Decompensated NASH Cirrhosis with Symptomatic Hyponatremia and Endocrine Comorbidities: A Clinical Pharmacist–Led Intervention

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Abstract:

This case report describes the acute decompensation of a 42-year-old male with end-stage nonalcoholic steatohepatitis (NASH)–related cirrhosis complicated by chronic hypothyroidism and hypoadrenalism. The patient presented with progressive jaundice, profound fatigue, and life-threatening electrolyte disturbances, including severe hyponatremia and hypokalemia, with a documented history of hyponatremia-related seizures. Management required strict fluid restriction, albumin infusion, electrolyte correction, infection prophylaxis, and neurological protection. Critical review of inpatient and discharge management revealed systemic gaps, including delayed diuretic initiation and lack of clarity regarding lactulose titration. Clinical pharmacist intervention was pivotal in preventing high-risk drug–drug interactions, particularly between levothyroxine and calcium supplementation, and in optimizing hepatic encephalopathy prophylaxis. The patient was discharged with a MELD score of 24 and remains active on the deceased donor liver transplantation waitlist. This case highlights the indispensable role of clinical pharmacy services in complex hepatology care.

Keywords—NASH cirrhosis; Hyponatremia-related seizures; Clinical pharmacy; MELD score; Hepatic encephalopathy; Drug–drug interaction

INTRODUCTION

Nonalcoholic steatohepatitis (NASH) has emerged as one of the leading causes of chronic liver disease and cirrhosis worldwide and is a rapidly increasing indication for liver transplantation. Decompensated cirrhosis is frequently associated with ascites, electrolyte disturbances, hepatic encephalopathy, and increased short-term mortality, as reflected by elevated Model for End-Stage Liver Disease (MELD) scores. Hyponatremia is a particularly poor prognostic marker and is associated with neurological complications, including seizures.

This report describes a patient with decompensated NASH cirrhosis who presented with symptomatic hyponatremia in the presence of coexisting hypothyroidism and hypoadrenalism. These endocrine disorders significantly complicated fluid and electrolyte homeostasis. The case emphasizes the pathophysiological mechanisms, therapeutic challenges, and the critical role of the clinical pharmacist in optimizing medication safety and discharge planning in a pre-transplant patient.

Patient Information

A 42-year-old male (42 years, 9 months, and 4 days) was admitted on **6 August 2025** with complaints of progressive yellowish discoloration of the eyes, profound tiredness, decreased appetite, and sleep disturbance for five days.

Past Medical History

The patient was a known case of **decompensated NASH-related cirrhosis** with portal hypertension. He had a previous episode of **fundal variceal bleeding**, which was managed with endoscopic glue

therapy. The neurological risk was significant due to a documented history of **hyponatremia-related seizures**.

The patient also had chronic endocrine comorbidities:

- **Hypothyroidism**, treated with levothyroxine (Eltroxin)
- **Hypoadrenalism**, treated with hydrocortisone (Hisone)

Clinical Examination and Investigations

On examination, the patient had **moderate ascites**. The initial MELD score was **23**, which increased to **24** during hospitalization. Laboratory investigations revealed severe hepatic dysfunction, coagulopathy, electrolyte derangements, and thrombocytopenia.

Table 1: Laboratory Parameters During Hospitalization

Parameter	Reference Range	On Admission	Peak / Worst Value	At Discharge
Total Bilirubin (mg/dL)	0.3–1.2	Elevated	Markedly elevated	Improved
INR	0.8–1.2	2.2	2.2	Slight improvement
Serum Sodium (mmol/L)	135–145	Low	Critically low	Improved
Serum Potassium (mmol/L)	3.5–5.0	Low	Low	Normalized
Platelet Count ($\times 10^3/\mu L$)	150–450	Severely reduced	Severely reduced	Persistently low
MELD Score	—	23	24	24

In-Hospital Management

Electrolyte and Volume Management

The primary therapeutic objective was the safe correction of symptomatic hyponatremia, given the patient's prior seizure history. Management included **strict fluid restriction** and **intravenous albumin infusion** to improve effective circulating volume. Serum sodium levels gradually improved without neurological complications.

Hypokalemia was corrected using a **potassium-rich diet** and **potassium chloride syrup (POTKLOR)**, resulting in normalization of serum potassium levels.

Infection and Neurological Prophylaxis

Given the presence of ascites and decompensated cirrhosis, **intravenous antibiotics (cefotaxime/cefixime)** were initiated for spontaneous bacterial peritonitis prophylaxis. Due to the history of hyponatremia-related seizures, **levetiracetam (LEVIPIL)** was prescribed as secondary seizure prophylaxis.

Portal Hypertension Management

Upper gastrointestinal endoscopy revealed residual low-risk esophageal varices. **Carvedilol** was initiated for prophylaxis against variceal rebleeding.

Discharge Plan

At discharge, the patient was prescribed **lactulose syrup and rifaximin** for hepatic encephalopathy prophylaxis. Diuretic therapy with **spironolactone and furosemide** was planned for initiation during follow-up. The patient continues to remain active on the **deceased donor liver transplantation (DDLT) waitlist**.

Discussion

Hyponatremia in advanced cirrhosis is predominantly dilutional and results from non-osmotic vasopressin release secondary to systemic vasodilation and perceived hypovolemia. In this patient, the presence of prior hyponatremia-related seizures necessitated cautious correction with strict fluid restriction to ensure neurological safety.

Concomitant hypokalemia further increased the risk of hepatic encephalopathy by enhancing renal ammoniagenesis. Prompt correction of potassium was therefore essential.

Role of the Clinical Pharmacist

A comprehensive medication review by the clinical pharmacist identified two critical concerns:

Drug–Drug-Interaction

The concurrent administration of

levothyroxine (Eltroxin) *and* ***calcium supplementation (Shelcal)*** *posed a risk of reduced levothyroxine absorption. The pharmacist ensured patient counseling and mandated a* ***minimum 4-hour separation*** *between the two agents, preventing therapeutic failure of hypothyroidism management.*

Therapeutic-Optimization

The discharge plan lacked clarity regarding ***lactulose titration****. The pharmacist counseled the patient to titrate lactulose to achieve* ***2–3 soft bowel movements per day****, rather than adhering to a fixed dose. Guidance was also provided regarding the appropriate* ***spironolactone-to-furosemide ratio*** *to counteract secondary hyperaldosteronism.*

Conclusion

This case highlights the complexity of managing decompensated NASH cirrhosis with symptomatic hyponatremia in the presence of endocrine comorbidities. The patient's high MELD score and prior seizure history necessitated meticulous electrolyte management and neurological prophylaxis. Clinical pharmacist involvement was crucial in preventing serious drug–drug interactions and ensuring effective hepatic encephalopathy prophylaxis. Integration of clinical pharmacy services is essential for optimizing outcomes in high-risk hepatology patients awaiting liver transplantation.

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