AJKD

Acid-Base and Electrolyte Teaching Case

Approach to the Hemodialysis Patient With an Abnormal Serum Bicarbonate Concentration

Panupong Lisawat, MD, and F. John Gennari, MD

We present a patient receiving hemodialysis with a persistently high serum bicarbonate concentration to illustrate the evaluation and management issues for patients with both high (>25 mEq/L) and low (<20 mEq/L) pretreatment values. Patients with high serum bicarbonate concentrations typically are malnourished and have low rates of endogenous acid production. Evaluation should begin with assessment of whether an acute and potentially reversible cause of metabolic alkalosis is present. If not, management should be directed at treating malnutrition. By contrast, patients with low predialysis serum bicarbonate concentrations, in the absence of an acute and reversible cause, may benefit from increasing the level by an adjustment in dialysate bicarbonate concentration. However, the level at which one should intervene and to what extent serum bicarbonate concentration should be increased are unresolved issues. Whether such an intervention will reduce mortality risk has not been determined.

Am J Kidney Dis. ■(■):■-■. © *2014 by the National Kidney Foundation, Inc.*

INDEX WORDS: Hemodialysis; serum bicarbonate concentration; mortality; alkalosis; acidosis; malnutrition.

Note from Feature Editor Jeffrey A. Kraut, MD: This article is part of a series of invited case discussions highlighting the diagnosis and treatment of acid-base and electrolyte disorders.

INTRODUCTION

In patients without kidney function, body buffers consumed by acid accumulation are restored by alkali administration during dialysis. The administered alkali (bicarbonate and acetate in conventional hemodialysis) abruptly increases serum bicarbonate concentration, and then the bicarbonate is slowly consumed again by endogenous acid production in the interval between treatments (Fig 1).[1] The amount of alkali administered during the treatment is dependent on the dialysate bicarbonate concentration and traditionally has been set to avoid marked postdialysis alkalemia and minimize predialysis acidemia. Most patients receiving hemodialysis now are dialyzed with a dialysate containing bicarbonate in a final concentration ≥ 35 mEq/L and have predialysis serum bicarbonate concentrations of 19-22 mEq/L after the long interval between treatments.[2-4] Concerns have been raised about patients with predialysis values < 22 mEq/L and >25 mEq/L because of increased morbidity and mortality.[2-8] We present a patient with a consistently high predialysis serum bicarbonate concentration and review the risks, causes, and management of hemodialysis patients at the extremes of alkalosis and acidosis.

CASE REPORT

Clinical History and Laboratory Data

A 65-year-old woman with congestive heart failure secondary to mitral valve disease developed progressive chronic kidney disease, reaching end-stage kidney disease over 3 years. The cause of her kidney disease was undefined, but presumed to be nephrosclerosis complicated by cardiorenal syndrome. She was started on hemodialysis therapy because of uremic symptoms and metabolic acidosis with a bicarbonate concentration of 16 mEq/L. With hemodialysis, her symptoms improved and metabolic abnormalities were partially controlled, but her predialysis serum bicarbonate concentration, measured as total carbon dioxide, rapidly increased to 30-33 mEq/L and remained there (Table 1). She had persistent hypoalbuminemia and thrombocytopenia, with the latter problem diagnosed by bone marrow biopsy to be due to malnutrition. She was dialyzed with a citrate dialysate for anticoagulation because of nosebleeds with heparin (Table 2). The dialysate contained a bicarbonate concentration of 37 mEq/L after dilution and reaction with citric acid and also featured a variable dialysate sodium concentration to attempt to ameliorate her persistent hypotension and cramps. Her target weight was 42 kg, and she typically gained 1 kg between treatments. The patient reported no nausea or vomiting. She described a good appetite, but a detailed dietary history revealed very low caloric intake. Physical examinations on dialysis showed her to be afebrile, with blood pressure typically ~75/45 mm Hg and respiration rate of 18 breaths/min. Oxygen saturation was 98% on room air. She had a tunneled right-sided internal jugular catheter for access. She was alert and oriented, but had notable muscle wasting. Lungs were clear, heart rate was paced after an ablative procedure, and abdomen was scaphoid. She had no edema.

Additional Investigations

Serum bicarbonate concentration measured postdialysis was 33 mEq/L. After 2 days without dialysis, the predialysis value was 30 mEq/L. Using an estimated bicarbonate space of distribution

From the University of Vermont College of Medicine, Burlington, VT.

Received August 27, 2013. Accepted in revised form December 24, 2013.

Address correspondence to F. John Gennari, MD, Division of Nephrology and Hypertension, Fletcher Allen Health Care, University of Vermont College of Medicine, 1 S Prospect St, Burlington, VT 05401. E-mail: fgennari@uvm.edu

0272-6386/$36.00

http://dx.doi.org/10.1053/j.ajkd.2013.12.017

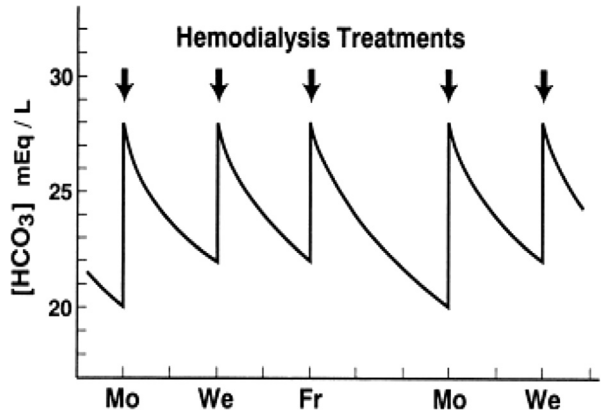

Figure 1. Schematic representation of the time course of serum bicarbonate concentration ([HCO_3^-]) over 9 days in a patient receiving conventional outpatient hemodialysis. The value increases abruptly with each treatment and then gradually decreases between treatments as it is consumed by endogenous acid production. Serum [HCO_3^-] is lower than normal only intermittently and decreases to its nadir once a week at the end of the longest interval between treatments (68-70 hours). Abbreviations: Mo, Monday; Fr, Friday; We, Wednesday. Reproduced from Gennari[21] with permission of Lippincott Williams & Wilkins.

of 0.5, the patient's weight (42 kg), and number of hours between these 2 treatments (69 hours), her calculated net acid production rate was 22 mEq/d, or 0.52 mEq/kg/d. The bicarbonate volume of distribution could have been <0.5 in our patient due to her high serum bicarbonate concentration and decreased muscle mass because muscle cells have an important role in buffering changes in serum bicarbonate concentrations. To the extent that the volume of distribution is smaller, her estimated net acid production would be even lower. Normalized protein catabolic rate, calculated from her urea reduction rate, was correspondingly very low at 0.65 g/kg/d.

Diagnosis

High predialysis serum bicarbonate concentration because of very low endogenous acid production, most likely due to malnutrition.

Clinical Follow-up

Despite our efforts to improve the patient's nutrition, she remained malnourished. Within 3 months, she developed sepsis, went on to comfort care, and died.

DISCUSSION

Dialysate bicarbonate concentration in conventional hemodialysis has not changed notably during the past 25 years, and a typical treatment produces a posttreatment serum bicarbonate concentration of 28-30 mEq/L.[5] After the long interval between treatments, serum bicarbonate concentrations gradually decrease to 19-22 mEq/L in most patients. Not surprisingly, there is wide variability in predialysis serum bicarbonate concentrations, and the level achieved is related inversely to protein catabolic rate (Fig 2).[2,5] A lingering mystery is why the postdialysis serum bicarbonate concentration is not higher. Not only is the usual dialysate bicarbonate concentration 35 mEq/L, but it also contains 4 mmol/L of acetate, which forms additional bicarbonate when metabolized in the body. The dialysance of bicarbonate is ~65% of blood flow rate, and this high value is reflected by a rapid initial increase in serum bicarbonate concentration in the first 1-2 hours of treatment.[1,9] However, in the latter part of the treatment, the value increases only slightly or levels off (and even decreases in rare cases). By the end of the treatment, serum bicarbonate concentration is about 4-7 mEq/L less than the dialysate bicarbonate concentration.[1] The most likely cause for the failure of serum bicarbonate concentration to increase further is stimulation of organic acid production by the rapidly added alkali, but this surge has been difficult to quantify.[1] Without such a surge, serum bicarbonate concentration should approximate the dialysate concentration given its high dialysance. With slow continuous low-efficiency dialysis, in fact, serum bicarbonate concentration essentially equals dialysate bicarbonate concentration within 12-18 hours. In our patient, little net bicarbonate was added during each dialysis session, and postdialysis serum bicarbonate concentration did not increase above 33 mEq/L. Given the patient's low blood pressure and cramps, it is

Table 1. Representative Laboratory Values

Measurement	Value
[K^+] (mEq/L)	4.3
[Cl^-] (mEq/L)	99
[HCO_3^-] (mEq/L)[a]	31
Calcium (mg/dL)	8.1
Calcium, corrected (mg/dL)[b]	9.9
Phosphorus (mg/dL)	5.8
Albumin (g/dL)	2.6
Hemoglobin (g/dL)	8.8
WBC count ($\times 10^3/\mu L$)	2.110
Pre/post SUN (mg/dL)	41/7
Urea reduction (%)	82.9
nPCR (g/kg/d)	0.65
PTH (pg/mL)	4.43
TSAT (%)	22
Ferritin (ng/mL)	703
Hematocrit (%)	26.7
Platelet count ($\times 10^3/\mu L$)	85

Note: Unless specifically noted, all values are from blood samples obtained predialysis after the longest interval between treatments. Serum sodium was not measured. Conversion factors for units: calcium in mg/dL to mmol/L, ×0.2495; phosphorus in mg/dL to mmol/L, ×0.3229; SUN in mg/dL to mmol/L, ×0.357.

Abbreviations: [Cl^-], chloride concentration; [HCO_3^-], bicarbonate concentration; [K^+], potassium concentration; nPCR, normalized protein catabolic rate; PTH, parathyroid hormone; SUN, serum urea nitrogen; TSAT, transferrin saturation; WBC, white blood cell.

[a]Measured as total carbon dioxide.

[b]$Calcium_{corrected}$ (mg/dL) = Calcium (mg/dL) + (4.4 − [albumin] g/dL).