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Hypernatremia in Decompensated Cirrhosis—Is Lactulose Boon or Bane?

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Abstract

Background: Hyponatremia is common in cirrhosis and is associated with high mortality. In contrast, hypernatremia is less frequent and is typically caused by hypotonic fluid losses due to osmotic diuresis, increased insensible water losses, and reduced water intake, often associated with encephalopathy. Lactulose, commonly used for the management of hepatic encephalopathy (HE), sometimes paradoxically worsens the encephalopathy due to the development of dehydration and hypernatremia. Prompt identification of the cause of hypernatremia and slow correction of serum sodium is associated with improved short-term survival. **Case Presentation:** We report a case of acute on chronic liver failure with HE, who developed hypernatremia during hospital stay due to lactulose-induced diarrhoea that worsened his sensorium further. After adjusting the dose of lactulose and correcting hypernatremia, his encephalopathy improved, and he was subsequently discharged. **Conclusion:** Lactulose may cause hypernatremia, especially in cirrhosis patients with hepatic encephalopathy. Early identification of hypernatremia, along with its cause and slow correction of sodium, is associated with improved short-term survival in decompensated cirrhosis.

Keywords

Lactulose, Decompensated Cirrhosis, Hypernatremia

1. Introduction

Hyponatremia is the most frequently observed electrolyte disturbance in decompensated cirrhosis, with a prevalence of approximately 50% [1]. Hypernatremia in cirrhosis, on the other hand, is an uncommon condition, with serum sodium > 145 mmol/L and is reported in up to 4% of patients, particularly those receiving

vaptan therapy. Moderate to severe hypernatremia (sodium > 150 mmol/L) is even less frequent, seen in only 0.4% of patients on the transplant waiting list [2]. In cirrhosis, hypernatremia is linked to encephalopathy and a higher mortality rate. Lactulose has been commonly used in the treatment and prevention of hepatic encephalopathy [3]. In general, lactulose is considered safe. Like other laxatives, it can cause dehydration and various electrolyte abnormalities, including hypernatremia [4] [5]. These disturbances, in turn, can paradoxically worsen HE and lead to further complications. Prompt identification of the cause of hypernatremia and slow correction is associated with improved short-term survival in decompensated cirrhosis patients. We are reporting a rare case of hypernatremia in decompensated cirrhosis with hepatic encephalopathy.

2. Case Presentation

A 42-year-old gentleman with past medical history of chronic pancreatitis with Type 3c diabetes mellitus presented to our emergency with jaundice for 2 weeks, abdominal distension for 1 week and worsening sensorium for 3 days. He had a history of chronic alcohol abuse and consumed alcohol in cirrhogenic doses, with the last alcohol intake 1 month ago. On admission, he had deep icterus, gross abdominal distension, hepatic encephalopathy (West Haven grade III) and 86% SpO_2 on room air. On evaluation, he had anemia (8.8 gm/dl), thrombocytopenia 70,000 cells/mm^3, leukocytosis (12,200 cells/mm^3), deranged liver function tests (Total bilirubin-14 mg/dl, Direct bilirubin-8.4 mg/dl, AST/ALT-70/15 U/L, serum albumin-2.5 g/dl and INR-2.5). His serum sodium was 134 mEq/L, Urea-30 mg/dl, Cr-0.9 mg/dl. Ultrasound of the abdomen was suggestive of chronic liver disease with features of portal hypertension and gross ascites. He also had spontaneous bacterial peritonitis (SBP) with ascitic fluid total cells being 1320/mm^3 with 70% neutrophils. Diagnosis of acute on chronic liver failure with hepatic encephalopathy and SBP was made (MELD-28, CLIF-C ACLF score 55). After prognostication, he started oxygen supplementation (2 L/min), intravenous antibiotics (Ceftriaxone 1 gm twice daily), 20% Albumin infusion, Enteral nutrition (Branched-chain amino acid based) and anti-hepatic encephalopathy measures (Lactulose syrup 20 gm every 6 hourly and Rifaximin 550 mg twice daily) via nasogastric tube. He started showing slow and gradual improvement in his sensorium. On day 4 of admission, he developed relative hypernatremia (Na-151 mmol/L) and had a dip in his sensorium. The free water deficit (FWD) was calculated using the formula:

$$\text{FWD} = \text{Total Body Water} \times ((\text{Serum Sodium}/140) - 1).$$

In patients with cirrhosis, estimating total body water can be challenging due to fluid shifts, so it was conservatively approximated as 0.4 to 0.6 × body weight (kg). The patient's FWD was estimated to be 2.6 Liters. To minimize the risk of cerebral edema and osmotic demyelination syndrome, a slow correction over 48 hours was planned. Intravenous 5% dextrose was administered at 40 mL/hour, and 50 mL of water was given via Ryle's tube every 4 hours. Over the next 24

hours, the patient's hypernatremia worsened, with serum sodium rising to 158 mmol/L. At that time, he was experiencing eight bowel movements per day. He had no evidence of gastrointestinal bleeding, polyuria, or excessive intravenous sodium administration. Therefore, gastrointestinal losses were considered a possible contributing factor to his hypernatremia. As a result, lactulose dose was reduced to 10 grams twice daily, and free water replacement was continued as deemed appropriate. Following these changes, stool frequency decreased to three times per day within 48 hours. During the hospital stay, both serum sodium levels and hepatic encephalopathy improved gradually, with sodium normalizing to 142 mmol/L and encephalopathy improving to grade I by day 10. He was discharged in a hemodynamically stable condition on day 11. At his two-week follow-up, the patient showed complete resolution of hepatic encephalopathy, noticeable improvement in ascites and was actively participating in an alcohol de-addiction program.

3. Discussion

Hypernatremia is defined as serum sodium concentration > 145 mmol/L and it represents a state of total body water deficiency absolute or relative to total body sodium [6]. Mortality in patients with hypernatremia reaches 20% to 60% and varies according to comorbidities, associated illness and whether hypernatremia is present at admission or acquired during hospitalization [7]. Hypervolemic hyponatremia is the most common sodium abnormality in cirrhosis, with a prevalence close to 50% in patients with ascites. Hypernatremia is uncommon in decompensated cirrhosis [1]. According to a prospective multicentre study, the prevalence of hypernatremia in cirrhosis was 5 percent [8]. In a retrospective analysis, the prevalence of hypernatremia in decompensated cirrhosis patients admitted to medical ICU with HE was 53% and was associated with high mortality rate [9]. Probable causes of hypernatremia in decompensated cirrhosis include increased insensible water losses (fever, sepsis, hyperventilation), impairment of water intake due to encephalopathy, the use of osmotic cathartics with hypotonic enteric losses, gastrointestinal bleeding (due to increased blood urea nitrogen due to the absorption of nitrogenous compounds from the gastrointestinal tract that results in osmotic diuresis) and rarely administration of parenteral solutions with high sodium concentration. In our index patient, the likely cause of hypernatremia could be osmotic laxative (Lactulose). Few studies available in literature show that hypernatremia is rare in decompensated cirrhosis and lactulose may be the cause of hypernatremia [4] [5]. The acute onset of hypernatremia leads to an abrupt shrinkage of the brain due to intracellular dehydration that can lead to worsening or new onset altered sensorium, seizures, coma, hypertonia, high fever, intracranial haemorrhages and thrombosis of the dural sinuses and demyelination syndromes [1]. In our case, after an initial improvement in sensorium with anti-hepatic coma measures, the patient's sensorium worsened due to development of hypernatremia secondary to lactulose induced diarrhoea. Hypernatremia in decompensated cirrhosis is associated with high mortality. In one retrospective anal-

ysis of patients admitted with end stage liver disease, patients with hypernatremia had high mortality of 87% compared with 60% in patients without hypernatremia [10]. Early identification of hypernatremia along with its cause and slow correction (6 - 8 meq/day) of sodium, is associated with improved short-term survival in decompensated cirrhosis, as seen in our patient. A key limitation of this case report is that the findings are derived from a single patient, restricting broader applicability. Although the temporal relationship between lactulose-induced diarrhea and hypernatremia was evident, the precise contribution of lactulose compared to other potential factors, such as insensible fluid losses or decreased oral intake, remains speculative. This is because key diagnostic measures, such as stool osmolality or urinary sodium excretion, were not performed to conclusively determine the underlying mechanism.

4. Conclusion

Hypernatremia is rare in cirrhosis and is associated with a high mortality rate. Lactulose may cause hypernatremia, especially in cirrhosis patients with hepatic encephalopathy. Early identification of hypernatremia, along with its cause and slow correction of sodium, is associated with improved short-term survival in decompensated cirrhosis.

Author Contributions

SD—Conceptualization, Methodology, Writing-Original draft preparation;
GM—Writing-Reviewing and Editing;
RR—Data curation;
SA—Supervision.

Ethics Approval and Consent to Participate

Prior ethics approval was taken.

Consent for Publication

Written informed consent to publish was obtained from the patient.

Conflicts of Interest

All authors have no financial and non-financial competing interests.

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Abbreviations

AST—Aspartate transferase

ALT—Alanine transferase

MELD—Model for end stage liver disease

CLIF—Chronic liver failure

ACLF—Acute on chronic liver failure