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Original Research Article

Correlation Between Minimal Hepatic Encephalopathy and Features of Decompensated Liver Disease

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Abstract: ***Background***: Minimal Hepatic Encephalopathy (MHE) is a subclinical neurocognitive disorder seen in cirrhosis, characterized by cognitive impairments undetectable on routine clinical examination. Its impact on disease progression and quality of life remains under-recognized. This study aimed to assess the association between MHE and features of decompensated liver disease. ***Methods***: A facility-based cross-sectional study was conducted on 70 patients with cirrhosis at Gandhi Medical College, Bhopal, from July 2023 to December 2024. MHE was diagnosed using the Psychometric Hepatic Encephalopathy Score (PHES). Patients were evaluated for ascites, upper gastrointestinal (GI) bleeding, Child-Pugh class, and laboratory parameters. Statistical analysis was performed using SPSS v20, with significance set at $p<0.05$. ***Results***: MHE was identified in 57.1% of patients. Significant associations were observed between MHE and higher ascites grades ($p=0.002$), history of upper GI bleeding ($p=0.001$), and advanced Child-Pugh class ($p=0.001$). Laboratory findings revealed significantly higher total bilirubin (5.78±5.71 vs. 1.29±0.75 mg/dL), prothrombin time (27.29±8.17 vs. 22.04±5.05 seconds), and INR (2.01±0.77 vs. 1.49±0.48) in MHE patients ($p<0.05$). No significant associations were found between MHE and cirrhosis etiology, viral markers, or serum ammonia levels. ***Conclusions***: MHE is prevalent among cirrhotic patients and significantly correlates with features of decompensated liver disease and markers of hepatic dysfunction. Routine screening using standardized psychometric tools is essential for early detection and management to mitigate cognitive decline and improve clinical outcomes.

Keywords: Minimal hepatic encephalopathy, cirrhosis, decompensated liver disease, PHES, ascites, Child-Pugh score, cognitive dysfunction.

INTRODUCTION

Cirrhosis of the liver represents the end stage of any chronic liver disease, which is characterized by distortion of the normal lobular architecture of the liver, with the formation of fibrotic tissue and regenerative nodules, typically resulting from prolonged hepatic injury. [1,2] Chronic liver disease can present in either a compensated or decompensated state. Decompensated liver disease refers to the acute deterioration of liver function in individuals with pre-existing chronic liver disease or cirrhosis. It is clinically defined by the development of complications such as ascites, jaundice, hepatorenal syndrome, hepatic encephalopathy, or variceal bleeding.[2]

Cirrhosis is associated with numerous complications, including hepatic encephalopathy, portal hypertension and end-stage liver disease, and contributes significantly to global morbidity and mortality.[1] Hepatic encephalopathy (HE) is a neurocognitive disorder associated with both acute and chronic liver dysfunction, in which brain function becomes impaired. The earliest stage in the HE spectrum is minimal hepatic encephalopathy (MHE), formerly referred to as subclinical or latent

encephalopathy. According to consensus statement 5D, MHE is defined by the presence of quantifiable cognitive impairments in patients with portal-systemic shunting and/or liver disease, without any other identifiable cause of cognitive dysfunction.[3]

The concept of Minimal Hepatic Encephalopathy (MHE) emerged after clinicians observed that some patients with cirrhosis, despite appearing neurologically normal during standard clinical evaluations, demonstrated abnormalities in electroencephalogram (EEG) findings and cognitive performance on basic testing.[4] MHE is primarily characterized by subtle dysfunctions in central nervous system activity, including impairments in attention, psychomotor speed, visuospatial perception, response inhibition, and delayed information processing.[5,6] The reported prevalence of MHE among individuals with liver cirrhosis varies widely, ranging from 22% to 74%. This variation is likely influenced by factors such as patient age, the presence of oesophageal varices, severity of liver dysfunction, history of overt hepatic encephalopathy, and the existence of surgical porto-systemic shunts. Notably, the underlying etiology of liver disease does not appear to affect the prevalence of MHE.[3]

Due to its subclinical nature, MHE cannot be detected through routine neurological examinations and clinical history. Instead, its diagnosis relies on specialized neuropsychometric or neurophysiological assessments, which can be administered at the bedside or in outpatient settings.[5,6] The Psychometric Hepatic Encephalopathy Score (PHES) is a widely validated diagnostic tool for MHE and comprises five individual tests: the Digit Symbol Test (DST), Serial Dotting Test (SDT), Line Tracing Test (LTT), and Number Connection Tests A and B (NCT-A and NCT-B).[7]

Given the subtle yet significant impact of MHE on cognitive function and daily living, understanding its relationship with clinical features of decompensated liver disease is essential. Since MHE may precede or coexist with complications associated with decompensated liver cirrhosis, evaluating its correlation with the features of decompensated cirrhosis could help in identifying patients at greater risk for disease progression and neurocognitive decline. Therefore, the current study was undertaken to investigate the association between MHE and features of decompensated liver disease at our center.

METHODOLOGY

This study was conducted as a facility based cross sectional study on 70 cases with liver cirrhosis at Department of Medicine, Gandhi Medical college (Hamidia Hospital) Bhopal, during the study period of 18 months i.e. from 1st July 2023 to 31st December 2024.

All the cases with confirmed cirrhosis (based on USG) belonging to more than 18 years of age were included whereas patients with history of overt hepatic encephalopathy, Psychiatric/Neurological disorders, history of Hepatocellular Ca or Other malignancy, previous TIPS or shunt surgery, history of consumption of Psychoactive drugs/antibiotics during past 2 weeks and alcohol >50g/day within past 3 months were excluded from the study.
Sample size was estimated using the formula
$n=(z)2p(1–p)/d2$
where; z=1.96 at 95% CI,
p=prevalence of MHE= 22%[3]
q=-p=26%
d=10% allowable error

Sample size was estimated to be 66 and hence a total of 70 cases were included.

For diagnosis of MHE, PHES scale was used and hence the due permission was obtained for using PHES scoring system. Following approval from the Institutional Ethics Committee, patients diagnosed with liver cirrhosis who met the study's inclusion criteria were recruited after obtaining written informed consent. All participants underwent comprehensive history taking, followed by detailed general and systemic examinations. Each participant underwent a set of diagnostic investigations, along with abdominal ultrasonography (USG), and electroencephalogram (EEG). Serum ammonia levels could not be measured in all participants. The severity of cirrhosis was evaluated using the Child-Pugh-Turcotte score.[8]

Minimal Hepatic Encephalopathy (MHE) was diagnosed using the Psychometric Hepatic Encephalopathy Score (PHES), which has been validated and standardized for the Indian population.[9] All assessments were administered sequentially in a quiet environment after explaining the procedure to the participants and allowing a trial run. Pencils were used for all written components.

- Digit Symbol Test (DST): Participants completed a table by matching 80 numbers with their corresponding symbols within 90 seconds. The number of correctly matched symbols was recorded as the final score.[7]
- Number Connection Test-A (NCT-A): Participants were asked to sequentially connect numbers from 1 to 25 as quickly and accurately as possible. Mistakes had to be corrected while the timer continued running. Completion time (in seconds) was recorded.[7]
- Number Connection Test-B (NCT-B): This test was adapted to the local language (Hindi). Instead of English

alphabets, Hindi letters (क, ख, ग, etc.) were used. Participants connected a sequence such as "1–क – 2–ख" up to 13, with corrections made during the timing. Total completion time (in seconds) was recorded.[7]

- Serial Dotting Test (SDT): Participants placed a dot in the center of 100 pre-drawn circles as quickly as possible. The final score was the time taken in seconds.[7]
- Line Tracing Test (LTT): Participants traced a path through a maze formed by two parallel lines without touching the edges. Both the time taken and number of errors were recorded.[7]

To analyze the results, the mean and standard deviation (SD) for each test were first calculated from the control group. For each test (except DST), scores falling between ±1 SD were assigned a value of 0. Scores between +1 to +2 SD received -1, +2 to +3 SD were scored as -2, and those beyond +3 SD were given -3. Conversely, scores below -1 SD were given +1. For DST, a reverse scoring was applied: scores between ±1 SD were scored as 0; scores between -1 to -2 SD were assigned -1, -2 to -3 SD were scored as -2, and scores below -3 SD were assigned -3. Scores above +1 SD were assigned +1. The total PHES score was calculated by summing the individual test scores. A cumulative score below -4 was considered indicative of MHE. EEG findings were also documented for each participant.

Data Analysis

All collected data were entered and compiled in Microsoft Excel and analyzed using IBM SPSS Statistics version 20 (IBM Corp. Statistical Package for Social Sciences, Illinois Chicago). Categorical variables were presented as frequencies and percentages, while continuous variables were summarized using mean and standard deviation. The association between MHE and specific features of decompensated liver disease was examined using chi-square tests for categorical data and independent t-tests for continuous data. A p-value of less than 0.05 was considered statistically significant.

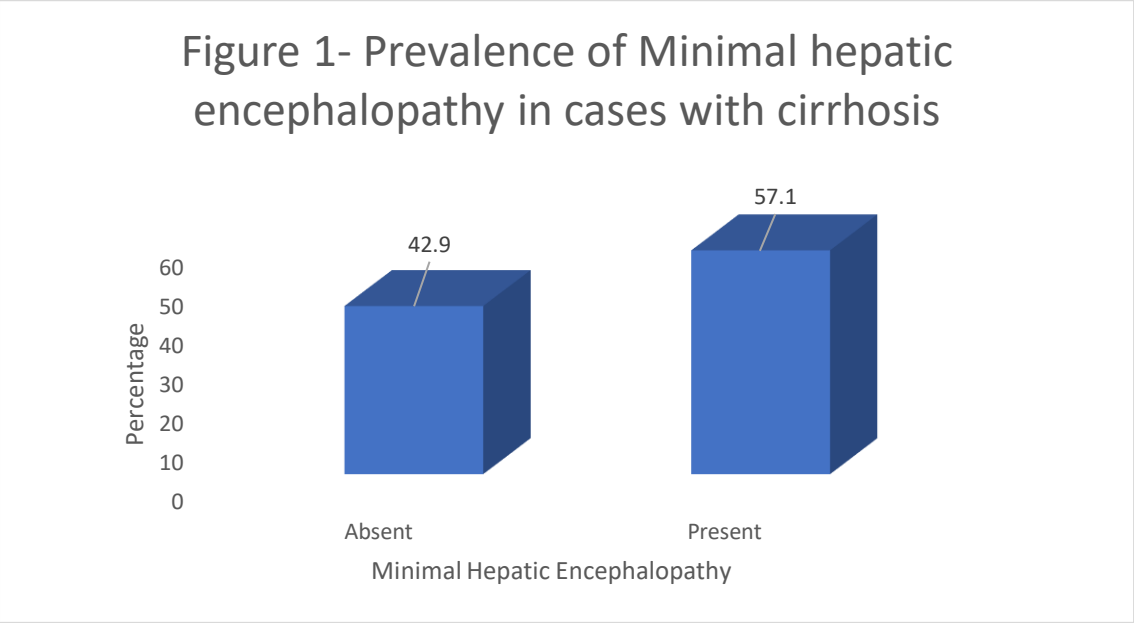
RESULTS

The present study was conducted on a total of 70 patients with cirrhosis with mean age of 45.66±10.96 years.

Table 1- Distribution of cases according to baseline variables

Baseline variables			No. of patients (n=70)	Percentage
Age (years)		≤30	5	7.1
		31– 40	18	25.7
		41 – 50	24	34.3
		51– 60	17	24.3
		≥60	6	8.6
Gender		Male	55	78.6
		Female	15	21.4
Ascites		No	29	41.4
		Mild	23	32.9
		Moderate	13	18.6
		Severe	5	7.1
Upper GI bleed		No	44	62.9
		Yes	26	37.1
Viral markers	HBsAg	Non-reactive	55	78.6
		Reactive	15	21.4
	AntiHCV	Non-reactive	66	94.3
		Reactive	4	5.7
Aetiology of CLD		Alcohol	42	60
		Auto-immune	2	2.9
		Cryptogenic	4	5.7
		Hepatitis B	15	21.4
		Hepatitis C	4	5.7
		NASH	3	4.3
Child Pugh score		A	14	20.0
		B	32	45.7
		C	24	34.3
EEG suggestive of MHE		Not Suggestive	63	90
		Suggestive	7	10

Majority of patients with Cirrhosis belonged to 41 to 50 years of age (34.3%) andwe observed a male predominance for cirrhosis with male:female ratio of 3.7:1. History of upper GI bleeding was observed in 37.1% cases with cirrhosis and 58.6% patients had ascites, among them, mild, moderate and severe ascites was observed in 32.9%, 18.6% and 7.1% cases respectively. HBsAg was reactive in 21.4% cases and anti-HCV was reactive in 5.7% cases. Most common cause of cirrhosis was alcoholic liver disease (60%), followed by Hepatitis B (21.4%). Majority of patients belonged to Child Pugh score B (45.7%), followed by Child Pugh class C (34.3%) and Child Pugh A class (20%). EEG was suggestive of hepatic encephalopathy in 10% cases of cirrhosis (Table 1).



Based on PHES scale, we observed MHE in 57.1% cases with cirrhosis (Figure 1).

Table 2- Association of Minimal Hepatic encephalopathy with features of decompensated liver disease

Features of decompensated liver disease		MHE				P value
		Absent (n=30)		Present (n=40)		
		n	%	n	%	
Upper GI bleed	No	27	61.4	17	38.6	**0.001**
	Yes	3	11.5	23	88.5	
Ascites	No	20	69.0	9	31.0	**0.002**
	Mild	7	30.4	16	69.6	
	Moderate	3	23.1	10	76.9	
	Severe	0	0.0	5	100.0	
HBsAg	Non-reactive	22	40.0	33	60.0	0.36
	Reactive	8	53.3	7	46.7	
Anti-HCV	Non-reactive	28	42.4	38	57.6	0.77
	Reactive	2	50.0	2	50.0	
Aetiology	Alcohol	15	37.2	27	62.8	0.62

	Auto-immune	1	50.0	1	50.0	
	Cryptogenic	1	25.0	3	75.0	
	Hepatitis B	8	53.3	7	46.7	
	Hepatitis C	2	50.0	2	50.0	
	NASH	3	100.0	0	0	
Severity of cirrhosis	A	10	71.4	4	28.6	**0.001**
	B	18	6.2	14	43.8	
	C	2	8.3	22	91.7	

MHE was significantly associated with higher grades of ascites, severe cirrhosis and upper GI bleeding ($p<0.05$). However, we observed no significant association of viral markers and aetiology with MHE (Table 2).

Table 3- Association of Minimal Hepatic encephalopathy with findings of blood investigations

Investigations	MHE				P value
	Absent (n=30)		Present (n=40)		
	Mean	SD	Mean	SD	
Total bilirubin (mg/dl)	1.29	0.75	5.78	5.71	**0.001**
Sodium (mmol/L)	134.65	4.60	132.37	5.73	0.079
Potassium (mmol/L)	3.91	0.53	3.96	0.65	0.714
WBC count (cells/µl)	7334.33	3312.13	8062.25	4241.44	0.439
Platelet count (/µl)	1.45	0.81	5401.16	19289.36	0.131
PT (seconds)	22.04	5.05	27.29	8.17	**0.003**
INR	1.49	0.48	2.01	0.77	**0.002**
Serum albumin (g/dl)	2.44	0.53	2.25	0.49	0.117
Blood urea (mg/dL)	42.23	22.71	40.47	24.21	0.759
Serum creatinine (mg/dL)	1.10	0.53	1.13	0.67	0.819
Ammonia (µg/dL)	69.5	7.78	86.7	14.33	0.14

Mean total bilirubin levels were found to be significantly higher in cases with MHE as compared to patients with no MHE (5.78±5.71 vs. 1.29±0.75; $p<0.05$). Similarly, mean PT and INR were significantly higher in cases with MHE as compared to patients with no MHE (27.29±8.17 vs. 22.04±5.05 seconds and 2.01±0.77 vs. 1.49±0.48; $p<0.05$). However, we found no significant difference in other investigation findings in patients with and without MHE ($p>0.05$) (Table 3).

DISCUSSIONS

The present study was conducted at tertiary care centre to assess the correlation between Minimal Hepatic Encephalopathy and features of Decompensated Liver Disease. This study included 70 patients with cirrhosis. Although minimal hepatic encephalopathy is common a feature of cirrhosis, noncirrhotic patients may also experience MHE.[10] We conducted PHES battery of tests on cases with cirrhosis and observed MHE in more than half of the patients with cirrhosis (57.1%). Our study findings were supported by the findings of Thanapirom et al, in which MHE was observed in 26.6% cases with cirrhosis.[11] Gairing et al found MHE in 35% cases

of cirrhosis.[12] Kavya et al found MHE in 62% cases [13] whereas Pessidjo Djomatcho et al documented much higher prevalence of MHE (74%).[14]

One of the most common complications of cirrhosis is ascites, which is characterized by a pathological accumulation of excess fluid in the peritoneal cavity. Along with portal hypertension, ascites may be the initial indication of cirrhosis progression from the compensated state to the decompensated state, and it is seen in around 50% of cases of decompensated cirrhosis.[15] We observed ascites in 58.6% patients with cirrhosis and observed a significant association of MHE with higher severity of ascites i.e. 69.6% cases with mild ascites, 76.9% cases with moderate ascites and 100% cases with severe ascites had MHE ($p<0.05$). About 37.1% cases presented with upper GI bleeding and we observed MHE in significantly higher proportions of patients with history of upper GI bleeding as compared to patients with no history of upper GI bleeding (88.5% vs. 38.6%; $p<0.05$).

The findings of present study were in line with the findings of Thanapiom et al, in which about 43.8% cases with cirrhosis had decompensated cirrhosis.[11] Wang et al reported significant association of ascites with minimal hepatic encephalopathy, supporting our study findings. 63.3% cases with MHE and 44.6% cases with no MHE had prior history of ascites ($p<0.05$).[16]

Cirrhosis could be attributed to a number of underlying causes, but the most common ones are alcoholic liver disease, viral hepatitis (hepatitis B and C), and non-alcoholic fatty liver disease. Hemochromatosis, Wilson disease, autoimmune hepatitis, primary biliary cirrhosis (PBC), primary sclerosing cholangitis (PSC), and genetic variables (alpha-1 antitrypsin deficiency) are other rare causes of chronic liver disease (CLD).[17]

We included patients with various aetiologies of cirrhosis, most common being alcohol (60%), followed by hepatitis (27.1%) i.e. hepatitis B in 21.4% and Hepatitis C in 5.7%. Other causes documented in small proportions of cases were cryptogenic, NASH and autoimmune cirrhosis. In the present study, MHE was found in 46.7% of cases with Hepatitis B, 50% of cases with autoimmune aetiology, 75% of cases with cryptogenic cirrhosis, 62.8% of cases with alcoholic liver disease, 50% of cases with Hepatitis C and none of the cases with NASH had MHE. We observed no significant association of aetiology with MHE ($p>0.05$). However, in a study of Thanapirom et al, viral aetiologies, Hepatitis C and Hepatitis B (24.6% cases each) were the most common aetiologies, followed by non-alcoholic steatohepatitis (18.7%) and alcoholic-related disease(18.2%).[11] Similar findings were documented by Pessidjo Djomatcho et al, the authors found no significant association of onset of MHE with etiology of cirrhosis ($p>0.05$).[14]

Child Pugh Score was used to assess the severity of Cirrhosis and majority of cases in our study had moderate to severe cirrhosis (Child Pugh class B-45.7%). MHE was significantly associated with higher severity of cirrhosis i.e. 91.7% cases with Child Pugh Class C, 43.8% cases with Child Pugh class B and 28.6% cases with Child Pugh class A had MHE ($p<0.05$). In a study of Thanapirom et al, about 43.8% cases presented with Child Pugh class B and C and the authors found a significant association of MHE with severity of cirrhosis. Significantly higher proportions of patients with MHE had CTP class B/C (53.7% vs. 40.3%, $p=0.08$). The authors also found a weak inverse correlation of PHES with MELD score ($r=-0.20$, $p=0.008$ and CTP score ($r=-0.19$, $p=0.009$).[11] The findings of present study were supported by the findings of Gairing et al, where the authors found that the prevalence of MHE was low in cases with CP class A (25%) whereas the prevalence of MHE was higher in cases with CP class B and Class C (42% and 52% respectively).[12] Although higher proportions of patients with MHE in a study of Pessidjo Djomatcho et al had cirrhosis of child Pugh Class B or C (48.4%) as compared to patients with no MHE (16.7%), the authors found no significant association of severity of cirrhosis with MHE ($p>0.05$).[14]

In our study, Patients with MHE had significantly higher mean total bilirubin levels than those without MHE (5.78±5.71 vs. 1.29±0.75; $p<0.05$). Similar to this, patients with MHE had significantly higher mean PT and INR (27.29±8.17 vs. 22.04±5.05 seconds and 2.01±0.77 vs. 1.49±0.48; $p<0.05$) than patients without MHE. Thanapirom et al found significantly lower serum albumin levels in patients with MHE as compared to those without MHE (3.4±0.7 vs. 3.6±0.8g/dL, $p=0.09$) whereas the authors found no significant association of MHE with serum bilirubin levels or INR ($p>0.05$).[11] Gairing et al observed a negligible but significant correlation of serum ammonia levels with PHES ($r=-0.16$, $P < 0.001$).[12] However, we found no significant association of serum ammonia with MHE, and it could be due to serum ammonia estimation in small proportions of cases (only 12 cases) in our study. Pessidjo Djomatcho et al found no significant difference in mean serum bilirubin, albumin, INR, creatinine and platelets between patients with MHE and no MHE in their study group ($p>0.05$).[14]

Our study had certain limitations, first, serum ammonia levels were assessed in only small proportions of patients due to its high cost, i.e. it could be assessed in only 12 patients with cirrhosis. As it was done in only few patients, actual association of ammonia levels with MHE could not be assessed. Since the study was conducted as cross sectional study, both short term and long term outcome of the

patients with and without MHE could not be assessed. As the study was conducted at a single hospital, results may vary in different healthcare settings with different patient demographics and healthcare practices.

CONCLUSIONS

Minimal hepatic encephalopathy is common in cases with cirrhosis. Patients with MHE show significantly worse outcomes in terms of ascites severity, frequency of upper GI bleeding, and advanced Child-Pugh class, indicating a strong association between MHE and the clinical features of decompensated liver disease. Furthermore, laboratory parameters such as total bilirubin, prothrombin time (PT), and international normalized ratio (INR) were significantly elevated in patients with MHE, suggesting greater hepatic dysfunction in this subgroup. These findings underscore the high prevalence of MHE among patients with decompensated cirrhosis and highlight the importance of early cognitive screening using standardized psychometric tools. Early detection and management of MHE may play a critical role in improving patient outcomes, reducing complications, and enhancing quality of life in individuals with chronic liver disease.

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