Original Article

HELICOBACTER PYLORI AND PEPTIC ULCER IN CIRRHOTIC PATIENTS

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ABSTRACT

Objective: H.pylori infection increases the risk of peptic ulcer in cirrhotic patients. The role of H.pylori in inducing peptic ulcer disease in chronic liver disease patients remains controversial. The objective was to see if etiology of peptic ulcer in cirrhotic patients is related to H.pylori infection.

Methodology: Sixty cirrhotic patients were enrolled in the study. Peptic ulcer was detected in patients by means endoscope. Sera from patients were tested for Helicobacter pylori antibodies by a commercial ELISA kit. t student and fisher test was used for statistical analysis.

Results: Active peptic ulcer was detected in nine cirrhotic patients. Of the nine patients with peptic ulcer eight (88.9%) tested positive for Helicobacter pylori antibodies and of the remaining 51 patients, 31(60.8%) tested positive for Helicobacter pylori antibodies.

Conclusion: Helicobacter pylori infections in our series of cirrhotic patients with peptic ulcer seems to show the same pattern as described in cirrhotic patients without peptic ulcer (P=0.078). The suggestion that the etiology of the peptic ulcer in cirrhotic patients could be related to Helicobacter pylori infection was not confirmed by our study.

KEY WORDS: H.pylori, Peptic ulcer, Cirrhosis.

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INTRODUCTION

Helicobacter pylori(H.pylori) is established as an important etiologic factor for chronic gastritis and peptic ulcer diseases.[1] Eradication of H.pylori gastric infection markedly decreases peptic ulcer recurrence in patients.[2-8] Moreover, cure of H.pylori infection decreases the risk of recurrence in patients with bleeding peptic ulcer.[3]

Factors related to peptic ulcer development and the causes of increased prevalence of the disease in cirrhotic patients are poorly understood. Patients with liver cirrhosis are frequently subjected to a number of disorders of the gastric mucosa. Peptic lesions in the gastroduodonal mucosa have been found to be more frequent in cirrhotic than in control.[4]

A previous report indicates that H.pylori infection increases the risk of peptic ulcer in cirrhotic patients by 2.7 fold; however, subsequent study does not confirm this finding.[5] Some studies have reported that there is some

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degree of difference between the hepatic pressure gradient in patients with and without gastric ulcer.[6] Thus the role of H.pylori in inducing peptic ulcer disease in chronic liver disease patients remains controversial.[7] This study was conducted to see if etiology of peptic ulcer in cirrhotic patients is related to H.pylori infection.

METHODOLOGY

Sixty cirrhotic patients between July 2006 and March 2008 were enrolled in the study. A diagnosis of cirrhosis was made on the basis of clinical findings, abdominal sonogram; endoscopic procedures done by fellows in gasteroenterology besides laboratory parameters and/or liver biopsy. At the time of inclusion a questionnaire was completed and patient serum was obtained and stored at -70°C until analyzed. Etiology of the cirrhosis was defined as viral when HBS Ag or antibodies to the hepatitis C virus were present. Less common diseases such as alcoholic cirrhosis, Wilson disease, primary billiary cirrhosis, cancer and autoimmune was detected. According to the current clinical criteria. Cancer in these patients was etiological factor for cirrhosis. Finally some patients were defined as unknown etiology of cirrhosis.

Sera from patients were tested for Helicobacter pylori antibodies by a commercial ELISA kit (PISHTAZ TEB). Its sensivity for the diagnosis of Helicobacter pylori infection has been reported to be >98% and its specificity >98. In accordance with the manufacturers' instructions, ELISA values lower than 15 were considered negative and values 15 or over positive.

Statistical Analysis: Statistical analyses were performed with independent students t test and chi-square test accordingly. Results were considered statistically significant at $P<0.05$.

RESULTS

A total sixty cirrhotic patients who attended the gastrointestinal clinic during twenty month study period were enrolled in the study. Thirty eight patients were male and twenty two female with mean age+-sd, 47.22+-17.12(15-72) years. Table-I shows the age, sex and etiology of patients.

Table-I: Results of the univariate analysis

Analysis		*Number*	*%*
Sex	Male	38	63.3
	Female	22	36.7
	Total	60	100
Age	<20	6	10
	21-30	8	13.4
	31-40	6	10
	41-50	14	23.3
	51-60	11	18.3
	>60	15	25
	Total	60	100
Cirrhosis etiology			
Alcoholic		2	3.3
HCV		6	10
HBV		16	26.7
Primary billiary cirrhosis		3	5
Wilson		1	1.7
Cancer		5	8.3
Autoimmune		1	1.7
Unknown		24	40
Total		60	100

Active peptic ulcer was detected in nine cirrhotic patients by means endoscope (model GFI-XQ230). Of the nine patients with peptic ulcer eight (88.9%) tested positive for Helicobacter pylori antibodies and of the remaining 51 patients, thirty one (60.8%) tested positive for Helicobacter pylori antibodies (Table-II). Peptic ulcer was detected in anterum, fundus and duodenal area.

Esophageal varices were diagnosed in 13 of the 60 patients by means endoscope. Of the 13 patients with esophageal varices eleven (84.6%) tested positive for Helicobacter pylori antibodies and of the remaining 47 patients, twenty eight (59.6%) tested positive for Helicobacter pylori antibodies (Table-III).

Table-II: Peptic ulcer and H.pylori IgG in cirrhotic patients

Peptic ulcer IgG	*Negative*	*Positive*	*Total*	
Positive	31(60.8%)	8(88.9%)	39(100%)	
Negative	20(39.2%)	1(11.1%)	21(100%)	X =3.105
Total	51(100%)	9(100%)	60(100%)	P=0.078

DISCUSSION

There are many factors that could have induced peptic ulcer. As regards gastric acidity in cirrhosis, there was marked hypogastric acidity over the entire circadian cycle which was evaluated from 24-h gastric acidity. Nevertheless, the actual mechanism of hypoacidity is poorly understood. The degree of acidity and modulators of gastric mucosal response needs further investigation to confirm its association with the pathogenesis of peptic ulcer in cirrhosis.[5] The most important etiology of peptic ulcer is H.pylori infection. But the etiology of cirrhosis was not related to the seroprevalence of Helicobacter pylori infection and on the other hand Helicobacter pylori infection was not related to sex, age and etiology of cirrhosis.

Peptic ulcer is a major problem in patients with liver cirrhosis and the risk of the peptic ulcer is greater in these patients.[7] Prospective endoscopic surveys have shown the incidence of peptic ulcer of 4.3% in patients with cirrhosis which is between 20 and 47 times greater than in the general population.[7]

It has been demonstrated that patients with liver cirrhosis are frequently subjected to a number of disorders of the gastric mucosa which have been observed more often in cirrhotic patients than in controls. Helicobacter pylori infection is also an important factor in the pathogenesis of peptic ulcer.[9] But H.pylori eradication does not protect all cirrhotics from ulcer recurrence.[10]

The prevalence of peptic ulcer observed in this study is 15% of total 60 cirrhotic patients more than previous studies).

Our results demonstrate that, H.pylori infection was an independent factor associated with cirrhosis and peptic ulcer. The difference in the prevalence of H.pylori infection between cirrhotic patients with peptic ulcer (88.9%) and without peptic ulcer (51.7%) did not reach statistical significance (P=0.078).

Esophageal varicies and variceal bleeding is most related to death in cirrhotic patients. There seems to exist associations between infection and other complications such as malnutrition, hepatic encephalopathy and variceal bleeding.[11,12] In this study we obtained the same results about relationship between esophageal varicies and H.pylori infection. The difference in the prevalence of H.pylori infection between cirrhotic patients with esophageal varicies (84.6%) and without esophageal varicies (59.6%) did not reach statistical significance (P=0.078).

In conclusion, Helicobacter pylori infections in our series of cirrhotic patients with peptic ulcer seems to show the same pattern as described in cirrhotic patients without peptic ulcer (P=0.078). The suggestion that the etiology of the peptic ulcer in cirrhotic patients

Table-III: Esophageal varicies and H.pylori IgG in cirrhotic patients

Esophageal varicies IgG	*Negative*	*Positive*	*Total*	
Positive	28(59.6%)	11(84.6%)	39(100%)	
Negative	19(40.4%)	2(15.4%)	21(100%)	X =3.109
Total	47(100%)	13(100%)	60(100%)	P=0.078

could be related to Helicobacter pylori infection was not confirmed by our study. This study was carried out in only one centre so the study patients may not represent all cirrhotic patients. Therefore, more studies performed in other regions with more patients are needed to confirm our findings.

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