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ACUTE RENAL FAILURE, ELECTROLYTE AND ACID-BASE CHANGES IN THREE CASES WITH ILEOSTOMY

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ABSTRACT

Ileostomy is a standard surgical procedure performed in order to protect the anastomosis and reduce the complication risk in colorectal surgery. Dehydration is one of the most important complications of ileostomy and dehydration-induced renal failure. We present three ileostomy cases that had undergone bowel resection for small bowel perforation, two of which due to mesenteric ischemia, and the other one due to a gunshot injury, all three developing acute renal failure. As ileostomy patients carry the risk of life-threatening renal failure and electrolyte and acid-base changes due to dehydration, clinicians should follow-up these patients closely considering the possible complications of ileostomy.

Keywords: Ileostomy, Acute renal failure, Acidosis, Alkalosis, Electrolyte imbalance, Dehidratation.

1. INTRODUCTION

An ileostomy is a common surgical procedure which prevents anastomotic complications in sphincter–preserving colorectal surgery and minimizes the risk of peritoneal sepsis due to anastomotic leak. Ileostomies are associated with increased morbidity and mortality, while dehydration is the most common complication [1]. A well-performed ileostomy causes 200–1000 mL of daily fluid loss [2]. Many patients adapt to this daily loss by making changes in fluid and salt intake, urine volume, and electrolyte and acid-base excretions in order to maintain volume, electrolyte, and acid base equilibrium [3]. This balance is so sensitive that it can be impaired by ileostomy-induced daily fluid losses exceeding 1 L, resulting in acute renal failure (ARF). We present three cases of ARF and electrolyte and acid-base changes following an ileostomy.

2. CLINICAL PRESENTATION AND INTERVENTION

Case 1: A 48-year-old male patient presented with complaints of anorexia, malaise, and oliguria which had persisted for three days. He had a history of ileostomy secondary to mesenteric ischemia 20 days earlier. He reported that he had received 5 L oral fluid daily and excreted approximately 4 L daily from the ileostomy. He had been re-admitted to the hospital a week earlier with similar complaints and was given intravenous (IV) fluids. In his medical history, he had received Coumadin for 6 months due to deep venous thrombosis diagnosed three years ago. On physical examination, he was conscious, cooperative, and orientated. Blood pressure was 110/70 mmHg, heart rate was 90/min, and body temperature was 37ºC. The patient was dehydrated and had reduced turgor and tonus of the skin. The laboratory findings were as follows: glucose: 125 mg/dL; urea: 103 mg/dL; creatinine: 4.1 mg/dL; sodium: 114 mmol/L; potassium: 4.2 mmol/L; chlorine: 69 mmol/L; and albumin: 3.8 g/dL. Arterial blood gases were as follows: pH: 7.5; pCO_2: 52 mmHg; and HCO_3: 40 mEq/L. He was hospitalized with a diagnosis of pre-renal ARF consequent to dehydration caused by ileostomy losses. He was monitored for fluid intake, excretion, and daily fluid replacement. His creatinine levels decreased to normal with IV fluid replacement. At follow-up, the patient's urea and creatinine levels were found to be elevated again despite oral fluid intake when parenteral fluid infusion was stopped. He was transferred to the general surgery department, and the ileostomy was closed by ileoileal anastomosis. At follow-up, the patient's creatinine levels had decreased to 0.6 mg/dL, parenteral fluids had ceased, and urea and creatinine levels remained normal.

Case 2: A 50-year-old male patient presented with complaints of malaise, anorexia, and oliguria which had persisted for three days. He had a history of ileostomy secondary to mesenteric ischemia 17 days earlier. On physical examination, blood pressure was 90/60 mm/Hg, heart rate was 92/min, and body temperature was 36ºC. The patient looked dehydrated, his tongue was dry, and skin turgor was decreased. Laboratory findings were as follows: glucose: 107 mg/dL; urea: 257 mg/dL; creatinine: 4.1 mg/dL; uric acid: 12.15 mg/dL; sodium: 117 mmol/L; and potassium: 7.0 mmol/L. Arterial blood gases were as follows: pH: 7.41; pCO_2:25 mmHg; and HCO_3: 16 mEq/L. The patient had approximately 3 L daily drainage through the ileostomy and was admitted to the internal medicine clinic for volume loss-induced pre-renal ARF. His fluid balance was followed up, and creatinine levels (0.6 mg/dl) decreased with the administration of IV isotonic solutions. At follow-up, the patient's urea and creatinine levels had increased again despite oral fluid intake when parenteral fluids were stopped. Urea and the creatinine levels remained within the normal limits with parenteral fluid support, so the patient was referred to the general surgery department. After closure of the ileostomy, electrolyte disorder and renal failure did not resume.

Case 3: A 25-year-old male patient presented with mental confusion and oliguria that had begun a day prior to admission. He had an ileostomy for small bowel perforation caused by a shotgun injury 20 days earlier. On physical examination, he was mentally confused. Blood pressure was 90/60 mm/Hg, heart rate was 96/min, and body temperature was 37ºC. He had

decreased turgor and tonus. Laboratory findings were as follows: glucose: 131 mg/dL; urea: 455 mg/dL; creatinine: 9.5 mg/dL; sodium: 119 mmol/L; potassium: 5.9 mmol/L; chlorine: 103 mmol/L. Arterial blood gases were as follows: pH: 7.52; pCO_2: 48 mmHg; and HCO_3: 39 mEq/L. He was diagnosed with ARF related to dehydration due to ileostomy loss. IV isotonic fluid replacement reduced the creatinine level to 0.8 mg/dL on the third day of admission. Although the patient's oral fluid intake was good, ileostomy drainage was approximately 5 L daily. Ceasing parenteral fluids caused a relapse of dehydration, so surgical closure of the ileostomy was performed. Biochemical analyses at the time of discharge returned the following results: urea: 21 mg/dL; creatinine: 0.4 mg/dL; sodium: 134 mmol/L; and potassium: 4.4 mmol/L.

3. DISCUSSION

Ileostomy-related complications include dehydration, wound infection, skin irritation, prolapsus, retraction, necrosis, stenosis, parastomal hernia, sepsis, and hemorrhage. Rates of ileostomy-related dehydration have been reported from 0.8% to 20% [4], while a 50% rate of dehydration-related renal failure has been reported. However, the exact rate of renal failure associated with ileostomy is not known because ileostomy patients are not closely followed after discharge and re-present to hospitals when severe dehydration due to excessive stoma discharge occurs [1].

In a retrospective study of 107 ileostomy patients, Beck-Kaltenbach, et al. [1] compared the glomerular filtration rate (GFR) after the ileostomy procedure and at ileostomy closure. Independent of the underlying disease and the length of time since ileostomy closure, patients' mean GFR were found to be significantly lower at ileostomy closure. Severe renal failure developed in 30% of cases (GFR<30 ml/dk). This indicates that ileostomy patients face a high risk of renal failure. That our cases had 4, 3 and 5 L of ileal drainage and that parenteral fluid replacement completely resolved the renal failure supports that dehydration is the main cause of this renal failure.

The literature review identified only a few cases of ileostomy-related metabolic alkalosis [5]. In all cases, metabolic alkalosis was connected to high-volume ileostomy drainage and excessive chlorine loss through ileal drainage. Our case 1 and case 3 developed metabolic alkalosis. The cases had high ileostomy drainage volumes, but the chlorine level in ileal drainage could not be measured.

Although normal ileal drainage includes low amounts of potassium (5–10 mmol/L) [6], the hyperkalemia observed in cases 2 and 3 shows that the small bowel excretes a very small amount of potassium. In cases when ileal drainage increases, this volume loss, in addition to the negligible quantity of potassium loss, contributes to hyperkalemia in ARF. In case 1, the absence of hyperkalemia might have been caused by renal potassium loss accompanying metabolic alkalosis. Isotonic replacement, such as in our cases, increases urine potassium excretion and helps correct hyperkalemia. As in case 2 in which potassium levels exceeded 6 mmol/L, additional treatment (e.g., insulin, glucose) might be necessary to introduce potassium to cells.

Various medications are used to decrease ileostomy volume and sodium loss. Codein phosphate and the less effective diphenoxylate hydrochloride, combined with atropine sulphate, decrease the loss of sodium, potassium, and water [7]. In rare cases, high ileal drainage, which occurred despite these precautions, was successfully treated with a glucose polymer electrolyte solution [8]. We also used diphenoxylate hydrochloride combined with atropine sulphate for high-volume ileostomy loss. However, high-output ileal drainage continued despite a partial decline in ileostomy volume, so ileostomy closure was considered.

In conclusion, ileostomy patients face the risk of life-threatening renal failure and electrolyte and acid-base changes due to dehydration. Therefore, clinicians should closely follow up with these patients and consider the possible complications of ileostomies. Additionally, the decision for and timing of ileostomy closure to resolve these complications should be made carefully.

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