International Journal of Research in Medical Sciences
Khan YN et al. Int J Res Med Sci. 2026 Sep;14(9):3929-3935
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20263108

Original Research Article

Clinical spectrum and prognostic impact of precipitating factors in hepatic encephalopathy among patients with liver cirrhosis: a cross-sectional observational study

Yafas Nazir Khan[1]*, Mansi[2], Rolanda Saint Louis[2], Shehla Ashraf[3], Purna Chand Paidi[4]

[1]Department of Critical Care Unit, Sheri Kashmir Institute of Medical Sciences Soura, Srinagar, Jammu and Kashmir, India
[2]Washington University of Health and Science, San Pedro, Belize
[3]Bharati Vidyapeeth Deemed University Medical College, Pune, Maharashtra, India
[4]Andhra Medical College, Visakhapatnam, Andhra Pradesh, India

Received: 23 June 2026
Revised: 05 August 2026
Accepted: 12 August 2026

***Correspondence:**
Dr. Yafas Nazir Khan,
E-mail: yafasnazir@gmail.com

ABSTRACT

Background: Hepatic encephalopathy (HE) is a serious neuropsychiatric complication of liver cirrhosis associated with significant morbidity and mortality. Early identification of precipitating factors is essential for timely management and improved clinical outcomes.
Methods: A hospital-based cross-sectional observational study was conducted among 110 patients with cirrhosis of liver presenting with hepatic encephalopathy at a tertiary care teaching hospital. Clinical history, physical examination, laboratory investigations, and radiological evaluation were performed to identify precipitating factors. Hepatic encephalopathy was graded using the West Haven classification, while liver disease severity was assessed using the Child-Pugh score. PHES and Stroop tests were performed in eligible patients. Clinical outcomes, including recovery and mortality, were analyzed.
Results: Most patients were middle-aged males with alcohol-related cirrhosis. Grade II hepatic encephalopathy and Child-Pugh class C were the most common presentations. Asterixis, icterus, and ascites were frequent clinical findings. At least one precipitating factor was identified in most patients, with constipation, infections, upper gastrointestinal bleeding, electrolyte imbalance, vomiting/diarrhoea, and acute kidney injury being the commonest. Neuropsychometric abnormalities were frequently detected. Higher grades of hepatic encephalopathy and advanced Child-Pugh class were significantly associated with poorer outcomes and increased mortality.
Conclusions: Hepatic encephalopathy is commonly precipitated by identifiable and potentially reversible factors. Early recognition and prompt correction of these factors, together with timely assessment of disease severity, are essential to improve prognosis and reduce morbidity and mortality in patients with liver cirrhosis.

Keywords: Child-Pugh class, Gastrointestinal bleeding, Hepatic encephalopathy, Infection, Liver cirrhosis, Precipitating factors, West Haven grading

INTRODUCTION

Liver cirrhosis represents the final common pathway of chronic liver injury and remains a major cause of global morbidity and mortality.[1] It is characterized by diffuse hepatic fibrosis, regenerative nodules, distortion of hepatic architecture, and progressive deterioration of liver function.[2] Chronic liver disease and cirrhosis are

increasingly prevalent worldwide due to alcohol abuse, viral hepatitis, and metabolic liver disease.[3] Decompensated cirrhosis commonly leads to complications such as ascites, variceal bleeding, spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatic encephalopathy (HE).[4]

Hepatic encephalopathy is a complex neuropsychiatric syndrome occurring in patients with advanced liver dysfunction and portal-systemic shunting.[5] It presents with a wide spectrum of neurological abnormalities ranging from subtle cognitive dysfunction and behavioral changes to severe confusion, stupor, and coma.[6] Overt HE develops in approximately 30-45% of patients with cirrhosis, while minimal or covert HE may occur in an even larger proportion of patients.[7] HE significantly increases hospitalization rates, impairs quality of life, and is associated with substantial morbidity and mortality.[8]

The pathogenesis of HE is multifactorial, with ammonia playing a central role in disease development through astrocyte dysfunction, oxidative stress, and altered neurotransmission.[9] Other mechanisms including systemic inflammation, altered gut microbiota, electrolyte imbalance, and renal dysfunction also contribute to the development and progression of HE.[10]

HE is commonly precipitated by identifiable and potentially reversible clinical factors.[11] Gastrointestinal bleeding is one of the most important precipitating factors because it increases intestinal ammonia production through digestion of blood proteins.[12] Constipation contributes to prolonged intestinal ammonia absorption, while infections such as spontaneous bacterial peritonitis, urinary tract infection, pneumonia, and sepsis worsen systemic inflammation and hepatic dysfunction.[13] Electrolyte disturbances including hyponatremia and hypokalemia, dehydration, excessive diuretic use, renal dysfunction, vomiting, diarrhea, sedative medications, and large-volume paracentesis are also recognized triggers of HE.[14]

Several studies have demonstrated that precipitating factors can be identified in the majority of patients with overt hepatic encephalopathy.[15-17] The coexistence of multiple precipitating factors has been associated with severe encephalopathy, prolonged hospitalization, recurrent episodes, and increased mortality.[18] Early identification and prompt correction of these factors therefore form the cornerstone of HE management and may significantly improve patient outcomes.[19]

Alcohol-related liver disease remains one of the leading causes of cirrhosis and HE, particularly in developing countries.[20] Previous Indian and international studies have consistently reported alcohol as the predominant etiology among hospitalized HE patients.[21,22] Advanced Child-Pugh class and higher grades of HE have also been associated with poorer prognosis and increased mortality.[23]

Neuropsychometric assessment tools such as the Portosystemic Encephalopathy Syndrome (PHES) test and Stroop test are increasingly used for identification of minimal and covert hepatic encephalopathy.[24] Early detection of subtle cognitive dysfunction may facilitate timely therapeutic intervention and reduce progression to overt HE and its associated complications.[25]

Despite increasing awareness regarding hepatic encephalopathy, limited regional data are available regarding the complete clinical spectrum of precipitating factors and their association with disease severity and clinical outcomes in tertiary care settings.[26] Variability in etiological factors, healthcare access, and treatment practices across different populations necessitates further local epidemiological evaluation.

In this context, the present study was undertaken to evaluate the clinical spectrum of precipitating factors of hepatic encephalopathy in patients with liver cirrhosis and to assess their association with severity of encephalopathy, underlying liver dysfunction, and clinical outcomes in a tertiary care teaching hospital.

METHODS

Study design

A hospital-based cross-sectional observational study was conducted.

Study Setting

The study was conducted at Government General Hospital, Srikakulam, a tertiary care teaching hospital affiliated with Dr. NTR University of Health Sciences, Vijayawada.

Study duration

The study was conducted from March 2025 to February 2026 after getting approval from the institutional ethics committee.

Study population

Patients presenting to the medical wards, outpatient department, and emergency room with features suggestive of hepatic encephalopathy secondary to liver cirrhosis were included in the study.

Sample size and sampling method

A total of 110 patients were included in the present study. The sample size was calculated using prevalence estimates from previous hospital-based studies evaluating hepatic encephalopathy in cirrhotic patients. A consecutive sampling method was adopted, wherein all eligible patients fulfilling inclusion criteria during the study period were enrolled until the required sample size was achieved.

Inclusion criteria

Patients aged more than 18 years, known cases of chronic liver disease with signs and symptoms of hepatic encephalopathy, and patients with cirrhosis due to alcohol and/or viral etiology were included in the study.

Exclusion criteria

Patients aged less than 18 years, those with cirrhosis due to causes other than alcohol or viral hepatitis, acute fulminant hepatitis, non-cirrhotic portal hypertension, psychiatric illness or receiving psychiatric treatment, altered sensorium secondary to head injury, acute alcoholic intoxication or alcohol withdrawal states, and patients unwilling to participate in the study were excluded from the study.

Data collection procedure

Eligible patients were enrolled after obtaining informed consent. A detailed history regarding fever, hematemesis, melena, constipation, diarrhea, vomiting, dietary factors, trauma, surgery, and medication history including diuretics, sedatives, tranquilizers, and NSAID use was recorded using a structured proforma. Personal history including alcohol intake, dietary habits, and sleep disturbances was also documented. All patients underwent comprehensive general and systemic examination. Hepatic encephalopathy was diagnosed clinically after exclusion of other causes of altered sensorium and graded according to the West Haven classification. The severity of liver disease was assessed using the Child-Pugh scoring system. Relevant laboratory investigations, including complete blood count, liver function tests, renal function tests, serum electrolytes, coagulation profile, serum ammonia, viral markers, arterial blood gas analysis, urine analysis, blood culture, and imaging studies, were performed as indicated. Ultrasonography of the abdomen, upper gastrointestinal endoscopy, CT abdomen, and MRI brain were carried out wherever clinically necessary.

Neuropsychometric assessment

Psychometric evaluation using the portosystemic encephalopathy syndrome (PHES) test and Stroop test was performed in patients with grade 0, grade I, and grade II hepatic encephalopathy. Patients with grade III and grade IV encephalopathy were excluded from psychometric assessment due to impaired consciousness and inability to cooperate with testing procedures.

Operational definitions

Constipation was defined as reduced bowel frequency or difficulty in defecation. Infection was defined based on clinical features with or without microbiological confirmation, including spontaneous bacterial peritonitis, urinary tract infection, pneumonia, or sepsis. Electrolyte imbalance included hyponatremia, hypokalemia, and hyperkalemia. Acute kidney injury was defined according to KDIGO criteria.

Outcome measures

Clinical outcomes including recovery, mortality, and discharge against medical advice were recorded during hospital stay. Association between precipitating factors, severity of hepatic encephalopathy, Child-Pugh classification, and mortality was analyzed.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using SPSS software. Continuous variables were expressed as mean±standard deviation, while categorical variables were represented as frequencies and percentages. Associations between variables were analyzed using appropriate statistical tests. A p value of less than 0.05 was considered statistically significant.

Ethical considerations

Ethical approval was obtained from the institutional ethics committee prior to commencement of the study. Written informed consent was obtained from all participants or their attendants, and confidentiality of patient information was maintained throughout the study period.

RESULTS

A total of 110 patients with hepatic encephalopathy secondary to liver cirrhosis were included in the present study. Demographic profile, etiology of cirrhosis, clinical presentation, severity of hepatic encephalopathy, precipitating factors, psychometric assessment, and clinical outcomes were analyzed.

Table 1: Demographic profile and etiology of cirrhosis among study participants (n=110).

Variables	Number of patients	Percentage
Age group (years)		
21-30	6	5.5
31-40	18	16.4
41-50	34	30.9
51-60	30	27.3
61-70	17	15.5
>70	5	4.5
Gender		
Male	84	76.4
Female	26	23.6
Etiology of cirrhosis		
Alcohol-related cirrhosis	78	70.9
Viral hepatitis-related cirrhosis	22	20
Mixed etiology	10	9.1

Table 2: Clinical presentation and examination findings among study participants (n=110).

Clinical features	Number of patients	Percentage
Irrelevant speech	68	61.8
Excessive drowsiness	60	54.5
Constipation	44	40
Fever	38	34.5
Disorientation/confusion	40	36.4
Upper gastrointestinal bleeding	30	27.3
Vomiting/diarrhea	26	23.6
Reduced urine output	18	16.4
Asterixis	96	87.3
Icterus	88	80
Ascites	84	76.4
Pedal edema	70	63.6

The majority of patients belonged to the 41-60 years age group, with a mean age of 53.4±11.2 years. A marked male predominance was observed. Alcohol-related cirrhosis constituted the predominant etiology, either alone or in combination with viral hepatitis.

Table 3: Distribution of hepatic encephalopathy grades and Child-Pugh classification (n=110).

Variables	Number of patients	Percentage
West Haven Grade		
Grade 0	4	3.6
Grade I	36	32.7
Grade II	40	36.4
Grade III	20	18.2
Grade IV	10	9.1
Child-Pugh Class		
Class A	8	7.3
Class B	32	29.1
Class C	70	63.6

Irrelevant speech and excessive drowsiness were the most common presenting neurological symptoms. Asterixis, icterus, and ascites were frequently observed examination findings, indicating advanced liver dysfunction in the majority of patients.

Table 4: Neuropsychometric assessment among eligible study participants.

Test	Patients tested (n=80)	Abnormal findings
PHES test	80	78
Stroop test	80	79

Grade II hepatic encephalopathy was the most common presentation, followed by grade I. Advanced encephalopathy (grade III and IV) was observed in more than one-fourth of patients. The majority of patients belonged to Child-Pugh class C, indicating advanced liver disease.

Table 5: Distribution of identifiable precipitating factors of hepatic encephalopathy (n=110).

Precipitating factors	Number of patients	Percentage
Constipation	44	40
Infection	42	38.2
Upper gastrointestinal bleeding	30	27.3
Electrolyte imbalance	28	25.5
Vomiting/diarrhea	26	23.6
Acute kidney injury	18	16.4
Sedative/drug use	10	9.1
High protein intake	8	7.3

Psychometric evaluation using PHES and Stroop tests was performed in patients with grade 0, I, and II hepatic encephalopathy. Abnormal neuropsychometric findings were observed in the majority of evaluated patients, indicating a high prevalence of cognitive dysfunction among testable participants.

Table 6: Association between hepatic encephalopathy grade and Child-Pugh classification.

HE Grade	Child-Pugh B	Child–Pugh C
Grade I	20	16
Grade II	10	30
Grade III	2	18
Grade IV	0	10

Constipation and infections were the most common precipitating factors identified in the present study, followed by upper gastrointestinal bleeding and electrolyte imbalance. Multiple precipitating factors were observed in several patients.

Table 7: Hospital outcome among study participants (n=110).

Outcomes	Number of patients	Percentage
Improved/discharged	78	70.9
Mortality	24	21.8
DAMA	8	7.3

Higher grades of hepatic encephalopathy were predominantly observed among patients belonging to Child-Pugh class C, indicating a strong association between severity of encephalopathy and advanced liver dysfunction.

The majority of patients improved with treatment and were discharged successfully. However, mortality remained

considerable among patients with advanced hepatic encephalopathy and severe liver disease.

Table 8: Mortality according to hepatic encephalopathy grade and Child-Pugh classification.

Variables	Mortality (%)
Grade I HE	2.8
Grade II HE	10
Grade III HE	40
Grade IV HE	70
Child-Pugh Class B	9.4
Child-Pugh Class C	28.6

Mortality increased progressively with higher grades of hepatic encephalopathy and worsening Child-Pugh class. Patients with grade IV encephalopathy and Child-Pugh class C demonstrated the poorest outcomes.

DISCUSSION

The present study evaluated the clinical spectrum of precipitating factors of hepatic encephalopathy (HE) in patients with liver cirrhosis and assessed their association with disease severity and clinical outcomes. The findings demonstrate that HE commonly occurs in the setting of advanced liver disease and is frequently associated with identifiable and potentially reversible precipitating factors.

In the present study, the majority of patients belonged to the 41-60 years age group, with a mean age of 53.4±11.2 years, and a clear male predominance was observed. Similar demographic patterns were reported in studies conducted by Gautam et al, Singh et al, Khan et al, and Sethuraman et al, where middle-aged males constituted the majority of patients with HE. The observed male predominance is likely attributable to the high prevalence of alcohol-related liver disease among males in developing countries.[11,12,17,19]

Alcohol-related cirrhosis was the predominant etiology in the present study, either alone or in combination with viral hepatitis. Similar findings were reported by Bharathi et al, Khan et al, Bhattarai et al, and Singh et al, where alcohol constituted the leading cause of cirrhosis among patients presenting with HE. Chronic alcohol consumption contributes to progressive hepatic fibrosis, portal hypertension, malnutrition, and metabolic derangements, thereby increasing susceptibility to hepatic decompensation and encephalopathy.[15,17,20,24]

Irrelevant speech, excessive drowsiness, and disorientation were among the most common presenting neurological manifestations in the present study. Asterixis, icterus, and ascites were frequently observed examination findings, reflecting advanced hepatic dysfunction and portal hypertension. Similar clinical profiles were reported in earlier studies evaluating HE among cirrhotic patients.[15,17]

The majority of patients in the present study belonged to West Haven grade II hepatic encephalopathy, while advanced encephalopathy (grade III and IV) was observed in more than one-fourth of cases. Comparable findings were reported by Poudyal et al, Bhattarai et al, and Singh et al, where grade II HE was the most common clinical presentation. Advanced grades of encephalopathy are generally associated with severe hepatic dysfunction, higher ammonia levels, systemic inflammation, and poorer prognosis.[14,20,24]

Most patients in the present study belonged to Child-Pugh class C, indicating advanced liver disease. Similar observations were reported in multiple earlier studies, where the majority of HE patients had severe cirrhosis with decompensated liver function. The present study also demonstrated a clear association between worsening HE grade and advanced Child-Pugh class, suggesting that severity of hepatic dysfunction strongly influences neurological deterioration and clinical outcomes.[11,12,17,19]

Psychometric assessment using PHES and Stroop tests demonstrated a high prevalence of cognitive dysfunction among eligible patients in the present study. Similar findings have been reported in previous studies evaluating covert and minimal hepatic encephalopathy. These psychometric tools are valuable for identifying early neurocognitive impairment before development of overt encephalopathy and may help facilitate early therapeutic intervention.[25,26]

Constipation emerged as the most common precipitating factor in the present study, followed by infections, upper gastrointestinal bleeding, and electrolyte imbalance. Similar findings were observed by Khan et al, Khadka et al, and Singh et al, where constipation and gastrointestinal bleeding were among the predominant triggers of HE. Constipation prolongs intestinal transit time and increases ammonia absorption, thereby contributing to encephalopathy development.[17,18,24]

Infections constituted another major precipitating factor in the present study. Similar observations were reported by Handady et al, Poudyal et al, and Sîngeap et al, where infections such as spontaneous bacterial peritonitis, urinary tract infection, and respiratory tract infection were strongly associated with HE episodes. Infections increase systemic inflammation, worsen hepatic dysfunction, and enhance ammonia-related neurotoxicity, thereby precipitating encephalopathy.[13,14,23]

Upper gastrointestinal bleeding was identified as an important precipitating factor in the present study. Similar findings were reported in several earlier investigations. Gastrointestinal bleeding contributes to increased intestinal nitrogen load and ammonia production due to digestion of blood proteins, thereby exacerbating encephalopathy. Electrolyte imbalance, particularly hyponatremia and hypokalemia, also played a significant role in precipitation of HE in the present study. Similar

associations were demonstrated in previous studies evaluating metabolic precipitants of encephalopathy.[12,15,12,19,20,24]

Acute kidney injury was identified in a considerable proportion of patients and was associated with severe disease. Similar findings were reported by Rudler et al, where renal dysfunction and acute kidney injury were significantly associated with poor prognosis and mortality in overt HE. Impaired renal clearance of ammonia and worsening systemic hemodynamic instability may contribute to this association.[25]

The present study demonstrated that mortality increased progressively with worsening HE grade and advanced Child-Pugh class. Patients with grade IV encephalopathy and Child-Pugh class C had the poorest outcomes. Similar findings were reported by Singh et al, Khan et al, and Modhavadiya et al, where advanced encephalopathy and severe hepatic dysfunction were associated with significantly increased mortality. Presence of multiple precipitating factors, severe systemic illness, and advanced hepatic decompensation likely contribute to adverse outcomes in these patients.[12,17,21]

The majority of patients in the present study improved following identification and management of precipitating factors, emphasizing the reversible nature of HE when precipitating conditions are promptly recognized and corrected. Early diagnosis, timely treatment of infections, correction of electrolyte imbalance, prevention of constipation, control of gastrointestinal bleeding, and optimization of supportive care remain essential components in the management of hepatic encephalopathy.

Overall, the findings of the present study highlight that hepatic encephalopathy in cirrhotic patients is frequently precipitated by identifiable and potentially preventable factors. Early recognition and prompt correction of these precipitating factors, along with assessment of liver disease severity, are crucial for improving prognosis and reducing morbidity and mortality associated with hepatic encephalopathy.

Limitations

The present study has certain limitations. Being a single-center hospital-based study with a relatively small sample size, the findings may not be generalizable to the wider population. The cross-sectional study design limits the ability to establish causal relationships between precipitating factors and clinical outcomes. In addition, long-term follow-up of patients and assessment of recurrence of hepatic encephalopathy could not be performed. Further multicenter prospective studies with larger sample sizes and longer follow-up are recommended to validate these findings and better evaluate prognostic factors and long-term outcomes in patients with hepatic encephalopathy.

CONCLUSION

The present study demonstrated that hepatic encephalopathy in patients with liver cirrhosis is commonly associated with identifiable and potentially reversible precipitating factors. Alcohol-related cirrhosis constituted the predominant underlying etiology, and the majority of patients presented with advanced liver disease belonging to Child-Pugh class C. Constipation, infections, upper gastrointestinal bleeding, electrolyte imbalance, vomiting/diarrhea, and acute kidney injury were the major precipitating factors identified in the present study. Higher grades of hepatic encephalopathy showed strong association with severe hepatic dysfunction and poorer clinical outcomes. Mortality increased progressively with worsening West Haven grade and advanced Child-Pugh classification. Neuropsychometric abnormalities were highly prevalent among eligible patients, indicating significant cognitive impairment even in early stages of hepatic encephalopathy. The findings emphasize that early identification and prompt correction of precipitating factors, along with timely assessment of disease severity, play a critical role in improving prognosis and reducing morbidity and mortality among patients with hepatic encephalopathy.

Recommendations

Routine evaluation for precipitating factors should be performed in all cirrhotic patients presenting with hepatic encephalopathy to facilitate early diagnosis and prompt management. Constipation, infections, gastrointestinal bleeding, electrolyte disturbances, and renal dysfunction should be aggressively identified and corrected to prevent progression of encephalopathy and reduce recurrence. Early screening for minimal and covert hepatic encephalopathy using psychometric tools such as PHES and Stroop tests may help detect cognitive dysfunction before development of overt encephalopathy. Strict infection surveillance, careful electrolyte monitoring, rational use of diuretics and sedatives, nutritional optimization, and patient education regarding medication adherence and avoidance of alcohol are essential components of management. Patients with advanced Child-Pugh class and higher grades of hepatic encephalopathy require intensive monitoring and early referral to higher centers when necessary, due to increased risk of mortality. Further multicentric prospective studies with larger sample sizes are recommended to better evaluate regional patterns of precipitating factors and long-term outcomes in hepatic encephalopathy.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee of Government Medical College, Srikakulam

REFERENCES

1. Ginès P, Krag A, Abraldes JG, Solà E, Fabrellas N, Kamath PS. Liver cirrhosis. Lancet. 2021;398(10308):1359-76.
2. Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. Lancet. 2014;383(9930):1749-61.
3. Asrani SK, Devarbhavi H, Eaton J, Kamath PS. Burden of liver diseases in the world. J Hepatol. 2019;70(1):151-71.
4. European Association for the Study of the Liver. EASL clinical practice guidelines on the management of hepatic encephalopathy. J Hepatol. 2022;77(3):807-24.
5. Vilstrup H, Amodio P, Bajaj J, Cordoba J, Ferenci P, Mullen KD, et al. Hepatic encephalopathy in chronic liver disease: 2014 practice guideline. Hepatology. 2014;60(2):715-35.
6. Ferenci P, Lockwood A, Mullen K, Tarter R, Weissenborn K, Blei AT. Hepatic encephalopathy-definition, nomenclature, diagnosis and quantification. Hepatology. 2002;35(3):716-21.
7. Bajaj JS. Review article: the modern management of hepatic encephalopathy. Aliment Pharmacol Ther. 2010;31(5):537-47.
8. Butterworth RF. Pathogenesis of hepatic encephalopathy and brain edema in acute liver failure. J Clin Exp Hepatol. 2015;5(Suppl 1):S96-103.
9. Dhiman RK, Saraswat VA, Sharma BK, Sarin SK, Chawla YK, Butterworth R, et al. Minimal hepatic encephalopathy: consensus statement of a working party of the Indian National Association for Study of the Liver. J Gastroenterol Hepatol. 2010;25(6):1029-41.
10. Amodio P, Montagnese S, Gatta A, Morgan MY. Characteristics of minimal hepatic encephalopathy. Metab Brain Dis. 2004;19(3-4):253-67.
11. Gautam A, Sharma BC, Sarin SK. Precipitating factors of hepatic encephalopathy in liver cirrhosis. Indian J Gastroenterol. 2012;31(4):174-8.
12. Singh J, Sharma BC, Puri V, Sachdeva S, Srivastava S. Clinical profile and precipitating factors of hepatic encephalopathy in cirrhosis of liver. J Assoc Physicians India. 2018;66(9):38-42.
13. Handady SO, Ahmed ME, Elbashir MM. Precipitating factors and outcome of hepatic encephalopathy among Sudanese patients with liver cirrhosis. Sudan J Med Sci. 2017;12(1):15-22.
14. Poudyal NS, Chaudhary S, Kc S, Mandal BR. Clinical profile and precipitating factors of hepatic encephalopathy in liver cirrhosis. JNMA J Nepal Med Assoc. 2019;57(216):95-100.
15. Bharathi V, Reddy KR, Kumar A. Clinical spectrum and precipitating factors of hepatic encephalopathy in cirrhosis. Int J Adv Med. 2020;7(3):428-33.
16. Khungar V, Poordad F. Hepatic encephalopathy. Clin Liver Dis. 2012;16(2):301-20.
17. Khan M, Jamil Z, Ahmed M, Khan RA. Clinical spectrum and precipitating factors of hepatic encephalopathy in cirrhosis patients. Pak J Med Sci. 2019;35(2):468-73.
18. Khadka S, Acharya S, Shrestha DB. Precipitating factors of hepatic encephalopathy among cirrhotic patients admitted to tertiary care center. Ann Med Surg. 2022;81:104455.
19. Sethuraman R, Balasubramanian K, Prakash JA. Clinical profile and predictors of outcome in hepatic encephalopathy. Trop Gastroenterol. 2016;37(3):190-6.
20. Bhattarai S, Gyawali M, Khanal S, Bhandari R. Hepatic encephalopathy in liver cirrhosis: precipitating factors and outcome analysis. Kathmandu Univ Med J. 2021;19(74):141-6.
21. Modhavadiya Y, Shah M, Patel H. Mortality predictors in overt hepatic encephalopathy among cirrhotic patients. Cureus. 2023;15(2):e34821.
22. Sîngeap AM, Stanciu C, Huiban L, Cojocariu C, Girleanu I, Chiriac SA, et al. Clinical profile and outcomes in patients with hepatic encephalopathy. Exp Ther Med. 2021;22(2):822.
23. Rudler M, Cluzel P, Corvec TL, Benosman H, Massard J, Thabut D. Prognostic factors of overt hepatic encephalopathy in cirrhotic patients. Clin Res Hepatol Gastroenterol. 2018;42(2):124-31.
24. Weissenborn K. Diagnosis of minimal hepatic encephalopathy. J Clin Exp Hepatol. 2015;5(1):S54-9.
25. Bajaj JS, Thacker LR, Heuman DM, Fuchs M, Sterling RK, Sanyal AJ, et al. The Stroop smartphone application is a short and valid method to screen for minimal hepatic encephalopathy. Hepatology. 2013;58(3):1122-32.
26. Sharma P, Sharma BC. Management of overt hepatic encephalopathy. J Clin Exp Hepatol. 2015;5(1):S82-7.

Cite this article as: Khan YN, Mansi, Louis RS, Ashraf S, Adari CLB. Clinical spectrum and prognostic impact of precipitating factors in hepatic encephalopathy among patients with liver cirrhosis: a cross-sectional observational study. Int J Res Med Sci 2026;14:3929-35.