which was difficult to obtain in a routine dialysis. Petitclerc et al. [18] converted the sodium kinetic model to the conductivity kinetic model by replacing $Na^+$ levels with conductivity values and sodium dialysance with conductivity dialysance. Locatelli et al. [19] assessed the validity of the conductivity kinetic model, and the results demonstrated an imprecision of less than 0.14 mS/cm, which is equivalent to a $Na^+$ imprecision of less than 1.4 mEq/L. Despite the higher imprecision of the kinetic equation in our study compared to the latter two models, it can be considered clinically satisfactory as the 95% limits of agreement are clinically not important.

No neurological complications were reported in the study population as a result of hyponatremia correction. This was not unexpected due to several factors: First, the daily change in serum $Na^+$ did not exceed the

recommended value in any patient. Second, patients with a high risk of ODS were excluded. Third, elevated urea levels in patients with kidney failure have a protective effect against ODS [4].

It is important to emphasize that (1) The dialysis dose in our study is small to guarantee slow correction of severe hyponatremia, so the described methodology may not be suitable for kidney failure patients who need high-efficiency dialysis. Delivering a small dialysis dose may be considered better than deferring the dialysis initiation till correction of severe hyponatremia with hypertonic saline, which may be complicated by inducing/exacerbating hypervolemia in oliguric kidney failure. (2) Although we unified the dialysis prescription for all patients, using different blood and dialysate flow rates, or dialyzer surface areas is expected to give similar results as long as the session is terminated at the predetermined $D_{Na}.t/V$.

The discrepancy between observed and predicted end-dialysis serum $Na^+$ in 15 HD sessions is not necessarily due to an equation error; it could alternatively be the result of the isolated or combined effects of the following factors:

(1) Inaccuracy in measurement of the prescribed dialysate $Na^+$: The prescribed dialysate $Na^+$ is derived from dialysate conductivity measurement. Previous studies found that the bias between the prescribed dialysate $Na^+$ derived from conductivity measurement and measured dialysate $Na^+$ by indirect ISE analyzer is small and irrelevant when the analyzer is set to the urine mode rather than the plasma mode (mean difference: 1.5 mEq/L, 95% CI 0.6–2.9) [20].
(2) Inaccuracy in estimation of the diffusible plasma $Na^+$: The diffusible $Na^+$ is the plasma water $Na^+$ adjusted for the Donnan effect. We did not correct plasma $Na^+$ for plasma water fraction or the Donnan effect, given that each of these correction factors cancels out the other and we considered the diffusible plasma $Na^+$ equal to the plasma $Na^+$.
(3) Some of the transferred sodium mass might be stored in an osmotically inactive form in connective tissue, cartilage, or bone [21].
(4) Inaccuracy in TBW estimation by Watson's equation: Earlier studies revealed that Watson's equation may overestimate TBW in dialysis patients [22, 23]. On the other hand, Pozzoni et al. [5] did not report a clinically significant difference between the inaccuracy and imprecision of prediction of end-dialysis plasma $Na^+$ using sodium and conductivity kinetic models when TBW was estimated by Watson's equation compared with the direct dialysate quantification method.
(5) Random error in serum $Na^+$ measurement by indirect ISE.
(6) Inaccuracy in ionic dialysance measurement.

This study is subject to some limitations. First, we did not measure dialysate $Na^+$ and considered it equal to the prescribed dialysate $Na^+$ derived by conductivity measurement. Second, Watson's equation was used to estimate TBW, and this method, which is dependent on anthropometric measures, may be inaccurate and overestimate TBW compared to the more accurate methods such as bioimpedance spectroscopy. Despite this disadvantage, it is simple and can be applied easily in practice. Third, the study was conducted on a small sample size, and it may be claimed that this caused unreliable estimates of the 95% limits of agreement. Fourth, the duration of HD sessions was short, so it is unknown whether the application of this sodium kinetic equation to longer HD sessions will give similar results or not. The latter hypothesis is risky to be investigated, as the longer HD sessions will increase $D_{Na}.t/V$ with a risk of rapid correction of hyponatremia, except if dextrose 5% is infused concomitantly, which will change the diffusive sodium gradient and TBW, and thus may affect the prediction power of the sodium kinetic equation. Fifth, serum $Na^+$ was measured by indirect ISE, which is affected by plasma levels of proteins and lipids like flame photometry, and we might obtain different imprecision values if direct ISE was used instead, as observed by Di Filippo et al. [17], who reported decreasing the imprecision value of the original kinetic model by Gotch et al. [16] when plasma $Na^+$ was measured using direct ISE instead of flame photometry.

## Conclusion

This study revealed that application of the tested sodium kinetic equation using ionic dialysance measured by OCM software embedded in dialysis machines as a substitute for sodium dialysance is clinically effective in the prediction of end-dialysis serum $Na^+$ in severely hyponatremic kidney failure patients undergoing conventional HD.

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**Author contributions**
The author designed the research, collected the data, interpreted the results and wrote the manuscript. The author read and approved the final manuscript.

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**Availability of data and materials**
The datasets of the study are not publicly available as it is private date, but are available from the corresponding author on reasonable request.

## Declarations

### Ethics approval and consent to participate

The study was approved by Ain Shams University Faculty of Medicine Research Ethics Committee with registration number FMASU R 163 2022. Written consent was obtained from patients involved in the study.

### Consent for publication

Not applicable.

### Competing interests

The author declares no competing interests.

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