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Predictors of Inpatients Mortality in Patients with Liver Cirrhosis with Hepatic Encephalopathy

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ABSTRACT

Background

Hepatic encephalopathy has high risk of recurrence and is associated with advanced cirrhosis of liver. It features a spectrum of reversible neuropsychiatric abnormalities. The purpose of the research was to study the inpatients mortality and its predictors among cirrhotic patients with hepatic encephalopathy.

Methods

A cross-sectional, hospital-based study comprising of 182 cirrhotic patients with hepatic encephalopathy were evaluated. West Haven Classification (Grade I to IV) was used for grading of hepatic encephalopathy. The data analysis was done using SPSS version 20 and p-value of ≤0.05 was considered significant.

Results

Majority of patients (39.6%) presented with Grade II encephalopathy followed by Grade III (26.4%). Inpatient mortality was 22.5%. Mortality was seen in 95% patients with HE of grade IV. Univariate analysis showed male sex [p-value=0.01, OR-0.37, CI (0.17-0.820)], shock at presentation [p-value=0.0001, OR-42, CI (16.05 -109.9)], CTP [p-value=0.03, OR-3.8, CI (1.1-13.3)], presence of more than two precipitating factors [P=0.0001, OR-48, CI (10.3-216.3)], presence of SBP [p-value=0.0001, OR-23.4, CI (9.1-60.4)] as statistically significant predictive factors. Multivariate analysis showed shock [P=0.0001, OR-85.3, CI (12.7-571.7)], presence of more than two precipitating factors [p-value=0.0001, OR-101.8, CI (3.32-3119.32)] and SBP [p-value=0.0001, OR-44.7, CI (6.49-309.5)] as statistically significant predictive factors for mortality in patients with liver cirrhosis with hepatic encephalopathy.

Conclusions

Inpatient mortality was high with increasing grades of encephalopathy among cirrhotic patients. Initial presentation with shock, presence of more than two precipitating factors and SBP were independent predictors of inpatient mortality in patients with liver cirrhosis with hepatic encephalopathy.

Keywords: cirrhosis of liver; hepatic encephalopathy; mortality; predictors.

INTRODUCTION

Hepatic encephalopathy encompasses a spectrum of reversible neuropsychiatric abnormalities in patients with cirrhosis of liver in the absence of any organic brain disease.[1] Hepatic encephalopathy may be latent or overt with abnormalities in cognitive functions, disorientation, loss of consciousness, asterixis and other clinical signs.[2-4] It results due to hyperammonemia and accumulation of various neuro-toxins into the brain.[5,6] Infections and gastro intestinal bleeding are the most common precipitating factors.[7,8] Cirrhotic patients with HE can have many other co morbidities and varied complications like ascites, splenomegaly, spontaneous bacterial peritonitis, and upper gastrointestinal bleed from ruptured esophagogastric varices that increases morbidity and mortality.[1] Patient outcome depends

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on early identification of the precipitating factor and early treatment and management.[9] National data on mortality of hepatic encephalopathy are scanty. This research was undertaken to study the mortality and its predictors among cirrhotic patients with hepatic encephalopathy admitted at a tertiary care teaching hospital in Nepal.

METHODS

This hospital based cross-sectional study was carried out in the unit of Medical Gastroenterology, department of Medicine at Manipal College of Medical Sciences and Teaching Hospital, Nepal from October 2021 to March 2024 for a period of 30 months.

The sample size was collected using the formula,

$n= 1.96*1.96x\ p\ x\ q\ /e^2$

where, p= prevalence

q= 1-p

e = margin of error (0.05 i.e., 5%)

From a previous study,

Iqbal et al,[8] mortality was 11 % in cirrhotic patients with hepatic encephalopathy (p=11), sample size (n) = (1.96*1.96 x 11 x 89)/25 = 157. Adding 10% for missing data, the sample size of 173 was reached.

So, the minimal sample size adequate for the study was 173. The study was approved and verified by the Institutional Review (MEMG/IRC/373/GA). Informed consent was taken from patient relatives. All consecutive cirrhotic cases admitted in Intensive care Units (ICU) and / or ward with features of hepatic encephalopathy were included in the study. Clinical diagnosis of cirrhosis was made in presence of jaundice, ascites, splenomegaly and other stigmata of chronic liver disease. All these study subjects had sonological and/ or endoscopic evidence of portal hypertension. West Haven Classification (Grade I to IV) was used for grading of hepatic encephalopathy in patients with cirrhosis of liver. Grade 1 represents cirrhotic patients with mild confusion, lack of awareness and shortened attention span. Grade 2 represents patients with drowsiness, lethargy or apathy, minimal disorientation for time or place, subtle personality changes, inappropriate behavior and impaired simple mathematical calculations and presence of asterixis. Patients with increased somnolence but arousable, marked confusion with gross disorientation were included in grade 3 and patients with Coma (unresponsive to verbal or noxious stimuli) were included in Grade 4 hepatic encephalopathy. Child Turcotte Pugh (CTP A, B or C) scoring system was used to assess the severity. Patients with hepatic encephalopathy without evidence of cirrhosis of liver, those with acute fulminant hepatitis or those with non cirrhotic portal hypertension and those with incomplete records were excluded from the study. Data regarding demographic variables, complications and precipitating factors etc. were studied and alongside blood investigations like complete blood count, platelets count, blood grouping, liver function test, prothrombin time / international normalized ratio (PT / INR), coagulation profile and viral serologies were collected. Ultrasonography (GE Healthcare GE Logiq P3, Chicago, Illinois, United States) of the abdomen was done for assessment of liver, spleen, parenchymal echogenicity, portal vein diameter and ascites. Computed Tomography scan (PHILIPS Ingenuity 128 CT Scanner, NETHERLANDS Inc) of Brain was also done in few selected cases to rule out any other causes of loss of consciousness or altered sensorium. Upper GI endoscopy (PENTAX EPK 700, PENTAX JAPAN Inc) was done for screening of varices and therapeutic interventions in the form of variceal banding and/ or hemostatsis was achieved in patients with upper GI bleed. The data analysis was done using SPSS version 20. All categorical data were expressed in percent and absolute number. All numerical continuous data were expressed in mean ± SD. Statistical analysis was done with student's t test for continuous variables. Chi squared test or Fisher's exact test (wherever Chi squared test was not feasible) were used to test for significant difference of proportions for categorical data. All tests were analyzed with a 95% confidence interval and a P value of ≤ 0.05 was considered significant. Univariate and multivariate analysis models were used to test significance of predictive factors for mortality.

RESULTS

A total of 191 cirrhotic patients with hepatic encephalopathy were admitted during the study period.

But nine patients were excluded because of inadequate data. Finally, a total of 182 cirrhotic subjects were taken up for the study. The mean age of subjects was 55.61±11.45 years with a range of 32-74 years and maximum cases in 40-60 years of age group. The study population comprised of 143 (78.5%) males and 39 (21.5%) females (M: F=3.7:1). The most common etiology of cirrhosis was chronic alcohol consumption leading to alcoholic liver disease in 165 (90.7%). Ten (5.5%) cases were of chronic Hepatitis B and rest 7(3.8%) were of chronic Hepatitis C (Table 1).

Table 1. Etiologies and co morbidities in cirrhotic subjects. (n=182)

Variables	Frequency (%)
Etiology of Cirrhosis	
Alcohol related	165 (90.7)
Chronic HBV	10 (5.5)
Chronic HCV	7 (3.8)
Co morbidities	
Bronchial Asthma	3 (1.6)
Congestive Cardiac Failure	18 (9.9)
Chronic Kidney Diseases	5 (2.7)
COPD	9 (4.9)
CVA	6 (3.3)
Diabetes Mellitus	36 (19.8)
Hypertension	32 (17.6)
Ischemic Heart Disease	10 (5.5)
None	23 (12.6)
Pulmonary Tuberculosis	3 (1.6)
Rheumatoid Arthritis	7 (3.8)
Others	30 (16.5)

*COPD: Chronic Obstructive Pulmonary Disease, CVA: Cerebro Vascular accident

Cirrhotic subjects with encephalopathy were classified according to CTP classes. Majority of patients (n=146, 80.2%) were of Child grade C. The most common presentation was irrelevant talking (n=156, 85.7%) followed by disorientation (144, 79.1%), increased day time sleepiness (117, 64.3%) and loss of consciousness (69, 37.9%). Majority of patients (n=72, 39.6%) presented with Grade II encephalopathy followed by Grade III (n=48, 26.4%) and Grade I encephalopathy (n=42, 23.1%). Twenty patients (11%) had Grade IV encephalopathy (Table 2).

The most common precipitating factor was upper GI bleed (n=126, 69.2%) followed by dyselectrolytemia (n=64, 35.2%), repeated large volume paracentesis (n=59, 32.4%) and spontaneous bacterial peritonitis

Table 2. Information on various parameter of the study subjects. (n=182)

Variables	Frequency (%)
HE Grades	
I	42 (23.1)
II	72 (39.6)
III	48 (26.4)
IV	20 (11.0)
UGI Bleed	
No	56 (30.8)
Yes	126 (69.2)
Acute Kidney Injury	
No	151 (83.0)
Yes	31 (17.0)
Spontaneous Bacterial Peritonitis	
No	150 (82.4)
Yes	32 (17.6)
Sepsis	
No	154 (84.6)
Yes	28 (15.4)
CTP	
B	36 (19.8)
C	146 (80.2)
Ascites	
Gross	122 (67.0)
Min	58 (31.9)
None	2 (1.1)
Repeated Large Volume Paracentesis	
No	123 (67.6)
Yes	59 (32.4)
Shock	
No	139 (76.4)
Yes	43 (23.6)
More than two precipitating factors	
No	104 (57.1)
Yes	78 (42.9)

(SBP) (n=32, 17.6%). Other precipitating factors were sepsis (n=28, 15.4 %), chronic constipation (n=48, 26.4%), continuous alcohol use in last one week (n=26, 14.3%), and use of sedatives or hypnotics (n=8, 4.4%). In this study, 78 (42.9%) had more than 2 precipitating factors. Average hospital stays of the cirrhotic patients with HE was 9 days. Patients presenting with ≥2 precipitating factors and advanced grade of HE i.e., Grade > II and CTP class C had a prolonged mean hospital stay of 13 days. Hospital stay was also prolonged (>9 days) in patients with co morbidities and in those requiring regular large volume paracentesis. One Hundred and forty-one (77.5%) patients were discharged home after

Table 3. Association of death with various variables. (n=182)				
Variables	Death		Chi-square Value	p-value
	Yes (%)	No (%)		
Sex				
Female	14(37.8)	23(62.2)	1.24	0.075
Male	27(18.6)	118(81.4)		
HE Grades				
I	0(0)	41(100)	83.186	0.001*
II	6(8.2)	67(91.8)		
III	16(33.3)	32(66.7)		
IV	19(95)	1(5)		
Etiology of CLD				
Alcohol related	37(22.4)	128(77.6)	0.184	0.912
Chronic HBV	2(20)	8(80)		
Chronic HCV	2(28.6)	5(71.4)		
UGI Bleed				
No	2(3.5)	54(96.5)	3.405	0.03*
Yes	39(31)	87(69)		
Acute Kidney Injury				
No	32(21.2)	119(78.8)	3.906	0.048*
Yes	9(29)	22(71)		
SBP				
No	17(11.3)	133(88.7)	61.254	0.023*
Yes	24(75)	8(25)		
Sepsis				
No	34(22.1)	120(77.9)	0.116	0.73
Yes	7(25)	21(75)		
CTP				
B	3(8.3)	33(91.7)	5.18	0.001*
C	38(26)	108(74)		
Ascites				
Gross	33(27)	89(73)	6.266	0.044*
Min	7(12.1)	51(87.9)		
None	1(50)	1(50)		
Shock				
No	9(6.5)	130(93.5)	88.86	0.002*
Yes	32(74.4)	11(25.6)		
More than 2 precipitating factors				
No	1(1)	103(99)	64.66	<0.001*
Yes	40(51.3)	38(48.7)		
Blood Transfusion				
≥3 units	40(23.8)	128(76.2)	2.05	0.15
<3 units	1(7.1)	13(92.9)		

*Statistically significant at 5% level of significance.

successful treatment. Forty-one (22.5%) had inpatient mortality. Mortality was maximum accounting to 19 (95%) patients with HE grades IV, followed by 16(33.3%) in HE grades III and 6(8.2%) in HE grades II. Significant association was observed between higher grades of encephalopathy with mortality (P=0.001). No mortality was observed in patients with HE grades I. Mortality of patients was 26% in class C and only 8.3% in class B. Significant association was observed between CTP class C with mortality (p=0.001). When more than 2 precipitating factors were present, mortality was seen in 40(51.3 %) patients (p<0.001) (Table 3). Upper GI bleed, Acute Kidney Injury (AKI), SBP, gross ascites and presentation with shock were complications associated with mortality (P<0.05) (Table 3).

Similarly, leukocytosis (≥15000), hyponatremia (<130), raised ALT (> 80) and increased INR (>2.3) were the laboratory parameters with significant predictors of mortality (P<0.05) (Table 4).

Table 4. Association of death with various lab parameters. (n=182)				
Lab parameter	Death		Chi-square value	p-value
	Yes	No		
Hemoglobin				
<10	39(23.1)	130(76.9)	0.409	0.522
≥10	2(15.4)	11(84.6)		
WBC				
<15000	23(14.9)	131(85.1)	33.06	<0.001*
≥15000	18(64.3)	10(35.7)		
Serum Sodium				
<130	17(40.5)	25(59.5)	4.06	0.045*
≥130	24(18.5)	106(81.5)		
Total bilirubin				
>3	22(25.3)	65(74.7)	0.727	0.394
<3	19(20)	76(80)		
ALT				
≥80	34(24.8)	103(75.2)	7.96	0.005*
<80	17(37.8)	28(62.2)		
Albumin				
<2.8	26(22.8)	88(77.2)	0.014	0.907
≥2.8	15(22.1)	53(77.9)		
INR				
≥2.3	34(23.4)	111(76.6)	5.34	0.04*
<2.3	7(18.9)	30(81.1)		

*Statistically significant at 5% level of significance.

Table 5. Univariate and multivariate analysis of death for different variables.				
Risk Factors Level	**No mortality**	**Mortality**	**Univariate**	**Multivariate**
Sex				
Male	118	27	**P= 0.01; OR= 0.3 7 CI (0.17-0.82)**	
Female	23	14		
UGI Bleed				
No	54	2		
Yes	87	39	P= 0.11; OR= 0.13 CI (0.01-1.57)	
AKI				
No	119	32		
Yes	22	9	P= 0.34; OR=1.5 CI (0.63-3.6)	
Sepsis				
No	120	34		
Yes	21	7	P= 0.73; OR= 1.1 CI (0.46-3.0)	
Ascites				
No	1	1		
Yes	140	40	P= 0.38; OR= 0.28 CI (0.01-4.6)	
Shock				
No	130	9		
Yes	11	32	P= 0.0001; OR= 42 CI (16.05-109.9)	P= 0.0001; OR= 85.3 (12.7-571.7)
CTP				
B	33	3		
C	108	38	P= 0.03; OR= 3.8 (1.1-13.3)	
More than 2 precipitating factors				
No	103	1		
Yes	38	40	P= 0.0001; OR= 48 (10.3-216.3)	P= 0.0001; OR= 101.8 (3.32 -3119.32)
SBP				
No	133	17		
Yes	8	24	P= 0.0001; OR= 23.4 (9.1-60.4)	P= 0.0001; OR= 44.7 (6.48-309.5)
INR>2.3				
No	114	34		
Yes	27	7	P= 0.76; OR= 0.86 (0.34-2.1)	

Table 5 depicts the associated risk factors that are responsible for mortality in patients presenting with liver cirrhosis with hepatic encephalopathy. Univariate analysis showed risk factors like male sex [P= 0.01, OR= 0.37 CI- 0.17-0.82], patients presenting with shock [P= 0.0001, OR= 42.0 CI- 16.05-109.9], Child's C [P= 0.03, OR= 3.8 CI- 1.1-13.3], patients with more than 2 precipitating factors [p= 0.0001, OR= 48, CI-10.3-216.3] and SBP [P=0.001, OR= 23.4 CI- 9.11-60.4] to be clinically significant. However, multivariate analysis showed shock at initial presentation [P= 0.0001, OR- 85.3 CI- 12.7-571.7], presence of more than 2 precipitating factors [p= 0.0001, OR= 101.8, CI- 3.32-3119.32] and SBP [P= 0.0001, OR= 44.7 CI- 6.49- 309.5] as clinically significant risk factors to predict mortality in patients with liver cirrhosis with HE.

DISCUSSION

The mean age of subjects was 55.61±11.45 years with male predominance (78.5%) (M: F=3.7:1). The mean age of subjects were lower; 47 years, 49.58 years and 51.25 years and all had male dominance in the studies by Raphael et al.,[10] Sethuraman et al[11] and Kumar et al.[12] respectively. Alcohol being the most common aetiology of cirrhosis and alcohol drinking patterns and age of starting taking excessive alcohol may differ in various places. Majority of patients (39.6%) presented with Grade II encephalopathy followed by Grade III (26.4%) and Grade I encephalopathy 23.1%) in the current study. Grade IV encephalopathy was present in rest 11%. Similar were the findings in the the studies by Sethuraman et al.[11], Kumar et al.[12], Mumtaz et al.[13], Singh et al.[14] and Poudyal et al.[15] as patients

presented commonly with Grade II and III HE. Contrary to above studies, majority (36%) of the patients were in grade IV HE followed by 30% in grade III ,10% in grade II and 24% had grade I HE in the study by Nayak et al.[16] This study confirmed the previous findings by Raphael et al[10] and Sethuraman et al.[11], who described majority of the cirrhotic patients who present with HE were of Child grade C. Earlier grades or latent HE may not come to hospital for admissions. Average days of hospital stay was 9 days in the current study, which was lesser by 2 days compared to the study by Iqbal et al.[17] Patient co morbidities and other complication due to liver cirrhosis have a detrimental role in prolonging hospital stays. Inpatient mortality was 22.5% in the current study. It was almost similar (23%) in the study by Devrajani et al[18] in Hyderabad, India. Inpatient mortality was higher, 29% and 30% in the studies by Arisar et al.[19] and Maqsood et al.[20] in Pakistan respectively. It was however, lower in the studies by Sethuraman et al[11], Khan et al.[21] and Iqbal et al.[17] who reported mortalities of 17.48%, 15.3% and 15 % respectively. In a previous study by Poudyal et al.[15] in Nepal, mortality was observed in 16.7% patients. Mortality in these patients of liver cirrhosis depend on the stages of HE as well as many other varied complications and other co morbidities. The most important predictor of mortality in these cirrhotic patients were HE of grade IV, Child class C and presence of more than 2 precipitating factors. Mortality was maximum accounting to 95% with HE grades IV and no mortalities in grade I. Mortality was 50% patients in Grade IV hepatic encephalopathy by Singh et al.[14] Similar were the findings in the study by Nyak et al.[16], where the highest mortality rate was found in grade IV. No mortalities were observed in HE grades I, findings in consistency with the present study. The higher grades of HE need vigilant ICU care and these cases are usually associated with more precipitant factors and other complications of liver cirrhosis as well. Among patients who died, 92.7% patients belonged to Child C class in the current study and 66.7% in the study by Singh et al.[14] Majority of patients who expired belonged to class C according to Sethuraman et al.[10] and Poudyal et al.[15] Overall mortality was 26% among CTP class C in the current study. The mortality rate of patients in Child class C were 20% and 37% in the studies by Sethuraman et al.[11] and Nayak et al.[15] respectively. The higher the CTP class, more is the grading of HE and other complications like ascites and coagulability. In this study, mortality rose to 51.3% in presence of more than 2 precipitating factors. The mortality was even higher (66.7 %) in presence of more than 2 precipitating factors in the study by Mumtaz et al.[12] Infections, gastro intestinal bleeding and dyselectrolytemia have been described as the most common precipitating factors and causes of mortality in HE in published literatures.[11-15] Upper GI bleed, Acute Kidney Injury (AKI), SBP, gross ascites and shock were other important and significant predictors of mortality ($P<0.05$) in this study. Infections, gastro intestinal bleeding are important aetiologies for SBP (spontaneous bacterial peritonitis) whereas, these factors along with shock, any form of kidney injury causes azotemia that adds insults on HE. Renal disease is associated with impaired immune function, characterized by abnormalities in the function of monocytes, neutrophils, and the associated leukopenia . Evidence also indicates that AKI is associated with spontaneous bacterial peritonitis.[24] Similarly, leukocytosis (≥15000), hyponatremia (<130) raised ALT(>80) and increased INR (>2.3) were the laboratory parameters with significant predictors of mortality in the current study ($P<0.05$). Hyponatremia (serum sodium < 130 mEq / l) was the independent predictive factor of overt HE in the study by Guevara et al.[22] Similarly, cirrhotic patients presenting with acute variceal bleeding with Child C, serum potassium < 3.5 mmol/L, WBC > 10,000 cells/mm3, and hemoglobin < 8 gm/dL were significant predictors for mortality in hepatic encephalopathy according to Rattanasupar et al.[23]

CONCLUSIONS

Majority of patients presented with Grade II Hepatic encephalopathy. Inpatient mortality was high with

increasing grades of encephalopathy. Shock at initial presentation, presence of more than two precipitating factors and SBP were statistically significant risk factors that predicted inpatients mortality in patients with liver cirrhosis with hepatic encephalopathy.

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