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# FREQUENCY OF HYPOMAGNESEMIA IN PATIENTS WITH DECOMPENSATED CHRONIC LIVER DISEASE PRESENTING WITH HEPATIC ENCEPHALOPATHY

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**Abstract**

***Objective:*** *To determine the frequency of hypomagnesemia in patients with decompensated chronic liver disease (CLD) presenting with hepatic encephalopathy (HE).*

***Methods:*** *This cross-sectional study was carried out at the Department of Medicine, Central Park Teaching Hospital, Lahore, from May 2024 to January 2025. We enrolled 163 patients with hepatic encephalopathy caused by decompensated CLD. Patients having serum magnesium levels <1.46 mg/dL were labelled as having hypomagnesemia.*

***Results:*** *The mean age was 48.3 (±12.6) years. Most were male, with 106 males (65.1%) and 57 females (34.9%). Regarding the severity of hepatic encephalopathy (HE), 71.2% of participants (116 individuals) were classified as Stage I-II, while 28.8% (47 individuals) were classified as Stage III-IV. Hypomagnesemia was present in 105 (64.4%) participants. Among patients with Stage I-II HE, 69 out of 105 (59.5%) had hypomagnesemia, while 47 out of 58 (40.5%) did not. In contrast, for those with Stage III-IV HE, a much higher proportion, 36 out of 105 (76.6%), experienced hypomagnesemia, compared to only 11 out of 58 (23.4%) who did not (p-value 0.03).*

***Conclusion:*** *Hypomagnesemia is very common in decompensated CLD patients. Moreover, the incidence of hypomagnesemia is directly linked to the severity of hepatic encephalopathy*

## INTRODUCTION

Liver cirrhosis refers to the progressive scarring and fibrosis of liver tissue, characterized by the formation of regenerative nodules interspersed within fibrous bands, typically resulting from chronic liver disorders.[1] This condition is a major contributor to illness and death among individuals suffering from chronic liver diseases. In 2019, it was reported that cirrhosis accounted for approximately 2.4% of global fatalities.[2] As of 2017, the estimated number of individuals living with compensated cirrhosis stood at around 112 million, whereas the figure for those with decompensated cirrhosis was close to 10.6 million.[3]

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Hepatic encephalopathy (HE) is a complex neurological disorder that occurs in individuals with advanced cirrhosis, leading to diminished brain function.[4, 5] The precise mechanisms underlying HE remain unclear, with several potential pathways thought to contribute to its onset and worsening.[6] There are numerous recognized factors that can trigger HE, and managing these factors effectively may help to reduce the severity of the condition and enhance cognitive function.[7]

Recent studies indicate that imbalances in electrolyte levels, along with trace elements like magnesium ($Mg^{2+}$), may significantly influence the onset and severity of hepatic encephalopathy (HE).[8, 9] Magnesium is a crucial intracellular cation that is essential for neuromuscular signaling and the proper operation of the central nervous system. Many patients with cirrhosis exhibit hypomagnesemia, characterized by low magnesium levels. This deficiency may stem from several factors, including malnutrition, issues with trace element absorption, decreased albumin production, and the use of diuretics. Collectively, these factors can lead to magnesium deficiency, which is important for both hepatic health and neurological function.[10]

The aim of the present study was to determine the frequency of hypomagnesemia in patients with decompensated liver disease presenting with HE.

## METHODS:

This cross-sectional study was carried out at the Department of Medicine, Central Park Teaching Hospital Lahore, from May 2024 to January 2025. We enrolled 163 patients with hepatic encephalopathy caused by DCLD, all over 18 years old, of either gender. Hepatic encephalopathy was diagnosed based on the appearance of neuropsychiatric symptoms in patients with clinically diagnosed liver cirrhosis, and graded according to the West Haven criteria, after ruling out other causes of altered sensorium through clinical assessment or investigations. Patients with DCLD who showed no signs of HE, or those with HE caused by sepsis or uremia, were excluded.

After taking aseptic measures, three cc of venous blood were sent to the hospital lab and centrifuged at 5000 rpm for 30 minutes to separate serum. Serum magnesium levels, which were assessed using commercially available kits. Patients having serum magnesium levels <1.46 mg/dL were labelled as having hypomagnesemia.

## RESULTS:

The study sample of 163 participants has a mean age of 48.3 years (±12.6). Most were male, with 106 males (65.1%) and 57 females (34.9%). The average Body Mass Index (BMI) is 24.7 $kg/m^2$ (±1.29). Common signs and symptoms include ascites in 28 cases (17.2%), anemia in 35 cases (21.5%), and constipation in 39 cases (23.9%). Fever was reported by 25 participants (15.3%), and gastrointestinal bleeding was observed in 36 cases (22.1%). Regarding hepatic encephalopathy (HE), 71.2% (116 individuals) were classified as Stage I-II, while 28.8% (47 individuals) were Stage III-IV. Hypomagnesemia was found in 105 participants (64.4%), with 58 participants (35.6%) not affected (Table 1).

Examining the link between hypomagnesemia and the severity of hepatic encephalopathy (HE) revealed significant results. Among patients with Stage I-II HE, 69 out of 105 (59.5%) had hypomagnesemia, while 47 out of 58 (40.5%) did not. In contrast, for those with Stage III-IV HE, a much higher proportion, 36 out of 105 (76.6%), experienced hypomagnesemia, compared to only 11 out of 58 (23.4%) who did not. The association showed a p-value of 0.03, indicating statistical significance. These findings suggest a clear correlation between HE severity and hypomagnesemia (Table 2).

**Table 1. Baseline Characteristics (N=163).**

| | |
|---|---|
| Age | 48.3±12.6 |
| Male/Female Gender | 106 (65.1%) / 57 (34.9%) |
| Body Mass Index (BMI) ($Kg/m^2$) | 24.7±1.29 |
| ***Signs and Symptoms*** | |
| Ascites | 28 (17.2%) |
| Anemia | 35 (21.5%) |

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| | |
|---|---|
| Constipation | 39 (23.9%) |
| Fever | 25 (15.3%) |
| GI Bleeding | 36 (22.1%) |
| ***Severity of HE (%)*** | |
| Stage I-II | 116 (71.2%) |
| Stage III-IV | 47 (28.8%) |
| ***Hypomagnesemia (%)*** | |
| Yes | 105 (64.4%) |
| No | 58 (35.6%) |

**Table 2. Association of Hypomagnesemia with Severity of HE.**

| **Severity of HE** | **Hypomagnesemia** | | **P-value** |
|---|---|---|---|
| | **Yes (N=105)** | **No (N=58)** | |
| **Stage I-II** | 69 (59.5%) | 47 (40.5%) | **0.03** |
| **Stage III-IV** | 36 (76.6%) | 11 (23.4%) | |

**DISCUSSION:**

HE is a recognized neuropsychiatric complication associated with liver cirrhosis, commonly observed in patients with chronic liver disease. It occurs due to dysfunction of the central nervous system, with hypomagnesemia identified as one of the contributing risk factors.[11, 12] This research aimed to evaluate the prevalence of hypomagnesemia in individuals with chronic liver disease (CLD) and to explore its correlation with the severity of hepatic encephalopathy (HE). In our findings, approximately 64.4% of patients exhibited low magnesium levels, and a significant relationship was observed between hypomagnesemia and the advancement of HE.

In a hospital-based research study conducted in Bareilly, India, Das and colleagues aimed to investigate serum magnesium levels in patients suffering from liver cirrhosis. The study involved comparing these levels with those in a healthy control group. The research included 50 patients diagnosed with alcohol-induced liver cirrhosis, referred to as Group I, and a control group of 50 individuals with no liver disease, labeled as Group II. The results revealed that patients in Group I exhibited significantly lower serum magnesium levels compared to the control group, with a highly significant statistical difference ($p<0.0001$).[13] Gurudevarahalli and colleagues carried out a hospital-based investigation examining serum magnesium levels in patients with liver conditions. Their findings indicated that individuals with cirrhosis of the liver tended to have reduced serum magnesium concentrations. Specifically, among a group of 25 patients suffering from chronic liver disease, 23 exhibited decreased magnesium levels in their blood.[14] Kar et al. conducted a study in Andhra Pradesh, India, to evaluate micronutrient imbalances in liver cirrhosis. They measured serum magnesium in 34 compensated and 31 decompensated patients, compared to 36 healthy controls, using post hoc ANOVA. Results showed reduced magnesium levels in cirrhosis patients versus controls ($p<0.05$), but no significant difference between compensated and decompensated groups ($p>0.05$).[15]

Researchers from Japan, led by Arakawa et al., conducted a comprehensive study examining serum levels of various trace elements, including magnesium, in different patient groups. The study compared individuals diagnosed with chronic hepatitis, liver cirrhosis, or primary biliary cirrhosis with healthy control subjects. Results indicated that magnesium levels tend to decline as liver disease progresses, with the most significant reductions observed in advanced stages such as cirrhosis.[16]

The article did not thoroughly account for all variables that may impact magnesium levels, including diet, medication use like diuretics, and kidney health. Accordingly, it is advisable to conduct larger, regionally diverse studies across Pakistan to validate these preliminary results and investigate if

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magnesium supplements can enhance recovery from hepatic encephalopathy.

**CONCLUSION:**

Hypomagnesemia is very common in decompensated CLD patients. Moreover, the incidence of hypomagnesemia is directly linked to the severity of hepatic encephalopathy.

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