Clinical Kidney Journal, 2016, vol. 9, no. 4, 540–542

doi: 10.1093/ckj/sfw036
Advance Access Publication Date: 26 May 2016
Exceptional Case

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Continuous venovenous hemofiltration using customized replacement fluid for acute kidney injury with severe hypernatremia

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Abstract

The initiation of continuous renal replacement therapy (CRRT) in acute kidney injury (AKI) with severe hypernatremia is challenging since sodium concentrations in commercial replacement fluid (RF) and dialysate solutions are usually fixed at 140 mEq/L. We present a case of AKI with severe hypernatremia successfully treated with CRRT using commercial RF solutions customized to prevent rapid correction of hypernatremia. None of the few case reports published on hypernatremia and AKI requiring CRRT have included formulas to help modulate the sodium content in the solutions. We present an equation to facilitate adjustment of the sodium concentration in this setting.

Key words: acute kidney injury, acute renal failure, continuous renal replacement therapy, dialysis, hypernatremia

Background

Hypernatremia is common in critically ill patients, with a prevalence as high as 26% [1]. Whether hypernatremia represents a marker of disease severity or an independent prognostic factor remains a subject of controversy [2]. Patients with hypernatremia have a higher risk of mortality and longer lengths of stay compared with critically ill patients with normal sodium concentrations [1, 2]. When treating a patient with severe hypernatremia and acute kidney injury (AKI) requiring dialysis, clinicians face a therapeutic challenge, as the sodium content in replacement fluid (RF) and/or dialysate solutions is not easily adjustable. Therefore, they must be aware of the potential risk for a rapid decline in serum sodium concentrations if no modifications are made to these solutions. We present a case of severe hypernatremia and AKI successfully treated with continuous venovenous hemofiltration (CVVH), along with an equation to help calculate the required adjustments in sodium concentrations in the RF and/or dialysate.

Case report

A 53-year-old 56-kg man was brought to our emergency department for a decreased level of consciousness for 3 days. His past medical history was relevant for hypertension and bipolar disorder. His medications included hydrochlorothiazide 25 mg daily, triamterene 50 mg daily and lithium 300 mg thrice daily.

On arrival, he had decreased responsiveness and his Glasgow coma scale (GCS) was 9. His blood pressure was 121/95 mmHg, heart rate was 124 beats/min, respiration rate was 30 breaths/min, pulse oximetry was 98% on room air and temperature was 36.4°C.

Received: January 11, 2016. **Accepted:** April 11, 2016

Table 1. Target sodium concentrations in RF solution with required volume of 23% NaCl and 3% NaCl to add to the RF solution

Target final sodium concentration in RF	170 mEq/L	165 mEq/L	160 mEq/L	155 mEq/L	150 mEq/L	145 mEq/L
Volume of 23.4% NaCl to add in a 5-L bag of RF[a]	38 mL (150 mEq)	31 mL (125 mEq)	25 mL (100 mEq)	19 mL (75 mEq)	13 mL (50 mEq)	6 mL (25 mEq)
Volume of 3% NaCl to add in a 5-L bag of RF[b]	294 mL (150 mEq)	245 mL (125 mEq)	196 mL (100 mEq)	147 mL (75 mEq)	98 mL (50 mEq)	49 mL (25 mEq)

[a]1 mL 23.4% NaCl = 234 mg/mL × 17 mEq/g = 3.98 mEq NaCl/mL.
[b]1 mL 3% NaCl = 50 mg/mL × 17 mEq/g = 0.51 mEq NaCl/mL.

He presented signs of profound dehydration. The rest of the physical exam was unremarkable. Initial laboratory findings showed the following: serum sodium concentration, 184 mEq/L; serum creatinine level, 755 µmol/L (8.5 mg/dL) with a baseline level of 107 µmol/L (1.2 mg/dL) 3 months before; and blood urea nitrogen (BUN) level, 57.1 mmol/L (160 mg/dL). Other laboratory results were as follows: chloride, 133 mEq/L; potassium, 4.0 mEq/L; bicarbonate, 14.1 mEq/L; lactate level, 5.5 mmol/L (0.6 mg/dL); and creatine kinase level, 1500 U/L. Other relevant findings included a negative toxic screen, normal osmolal gap and glucose levels and undetectable alcohol, aspirin, methanol, ethylene glycol and lithium concentrations. Arterial blood gas showed a pH of 7.26, partial pressure of CO_2 30 mmHg and HCO_3^- 13 mmol/L. Complete blood count showed increased white blood cells (32.8×10^9) and hemoglobin level [225 g/L (22.5 g/dL)]. The patient was nonoliguric (urine output 1.1 L/day) and his urinalysis was normal. His urine osmolality was 394 mOsm/L and urine sodium concentration was 38 mmol/L. Additional investigations included normal head computed tomography, abdominal ultrasound and chest X-ray. The AKI diagnosis was attributed to an acute tubular necrosis (ATN) caused by severe dehydration and sepsis. A partial nephrogenic diabetes insipidus caused by chronic lithium administration was also suspected. The patient was brought to the ICU, where he received intravenous antibiotics for a suspected gastrointestinal source.

The patient initially received 2 L of 0.9% NaCl followed by an infusion at 150 mL/h to correct hypovolemia. We then estimated the total body water deficit at 10.5 L and subsequently modified the fluid composition for a 5% dextrose water at 80 mL/h, aiming to decrease the sodium concentration level to a maximum rate of 10 mEq/L in 24 h. Given the small improvement in creatinine [719 µmol/L (8.1 mg/dL)], BUN [60.8 mmol/L (170 mg/dL)] and sodium concentration (177 mEq/L) after 24 h, and in the absence of improvement in the patient clinical status, we initiated CVVH (Aquarius, Baxter, Mississauga, ON, Canada) to treat severe AKI with suspected uremic encephalopathy and to lower sodium safely. At our institution, commercially available RF solutions are used and custom-made RF solutions are not routinely available. The dialysis prescription was as follows: hemofilter Aquamax HF19 (Baxter), with a blood flow rate of 250 mL/min, and an initial RF rate of 20 mL/kg/h with a modified hypertonic RF solution, adding 23% NaCl to Prismasol 4 mEq/L solution (Baxter) (Table 1). The initial corrections in serum sodium levels were too conservative, so we modified sodium levels in the RF. We also increased the RF flow rate to 25 mL/kg/h on Day 3. The patient received CVVH for 72 h without ultrafiltration. His sodium levels decreased as expected (Figure 1), with gradual improvement in his level of consciousness. The patient was discharged from hospital 2 weeks later with a creatinine of 134 mmol/L (1.5 mg/dL).

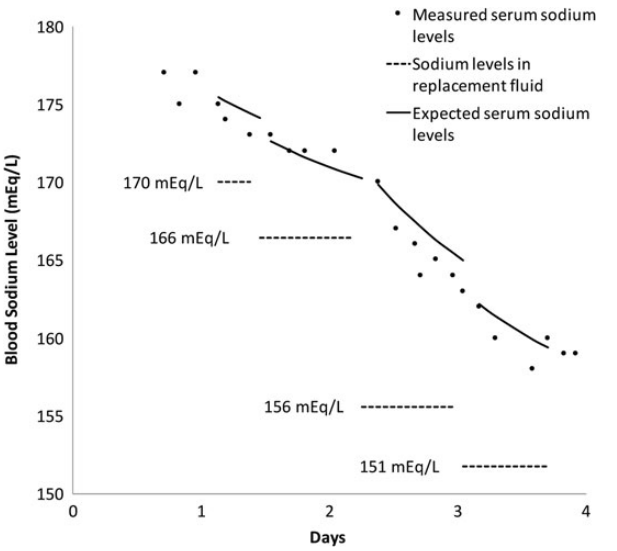

Fig. 1. Serum sodium concentrations before and during CVVH with customized RF solutions. The expected decline in serum sodium concentrations was calculated with the equation provided in the text.

Discussion

Chronic hypernatremia is associated with an accumulation of organic osmolytes in brain cells, which constitutes a cerebral adaptation to this hypertonic environment. Therefore, when treating chronic hypernatremia, a rapid correction of the total body water deficit can lead to cerebral edema. Current recommendations suggest a maximal decrease in sodium concentration of 0.5 mEq/L/h mainly based on results from pediatric patients [3].

Management of severe hypernatremia and AKI requiring dialysis represents a therapeutic challenge. One must choose the composition of the RF for the renal replacement therapy wisely to prevent a rapid decrease in serum sodium concentrations. Expected serum sodium changes during conventional intermittent hemodialysis (IHD), sustained efficiency dialysis (SLED) and CVVH are usually higher than the suggested decrease of 0.5 mEq/L/h [4]. In CVVH, the rate of electrolyte equilibration with the concentration in the RF is influenced by the initial difference between the serum and RF sodium concentration and the CVVH prescription itself [5]. A more rapid decrease in sodium levels is seen with higher RF rates and higher blood flow rates. Some kinetic models can help predict serum sodium changes during treatment [4]. Therefore, CVVH with customized RF solution can safely lower serum sodium concentration in a stepwise manner. There are only limited cases reported in the literature, and none of them have included equations to help adjust the sodium content in the RF and/or dialysate [6–8].

In our case, the customized RF solution was prepared by adding 23% NaCl to the initial RF solution (Na 140 mEq/L) following the data presented in Table 1. The addition of 3% NaCl instead of 23% NaCl would require larger volumes to be added in the RF solution, which would have affected the concentration of other electrolytes. We estimated the RF sodium concentration using the following kinetic equation [4]:

$$\text{Replacement fluid } [Na^+] = \frac{\text{desired serum } \Delta\,[Na^+]}{(1 - e^{(-Cl \times 24\text{ h})/V})} + \text{initial serum } [Na^+]$$

where Cl is the Na^+ filter clearance, V is the total body water volume and desired serum $\Delta[Na^+]$ represents a negative value when treating hypernatremia and a positive value when treating hyponatremia.

The clearance depends on the predilution or postdilution mode:

$$Cl\,\text{predilution} = \left(\frac{Q_b}{Q_b + Q_{rf}}\right) \times SC_{Na^+} \times (Q_{rf} + Q_{uf})$$

$$Cl\,\text{postdilution} = SC_{Na^+} \times (Q_{rf} + Q_{uf})$$

where Q_b is the blood flow rate (L/h), Q_{rf} is the RF flow rate (L/h), Q_{uf} is the ultrafiltration rate (L/h) and SC_{Na} is the Na^+ sieving coefficient (~1).

For example, if the initial blood sodium concentration is 177 mEq/L and the target blood sodium concentration is 170 mEq/L, the desired correction rate would be −7 mEq/L in 24 h. V is the total body water volume, which equals 0.5 × estimated dry weight for females and 0.6 × estimated dry weight for males; V was estimated as 33.5 L in this case, Q_b was 250 mL/min (15 L/h), Q_{rf} was 17 mL/min (1 L/h), Q_{uf} was 0 L/h, e was 2.72 (constant) and Cl was 0.94, as calculated with previous values. Therefore, the customized sodium concentration in the RF should be 163 mEq/L.

In conclusion, the treatment of AKI and severe hypernatremia using CVVH is facilitated by the use of customized commercially available RF solutions. The solutions should be prepared cautiously to prevent any sterility breach, and sodium levels can easily be estimated based on the sodium kinetic model presented in this report.

Acknowledgements

J.B. and S.T.'s research efforts are supported by the Fonds de la Recherche du Québec-Santé.

Conflict of interest statement

None declared. The results presented in this paper have not been published previously in whole or part, except in abstract format.

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