



Electrolyte Disturbances in Chronic Liver Disease: Correlation of Serum Sodium, Potassium, and Magnesium Levels with Disease Severity

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Abstract

Background: Electrolyte imbalances are common in cirrhosis and may indicate disease progression. While magnesium has been explored as a marker of severity, limited studies evaluate the combined correlation of sodium, potassium, and magnesium with chronic liver disease severity. **Aims:** To estimate serum sodium, potassium, and magnesium levels in cirrhosis patients, and to assess their correlation with the severity of liver disease as measured by Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores. **Methods:** This cross-sectional study enrolled 121 cirrhotic patients. Serum electrolyte levels were measured and correlated with MELD and CTP scores. Statistical tests were done using SPSS v26. **Results:** Magnesium levels declined significantly across worsening CTP classes ($p = 0.012$), and inversely correlated with MELD ($r = -0.336$, $p < 0.001$). However, sodium ($p = 0.22$) and potassium ($p = 0.792$) levels showed no significant variation across CTP classes. Magnesium also negatively correlated with bilirubin ($r = -0.258$, $p = 0.004$) and positively with albumin ($r = 0.239$, $p = 0.008$). **Conclusion:** Magnesium correlates significantly with cirrhosis severity, suggesting its utility as a prognostic marker. Sodium and potassium did not show a consistent association. Routine evaluation of magnesium may enhance the assessment of cirrhosis progression.

Keywords: Cirrhosis, CTP, Electrolytes, Magnesium, MELD, Potassium, Sodium

1. Introduction

Cirrhosis is a long-term liver condition where normal liver tissue is gradually replaced by scar tissue, leading to reduced liver function and a host of complications. One often overlooked yet crucial aspect of this disease is the disturbance in electrolyte balance—especially involving magnesium, sodium, and potassium^{1,2}.

Magnesium, for instance, is vital for energy production and maintaining cellular health. When magnesium levels drop, which is common in cirrhosis, it can worsen liver damage by increasing oxidative stress and impairing mitochondrial function^{3,4}. Similarly, low sodium (hyponatremia) and abnormal potassium levels (either too high or too low) are frequently seen in patients with advanced liver disease. These imbalances can lead

to serious complications such as hepatic encephalopathy, ascites, and even increased risk of death^{1,5}.

To better understand how sick a patient with cirrhosis is and to predict their chances of survival, clinicians often use scoring systems like the CTP and the Model for End-Stage Liver Disease (MELD) scores^{6,7}. These scores are calculated using various lab values and clinical symptoms, but they do not typically include electrolyte levels.

This study aims to explore whether sodium, potassium, and magnesium levels can provide additional insights into the severity of liver disease. By correlating these electrolytes with MELD and CTP scores, we hope to better understand their role in the clinical picture—and whether they could serve as low-cost, accessible markers for prognosis in cirrhosis.

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2. Aim and Objectives

1. To estimate serum sodium, potassium, and magnesium levels in patients with chronic liver disease.
2. To correlate individual electrolyte levels with the severity of liver disease as measured by CTP and MELD scores.

3. Review of Literature

Electrolyte imbalances are a common and often underappreciated aspect of chronic liver disease. Among the many metabolic changes seen in cirrhosis, disturbances in magnesium, sodium, and potassium are particularly important—not just for clinical management but also for understanding how far the disease has progressed.

3.1 Magnesium: The Overlooked Indicator

Magnesium plays a quiet but powerful role in liver health. It's involved in hundreds of enzymatic reactions, keeps muscle and nerve functions stable, and helps control oxidative stress—something cirrhotic livers struggle with. Research has shown that patients with liver disease, especially those with alcohol use or poor nutrition, often have low magnesium levels¹⁻³.

3.2 Sodium and Potassium: Clinically Crucial, Prognostically Limited

Sodium and potassium levels are closely monitored in patients with cirrhosis—for good reason. Hyponatremia (low sodium) is common in decompensated liver disease and is associated with fluid retention, hepatic encephalopathy, and even death^{4,5}. It's particularly worrying in patients with acute-on-chronic liver failure, where it can signal a sudden downturn in health.

Potassium levels also matter. Hypokalemia (low potassium) often shows up in patients on diuretics, while hyperkalemia (high potassium) may indicate renal impairment - a frequent companion to advanced cirrhosis⁶.

3.3 Dyselectrolytemia in Cirrhosis

More importantly, these low levels are not just random findings—they tend to correlate with how severe the liver damage is. Multiple studies, including work by Wu *et al.*⁷ and Chavan *et al.*⁸, found that falling magnesium levels went hand-in-hand with rising MELD and

CTP scores—both widely used indicators of disease progression. Veena *et al.*⁹ demonstrated a significant negative correlation between serum magnesium levels and cirrhosis severity as demonstrated by CTP scores.

Other researchers like Rani *et al.*¹⁰ and Das *et al.*¹¹ backed this up, showing that magnesium levels were consistently lower in patients with more advanced cirrhosis. Gowda *et al.*¹² demonstrated that serum magnesium levels were lower in alcoholic hepatitis and cirrhosis patients. Koivisto's¹³ study even highlighted how magnesium deficiency could be linked to muscle cramps, fatigue, and poor quality of life in terminal liver disease.

What makes magnesium stand out is its relative stability. While sodium and potassium levels may fluctuate due to medications or fluid shifts, magnesium seems to offer a more reliable signal of underlying liver dysfunction. Broader reviews on trace element imbalances also consistently list magnesium as one of the most depleted nutrients in cirrhotic patients^{14,15}.

Some trials have even explored the therapeutic potential of magnesium supplementation. For instance, Poikolainen and Alho¹⁶ conducted a randomised study in alcoholics and found that magnesium therapy could help reduce fatigue and neuromuscular symptoms. Another study by Gullestad *et al.* compared oral vs. IV magnesium and suggested that IV might be more effective in advanced disease¹⁷.

And there's more — even in Non-Alcoholic Fatty Liver Disease (NAFLD), low magnesium levels have been linked to worse outcomes, suggesting its potential as a pan-hepatic marker of metabolic distress¹⁸.

Dumea *et al.*¹⁹ even found that potassium disturbances could help predict complications like variceal bleeding. However, these electrolytes are often influenced by external factors—diet, medications, fluid balance—which makes them less reliable as long-term markers of disease severity. As Rahelić *et al.*²⁰ pointed out, while they're essential in day-to-day management, their diagnostic value as stable predictors is limited compared to magnesium.

3.4 The Bigger Picture: Micronutrients and Liver Disease

Looking beyond these three electrolytes, the literature suggests that trace element deficiencies are widespread in chronic liver disease. Studies from Llibre-Nieto², Kar

et al.¹⁴, and others found that patients with cirrhosis often have simultaneous deficits in zinc, copper, selenium, and magnesium — all of which can worsen immune function, delay tissue repair, and increase oxidative stress¹⁵.

All these findings point to a growing consensus: magnesium is not just a marker, but a meaningful contributor to patient outcomes. And while sodium and potassium still have an essential role in acute care, magnesium might offer a more stable window into the liver's long-term health.

4. Materials and Methods

This cross-sectional study was carried out in the Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai, for a period of 6 months from June 2024 to November 2024. Data was collected from patients with confirmed cases of chronic liver diseases admitted to wards and emergencies under the Department of Internal Medicine, RGGGH.

- Informed consent was taken from all cases.
- A detailed history of the duration of the illness and other significant medical illnesses, and a clinical examination was done.
- Basic investigations, including Complete Blood Count (CBC), Renal Function Test (RFT), Liver Function Test (LFT), PT-INR and estimation of serum sodium, potassium and magnesium levels, were carried out in all the patients included in the study.
- MELD score and Child-Pugh class were assessed for the patients.

4.1 Sample Size Calculation

According to the study⁹,

Prevalence of hypomagnesemia among liver cirrhosis patients = 71.8% Level of significance (P) = 71.8%

Absolute precision (d) = 8% Sample size (N) = Z^2PQ/d^2

$Q = 100 - 71.8 = 28.2$

$N = (1.96 \times 1.96 \times 71.8 \times 28.2) / (8 \times 8) = 121$ Sample Size = 121

4.2 Study Population

Patients >18 years of age presenting to the emergency and OPD diagnosed with chronic liver diseases after obtaining informed consent.

Inclusion Criteria

Patients of both sexes diagnosed with chronic liver diseases.

- Patients above 18 years of age.
- Patients with informed consent.

Exclusion Criteria

- Patients taking or had previously taken any treatment with magnesium and or potassium supplements in the last 6 months.
- Patients with chronic kidney diseases.
- Patients on lithium therapy, metformin, insulin, and diuretics.
- Patients with hypothyroidism.
- Patients with chronic diarrhoea.

4.3 Statistical Analysis Plan

- Patients were categorised according to the study sample, and results were analysed by SPSS software.
- Characteristics of the study participants, like clinical features and laboratory investigations, were described as means with standard deviation for continuous variables and frequency with percentage for categorical variables.
- MELD score was described as the mean with standard deviation, and Patients were categorised into A, B, and C classes based on Child-Pugh score. The proportion of patients in each category was described as a proportion with a 95% confidence interval.
- The correlation between serum sodium, potassium and magnesium levels and MELD score was studied, and the pvalue was calculated.
- Differences in serum sodium, potassium and magnesium levels among various classes of Child-Pugh scores were studied using an unpaired t-test.

5. Results (Including Observations)

A total of 121 patients were enrolled. Median age was 45 years with a male predominance (85.12%). Alcoholic liver disease was the most common aetiology. Electrolyte disturbances included hypomagnesemia in 58.33% of cases, while sodium and potassium levels varied less significantly across CTP grades. Mean MELD score value was 24.

Table 1 presents the median values of magnesium, sodium, and potassium across CTP Classes A, B, and C. The data clearly demonstrate a stepwise decline in magnesium levels as liver disease worsens, whereas sodium and potassium show only minimal variations. This supports the hypothesis that magnesium may be a more consistent marker of cirrhosis severity.

Table 2 compares sodium levels across different CTP grades. The lack of statistically significant differences suggests that sodium is not a reliable standalone marker for disease severity progression in cirrhosis.

In Table 3, Potassium levels are compared across CTP classes in this table. Although potassium disturbances are clinically important in cirrhosis, this table indicates that they do not consistently correlate with CTP grade progression.

Table 1. Median electrolyte levels by CTP class

CTP Grade	Magnesium (mg/dL)	Sodium (mEq/L)	Potassium (mEq/L)
A	2.0	136	4.3
B	1.75	134	4.2
C	1.6	132	4.1

Table 2. Difference of sodium levels between different CTP grades (A, B and C)

	CTP Grade	N	Median	IQR	pvalue
Sodium levels	Grade A	1	137	0	0.22
	Grade B	24	133	7.5	
	Grade C	96	130	8	
	Total	121			

Table 3. Difference of potassium levels between different CTP Grades (A, B and C)

	CTP Grade	N	Median	IQR	pvalue
Potassium levels	Grade A	1	4.2	0	0.792
	Grade B	24	4.35	0.93	
	Grade C	96	4.3	1.1	
	Total	121			

Table 4 highlights significant differences in magnesium levels across CTP grades. The observed trend of decreasing magnesium with increasing CTP score adds further weight to its prognostic value.

In Table 5, electrolyte imbalances are assessed in the context of hepatic encephalopathy. Among the three, magnesium appears most associated with the presence of encephalopathy, suggesting a potential role in its pathophysiology. Table 5 summarises the results of the Kruskal-Wallis H test conducted to determine the statistical significance of electrolyte differences across CTP grades. Magnesium levels vary significantly ($p = 0.012$), reinforcing its diagnostic relevance. Sodium and potassium levels, however, show no significant differences.

Table 6 evaluates electrolyte profiles in patients with hepato-renal syndrome. Although all three electrolytes can be disrupted, magnesium and sodium appear to show more consistent trends compared to potassium.

Table 4. Difference of magnesium levels between different CPT Grades (A, B and C)

	CTP Grade	N	Median	IQR	pvalue
Magnesium levels	Grade A	1	2	0	0.012*
	Grade B	24	1.75	0.45	
	Grade C	96	1.6	0.6	
	Total	121			

Table 5. Comparison of electrolyte levels with hepatic encephalopathy

Electrolyte	HE (n = 68)	No HE (n = 53)	pvalue
Mg (Median and IQR)	1.2 (1.1 – 1.6)	1.9 (1.6 – 2.1)	<0.001*
Na (Median and IQR)	130 (125 – 134)	133 (129 – 136)	0.021*
K (Median and IQR)	4.4 (3.8 – 5.0)	4.2 (4.0 – 4.6)	0.367*

Table 6. Comparison of electrolyte levels with hepato-renal syndrome

Electrolyte	HRS (n = 26)	No HRS (n = 95)	pvalue
Mg (Median and IQR)	1.2 (1.1 – 1.6)	1.6 (1.6 – 2.1)	0.012*
Na (Median and IQR)	128(125 – 134)	133 (127 – 136)	0.003*
K (Median and IQR)	4.5 (3.8 – 5.0)	4.3 (4.0 – 4.6)	0.363

Table 7. Comparison of electrolyte levels with different MELD scores

Electrolyte	MELD ≤ 14 (n = 25)	MELD 15–21 (n = 25)	MELD ≥ 22 (n = 71)	pvalue
Mg (Median and IQR)	1.6 (1.5 – 2.0)	1.6 (1.2 – 1.9)	1.6 (1.2 – 1.9)	0.23
Na (Median and IQR)	133 (131 – 137)	133 (129 – 134)	129 (125 – 135)	0.017
K (Median and IQR)	4.3 (4.1 – 5.0)	4.1 (3.6 – 4.8)	4.3 (3.9 – 4.9)	0.308

Table 7 compares magnesium, sodium, and potassium across MELD score categories. A notable decline in magnesium with higher MELD scores underscores its correlation with liver disease severity. Sodium levels were significantly correlated with MELD levels.

Figure 1 illustrates a negative correlation between serum magnesium levels and MELD scores. As the MELD score increases, indicating more severe liver dysfunction, magnesium levels tend to decline. This supports the idea that hypomagnesemia may be an early and consistent marker of cirrhosis progression.

Figure 2 shows that magnesium levels decrease progressively from CTP Class A to Class C. This visual representation confirms the statistical trend seen in the data, reinforcing magnesium's role as a biomarker of liver disease severity.

Figure 3 presents the relationship between sodium levels and MELD scores. The distribution appears more scattered with no consistent pattern, indicating that sodium may not be strongly correlated with overall liver disease severity.

Figure 4 shows that sodium levels decrease progressively from CTP Class A to Class C, with no particular gradient, unlike magnesium.

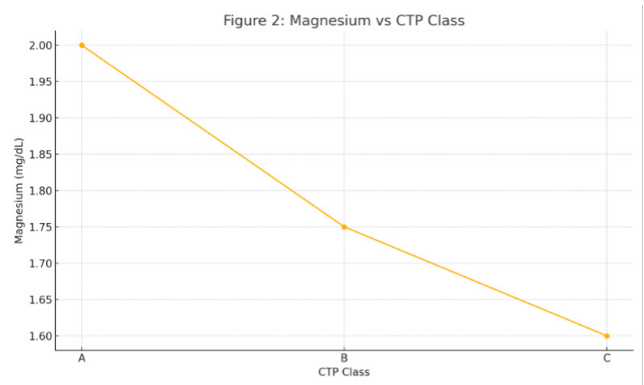
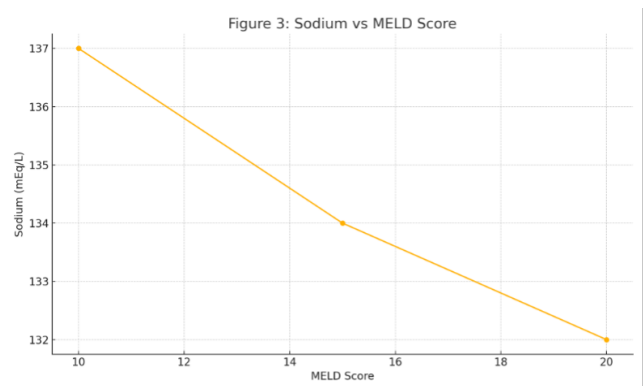
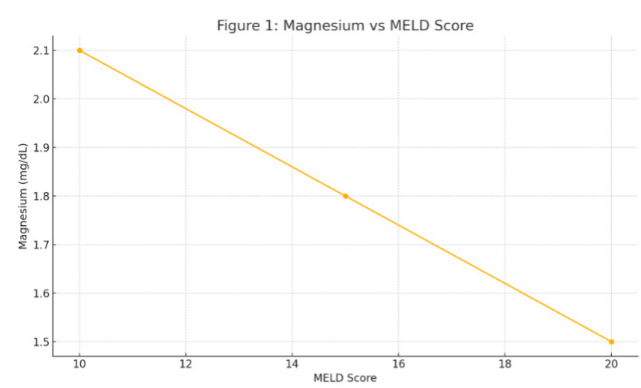
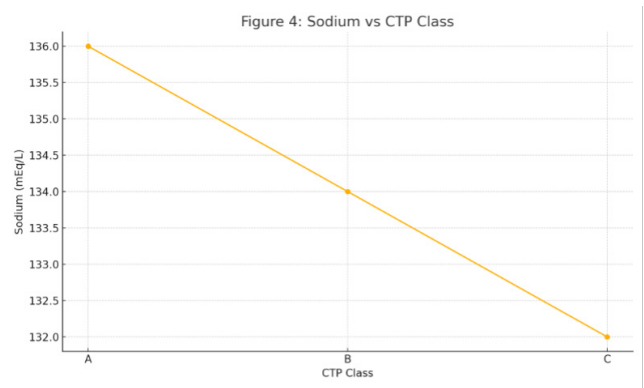
**Figure 2.** Magnesium vs CTP class.**Figure 3.** Sodium vs MELD score.**Figure 1.** Magnesium vs MELD score.**Figure 4.** Sodium vs CTP class.

Figure 5 shows potassium levels plotted against MELD scores. The graph lacks a clear directional trend, supporting findings that potassium disturbances are influenced more by acute renal and drug-related factors rather than baseline liver function.

Figure 6 visualises potassium levels across CTP classes. The absence of a significant gradient implies potassium is less reliable as a marker for chronic liver disease progression when compared to magnesium.

6. Discussion

Our results highlight the potential value of monitoring magnesium in patients with cirrhosis. We observed that as liver disease worsens—reflected by higher MELD and CTP scores—magnesium levels tend to fall significantly. This supports earlier findings and underscores magnesium's role as a possible indicator of disease severity^{7,8}.

Sodium and potassium, while clinically important in managing cirrhosis complications, did not show a

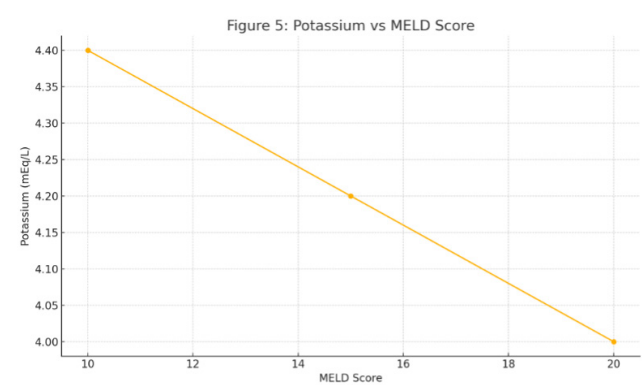


Figure 5. Potassium vs MELD score.

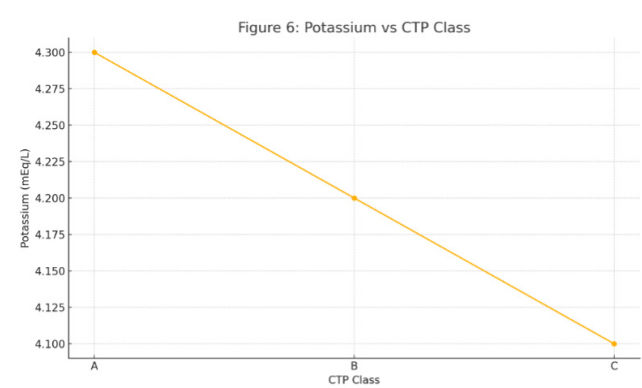


Figure 6. Potassium vs CTP class.

strong correlation with MELD or CTP scores in our study. This might be because these levels fluctuate more with medications and fluid shifts. For example, sodium can drop in patients with ascites who are on diuretics or fluid restrictions, and potassium levels can vary depending on renal function and aldosterone levels^{1,4}.

Even though we didn't find significant trends with sodium or potassium in this sample, these electrolytes still matter in day-to-day care. But from a prognostic standpoint—if we're trying to predict how advanced the liver disease is—magnesium appears to offer a more stable and meaningful signal.

In essence, our findings add to a growing body of research suggesting that magnesium deserves more attention in both clinical monitoring and research on cirrhosis.

7. Summary and Conclusion

This study reinforces that magnesium levels can provide meaningful insight into the severity of liver disease. We found a clear and statistically significant link between falling magnesium levels and worsening MELD and CTP scores.

While sodium and potassium abnormalities are certainly common and important in managing cirrhosis, they did not show the same strong relationship with severity scores in our sample. This doesn't make them less clinically relevant—but it does suggest that magnesium might be a better marker for overall disease progression.

Given that magnesium testing is inexpensive and readily available, incorporating it into routine evaluations could help clinicians track disease status more effectively. Future research should explore whether correcting magnesium deficiencies might also improve outcomes in patients with cirrhosis.

8. References

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