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THE STUDY ON PRECIPITATING FACTORS OF HEPATIC ENCEPHALOPATHY IN CIRRHOTIC PATIENT IN WEST BENGAL

General Medicine

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ABSTRACT

Introduction: Hepatic encephalopathy is a frequent complication and one of the most debilitating manifestations of liver diseases, severely affecting the lives of patients.

Aims and objective:

i. To study the precipitating factors of hepatic encephalopathy.
ii. To observe the clinical manifestation at the time of presentation of hepatic encephalopathy.
iii. To observe the seasonal variation of hepatic encephalopathy in cirrhotic patient.

Materials and method: General Medicine, Murshidabad Medical college and R. G. Kar Medical college in West Bengal**.** All adult cirrhotic Patients admitted with encephalopathy in the medicine ward within the study period**.** One year starting from 1st Nov 2019 to 31st October 2020.

Conclusion: This study found that most common precipitating factors of hepatic encephalopathy in our patients are infection, constipation electrolyte imbalance and Variceal Haemorrhage bleeding. These are potentially preventable and reversible.

KEYWORDS

Hepatic Encephalopathy, Cirrhotic, Precipitating Factors

INTRODUCTION

Hepatic encephalopathy is a frequent complication and one of the most debilitating manifestations of liver diseases, severely affecting the lives of patients. The alteration of brain functioning, which can produce behavioral, cognitive and motor effects were termed porto systemic encephalopathy and later included in the term Hepatic Encephalopathy. Unless the underlying liver diseases is successfully treated, Hepatic Encephalopathy is associated with poor survival and high risk of recurrences [1].The incidence and prevalence of hepatic encephalopathy are related to the severity of the underlying liver insufficiency[2].In patients with cirrhosis, fully symptomatic overt hepatic encephalopathy is an event that defines the decompensated phase of the disease ,also variceal bleeding or ascites[3].The manifestation of hepatic encephalopathy may not be an obvious clinical finding and there are multiple tools used for its detection, which influences the variation in the reported incidence and prevalence rates. The prevalence of overt hepatic encephalopathy at the time of diagnosis of cirrhosis is 10%-14% in general, 16%-21% in those with decompensated cirrhosis, and 10% to 50% in patients with trans jugular intrahepatic portosystemic shunt. The cumulated numbers indicate that overt hepatic encephalopathy will occur in 30%-40% of those with cirrhosis at some time during their clinical course and in the survivors in most cases repeatedly .Minimal hepatic encephalopathy or covert hepatic encephalopathy occurs in 20%-80% of patients with cirrhosis .The risk for the first bout of overt hepatic encephalopathy is 5%-25% within 5 years after cirrhosis diagnosis, depending on the presence of risk factors ,such as other complications of cirrhosis and probably diabetes and hepatitis C .

Hepatic encephalopathy can occur in those with acute or chronic liver disease. Episodes can be triggered by infections, GI bleeding, constipation, electrolyte problems, or certain medications. The underlying mechanism is believed to involve the buildup of ammonia in the blood, a substance that is normally removed by the liver. The diagnosis is typically made after ruling out other potential causes. It may be supported by blood ammonia levels, an electroencephalogram, or a MRI brain.

In those with cirrhosis, the risk of developing hepatic encephalopathy is 20% per year, and at any time about 30–45% of people with cirrhosis exhibit evidence of overt encephalopathy. The prevalence of minimal hepatic encephalopathy detectable on formal neuropsychological testing is 60–80%; this increases the likelihood of developing overt encephalopathy in the future. Once hepatic encephalopathy has developed, the prognosis is determined largely by other markers of liver failure, such as the levels of albumin (a protein produced by the liver), the prothrombin time (a test of coagulation, which relies on proteins produced in the liver), the presence of ascites and the level of bilirubin (a breakdown product of hemoglobin which is conjugated and excreted by the liver). Together with the severity of encephalopathy, these markers have been incorporated into the Child-Pugh score; this score determines the one- and two-year survival and may assist in a decision to offer liver transplantation. [4]

i. To study the precipitating factors of hepatic encephalopathy.
ii. To observe the clinical manifestation at the time of presentation of hepatic encephalopathy.
iii. To observe the seasonal variation of hepatic encephalopathy in cirrhotic patient.

MATERIALS AND METHOD:

STUDY POPULATION:

All adult cirrhotic Patients admitted with encephalopathy in the medicine ward within the study period

STUDY PERIOD:

One year starting from 1st Nov 2019 to 31st October 2020.

SAMPLE SIZE:

All the patient admitted with hepatic encephalopathy, size was depend on the total admission within the study period in General Medicine, Murshidabad Medical college and R. G. Kar Medical college, West Bengal**.**

INCLUSION CRITERIA

1. Cirrhotic patient diagnosed to have hepatic encephalopathy.
2. Patients should be 12 years or older.
3. Patients should be willing to participate in the study and provide informed consent after due ethical clearance. In case of patients less than 18 years of age, informed consent is to be taken from the guardian.

EXCLUSION CRITERIA:

1. Coma or encephalopathy of other reason like hypoglycemia, keto acidosis, dyselectrolytemia, hypercarbia,stroke.
2. Alcohol intoxication, or drug overdose
3. Epilepsy
4. Patient not willing to be included in the study

STATISTICAL ANALYSIS:

For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS (version 25.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. Z-test (Standard Normal Deviate) was used to test the significant difference of proportions. p-value ≤ 0.05 was considered for statistically significant.

RESULT & DISCUSSION

We found that 30(50.0%) patients had 51-60 years of age which was more common and this was statistically significant (p= .00854). It was found that 12(20.0%) patients had female and 48(80.0%) patients had male. The value of p is < .00001.

Handady SO et al [5] (2015) found that Ninety one (n= 91, 75.8%) were male and twenty nine (29, 24.2 %) were females. The mean age was 50.23 years with STD ± 16.2.

Siva PK et al [6] (2009) found that including 48(96%), male and 02 (4%) females, presenting or complicating into hepatic encephalopathy were studied. Majority i.e. 43 (86%) patients were older than 40 years including 41 (82%) males and 02 (04%) females in this group. Seven (14%) patients were between 20 and 40 years old, all of them were males.

Rovira A et al [7] (2008) found that representing disturbances in cell-volume homeostasis secondary to brain hyperammonemia. All these MR imaging abnormalities, which return to normal with restoration of liver function, probably reflect the presence of mild diffuse brain edema, which seems to play an essential role in the pathogenesis of HE. It is likely that MR imaging will be increasingly used to evaluate the mechanisms involved in the pathogenesis of HE and to assess the effects of therapeutic measures focused on correcting brain edema in these patients.

We found that higher frequency 15(25.0%) patients had admitted on April and 21(35.0%)patients had admitted on May. This was not statistically significant.

We found that 20(33.33%) patients had anaemia (haemoglobin less than 7gm/dl) which was statistically significant (p=.00026)

It was found that 20(33.33%) patients had grade 3 varix and stool OBT positive which was statistically significant (p=.00026)

It was found that 9(15.00%) patients had Hypokalemia K (less than 3 in HE patients) this was significantly lower (p<0.0001).

We found that 16(26.66%) patients had Hyponatremia Na less than 135 this was significantly lower (p<0.0001). 5(8.33%) patients had diarrhea this was significantly lower (p<0.0001).

We found that 12(20.00%) patients had constipation. The value of z is 6.5727. The value of p is < .00001. The result is significant at p < .05.

Gad YZ et al [8] (2012) found that the precipitating factors of HE in our studied cases were upper GI bleeding in 87 patients (36.7%), advanced HCC in 43 patients (18.14%), infections in 37 patients (15.61%), excess protein intake in 27 patients (11.39%), electrolyte imbalance in 7 patients (2.95%), and altered bowel habits in 33 patients (13.92%). Males were commonly affected by HCC ($P < 0.05$) and upper GI bleeding more than female patients ($P < 0.05$). Male patients were commonly presented with HE (63.29%), and this gender difference reached a statistical significance among those presented with upper GI bleeding ($P < 0.05$) and HCC ($P < 0.05$). Upper GI bleeding, advanced HCC, and infections were the leading causes of death with a higher prevalence in grade IV comatose ($P < 0.05$), child C ($P < 0.05$), male patients ($P < 0.05$).

Coggins CC et al [9] (2013) found that promote comprehensive assessment, differential evaluation and provision of care which optimizes benefit while minimizing burden. Delirium is a debilitating neuropsychiatric complication that is highly prevalent in palliative care.

Tariq M et al [10] (2009) found that 29% upper gastrointestinal bleed (GIB), 8% spontaneous bacterial peritonitis (SBP), 19.5% urinary tract infection (UTI), 2.5% lower respiratory tract infection and 3% had diarrhea. There was hyponatremia in 1.5% and hypokalemia in 4.5% patients.

We found that 43(71.67%) patients had infection/ Leucocytosis (more than 10000 total count) and increase CRP (more than 6) in HE patients this was significantly lower (p<0.0001).

It was found that 39(65%) patients had incidence of Spontaneous Bacterial Peritonitis in HE Patients (Ascitic fluid cell count more than 250 and Cell Type Neutrophil) this was significantly lower (p<0.0001).

It was found that 6(10%) patients had Hbs Antigen Positive, 6(10%) patients had Anti HCV positive and 48(80%) patients had Non Reactive this was significantly lower (p<0.0001).

We also found that patients who have grade IV encephalopathy are in deep coma stage . They do not have Flapping Tremor. Their EEG are abnormal deep flat wave seen. Grade III encephalopathy patient have flapping treamer or asterixis. They have triphasic wave in EEG. It also shows that patient having minimal,grade I encephalopathy take more time to do stroop test than normal patient. They also take more time to do number connection test. GRADE II encephalopathy patient have flapping treamer or asterixis.

Alam I et al [11] (2005) found that Fifty patients (32 males and 18 females) were enrolled. 47 patients had hepatitis B, C or both positive. 64% were in the age group of 45-60 years and 76% were having either grade III or IV coma. Thirty three (66%) patients had asterixis which was found to be a sensitive index for the diagnosis of HE in patients who were not in coma. Jaundice and Ascites were other common presenting features. Electrolytes imbalance in 28(56%) patients, diarrhea in 20(40%), constipation in 16(32%), infections in 12(24%) and gastrointestinal bleed in 11(22%) patients were amongst the commonest precipitating factors. None gave the history of alcoholism or recent surgery. Occurrence of precipitating factors for HE in patients with CL is a common phenomenon and all such patients must be hospitalized to ascertain and manage such factors.

Table: Distribution of all parameters

			Number	Percentage (%)
Incidence of Anaemiahaemoglobin less than 7gm/dl in HE Patients		Present	20	33.33
		Absent	40	66.67
		Total	**60**	**100**
Incidence of Variceal Haemorrhage in HE Patients	**Grade 3 Varix and Stool Obt positive**	Present	20	33.33
		Absent	40	66.67
		Total	**60**	**100**
Incidence of Hypokalemia K less than 3 in HE Patients		Present	9	15.00
		Absent	51	85.00
		Total	**60**	**100**
Incidence of Hyponatremia Naless than 135 in HE Patients		Present	16	26.66
		Absent	44	73.34
		Total	**60**	**100**
Incidence of Diarrhoea in HE Patients		Present	5	8.33
		Absent	55	91.67
		Total	**60**	**100**
Incidence of Constipation in HE Patients		Present	12	20.00
		Absent	48	80.00
		Total	**60**	**100**
Incidence of Infection/Leucocytosis (more than 10000 total count) and increase CRP (more than 6) in HE Patients		Present	43	71.67
		Absent	17	28.33
		Total	**60**	**100**
Incidence of Spontaneous Bacterial Peritonitis in HE Patients (Ascitic fluid cell count more than 250 and Cell Type Neutrophil)		Present	39	65
		Absent	21	35
		Total	**60**	**100**
Incidence of Hepatitis B and Hepatitis C in HE Patients		Hbs Antigen Positive	6	10
		Anti HCV positive	6	10
		Non Reactive	48	80
		Total	**60**	**100**

CONCLUSION

This study found that most common precipitating factors of hepatic encephalopathy in our patients are infection, constipation electrolyte imbalance and Variceal Haemorrhage bleeding. These are potentially preventable and reversible.

We also found that incidence of Spontaneous Bacterial Peritonitis in HE Patients was significantly higher.

Patients at risk of malnutrition are relatively difficult to identify since liver disease may interfere with biomarkers of malnutrition.

We also found that patients who have grade IV encephalopathy are in deep coma stage which was abnormal EEG. For the diagnosis of minimal and grade I encephalopathy stroop test and number connection test is essential.

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