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A Study Correlating Serum Magnesium and Child Pugh Score in Liver Cirrhosis

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HIGHLIGHTS

1. Serum magnesium levels decrease in cirrhosis.
2. Lower magnesium correlates with higher Child-Pugh.
3. Magnesium deficiency indicates worsening liver function.
4. Child-Pugh score predicts cirrhosis severity.
5. Magnesium monitoring aids in cirrhosis management.

ARTICLE INFO

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ABSTRACT

Introduction: Liver cirrhosis, a chronic condition characterized by widespread hepatic fibrosis, is associated with significant morbidity and mortality. Magnesium, an essential mineral for numerous physiological processes, has been shown to decrease in patients with advanced liver disease. This study aimed to investigate the correlation between serum magnesium levels and the Child-Pugh score in patients with liver cirrhosis. **Objectives:** To evaluate the relationship between serum magnesium levels and the severity of liver cirrhosis as measured by the Child-Pugh score. **Methods:** A prospective study was conducted on 105 liver cirrhosis patients. Demographic, clinical, and biochemical data were collected. Serum magnesium levels were measured, and the severity of cirrhosis was assessed using the Child-Pugh score. Statistical analysis was performed to determine the correlation between magnesium levels and cirrhosis severity. **Results:** The study included 105 patients with a mean age of 50.13 years, with 90.5% being male. Alcohol was the primary cause of cirrhosis in 86.7% of cases. Ascites was present in 90.5% of patients, and 27.6% experienced hepatic encephalopathy. Serum magnesium levels were found to decrease as the severity of cirrhosis increased, with a significant correlation observed between magnesium levels and the Child-Pugh score ($p < 0.05$). **Conclusion:** This study demonstrated a significant correlation between serum magnesium levels and the severity of liver cirrhosis as assessed by the Child-Pugh score. Monitoring serum magnesium may provide additional insights into disease severity and progression in cirrhosis patients.

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INTRODUCTION

Chronic liver inflammation and injury lead to cirrhosis, characterized by widespread hepatic fibrosis and the replacement of healthy liver tissue with regenerating nodules[1]. It represents the final stage of chronic liver disease (CLD) and can arise from various causes, including viral hepatitis, alcohol abuse, non-alcoholic fatty liver disease (NAFLD), and autoimmune conditions. The global burden of cirrhosis varies across regions, genders, racial groups, and socioeconomic statuses and has evolved over time. According to the 2017 Global Burden of Disease (GBD) Study, around 112 million cases of compensated cirrhosis were reported globally, with an age-standardized prevalence of 1,395 per 100,000 people. In 2017, cirrhosis accounted for 2.4% of global deaths. Risk factors for cirrhosis progression include age, comorbidities (such as HIV and HCV co-infection), and male sex (except in alcoholic liver disease, where females progress faster)[2]. Disease progression varies based on underlying causes, treatment availability, and the persistence of liver damage.

The Child-Pugh and Model for End-Stage Liver Disease (MELD) scores are commonly used to assess the stage and mortality risk in cirrhosis[3]. A MELD score of 12 or higher indicates an increased risk of cirrhosis-related complications, while a Child-Pugh grade B (7-9 points) suggests early hepatic decompensation. Magnesium, the fourth most abundant mineral in the body, is essential for physiological functions, including ion regulation and energy production as an enzyme cofactor[4]. Most magnesium is stored in soft tissues and bones, with diet and kidney function playing crucial roles in regulating magnesium levels.

Magnesium plays a crucial role in liver function and may contribute to the development of chronic liver disease[5]. A deficiency in magnesium is a common marker of liver cirrhosis. Studies have shown that serum magnesium levels tend to decrease as the severity of liver disease increases, as classified by the Child-Pugh score. Recent research suggests that higher magnesium intake may lower the risk of developing fatty liver disease and reduce mortality from liver disease, particularly in individuals who consume alcohol[6]. This relationship between the severity of liver cirrhosis and serum magnesium levels in affected patients, providing insights into the role of magnesium in liver health[7].

The study aims to evaluate the correlation between serum magnesium levels and the severity of liver cirrhosis using the Child-Pugh score. By assessing this relationship, the study seeks to determine how magnesium deficiency impacts the progression and prognosis of cirrhosis, potentially offering insights into disease management and patient outcomes.

METHODOLOGY

After ethics committee approval, patients meeting the inclusion criteria were enrolled after informed consent. Demographic details, illness history, and relevant information were collected, followed by a thorough clinical examination. Routine investigations, including serum magnesium, were conducted. The Child-Pugh score was calculated using clinical and lab data, and statistical analyses were performed.

RESULTS

The clinical, and etiological data for a group of 105 patients. The age of patients ranged from 24 to 88 years, with a mean age of 50.13 years (±14.09). The majority of patients were male (90.5%), while females accounted for only 9.5%. About 33.3% of patients had comorbidities, with diabetes mellitus being the most common (28.6%), followed by hypertension (15.2%). Regarding the cause of liver disease, alcohol was the predominant etiology, affecting 86.7% of patients, while 11.4% had cryptogenic cirrhosis. Ascites severity varied among patients: 9.5% had no ascites, 19% had mild ascites, 45.7% had moderate ascites, and 25.7% had gross ascites. The data highlights the male predominance, alcohol-related etiology, and the significant presence of comorbidities like diabetes and hypertension among patients.

Table 1: Hepatic Encephalopathy

Hepatic Encephalopathy	Frequency	Percentage
No	76	72.4
Grade 2	9	8.6
Grade 3	14	13.3
Grade 4	6	5.7

Among the research participants, 27.6% experienced hepatic encephalopathy. Specifically, 8.6% had Grade 2 encephalopat-hy, 13.3% had Grade 3, and 5.7% had Grade 4, reflecting varying severity levels within the group.

Table 2: Child Pugh Score

Child Pugh Score	Frequency	Percentage
A	9	8.6
B	35	33.3
C	61	58.1

The table presents the distribution of patients based on their Child-Pugh score, which assesses liver disease severity. of the patients, 8.6% were classified as Child-Pugh A (mild disease), 33.3% as Child-Pugh B (moderate disease), and 58.1% as Child-Pugh C (severe disease).

Table 3: Hematology

Hematology	Minimum	Maximum	Mean	Std. Deviation
HB	2.83	15.80	10.0089	2.60859
TLC	2680	34840	10391.24	5926.488
PLT	0.17	4.49	1.5106	0.88062
PT	11.4	67.2	21.572	9.8505
APTT	25.8	180.0	48.253	28.8601
INR	0.78	6.47	1.7095	0.96644

The table summarizes hematology data for patients, showing a wide range of values. Hemoglobin (HB) levels ranged from 2.83 to 15.80 g/dL, with a mean of 10.00. Total leukocyte count (TLC) varied from 2,680 to 34,840 cells/µL, and platelet count (PLT) ranged from 0.17 to 4.49 lakh/µL. Prothrombin time (PT), activated partial thromboplastin time (APTT), and INR also displayed broad ranges, reflecting clotting function abnormalities in the patients.

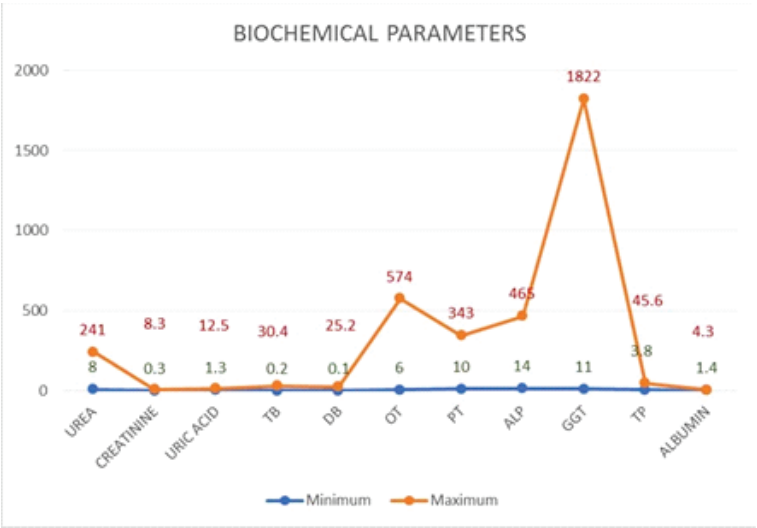

Figure 1: Biochemical Parameters

The table presents biochemical parameters with their respective ranges, means, and standard deviations. Urea levels ranged from 8 to 241 mg/dL (mean: 32.66), creatinine from 0.3 to 8.3 mg/dL (mean: 1.15), and uric acid from 1.3 to 12.5 mg/dL (mean: 4.09). Total bilirubin (TB) varied from 0.2 to 30.4 mg/dL, direct bilirubin (DB) from 0.1 to 25.2 mg/dL, and liver enzymes (OT, PT, ALP, GGT) showed extensive ranges, reflecting variability in liver function and other metabolic processes. Total protein (TP) and albumin levels also varied, indicating differences in protein synthesis and liver function among patients.

Table 4: Electrolyte

Electrolytes	Minimum	Maximum	Mean	Std. Deviation
Magnesium	0.40	2.10	1.4578	0.27569
Sodium	112	145	131.26	6.202
Potassium	2.2	6.7	4.210	0.8709
Chloride	76	116	100.05	7.028

The mean serum levels among research participants were as follows: magnesium was 1.4578 ± 0.27569 mg/dL, sodium was 131.26 ± 6.202 mg/dL, potassium was 4.210 ± 0.8709 mg/dL, and chloride was 100.05 ± 7.028 mg/dL. These values represent the average concentrations of these electrolytes in the study group.

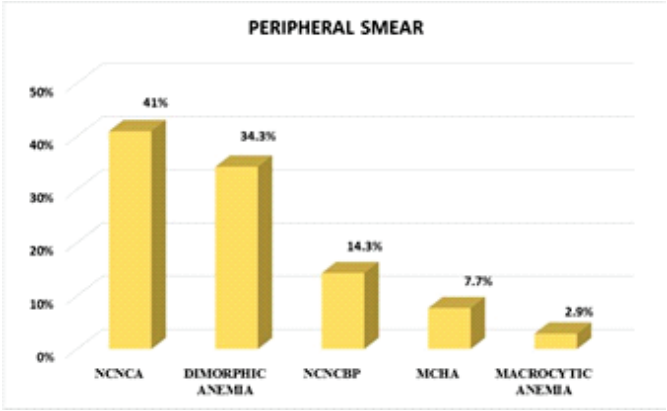

Figure 2: Peripheral Smear

Among individuals with liver cirrhosis, 41% had normocytic normochromic anemia, 34.3% had dimorphic anemia, 14.3% had a normocytic normochromic blood picture, 7.7% had microcytic hypochromic anemia, and 2.9% had macrocytic anemia.

Table 5: Association of Serum Magnesium with Grades of Ascites

ASCITIS	SERUM MAGNESIUM		P value
	Mean	Std. Deviation	
NO	1.6030	0.11480	0.068
MILD	1.5460	0.35581	
MODERATE	1.4419	0.25403	
GROSS	1.3998	0.25963	

The table shows the association between serum magnesium levels and the severity of ascites. Mean serum magnesium decreased as ascites severity increased: 1.6030 mg/dL for no ascites, 1.5460 mg/dL for mild ascites, 1.4419 mg/dL for mod-erate ascites, and 1.3998 mg/dL for gross ascites. The p-value is 0.068, indicating that the association is not statistically significant.

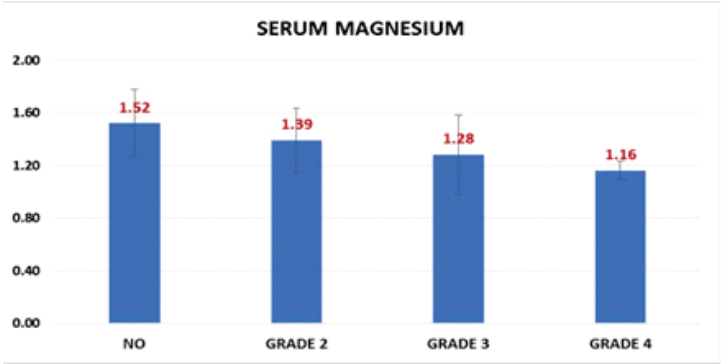

Figure 2: **Association of Serum Magnesium with Grades of Hepatic Encephalopathy**

The table shows the association between serum magnesium levels and the severity of ascites. Mean serum magnesium decreased as ascites severity increased: 1.6030 mg/dL for no ascites, 1.5460 mg/dL for mild ascites, 1.4419 mg/dL for moderate ascites, and 1.3998 mg/dL for gross ascites. The p-value is 0.068, indicating that the association is not statistically significant.

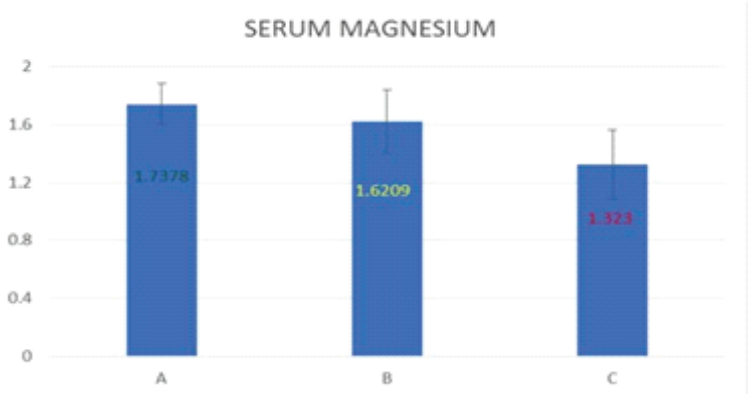

Figure 4 : Association of Serum Magnesium with Grades of Child Pugh Score

The study found that serum magnesium levels decreased with increasing liver cirrhosis severity according to the Child-Pugh score. There was a statistically significant correlation between blood magnesium levels and the severity of liver cirrhosis.

DISCUSSION

Magnesium is essential for various physiological and pathological processes, including energy metabolism, ion transport, and DNA repair. It maintains homeostasis through bone exchange, renal excretion, and gastrointestinal absorption. Deficiency in magnesium is associated with a range of conditions, such as respiratory issues, mental disorders, and cardiovascular diseases. Symptoms of magnesium deficiency include headaches, muscle spasms, arrhythmia, and osteoporosis. The liver, crucial for regulating trace elements like magnesium, often shows decreased magnesium levels in cirrhosis, a late stage of liver disease marked by permanent fibrosis. Historical studies, starting with Lim et al. in 1972, identified magnesium deficiency in cirrhosis patients. Magnesium deficiency is also linked to peripheral insulin resistance in alcoholic liver disease. Factors such as reduced dietary intake, increased renal excretion, and reduced absorption contribute to hypomagnesaemia in liver cirrhosis patients. However, some research has not confirmed a link between serum magnesium levels and cirrhosis, highlighting the need for further investigation[8].

The study participants had ages ranging from 24 to 88 years, with a mean age of 50.13 ± 14.09 years. Peng X et al. reported an average age of 58 years (range: 25–86 years), while SafariKish B et al. found a mean age of 64.1 ± 13.7 years. Chavan M et al. reported an average age of 46.9 years. Veena G et al. observed a mean age of 54.41 ± 7.75 years, with the majority in the 51-year age group (49.3%). Ali A et al. found a mean age of 51.1 ± 7.46 years. Das B et al. identified 36% of cases in the 47–54 year range, 32% in the 39–46 year range, 18% in the 55–62 year range, and 14% in the 31–38 year range. These findings align with the current study[99][10][11].

In this study, 9.5% of participants were female, and 90.5% were male. Peng X et al. reported 60.5% male patients, while Safari-Kish B et al. had 47.8% female and 52.2% male participants. Nangliya V et al. found 66% male and 34% female participants. Veena G et al. reported 62% male and 38% female participants. Ali A et al. found 65% male participants, and Das B et al. also noted a higher prevalence of liver cirrhosis in males. These results are consistent with the current study[12][13].

In the current study, 86.7% of liver cirrhosis cases were attributed to ethanol, 11.4% to cryptogenic disease, and 1% each to alcohol-induced with hepatitis B and autoimmune causes. Peng X et al. found 63.2% of patients had hepatitis B virus infection, 11.2% had alcoholic liver disease, 10.5% had cryptogenic liver disease, and 15.1% had other causes like hepatitis C or cholestatic liver disease. SafariKish B et al. identified hemosiderosis (4.4%), hepatitis C (8.9%), hepatitis B (7.8%), and cryptogenic disease (71.1%) as common causes. Nangliya V et al. reported 42.6% alcoholic, 20% NASH, 20.7% HBV, and 6.7% HCV cases. Veena G et al. found alcohol was the primary cause in 48.7% of those aged 51 to 60.

In terms of ascites, 90.5% of subjects had ascites: 19% mild, 45.7% moderate, and 25.7% gross. Peng X et al. reported ascites in 59.9% of cirrhotic patients, while Ali A et al. found 38% with ascites[11][12][13].

In the current study, 58.1% of patients had a Child-Pugh score of C, 33.3% had B, and 8.6% had A. SafariKish B et al. reported 38.9% in class A, 41.1% in B, and 20% in C. Nangliya V et al. found 34% in class A, 33.3% in B, and 32.7% in C, aligning with the current study's findings[14][15].

The average serum magnesium level in this study was 1.4578±0.27569. Lower magnesium levels were associated with higher Child-Pugh scores, reflecting greater liver cirrhosis severity. Peng X et al. noted a mean magnesium level of 0.69±0.12 mmol/L, with significant magnesium deficiency in classes B (68%) and C (83.3%). SafariKish B et al. reported a mean level of 1.84±0.3 mEq/L and a significant correlation between hypomagnesaemia and Child-Pugh classes B and C ($p=0.027$). Nangliya V et al. observed a marked decline in magnesium from class A to C, with a notable inverse relationship ($r=-0.35$; $p<0.001$). Veena G et al. recorded mean magnesium le-

-vels of 1.540±0.282 (Grade A), 1.423±0.23 (Grade B), and 1.186±0.298 (Grade C), with significant differences across grades (p=0.001). Ali A et al. found lower mean magnesium values in Child-Pugh class C. Das B et al. and Saxena T et al. also reported lower magnesium levels in cirrhosis patients. Patel M et al. highlighted that low magnesium levels are not solely due to alcohol but also other factors. Cirrhosis leads to reduced magnesium levels due to impaired liver function, malnutrition, and increased magnesium excretion, exacerbated by alcohol use and certain diuretics[10][11][16][17].

CONCLUSION

The study reveals a strong link between serum magnesium levels and the Child-Pugh score, reflecting liver disease severity. Consistent with previous research, low magnesium levels may indicate advanced liver cirrhosis. Larger, more diverse studies are needed to confirm these findings and explore potential treatments. Participants had a mean age of 50.13 years, with 90.5% male. Comorbidities affected 33.3% of patients, mainly diabetes (28.6%) and hypertension (15.2%). Alcohol was the primary cause of liver cirrhosis (86.7%), followed by cryptogenic causes (11.4%). Ascites was present in 90.5% of participants, with 27.6% having hepatic encephalopat hy. A Child-Pugh score of C was observed in 58.1% of individuals. Serum findings included a low albumin (mean 2.606 g/dL), high total bilirubin (mean 7.004 mg/dL), and low hemoglobin (mean 10 g/dL). The average serum magnesium level was 1.4578 mg/dL, with lower magnesium levels associated with higher Child-Pugh scores, hepatic encephalopa thy, and ascites severity.

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