ORIGINAL ARTICLE

Upper Gastrointestinal Bleed, a Grave yet Precipitating Factor of Hepatic Encephalopathy in Cirrhotic patients

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ABSTRACT

Aim: To determine the frequency of development of hepatic encephalopathy in cirrhotic patients, presented with upper gastrointestinal bleeding in medical emergency of tertiary care hospital.
Study design: Descriptive case series
Place and duration: Medical emergency of Services Hospital Lahore, Pakistan. Study was carried out over a period of six months from April 2013 to October 2013
Methods: A total of 300 cases were included in this study. Patient presenting with upper GI bleeding having cirrhotic liver were evaluated and followed up for occurrences of hepatic encephalopathy from the date of admission to 5th post admission day using West Haven criteria. Gastroscopy was done in all patients for diagnostic and therapeutic purposes.
Results: Regarding age distribution of patients, 42 patients (14%) were 18-40 years old, 185 patients (61.7%) were 41-60 years of age, 69 patients (24.3%) were between 61-90 years of age, while 4 patients (1.3 %) were between 81-100. Mean age of the patients was observed 53.1 ± 12.43
Out of 300 patients, 157 patients (52.33%) were male and remaining 143 patients (47.66) were female. .Hepatic encephalopathy in patients of upper gastrointestinal bleed was noted in patients (47%)
Conclusion: It is concluded that upper gastrointestinal bleeding is leading precipitating factor of hepatic encephalopathy.
Keywords: Cirrhosis, upper gastrointestinal bleeding, hepatic encephalopathy

INTRODUCTION

Liver cirrhosis is the twelfth leading cause of death in United States of America. It is an important cause of morbidity and mortality worldwide, one of the most frequent cause of hospitalization in Pakistan as well and costing a major burden on health system because of its grave complication i.e. upper gastrointestinal bleed, hepatic encephalopathy (HE) and infection[1].

Patients with advanced cirrhosis frequently develop hepatic encephalopathy, a disorder of neurological function comprising of a wide spectrum of clinical signs and symptoms ranging from minimal changes in neuropsychological function to profound coma. [2]Ammonia has a key importance in the pathogenesis of hepatic encephalopathy. Ammonia causes neurotransmitter abnormalities and induces injury to astrocytes partially mediated by oxidative stress. These disturbances lead to astrocyte swelling and brain edema, which appear to be involved in the pathogenesis of neurological manifestations. In addition, proinflammatory cytokines may have a major contributory role in impairing several brain functions.

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The leading factors that precipitate hepatic encephalopathy in patients with liver cirrhosis are infection, constipation and upper gastrointestinal bleeding. The frequency of infection as common precipitating factor appears to be declining and of gastrointestinal bleeding appears to be increasing[3]. The magnitude of gastrointestinal bleeding was 45 % as precipitating factor of hepatic encephalopathy[4].

Upper gastrointestinal bleeding presents with hematemesis, melena or hematochezia. Acute upper gastrointestinal bleed especially due to gastroesophageal varices in patients of liver cirrhosis is associated with high morbidity and frequent development of life-threatening complications.

Bleeding into intestinal tract may significantly increase the amount of protein in the bowel, resulting in increased ammonia production and precipitate rapid development of encephalopathy[5].

Management focuses evaluation of magnitude of hemorrhage and resuscitation of the patient by maintenance of intravenous line, administration of isotonic solutions and cross-matched blood. The magnitude of initial bleed increases likelihood of ongoing or recurrent bleed. Bleeding may be controlled by intravenous infusions of somatostatin analogs and/or vasopressor agents and proton pump inhibitors and use of upper gastrointestinal endoscopy to take definite measures like band ligation of varices and sclerotherapy. Untreated

patients surviving a variceal hemorrhage have a 1- to 2-year risk of rebleeding of about 60% and a risk of death of about 40% to 50%.

Treatment of hepatic encephalopathy includes assessment of the grade of hepatic encephalopathy, providing adequate nutrition, decreasing protein load, purging the blood by the use of lactulose, use of antibiotics, and branched chain amino acids, and an intensive care in case of coma or heavy gastrointestinal bleeding[8].

Although number of studies have been done in this context, but a comprehensive study focusing on upper gastrointestinal bleeding as the precipitating factor is still lacking in our part of the world and I am expecting higher magnitude of gastrointestinal bleeding as the precipitating factor. This deficiency and unavailability of emergency endoscopic facilities has motivated us to select the topic for this study.

MATERIAL AND METHODS

The study was carried out in medical emergency of Services Hospital Lahore over a period of six months from 01-04-2013 to 31-07-2013. Patients of either sex above the age of 18 years, known to have cirrhosis presenting with upper gastrointestinal bleeding were included in the study. Exclusion criteria included patients having other risk factors for hepatic encephalopathy such as infection (as presence of temperature of > 99 F and raised leukocyte count), hypoglycemia, electrolyte imbalance as high or low level of serum sodium or serum potassium, constipation (defined as failure to pass stool in 48 hours), dehydration (presence of dry tongue and loss of skin turgor) and use of sedative drugs. Secondly, patients suffering from other diseases causing encephalopathy such as metabolic disorders, infectious diseases, intracranial vascular events and intracranial space occupying lesions were excluded from the study.

Three hundred cases of liver cirrhosis presenting with hematemesis and/or melena presented in medical emergency of Services Hospital that fulfilled the inclusion criteria were enrolled in the study after taking informed consent. A detailed clinical history of the patient was taken regarding the present and past illnesses. Occurrence of hepatic encephalopathy was assessed by West Heaven scale on daily basis till 5th post admission day.

The routine and relevant investigations carried out were full blood count, urine examination, blood urea and creatinine, blood glucose, ches radiograph, serum electrolytes, serum albumin, coagulation profile, ultrasound of abdomen, liver function test (LFT), HBsAg and Anti-HCV.

RESULTS

Regarding age distribution of patients, 42 patients (14%) were 18-40 years old, 185 patients (61.7%) were 41-60 years of age, 69 patients (24.3%) were between 61-90 years of age, while 4 patients (1.3 %) were between 81-100. Mean age of the patients was observed 53.1± 2.43 (Table 4). Out of 300 patients, 157 patients (52.33%) were male and remaining 143 patients (47.66) were female (Table 5). Hepatic encephalopathy in patients of upper gastrointestinal bleed was noted in patients (47%) (Table 6).

Table 1: Distribution of cases by Age

Age (Year)	n	%age
18-40	42	4
41-60	185	61.7
61-80	69	24.3
81-100	4	1.3
Mean±SD	53.1±12.43	

Table 2: Distribution of cases by gender

Gender	n	%age
Male	157	52.3
Female	143	47.7
Total	110	100

Table 3: Helicobacter pylori

Helicobacter Pylori	n	%age
Yes	141	47 %
No	159	53 %
Total	300	100.0

DISCUSSION

Hepatic Encephalopathy is a life threatening complication of liver cirrhosis. According to our study, majority of patients were from periphery of Lahore, Sheikhupura, Gujranwala, Wazerabad Sargodha, Pak Pattan, Okara, Kasur etc, and this may be because of poor hygiene, lack of awareness, motivational deficiency and improper counseling. We also found that majority of the patients were middle aged people with male preponderance. This observation is quite comparable with other studies done in other parts of Pakistan[9,10].

In our study, the majority of patients was HCV positive and in our country this is a common cause of liver cirrhosis and this observation is comparable to other studies done in other parts of Pakistan[9].

On the other hand, in the western world alcoholism is the main cause of liver cirrhosis.

The leading factors that precipitate hepatic encephalopathy in patients with liver cirrhosis are infection, constipation and upper gastrointestinal bleeding. The frequency of infection as common

precipitating factor appears to be declining and of gastrointestinal bleeding appears to be increasing[3].

The gastrointestinal bleeding (GIB) was a leading precipitating factor of hepatic encephalopathy in our study. In one study conducted in Combined Military Okara, Pakistan on determination of factors precipitating encephalopathy in patients with liver cirrhosis also had gastrointestinal bleeding (GIB) the most common precipitating factor 37.2%. In another study conducted in Aga Khan University Hospital, from January 2005 to December 2007 also had gastrointestinal bleeding (GIB) the most common precipitating factor 34%[11]. In another study conducted on precipitating factors of hepatic encephalopathy at a tertiary care hospital Jamshoro, Hyderabad had gastrointestinal bleeding (GIB) as precipitating factor 45%[4].

Upper gastrointestinal bleeding presents with hematemesis, melena or hematochezia. Acute upper gastrointestinal bleed especially due to gastroesophageal varices in patients of liver cirrhosis is associated with high morbidity and frequent development of life-threatening complications.

Bleeding into intestinal tract may significantly increase the amount of protein in the bowel, resulting in increased ammonia production and precipitate rapid development of encephalopathy[5].

Ammonia causes neurotransmitter abnormalities and induces injury to astrocytes partially mediated by oxidative stress. These disturbances lead to astrocyte swelling and brain edema, which appear to be involved in the pathogenesis of neurological manifestations. In addition, proinflammatory cytokines may have a major contributory role in impairing several brain functions.

Hepatic encephalopathy is a diagnosis of exclusion hence [12], such a tool was used in our study to detect hepatic encephalopathy and this correlates with the study of Alam et al,[9] who studied the precipitating factors of hepatic encephalopathy without considering the serum ammonia levels as a diagnostic tool[26]. Evidence exists that serum ammonia has low sensitivity in hepatic encephalopathy and is not always raised in HE therefore, it is not a good screening tool.[13] Specific precipitating factors have specific treatment and with proper treatment and care hepatic encephalopathy is frequently reversible. In fact, complete recovery is possible, especially if the encephalopathy was triggered by a reversible cause. However, people with chronic liver disorders are susceptible to future episodes of encephalopathy. Therefore, our goal should be to identify and manage the particular precipitating factor and effective measures and steps must be taken especially in remote areas where there is lack of medical facilities. Therefore, in this context we can prolong the life expectancy and improve the quality of life in patients with hepatic encephalopathy.

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