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Severe Metabolic Alkalosis in Hemodialyzed Patients

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Abstract: Acid-base disorders are a major problem in the management of patients with end-stage renal disease. They mainly presented metabolic acidosis with an increased anion gap, however, other acid-base disorders are not excluded. Two hemodialyzed patients are presented who developed severe metabolic alkalosis, the first due to vomiting, which was occasionally exacerbated by respiratory alkalosis due to anxiety neurosis, and the second due to soda intake (prescription given ten years ago when she was in the pre-end stage of chronic kidney failure).

Keywords: Metabolic Alkalosis, Respiratory Alkalosis, Conventional Hemodialysis, Hemodiafiltration, Soda, Vomiting.

Case Report

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INTRODUCTION

It is known that end-stage renal disease (ESRD) is characterized by metabolic acidosis due to the inability of kidneys to remove metabolic acids such as phosphates and sulfates [1], and the simultaneous regeneration of bicarbonates (HCO_3^-), due to the basic inability of the kidneys to produce ammonia. This problem during the hemodialysis procedure is addressed by increasing the bicarbonates of dialysate [2-4], with dietary modifications [5], but also with exogenous administration of soda. With a bicarbonate content in the dialysate of 30-40 mEq/L, satisfactory levels of bicarbonates in the blood are achieved after the end of session [6], so that the patient has blood bicarbonate levels above 20-22 mEq/L before the beginning of the next hemodialysis session [1-7]. However, other factors such as vomiting, iatrogenic administration of e.g. non-absorbable alkalis as antacids [8], or accidental overload with alkali or soda [9, 10], are the main causes of metabolic alkalosis in hemodialyzed patients [11], as occurred in our cases. We present here two hemodialyzed patients they showed with metabolic alkalosis and review the literature.

CASE DESCRIPTION

Patient 1

A 90-year-old woman was on a conventional hemodialysis program with bicarbonate in the last 12 months (3 times a week, for 4 hours and 30 minutes per session, with a modified cellulose filter, with surface area 1.7 m^2). She reported fatigue and coffee-colored vomiting (which continued during the dialysis session that took place on the same day), at which time she visits the hospital. She had no residual renal function and the weight gain from session to session was 1-1.5 kg. Laboratory exams showed Hct 32.1%, Hb 9.5 gr%, white blood cells 8,820/m^3 (with a normal type), C-reactive protein 9.82 mg/L (normal value <5.0) and the other laboratory tests were normal. Blood gases obtained before the dialysis session from the central venous dialysis catheter she had, showed pH 7.56, $PaCO_2$ 36 mmHg, HCO_3^- 32.2 mEq/L, PaO_2 25 mmHg, and BE (base excess) +8.1 mEq/L (metabolic alkalosis + respiratory alkalosis, from the blood gas picture alone). An intravenous proton pump inhibitor (PPI) was added therapeutically, and the patient improved after 3 days of hospitalization and was discharged from the hospital.

After 10 days, she returned complaining of intractable vomiting, so blood gases obtained before the dialysis session and again from the central venous dialysis catheter (with dialysate bicarbonates 30 mmHg), which showed pH 7.52, $PaCO_2$ 45 mmHg, HCO_3^- 36.7 mEq/L, PaO_2 19 mmHg, BE +12.3 mEq/L (metabolic alkalosis + respiratory alkalosis). During this second hospitalization, an endoscopic exam (gastroscopy) was performed and a stomach full of nutritious content, purple in color, diffuse gastritis, wall inertia and a missing pylorus were found (Figure 1). Ultrasound of the

upper abdomen was not diagnostic, while according to the radiologist "at least the gallbladder was normal". An exploratory laparotomy was decided upon, during which an amorphous mass was found in the gallbladder area that included the gallbladder, the duodenum, and the transverse colon, in conditions of inoperable cancer of unknown origin.

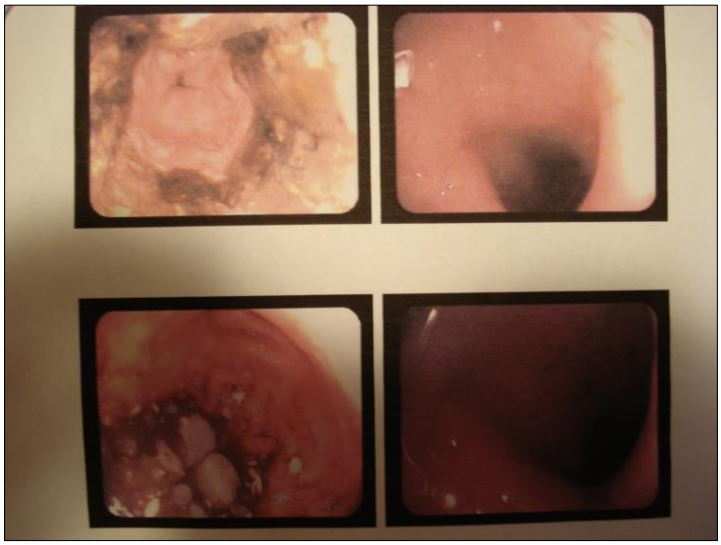

Figure 1: Gastroscopy findings (malignancy is visible in the left images)

Patient 2

An 84-year-old woman who was on an predilution online hemodiafiltration (HDF) program (3 times a week, for 4 hours and 15 minutes per session), with a polyethosulfone filter, surface area 1.9 m^2 and with a 42 L substitution volume/session, in a routine check-up that was performed and concerned all patients in our unit, it was found in the blood gases (from a sample taken from the arterial line of the fistula), before the begining of the session: pH 7.48, $PaCO_2$ 48 mmHg, HCO_3^- 35.7 mEq/L and BE +11.7, i.e. it was a pure metabolic alkalosis, at least from the blood gas picture alone. She had no residual renal function and the weight gain from dialysis session to session was approximately 1.5 kg. Because the dialysate (33 mEq/L bicarbonates) did not seem to be responsible for this alkalosis, a detailed history was taken regarding the diet and the medications she was taking (e.g., phosphate binders with carbonate) were checked and nothing was found that was responsible and explained the alkalosis. After one week, she came back with pH 7.45, $PaCO_2$ 43 mmHg, HCO_3^- 29.9 mEq/L and BE +6 mEq/L (again pure metabolic alkalosis), while on the same day when disconnected from the dialysis machine she had pH 7.46, $PaCO_2$ 40 mmHg, HCO_3^- 28.4 mEq/L and BE +4.5 mEq/L. An attempt was made again to detect the cause of metabolic alkalosis that the patient had and it was found that she was taking, at intervals of dialysis sessions, 1x3 tablespoons of baking soda daily (approximately 180 mEqx3 bicarbonates). This was an instruction that had been given to the patient, by another hospital, before the start of hemodialysis (pre-end stage chronic renal failure), although approximately 10 years had passed since then. The discontinuation of the soda was recommended and the problem of metabolic alkalosis was restored.

The severe metabolic alkalosis that our patients presented is highlighted and a review of the literature is made.

DISCUSSION

Although the expected acid-base disorders in hemodialyzed patients is the high anion gap metabolic acidosis, other types of disturbances cannot be excluded. The coexistence of metabolic alkalosis has also been noted by other investigators [12], which makes it more imperative to check the blood gases of these patients at regular intervals (at least monthly unless there is a special reason) [13]. However, it is worth noting that alkalemia from exogenous alkali administration is rare, unless the patient has renal failure.

The presence of vomiting helps the acidic contents of the stomach to be removed from the body, contributing to the retention in the circulation of the bicarbonate buffer which comes from the production of hydrogen ions, which are secreted into the lumen of the stomach to form HCl. This excess bicarbonate (which constitutes a temporary buffer and normally represents the alkaline tide) is removed from the body (as non-inactivated or non-neutralized bicarbonate), at the level of the duodenum. There, the acidic contents of the stomach constitute a homologous stimulus for the secretion of pancreatic liquids, which are rich in bicarbonates (Figure 2). In the case of vomiting, therefore, there is no stimulus for the secretion of pancreatic fluid, resulting in the gain of bicarbonates

remaining in circulation. A typical case was the case of a hemodialysis woman from Austria, who, due to induced vomiting (due to a problem in the upper gastrointestinal tract), arrived at the hospital with severe metabolic alkalosis (pH 7.52, BE +17) and was successfully treated with PPIs [12]. In these cases, the administration inhibitors of histamine H_2-receptor have proven successful for 10 years in non-dialysis patients and in patients in intensive care units [14].

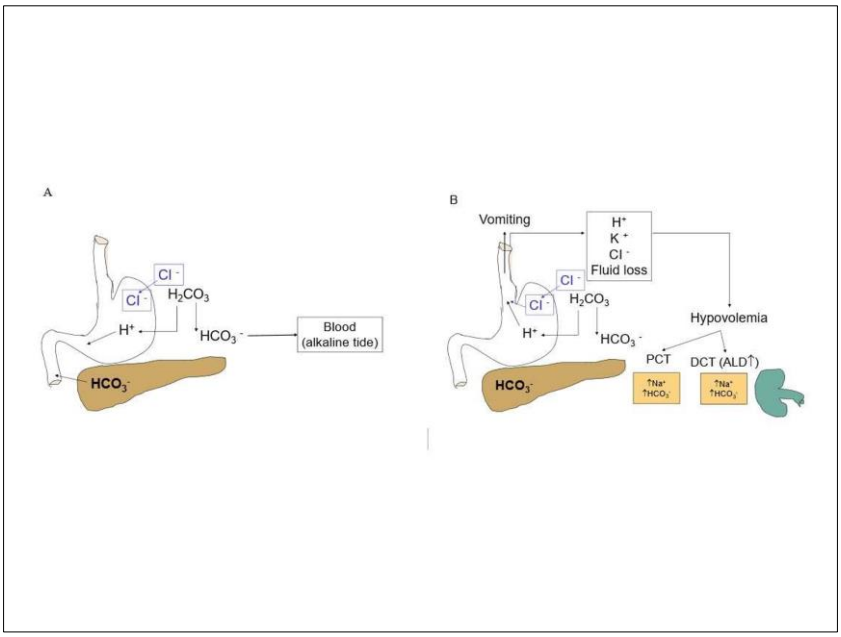

Figure 2: Initially, for every H^+ secreted in the stomach, one bicarbonate is gained, which returns to the circulation (alkaline tide) (A). Then, under normal conditions, the acidic contents of the stomach, when the food reach to the duodenum, stimulate the secretion of pancreatic fluid rich in bicarbonates, at which point the earned bicarbonates in the stomach are excreted in duodenum (A). When this does not happen and the content of the stomach is lost through vomiting, the gain of bicarbonates in the stomach are maintained in the body, but also because of hypovolemia produced aldosterone which act in DCT and reabsorb sodium in excretion of H^+, where earned bicarbonates and they cause this way metabolic alkalosis (B). (PCT = proximal convoluted tubule, DCT = distal convoluted tubule, ALD = aldosterone)

Of course, other causes of metabolic alkalosis in hemodialyzed patients have been reported in the literature, such as from taking preparations containing soda (apparently for indigestion reasons) [15], as happened in our second patient and often without medical advice [9, 10], or even in cocaine-addicted individuals.

Something similar happened to a hemodialyzed patient (who had been on dialysis since 2 years ago, 3 times a week, with a dialysate containing 35 mEq/L bicarbonates and 4 mEq/L acetate), who was taking baking soda due to hiccups that she was having and who was relieved by taking it (she took ¾ of a 126 ml bottle of Bromoseltzer solution, containing 89 mEq HCO_3^- and 71 gr citric acid, within 48 hours). At the hospital, hiccups, disorientation, reduced sensation in the left upper limb, blood pressure 170/100 mmHg, pulse 108/min and 24 breaths/min were found. Arterial blood gases revealed metabolic alkalosis (pH 7.53, $PaCO_2$ 47 mmHg, HCO_3^- 40 mEq/L and PaO_2 59 mmHg) and was successfully treated with conventional hemodialysis with reduced dialysate bicarbonates (28 mEq/L in the first hour and 30 mEq/L in the next two hours) [10], as was observed much earlier by others [16], but confirmed by us. A similar case has also been reported of a 52-year-old hemodialyzed patient who received baking soda daily for several weeks because she particularly liked it (i.e. without suffering from nausea or vomiting) [9].

Clinically, however, in addition to the above, a correlation between sleep apnea and the presence of arterial hypertension with metabolic alkalosis has been observed in a hemodialyzed patient who was taking antacids alone (4-5 gr of baking soda/24 hours). With these conditions, he appeared in the hospital with a pH of 7.47, $PaCO_2$ 55.6 mmHg and HCO_3^- 40.1 mEq/L. During his hospitalization, bicarbonates and pH progressively decreased, so that episodes of sleep apnea also was reduced, while blood pressure was restored to normal after two months, without the need for antihypertensive drugs [11]. These disorders were attributed to the reduction of respiratory function (respiratory depression), as compensation for metabolic alkalosis, where hypoxia also stimulates the activity of the sympathetic nervous system, which is responsible for hypertension, which is somewhat exacerbated by the sodium load of soda. Because of this, the mortality of

metabolic alkalosis is increased [7, 17], which is why aggressive and urgent treatment is required [18].

Similarly, Pathak *et al*., at the National Kidney Foundation Clinical Meeting in 2008 reported a hemodialyzed woman who developed a decrease in the number and range of breaths (hypopnea) during a dialysis session. The session was interrupted and the blood gases showed pH 7.46, $PaCO_2$ 94 mmHg and HCO_3^- 69 mEq/L. Although she was intubated, she immediately experienced ventricular fibrillation and arrest. Cardiopulmonary resuscitation was successful, and her heart rate and blood pressure were restored. After restoring the bicarbonates and pH levels, she was discharged from the hospital healthy 10 days. It was obvious that the patient received large amounts of bicarbonates due to poor functioning of the hemodialysis system during the session, resulting in rapid metabolic alkalosis [19].

It is noted that hypopnea in such patients is dealt with by treating the disease that is responsible for it, such as metabolic alkalosis, and not by treating hypoxemia. In fact, in the past, caution was drawn to the use of acetate hemodialysis, because it aggravated the alkalosis (due to it led to a decrease in CO_2) [20]. However, in our patients, no disturbance in blood gases ($PaCO_2$ and PaO_2) were found, so their general condition was good.

There did not appear to be any other cause of metabolic alkalosis from the history or laboratory exams of our patients, such as hypochloremia and hypokalemia, which are typically not observed in metabolic alkalosis in patients without renal function (it is noted that no one of our patients had residual renal function) [8], although hypokalemia may also be due to intracellular potassium movement due to alkalosis, while it should also be emphasized that an increase in bicarbonates in a patient with ESRD always indicates the presence of metabolic alkalosis, because the secondary increase in bicarbonates in respiratory acidosis requires the presence of renal function. In contrast, hemodialyzed patients are prone to the development of metabolic alkalosis in cases of acid loss through the gastrointestinal tract, because they do not have the ability to eliminate excess bicarbonates through the affected kidneys.

It is concluded that for various reasons, hemodialyzed patients may experience metabolic alkalosis, a fact that should be considered. It would perhaps be preferable for each hemodialysis unit to perform at least monthly blood gas analysis to detect such cases as early as possible.

REFERENCES

1. McGill, R.L., Weiner, D.E. (2017). Dialysate composition for hemodialysis: Changes and changing risk. *Semin Dial*, 30(2), 112-120.
2. Ahmad, S., Pagel, M., Vizzo, J., Scribner, B.H. (1980). Effect of the normalization of acid base balance on postdialysis plasma bicarbonate. *Trans Am Soc Artif Intern Organs*, 26, 318-321.
3. Kobrin SM, Raja RM (1989). Effect of varying dialysate bicarbonate concentration on serum phosphate. *Trans Am Soc Artif Intern Organs*, 35, 423-425.
4. Williams, A.J., Dittmer, I.D., McArley, A., Clarke, J. (1997). High bicarbonate dialysate in haemodialysis patients: effects on acidosis and nutritional status. *Nephrol Dial Transplant*, 12, 2633-2637.
5. Gotch, F.A., Sargent, J.A., Keen, M.L. (1982). Hydrogen ion balance in dialysis therapy. *Int J Artif Organs*, 6, 388-395.
6. Tentori, F., Karaboyas, A., Robinson, B.M., Morgenstern, H., Zhang, J., Sen, A. (2010) Association of dialysate bicarbonate concentration with mortality in the dialysis outcomes and practice patterns study (DOPPS). *Am J Kidney Dis*, 62(4), 738-746.
7. Wu, D.Y., Shinaberger, C.S., Regidor, D.L., et al. (2006) Association between serum bicarbonate and death in hemodialysis patients: is it better to be acidotic or alkalotic? *Clin J Am Soc Nephrol*, 1, 70-78.
8. Ostermann, M.E., Girgis-Hanna, Y., Nelson, S.R., Eastwood, J.B. (2003) Metabolic alkalosis in patients with renal failure. *Nephrol Dial Transplant*, 18, 2442-2448.
9. Bleyer, A.J., Appel, R.G. (1998) Metabolic alkalosis due to Pica in a hemodialysis patient. *Nephron*, 79, 483-484.
10. Sahani, M.M., Brennan, J.F., Nwakanma, C., Chow, M.T., Ing, T.S., Leehey, D.J. (2001) Metabolic alkalosis in a hemodialysis patient after ingestion of a large amount of an antacid medication. *Artif Organs*, 25(4), 313-315.
11. Okada, H., Inoue, T., Takahira, S., Sugahara, S., Nakamoto, H., Suzuki, H. (1999) Daytime hypertension, sleep apnea and metabolic alkalosis in a haemodialysis patient-the result of sodium bicarbonate abuse. *Nephrol Dial Transplant*, 14, 452-454.
12. Kirsch, B.M., Sunder-Plassmann, G., Schwarz, C. (2006) Metabolic alkalosis in a hemodialysis patient-successfull treatment with a proton pump inhibitor. *Clin Nephrol*, 66(5), 391-394.
13. Kovacic, V., Roguljic, L., Kovacic, V. (2003) Metabolic acidosis of chronically hemodialyzed patients. *Am J Nephrol*, 23(3), 158-164.
14. Barton, C.H., Vaziri, N.D., Ness, R.L., Saiki, J.K., Mirahmadi, .KS. (1979) Cimetidine in the management of metabolic alkalosis is induced by nasogastric drainage. *Arch Surg*, 114, 70-74.
15. Panichi, V., Rizza, G.M., Taccola, D., Consani, C., Barsotti, G. (2004) Severe hypotension during hemofiltration in an uremic patient with metabolic alkalosis. *Ren Fail*, 26(1)m 73-75.

16. Aynus, J.C., Olivero, J.J., Androgué, H.J. (1980) Alkalemia associated with renal failure: correction by hemodialysis with low bicarbonate dialysate. *Arch Intern Med*, 140, 513-515.
17. Anderson, L.E., Henrich, W.L. (1987) Alkalemia-associated morbidity and mortality in medical and surgical patients. *South Med J*, 80, 729-733.
18. Palmer, B.F., Alpern, R.J. (1997) Metabolic alkalosis. *J Am Soc Nephrol*, 8, 1462-1469.
19. Pathak, M., Zsom, L., Fulop, T. (2008) A case of extreme metabolic alkalosis in hemodialysis patient. *Am J Kidney Dis*, 51(4).
20. Blank, M.J., Lew, S.Q. (1991) Hypoventilation in a dialysis patient with severe metabolic alkalosis: treatment by hemodialysis. *Blood Purif*, 9, 109-113.