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ORIGINAL ARTICLE

Frequency of Hepatic Encephalopathy (HE) among Hepatitis Patients at Mardan Medical Complex, Mardan

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ABSTRACT

Introduction: Hepatic encephalopathy (HE) represents severe neuropsychiatric condition of acute and chronic liver disease. Approximately 70% HE found in patients already suffering from liver cirrhosis. The aim of this study was to find the risk factors for developing Hepatic Encephalopathy in patients suffering from Chronic liver disease.

Materials and Methods: This descriptive study was conducted in Mardan medical complex in four months duration. Data was collected from one hundred patients using non-probability sampling technique. All the data were analyzed through statistical software, JMP, 10.0 SAS, USA.

Results: This study reports the frequency of HE among 100 hepatitis patients with mean age 55±9years. Out of these patients, 58% were males and 42% females. In our study, infection was the main precipitating factor found in most of the patients examined, followed by spontaneous bacterial peritonitis (20% patients), constipation (18% patients), GI bleeding (13% patients), and electrolyte imbalance such as hypokelmia (6% patients) and hypernatremia (3% patients).

Conclusion: It is concluded that HE is more common in male patients than female. The risk of HE can be minimized by providing good hygienic conditions and germs free food in hospital.

Keywords: Hepatic encephalopathy, Hepatitis, Cirrhosis, Chronic liver diseases

INTRODUCTION

Hepatic encephalopathy (HE) is a common liver disease in children caused by metabolic disturbance. In children the physiology of hepatic encephalopathy (HE) is very different than adults. In adult, the HE usually leads to chronic liver disease and cirrhosis, and acute liver failure in case of children[1]. The survival rate in patients suffering from acute encephalopathy is lower (70%) than chronic encephalopathy (100%). The mildest form of HE was described for first time by Zeegan et al in 1970[2]. In HE increase was observed in the level or concentration of serum potassium, billirubin, alkaline phosphatase, and blood nitrogen in male patients and decrease in the serum albumin and prothrombin activity[3]. HE was classified into four categories at the 11th World Congress of Gastroenterology, Vienna, by Working Party They proposed a multiaxial definition of HE that defined the type of hepatic abnormality, the duration and characteristics of neurological manifestations in chronic liver disease. Type A Encephalopathy is linked with severe liver malfunction, Type B with no intrinsic hepatocellular and portosystemic bypass, Type C with cirrhosis and Type D with disorders of the urea cycle[4]. Type-D syndromes are rare hereditary disorders of urea biosynthesis caused by genetic defects in the urea cycle for which six enzymes participate in urea formation[3]. For HE diagnosis, at least two neurophysiologic tests were recommended by working party. Number connection test (NCT-A), (NCT-B) block design test (BDT) and the digit symbol test[5]. Factors such as Ammonia, Manganese, glutamine, certain amino acid and inflammatory mediators can directly or in directly affect the brain function in hepatic encephalopathy[6].

Non absorbed disaccharides (ex-lactulose, lactitol) and antibiotics (ex-neomycin, rifaximin) represent the foundation of treatment[7]. Some factors can lead to increase in number of cirrhotic patients and also in mortality rate. These factors include less education, dearth, insufficient nutrition, lack of clean environment and counselling[8].

Patients with Hepatic encephalopathy may lead to turn down in the level of consciousness often characterized as comma. At this stage, the precipitating factor is usually recognized. The treatment can be continued till disappearance of precipitating factor. The treatment of the precipitating factor diminishes the encephalopathy[9]. These precipitating events should be identified quickly, patients' attendants counselled properly and provided with quick medical care. Our study was aimed to evaluate the precipitant factors of hepatic encephalopathy released from patients admitted in hospitals.

PATIENTS AND METHODS

A hospital base descriptive study was carried out on 100 patients in medical unit of Mardan medical complex Mardan from March 2015 to June 2015. We included the manifesting signs and symptom of hepatic encephalopathy from patients aged 13 or above. They came to hospital for treatment and were admitted. All cases of unconscious patients from our study were excluded that had renal failure due to any cause comprise hepatorenal disorder, hypertension, diabetes, hypotension, sedative overdose, electrolyte disturbances, cerebrovascular accidents including haemorrhage and infarctions. Permission from hospital ethical committee was taken before start of study. All subjects for hepatitis, viral antibodies, anti-HV (hepatitis antibody) in current study were enrolled in OPD.

Tests conducted for each of patients include full blood count, renal function tests, random blood sugar, coagulation profile, serum albumin, liver function tests and serum electrolyte. Through abdominal ultrasound, we looked for liver and ascites, parenchymal echogenicity, portal vein diameter, splenic size. For spontaneous bacterial peritonitis ascites, an ascetic tap was also done. Indication of the existence of other concurrent problem of cirrhosis liver developing hepatic encephalopathy was too be evidence and Child's achieve was assess for each patient based on parameters.

Based on experimental work and relevant investigations, we diagnosed the patients having hepatic encephalopathy. Experimental facial appearance integrated palmar erythema, spider naevi, gynaecomastia, jaundice, caput medusa, fetor hepaticas, reduced ascites , splenomegaly, and unconsciousness, liver span. The study incorporated hepatitis A, B, C and D class, serum albumin, CT brain, blood sugar levels, blood urea and creatinine, complete blood count, liver function tests, serum globulin, ascetic fluid examination, serum electrolytes, ultrasound tummy for liver and spleen, endoscopy for variceal bleed. During data collection, check list and structured Performa were used. We recorded a comprehensive experimental account of the patients about the present and past illnesses. All the laboratory investigations were done from hospital laboratory under

supervision of well experienced and skilled microbiologists and technicians.

In a pre-designed Proforma, important information including name, age, gender and address was recorded. Strictly exclusion criteria were followed to control any biasness in the study results. Encephalopathy was graded according to the experimental condition (West Haven scale) as specified. We can record all the information of the patients who have been admitted in hospitals.

RESULTS

A total of 100 patients of cirrhosis with HE were studied, out of these patients, 58 were males and remaining females. Mean age was 55 ± 9 years, laboratory parameters are shown in Table I. All the patients belong to Mardan and their local areas. Different parameters recorded during this study (Table-1) helped us in diagnosing the patients of hepatic encephalopathy.

Table 1: Laboratory parameters of patients suffering from Hepatic Encephalopathy (HE).

S. No.	Variables	Mean ± SD	Normal Range
1	Blood Urea (mg/dl)	29.6±2.6	5-20
2	Haemoglobin (g/dl)	10.8±1.3	13.7-17.2
3	WBC count (x10^9/l)	9.4±2.8	3.8 - 10.8 x 10^9
4	Platelet count (x10^9/l)	101.3±28.5	150 - 450 x 10^9
5	Total billirubin (mg/dl)	4.2±2.3	< 1.3 (0.1 -1.3)
6	Albumin (g/l)	2.4±0.4	3.4-4.8
7	Prothrombin time (seconds)	21.8±4.3	9 - 12.5
8	Sodium (mEq/l)	130.3±4.8	135-145
9	Potassium (mEq/l)	4.3±0.4	3.5-5.0
10	Creatinine (mg/dl)	1.9±0.6	0.7-1.4

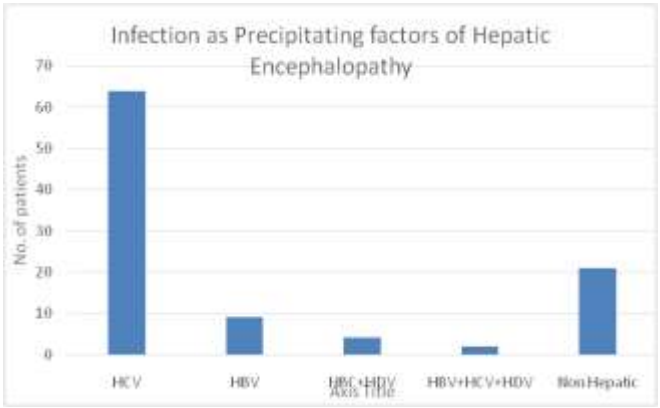

Fig-1: Indicates 64 patients with cirrhosis due to hepatitis C infection, 9 patients with hepatitis B,4 patients with Hepatitis B and D related cirrhosis ,2 patients with hepatitis B, C,D infections.

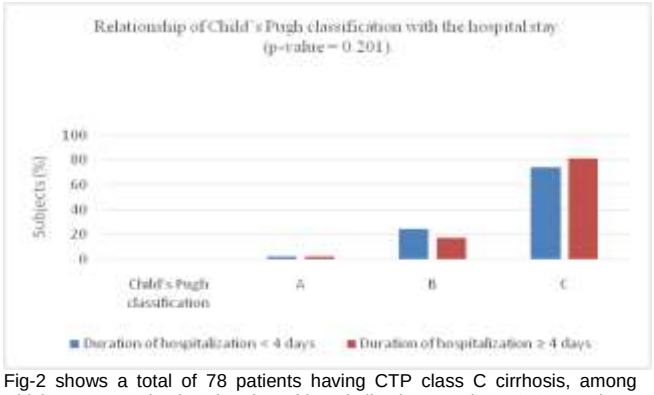

Fig-2 shows a total of 78 patients having CTP class C cirrhosis, among which 74% were having duration of hospitalization < 4 days, 81% ≥ 4 days, similarly patients with CTP class B cirrhosis, here 24% having duration of hospitalization < 4 days and 17% had hospitalization ≥ 4 days2 patients with CTP class A cirrhosis.

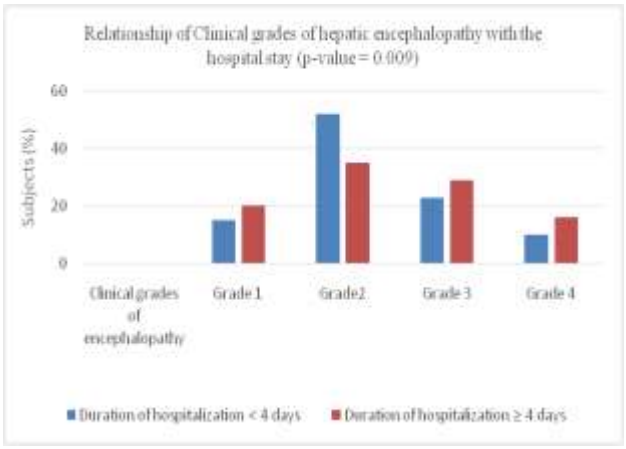

Fig-3 Indicates that 18 patients were in grade 1 hepatic encephalopathy (15% having duration of hospitalization < 4 days, while 20 % having duration of hospitalization ≥ 4 days), while 43 had grade 2 (52% of hospitalization < 4 days while 35% of hospitalization ≥ 4 days) , 26 grade 3 (23% of hospitalization < 4 days while 29% of hospitalization ≥ 4 days) and 13 patients had grade 4 HE (10% of hospitalization < 4 days while 16% of hospitalization ≥ 4 days) on presentation based on the West Haven criteria and NCT-A.

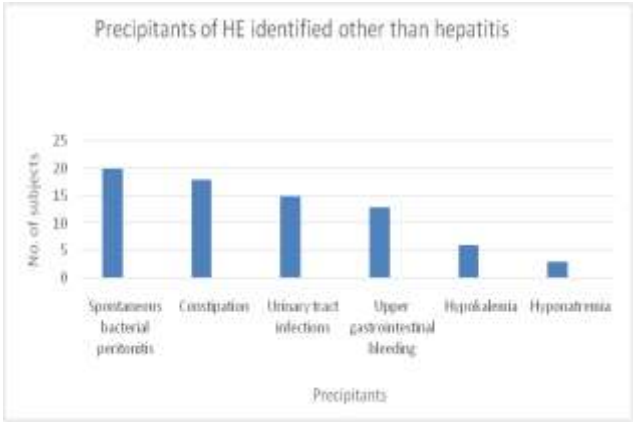

Fig- 4: shows that spontaneous bacterial peritonitis (SBP) is the most common precipitant seen in 20 patients. Constipation seen in 18, urinary tract infections in 15 and upper gastrointestinal bleeding in 13 patients Electrolyte abnormality including hypokalemiain 6 while hyponatremia in 3 patients.

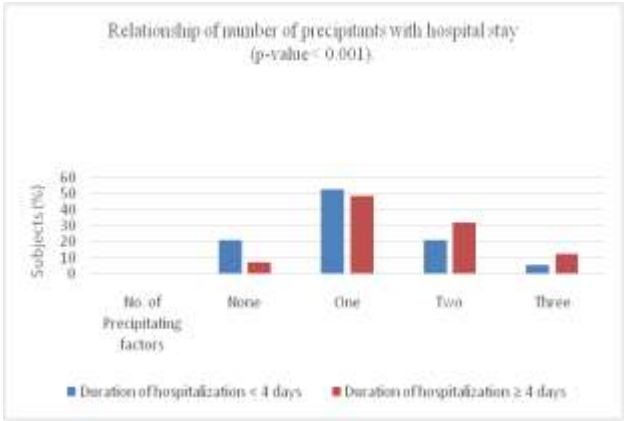

Fig- 5 shows that 51 patients had one identifiable precipitant, 27 patients had 2, 8 patients had three precipitating factors at the time of presentation. Mean hospital stay was 4 ± 3 days; the duration of hospital stay of 50 patients was < 4 days, of which 53% were found out to have only one precipitating factor. 50 patients had a mean stay of > 4 days.

DISCUSSION

Hepatic encephalopathy is a fatal disease of liver characterized by potentially reversible impairment of brain function and have unsolved problem for physician and researcher all over the world since at the time of Hippocrates. There is no diagnosis mechanism of hepatic encephalopathy. For modern research, it has key steps to remove precipitating factors in whole administration[10]. The present study has been carried out on 100 patients having mean ages of 55+9 years. Out of 100 patients, 58 were males and 42 females. The increasing number of hepatitis-C Virus developed a pandemic and vital cause of liver cirrhosis in Pakistan.

Feasible clarification of the study the majority of patients in our study were at the end stage of cirrhosis and hepatitis C, the most common form in these patients. In our study, HCV antibodies are found in 64% patients is comparable with 64% and 61.05% observed[11]. Our results are in contrast to western studies where alcohol is the main precipitating factor being an industrial nation[12]. In our study, the main precipitating factors were spontaneous bacterial peritonitis (SBP), constipation, and GI bleeding. Frequency of different precipitating factors reported nationally and internationally have been given in Table no 4 and compared with one which has been reported during this study.

Table 2: Show comparison of precipitating factors of hepatic encephalopathy reported in various studies.

Precipitating factor of HE.	Mehboob *et al.,*	Alam *et al.,*	Tariq *et al.,*	Ahmed *et al.,*	Sheikh *et al.,*	Present study
Infections(SBP, UTI)	47	24	30	28	15	35
Constipation	19	32	29	52	52	18
GI Bleeding	30	22	30	56	56	13
Hypokelmia	-	18	4.5	68	70	6
Hyponatremia	-	36	1.5	28	28	3
Diarrhea	5	40	3	22	12	-

Mehboob, Tariq, Alam and our study shows that infection (SBP, UTI), constipation, and GI Bleeding were the main precipitating factor of hepatic encephalopathy. In this study, constipation is second major factor which can easily be treated by taking well balanced fiber food, excess water, taking exercise and avoids caffeine. GI bleeding is found in 13% patients in our study this is in contrast 27% patients observed by Ashoor *et al.,* 2012[13]. Low grade cerebral edema is an important factor in pathogenesis of HE in cirrhotic patients produced due to hypokelmia[14]. Our results are in contrast to that of Ahmad and Sheikh Results where hypokelmia and hypernatremia were the main causes of hepatic encephalopathy. Some of the foreign studies show that infection was not the cause of hepatic encephalopathy there because of the presence of best sterile/hygienic conditions in hospital and health care center[15].

CONCLUSIONS

Frequencies of different precipitating factors were investigated to get an idea which factor is responsible for hepatic encephalopathy in District Mardan. Once we know the main precipitating factor, then it is easy to devise a strategy to control it to large extent. The most common precipitating factors in our study are infection, constipations, GI bleeding. If we can look at the foreign studies there is no infection found responsible for hepatic encephalopathy because of their well hygienic and sterile conditions. We should provide good sterile and hygienic conditions to patients in hospitals to minimize the risk of hepatic encephalopathy.

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