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OSMOTIC DEMYELINATION SYNDROME IN CIRRHOTIC PATIENTS: A RISK OF OVERCORRECTION

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ABSTRACT

Background: Osmotic demyelination syndrome (ODS), though rare, is a potentially fatal neurological disorder arising from rapid correction of chronic hyponatremia. Patients with decompensated liver cirrhosis are especially vulnerable due to their predisposition to sodium imbalance and altered cerebral osmoregulation.This study aims to explore the risk of ODS in cirrhotic patients with hyponatremia and highlight the importance of cautious sodium correction.**Methods:** A prospective observational study was conducted over 18 months. Cirrhotic patients admitted with acute decompensation were assessed for hyponatremia severity and monitored throughout hospitalization. Daily serum sodium levels and clinical signs of encephalopathy or neurological deterioration were recorded. Patients undergoing rapid sodium correction (>8 mEq/L/24 hr) were closely evaluated for symptoms suggestive of ODS. **Results:** Among 71 cirrhotic patients, 51 (71.8%) had hyponatremia. Of these, 13 (18.3%) had severe hyponatremia (<125 mEq/L). Sodium correction >8 mEq/L/day occurred in 5 patients, all from the severe hyponatremia group. Two patients exhibited clinical signs suggestive of central pontine myelinolysis—progressive dysarthria, quadriparesis, and altered mental status. Both had serum sodium corrected by >10 mEq/L within 24 hours using hypertonic saline. One succumbed to complications; the other showed partial recovery.**Conclusion:** Cirrhotic patients with severe hyponatremia are highly susceptible to osmotic demyelination when serum sodium is corrected too rapidly. Clinicians must adopt a controlled correction approach (<6–8 mEq/L/day), particularly in the presence of chronic sodium derangement, to prevent iatrogenic neurological injury.

INTRODUCTION

Osmotic demyelination syndrome (ODS), particularly central pontine myelinolysis (CPM), is a devastating neurological condition precipitated by rapid correction of chronic hyponatremia. In patients with liver cirrhosis, the risk is heightened due to multiple factors, including altered blood-brain barrier permeability, persistent neuroinflammation, and cerebral edema from ammonia accumulation and sodium imbalance [1–3]. Though uncommon, ODS poses serious clinical and prognostic implications, particularly in transplant candidates or those receiving hypertonic therapy.

Cirrhotic hyponatremia typically develops via non-osmotic release of antidiuretic hormone (ADH), leading to disproportionate water retention relative to sodium, resulting in dilutional hyponatremia [1,2,4]. Neurological consequences range from mild cognitive impairment to coma. However, rapid correction of long-standing hyponatremia—particularly >8–10 mEq/L/day—can lead to cellular dehydration and myelinolysis, especially in pontine neurons [1,5,6].

This study investigates the incidence and characteristics of ODS in hyponatremic cirrhotic patients admitted to a tertiary center in South India, with a focus on the role of rapid sodium correction.

MATERIALS AND METHODS

1. Study Design and Setting

This prospective observational study was conducted at Sri Siddhartha Medical College and Hospital, Tumkur, over a period of 18 months. The study was approved by the Institutional Ethics Committee. Written informed consent was obtained.

2. Inclusion Criteria

Adults (≥18 years) with ultrasound and lab-confirmed liver cirrhosis admitted for acute decompensation were included.

3. Exclusion Criteria

Patients with pre-existing CKD, cardiac failure, diuretic-induced hyponatremia, or grade IV hepatic encephalopathy were excluded to minimize confounding [6,8].

4. Data Collection

Serum sodium levels were measured daily. Hyponatremia was classified as:

- Mild: 130–134 mEq/L
- Moderate: 125–129 mEq/L
- Severe: <125 mEq/L

Sodium correction was monitored closely. Any increase >8 mEq/L/24 hrs was flagged. Neurological status was evaluated daily. MRI was advised for suspected ODS.

RESULTS:

Patient Distribution

Out of 71 cirrhotic patients:

- 51 (71.8%) had hyponatremia
- 13 (18.3%) had severe hyponatremia (<125 mEq/L)

Sodium Correction Trends

Among the 13 with severe hyponatremia:

- 5 received hypertonic saline
- 3 had correction >8 mEq/L/day
- 2 had correction >10 mEq/L/day

Clinical Manifestations

Both patients who exceeded correction thresholds developed progressive dysarthria, quadriparesis, and altered sensorium by day 3. One of these patients succumbed to multi-organ failure; the other partially recovered with supportive care. MRI was suggestive (though not conclusive) of pontine demyelination.

No signs of ODS were noted in patients with slower correction (<6 mEq/L/day), in agreement with previous findings [1,5,9].

DISCUSSION

ODS results from osmotic stress following rapid correction of chronic hyponatremia. In cirrhotics, brain osmoregulation is already compromised due to ammonia toxicity,

neuroinflammation, and impaired blood-brain barrier integrity [1,3,4,6]. Overcorrection leads to cellular dehydration and myelin damage, primarily in the central pons [1,2].

Risk factors include:
- Chronic severe hyponatremia
- Malnutrition
- Liver failure
- Hypokalemia
- Use of hypertonic saline without intensive monitoring [1,5,10]

Guidelines suggest limiting correction to <6–8 mEq/L/day to reduce ODS risk [1,6]. Our findings underscore these recommendations, as both patients who exceeded this limit developed neurological deterioration, while those corrected slowly had no such issues.

The literature further supports that correction of sodium levels, although necessary, does not always improve survival outcomes and may, if mishandled, cause irreversible harm [2,5,7]

CONCLUSION

ODS is a rare but potentially fatal complication of rapid sodium correction in cirrhotic patients. It is preventable with careful fluid and electrolyte management, especially in those with severe chronic hyponatremia. A controlled correction strategy (<6–8 mEq/L/day) and close ICU monitoring are essential for safety.

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