CASE REPORT

Hypokalemia-associated paralysis and metabolic acidosis in a patient with bilateral ureterosigmoidostomy

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Abstract Ureterosigmoidostomy is a urological intervention performed to treat various conditions such as invasive bladder cancer, bladder exstrophy, vesicovaginal fistula, or urethral trauma. However, this intervention may lead to several metabolic complications. Here, we report an interesting case with quadriparesis and intestinal paralysis resulting from severe hypokalemia (the serum potassium level, 1.8 mEq/L) and hyperchloremic metabolic acidosis [pH 6.927 and the arterial bicarbonate level, 8.0 mEq/L] in a 65-year-old man who had undergone bilateral ureterosigmoidostomy for bladder cancer 16 years earlier. The abdominal computed tomography scan also showed that massive fluid consisting of the mixture of the diverted urinary stream and feces was accumulated in the dilated distal colon. The treatment with intravenous potassium and sodium bicarbonate administration combined with the drainage of the diverted urinary stream from the distal colon resulted in the restoration of hypokalemia and acidosis followed by the improvement of quadriparesis and intestinal paralysis. The underlying mechanism and the treatment of metabolic complications after ureterosigmoidostomy are briefly discussed.

Keywords Hypokalemia · Intestinal paralysis · Metabolic acidosis · Quadriparesis · Ureterosigmoidostomy

Introduction

Ureterosigmoidostomy is a surgical procedure in which the ureters are diverted into the sigmoid colon, although it often results in metabolic complications [1]. One of these complications is hypokalemia, which can cause muscular paralysis such as quadriparesis [2–6]. This diversion technique was previously performed in patients with invasive bladder cancer, bladder exstrophy, or urethral trauma. However, this technique is rarely applied now because of the high rate of metabolic complications [7]. Herein, we report an interesting case of ureterosigmoidostomy-associated hypokalemia, quadriparesis, intestinal paralysis, and hyperchloremic metabolic acidosis.

Case report

A 65-year-old man was transferred to the emergency department of our hospital with weakness of all four limbs after a 1-month history of fever and anorexia. The weakness was more marked in lower limbs and he could not walk and defecate by himself. The patient had undergone bilateral ureterosigmoidostomy for bladder cancer 16 years earlier.

On arrival at the department, the patient was drowsy (Glasgow Coma Scale, E2V1M4) and had abdominal distention. His blood pressure was 122/81 mmHg and the electrocardiogram showed sinus tachycardia at a rate of 113 beats/min. The arterial blood gas analysis showed severe metabolic acidosis [pH 6.927 and bicarbonate (HCO_3^-), 8.0 mEq/L]. The other laboratory data were as follows: white blood cells, $17,900/\text{mm}^3$; red blood cells, $428 \times 10^4/\text{mm}^3$; hemoglobin, 12.5 g/dL; platelets, $39.1 \times 10^4/\text{mm}^3$; serum creatine kinase, 339 U/L; blood

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Fig. 1 Findings of abdominal CT scan on admission. Massive fluid consisting of the mixture of diverted urine and feces was observed in the dilated distal colon (arrows)

urea nitrogen, 111 mg/dL; serum creatinine, 2.98 mg/dL; sodium (Na), 142 mEq/L; potassium (K), 1.8 mEq/L; chloride (Cl), 114 mEq/L; C-reactive protein, 5.6 mg/dL and blood glucose, 259 mg/dL. The abdominal computed tomography (CT) scan showed that massive fluid, consisting of the mixture of diverted urine and feces, had accumulated in the dilated distal colon (Fig. 1). Although obvious abnormalities were not found in the thyroid function and the adrenocorticotrophic hormone-cortisol axis, the renin-aldosterone system was extremely activated [plasma renin activity, 68 ng/mL/h and plasma aldosterone concentration, 353 pg/mL, respectively] on admission (Fig. 2; Table 1). A urinalysis could not be performed because the patient had undergone bilateral ureterosigmoidostomy and there was no “bladder urine” excretion.

The patient was immediately treated with intravenous administration of potassium chloride and sodium bicarbonate. In addition, the massive fluid in the distal colon was removed with frequent rectal drainage. The patient was also treated with intravenous antibiotics (Cefepime) for the management of a urinary tract infection.

After initiating treatment, the serum levels of K and HCO_3^- gradually increased. Along with the improvement of hypokalemia, the patient was able to walk and defecate by himself. On day 16 of hospitalization, the plasma renin activity and the plasma aldosterone concentration also normalized to 0.7 ng/mL/h and 105 pg/mL, respectively (Fig. 2). The patient fully recovered and was discharged.

Fig. 2 Clinical course

Table 1 Laboratory data on admission

WBC	17,900/mm ³	HbA1c	8.0 %
RBC	428 × 10 ⁴ /mm ³	BNP	62.6 pg/mL
Hb	12.5 g/dL	CRP	5.6 mg/dL
Plt	39.1 × 10 ⁴ /mm ³	Procalcitonin	5.66 ng/mL
TP	7.1 g/dL	pH	6.927
Alb	3.6 g/dL	PCO ₂	39.2 mmHg
T-bil	0.4 mg/dL	PO ₂	130.4 mmHg
AST	18 U/L	HCO_3^-	8.0 mEq/L
ALT	6 U/L	BE	−23.8
LDH	339 U/L	Lactate	16.90 mg/dL
CK	339 U/L	Anion gap	14.4
BUN	111 mg/dL		
Cr	2.98 mg/dL	free T3	1.54 pg/mL
UA	4.3 mg/dL	free T4	0.82 pg/mL
Na	142 mEq/L	TSH	0.7676 pg/mL
K	1.8 mEq/L	ACTH	9.9 pg/mL
Cl	114 mEq/L	Cortisol	57.5 μg/dL
Ca	8.7 mg/dL	Renin activity	68 ng/mL/h
Pi	3.2 mg/dL	Aldosterone	353 pg/mL
Glucose	259 mg/dL		

Discussion

Ureterosigmoidostomy is a urological intervention performed to treat various conditions such as invasive bladder cancer, bladder exstrophy, vesicovaginal fistula, and urethral trauma. However, this intervention may lead to several metabolic complications [1, 7]. One of these complications is hypokalemia, which can cause muscular paralysis (quadriparesis or paralysis of the intestines and respiratory muscles).

In epithelial cells of the distal colon, the epithelial sodium channel (ENaC) and the potassium (K) channel are expressed and play roles in the reabsorption of sodium (Na) and the secretion of K, which is similar to the kidneys [8]. These actions are regulated by the renin-angiotensin system via the mineralocorticoid receptor. In our case, both the renin activity and the aldosterone concentration on admission were extremely elevated likely due to dehydration, which was caused by fever and anorexia associated with the urinary tract infection (Fig. 2; Table 1). Therefore, this increased activity of the renin-angiotensin system was supposed to induce the secretion of K and the reabsorption of Na in both the kidneys and the distal colon. As a result, the patient developed severe hypokalemia followed by quadriparesis and intestinal paralysis. In accordance with this possibility, both the renin activity and the aldosterone concentration were normalized on day 16 after hospitalization when his hypokalemia had already improved (Fig. 2).

Hyperchloremic metabolic acidosis secondary to ureterosigmoidostomy was first described by Boyd et al. [9] in a child who developed osteomalacia (rickets), although the mechanism is not completely understood. In epithelial cells of the sigmoid colon, the chloride ion/bicarbonate ion ($\text{Cl}^-/\text{HCO}_3^-$) exchanger is expressed and is quite activated [10]. As the diverted urinary stream bathes the intestinal lining, Cl^- is reabsorbed in exchange for secreted HCO_3^- . Thus, the extensive contact time with the colon increases the magnitude of the $\text{Cl}^-/\text{HCO}_3^-$ exchange. The loss of alkali coupled with chloride accumulation results in hyperchloremic metabolic acidosis. In our case, prolonged contact time of the diverted urinary stream with the colon due to the hypokalemia-associated intestinal paralysis was likely to cause severe metabolic acidosis (Fig. 1).

Patients treated with ureterosigmoidostomy should be instructed to empty the rectum as frequently as possible and, if necessary, receive potassium and bicarbonate supplementation to prevent the development of hypokalemia and metabolic acidosis. A rectal tube insertion at night in order to drain the diverted urine is also effective to prevent these complications. In our case, the treatment with intravenous potassium and sodium bicarbonate administration combined with frequent drainage of the diverted urinary stream from the distal colon resulted in the restoration of hypokalemia and acidosis followed by quadriparesis and intestinal paralysis (Fig. 2). On the other hand, the metabolic complications occur less frequently in patients treated with the ureteroileostomy, because the rapid drainage of urine into the bag reduces the contact time with the intestines [1, 7]. Therefore, we considered constructing an

ileal conduit to prevent further episodes of hypokalemia and metabolic acidosis. However, the patient was unwilling to undergo the surgery.

There are several limitations in our case. First, we did not evaluate muscle strength test quantitatively before and after treatment. Second, the balance study of urinary electrolytes was needed normally, although it is practically difficult to separate urine from feces in patients with bilateral ureterosigmoidostomy.

In summary, we treated a case of quadriparesis and intestinal paralysis associated with severe hypokalemia and hyperchloremic metabolic acidosis after bilateral ureterosigmoidostomy. Awareness of this condition and timely management are necessary to avoid this life-threatening situation.

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Compliance with ethical standards

Conflict of interest The authors have declared that no conflict of interest exists.

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