

CLINICAL PROFILE OF PATIENTS WITH HEPATIC ENCEPHALOPATHY**Dr Gopalakrishna P¹, Dr Raghavendra B C², Dr K S Satish Ramana³, Dr Prashanth B V⁴**

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Abstract**INTRODUCTION**

Hepatic encephalopathy is a complex, potentially reversible neuropsychiatric condition that occurs as a consequence of acute or chronic liver disease. Hepatic encephalopathy may arise spontaneously but more commonly will develop as a result of some precipitating factor in the course of acute or chronic liver disease. Hepatic encephalopathy is a major clinical complication of Cirrhosis of liver. In our sub-continent due to the high prevalence of Hepatitis B and C, Cirrhosis of liver is becoming more common in our day-to-day clinical practice. Early identification of precipitating factors is extremely important in diagnosis and treatment of this fatal condition. The clinical course of hepatic encephalopathy can be interrupted in majority of patients by controlling these precipitating factors.

METHODS AND METHODOLOGY

The present is Observational study included 50 cases of hepatic encephalopathy diagnosed clinically admitted in Department of General Medicine, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru over a period of 1 year. On admission a detailed history was noted and thorough clinical examination was done. For data collection, a questionnaire was developed. History of previous episodes of jaundice, blood transfusions, IV abuse, drug intake and alcoholism with reference to the type, duration, amount & frequency was elicited. History regarding alteration in sleep pattern & neurological symptoms were enquired into. Diagnosis of hepatic encephalopathy was done on clinical grounds only in most of the cases. The use of neuroimaging modalities was limited to circumstances where there were strong clinical indicators for other diagnoses. Neuropsychiatric assessment along with EEG was done if the diagnosis was in doubt.

RESULTS

In the present study hepatic encephalopathy was most commonly associated with cirrhosis / chronic liver disease in 37 (74%) patients. One patient developed hepatic encephalopathy after 20 days following TIPPS procedure. Acute liver cell failure was seen in 12 (24%) patients of whom 9 cases were due to malaria. Majority of patients of hepatic encephalopathy in the present study belong to Grade IV (36%) and Grade III (31%) of West Haven classification followed by 22% in Grade I and 11% in Grade II. Among 50 patients, 9 patients (24%) belong to type A and 37 patients (74%) belong to type C. There was 1 patient (2%) in type B. About 61% of patients in the study had history of consumption of alcohol in significant amounts. Hepatitis B virus infection was found in 12 (24%) patients, of them 4 (8%) were also positive for hepatitis C. Hepatitis C virus infection was found in 6 (12%) patients.

CONCLUSION

From this study, it was concluded that in most of the cases there are different factors which play a key role in precipitating hepatic encephalopathy which is a common phenomenon in patients with liver disease. Infections, upper GI bleed, constipation and electrolyte imbalance were the most common precipitating factors. There is definite need for health education in patients who are diagnosed with cirrhosis of liver regarding the risk of hepatic encephalopathy and its precipitating factors.

Keywords: Hepatic encephalopathy, Cirrhosis of liver, GI bleed.

INTRODUCTION

Hepatic encephalopathy is a complex, potentially reversible neuropsychiatric condition that occurs as a consequence of acute or chronic liver disease. Hepatic encephalopathy may arise spontaneously but more commonly will develop as a result of some precipitating factor in the course of acute or chronic liver disease. ^[1]

Hepatic encephalopathy is a major clinical complication of Cirrhosis of liver. In our sub-continent due to the high prevalence of Hepatitis B and C, Cirrhosis of liver is becoming more common in our day-to-day clinical practice. ^[2]

Although the onset of hepatic encephalopathy can rarely be pinpointed clinically, it is a clinical landmark in patients with advanced liver disease, invariably signaling a worsening medical condition. Severe hepatic encephalopathy in patients with cirrhosis is associated with a mortality of more than 50% in the first year alone. ^[3,4]

Hepatic encephalopathy in patients with cirrhosis does not decisively limit eligibility for liver transplantation, although patients often die while they are on the waiting list for a transplant. ^[5] Similarly, among patients with fulminant hepatic failure, progression from acute hepatic encephalopathy to brain edema is associated with a high mortality. The rate of death is substantially lower for patients who receive a transplant. ^[4]

The survival rate is more than 70% in the first 5 years after transplantation, although only one of five patients with fulminant hepatic failure receives a transplant. Hepatic encephalopathy is not diagnosed and graded exclusively by specialists in chronic liver failure. Patients may first see a general practitioner, an emergency physician, or a hospitalist. ^[5]

Early identification of precipitating factors is extremely important in diagnosis and treatment of this fatal condition. The clinical course of hepatic encephalopathy can be interrupted in majority of patients by controlling these precipitating factors. Thus, this study aims at studying the clinical profile and the spectrum of precipitating factors of hepatic encephalopathy. ^[6]

Hence early and accurate diagnosis and proper identification of precipitating factors will help in initiating the appropriate treatment and thereby bringing down the morbidity and mortality.

METHODS AND METHODOLOGY

The present is Observational study included 50 cases of hepatic encephalopathy diagnosed clinically admitted in Department of General Medicine, Sathagiri Institute of Medical Sciences and Research Centre, Bengaluru, over a period of 1 year.

On admission a detailed history was noted and thorough clinical examination was done. For data collection, a questionnaire was developed. History of previous episodes of jaundice, blood transfusions, IV abuse, drug intake and alcoholism with reference to the type, duration, amount & frequency was elicited. History regarding alteration in sleep pattern & neurological symptoms were enquired into.

A complete history regarding precipitating events of hepatic encephalopathy like GI bleeding, high protein intake & overzealous use of diuretics or vigorous paracentesis were noted.

A detailed systemic examination was carried out including a general examination pertinent to the stigmata of chronic liver disease like anemia, clubbing, jaundice, testicular atrophy, loss of axillary hair & gynaecomastia. The skin was carefully examined to detect purpuric spots, ecchymoses, bruises, palmar erythema and spider angiomas.

Examination of the abdomen was carried out to detect any distention, dilated veins, signs of free fluid, organomegaly & other palpable mass lesions. The abdomen was carefully auscultated with a view to detect any venous hum.

Evaluation of the mental status and neurological examination was done to know the presence of flapping tremor, level of consciousness and personality changes. Evidence of any constructional apraxia was recorded using simple number connecting test.

Routine haematological investigations, biochemical parameters and specific investigations such as liver function tests, viral markers etc were performed. Special investigations like serum iron levels, ceruloplasmin levels, levels of alpha 1 antitrypsin were also performed as and when required. An abdominal ultrasound was performed to look for liver size, its parenchymal echogenicity, portal vein diameter, splenomegaly and free fluid. In patients with ascites, paracentesis was done and fluid was sent for biochemical and microbiological analysis to find out any evidence of infection. Upper GI endoscopy was done to detect any bleeding varices. Any evidence of other co-existent complications of cirrhosis liver like portal hypertension, hepatorenal syndrome and hepato-pulmonary syndrome was also recorded.

Diagnosis of hepatic encephalopathy was done on clinical grounds only in most of the cases. The use of neuroimaging modalities was limited to circumstances where there were strong clinical indicators for other diagnoses. Neuropsychiatric assessment along with EEG was done if the diagnosis was in doubt. Hepatic encephalopathy was graded according to the West Haven criteria.

Inclusion Criteria:

1. Patients with clinical symptoms and signs of hepatic encephalopathy.
2. Patients with age more than 18 years irrespective of sex

Exclusion Criteria :

The following patients were excluded from the study.

1. The cases of hepatic encephalopathy arising due to obstructive jaundice because of surgical causes like stones, strictures, mass lesions etc.
2. All cases of cirrhosis due to cardiovascular causes.

Statistical Analysis :

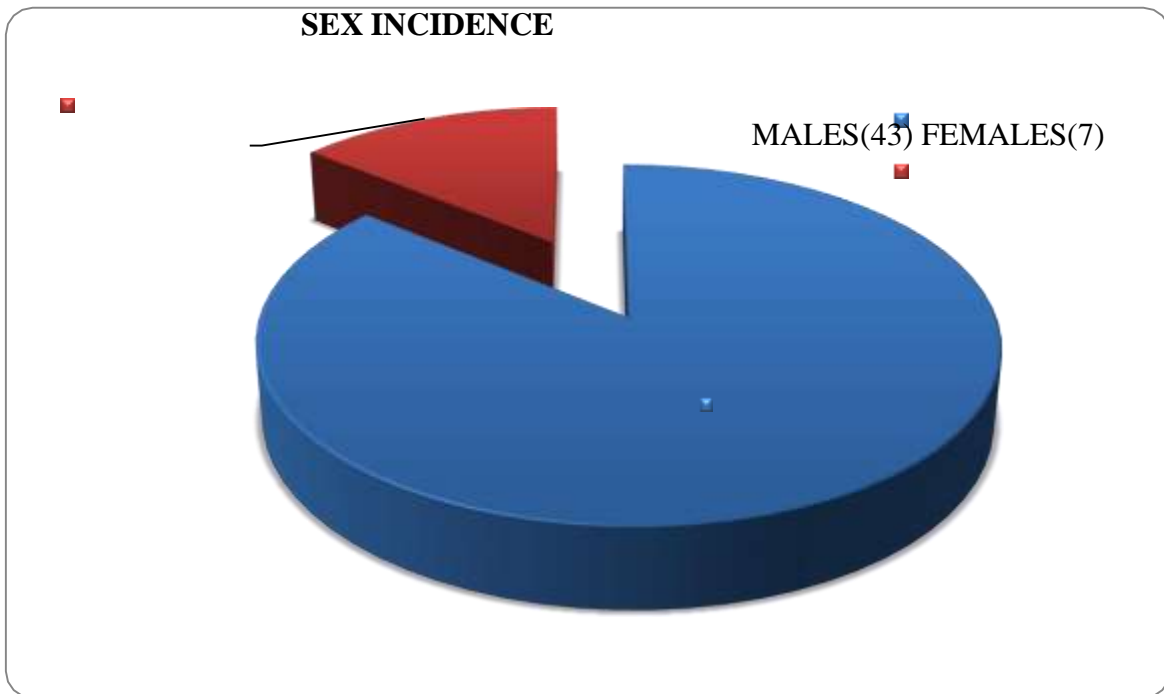
Results will be expressed in percentages (%).

RESULTS

Sex Incidence :

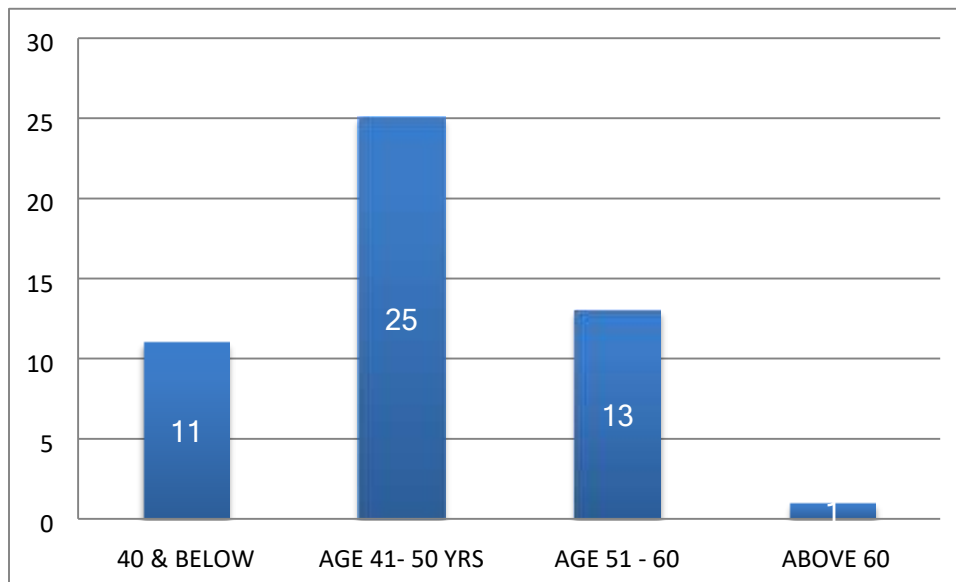
In the present study which included 50 patients, there was a male preponderance with 43 (86%) to 7(14%) females.

GRAPH 1: SEX INCIDENCE



Age Incidence:

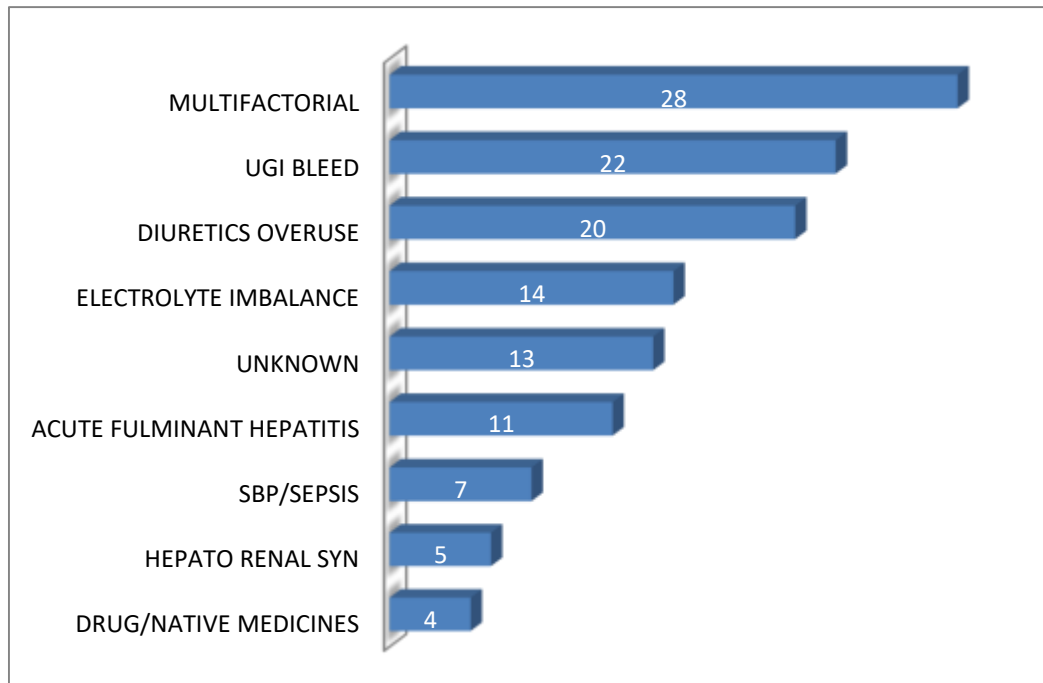
Most of the patients i.e. 25 (50%) are in the age group between 41 — 50 yrs, out of these 24 (96%) were males and 1(4%) female. Eleven (22%) patients were in the age group 40 and below, of these eight (73%) were males and 3 (27%) females. There were 13 (26%) patients in the age group 51 -60 yrs and only one patient above 60 years.

GRAPH 2 : AGE INCIDENCE**Precipitating factors :**

In the present study the most common precipitating factors of Hepatic encephalopathy were GI bleeding 22 (44%), dehydration with diuretic overuse 20 (40%) and hypokalemia 14 (28%). However, in about 28 (56%) patients precipitating cause was multifactorial which predominantly included sepsis (LRTI), constipation, and increased protein intake. In about 13 (26%) patients no precipitating cause could be elicited. Of the 11 (22%) patients who had evidence of acute Fulminant hepatitis Plasmodium Falciparum was found to be positive in 7 (63%) patients.

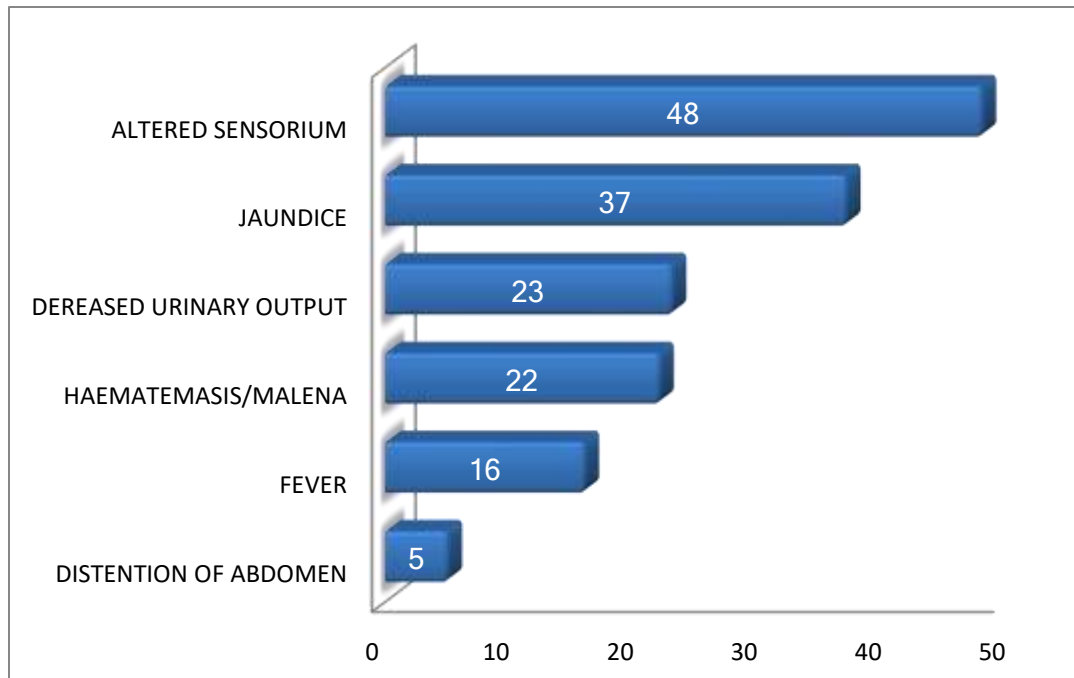
The other factors are as charted below: -

GRAPH 3 : PRECIPITATING FACTORS



Analysis of symptoms:

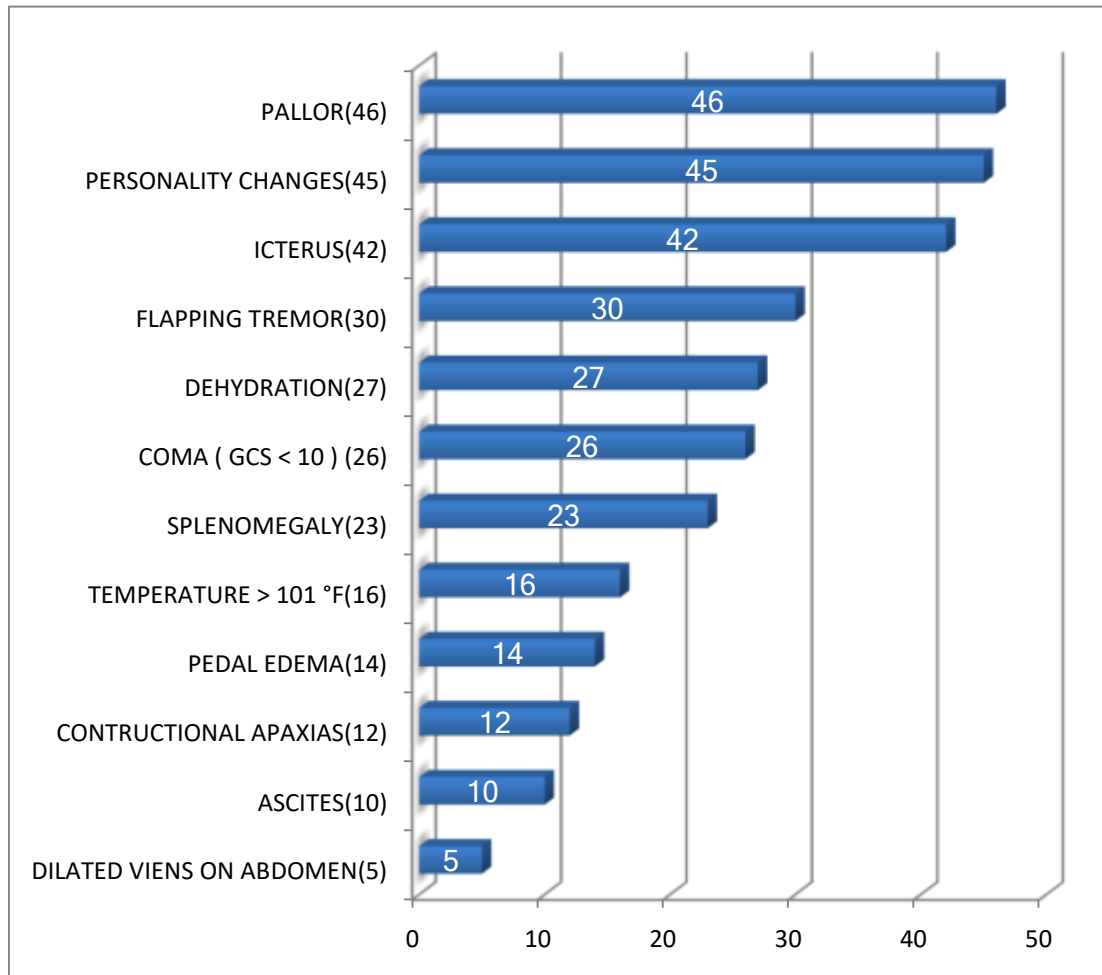
In the present study, altered sensorium ranging from drowsiness to coma was seen at presentation in 48 (96%) patients, the other two developed hepatic encephalopathy during hospital stay. Thirty seven (74%) patients had jaundice at time of presentation. Twenty three (46%) patients complained of decreased urine output at presentation. Fever was documented in 16(32%) of patients. Although only 5 patients complained distention of abdomen, ascites was clinically documented in 10 (20%) patients. Symptoms of GI bleeding in the form of hematemesis and malena were seen in 22 (44%) patients.



GRAPH 4 : ANALYSIS OF SYMPTOMS

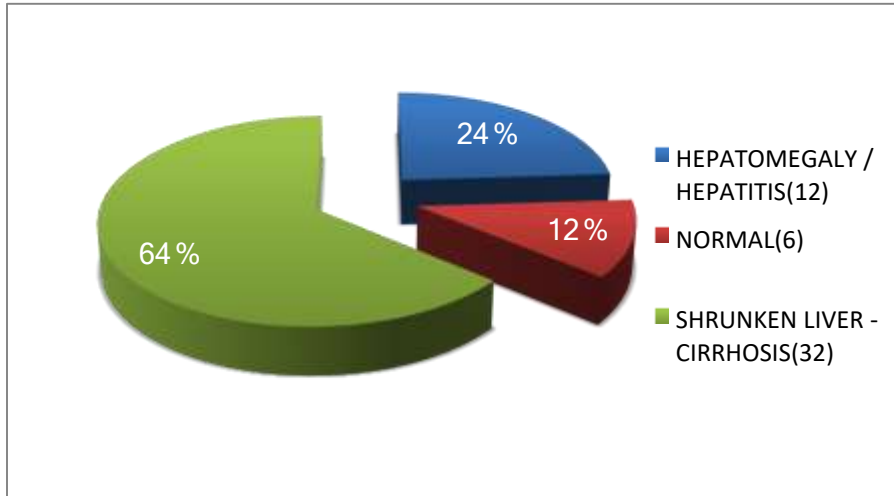
Analysis of signs:

Grade IV hepatic encephalopathy was seen in 26 (52%) patients at presentation. Signs of dehydration were evident in 27 (54%) patients. Flapping tremor was elicited in 60% of patients, if not at presentation, later during their course in hospital. Constructional apraxia could be elicited only in 24% of patients, where as inversion of sleep rhythm was observed in up to 95% of patients. The other signs are charted as below.

GRAPH 5 : ANALYSIS OF SIGNS**USG abdomen findings:**

Liver size was shrunken in 32 (64%) patients. Hepatomegaly was seen in 12 patients majority of them presented with acute fulminant hepatitis.

GRAPH 6: LIVER – ULTRASOUND ABDOMEN FINDINGS



Etiology :

In the present study hepatic encephalopathy was most commonly associated with cirrhosis / chronic liver disease in 37 (74%) patients. One patient developed hepatic encephalopathy after 20 days following TIPPS procedure. Acute liver cell failure was seen in 12 (24%) patients of whom 9 cases were due to malaria.

GRAPH 7: ETIOLOGY OF HEPATIC ENCEPHALOPATHY (HE)

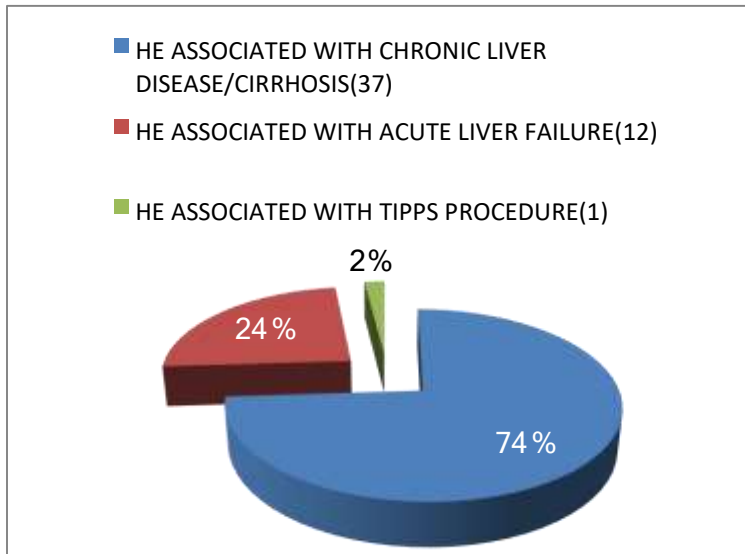


TABLE 1 : WEST HAVEN CLASSIFICATION

Stage	No. of Patients
I	11
II	5
III	16
IV	18

Majority of patients of hepatic encephalopathy in the present study belong to Grade IV (36%) and Grade III (31%) of West Haven classification followed by 22% in Grade I and 11% in Grade II.

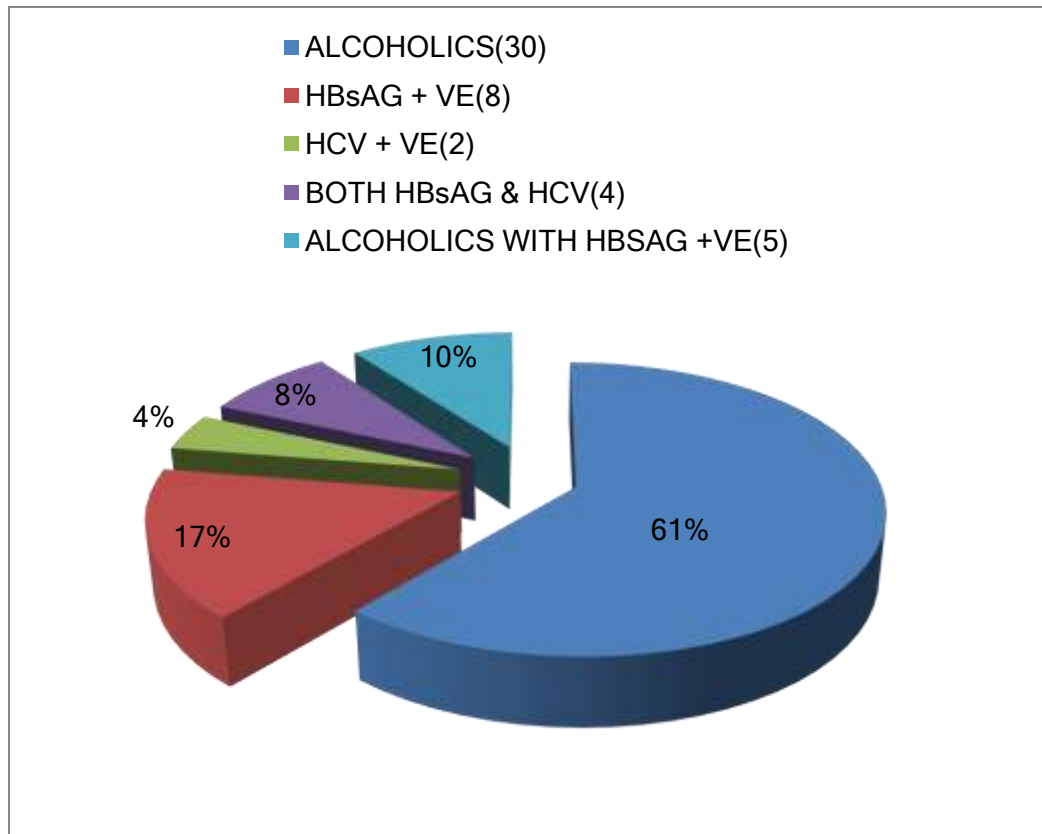
TABLE 2: TYPE OF HEPATIC ENCEPHALOPATHY

Type of Hepatic Encephalopathy	No. of Patients
Type – A	12
Type – B	1
Type – C	37
Total	50

Among 50 patients, 9 patients (24%) belong to type A and 37 patients (74%) belong to type C. There was 1 patient (2%) in type B.

Risk factors for chronic liver disease:

About 61% of patients in the study had history of consumption of alcohol in significant amounts. Hepatitis B virus infection was found in 12 (24%) patients, of them 4 (8%) were also positive for hepatitis C. Hepatitis C virus infection was found in 6 (12%) patients.

Graph 8: RISK FACTORS FOR CHRONIC LIVER DISEASE

DISCUSSION

The most common precipitating factors for hepatic encephalopathy detected were infection (67%), constipation (49%) and gastrointestinal bleeding(45%) by Bikha Ram Devrajani et al. [8] Most common precipitants of hepatic encephalopathy were infections (20.5%) and constipation (18.3%) by Mumtaz K et al. [9] The identified precipitants for hepatic encephalopathy were sepsis (29%), electrolyte imbalance (14%), gastrointestinal bleed (24%) and drugs (5%) by Onyekwere CA et al. [10] Infection (44%), gastrointestinal bleeding (38%) and constipation (38%) stood out as the most common precipitating factors for hepatic encephalopathy by Maqsood S et al. [11]

Electrolyte imbalance in 56% patients, diarrhea in 40%, constipation in 32%, infections in 24% and gastrointestinal bleed in 22% patients were amongst the commonest precipitating factors for hepatic encephalopathy by Islam et al, [12] whereas 30% had constipation, 29% had upper gastrointestinal bleed in patients with HE by Mohammad Tariq et al. [13] Constipation (32.9%) and upper GI bleed (31.4%) were the commonest precipitating factors by Khurram et al. [14]

Majority of the patients (80%) in the present study had encephalopathy complicating underlying chronic liver disease with only 18% patients had acute fulminant hepatic failure. 1 (2%) case of type B were seen in the present study. Type C was the most common type in majority of the studies as shown in the table.

Caution must be exercised in putting cirrhotic patients on diuretics early and effective infection control measures and better hygienic conditions in hospitals should be maintained. [15] Consistent use of lactulose and fibre should be encouraged to prevent constipation. More and more endoscopic facilities should be made available nationwide for prompt control of gastrointestinal bleeding. Only then there are any chances of combating cirrhosis and even worse hepatic encephalopathy. [16]

From this study, it was concluded that in most of the cases there are different factors which play a key role in precipitating hepatic encephalopathy which is a common phenomenon in patients with liver disease. Infections, upper GI bleed, constipation and electrolyte imbalance were the most common precipitating factors.

There is definite need for health education in patients who are diagnosed with cirrhosis of liver regarding the risk of hepatic encephalopathy and its precipitating factors. Priority should be given to these factors in terms of hospital funds, blood banks, medicines and human efforts. No stone should be left unturned in providing better and effective infection control measures and better hygienic conditions in hospitals. The proper disposal of infected hospital waste and especially syringes, blades should be performed. Consistent use of lactulose and fiber, lifestyle modifications like exercise, low-fat diet and activity should be encouraged to prevent constipation.

Emergency endoscopic facilities should be made available nationwide in public sector hospitals for prompt control of gastrointestinal bleeding. Every effort should be made to control increasing incidence of hepatitis B and C through health education, prevention and immunization. Judicial use of sedatives and diuretics and proper advice regarding diet must be an integral part of all counselling protocol to cirrhotic patients. Hence, the early detection and diagnosis of these precipitating factors help in starting treatment of this fatal condition, hence reducing the mortality.

CONCLUSION

From this study, it was concluded that in most of the cases there are different factors which play a key role in precipitating hepatic encephalopathy which is a common phenomenon in patients with liver disease. Infections, upper GI bleed, constipation and electrolyte imbalance were the most common precipitating factors. There is definite need for health education in patients who are diagnosed with cirrhosis of liver regarding the risk of hepatic encephalopathy and its precipitating factors. Priority should be given to these factors in terms of hospital funds, blood banks, medicines and human efforts. No stone should be left unturned in providing better and effective infection control measures and better hygienic conditions in hospitals. The proper disposal of infected hospital waste and especially syringes, blades should be performed. Consistent use of lactulose and fibre, lifestyle modifications like exercise, low-fat diet and activity should be encouraged to prevent constipation. Emergency endoscopic facilities should be made available nationwide in public sector hospitals for prompt control of gastrointestinal bleeding.

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