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Single Case

Chronic Liver Disease Primarily Presenting with Motor Weakness by Intractable Hypokalemia with Combined Respiratory Alkalosis and Chronic Diarrhea: A Case Report

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Keywords
Liver cirrhosis · Portal hypertension · Hepatopulmonary syndrome · Diarrhea · Hypokalemia

Abstract
Introduction: Most patients with compensated cirrhosis remain asymptomatic. However, with the onset of decompensation, electrolyte and acid-base disturbances are frequent in patients with chronic liver disease, including hypokalemia. We encountered a case of chronic liver disease with portal hypertension, primarily presenting with motor weakness caused by intractable hypokalemia, hypoxia-associated respiratory alkalosis, and chronic diarrhea. ***Case Presentation:*** A 54-year-old male presented to the emergency department with motor weakness. He reported experiencing exertional dyspnea and watery diarrhea for the past 3 months, approximately ten times daily. Arterial blood gas analysis indicated hypoxia and hypocapnia compatible with chronic respiratory alkalosis. The transtubular potassium gradient was 1.69, and the aldosterone/renin ratio was 17.6 (ng/dL)/(ng/mL/h). The patient had a 30-year history of consuming 360–720 mL of 20% alcohol almost daily. Abdominal computed tomography revealed multiple regenerative and dysplastic nodules in the liver, splenomegaly, ascites, esophageal varices, and diffuse edematous wall thickening in the bowel, suggesting portal hypertensive enteropathy. Computed tomography of the lungs showed no specific abnormalities in the lungs, pleura, or thoracic wall. ***Conclusion:*** We

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present a case of liver cirrhosis complicated by intractable hypokalemia, respiratory alkalosis, portal hypertension, and chronic diarrhea. A 24-h urine analysis showed renal excretion levels of Na^+, K^+, and Cl^- at 6.0, 2.5, and 11.0 mmol, respectively, suggesting renal retention of these electrolytes. Meanwhile, the serum levels of Na^+, K^+, and Cl^- were 136, 1.8, and 98 mEq/L, respectively, indicating a preserved balance of sodium and chloride but not potassium. This case underscores the importance of clinicians considering both liver cirrhosis-associated hypoxia and chronic liver disease-induced chronic diarrhea as potential underlying causes, especially when more common causes of hypokalemia have been excluded.

Introduction

Electrolyte and acid-base disorders are common in patients with end-stage liver disease [1, 2]. In general, these disorders develop in patients with decompensated cirrhosis. Hypokalemia is expected in this setting [3] due to several potassium-depleting conditions, including hypoxia, a common cause of respiratory alkalosis in which intracellular K^+ translocation occurs, and chronic diarrhea resulting in gastrointestinal K^+ loss [4]. The prevalence of hypoxia in liver cirrhosis ranges from 10 to 30%, depending on the diagnostic criteria and study population [5]. Chronic liver disease with portal hypertension can lead to changes in the gastrointestinal system, including increased intestinal permeability, bacterial overgrowth, and bile acid malabsorption, all of which contribute to both osmotic and secretory chronic diarrhea [6, 7].

We recently encountered a patient with chronic liver disease who presented with motor weakness caused by intractable hypokalemia associated with respiratory alkalosis, chronic diarrhea, and portal hypertension. The hypokalemia remained refractory despite substantial potassium replacement for several days. Notably, the patient had no previous diagnoses of chronic liver disease.

Case Presentation

On June 17, 2024, a 54-year-old male patient (height, 168 cm; body weight, 52 kg) presented to our hospital's emergency department. His primary complaint was motor weakness, particularly in his lower extremities, which made him unable to stand or walk. He reported experiencing exertional dyspnea and watery diarrhea approximately ten times a day over the past 3 months.

Upon examination, he appeared cachectic but alert, with vital signs of blood pressure of 108/53 mm Hg, pulse rate of 80/min, respiratory rate 20–25/min at rest, and body temperature of 36.5°C. Physical examination revealed mild jugular venous distension and no abdominal tenderness. Bowel sounds were normally audible, and abdominal percussions were dull with minimal fluid shift. Palmar erythema and spider nevi were observed on the face and anterior chest wall. The liver was palpated firmly from the right subcostal margin using two finger breaths. Of note, the patient had a history of consuming 360–720 mL of 20% alcohol (equivalent to 1–2 bottles of soju, a Korean distilled spirit) almost daily for 3 decades.

Laboratory findings upon arrival at the emergency room are presented in Table 1. Prothrombin time (PT) was 18.7 s (1.53, INR), and partial thromboplastin time (PTT) was

48.2 s. Electrocardiography revealed a prolonged PR interval, wide QRS, and a flat T wave in the precordial leads. The albumin-corrected anion gap was 19.1 mEq/L, which suggested metabolic acidosis with a high anion gap. However, arterial blood gas analysis performed on the 1st and the 3rd hospital days was compatible with partially compensated respiratory alkalosis (Table 1) [8]. Computed tomography of the lungs revealed no specific abnormalities in the lungs, pleura, or thoracic wall.

Clinical Findings and Follow-Up Assessment

Enterocolitis was initially suspected. However, stool tests were negative for fecal leukocytes, stool occult blood, and parasites. In addition, no common intestinal pathogens, such as Salmonella and Shigella, were cultured. The plasma renin level was 1.64 ng/mL/h (reference range: 0.31–1.84 in the supine position). The plasma aldosterone level was 1.76 ng/dL (reference range: 6.8–24.9 in the supine position). The aldosterone/renin ratio [9] was 17.6 (ng/dL). The transtubular potassium gradient (TTKG) calculated as urine K^+ × plasma osmolality/plasma K^+ × urine osmolality [10] was 1.69. Cortisol and ACTH levels measured in the morning were 22.3 μg/dL (reference range: 0.7–22.6) and 17.6 pg/mL (reference range: 7.2–63.3), respectively. Abdominal computed tomography shown in Figure 1 revealed multiple regenerative and dysplastic nodules in the liver, splenomegaly, ascites, esophageal varices, and diffuse edematous wall thickening in the bowel, suggesting portal hypertensive enteropathy [11].

Therapeutic Intervention

Given that his diarrhea worsened with food intake, oral feeding was stopped and the patient started parenteral nutrition. Intravenous potassium administration was initiated at 80 mEq K^+ daily in 2 L of normal saline [4]. Although the diarrhea frequency decreased to 1–2 times per day, it was not fully resolved after several days of hospitalization. Potassium levels, shown in Figure 2, remained below the reference range despite daily administration of 80 mEq for 6 consecutive days. A week after admission, he was referred to a tertiary hospital in the hope of optimal care for cirrhosis, chronic diarrhea, and hepatopulmonary syndrome based on further assessments available at a tertiary hospital. On a follow-up phone call made 3 months later, he reported having diarrhea 2–3 times a day on a soft diet and was unable to walk without assistance. He reported that he had no more consumed alcohol since leaving the hospital. Thus, the symptoms could be alleviated to some extent with complete abstinence from alcohol and proper nutrition.

Discussion

Imbalances in sodium, chloride, and potassium levels are common electrolyte abnormalities in cirrhosis. However, no one has reported a case of chronic liver disease presenting primarily with motor weakness due to intractable hypokalemia with combined respiratory alkalosis and chronic diarrhea. The major challenge in this case was the severe hypokalemia that was refractory to potassium supplementation. The renal excretion of Na^+, K^+, and Cl^- over 24 h was 6.0, 2.5, and 11.0 mmol, respectively (Table 2), indicating that the kidneys compensated for the changes in these electrolytes. Serum Na^+, K^+, and Cl^- levels were 136, 1.8, and 98 mEq/L, respectively, indicating a well-maintained sodium and chloride balance but not for potassium. The pathophysiology of this electrolyte imbalance likely involves several factors. One primary consideration is the shift of potassium into the cells induced by alkalosis. Pulmonary vascular dilation assessments, such as contrast-enhanced echocardiography or radioactive lung perfusion studies [5], were not performed

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Table 1. Laboratory findings at the emergency room

	Result
WBC, /μL	14,360
RBC, /μL	3.7
Hb, g/dL	13.1
HCT (%)	35.6
PLT, /μL	213,000
Neutrophil (seg), %	83.7
Lymphocyte, %	7.3
Monocyte, %	8.0
Blood chemistry	
T protein, g/dL	8.5
Albumin, g/dL	3.4
Glucose, mg/dL	148
BUN, mg/dL	13.0
Creatine, mg/dL	0.73
Bilirubin, total, mg/dL	**4.2**
AST, U/L	**234**
ALT, U/L	**81**
ALP, U/L	**68**
Total calcium, mg/dL	8.8
Total amylase, U/L	75
LDH, U/L	381
CPK, U/L	4,437
Bilirubin, direct, mg/dL	**2.2**
CRP, mg/dL	7.8
Sodium, mEq/L	136
Potassium, mEq/L	1.8
Chloride, mmol/L	98
α-Fetoprotein, ng/mL	7.42

As the patient had not presented with liver dysfunction before this episode, the abnormal liver function tests were the initial clue for exploring chronic liver disease with portal hypertension. Abnormal liver function is indicated in bold.

in this case; therefore, classical hepatopulmonary syndrome could not be confirmed [2, 5]. However, the patient exhibited hypoxia, tachypnea, and hypocapnia, with respiratory alkalosis. Given the absence of significant abnormalities on lung CT, we believe that hypoxia related to liver cirrhosis plays a critical role in the development of respiratory alkalosis. Although stool electrolyte levels were not assessed, previous studies have indicated that diarrheal fluid in similar cases often contains high potassium concentrations (130–170 mEq/L) and low sodium levels (4–15 mEq/L) driven by electrochemical gradients that promote potassium loss through active chloride secretion and potential secondary hyperaldosteronism [12]. Therefore, we believe that chronic diarrhea in the

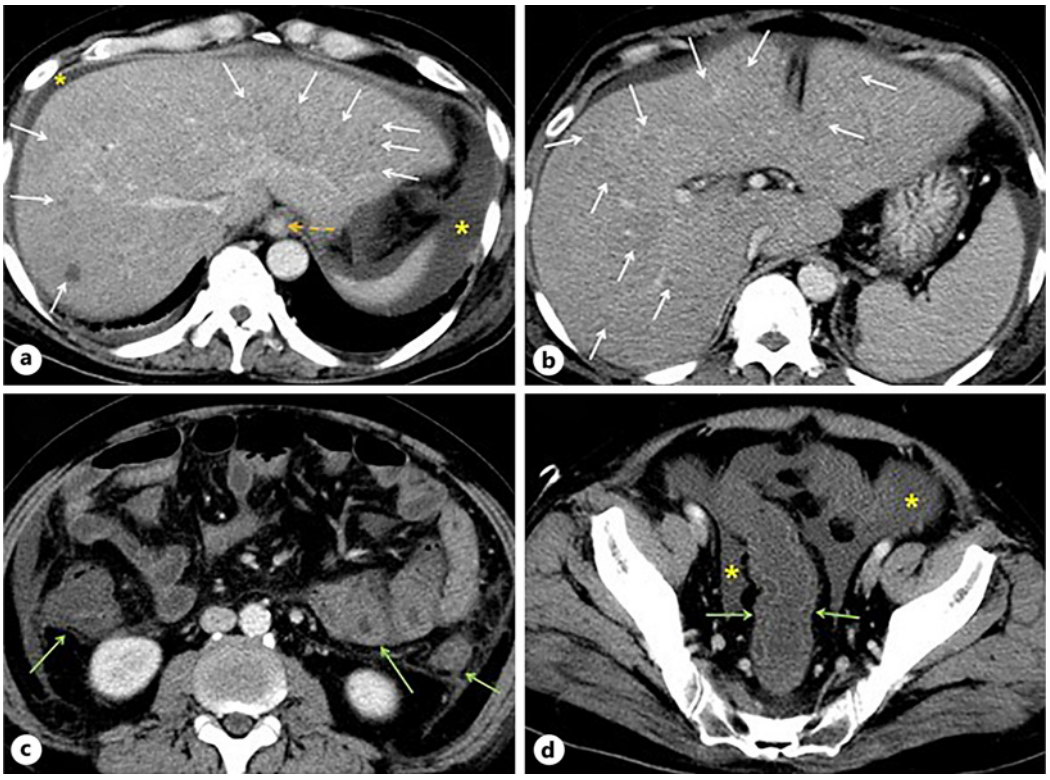

Fig. 1. Contrast-enhanced abdominal CT images of a 52-year-old man. **a**, **b** Axial CT images show mild hypertrophy of the caudate lobe and lateral segments of the left lobe. Multiple low-attenuating small nodules (white arrows) are scattered in the liver. Circumferential enhancing lesions in the esophageal wall suggest the presence of esophageal varices (dashed arrow). Additionally, mild splenomegaly and ascites (asterisks) are present. These findings indicate liver cirrhosis with multiple regenerative and dysplastic nodules, along with the presence of portal hypertension. **c** Axial CT image reveals diffuse edematous wall thickening in the ascending and descending colon and the small bowel (green arrows). **d** Diffuse edematous wall thickening is also noted in the sigmoid colon (green arrows). Pelvic ascites is present (asterisks). The diffuse edematous wall thickening in this patient with underlying liver cirrhosis and portal hypertension suggests the possibility of portal hypertensive enteropathy.

current patient may have contributed to the significant depletion of potassium stores in the body. Accurate identification of the causes of diarrhea and hypoxia is crucial for effective management. However, the diagnosis of chronic diarrhea due to portal hypertension and cirrhosis-associated hypoxia can be difficult without clear clinical suspicion. In this case, the presenting symptoms of muscle weakness and hypokalemia were ultimately attributed to chronic liver disease-associated respiratory alkalosis and diarrhea. Conversely, hypoxia and hypokalemia may also affect liver metabolism. These findings underscore the potential for organ crosstalk between the liver and lungs and between the liver and gut [13, 14].

Limitations

We have not performed an intensive test for Whipple's and celiac diseases. However, the prevalence of both Whipple's and celiac disease is scarce in Korea [15, 16]. Alcoholic myopathy [17] with vitamin deficiency may have contributed to the muscle weakness observed in this case. Another limitation is that we did not perform a

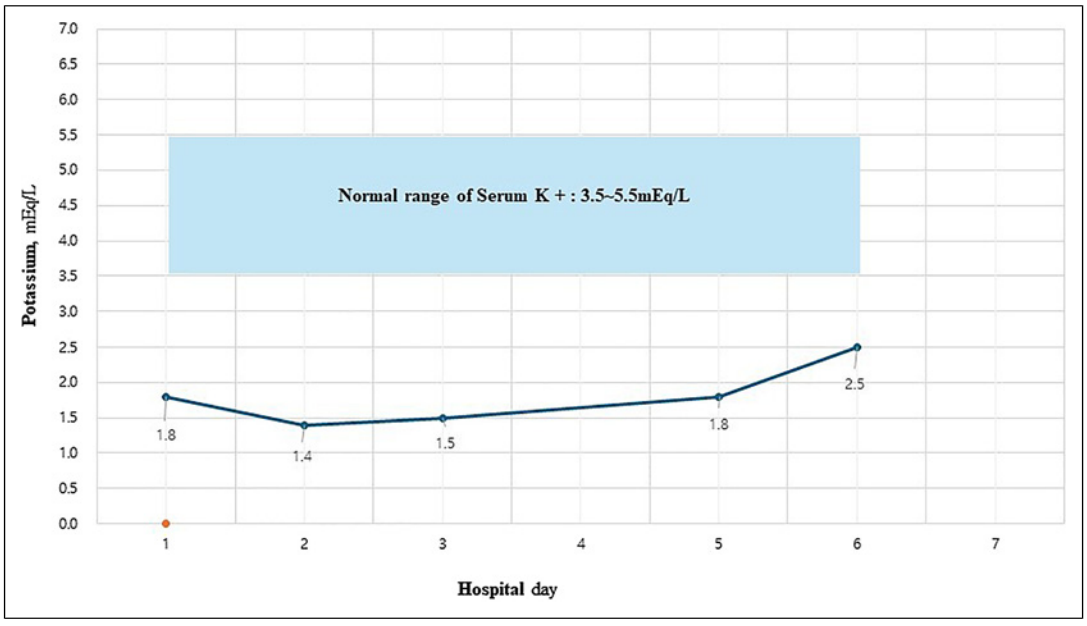

Fig. 2. Sequential measurement of serum potassium levels. During the observation period, 80 mEq potassium in 2 L normal saline was administered intravenously daily. Serum potassium levels remained below the normal range of 3.5–5.5 mEq/L.

Table 2. Arterial blood gas analysis

ABGA	Hospital day 1	Hospital day 3
pH	7.534	7.453
PCO_2, mm Hg	23.9	30.9
PO_2, mm Hg	57.7	71.3
HCO_3, mmol/L	20.4	21.8
$ctCO_2$, mmol/L	21.1	22.8
BE (ecf)	−2.4	−2.3
BE	0.1	−0.8
HCO_3std, mmol/L	24.4	23.7
O_2SAT, %	93.2	95.1

The ABGA results are compatible with partially compensated respiratory alkalosis.

colonoscopy to rule out inflammatory bowel disease, microscopic colitis, or ischemic colitis.

In conclusion, we present a case of liver cirrhosis complicated by intractable hypokalemia, respiratory alkalosis, portal hypertension, and chronic diarrhea. These results highlight the importance of considering both cirrhosis-associated hypoxia and portal hypertension-induced chronic diarrhea as potential underlying causes, especially when the more common causes of hypokalemia in chronic liver disease have been excluded. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000544099).

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Statement of Ethics

The case report protocol was reviewed and approved by the Public Institutional Review Board of the Ministry of Health and Welfare of South Korea (http://irb.or.kr/menu02/summary.aspx; approval No.: P01-202412-01-022). Written informed consent was obtained from the patient to publish the details of their medical case and any accompanying images.

Conflict of Interest Statement

The authors declare that they have no competing interests.

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Author Contributions

Conceptualization and edits: S.-Y.H. Draft of the manuscript (partial): Y.-O.J. and N.-S.B. Chest CT reading: K.-H.L. Graphic arts and production of figures: E.-M.J. Supervision: J.-I.I. All authors read and approved the final manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

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