Original Article

Psychometric Hepatic Encephalopathy Score detects minimal hepatic encephalopathy in cirrhosis: a hospital-based case–control study from North India

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Abstract

Background: Minimal hepatic encephalopathy (MHE) impairs daily functioning in cirrhosis. The Psychometric Hepatic Encephalopathy Score (PHES) is a practical bedside battery for MHE detection. **Material and Methods:** Hospital-based case–control study over 18 months (July 2023– December 2024), including 70 radiologically proven cirrhosis cases without overt HE and 70 age-/sex-matched healthy controls. PHES (NCT-A, NCT-B, Digit Symbol, Line Tracing, Serial Dotting) was administered; MHE = PHES <-4. EEG was performed in cases. Data were analysed with t-test/chi-square (α=0.05). **Results:** Mean PHES was significantly lower in cases than controls (−5.97±4.89 vs −0.21±1.39; p<0.001). MHE prevalence was 57.1% (40/70) in cases versus 1.4% (1/70) in controls. In some cases, PHES was worse in MHE than in non-MHE (−9.45±3.37 vs −1.33±1.63; p<0.001). Child-Pugh class correlated with MHE (A 28.6%, B 43.8%, C 91.7%; p=0.001). Ascites severity also correlated (mild 69.6%, moderate 76.9%, severe 100%; p<0.05). EEG was suggestive in 7 patients, all had MHE (p=0.016). Laboratory indices were higher in MHE: total bilirubin (5.78±5.71 vs 1.29±0.75 mg/dL), PT (27.29±8.17 vs 22.04±5.05 s), INR (2.01±0.77 vs 1.49±0.48); all p<0.05. **Conclusion:** PHES detects a high burden of MHE in cirrhosis and aligns with disease severity (Child-Pugh class, ascites), EEG abnormalities, and coagulopathy. Routine PHES screening can unmask early neurocognitive impairment and support risk-stratified care in resource-limited settings.

Keywords: Minimal hepatic encephalopathy; Psychometric Hepatic Encephalopathy Score; Cirrhosis; Child-Pugh; Ascites; EEG.

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Introduction

Hepatic encephalopathy (HE) spans a spectrum from subtle cognitive impairment to coma. Minimal HE (MHE) represents the covert end of this spectrum: routine neurological examination is often normal, yet patients exhibit deficits in attention, psychomotor speed, visuospatial processing, and executive function that impair daily activities, including driving and operating machinery.[1,2]

Neuropsychological testing is therefore central to detection. The Psychometric Hepatic Encephalopathy Score (PHES), a paper-and-pencil battery comprising Number Connection Tests A/B, Digit Symbol, Line Tracing, and Serial Dotting, captures cognitive domains typically affected in MHE.[3-5] It is straightforward to administer, culturally adaptable, and recommended by expert groups; PHES has been validated/standardized across multiple countries, including India.

In clinical settings, adjunct EEG and other quantitative neurophysiological measures may be useful when feasible, but psychometrics remains the most practical first-line approach.[6]

Despite growing evidence, regional data from Central/North India remain limited. This study addresses that gap by applying PHES in a hospital-based case–control design to quantify the burden of MHE among adults with cirrhosis and to explore clinical correlates of disease severity.[7]

Aim: to assess the effectiveness of PHES for detecting MHE in cirrhosis and to examine its association with indicators of decompensation and related clinical/laboratory features in our setting.

Materials and Methods

Study design and setting: This cross-sectional observational study was conducted in the Department of Medicine at Gandhi Medical College and the associated Hamidia Hospital, Bhopal, over ~18 months (July 2023 to December 2024).

Participants: Cases: adults with ultrasonography-proven liver cirrhosis without overt signs of hepatic encephalopathy.

Controls: age- and sex-matched healthy adults. Sample size

Using n=z2p(1−p)/d2 with z=1.96 (95% CI), expected prevalence of minimal hepatic encephalopathy (MHE) p=22%,

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and allowable error d=10%, the minimum sample size was 66. We enrolled 70 cases and 70 controls.

Inclusion criteria

Age >18 years; confirmed cirrhosis on ultrasonography.

Exclusion criteria

Controls: psychiatric/neurological disorders; psychotropic drug use; alcohol intake >50 g/day within the past 3 months; inability to read/write; presence of chronic liver disease. Cases: prior overt hepatic encephalopathy; psychoactive drugs/antibiotics in the past 2 weeks; psychiatric/neurological disorders; alcohol >50 g/day within past 3 months; inability to read/write; hepatocellular carcinoma/other malignancy; prior TIPS/shunt.

Ethical considerations and consent

Written informed consent was obtained from all participants after explaining the study purpose, with confidentiality assured and the right to withdraw preserved. Prior permission to use the PHES scoring system was obtained; institutional ethics approval was secured.

Data collection and clinical assessment

After enrolment, all participants underwent a structured history and general/systemic examination. Investigations included LFT, RFT, abdominal ultrasonography, PT-INR, complete blood count, HBsAg, anti-HCV, and EEG; serum ammonia was not performed for all. Cirrhosis severity was graded using the Child–Pugh–Turcotte score.

PHES administration (psychometric testing)

PHES (validated and standardised for the Indian population) comprised five tests administered in a quiet area after a trial run, using pencil and uniform instructions: Digit Symbol Test (DST), Number Connection Test-A (NCT-A), Number Connection Test-B (NCT-B; adapted with Hindi varnmala), Line Tracing Test (LTT), and Serial Dotting Test (SDT). Timing, accuracy, and error counts were recorded per standard procedures.

PHES scoring and case definition

For each subtest, control means and SDs were calculated. For NCT-A, NCT-B, LTT, and SDT: scores between +1 and −1 SD were 0; +1 to +2 SD = −1; +2 to +3 SD = −2; >+3 SD = −3. values <−1 SD = +1. For DST: +1 to −1 SD = 0; −1 to −2 SD = −1; −2 to −3 SD = −2; <−3 SD = −3; >+1 SD = +1. The five sub scores were summed; a total score <-4 defined MHE.

Electroencephalography

Standard EEG was performed and documented for all cases to assess electrophysiological correlates.

Outcomes

The primary outcome was the prevalence of MHE (per PHES). Secondary analyses evaluated associations of MHE with clinical severity (Child–Pugh class), ascites, viral markers, history of upper GI bleeding, EEG findings, and laboratory parameters. (Analytic approach below.)

Statistical analysis: Data were compiled in MS-Excel and analysed in IBM SPSS v20. Categorical variables are presented as frequencies/percentages and compared using the chi-square test; continuous variables are presented as means ± SD and compared using the independent-samples t-test. Associations of MHE with categorical and continuous variables were assessed using chi-square and t tests, respectively. Two-sided $P < 0.05$ was considered statistically significant.

Results

Study sample and matching: A total of 140 participants were enrolled: 70 cirrhosis cases and 70 age- and sex-matched controls. Mean age did not differ (cases 45.66 ± 10.96 vs controls 44.26 ± 12.53 years; $\chi^2 = 3.60$, $P = 0.46$). Males comprised 78.6% of cases and 77.1% of controls ($\chi^2 = 0.041$, $P = 0.84$).

Age-group distribution among cases: ≤30 (7.1%), 31–40 (25.7%), 41–50 (34.3%), 51–60 (24.3%), ≥60 (8.6%) [Figure 1].

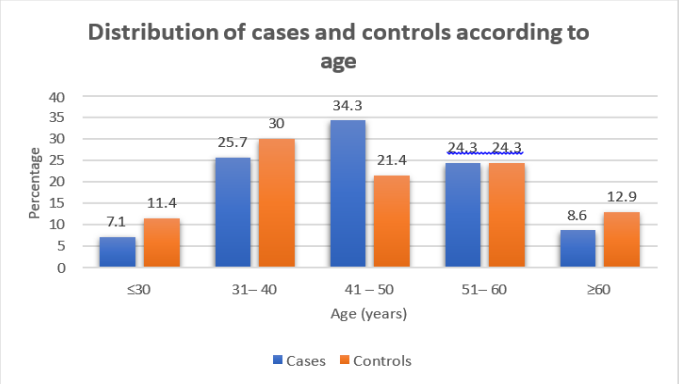

Figure 1: Distribution of cases and controls according to age PHES performance: cases vs controls.

Mean (±SD) values in cases vs controls were: NCT-A 81.99 ± 16.42 vs 63.74 ± 7.88 ($t = 8.382$, $P = 0.001$); NCT-B 119.21 ± 25.24 vs 94.81 ± 13.62 ($t = 7.118$, $P = 0.001$); DST 18.06 ± 4.69 vs 23.39 ± 3.85 ($t = 7.345$, $P = 0.001$); LTT 75.94 ± 11.20 vs 61.63 ± 9.22 ($t = 8.26$, $P = 0.001$); SDT 76.49 ± 13.66 vs 64.37 ± 6.80 ($t = 6.641$, $P = 0.001$). Overall, PHES was significantly worse in cases (−5.97 ± 4.89) than controls (−0.21 ± 1.39; $t = 9.479$, $P = 0.001$) [Table 1].

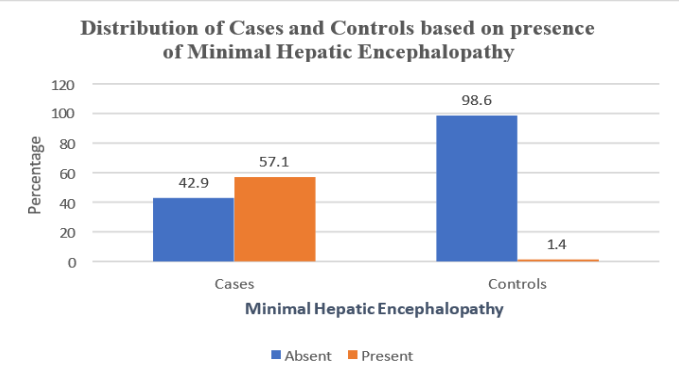

Figure 2: Distribution of Cases and Controls based on presence of Minimal Hepatic Encephalopathy

Prevalence of minimal hepatic encephalopathy (MHE)

MHE prevalence was 57.1% (40/70) in cases and 1.4% (1/70) in controls ($\chi^2 = 52.46$, $P = 0.001$) [Figure 2].

Clinical profile of cases: Upper GI bleeding history was present in 37.1% (26/70). Ascites on ultrasonography was present in 58.6% overall: mild 32.9%, moderate 18.6%, severe 7.1% [Table 2]. Viral markers: HBsAg reactive 21.4% (15/70); anti-

HCV reactive 5.7% (4/70). Etiology: alcohol 60.0%, hepatitis B 21.4%, hepatitis C 5.7%, cryptogenic 5.7%, NASH 4.3%, autoimmune 2.9%. Child- Pugh class: A 20.0%, B 45.7%, C 34.3% (mean CTP score 8.86 ± 2.38). EEG was suggestive of HE in 10.0% (7/70) [Figure 3].

Table 1: Distribution of Cases and Controls based upon PHES scale

PHES	Cases (n=70)	Controls (n=70)	T value	P value
NCT A	81.99±16.42	63.74±7.88	8.382	0.001
NCT B	119.21±25.24	94.81±13.62	7.118	0.001
DST	18.06±4.69	23.39±3.85	7.345	0.001
LTT	75.94±11.199	61.63±9.22	8.26	0.001
SDT	76.49±13.66	64.37±6.80	6.641	0.001
PHES	-5.97±4.89	-0.21±1.39	9.479	0.001

Table 2: Distribution of cases based on severity of ascites based on Ultrasonography

Ascites	Number of patients (n=70)	Percentage
No	29	41.4
Mild	23	32.9
Moderate	13	18.6
Severe	5	7.1

Table 3: Distribution of Laboratory Investigations in cases

Investigations	Mean	SD
Total bilirubin (mg/dl)	3.86	4.87
Sodium (mmol/L)	133.35	5.36
Potassium (mmol/L)	3.94	0.59
WBC count (cells/µl)	7750.29	3861.41
Platelet count (/µl)	3087.00	1475.57
PT (seconds)	25.04	7.43
INR	1.78	0.71
Serum albumin (g/dl)	2.33	0.51
Blood urea (mg/dL)	41.23	23.43
Serum creatinine (mg/dL)	1.11	0.611
Ammonia (µg/dL)	83.83	14.78

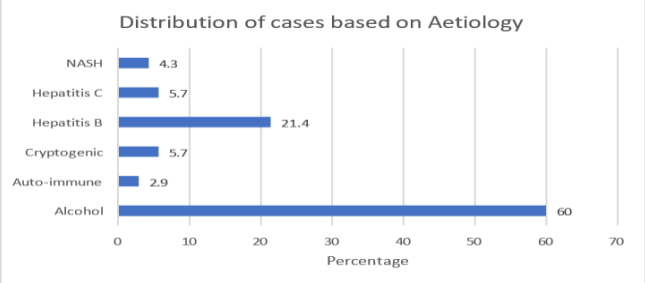

Figure 3: Distribution of cases based on Etiology

Laboratory profile of cases: Mean (±SD): total bilirubin 3.86 ± 4.87 mg/dL, sodium 133.35 ± 5.36 mmol/L, potassium 3.94 ± 0.59 mmol/L, WBC 7750.29 ± 3861.41/µL, platelets (unit as recorded), PT 25.04 ± 7.43 s, INR 1.78 ± 0.71, serum albumin 2.33 ± 0.51 g/dL, blood urea 41.23 ± 23.43 mg/dL, serum creatinine 1.11 ± 0.61 mg/dL. Ammonia was measured in 12 cases (mean 83.83 ± 14.78 µg/dL) [Table 3].

Associations with MHE among cases

- Upper GI bleeding: MHE in 88.5% with bleeding vs 38.6% without ($\chi^2 = 16.57$, P = 0.001).
- Ascites severity: MHE in 31.0% (no ascites), 69.6% (mild), 76.9% (moderate), 100% (severe) ($\chi^2 = 15.35$, P = 0.002).
- Viral markers: Not significant HBsAg ($\chi^2 = 0.86$, P = 0.36); anti-HCV ($\chi^2 = 0.09$, P = 0.77).
- Etiology: Not significant ($\chi^2 = 3.53$, P = 0.62).
- Child-Pugh: MHE in 28.6% (A), 43.8% (B), 91.7% (C); mean CTP 9.88 ± 2.47 in MHE vs 7.50 ± 1.38 in non-MHE ($\chi^2 = 18.69$, P = 0.001).
- EEG: Suggestive in 7 patients, all (100%) had MHE ($\chi^2 = 5.83$, P = 0.016).

PHES subtests by MHE status

Means (MHE present vs absent): NCT-A 92.30 ± 13.09 vs 68.23 ± 8.23 (t = 8.827, P = 0.001); NCT-B 132.15 ± 25.89 vs 101.97 ± 18.68 (t = 6.123, P = 0.001); DST 16.30 ± 4.40 vs 20.40 ± 4.10 (t = 3.989, P = 0.001); LTT 81.80 ± 10.55 vs 68.13 ± 6.20 (t = 6.32, P = 0.001); SDT 85.35 ± 10.73 vs 64.67 ± 6.03 (t = 9.486, P = 0.001). Total PHES: −9.45 ± 3.37 vs −1.33 ± 1.63 (t =12.17, P = 0.001) [Table 4].

Table 4: Association of Minimal Hepatic encephalopathy with PHES score

PHES score	MHE		T value	P value
	Absent (n=30)	Present (n=40)		
NCT A	68.23±8.23	92.3±13.09	8.827	0.001
NCT B	101.97±18.68	132.15±25.89	6.123	0.001
DST	20.40±4.1	16.30±4.4	3.989	0.001
LTT	68.13±6.2	81.8±10.55	6.32	0.001

SDT	64.67±6.03	85.35±10.73	9.486	0.001
PHES	-1.33±1.63	-9.45±3.37	12.17	0.001

Laboratory associations with MHE

Significantly higher in MHE vs non-MHE: total bilirubin 5.78 ± 5.71 vs 1.29 ± 0.75 mg/dL (t= −4.277, P = 0.001); PT 27.29 ± 8.17 vs 22.04 ± 5.05 s (t = −3.099, P = 0.003); INR 2.01 ±0.77 vs 1.49 ± 0.48 (t = −3.26, P = 0.002). Differences in sodium, potassium, WBC, platelets, albumin, urea, creatinine, and ammonia were not significant (all P > 0.05; ammonia available for 12 cases).

Discussion

In this hospital-based case–control study (70 cirrhosis; 70 controls), the PHES battery differentiated cases from controls across all components, with a markedly lower total PHES in cirrhosis (−5.97 ± 4.89 vs −0.21 ± 1.39; P<0.001). The prevalence of MHE was 57.1% in cases versus 1.4% in controls. Consistent with decompensation biology, MHE correlated with greater disease severity: proportions rose stepwise from 28.6% (Child-Pugh A) to 43.8% (B) and 91.7% (C), and the mean CTP score was higher in MHE vs non-MHE (9.88 ± 2.47 vs 7.50 ± 1.38). Ascites severity showed a similar gradient: 69.6% (mild), 76.9% (moderate), 100% (severe), supporting PHES sensitivity to clinical decompensation. Upper-GI bleeding was strongly associated with MHE (88.5% with history vs 38.6% without; P=0.001). Electrophysiology aligned with cognition: EEG was suggestive in 10% (7/70) of cases, and all 7 had MHE (P=0.016), reinforcing that abnormal EEG tracks psychometric impairment while acknowledging lingering debate about EEG as a primary diagnostic test.

Biochemically, bilirubin, PT, and INR were higher in MHE (5.78 vs 1.29 mg/dL; 27.29 vs 22.04 s; 2.01 vs 1.49; all P≤0.003), whereas other routine analyses did not differ significantly. Viral markers and etiologic class were not significantly associated with MHE. Taken together, these findings support PHES as a practical first-line screen that captures clinically meaningful gradients (Child-Pugh class, ascites, bleeding history) and aligns with EEG abnormalities. The solitary control with "MHE-positive" PHES noted alongside potential influences of education, age, language/motor factors, and test conditions underscores why standardised administration and local norms matter in practice.

Limitations

Serum ammonia sparsity: Ammonia was measured in 12/70 cases only, limiting correlation analyses.

Sample size: Single-centre sample of 70 cases may limit precision, especially for subgroup comparisons.

Design: Cross-sectional case–control design precludes causal inference and does not capture outcomes or treatment response over time.

Generalisability: Single-hospital context; results may vary across settings with different demographics and care practices.

Psychometric susceptibility: A positive PHES in 1 control and the documented influence of education/language/motor and test-environment factors highlight potential measurement bias without comprehensive local normative adjustment.

Conclusion

PHES identified a high burden of MHE (57.1%) in cirrhosis and tracked clinical severity rising with Child-Pugh class, ascites grade, and history of upper-GI bleeding while aligning with EEG abnormalities. Bilirubin, PT, and INR were higher in MHE, whereas viral markers and etiology were not discriminative. These data support routine PHES screening to unmask subclinical impairment and inform risk-stratified care in resource-limited settings, with future work needed to expand ammonia testing, adopt local norms, and evaluate longitudinal outcomes.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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