AJKD

Acid-Base and Electrolyte Teaching Case

Treatment of Severe Hyponatremia in Patients With Kidney Failure: Role of Continuous Venovenous Hemofiltration With Low-Sodium Replacement Fluid

Lenar Yessayan, MD, Jerry Yee, MD, Stan Frinak, MSEE, and Balazs Szamosfalvi, MD

Patients with hypervolemic hyponatremia and kidney failure pose a special therapeutic challenge. Hemodialysis to correct volume overload, azotemia, and abnormal electrolyte levels will result in rapid correction of serum sodium concentration and place the patient at risk for osmotic demyelination syndrome. We present a patient with acute kidney injury and severe hypervolemic hypotonic hyponatremia (serum sodium < 100 mEq/L) who was treated successfully with continuous venovenous hemofiltration. This teaching case illustrates the limitations of hemodialysis and demonstrates how to regulate the sodium correction rate by single-pool sodium kinetic modeling during continuous venovenous hemofiltration. Two methods to adjust the replacement fluid to achieve the desired sodium concentration are outlined.
Am J Kidney Dis. ■(■):■-■. © *2014 by the National Kidney Foundation, Inc.*

INDEX WORDS: Hyponatremia; treatment; continuous venovenous hemofiltration (CVVH); hemodialysis.

INTRODUCTION

In patients with chronic severe symptomatic hyponatremia and concomitant kidney failure, hypertonic saline should be used until resolution of the symptoms. Thereafter, the goal should be gradual correction of serum sodium level, by not more than 8-10 mEq/L over 24 hours.[1] In those who require hemodialysis, rapid correction of serum sodium concentration [Na^+] could ensue and put the patient at risk for osmotic demyelination syndrome.[2,3] Management using continuous renal replacement therapy also is challenging due to the lack of commercially available hypotonic dialysate or replacement fluids. This teaching case presents a patient with acute kidney injury and severe hyponatremia (serum [Na^+] < 100 mEq/L) treated with continuous venovenous hemofiltration (CVVH) in which gradual correction of serum [Na^+] was achieved using hypotonic replacement fluid composed of successively higher [Na^+].

CASE REPORT

Clinical History and Initial Laboratory Data

A 54-year-old woman presented to the emergency department with confusion, decreased responsiveness, and generalized weakness for 24 hours. She had nausea and decreased oral intake for a few weeks, with reduced urine output for a few days. Her medical history was significant for uncontrolled hypertension and alcohol abuse. Blood pressure was 246/115 mm Hg, heart rate was 106 beats/min, respiration rate was 21 breaths/min, and pulse oximetry was 94% on room air.

The patient's physical examination revealed bilateral basilar rales. Serum laboratory studies showed the following values: sodium, 96 mEq/L; potassium, 5.6 mEq/L; chloride, 64 mEq/L; bicarbonate, 16 mEq/L; serum urea nitrogen, 51 mg/dL; creatinine, 9.9 mg/dL; phosphorus, 8.1 mg/dL; serum osmolality, 226 mOsm/kg; and brain natriuretic peptide, 2,800 pg/mL. Arterial blood gas analysis showed pH 7.36, $Paco_2$ of 27.9 mm Hg, Pao_2 of 57.5 mm Hg, and bicarbonate level of 15.4 mEq/L. Urine sediment showed few granular casts. Urine osmolality was 157 mOsm/kg, sodium was 40 mEq/L, and creatinine was 33 mg/dL, with fractional sodium excretion of 12.5%.

Additional Investigations

Computed tomography of the brain demonstrated no intracranial pathology. The radiograph of the chest showed mild to moderate cardiomegaly with pulmonary vascular congestion.

Diagnosis

Acute kidney injury and severe hypervolemic hypotonic hyponatremia was diagnosed. Acute tubular necrosis was the presumptive cause of the acute kidney injury.

Clinical Follow-up

Treatment goals were identified to include gradual correction of hyponatremia, alleviation of congestive heart failure, and management of hyperkalemia, azotemia, and hyperphosphatemia. The patient began CVVH using a modified hypotonic replacement fluid. Standard fluid bags (NxStage PureFlow dialysate solution RFP 401; NxStage Medical Inc) were diluted by the addition of pure water to 112 mEq/L at the start of CVVH, 120 mEq/L 24 hours later, and 128 mEq/L 56 hours after initiating CVVH. CVVH was performed for a total of 72 hours at a rate of correction recommended by therapeutic guidelines[4] (Fig 1). Mentation improved without neurologic sequelae. On hospital day 2, an ultrasonogram showed bilateral hydronephrosis, which was treated by bilateral nephrostomy tube insertions on day 4 that produced complete recovery of kidney function. The patient was essentially anuric (urine < 100 mL/d) until bilateral nephrostomy placement.

From the Division of Nephrology and Hypertension, Henry Ford Hospital, Detroit, MI.

Received September 12, 2013. Accepted in revised form January 13, 2014.

Address correspondence to Lenar Yessayan, MD, Division of Nephrology and Hypertension, Henry Ford Hospital, 2799 West Grand Blvd, Detroit, MI 48202. E-mail: lyessay1@hfhs.org

0272-6386/$36.00
http://dx.doi.org/10.1053/j.ajkd.2014.01.451

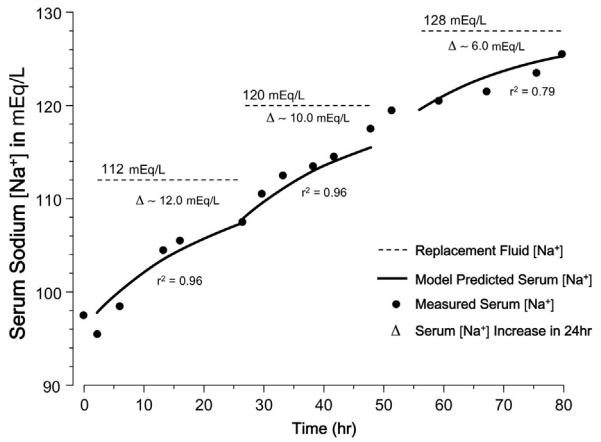

Figure 1. Serum sodium concentrations at baseline and during continuous venovenous hemofiltration (CVVH) while using successively higher replacement fluid concentrations. The r^2 denotes the coefficient of determination between predicted and measured serum sodium levels for each 24-hour CVVH period.

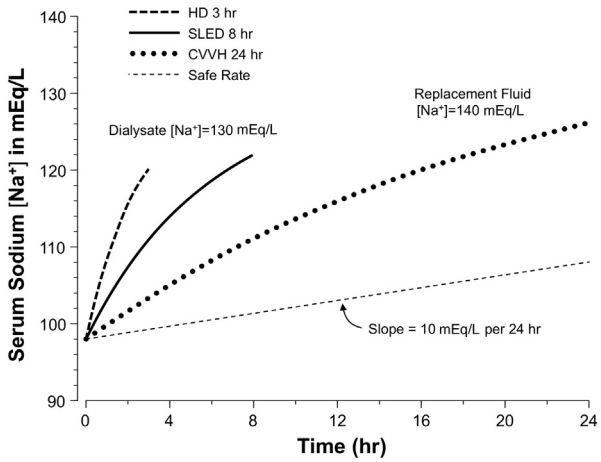

Figure 2. The expected serum sodium change during hemodialysis (HD), sustained low-efficiency dialysis (SLED), and continuous venovenous hemofiltration (CVVH) with noncustomized fluid bags. For HD, treatment time is 3 hours, dialysate sodium is 130 mEq/L, blood flow rate is 200 mL/min, and approximate delivered urea clearance is 180 mL/min (10.8 L/h). For SLED, treatment time is 8 hours, dialysate sodium is 130 mEq/L, dialysate flow is 100 mL/min, blood flow rate is 200 mL/min, and approximate delivered urea clearance is 78 mL/min (4.7 L/h). For CVVH, treatment time is 24 hours, replacement fluid sodium is 140 mEq/L, blood flow rate is 300 mL/min, replacement fluid is 1.2 L/h, and approximate delivered urea clearance is 1.2 L/h.

The final diagnosis was obstructive acute kidney injury due to cervical cancer.

DISCUSSION

Management of hypotonic hyponatremia should be based on its presumed duration and severity. In acute hyponatremia, free water movement into brain cells results in varying degrees of cerebral edema.[5] The risk of electrolyte disturbance exceeds that of osmotic demyelination syndrome secondary to rapid correction. Chronic hyponatremia is associated with brain cells adaptively losing organic osmolytes, and thus life-threatening cerebral edema is less likely.[6] This adaptation renders the brain vulnerable to osmotic demyelination syndrome in response to a rapid increase in serum [Na^+].[7-9] Therefore, management of hyponatremia entails balancing the risks of hyponatremia with those of rapid correction. For decades, a total increase of 12 mEq/L per day was considered safe[10,11]; however, osmotic demyelination syndrome has been reported with correction of only 10 mEq/L per day.[12,13] Consequently, even lower limits of correction have been recommended: 10 mEq/L in 24 hours and 18 mEq/L in 48 hours.[1]

Patients with severe hyponatremia and kidney failure with volume expansion pose a unique therapeutic challenge. Hypertonic saline is undesirable in patients with kidney failure and expanded volume status. Similarly, V_2 receptor antagonists are not likely to be effective at reduced glomerular filtration rates because of diminished fluid delivery to the distal nephron[14]; in the absence of published experience at reduced glomerular filtration rates, the use of these agents cannot be recommended.

Successful treatment of patients with kidney failure and hyponatremia using different extracorporeal modalities, including hemodialysis at low blood flow rates,[15] CVVH,[16] and continuous venovenous hemodialysis,[17,18] has been reported. However, all renal replacement modalities could be deleterious when used in severe cases of hyponatremia (Fig 2).

In patients with kidney failure and severe hyponatremia, hemodialysis would induce a rapid increase in serum Na^+ with the potential for osmotic demyelination syndrome.[2,19] The high blood flow rates (Q_b; $>$ 200 mL/min) and inability to reduce the dialysate bath's [Na^+] to $<$130 mEq/L are 2 reasons for the high Na^+ transfer when using conventional hemodialysis to treat hyponatremia. Theoretically, the dialysis machine's alarm system could be manipulated and calibrated to a lower [Na^+], at the expense of a treatment session with nonfunctional conductivity monitoring, posing an unwarranted and significant safety risk.

Complex Na^+ kinetic models were shown to be able to predict end-dialysis serum [Na^+] in previous clinical studies.[20] We present a simpler set of Na^+ kinetic equations that are more easily applicable in clinical practice and can be used to estimate serum [Na^+] at the end of treatment. By applying urea clearance principles to Na^+ dialysance (D), serum [Na^+] at the end of hemodialysis and CVVH treatments is predictable. The final serum [Na^+] is the sum of the initial serum [Na^+]