

Serum Magnesium Levels in Adults with Liver Cirrhosis and Its Association with Grade of Hepatic Encephalopathy

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Abstract

Background: Hepatic encephalopathy (HE) is a major neuropsychiatric complication of liver cirrhosis. Electrolyte and micronutrient abnormalities may contribute to its clinical manifestations. This study evaluated serum magnesium levels and their association with the severity of HE in adults with liver cirrhosis. **Material and Methods:** This hospital-based cross-sectional observational study included 154 adults with liver cirrhosis. Hepatic encephalopathy was graded according to the West Haven criteria. Serum magnesium was measured using the Xylidyl Blue I chromogenic method, and hypomagnesemia was defined as serum magnesium <1.6 mg/dL. Serum magnesium levels were compared according to the presence and grade of HE, and correlation analysis was performed. **Results:** The mean age was 50.81 ± 11.30 years, and 77.27% of participants were male. HE was present in 111 (72.08%) patients, while hypomagnesemia was observed in 62 (40.26%). Mean serum magnesium was significantly lower in patients with HE than in those without HE (1.56 ± 0.34 vs. 2.22 ± 0.28 mg/dL; $p < 0.0001$). Hypomagnesemia was present in 54.95% of patients with HE compared with 2.33% without HE ($p < 0.0001$). Mean serum magnesium progressively decreased from 2.22 ± 0.28 mg/dL in Grade 0 to 1.25 ± 0.19 mg/dL in Grade 4, while hypomagnesemia increased from 2.33% to 90.48%. Serum magnesium showed a strong negative correlation with HE grade ($r = -0.792$, $p < 0.0001$). **Conclusion:** Lower serum magnesium levels were significantly associated with the presence and increasing severity of hepatic encephalopathy in patients with liver cirrhosis. Serum magnesium assessment may provide a simple adjunctive biochemical parameter in the evaluation of these patients.

Keywords: Liver cirrhosis; Hepatic encephalopathy; Hypomagnesemia; Serum magnesium; West Haven criteria.

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INTRODUCTION

Liver cirrhosis is the advanced stage of chronic liver injury characterized by progressive fibrosis, architectural distortion, and regenerative nodules. It remains an important cause of morbidity and mortality worldwide, with complications such as portal hypertension, ascites, variceal bleeding, and hepatic encephalopathy (HE). Magnesium disturbances have increasingly been recognized in patients with cirrhosis and may be related to disease severity and adverse clinical outcomes.^[1]

Magnesium is an essential intracellular cation involved in numerous physiological processes, including energy metabolism, mitochondrial function, oxidative stress regulation, and inflammatory responses. Recent evidence suggests that magnesium deficiency is common in patients with chronic liver disease and may become more pronounced with increasing severity of cirrhosis.^[2,3] Peng et al. reported a significant association between magnesium deficiency and advanced liver cirrhosis, highlighting the potential clinical importance of monitoring magnesium status in these patients.^[4] Micronutrient deficiencies are also frequently observed in decompensated cirrhosis and may contribute to nutritional and metabolic abnormalities.^[5]

Low magnesium status has additionally been associated with hepatic fibrosis, while experimental and clinical evidence

suggests that magnesium may influence hepatic inflammation, oxidative injury, mitochondrial dysfunction, and fibrogenesis.^[6,7] Nutritional assessment and correction of clinically relevant deficiencies therefore constitute important components of comprehensive management in chronic liver disease.^[8] Hepatic encephalopathy is a major neuropsychiatric complication of cirrhosis, ranging from subtle cognitive impairment to deep coma. Although electrolyte abnormalities are recognized precipitating or aggravating factors, evidence regarding the relationship between serum magnesium and the severity of HE remains limited. Therefore, the present study was undertaken to evaluate serum magnesium levels in adults with liver cirrhosis and determine their association with the grade of hepatic encephalopathy.

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MATERIALS AND METHODS

Study Design and Setting: This hospital-based cross-sectional observational study was conducted at a tertiary care hospital among adult patients with liver cirrhosis. A total of 154 patients fulfilling the predefined eligibility criteria were included in the study.

Study Population: Adult patients with known liver cirrhosis admitted to the hospital during the study period were evaluated. Cirrhosis was diagnosed on the basis of clinical assessment and radiological findings on ultrasonography, including surface nodularity, coarse or heterogeneous hepatic echotexture, segmental hypertrophy, and/or features of portal hypertension such as altered Doppler flow, splenomegaly, or ascites.

Inclusion Criteria

Adult patients of either sex with established liver cirrhosis who were admitted during the study period were included.

Exclusion Criteria

Patients receiving medications known to affect serum magnesium levels, those with impaired renal function (creatinine clearance <60 mL/min), malignancy or other significant comorbidities, and patients with malabsorption syndromes were excluded.

Sample Size: The sample size was calculated based on a previous study by Cohen-Hagai et al., in which the proportion of patients with hypomagnesemia was approximately 10%. Considering an anticipated proportion (p) of 10%, absolute precision of 5%, 95% confidence level, and an allowance for error/non-response, the final sample size was 154 patients.

Data Collection and Clinical Assessment: After enrolment, demographic and relevant clinical details were recorded using a predefined study proforma. All participants underwent clinical evaluation for the presence and severity of hepatic encephalopathy.

Hepatic encephalopathy was graded according to the West Haven criteria as:

- **Grade 0:** No detectable abnormality.
- **Grade I:** Trivial lack of awareness, euphoria or anxiety, shortened attention span, and impaired ability to perform addition.
- **Grade II:** Lethargy or apathy, minimal disorientation,

subtle personality changes, inappropriate behaviour, and impaired ability to perform subtraction.

- **Grade III:** Somnolence to semi-stupor with responsiveness to verbal stimuli, confusion, and gross disorientation.
- **Grade IV:** Coma with absence of response to verbal or noxious stimuli.

Serum Magnesium Estimation: Venous blood samples were obtained from all enrolled patients for measurement of serum magnesium. Serum magnesium was estimated using the Xylidyl Blue I chromogenic method, a direct colorimetric assay, with absorbance measured at 660 nm. Hypomagnesemia was defined as serum magnesium <1.6 mg/dL.

The serum magnesium concentration was compared between patients with and without hepatic encephalopathy and across different West Haven grades. Correlation between serum magnesium level and HE grade was also assessed.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25.0. Categorical variables were expressed as frequencies and percentages, while quantitative variables were summarized as mean $\pm$ standard deviation or median with interquartile range, as appropriate. Normality of continuous variables was assessed using the Shapiro–Wilk test.

The independent-samples t-test was used for comparison of normally distributed continuous variables between two groups, while one-way ANOVA was used for comparisons involving more than two groups. Appropriate non-parametric tests were used for non-normally distributed data. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. Spearman's rank correlation coefficient was used to determine the relationship between serum magnesium concentration and grade of hepatic encephalopathy. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 154 patients with liver cirrhosis were included in the study. The mean age of the study population was 50.81 ± 11.30 years. Of the 154 patients, 119 (77.27%) were males and 35 (22.73%) were females. The largest proportions of patients belonged to the 41–50-year and 51–60-year age groups (27.27% each) [Table 1].

Table 1: Demographic characteristics of the study population (n=154)

Characteristic	n	Percentage (%)
Age group (years)		
<40	37	24.03
41–50	42	27.27
51–60	42	27.27
>60	33	21.43
Age, mean $\pm$ SD (years)	50.81 $\pm$ 11.30	—
Sex		
Male	119	77.27
Female	35	22.73

With regard to hepatic encephalopathy (HE), 43 (27.92%) patients had Grade 0, while 18 (11.69%), 40 (25.97%), 32 (20.78%), and 21 (13.64%) had Grades 1, 2, 3, and 4 HE, respectively. Thus, 111 (72.08%) patients had hepatic encephalopathy (Grades 1–4). Overall, hypomagnesemia

was present in 62 (40.26%) patients, while 92 (59.74%) had no hypomagnesemia. The overall mean serum magnesium concentration was 1.74 ± 0.44 mg/dL.

Serum magnesium levels differed significantly according to the presence of hepatic encephalopathy. Patients without HE

had a significantly higher mean serum magnesium level than those with HE (2.22 ± 0.28 vs. 1.56 ± 0.34 mg/dL; $p < 0.0001$). Hypomagnesemia was present in only 2.33% of patients

without HE compared with 54.95% of patients with HE ($p < 0.0001$) [Table 3].

Table 2: Distribution of hepatic encephalopathy grades and serum magnesium status

Parameter	n	Percentage (%)
Hepatic encephalopathy grade		
Grade 0	43	27.92
Grade 1	18	11.69
Grade 2	40	25.97
Grade 3	32	20.78
Grade 4	21	13.64
HE status		
Without HE (Grade 0)	43	27.92
With HE (Grades 1–4)	111	72.08
Hypomagnesemia		
Absent	92	59.74
Present	62	40.26
Serum magnesium, mean $\pm$ SD (mg/dL)	1.74 ± 0.44	—

Table 3: Association of serum magnesium with presence of hepatic encephalopathy

Serum magnesium parameter	Without HE (n=43)	With HE (n=111)	Total (n=154)	p-value
Hypomagnesemia, n (%)				
Absent	42 (97.67)	50 (45.05)	92 (59.74)	$< 0.0001^*$
Present	1 (2.33)	61 (54.95)	62 (40.26)	
Serum magnesium (mg/dL), mean $\pm$ SD	2.22 ± 0.28	1.56 ± 0.34	1.74 ± 0.44	$< 0.0001^\dagger$

Fisher's exact test; † Independent-samples t-test.

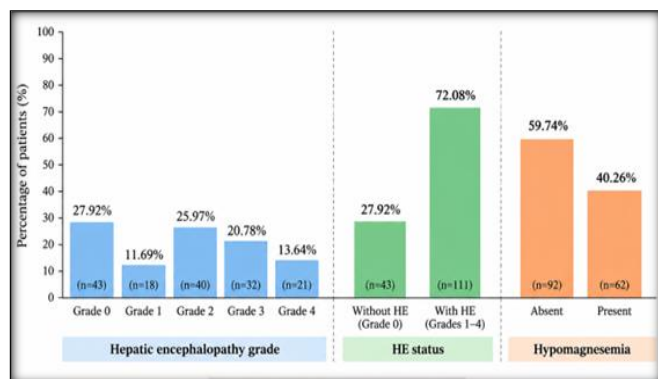


Figure 1: Distribution of hepatic encephalopathy grades and serum magnesium status.

There was a progressive reduction in serum magnesium concentration with increasing severity of hepatic encephalopathy. Mean serum magnesium decreased from 2.22 ± 0.28 mg/dL in Grade 0 to 1.87 ± 0.28 mg/dL in Grade 1, 1.68 ± 0.34 mg/dL in Grade 2, 1.44 ± 0.21 mg/dL in Grade

3, and 1.25 ± 0.19 mg/dL in Grade 4 ($p < 0.0001$). Similarly, the prevalence of hypomagnesemia increased progressively from 2.33% in Grade 0 to 90.48% in Grade 4 ($p < 0.0001$).

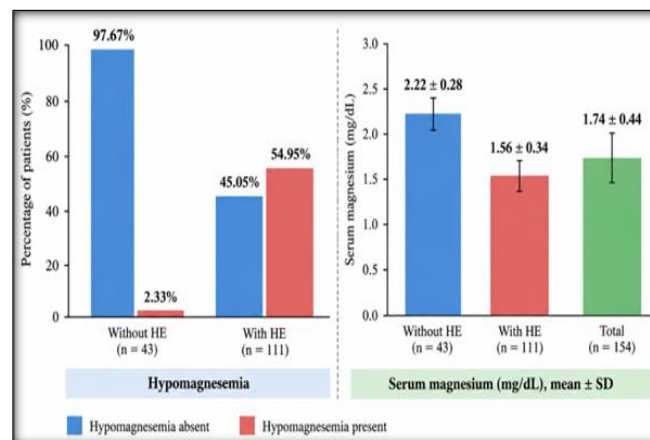


Figure 2: Association of serum magnesium with presence of hepatic encephalopathy.

Table 4: Association of serum magnesium with grade of hepatic encephalopathy

Parameter	Grade 0 (n=43)	Grade 1 (n=18)	Grade 2 (n=40)	Grade 3 (n=32)	Grade 4 (n=21)	p-value
No hypomagnesemia, n (%)	42 (97.67)	14 (77.78)	25 (62.50)	9 (28.13)	2 (9.52)	$< 0.0001^*$
Hypomagnesemia, n (%)	1 (2.33)	4 (22.22)	15 (37.50)	23 (71.88)	19 (90.48)	
Serum Mg, mean $\pm$ SD (mg/dL)	2.22 ± 0.28	1.87 ± 0.28	1.68 ± 0.34	1.44 ± 0.21	1.25 ± 0.19	$< 0.0001^\dagger$
Median (IQR), mg/dL	2.2 (2.1–2.4)	1.9 (1.7–1.975)	1.7 (1.4–1.8)	1.4 (1.3–1.6)	1.2 (1.1–1.3)	
Range, mg/dL	1.4–2.6	1.5–2.4	1.2–2.6	1.1–1.9	1.0–1.8	

Chi-square test; † one-way ANOVA.

Correlation analysis demonstrated a strong negative correlation between serum magnesium concentration and HE grade (Spearman $r = -0.792$, $p < 0.0001$), indicating that

serum magnesium levels decreased substantially with increasing severity of hepatic encephalopathy.

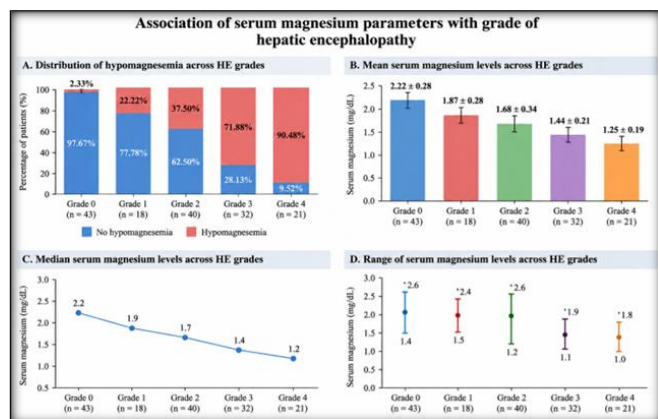


Figure 3: Association of serum magnesium with grade of hepatic encephalopathy

DISCUSSION

Hepatic encephalopathy (HE) is an important complication of cirrhosis and is associated with considerable morbidity, impaired quality of life, recurrent hospitalization, and poor clinical outcomes. The global burden of cirrhosis remains substantial, emphasizing the importance of identifying potentially modifiable factors associated with its complications.^[9,10] Current guidelines recognize HE as a spectrum ranging from subtle neurocognitive abnormalities to overt disorientation and coma, and recommend systematic assessment of severity and precipitating factors.^[11]

In the present study, 154 patients with liver cirrhosis were evaluated, with a mean age of 50.81 ± 11.30 years and a predominance of males (77.27%). Hepatic encephalopathy was present in 72.08% of patients, with Grade 2 being the most frequent overt HE grade. Contemporary literature emphasizes the heterogeneous clinical presentation of HE and the importance of recognizing potentially reversible metabolic and nutritional abnormalities during its assessment.^[12] Inflammation and systemic metabolic disturbances may interact with hyperammonemia and contribute to the neurological manifestations of cirrhosis.^[13,14]

A major finding of the present study was the significantly lower serum magnesium concentration among patients with HE compared with those without HE (1.56 ± 0.34 vs. 2.22 ± 0.28 mg/dL; $p < 0.0001$). Hypomagnesemia was present in 54.95% of patients with HE compared with only 2.33% of those without HE. These findings suggest an association between magnesium deficiency and the presence of hepatic encephalopathy. This is clinically relevant because metabolic abnormalities and nutritional deficiencies are increasingly recognized as potentially important contributors to the clinical course of cirrhosis and HE.^[15,16]

An important observation was the progressive decline in serum magnesium with increasing HE severity. Mean magnesium levels decreased from 2.22 ± 0.28 mg/dL in Grade 0 to 1.25 ± 0.19 mg/dL in Grade 4. Similarly, the prevalence of hypomagnesemia increased from 2.33% in Grade 0 to 90.48% in Grade 4. A strong negative correlation was demonstrated between serum magnesium concentration and HE grade ($r = -0.792$, $p < 0.0001$). Current HE guidance

also recommends correction of demonstrated or suspected micronutrient deficiencies because such deficiencies may compound HE.^[17]

However, the relationship between magnesium and HE remains incompletely established. Some contemporary studies have reported a high prevalence of hypomagnesemia in chronic liver disease without demonstrating a significant difference across HE grades.^[18] Such variation may reflect differences in study populations, severity of underlying liver disease, nutritional status, alcohol exposure, renal function, medication use, and definitions of magnesium deficiency. Therefore, the present findings establish a strong association, but they do not demonstrate that hypomagnesemia causes or aggravates HE.

The present study was limited by its single-centre, cross-sectional design, which prevents assessment of temporal or causal relationships. Dietary magnesium intake, intracellular magnesium status, medications, and other potential confounding factors were not comprehensively evaluated. Nevertheless, the marked progressive reduction in serum magnesium across HE grades suggests that serum magnesium may represent a simple biochemical parameter worthy of further investigation in cirrhotic patients. Prospective multicentre studies should determine whether magnesium independently predicts HE severity and whether correction of documented magnesium deficiency influences clinical outcomes.

CONCLUSION

Serum magnesium levels were significantly lower in cirrhotic patients with hepatic encephalopathy and showed a strong negative correlation with increasing HE grade. Hypomagnesemia became progressively more frequent with increasing severity of encephalopathy. These findings indicate that serum magnesium is significantly associated with the presence and severity of HE. Routine assessment of magnesium may therefore be useful as part of the biochemical evaluation of patients with cirrhosis, although prospective studies are required to determine its independent prognostic and therapeutic significance.

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Conflicts of interest

There are no conflicts of interest.

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