International Journal of Research in Medical Sciences
Mogalapu SS et al. Int J Res Med Sci. 2025 Jun;13(6):2529-2536
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20251647

Original Research Article

Correlation of serum magnesium levels with severity of liver disease in cirrhosis patients

Sai Samhitha Mogalapu*, Venkatesan Sivaprakasam, Umadevi Thirukonda Balasubramanian

Department of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, Tamil Nadu, India

Received: 21 April 2025
Revised: 14 May 2025
Accepted: 20 May 2025

***Correspondence:**
Dr. Sai Samhitha Mogalapu,
E-mail: samhithamogalapu@gmail.com

ABSTRACT

Background: Chronic liver disease (CLD) patients are prone to micronutrient disturbances particularly magnesium secondary to malnutrition and disturbed hepatic metabolism which may further exacerbate liver dysfunction. The child-turcotte-pugh (CTP) score and the model for end-stage liver disease (MELD) are established indices for cirrhosis prognostication. The ail of the study was to estimate serum magnesium levels in patients with chronic liver disease and to examine the correlation between serum magnesium levels and the severity of liver disease, as assessed by the MELD and child-pugh scores.
Methods: This cross-sectional observational study recruited 121 participants meeting the inclusion criteria over a period of six months. Informed consent was obtained. Basic investigations were done, including complete blood count, renal and liver function tests, PT-INR, and serum magnesium levels. MELD and child-pugh scores were calculated, and data analysis was performed using SPSS software.
Results: The median age of the participants was 45 years, with 85.12% being male and the predominant etiology being alcohol-related liver disease. The prevalence of Hypomagnesemia was 58.33%. A statistically significant correlation was observed between lower magnesium levels and higher child-pugh class, particularly in class C patients. There was a significant negative correlation between magnesium levels and MELD scores.
Conclusions: The findings underscore the need for further randomised controlled trials to evaluate the efficacy of magnesium supplementation in slowing disease progression and preventing cirrhosis complications. This potential treatment could significantly reduce morbidity and mortality, offering a promising outlook for the future of cirrhosis management.

Keywords: Cirrhosis, Hypomagnesemia, Nutrition, CTP score, MELD score

INTRODUCTION

Electrolyte disturbance and malnutrition are part of the issues confronting patients suffering from chronic liver disease, and these issues can worsen the prognosis and increase morbidity further.[1] Of all the trace elements, magnesium is perhaps the most "overlooked electrolyte" despite being an important cofactor in many biochemical processes. It is a significant cofactor in over 300 enzymatic reactions, including ATP metabolism, DNA/RNA synthesis, and cellular signalling, ensuring essential hepatic metabolic homeostasis.[2,3] Disruption of magnesium balance can be both a cause and a consequence of liver dysfunction. Magnesium deficiency in CLD patients stems from malnutrition, malabsorption, low albumin levels, diuretic therapy and hormonal imbalances.[1,2] Experimental and clinical studies have demonstrated that magnesium can accelerate liver injury by facilitating mitochondrial dysfunction, oxidative stress, and inflammatory signalling.[3]

Magnesium's role in cirrhosis is pathophysiologically and prognostically relevant. Clinicians use composite severity scores for staging and predicting outcomes in cirrhosis. The child-turcotte-pugh (CTP) score, including bilirubin, albumin, prothrombin time (INR), ascites, and encephalopathy, has historically been applied to determine the disease severity and the ability to forecast survival.[4,5] The model for end-stage liver disease (MELD) score first developed to assess the priority of liver transplant candidates uses bilirubin, INR, and creatinine to estimate 90-day mortality risk.[6] If serum magnesium correlates very highly with these tests, it is a readily available biomarker for the disease severity or could have prognostic significance.

Prior research has suggested an association between magnesium levels and liver disease outcomes. Wu et al., in a large NHANES cohort, reported that increased dietary magnesium intake was associated with reduced risk of mortality from liver disease, particularly alcohol-related liver disease and hepatic steatosis.[7] Directly, clinical research in cirrhosis has identified magnesium depletion to be familiar and potentially predictive. For example, a 2021 retrospective study of 152 cirrhosis patients reported that mean serum magnesium was low in the cohort, and patients with more severe disease - child-pugh B/C or MELD ≥21 had lower magnesium than those with less severe disease.[1] However, not all research has demonstrated clear correlations; an earlier publication reported no correlation between serum magnesium and isolated measures of liver function, such as total bilirubin, indicating that some laboratory measures may not reflect the multi-factorial effect of magnesium on the severity of disease.[8] With this background, this study aims to examine the correlation between serum magnesium levels and the severity of liver disease, as assessed by scores like CTP and MELD. As magnesium levels can easily be determined, they would reflect the nutritional status of the patient, which can be targeted in future interventions to reduce morbidity and mortality.

METHODS

This study was conducted after the approval of the Institutional Ethics Committee (IEC No. MMC/Approval /20042024). Written informed consent was obtained from all the participants. It was a hospital-based cross-sectional observational study conducted in the Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, Madras Medical College, over a period of six months from March to August 2024. The study was performed according to the STROBE guidelines for observational studies.

Sample size

The sample size (N) was calculated using the formula:

$N = Z^2 \times P \times Q/d^2$

Where:

Z = Z-score = 1.96

P = estimated proportion of an attribute present in the population = 71.8% 9

Q = 1 – P = 28.2

d = 8

N = 1.96×1.96×71.8×28.2/8×8 = 121

The sample size was calculated to be 121.

Inclusion criteria

Patients aged 18 years and above from both sexes diagnosed with chronic liver disease admitted either for the management of complications of cirrhosis or for outpatient follow-up visits were included.

Exclusion criteria

We excluded patients taking chronic magnesium supplementation or a recent high-magnesium diet (within 6 months) to rule out confounding supplementation effects and patients with comorbidities known to affect magnesium independently: chronic kidney disease (due to deranged excretion), chronic diarrhoea, hypothyroidism, or current use of magnesium-altering drugs like lithium, thiazide diuretics and insulin. Patients with hepatocellular carcinoma or other malignancies were also excluded.

A detailed history of the duration of the illness and other significant medical illnesses was elicited, and a clinical examination was performed. Basic investigations, including complete blood count (CBC), renal function test (RFT), liver function test (LFT), PT-INR and estimation of serum magnesium levels, were carried out in all the patients included in the study. MELD score and child-pugh class were assessed. Serum magnesium was determined by a colourimetric assay on an automated analyser, with an adult reference range of ~1.7–2.4 mg/dl. Internal quality controls were used to assure assay accuracy. For each patient, we calculated the MELD score by the formula: MELD = (0.957 · ln (Serum Creatinine) + 0.378 · ln (Serum Bilirubin) + 1.120. ln (INR) + 0.643) ×10, which yields a score from 6 to 40.[6] We documented child-pugh components - serum albumin, total bilirubin, INR, grade of ascites, and encephalopathy- to determine the child-pugh score (5–15 points), as shown in Table 1. Patients were grouped into child-pugh class A, B, and C for analysis.[4]

Statistical analysis

Statistical Analysis was performed using SPSS for Windows version 26. Continuous data were presented as mean ± standard deviation if normally distributed or as

median with interquartile range (IQR) when not normally distributed. Categorical variables were described as frequencies with percentages. The Kruskal-Wallis H test allowed comparison of serum magnesium levels between the three Child-Pugh classes (ordinal groups A, B, C) with Dunn's test for pairwise comparisons when significant. Correlation of magnesium values and MELD scores was tested by Pearson's correlation coefficient (r), and a scatter diagram showing serum Mg vs. MELD was plotted. We computed 95% confidence intervals (CI) for the correlation coefficient. A p-value <0.05 was taken to be statistically significant.

RESULTS

Patient characteristics

A total of 121 patients with chronic liver disease, of which 103 (85.12%) were male and 18 (14.88%) were female, were recruited with a median age of 45 years (range 39-52). The prevalence of hypomagnesemia was 58.33% and was seen in 70 participants. The predominant etiology was alcohol-related liver disease (55.4%).

As per Table 2, the median age and the gender distribution between participants with and without hypomagnesemia were 45 years and 46 years, respectively. It did not reveal much difference between the two groups.

As per Table 3, the gender distribution in patients with hypomagnesemia and those with normal magnesium levels showed no statistical significance. Males were predominant in both groups.

Table 1: Child-pugh staging system.

Parameter	1 point	2 points	3 points
Total bilirubin (mg/dl)	<2	02-mar	>3
Serum albumin (g/l)	>3.5	2.8-3.5	<2.8
INR	<1.7	1.7-2.3	>2.3
Ascites	Absent	Slight	Moderate
Encephalopathy	No encephalopathy	Grade 1-2	Grade 3-4

Table 2: Age and gender distribution among patients with and without hypomagnesemia.

Demographic variable	Normal (n=51)	Hypomagnesemia (n=70)
Age in years (median±IQR)	46 (39-52)	45 (39-53)
Gender - male	45 (88.24%)	58 (82.86%)
Gender - female	6 (11.76%)	12 (17.14%)

Table 3: Difference of gender and etiology in patients with and without hypomagnesemia.

Gender	Hypomagnesemia (n=70)	Normal (n=51)	Odd's ratio	95% CI	P value
Male (n=103)	58 (56.3%)	45 (43.7%)	0.64	0.22-1.85	0.412
Female (n=18)	12 (66.7%)	6 (33.3%)			

The chi-square test was used to assess statistical significance.

Table 4: Comparison of age and gender-based distribution with aetiology.

Age (years)/etiology	AI (n=5)	Ethanol (n=67)	HBV (n=19)	HCV (n=11)	MAFLD (n=15)	Wilson (n=4)	P value
<40 (n=39)	1	24	10	4	0	0	0.009
41-50 (n=45)	3	26	6	2	4	4	
51-60 (n=31)	1	14	2	5	9	0	
>60 (n=6)	0	3	1	0	2	0	

AI - autoimmune, HBV - hepatitis-b virus, HCV - hepatitis-c virus, MAFLD - metabolic-associated fatty liver disease. The chi-square test was used to assess statistical significance.

As seen in Tables 4 and 5, ethanol was the most common aetiology among all age groups. There was a statistically significant relationship (p-value <0.001) between gender and aetiology distribution, with ethanol being predominant among males and MAFLD being most common among females.

As seen in Table 6, the trend was decreasing serum magnesium with increasing child-pugh class. The median magnesium values for child-pugh A, B, and C were 2 mg/dl, 1.75 mg/dl and 1.6 mg/dl, respectively. The difference in magnesium levels between the three groups of the CTP grades was statistically significant. (p-value: 0.012).

As in Table 7, post-hoc testing revealed that class C patients had significantly lower magnesium than class B (p = 0.005), but class B vs. class A was not significant after adjustment (p = 0.543). The comparison of class C and A was not significant (p = 0.271). These results signify that advanced cirrhosis (CTP-C) is linked to more severe magnesium depletion. Figure 1 demonstrates the distribution of magnesium values in various CTP grades.

Table 5: Comparison of gender wise distribution with etiology among the study participants.

Gender/etiology	AI (n=5)	Ethanol (n=67)	HBV (n=19)	HCV (n=11)	MAFLD (n=15)	Wilson (n=4)	P value
Male (n=103)	0	65	19	9	6	4	<.001*
Female (n=18)	5	2	0	2	9	0	

AI - autoimmune, HBV - hepatitis-b virus, HCV - hepatitis-c virus, MAFLD - metabolic-associated fatty liver disease. The chi-square test was used to assess statistical significance.

Table 6: Difference of magnesium levels between different CTP Grades (A, B and C).

CTP grade	No. of patients	Median magnesium level (mg/dl)	IQR	P value
Grade A	1	2	0	
Grade B	24	1.75	0.45	.012*
Grade C	96	1.6	0.6	

We conducted a kruskal-wallis h test to compare the magnesium levels of the three groups. it revealed a significant difference in the magnesium levels between the three groups, h(2) = 8.92, p = 0.12. (*) indicates statistical significance.

Table 7: Comparison of mean difference in magnesium levels between various CTP grades.

CTP grade	CTP grade	Mean difference (mg/dl)	95% CI of mean difference	P value
Grade A	Grade B	0.204	-0.47 to 0.89	0.543
Grade A	Grade C	0.468	-0.37 to 1.31	0.271
Grade B	Grade C	0.284	0.08 to 0.45	0.005*

The chi-square test was used to assess the mean difference in magnesium levels between the various ctp groups. (*) indicates statistical significance.

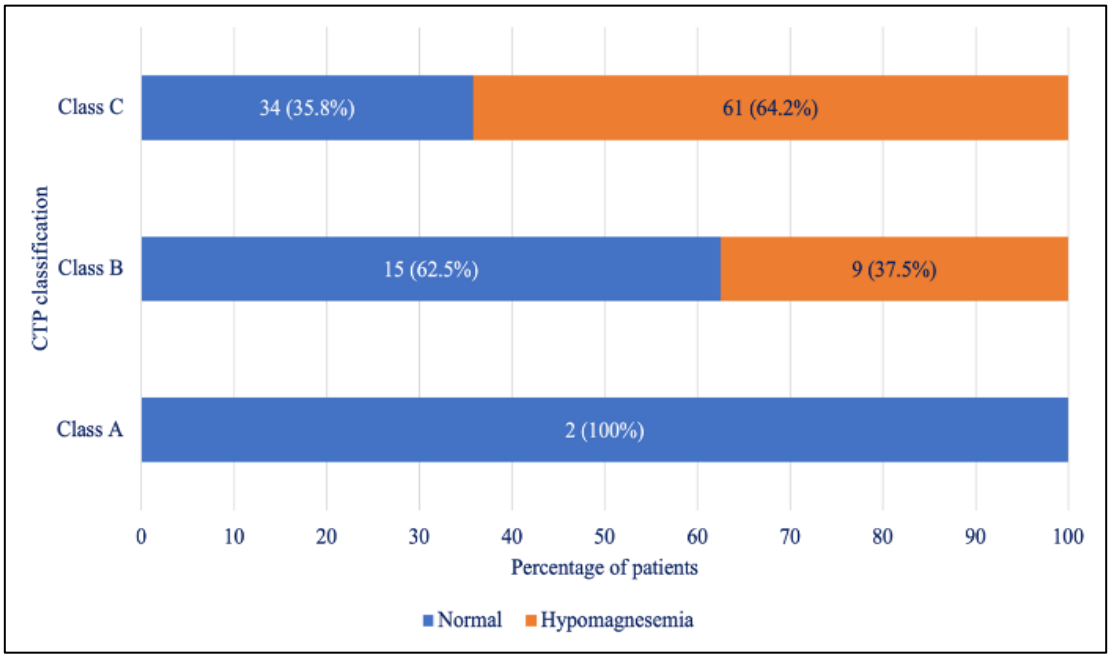

Figure 1: Distribution of magnesium levels and CTP classes.

As seen in Figure 2, serum magnesium correlated inversely with MELD. On Pearson correlation analysis, there was a moderately negative correlation between MELD and magnesium (r (119) = -.336, p = <.001) (Figure 2). Thus, patients with higher MELD scores had lower magnesium levels.

To further investigate this association, we examined the correlations between magnesium concentrations and the individual laboratory components of MELD and CTP. The serum magnesium was statistically significantly negatively correlated with total bilirubin (r (119) = -0.258, p =0.004) (Figure 3). This suggests that patients with raised bilirubin, a reflection of compromised liver excretory function, had decreased magnesium.

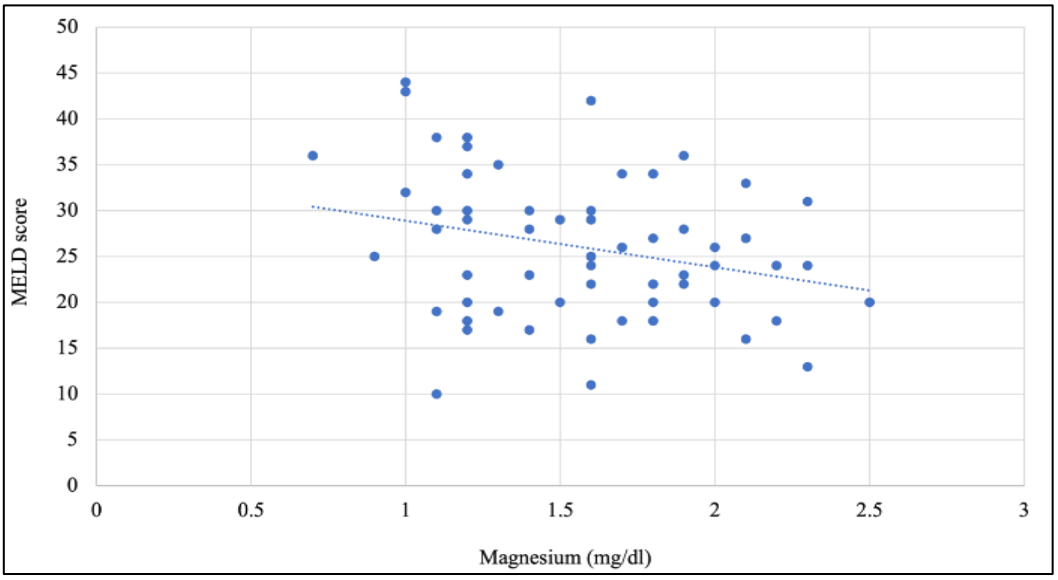

Figure 2: Scatter plot of MELD vs serum magnesium levels.

r(119) = .336, p = <0.001. Pearson's correlation was used to assess the statistical significance.

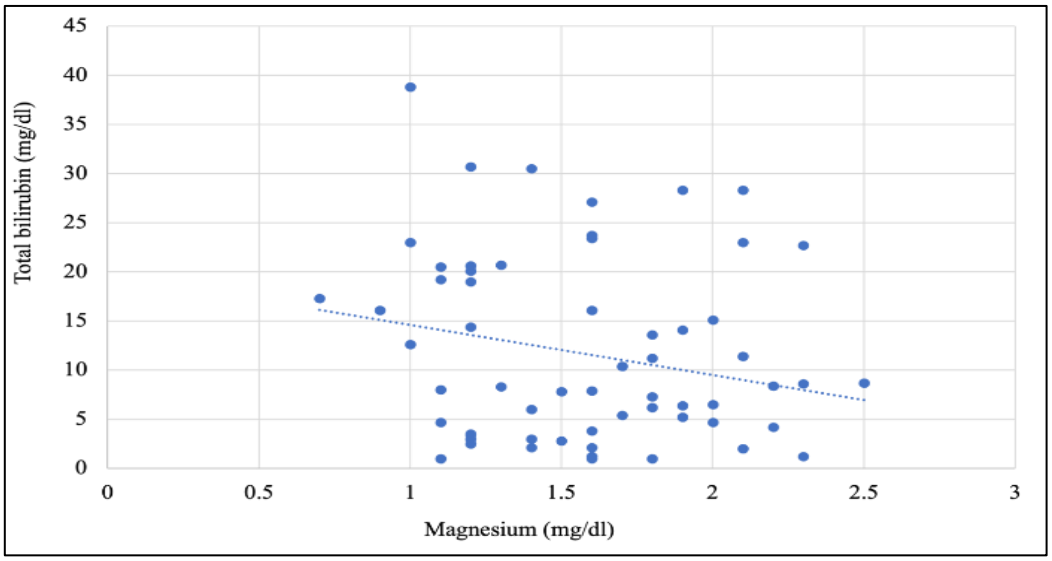

Figure 3: Correlation of serum bilirubin levels with magnesium levels.

r(119) = .258, p = .004; Pearson's correlation test was used.

DISCUSSION

This was a hospital-based cross-sectional study. One hundred twenty-one patients with chronic liver disease were included in this study. The median age of the participants was 45 years, with 85.12% being male, and most of the study participants were 41-50 years (37.19%). We observed a significant inverse relationship between serum magnesium and disease severity as assessed by child-pugh and MELD scores. Patients with more severe cirrhosis (CTP class C or elevated MELD) uniformly had lower magnesium levels. These observations prove that magnesium homeostasis is associated with hepatic functional reserve and prognosis in CLD.

Our findings are consistent with recent evidence globally. Peng et al. found magnesium deficiency was common in cirrhosis and was associated with more severe disease, reporting much lower Magnesium in Child-Pugh B/C patients and MELD ≥21. They also noticed that magnesium-deficient cirrhotic had poorer transplant-free survival chances.[1] In a study by Veena et al., the prevalence of hypomagnesemia was 71.8%, and a stepwise significant decline in mean magnesium from child-pugh A to C.[9] Our observation was in accord with these results in more samples. Another recent prospective study by Rani et al. illustrated that magnesium levels were significantly lower in cirrhotic patients than in healthy controls. Among cirrhosis patients, patients in higher child-pugh classes had lower magnesium levels. The authors concluded that

magnesium loss correlated with disease severity and speculated that correcting this imbalance may reduce complications.[10] Our findings support this conclusion since we also saw lower magnesium levels in patients with higher CTP and MELD scores, which is possibly associated with poor outcomes.

Das et al conducted a hospital-based study in Bareilly and analysed serum magnesium levels in patients with cirrhosis and compared them with the serum magnesium levels of controls. The study cohort included 50 cases of alcohol-induced liver cirrhosis (Group I) and 50 normal subjects (Group II). They found that serum magnesium levels in Group I were significantly lower ($p< 0.0001$).[11] Gurudevarahalli et al conducted a hospital-based study on serum magnesium levels in liver disease patients and observed lower serum magnesium levels in cirrhotic patients. Out of 25 patients with cirrhosis, 23 of them had low serum magnesium.[12]

Koivisto et al conducted a study to evaluate the magnesium levels in end-stage cirrhotic patients and healthy subjects in Finland. They performed a magnesium loading test in ten cirrhotics listed for liver transplantation and in six healthy control subjects. The uptake of magnesium was increased in cirrhotic patients as compared to controls. They found a lower magnesium uptake (8%) in healthy controls than in cirrhotics (34%).[13]

In a study done by Kar K, Dasgupta et al, they assessed the change of trace elements like zinc, copper, iron, magnesium, bilirubin and albumin during the process of decompensation of cirrhosis. Their study includes 34 patients with compensated cirrhosis and 31 patients with decompensated cirrhosis. They observed a statistically significant lower levels of trace element concentrations in the decompensated group.[14] Nangiya et al studied the association between levels of trace elements like zinc, selenium, and magnesium and CTP scores among cirrhotics. It was a cohort study. They observed that the serum zinc, magnesium and selenium levels decreased with the advancement of cirrhosis and correlated negatively with the CTP score.[15]

A double-blind, parallel group randomised control trial by Poikolainen et al assessed the serum liver enzyme levels after magnesium supplementation among alcoholics. They observed that the enzyme levels decreased significantly in the magnesium-treated group compared to the placebo group.[16] A Norwegian study conducted in chronic alcoholics observed that short-term magnesium supplementation can improve liver function and muscle strength. It was a double-blind randomised control study.[17]

In a study by Eshraghian A et al, 226 healthy participants who underwent liver biopsy were included. Biopsy samples were examined for the presence of steatosis and steatohepatitis. Serum magnesium levels were found to be independently associated with hepatic steatosis and steatohepatitis and were lower in those patients with evidence of steatosis and steatohepatitis in biopsy compared to other study participants. This shows that magnesium can also be associated with MAFLD.[18]

In a study conducted in 2015, divalent cations like magnesium, zinc and copper levels were studied among cirrhosis patients with variceal bleeding and healthy subjects as controls. They concluded that serum magnesium levels were lower in cirrhosis patients with variceal bleeding and correlated with complications like hepatic encephalopathy and ascites.[19] Rahelić D et al studied the role of zinc, copper, manganese and magnesium levels in cirrhosis patients. They observed no significant difference in magnesium concentrations in cirrhosis patients and controls. Serum manganese levels were found to correlate significantly with CTP groups ($p = 0.036$).[20]

Of note, not all previous research had positive correlations. Chavan et al conducted a study in Karnataka. It mostly looked at magnesium levels in patients with liver disease and found no correlation between serum magnesium and total bilirubin.[8] Their study involved both acute and chronic liver illness and employed bilirubin alone as a surrogate for severity, which will not reflect the multifactorial deterioration of cirrhosis. However, composite scores such as MELD and CTP give a more integrated severity measurement.

The correlation can be explained in terms of two not mutually exclusive mechanisms: magnesium as a victim of disease severity and magnesium as a contributor to disease progression. Advanced cirrhosis is first marked by malnutrition, malabsorption, secondary endocrine alterations like hyperaldosteronism and diuretic usage that facilitates renal wasting of magnesium. With worsening liver function, dietary intake tends to decrease, and gut oedema, including magnesium, may interfere with nutrient absorption. In addition, cirrhosis-related hypoalbuminemia diminishes the protein-bound fraction of magnesium and presumably reduces total serum magnesium levels. Our result of negative correlation with bilirubin favours the worsening excretory liver function occurring concurrently with falling magnesium by such nutritional/metabolic means.

However, the deficiency state per se could enhance liver damage and cause a vicious cycle. Low intracellular magnesium levels have been reported by experimental research to exacerbate oxidative stress and inflammation in the liver, affect energy-requiring cellular functions, and even facilitate fibrosis. Magnesium is necessary for enzyme activities that help to repair DNA and defend against oxidants; insufficiency may cause accumulation of oxidative injury to hepatocytes. A comprehensive review by Liu et al concluded that magnesium deficiency in chronic liver diseases is very prevalent and could speed up cirrhosis progression and even cause complications such as liver cancer.[3]

Limitations

It was a cross-sectional study, so the causal relationship could not be studied. Being a tertiary care referral centre, most of the patients were critically ill and belonged to higher CTP classes and MELD scores. We attempted to control for confounders by excluding patients on magnesium supplements or with renal failure, but minor confounders may still have influenced the findings. A healthy cohort group would have quantified the magnesium deficiency in cirrhotic patients more accurately.

Strengths

We quantitatively assessed magnesium in a well-phenotyped cirrhosis patient cohort and applied stringent statistical tools to determine this association with two popular severity scores. Concordant results obtained from group comparisons using a non-parametric technique and correlation analyses add credibility to the association. We also cross-checked our findings with current literature, which showed a high level of correspondence with several recent studies in various populations, further supporting external validity. To our knowledge, this study is among the first to explicitly explore and document the interaction between magnesium levels and both Child-Pugh and MELD scores simultaneously, giving a complete picture of the interaction.

CONCLUSION

The prevalence of hypomagnesemia in our study was 58.33%. The mean magnesium value was 1.6 mg/dl. A statistically significant correlation was observed between mean magnesium levels and the various child-pugh classes; lower levels were seen in Class C patients. Lower magnesium levels were seen in advanced cirrhosis patients with high CTP class or MELD score, indicating that magnesium levels may reflect the underlying severity of the disease. These results are congruent with the emerging data from the literature, suggesting that magnesium status may represent a significant but underappreciated part of patient evaluation in CLD.

Prospective cohort studies with long-term follow-up and randomised trials should be done to establish the role of magnesium in disease progression and to study the therapeutic role of magnesium supplementation in retarding the progression and preventing the complications of cirrhosis. Clinicians should be careful not to miss serum magnesium assay while managing cirrhosis patients, as hypomagnesemia is a potentially correctable and modifiable factor.

ACKNOWLEDGEMENTS

Authors would like to thank the faculty of the Institute of Internal Medicine and Department of Biochemistry, Madras Medical College, for their expert guidance and valuable support.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: This study was conducted with the approval of the Institutional Ethics Committee (IEC No. MMC/Approval /20042024)

REFERENCES

1. Peng X, Xiang R, Li X, Tian H, Li C, Peng Z, et al. Magnesium deficiency in liver cirrhosis: a retrospective study. Scandinavian journal of gastroenterology. 2021;56(4):463-8.
2. Llibre-Nieto G, Lira A, Vergara M, Solé C, Casas M, Puig-Diví V, et al. Micronutrient deficiencies in patients with decompensated liver cirrhosis. Nutrients. 2021;13(4):1249.
3. Liu M, Yang H, Mao Y. Magnesium and liver disease. Ann Transl Med. 2019;7(20):578.
4. Durand F, Valla D. Assessment of the prognosis of cirrhosis: Child-Pugh versus MELD. J Hepatol. 2005;42 Suppl(1):S100-7.
5. Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R. Transection of the oesophagus for bleeding oesophageal varices. Br J Surg. 1973;60(8):646-9.
6. Kamath PS, Wiesner RH, Malinchoc M, Kremers W, Therneau TM, Kosberg CL, et al. A model to predict survival in patients with end-stage liver disease. Hepatology. 2001;33(2):464-70.
7. Wu L, Zhu X, Fan L, Kabagambe EK, Song Y, Tao M, et al. Magnesium intake and mortality due to liver diseases: Results from the Third National Health and Nutrition Examination Survey Cohort. Sci Rep. 2017;7(1):17913.
8. Chavan M, Nadagoudar SB, Boke U. Study of serum magnesium in liver disease - a cross-sectional observational study conducted in Bagalkot District, Karnataka. J Evid Based Med Healthc. 2021;8(42):3646.
9. Veena G, James R. Prevalence of hypomagnesemia in cirrhosis of liver and its association with severity of the disease. Asian J Pharm Clin Res. 2022;15(8):92-5.
10. Rani P, Goel R, Madaan H. Assessment of serum magnesium levels in patients with liver cirrhosis and its correlation with severity of disease. Int J Acad Med Pharm. 2024;6(3):462-6.
11. Das B, Chandra P, Thimmaraju KV. Serum magnesium level in patients with liver cirrhosis. Int J Biol Med Res. 2011;2(3):709-11.
12. Gowda P, Tembad M. Study of serum magnesium in liver diseases. J Evol Med Dent Sci. 2015;4:3047-56.
13. Koivisto M, Valta P, Höckerstedt K, Lindgren L. Magnesium depletion in chronic terminal liver cirrhosis. Clin Transplant. 2002;16(5):325-8.
14. Kar K, Dasgupta A, Vijaya Bhaskar M, Sudhakar K. Alteration of micronutrient status in compensated and

decompensated liver cirrhosis. Indian J Clin Biochem. 2014;29(2):232-7.
15. Nangliya V, Sharma A, Yadav D, Sunder S, Nijhawan S, Mishra S. Study of trace elements in liver cirrhosis patients and their role in prognosis of disease. Biol Trace Elem Res. 2015;165(1):35-40.
16. Poikolainen K, Alho H. Magnesium treatment in alcoholics: a randomized clinical trial. Subst Abuse Treat Prev Policy. 2008;3:1.
17. Gullestad L, Dolva LØ, Birkeland K, Falch D, Fagertun H, Kjekshus J. Oral versus intravenous magnesium supplementation in patients with magnesium deficiency. Magnes Trace Elem. 1991;10(1):11-6.
18. Eshraghian A, Nikeghbalian S, Geramizadeh B, Malek-Hosseini SA. Serum magnesium concentration is independently associated with non-alcoholic fatty liver and non-alcoholic steatohepatitis. United Eur Gastroenterol J. 2017;6(1):97-103.
19. Dumea M, Cimpoesu DC, Nechifor M. Divalent cations profile in patients with liver cirrhosis and variceal bleeding. Rev Med Chir Soc Med Nat Iasi. 2015;119(4):1166-73.
20. Rahelić D, Kujundzić M, Romić Z, Brkić K, Petrovecki M. Serum concentration of zinc, copper, manganese and magnesium in patients with liver cirrhosis. Coll Antropol. 2006;30(3):523-8.

Cite this article as: Mogalapu SS, Sivaprakasam V, Balasubramanian UT. Correlation of serum magnesium levels with severity of liver disease in cirrhosis patients. Int J Res Med Sci 2025;13:2529-36.