MEDICAL CHANNEL

Original Article

SPONTANEOUS BACTERIAL PERITONITIS A LEADING PRECIPITATING CAUSE OF HEPATIC ENCEPHALOPATHY

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ABSTRACT:

Background: Spontaneous bacterial peritonitis (SBP) is defined as an ascitic fluid infection without an evident intra-abdominal surgically treatable source.Bacterial translocation is the presumed mechanism for the development of SBP, with gut flora traversing the intestine into mesenteric lymph nodes and ascites.
Objective: To evaluate the role of Spontaneous Bacterial Peritonitis and other associated factors as precipitating Cause of Hepatic Encephalopathy
Materials & Methods:A cross-sectional study at Department of Medicine,Isra University Hospital, Hyderabad, between March 2008 and December 2010 with hepatic encephalopathy was carried out. All patients with liver cirrhosis and portal hypertension presenting with hepatic encephalopathy were included.Patients were included in study with non-probability convenient sampling technique. Informed consent was taken before inclusion of patients in the study. Statically analysis and frequencies were calculated with MS Excel 2010.
Results: Different precipitants of HE were identified, the most common precipitant identified was SBP which was seen in 180 patients, constipation in 70 patients, upper GI bleeding in 50 patients, electrolyte abnormalities seen in 33 patients, benzodiazepines seen in 5 patients.
Conclusion:
SBP is found to be the most common precipitant of hepatic encephalopathy. Additional precipitating factors identified were constipation and GI bleeding.

Keywords: Spontaneous bacterial peritonitis, Hepatic Encephalopathy, constipation and GI bleeding

INTRODUCITON

Spontaneous bacterial peritonitis (SBP) is defined as an ascitic fluid infection without an obviousintraabdominal surgicallycurablecause.[1] The presence of infection is documented by a positive ascitic fluid bacterial culture and an elevated ascitic fluid absolute polymorphonuclear leukocyte (PMN) count (e”250 cells/mm3).SBP is common and severe complication of ascites ,severe enough for hospitalization, SBP can occur in up to 30% of individuals and can have a 25% in hospital mortality rate. Bacterial translocation is the presumed mechanism for the development of SBP, with gut flora traversing the intestine into mesenteric lymph nodes and ascites.
The most common organisms are Escherichia coli and streptococcus viridants, enterococcus. Patients with ascites may present with fever, altered mental status, abdominal pain. Or they may present without any symptoms.The vast majority of patients with SBP have advanced cirrhosis.[1,2] Other risk factors (most of which are associated with cirrhosis) include Ascitic fluid total protein concentration less than 1 g/dL (<10 g/L) , Prior

episode of SBP, Serum total bilirubin concentration above 2.5 mg/dL , Variceal hemorrhage , malnutrition,Use of proton pump inhibitors .HE is present in 60-70% of pts with cirrhosis.Precipitating factors can be identified, and the early prompt treatment of the precipitating cause can actually reduce the overall morbidity and mortality .Once the precipitating condition has been resolved, the encephalopathy also subsides usually. In patients with vriceal hemorrhage, the frequency of SBP is significantly increased, and prophylaxis against SPB is recommended.[3,4] HE secondary to a precipitating event are usually completely reversible following correction of the cause, and these patients have a good prognosis.

METHODOLOGY

A hospital based cross-sectional study of the patients presenting to the Isra University Hospital,Hyderabad, between March 2008 and December 2010with hepatic encephalopathy was carried out. All patients with liver cirrhosis and portal hypertension presenting with hepatic encephalopathy were included. The diagnosis of acute hepatic encephalopathy being made on the basis of history and physical examination. Different grades of HE were identified. Stage I: mental status change; Stage II: lethargy, confusion Stage III: stupor, but arousable;Stage IV: coma . Patients with acute fulminant failure, uremia , cerebral anoxic encephalopathy , are excluded. Liver cirrhosis was diagnosed on the basis of clinical , biochemical and ultrasonological examination . Data related to present and past medical history at the time of admission was collected from the medical records. The clinical findings on examination including presence of jaundice , pallor , fever , asterixis , and ascites were recorded. Demographics and laboratory parameters were collected including bilirubin, PT,Albumin/S. Ascitic tap was performed in all patients with ascites and sent for detail report in order to diagnose SBP.

All patients had management during their hospital stay which included lactulose and metronidazole, along with zinc and protein restriction. iv antibiotic , third generation cephalosporin was given in proper dose who were diagnosed having SBP on ascitic DR report.patients with upper GI bleed were given FFPS and Their EVBL was done.

Statistical analysis was performed using SPSS, precipitating factors were identified. All p values were considered as statically significant if <0.05.

RESULTS

A total of 356patients of cirrhosis with HE were studied of whom 260 were males Table I, mean age was 50 other demographic and laboratory parameters are shown, 280 patients with cirrhosis due to hepatitis c infection , rest with hepatitis B and D and few with non B , non C cirrhosis. 280 patients had CTP class c while 40 were CTP class B AND A cirrhosis respectively. 180 patients were in grade 2 HE, 60 patients in grade 3, 110 patients in grade 1-2.

Different precipitants of HE wereidentified, the most common precipitant identified was SBP which was seen in 180 patients, constipation in 70 patients, upper GI bleeding in 50 patients, electrolyte abnormalities seen in 33 patients, benzodiazepines seen in 5 patients, Table II.

DISCUSSION

Patients with cirrhosis are susceptible to a variety of complications and their life expectancy can be markedly reduced.[2,4]Once a patient develops complications of cirrhosis, they are considered to have decompensated disease.[5] The high morbidity and mortality of cirrhosis is secondary to these devastating complications. The quality of life and survival of patients with cirrhosis can be improved by the prevention and treatment of these complications. SBP is considered the important precipitating cause of HE ,Should be considered in patients with ascites who develop abdominal

TABLE I
GENDER DISTRIBUTION

Gender	Number	Percentage (%)
Males	260	73.03
Females	96	26.96
Total	356	100

TABLE II
PRECIPITANTS OF HE

	Number	Percentage (%)
SBP	180	50.56
Constipation	70	19.66
Upper GI bleeding	50	10.04
Electrolyte Abnormalities	33	9.26
Benzodiazepines	5	1.40

pain and fever and mental status changes.[6]Prevalence of SBP in cirrhotic on hospital admission is 10-30%.[7]Most episodes occur due to translocation of gut bacteria to mesenteric lymph nodes and ascites. So precipitating factors are recognized early including constipating, and GI bleed electrolyte abnormalities. A diagnostic paracentesis should be done on admission to hospital , especially in patients with hepatic encephalopathy.[8,9]

Patients who presented to us were most of the time were malnourished not only because of their disease but also because of taboos regarding their diet. And also most of them belonged to child score of more than 10,ie class c . electrolytes imbalances, increased dietary protein, constipation and gastrointestinal hemorrhage were also important and correctable precipitating causes of HE.[10-13] In patients with fever, abdominal pain or tenderness, or altered mental status, treatment can be started as soon as ascitic fluid, blood, and urine have been obtained for culture and analysis. In patients without these findings, it is reasonable to wait until the results of the PMN count are available.[14,15] Collection and processing of the specimen should take no more than one to four hours from the time of the paracentesis. Patients with ascitic fluid PMN counts e”250 cells/ mm3 in a clinical setting compatible with ascitic fluid infection should receive empiric antibiotic therapy.

It is therefore crucial that precipitating factors are identified earlier, especially in patients with severe liver diseases, and appropriate treatment initiated soon, with an aim to treat and manage them, and subsequently ensuring better outcomes. It is also important to educate the patients and their families about these precipitants to ensure earlier presentation, diagnosis and management ofHE.

CONCLUSION

SBP is a major and common precipitant of hepatic encephalopathy, constipation and GI bleeding was also identified as the important precipitants. Once the precipitating condition is resolved the encephalopathy also typically disappears. With the patient recovering to his or her previous state. Delay in identifying and treating the SBP leads to worse outcome.

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