Severe Respiratory Alkalosis during Hemodialysis

Abstract

Respiratory alkalosis during hemodialysis session is a rare complication. We managed two patients with severe respiratory alkalosis, a woman who developed this 75 min after the beginning of the session and a man who developed it about 1 h before the end of the session. In both, the cause was a hypotensive episode, and both hypotension and alkalosis were successfully treated.

Keywords: *Blood gases, cardiac arrhythmias, metabolic alkalosis, respiratory alkalosis*

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Introduction

Cases of respiratory alkalosis (RA) during the dialysis session have been reported, albeit rarely, in the literature.[1] This is responsible for many cardiac manifestations, such as tachycardia, ventricular and atrial arrhythmias, and angina pectoris of ischemic and nonischemic etiology.[2] Given the high mortality,[3] it is important to prevent any such complication and to treat it promptly. With two cases of RA that we faced, the literature on the subject is reviewed in this article.

Case Presentation

Patient 1

A 51-year-old woman, who had been on hemodialysis for 120 months (conventional bicarbonate hemodialysis, duration 4 h and 30 min/session, with a polysulfone filter, 1.8 m^2 surface area), complained of palpitations 75 min after starting of dialysis session. She had a blood pressure of 90/55 mmHg and heart rate of 140/min. The ultrafiltration rate was reduced and 200 ml of 0.9% NaCl and 5-amp D/W 35% were given.

Blood gas analysis revealed severe alkalemia (with pH = 7.74, $PaCO_2$ = 18 mmHg, PaO_2 = 125 mmHg, HCO_3^- = 24.4 mEq/l), that is, RA, which was explained by the patient's hypotension and severe.

She was immediately rebreathed (in paper bag), and the dialysate HCO_3^- was reduced. After 17 min, when blood gases were assessed again, the patient had recovered (pH = 7.41, $PaCO_2$ = 40 mmHg, PaO_2 = 134 mmHg, HCO_3^- = 25.4 mEq/l) [Table 1].

Patient 2

A 78-year-old man on hemodialysis for 12 months (on-line predilution hemodiafiltration, for 4 h, with a polyethersulfone filter, 1.9 m^2 surface area), complained of restlessness, chest discomfort, dizziness, palpitations, and imminent death 80 min before the end of the session. He had tachypnea, sweating, and tachycardia. His arterial blood pressure was 65/40 mmHg and heart rate 130–140/min with an irregular pulse. Electrocardiogram showed atrial fibrillation and anterior–lateral wall ischemia.

The ultrafiltration rate was reduced, the patient placed in Trendelenburg position, and 0.9% NaCl (200 ml) was given as bolus. Severe alkalemia was found (pH >7.8, $PaCO_2$ = 16 mmHg, PaO_2 = 145 mmHg), and he was rebreathed through a paper bag. Subsequent blood gas results showed a progressive improvement of alkalosis. One hour after the end of the session, his blood gases had fully recovered. It should be emphasized that the patient in the second sample (21 min after the first) had low HCO_3^- levels for the snapshot of the session (HCO_3^- = 24.1 mEq/l), that is, about an hour before the end of the dialysis session (at 16:45 pm), a fact that, although it was paradox, nevertheless, it helped him [Table 2].

Discussion

Hypotension during the hemodialysis is common and is caused by many factors such

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Table 1: Patient 1: blood gases at the beginning of the episode (A) and 17 min later (B) (after application of exhaled air rebreathing)

	Beginning of episode	After 17 min
Time/ parameters	13:24:51	14:02:47
pH	>7.74	7.41
$PaCO_2$ (mmHg)	18	40
PaO_2 (mmHg)	80	75
HCO_3^- (mEq/l)	24.4	25.4
Lactate (mmol/l)	0.6	0.9

Table 2: Patient 2: blood gases during the diagnosis of alkalemia, until shortly before the end of the session

	Beginning of episode	After 21 min	After 50 min	End of session
Time/ parameters	16:24	16:45	17:14	17:43
pH	>7.80	7.76	7.7	7.62
$PaCO_2$ (mmHg)	16	17	19	22
PaO_2 (mmHg)	145	95	102	91
HCO_3^- (mEq/l)	-	24.1	23.5	22.6
Lactate (mmol/l)	2.4	3.2	3.3	2.7

as those related to the patient (inability to rapidly redistribute fluids during their removal by ultrafiltration, reduced cardiac output, autonomic nervous system dysfunction, arrhythmias, anemia, drugs, etc.), the method used (rapid reduction of plasma osmolality by removing, e.g. urea), rapid fluid removal (change in electrolyte levels [hypocalcemia], temperature of dialysate, hypoxia), as well as various other causes (cardiac tamponade, myocardial infarction, aortic aneurysm, sepsis, air embolism, pneumothorax, hemolysis, etc.)[4,5]

Hypotension was the cause of tachypnea in both the cases presented. The first patient was taking a beta-blocker and was routinely hypokalemic, as she was on the day the episode of RA was recorded, and was known to have panic attacks whenever something happened to her during the dialysis session, which manifested with intense nervousness and tachyarrhythmias. The second patient, on the other hand, was elderly, with frequent episodes of hypotension during the session, receiving an angiotensin-II receptor blocker (ARB), that is, valsartan 80 mg/24 h, and had a high rate of ultrafiltration (lost 1500 ml of fluids at the time of the episode) and decreased cardiac. This patient's potassium was also low during the episode (3.1 mEq/l), as would otherwise be expected from the snapshot hemodialysis session, since this increases with any acute hypocapnia, during which the catecholamine levels also increase in plasma.[6]

The causes of RA are many.[7] However, in hemodialysis patients, a transient RA is found, which is due to an increase in the sensitivity of the respiratory center to CO_2, due to dialysis-induced disruption of the osmotic balance, as found experimentally, with or without urea in the dialysate.[8] As early as 1975, the opinion was expressed that during the dialysis session, there are pulmonary microemboli, which are responsible for the observed hypoxia,[9] a fact that was confirmed by detection of air bubbles in the lungs,[10] due to which tachypnea and thus hypocapnia are caused. These microemboli come from the lines of the dialysis extracorporeal circuit or from the filters, that is, from bubbles that travel through the venous line to the lungs, where they are trapped.

Unlike those with normal renal function, who tolerate hypocapnia well without serious complications, those with chronic kidney disease can experience severe and life-threatening complications due to alkalemia. Levels of pH >7.80 have been found in a hemodialysed patient, that is, almost respectively to those of our first patient.[11] Severe alkalemia (pH > 7.60) is associated with a reduction in blood supply to the brain and myocardium due to vasoconstriction, an effect that is more pronounced in RA than metabolic alkalosis, while at the same time, alkalemia shifts the O_2 dissociation curve from hemoglobin (Hb) to the left (more difficult release of O_2).[12] Neurologic disturbances include headache, tetany, delirium, and lethargy. The decrease in ionized calcium levels probably contributes to the appearance of these manifestations, as well as tissue hypoxia, due to the more difficult release of O_2 from Hb. Of course, alkalemia also lowers the threshold for angina pectoris and predisposes to persistent (resistant) supraventricular and ventricular arrhythmias. This arrhythmogenic effect is more evident in patients with underlying heart disease.[13] Ultimately, it appears that our patient's symptomatology is fully explicable by hypotension and RA.

The explanation for the low levels of HCO_3^- at the time of the episode (shortly before the end of the dialysis session snapshot in which it increased) is simple. In hypocapnia, an immediate decrease in plasma HCO_3^- is found. This acute adaptation is completed within 5–10 min of the beginning of hypocapnia and is due to HCO_3 – titration (consumption) by non-bicarbonate buffers of the body. To a lesser extent, this acute adaptation reflects an increase in the production of organic acids, especially lactate. Particularly, in acute RA, HCO_3^- is adaptively reduced by 0.2 mEq/l for every 1 mmHg of $PaCO_2$ reduction.[14,15] Something like this happened to our second patient, since shortly before the end of the session, he did not show increase of HCO_3^- (24.1 mEq/l).

Conclusion

It is concluded that the presence of severe alkalemia is an emergency and should be treated immediately. Such a simple instruction to rebreathe the exhaled air is

particularly effective as well as impressive (care should be taken not to sedate these patients if possible).

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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