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Case Report

Deterioration of Chronic Renal Dysfunction and Serum Electrolyte Disorders Associated with Massive Ileostomy Effluent

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A 27-year-old man with a 16-year history of Crohn's disease was referred to our hospital because of general fatigue and electrolyte disorders. The past medical history included chronic renal dysfunction due to renal amyloidosis and permanent ileostomy performed 2 years earlier. Laboratory findings on admission showed hypochloremia, hypokalemia, metabolic alkalosis, and acute deterioration of chronic renal dysfunction. The above disorders were considered to be due to dehydration caused by excessive loss of fluid and electrolyte from the ileostomy. Dehydration and electrolyte disorders were corrected by proper rehydration, which also improved renal dysfunction to the basal level. Although renal failure due to massive ileostomy effluent is rare, clinicians should be aware of possible dehydration in ileostomists, and treat such patients with water and electrolyte.

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Introduction

Ileostomy is performed in inflammatory bowel diseases, familial polyposis syndrome, and colorectal carcinoma when it is necessary to remove or bypass the entire colon and rectum. The ileostomy could lead to chronic water and sodium depletion due to ileostomy effluent, but almost all ileostomists do not develop significant symptoms attributable to dehydration or sodium depletion.[1] We report a case that showed gradual deterioration of chronic renal dysfunction due to excessive loss of water and sodium from ileostomy. Renal function impairment improved to the basal level following adequate rehydration.

Case report

A permanent ileostomy was performed in June 2000 for a 27-year-old man following the development of pelvic abscess caused by intra-abdominal fistula. The past medical history included a 16-year history of Crohn's disease and chronic renal dysfunction due to renal amyloidosis. In December 2001, he received 2000 mL per day of intravenous fluid (including 70 mEq of sodium chloride and 40 mEq of potassium) for loss of intestinal fluid from ileostomy. His body weight was 38.5 kg in those days. The laboratory findings on December 14, 2001 are shown in Table 1.

The patient was referred to our hospital on June 24, 2002 for further management of general fatigue and electrolyte derangement. On admission, he was 154 cm tall and weighed 36 kg. Arterial blood pressure was 98/66 mmHg and pulse rate was 104 bpm and regular. The conjunctivae indicated mild anemia, but no icterus. Examination of the neck, chest, and abdomen was negative and neurological examination was unremarkable. The skin was dry but no edema was detected in both extremities. Laboratory findings on admission (Table 2) revealed hypochloremia, hypokalemia, metabolic alkalosis, and deterioration of chronic renal dysfunction. The plasma total protein and albumin concentrations were elevated suggesting dehydration. Analysis of arterial blood gases indicated metabolic alkalosis. The urinary volume was about 1500 mL per day and sodium

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Table 1. Laboratory findings on December 14, 2001

Peripheral blood		Blood biochemistry		Urinalysis	
WBC (/mm^3)	8100	T. Bil (mg/dL)	0.1	protein	(+)
RBC (×10^4/mm^3)	282	T.P. (g/dL)	5.5	occult blood	(-)
Hb (g/dL)	8.3	Albumin (g/dL)	3.4	sugar	(-)
Hct (%)	25.4	GOT (IU/L)	22	Na (mEq/L)	18
Plt (×10^4/mm^3)	44.1	GPT (IU/L)	35	K (mEq/L)	28.8
MCV (fl)	90.1	T-Chol (mg/dL)	113	Cl (mEq/L)	9
MCH (pg)	29.4	LDH (IU/L)	101		
MCHC (g/dL)	32.7	BUN (mg/dL)	16		
		Cr (mg/dL)	1.9		
Serological tests		UA (mg/dL)	5.4		
CRP (mg/dL)	1.02	Na (mEq/L)	145		
		K (mEq/L)	4.5		
		Cl (mEq/L)	109		
		Ca (mg/dL)	9.6		
		IP (mg/dL)	4.9		

Table 2. Laboratory findings on admission

Peripheral blood		Blood biochemistry		Urinalysis	
WBC (/mm^3)	7800	T. Bil (mg/dL)	0.4	protein	(++)
RBC (×10^4/mm^3)	311	T.P. (g/dL)	8.3	occult blood	(-)
Hb (g/dL)	9.4	Albumin (g/dL)	4.5	sugar	(-)
Hct (%)	26.9	GOT (IU/L)	19	Na (mEq/L)	8
Plt (×10^4/mm^3)	26.8	GPT (IU/L)	15	K (mEq/L)	53.5
MCV (fl)	86.5	T-Chol (mg/dL)	88	Cl (mEq/L)	9
MCH (pg)	30.2	LDH (IU/L)	137		
MCHC (g/dL)	34.9	BUN (mg/dL)	52	**Arterial blood gas analysis**	
		Cr (mg/dL)	4.2	pH	7.470
Serological tests		UA (mg/dL)	13.0	PCO_2 (mmHg)	58.9
CRP (mg/dL)	0.30	Na (mEq/L)	135	PO_2 (mmHg)	93.0
		K (mEq/L)	2.9	HCO_3^- (mmol/L)	41.9
		Cl (mEq/L)	85	BE (mmol/L)	15.8
		Ca (mg/dL)	9.0	O_2 Sat (%)	97.5
		IP (mg/dL)	3.7		

urinary output was low while potassium output was high. Analysis of the daily ileostomy effluent showed a volume of about 2500 mL per day, pH 7.0, and sodium, potassium, and chloride concentrations of 128, 7.3, and 131 mEq/L, respectively.

Based on the physical examination (weight loss, hypotension, taphycardia, and dry skin) and the laboratory findings on admission (elevation of total protein concentration and hematocrit, and hypochloremia), and on the fact that the patient had been eating solid food and fluid without restriction since December 2001, the diagnosis was considered to be dehydration due to excessive loss of fluid and electrolyte from ileostomy. It was assumed that oral feeding resulted in increased volume of gastric and intestinal juice secreted from the gastrointestinal tract, which was eventually lost through the ileostomy. Therefore, we advised dietary restriction of fat in order to reduce intestinal secretions, but the patient refused the advice and continued to consume all food types. Accordingly, the hydration volume was increased to 3,500 mL per day (including 182 mEq of sodium, 202 mEq of chloride, and 100 mEq of potassium) by intravenous infusion. This resulted in a fall in serum creatinine concentration to 2.0 mg/dL and an increase in serum potassium concentration to 4.0 mEq/L. However, hypochloremia and metabolic alkalosis failed to respond to such treatment. In the next step, we increased the dose of sodium and chloride in the intravenous infusion fluid to 189 and 254 mEq/L per day, respectively. This resulted in elevation of serum chloride concentration to 98 mEq/L, and improvement of arterial pH (to 7.406) and serum bicarbonate concentration (to 26.1 mmol/L), and amelioration of fatigue state. The laboratory findings on discharge from hospital are shown in Table 3.

Table 3. Laboratory findings on dicharge from hospital

Peripheral blood		Blood biochemistry		Arterial blood gas analysis	
WBC (/mm^3)	5900	T. Bil (mg/dL)	0.4	pH	7.406
RBC (×10^4/mm^3)	289	T.P. (g/dL)	5.9	PCO_2 (mmHg)	42.0
Hb (g/dL)	8.6	Albumin (g/dL)	3.5	PO_2 (mmHg)	92.0
Hct (%)	25.6	GOT (IU/L)	20	HCO_3^- (mmol/L)	26.1
Plt (×10^4/mm^3)	30.1	GPT (IU/L)	26	BE (mmol/L)	2.8
MCV (fl)	88.6	T-Chol (mg/dL)	94	O_2 Sat (%)	97.3
MCH (pg)	29.8	LDH (IU/L)	139		
MCHC (g/dL)	33.6	BUN (mg/dL)	26		
		Cr (mg/dL)	2.3		
Serological tests		UA (mg/dL)	10.9		
CRP (mg/dL)	0.33	Na (mEq/L)	135		
		K (mEq/L)	4.1		
		Cl (mEq/L)	98		
		Ca (mg/dL)	9.6		
		IP (mg/dL)	3.7		

Discussion

Permanent ileostomy is the standard management for patients requiring panproctocolectomy for Crohn's disease. This technique is the accepted approach to the creation of an end stoma, because it prevents the development of serositis and resultant stricture formation. Although the risk of sodium and water depletion in ileostomists due to the loss of ileostomy effluent was described almost 40 years ago,[2] it is not always appreciated by clinicians. Clarke et al.[1] reported that ileostomists showed on average 11 % reduction in total body water and a 7 % deficit in total exchangeable sodium. To our knowledge, there are two reports of renal failure caused by massive ileostomy effluent in patients with normal renal function.[3,4] In our patient, however, renal dysfunction was already present due to the

associated amyloidosis, and hence it acutely deteriorated because of dehydration caused by ileostomy effluent loss.

Kennedy et al.[5] reported that the mean (±standard deviation) ileostomy effluent volume was 606±287 mL per day, and Christl et al.[6] indicated that the ileostomy output in some patients was more than 1000 mL per day and that the effluent was nearly isotonic with sodium concentrations of 100-130 mEq/L. In our patient, the measured volume of ileostomy effluent was about 2,500 mL per day. This massive volume of effluent was most likely due to the non-restricted oral intake of food and fluid. Although we advised restriction of fat-containing foods to reduce intestinal secretions, the patient refused the advice. Clarke et al.[1] advised increasing salt intake to maintain the sodium balance in ileostomists. Considered together, our reports and those of others emphasize the importance of diet control in ileostomists in order to prevent hyponatremia and dehydration.

Our case developed hypokalemia and metabolic alkalosis in addition to dehydration and hyponatremia. Kennedy et al.[5] reported that potassium urinary loss was elevated and the ratio of urinary sodium to potassium was low in ileostomists, as was the case in our patient, and that plasma renin and aldosterone concentrations were significantly higher in the ileostomists than in healthy subjects. Metabolic alkalosis seems to be associated with dehydration and hypokalemia. Both dehydration, including depletion of effective volume and chloride, and hypokalemia increase renal bicarbonate reabsorption.[7-9] Since fluid absorption is impaired in ileostomists, even high oral fluid intake may not lead to rehydration but rather to higher volumes of liquid stools.[4] In the two cases already reported,[3,4] serum electrolyte disorder was milder than in our case, and therefore dysfunction due to renal amyloidosis seems to have caused these disorders in our patient. Accordingly, our patient was treated with intravenous fluid containing high quantities of chloride and potassium.

In conclusion, we reported a patient with deterioration of chronic renal failure due to excessive loss of ileostomy effluent. Clinicians should be aware of possible dehydration in ileostomists, and treat them with appropriate fluid and electrolyte.

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