Metabolic alkalosis due to short bowel syndrome in a hemodialysis patient

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Abstract

A 53-year-old man on hemodialysis suffered from short bowel syndrome after subtotal colectomy and partial resection of the small intestine. Laboratory tests showed multiple electrolyte disorders and enlarged sodium and chloride ion (Cl^-) gaps despite treatment with large volume of sodium chloride replacement via central venous infusion. Blood gas analysis showed slightly high bicarbonate ion levels and metabolic alkalosis was suspected, which is uncommon in end stage kidney disease. The measurement of electrolytes in feces from ileostomy showed relatively high Cl^- excretion. The loss of Cl^- to feces may have caused the metabolic alkalosis; thus, additional Cl^- replacement may have been necessary.

Keywords Fecal electrolytes · Acid–base disorders · Hypochloremic alkalosis · Ileostomy

Introduction

Generally, patients with renal failure, especially those on dialysis, are more likely to develop metabolic acidosis. Short bowel syndrome can cause either acidosis or alkalosis, depending on the surgical procedure [1–3] and site. It has been reported that metabolic acidosis occurs in hemodialysis patients with short bowel syndrome [4, 5]. In addition to the fact that hemodialysis patients usually tend to be acidotic due to the accumulation of non-volatile acids, it seems that there was an overproduction of D-lactate in these cases [4–6]. However, to the best of our knowledge, there have been no reports of an alkalotic hemodialysis patient with short bowel syndrome.

Case report

We report the case of a 53-year-old man on hemodialysis because of diabetic nephropathy for 16 years. Five months ago, during his hospitalization for treatment of a left foot ulcer due to obstructive arteriosclerosis, intestinal necrosis occurred incidentally. His intestinal tract was extensively necrotic from the small intestine to the colon. Subtotal colectomy and partial resection of the small intestine were performed. As a result, the remaining intestinal tract was estimated to be about 1.5 m of the oral small intestine and a part of the rectum. He recovered in intensive care and was discharged from the hospital after prescription of a proton pump inhibitor. However, he was hospitalized again due to re-infection of the left foot ulcer. Laboratory tests showed lowered sodium (Na^+) and chloride (Cl^-) ions, and elevated potassium (K^+) ion compared with the preoperative findings (Table 1). Elevated blood glucose and C-reactive protein (CRP) levels were due to the inflammation in his left foot ulcer.

The results of the preoperative laboratory tests and after admission are shown in Table 1. A reduction in CRP level as a result of lower limb amputation and antibiotic treatment was observed; in addition, glycemic control was improved to some extent. However, despite these improvements in the patient's general condition, there was no improvement in the lowered Na^+ and Cl^- levels. Moreover, the gap of Na^+ and

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Table 1 Laboratory data obtained preoperatively, on the day of admission, and on day 11

	Preoperation	Day of admission	Day 11
Sodium (mEq/L)	136	130	127
Potassium (mEq/L)	4.8	5.9	3.4
Chloride (mEq/L)	101	90	84
Calcium (mg/dL)	8.1	9.0	9.9
Phosphorus (mg/dL)	6.4	3.3	3.4
Total protein (g/dL)	5.9	5.2	6.0
Albumin (g/dL)	3.4	2.2	2.4
Urea nitrogen (mg/dL)	55.0	62.0	145
Creatinine (mg/dL)	7.24	8.87	9.44
Uric acid (mg/dL)	4.5	6.7	9.6
Glucose (mg/dL)	221	484	211
CRP (mg/dL)	0.07	9.1	1.4
D-Dimer (μg/mL)	NA	4.4	2.2

CRP C-reactive protein, NA not available

Cl^- ($\text{Na}^+ - \text{Cl}^-$) was enlarged. Although we performed total parenteral nutrition treatment (total Na^+ : 295 mEq/day, K^+ : 106 mEq/day, Cl^- : 350 mEq/day, water: 3500 mL/day), there was no improvement. Blood gas analysis from the arterio-venous fistula showed higher bicarbonate ion (HCO_3^-) level than that in the arterial blood obtained immediately after the operation; therefore, metabolic alkalosis was suspected despite his renal function at the end stage of kidney disease (Table 2). In addition, measurement of electrolytes in feces from ileostomy at postoperative day 114 returned acidic (pH 5.0) with a relatively high Cl^- concentration (Table 3). Therefore, we changed the concentration of HCO_3^- in the dialysate from 30 to 25 mEq/L to regulate his blood gases and electrolytes.

Discussion

Metabolic alkalosis occurs in a specific clinical setting, requiring two mechanisms to be present. The first mechanism is an increase in plasma HCO_3^- concentration (generation of alkalosis). Secondly, it requires a mechanism to maintain the high plasma HCO_3^- [7]. Potential mechanisms of alkaline generation include acid loss, escaping from the digestive tract and kidney, intracellular shift, and exogenous or endogenous alkali increase. The maintenance mechanism is likely a decrease in glomerular filtration rate and loss of control of H^+ and HCO_3^- in the renal tubules [7, 8].

In this case, postoperative gastric acid secretion was excessive, and intestinal fluid (including HCO_3^-) could not be secreted due to short bowel syndrome. We speculated that because of the maintenance mechanism, which

Table 2 Blood gas data

	Postoperative day 1 (sample obtained from the artery)	Before hemodialysis (sample obtained from the arterio-venous fistula)	After hemodialysis (sample obtained from the arterio-venous fistula)
pH	7.30	7.40	7.47
PCO_2 (mmHg)	40.2	43.1	42.0
PO_2 (mmHg)	179	93.1	101
Bicarbonate (mmol/L)	20.5	26.2	30.3
Sodium (mmol/L)	141	129	133
Potassium (mmol/L)	3.5	3.2	2.9
Chloride (mmol/L)	106	91	100
Ionized calcium (mmol/L)	1.14	1.40	1.34
Glucose (mg/dL)	186	128	148
Lactate (mg/dL)	69	11	8.0
Hemoglobin (g/dL)	10.0	10.9	10.2

PO_2 partial pressure of oxygen, PCO_2 partial pressure of carbon dioxide

is usually seen in end stage kidney disease, HCO_3^- could not be excreted from the kidney and with the high level of HCO_3^- concentration of dialysate, metabolic alkalosis was maintained.

Short bowel syndrome often causes various metabolic acid–base disorders, and some cases of alkalosis have been reported [9]. Conversely, severe acidosis has been reported in a hemodialysis patient [4], likely caused by abnormal production of D-lactate [4–6]. However, in the present case, we almost totally resected the colon in which D-lactate producing bacteria grows; thus, this mechanism no longer had an effect on metabolic acidosis. Consequently, gastric acid was

Table 3 Laboratory analysis of the fecal matter from ileostomy (post-operative day 114)

		Reference range [12]
pH	5.0	
Sodium (mEq/L)	98.3	20–30
Potassium (mEq/L)	9.6	55–75
Chloride (mEq/L)	115.6	15–25
Calcium (mg/dL)	11.0	
Phosphorus (mg/dL)	1.8	
Urea nitrogen (mg/dL)	77.4	
Amylase (mg/dL)	3.0	
Bicarbonate (mEq/L)	< 3.1	0
Total amount (mL/day)	2700	

excreted in excess, and the intestinal tract was not able to neutralize it [10, 11], resulting in alkalosis due to excretion of acidic intestinal fluid.

We clarified this theory by actually measuring the state of electrolyte and acid–base equilibrium in our patient's feces.

It is important to consider the excretion of electrolytes in the feces especially in hemodialysis patients because electrolytes cannot be adjusted in the kidney. Although there are few reports of fecal electrolytes analyses, past studies (not in hemodialysis patients) have reported that excretion of electrolytes gradually begins to improve few weeks after surgery [12]. However, in the present case, even after 5 months of surgery, massive loss of Cl^- to the feces made it difficult to improve electrolyte balance. In addition, the rise in HCO_3^- level was slight compared with the Na^+ and Cl^- gap because there was an increased concentration of non-volatile acids secondary to renal failure.

In conclusion, we experienced a maintenance hemodialysis patient with short bowel syndrome exhibiting hypochloremic metabolic alkalosis. Sufficient amount of chloride should be administered in this morbidity.

Compliance with ethical standards

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee at which the studies were conducted (IRB approval number 31-52) and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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