ORIGINAL ARTICLE

Frequency of Helicobacter Pylori Infection in patients with Minimal Hepatic Encephalopathy

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ABSTRACT

Aim: To determine the frequency of Helicobacter Pylori infection in patients of cirrhosis with minimal hepatic encephalopathy.
Study design: Cross-sectional observational survey.
Place and duration of study: Department of Gastroenterology, Medical Unit-III, Services Hospital, Lahore from Jan 2013 to July 2013.
Methods: A total of 110 cases were included in this study. Bispectral Index Score of the patients was calculated. Endoscopic biopsies from gastric antrum were taken and sent for histopathology to confirm Helicobacter Pylori.
Results: Regarding age distribution of patients, 17 patients (15.5%) were 18-40 years old, 67 patients (60.9%) were 41-60 years of age while 26 patients (23.6%) were between 61-70 years of age. Mean age of the patients was observed 52.14±9.61. Out of 110 patients, 63 patients (57.3%) were male and remaining 47 patients (42.7%) were female. Helicobacter pylori infection in patients of minimal hepatic encephalopathy was noted in 67 patients (60.9%).
Conclusion: It is concluded that Helicobacter Pylori Infection is highly prevalent among the patients of minimal hepatic encephalopathy.
Keywords: Helicobacter Pylori, Minimal hepatic encephalopathy, Cirrhosis, Bispectral index.

INTRODUCTION

Hepatic encephalopathy is a frequent complication of liver cirrhosis. It is a reversible, complex neuropsychiatric syndrome. Clinical findings in hepatic encephalopathy include forgetfulness, alterations in handwriting, difficulty with driving, and reversal of the sleep-wake cycle[1,2]. Symptoms may range from mild neurologic disturbances to overt coma.

The mildest form of spectrum of hepatic encephalopathy is minimal hepatic encephalopathy. It refers to grade 0 on West Haven criteria. It is not associated with overt neuropsychiatric symptoms but rather with minimal changes detected only by special tests and is typically reversible with therapy. The diagnosis of minimal hepatic encephalopathy may have clinical importance as it influences the patient's quality of life, driving ability and independent survival [3] and confers an increased risk of becoming overt hepatic encephalopathy. Whether treatment of minimal hepatic encephalopathy confers any benefit is an area of active investigation[1,3,4,5]. Absence of clinical evidence of hepatic encephalopathy is the key to the diagnosis of minimal hepatic encephalopathy.

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The prevalence of minimal hepatic encephalopathy in adult cirrhotic patients varies from 30% to 84% depending upon the use of appropriate methods[6].

Psychometric tests such as number connection test and neurophysiological tests such as Bispectral Index scoring are used as a diagnostic screen for minimal hepatic encephalopathy. Bispectral Index is a bedside tool to monitor electroencephalographic activity.[7] It is helpful in early detection of cerebral dysfunction and classifying the degree of progress in the hepatic encephalopathy. It avoids the learning affects usually seen in psychometric tests and is also helpful in patients with low educational background in whom psychometric test will be difficult to perform.

There are many precipitating factors, which may lead to development of porto-systemic encephalopathy such as accumulation of ammonia, production of false neurotransmitters, decreased activity of urea-cycle enzymes due to zinc deficiency, deposition of manganese in the basal ganglia and increased endogenous activity of benzodiazepines.

One probable but important source of hyperammonemia in cirrhotic patients is Helicobacter Pylori infection[8]. These bacteria are rich in urease enzyme and are known to produce ammonia from urea that is rapidly absorbed from gastric lumen into circulation. Several studies show that the level of ammonia in gastric secretions of Helicobacter Pylori

infected cirrhotic patients is higher than in non-infected patient.

The importance of Helicobacter Pylori as an independent risk factor for the development of hepatic encephalopathy is not yet clear, but it may play a contributory role and its eradication has been shown to be associated with reduction in blood ammonia levels and improvement in hepatic encephalopathy.

However, the role of Helicobacter Pylori in causation of minimal hepatic encephalopathy has not been studied in detail. In a study conducted in China, the frequency of Helicobacter Pylori infection in cirrhotic patients, with minimal hepatic encephalopathy was 69.1%[8]. Similarly in 2011 an Indian study revealed its frequency as 63%[9].

To the best of our knowledge, very limited local data is available regarding Helicobacter Pylori infection in cirrhotic patients, with minimal hepatic encephalopathy. Therefore this study was designed to calculate the frequency of Helicobacter Pylori infection in our population. This study will provide baseline data regarding the magnitude of problem.

MATERIAL AND METHODS

The study was conducted at Gastroenterology Unit, Department of Medical Unit-III, Services Hospital, Lahore. One hundred and ten patients were enrolled in the study. Patients of both genders having age between 18 and 70 years having liver cirrhosis secondary to chronic hepatitis B & chronic hepatitis C with normal Mini Mental State Examination were included in the study. Patients with overt hepatic encephalopathy, recent upper gastrointestinal bleeding, alcoholic patients (minimum of two units of alcohol per week), patients with neurological illness, poor vision, un-controlled diabetes mellitus, chronic renal failure and hypertension, history of recent (less than 6 weeks)use of benzodiazepines or antiepileptic, history of Helicobacter Pylori eradication treatment within the previous three months, patients with platelet count <50,000 and INR >1.3 were excluded from the study.

According to the inclusion and exclusion criteria, 110 patients were selected from out patients department. After informed consent, patients were offered short admission at Medical unit-III. During admission their demographic data, history and clinical examination were performed. Bispectral index was calculated by researcher himself by applying bispectral index monitor's pad on the forehead and graded from 0 to 100. Patients having score between 85 and 100 were classified as having minimal hepatic encephalopathy. Endoscopic biopsies from gastric antrum of these patients were taken by a single consultant and were sent for histopathology in Services institute of medical sciences (SIMS) Lab to confirm Helicobacter Pylori.

RESULTS

Regarding age distribution of patients, 17 patients (15.5%) were 18-40 years old, 67 patients (60.9%) were 41-60 years of age while 26 patients (23.6%) were between 61-70 years of age. Mean age of the patients was observed 52.14±9.61 (Table 1). Out of 110 patients, 63 patients (57.3%) were male and remaining 47 patients (42.7%) were female (Table 2). Helicobacter pylori infection in patients of Minimal hepatic encephalopathy were noted in 67 patients (60.9%) (Table 3)

Table 1: Distribution of cases by age

Age (years)	n	%age
18-40	17	15.5
41-60	67	60.9
61-70	26	24.5

Table 2: Distribution of cases by sex

Gender	n	%age
Male	63	57.3
Female	47	42.7

Table 2: Frequency of Helicobacter Pylori

Helicobacter Pylori	n	%age
Yes	67	60.9
No	43	39.1

DISCUSSION

Minimal hepatic encephalopathy (HE) is complication of cirrhosis. The cirrhotic patients who do not show the symptoms of clinically overt hepatic encephalopathy (HE) present with mild cognitive impairment i.e., minimal hepatic encephalopathy. Various tools have been used for the diagnosis of Minimal Hepatic Encephalopathy but its results clearly differ between different studied populations[10].

Minimal hepatic encephalopathy impairs the quality of life, increases the risk of suffering accidents, predicts the appearance of clinical HE, and is associated with shortened lifespan. Early detection of MHE would be very useful[11]. Acetyl-L-carnitine (ALC) has been shown to be useful in improving blood ammonia and cognitive functions in cirrhotic patients with MHE[12].

There are many precipitating factors, which may lead to development of Minimal Hepatic encephalopathy such as accumulation of ammonia, production of false neurotransmitters, decreased activity of urea-cycle enzymes due to zinc deficiency, deposition of manganese in the basal ganglia and

increased endogenous activity of benzodiazepines. Ammonia has a key importance in the pathogenesis Minimal Hepatic encephalopathy [13-14.] Frequency and role of H. pylori in the pathogenesis of Minimal Hepatic encephalopathy has been a subject of ongoing debate.

In our study we found significant frequency of H. pylori among the patients of Minimal Hepatic encephalopathy (60.9%). Similar results were seen in a study conducted at Zhejiang UniversityChina From July 2003 to January 2005. H pylori prevalence was found among cirrhotic patients with Hepatic Encephalopathy (74.4%), Sub clinical Hepatic Encephalopathy (69.1%) and those without overt Hepatic Encephalopathy (53.2%)[8].

Another study conducted at Department of Medicine, Chattarpati Shahuji Maharaj Medical University, Lucknow, Uttar Pradesh, India in 2011. H. pylori infection was found in 22 (63%) of 35 patients with MHE. This study also measured the fasting blood ammonia levels in patients with MHE before and after giving H. Pylori eradication therapy. Fasting blood ammonia levels were found significantly higher in patients of Minimal Hepatic Encephalopathy who tested positive for H. pylori than in those who tested negative. Patients of MHE who were also positive for H. Pylori were given one-week triple anti-H. Pylori treatment that showed a significant reduction in blood ammonia levels[9].

CONCLUSION

Data from our study has further strengthened the causal association of H. pylori infection in Minimal Hepatic encephalopathy. It can negatively impacts the driving capability and work performance and eradication of this infection may help ameliorate the manifestations of this complication

Although testing for Helicobacter Pylori in MHE and subsequent therapy is not in routine, it is important to consider this infection in all cirrhotics in order to improve their quality of life. Further studies are needed to evaluate the arguments in favour of and against the role of H pylori in the pathogenesis of Minimal Hepatic Encephalopathy.

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