

**Original article:**

## Assessment of correlation of serum Mg level with hepatic encephalopathy in cirrhotic patients

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### Abstract:

**Introduction:** Hepatic encephalopathy (HE) is a neurometabolic syndrome characterized by impaired brain function in patients with decompensated cirrhosis.<sup>1-3</sup> The pathogenesis of HE is not completely understood and several proposed pathways are implicated in the initiation and exacerbation of this syndrome. Magnesium is an essential component of human body and other mammals, whose role in liver cirrhosis and its complications is still a matter of research. There are contrary reports about their serum concentrations in patients with liver cirrhosis. Magnesium is associated with more than 300 enzymatic reactions involving energy metabolism and protein and nucleic acid synthesis

**Aim:** To assess the correlation of serum Mg level with hepatic encephalopathy in cirrhotic patients.

**Objectives:** To study the serum electrolyte levels in hepatic encephalopathy in cirrhotic patients (To assess the correlation of serum Mg level with hepatic encephalopathy in cirrhotic patients)

**Result :** The mean Serum Sodium, Potassium, Calcium, Chloride, Bicarbonate and Magnesium levels of the patients were  $134.6 \pm 3.64$ ,  $4.0 \pm 0.8$ ,  $8.8 \pm 1.3$ ,  $95.6 \pm 8.9$ ,  $22.3 \pm 5.7$  and  $1.2 \pm 0.2$  mEq/L, respectively. (Table No. 10) No correlation was observed. Correlation is significant at the 0.01 level (2-tailed). Correlation is significant at the 0.05 level (2-tailed). In our study we came to a conclusion that deficiency in the serum magnesium levels is associated with cirrhosis in alcoholic patients.

**Conclusions** In our study we came to a conclusion that deficiency in the serum magnesium levels is associated with cirrhosis in alcoholic patients.

### INTRODUCTION:

Hepatic encephalopathy (HE) is a neurometabolic syndrome characterized by impaired brain function in patients with decompensated cirrhosis.<sup>1-3</sup> The pathogenesis of HE is not completely understood and several proposed pathways are implicated in the initiation and exacerbation of this syndrome.<sup>3-5</sup> HE may be clinically apparent in as many as one third of cirrhotic patients and, if rigorously tested, up to two thirds have some degree of mild or subclinical HE.<sup>6</sup>

Ammonia plays a central role in HE as it crosses the blood brain barrier causing neurological insult mediated by a decrease in excitatory neurotransmission.<sup>7</sup> Multiple precipitating factors for HE have been recognized and if controlled, may alter the disease acuity and improve mental status.<sup>8</sup> The most common precipitating factors for

HE includes dehydration, acute kidney injury, non-adherence to medications (particularly non-absorbable disaccharides), constipation and infections.<sup>9-11</sup>

Magnesium is essential for many intracellular processes and structures in the human body, such as muscle contraction and relaxation, neuronal signal transduction, and conduction of the action potential in the myocardium<sup>12</sup>. Most of the body's magnesium is intracellular and less than 1% of the total is found in serum. Therefore, significant magnesium deficiency might be present even though the serum magnesium level is within normal limits. Magnesium deficiency has been associated with several systemic conditions, including metabolic syndrome, cerebrovascular diseases, malignancies, bacterial and fungal infections, osteoporosis, and liver cirrhosis<sup>13-16</sup>. Several studies demonstrated a higher prevalence of magnesium deficiency in patients with liver cirrhosis compared to the general population<sup>17-20</sup>. Suggested pathogenesis includes decreased magnesium intake, fat malabsorption, diuretic use, renal tubular acidosis, and increased serum levels of growth hormone and glucagon<sup>20</sup>. Patients with alcoholic liver cirrhosis were found to have decreased muscle mass and strength as well as lower magnesium and potassium content in muscle tissue as compared to an age-matched control group<sup>21,22</sup>. Magnesium levels were found to decrease as the severity of liver disease progressed (according to CHILD score)<sup>21</sup>, and treatment with spironolactone increased muscle strength and electrolytes<sup>21,23</sup>.

Magnesium is an essential component of human body and other mammals, whose role in liver cirrhosis and its complications is still a matter of research. There are contrary reports about their serum concentrations in patients with liver cirrhosis. Magnesium is associated with more than 300 enzymatic reactions involving energy metabolism and protein and nucleic acid synthesis<sup>24,25</sup>. Magnesium also involved in immunoglobulin synthesis, immune cell adherence, antibody dependent cytotoxicity, GM lymphocyte binding, Thelper B-cell adherence and additional responses<sup>26</sup>. Only 0.3% of total body magnesium exists in serum<sup>27-29</sup>. In spite of all this knowledge regarding importance of magnesium in human body, very little is known about magnesium metabolism in diseased states, in comparison to the extensive studies of calcium, sodium and potassium etc. Hence, the present study is planned to assess the correlation of serum Mg level with hepatic encephalopathy in cirrhotic patients

#### **AIM & OBJECTIVES:**

**Aim:** To assess the correlation of serum Mg level with hepatic encephalopathy in cirrhotic patients

#### **Objectives:**

1. To study the serum electrolyte levels in hepatic encephalopathy in cirrhotic patients
2. To assess the correlation of serum Mg level with hepatic encephalopathy in cirrhotic patients

#### **MATERIAL AND METHODS:**

**Study design:** Cross sectional analytical

**Study population:** Patients diagnosed with liver cirrhosis with hepatic encephalopathy visiting **Noor hospital**

**Study period:** 2 years

**Sample size:** 20

**Ethical clearance:** The study will be initiated after approval of Institutional Ethical committee.

**Selection criteria:** Patients diagnosed with liver cirrhosis with hepatic encephalopathy visiting **Noor hospital** will be subjected to the following inclusion and exclusion criteria.

**Inclusion criteria:**

1. Patients diagnosed with liver cirrhosis with hepatic encephalopathy visiting **NOOR HOSPITAL**
2. Patients of age 18 years or above of either gender.
3. Patients/Patients legally acceptable representative willing to give written informed consent to participate in the study.

**Exclusion criteria:**

1. Patients with active cancer.
2. Special populations such as pregnant women.
3. Individuals with mental retardation, dementia.
4. Current treatment with magnesium supplements.
5. Renal failure patients

Patients who will satisfy the above inclusion and exclusion criteria will be included in the study. Written informed consent will be taken from all patients.

**Study procedure:**

After taking consent, patient's demographic data will be collected. Data for the following variables will be collected:

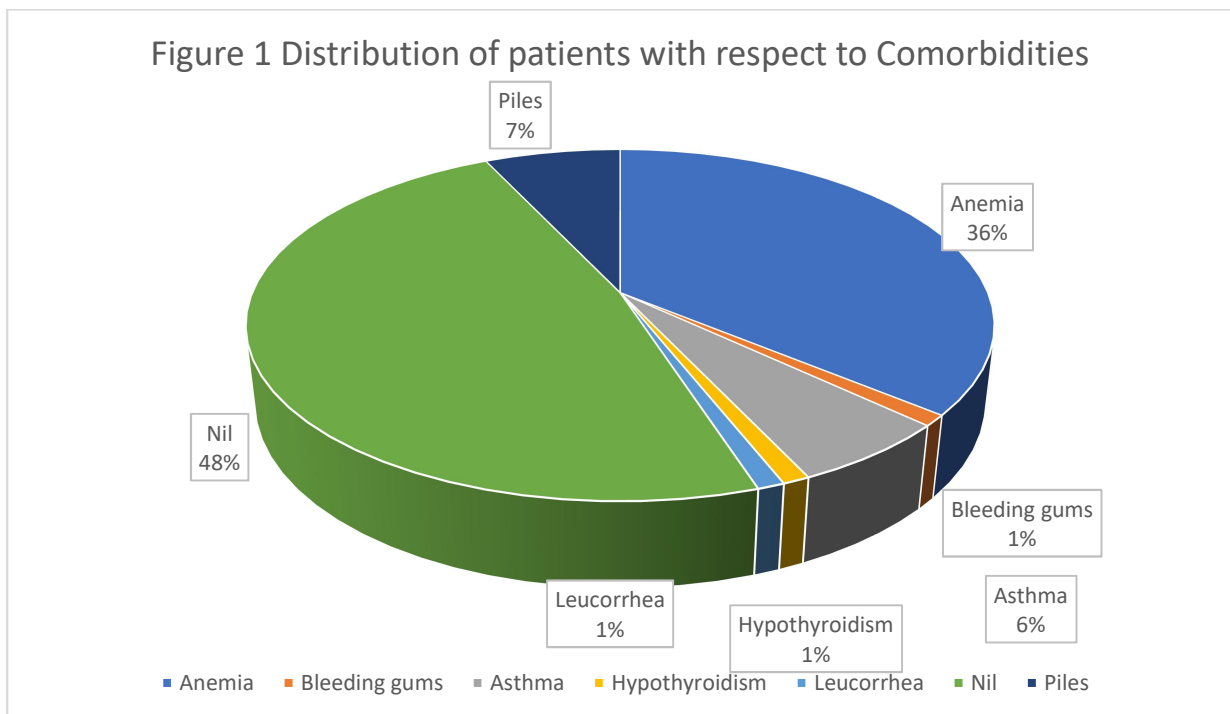
The following information regarding the patients will be collected:

S. No.	Variable	Method of measurement	Measurement scale	Descriptive statistics
1.	Age	Interview	Ratio	Mean, S.D.
2.	Gender	Interview	Nominal	Frequency, Proportion
3.	Occupation	Interview	Nominal	Frequency, Proportion
4.	Comorbidities	Examination	Ratio	Mean, S.D.
5.	Causes of liver cirrhosis	Record	Nominal	Frequency, Proportion
6.	Serum Electrolytes	Investigation	Ratio	Mean, S.D.
7.	Liver function test	Investigation	Ratio	Mean, S.D.
8.	Glasgow coma scale	Examination	Ordinal	Frequency, Proportion

**RESULTS:**

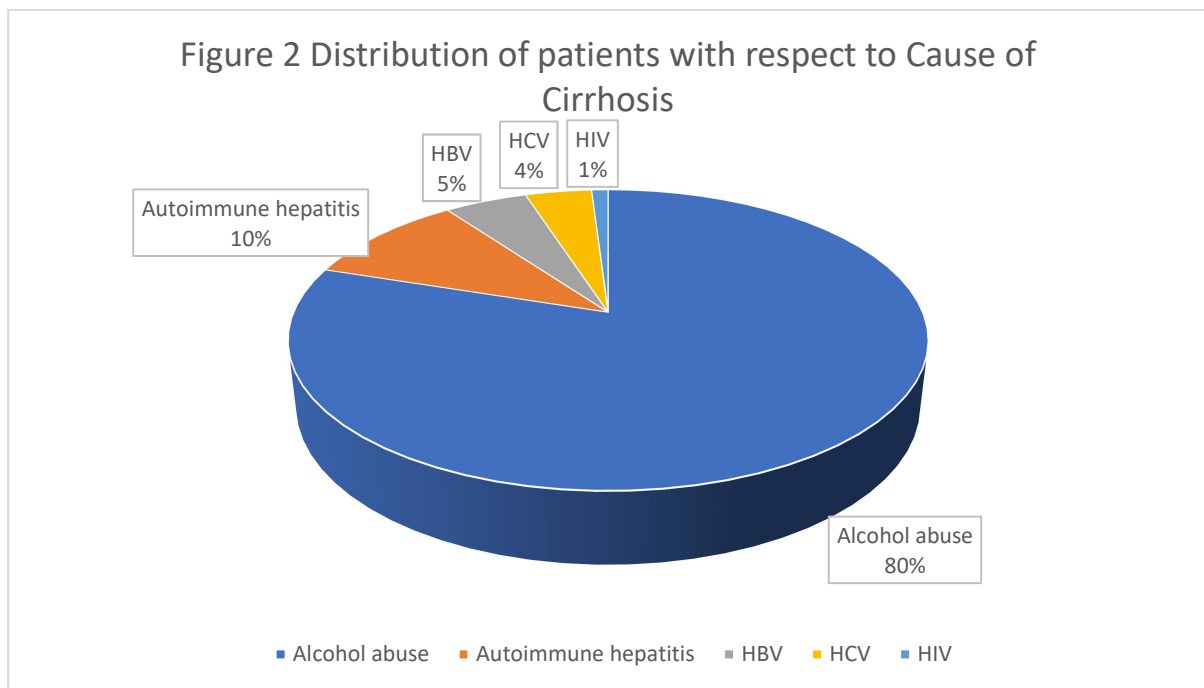
In the present study, 100 patients were included. All the patients were males. The mean Age of patients was 45.8 ± 13.3 years.

Table 1 Distribution of patients with respect to Comorbidities			
		Frequency	Percent
<b>Comorbidity</b>	Anemia	36	36
	Bleeding gums	1	1.0
	Asthma	6	6.0
	Hypothyroidism	1	1.0
	Leucorrhea	1	1.0
	Nil	48	48.0
	Piles	7	7.0
	Total	100	100.0



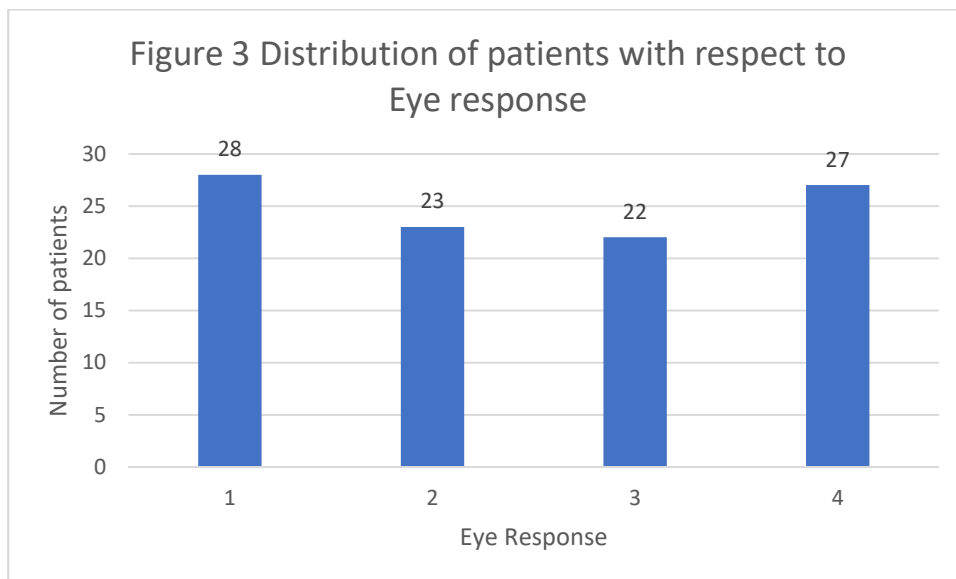
52 patients suffered from comorbidities. Most of the patients were suffering from anemia (36), followed by piles (7) and asthma (6).

		Frequency	Percent
<b>Cause of Cirrhosis</b>	Alcohol abuse	80	80.0
	Autoimmune hepatitis	10	10.0
	HBV	5	5.0
	HCV	4	4.0
	HIV	1	1.0
	Total	100	100.0



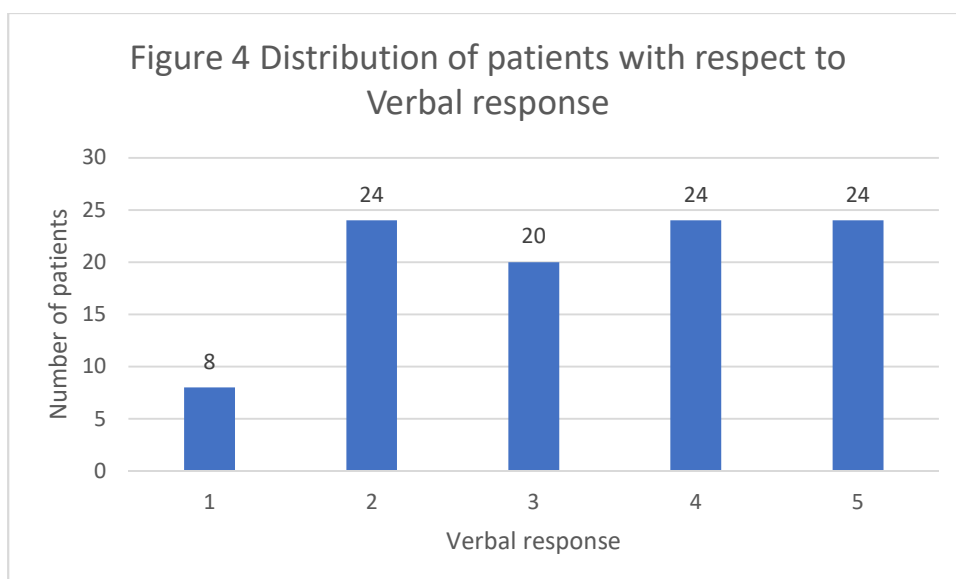
The most common cause of Cirrhosis was Alcohol abuse (80%) followed by Autoimmune hepatitis (10%) and HBV infection (5%).

		Frequency	Percent
<b>Eye response</b>	1	28	28.0
	2	23	23.0
	3	22	22.0
	4	27	27.0
	Total	100	100.0



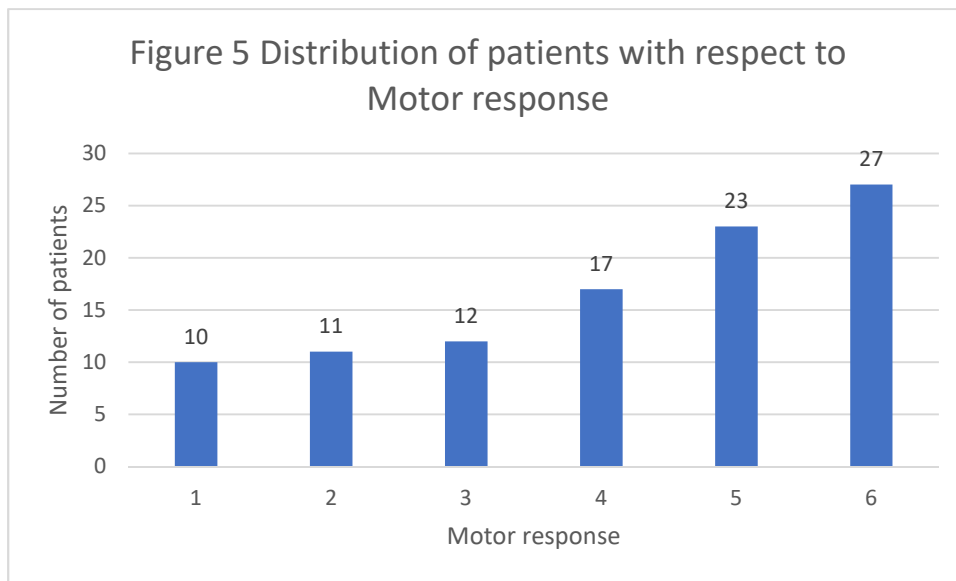
The most common eye response of the patients was 1 (28%) followed by 4 (27%).

		Frequency	Percent
Verbal response	1	8	8.0
	2	24	24.0
	3	20	20.0
	4	24	24.0
	5	24	24.0
	Total	100	99.0



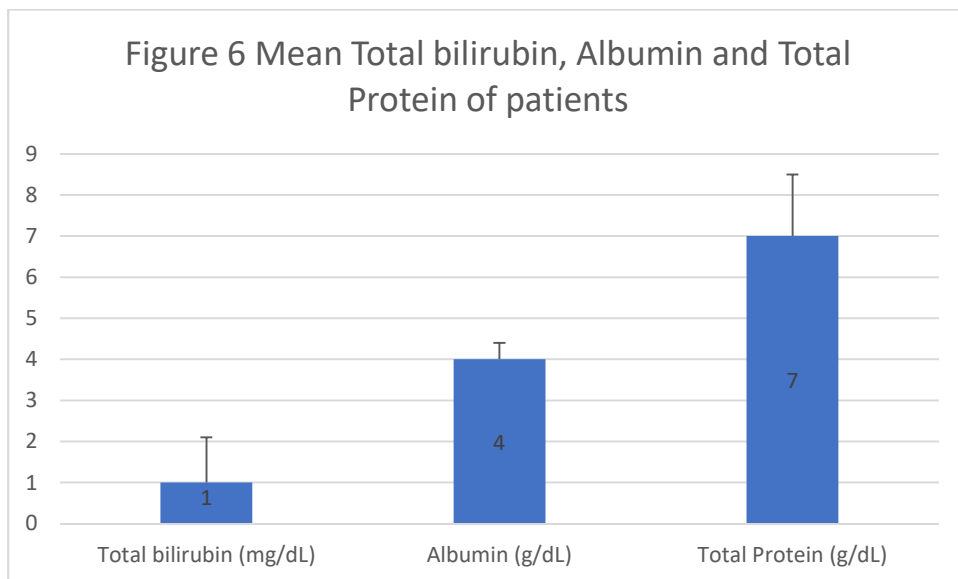
The most verbal response of the patients was 2, 4 and 5 (24% each).

		Frequency	Percent
Motor response	1	10	10.0
	2	11	11.0
	3	12	12.0
	4	17	17.0
	5	23	23.0
	6	27	27.0
	Total	100	100.0



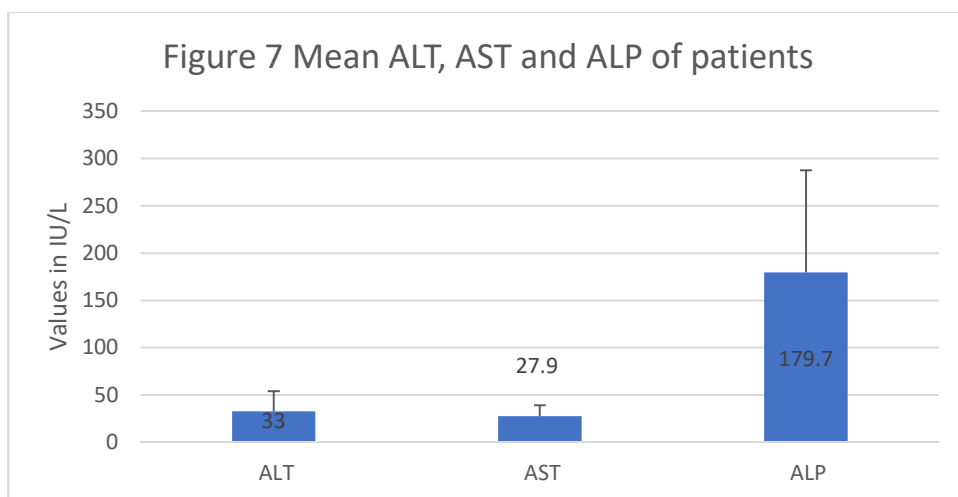
The most common Motor response of the patients was 6 (27%) followed by 5 (23%) and 4 (17%). The mean Glasgow coma scale of the patients was  $9.9 \pm 1.8$

	Mean	Std. Deviation
Total bilirubin (mg/dL)	1.0	1.1
Albumin (g/dL)	4.0	0.4
Total Protein (g/dL)	7.0	1.5



The mean Total bilirubin, Albumin and Total Protein of the patients was  $1.0 \pm 1.1$ mg/dL,  $4.0 \pm 0.4$ g/dL and  $7 \pm 1.5$ g/dL, respectively.

	Mean	Std. Deviation
ALT (IU/L)	33.0	21.0
AST (IU/L)	27.9	11.5
ALP (IU/L)	179.7	107.9



The mean ALT, AST and ALP of the patients was  $33.0 \pm 21$  mg/dL,  $27.9 \pm 11.5$  g/dL and  $179.7 \pm 107.9$ IU/L, respectively.



	Mean	Std. Deviation
S. Urea (mg/dL)	43.4	12.1
S. Creatinine (mg/dL)	1.5	0.3
S. Uric acid (mg/dL)	5.6	0.9

The mean S. Urea, S. Creatinine and S. Uric acid of the patients was  $43.4 \pm 12.1$  mg/dL,  $1.5 \pm 0.3$ mg/dL and  $5.6 \pm 0.9$  mg/dL, respectively.

	Mean	Std. Deviation
Serum Sodium (mEq/L)	134.6	3.6
Serum Potassium (mEq/L)	4.0	0.8
Serum Calcium (mEq/L)	8.8	1.3
Serum Chloride (mEq/L)	95.6	8.9
Serum Bicarbonate (mEq/L)	22.3	5.7
Serum Magnesium (mEq/L)	1.2	0.2

The mean Serum Sodium, Potassium, Calcium, Chloride, Bicarbonate and Magnesium levels of the patients were  $134.6 \pm 3.6$ ,  $4.0 \pm 0.8$ ,  $8.8 \pm 1.3$ ,  $95.6 \pm 8.9$ ,  $22.3 \pm 5.7$  and  $1.2 \pm 0.2$  mEq/L, respectively.

## DISCUSSION:

Hepatic encephalopathy (HE) is a neurometabolic syndrome characterized by impaired brain function in patients with decompensated cirrhosis.<sup>1-3</sup> The pathogenesis of HE is not completely understood and several proposed pathways are implicated in the initiation and exacerbation of this syndrome.<sup>3-5</sup> HE may be clinically apparent in as many as one third of cirrhotic patients and, if rigorously tested, up to two thirds have some degree of mild or subclinical HE.<sup>6</sup> Ammonia plays a central role in HE as it crosses the blood brain barrier causing neurological insult mediated by a decrease in excitatory neurotransmission.<sup>7</sup> Multiple precipitating factors for HE has been recognized and if controlled, may alter the disease acuity and improve mental status<sup>8</sup> (Table No. 1) In the present study, 100 patients were included. All the patients were males. The mean Age of patients was  $45.8 \pm 13.3$  years. (Fig. no.1) 52 patients suffered from comorbidities. Most of the patients were suffering from anemia (36), followed by piles (7) and asthma (6). (Table No. 2) The most common cause of Cirrhosis was Alcohol abuse (80%) followed by Autoimmune hepatitis (10%) and HBV infection (5%). Fig. no.3) here our study is in accordance with the study done by Vilstrup H, et al The most common eye response of the patients was 1 (28%) followed by 4 (27%). again here our study is in accordance with the study done by Pantham

Getal( Table No. 4) The most verbal response of the patients was 2, 4 and 5 (24% each).( Table No. 5) The most common Motor response of the patients was 6 (27%) followed by 5 (23%) and 4 (17%). The mean Glasgow coma scale of the patients was  $9.9 \pm 1.8$ (Fig. no.6) The mean Total bilirubin, Albumin and Total Protein of the patients was  $1.0 \pm 1.1$ mg/dL,  $4.0 \pm 0.4$ g/dL and  $7 \pm 1.5$ g/dL, respectively.(Fig. no.7) The mean ALT, AST and ALP of the patients was  $33.0 \pm 21$  mg/dL,  $27.9 \pm 11.5$  g/dL and  $179.7 \pm 107.9$ IU/L, respectively.(Fig. no. 8) The mean S. Urea, S. Creatinine and S. Uric acid of the patients was  $43.4 \pm 12.1$  mg/dL,  $1.5 \pm 0.3$ mg/dL and  $5.6 \pm 0.9$  mg/dL, respectively.(Fig. no. 9b) The mean Serum Sodium, Potassium, Calcium, Chloride, Bicarbonate and Magnesium levels of the patients were  $134.6 \pm 3.64$ ,  $4.0 \pm 0.8$ ,  $8.8 \pm 1.3$ ,  $95.6 \pm 8.9$ ,  $22.3 \pm 5.7$  and  $1.2 \pm 0.2$ mEq/L, respectively.( Table No. 10) No correlation w \*\*. Correlation is significant at the 0.01 level (2-tailed). Correlation is significant at the 0.05 level (2-tailed).

### CONCLUSION:

In our study we came to a conclusion that deficiency in the serum magnesium levels is associated with cirrhosis in alcoholic patients most of the studies like the study done by Iwasa M etal and Shechter M. etal are in accordance with us however multicentric studies with larger sample size are required to come to a conclusion Therefore, significant magnesium deficiency might be present even though the serum magnesium level is within normal limits

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Was informed consent obtained from the subjects involved in the study? YES

For any images presented appropriate consent has been obtained from the subjects: NA

Plagiarism Checked: Urkund Software

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