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Helicobacter Pylori Infection and Risk of Variceal Bleeding among Patients with Liver Cirrhosis

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Abstract

Inflammatory changes in the stomach caused by Helicobacter pylori indirectly and directly affect liver function. Moreover, the bacteria may worsen the course of the liver cirrhosis. The incidence of portal hypertension and esophageal varices correlates with the incidence of H. pylori infection. This study aims to detect H. pylori infection among patients with liver cirrhosis correlation with the degree of liver injury according to Child–Pugh classification and the stage of esophageal varices and its bleeding. This study was conducted in Hepatology and Gastroenterology and Infectious diseases of Al_Azhar medical Department from December 2018 till November 2020. 50 patients were included in bleeder group 32 patient had H.pylori and 18 patient negative H.pylori. 50 patient were included in non_bleeder group 28 patient had H.pylori and 22 patient were negative H.pylori. our results indicated statistically significant decrease in the prevalence of H. pylori infection among the bleeder group.

Keywords: Child-Pugh, H. pylori, esophageal varices.

INTRODUCTION

Helicobacter pylori is a Gram-negative

bacillus, resistant to the activity of gastric juice. The bacteria may take the vegetative or sporulation form.

H.pylori lives mainly on the surface of epithelial cells of mucous membranes of the prepyloric part of the stomach. The cilia present on the bacteria allow it to move into intercellular spaces and adhere to the surface of the cells.

Infection with these bacteria is one of the most common infection in the world [(1)].

Inflammatory changes in the stomach caused by Helicobacter pylori indirectly and directly affect liver function. Moreover, the bacteria may worsen the course of the liver cirrhosis. H. pylori infection is significantly more frequent among patients with post inflammatory liver cirrhosis related to HCV or HBV infection than in patients with alcoholic liver cirrhosis or primary biliary cirrhosis.[(2)]

The incidence of esophageal varices correlates with the incidence of H. pylori infection. Also, there is significant association between Helicobacter pylori infection and portal hypertensive gastropathy (PHG) in cirrhotic patients, also related to PHG severity, thereby necessitating H. pylori eradication.[(3)]

PATIENTS AND METHODS

The current study is a prospective case control study conducted on 150 patients admitted at Dar Elsalam Hospitals and EL Fayoum general Hospital with chronic liver diseases and portal hypertension after given their informed consent to the study

All patients are divided into three groups: GroupI: Contains 50 patients complaining of hematemesis as a result of bleeding Ovs. **(bleeders group).**

GroupII: Contains 50 patients attending

to endoscopy unit for prophylactic treatment of Ovs. **(non bleeders group). GroupIII:** Contains 50 patients with chronic liver disease without Ovs as a (**control group).**

Results

		Non bleeding (No.50)		Bleeding (No.50)		Control (No.50)		Chi square test/ one way ANOVA	
		No	%	No	%	No	%	X^2	P value
Age	Mean ± SD	45.2±2.15		48.65±3.2		53.25± 1.2		0.165	0.354
	Range	30-70		35-70		35-70			
Sex	Male	27	54.0%	26	52.0%	28	56.0%	0.161	0.922
	Female	23	46.0%	24	48.0%	22	44.0%		

Table (1): Discription of demographic data (age and gender) in between the studied gr

		Bleeding (No.50)		Non-Bleeding (No.50)	
		No	%	No	%
OV grade	II	8	16.0%	18	36.0%
	III	20	40.0%	18	36.0%
	IV	22	44.0%	14	28.0%

Table (2): OV grade in studied groups

		Non bleeding (No.50)		Bleeding (No.50)		Control (No.50)		Chi square test	
		No	%	No	%	No	%	X^2	P value
Child stage	A	18	36.0%	0	0.0%	50	100.0 %	11.088	0.001
	B	23	46.0%	23	46.0%	0	0.0%		
	C	9	18.0%	27	54.0%	0	0.0%		
H. pylori	Negative	22	44.0%	32	64.0%	25	50.0 %	4.225	0.121
	Positive	28	56.0%	18	36.0%	25	50.0 %		
ultrasound	No	7	14.0%	0	0.0%			13.894	0.003
	Mild	13	26.0%	14	28.0%				
	Moderate	24	48.0%	18	36.0%				
	Sever	6	12.0%	18	36.0%				

Table (3): child Pugh score, H.pylori distribution and degree of acitis in the studied groups.

	Non bleeding		Bleeding		Control (No.50)		one way ANOVA	
	Mean	SD	Mean	SD	Mean	SD	t	P value
Bilirubin	2.06	0.84	2.94	1.14	1.11	0.11	٥٥,١٤٦	0.001
Albumin	3.67	4.08	2.39	0.63	4.64	0.56	١١,٤٣١	0.001

Table (4): Serum bilirubin and serum albumin in the studied group:

There was no statistically significant difference between demographic data among studied groups(Table 1). From bleeding group 8 patients (16%) were OV grade II and 20 patients (40%) were OV grade III and 22 patients were OV grade IV (44%) (Table 2). There was statistically significant increase stage A in control group and non-bleeding group and increase stage C in bleeding group and statistically significant increase H. pylori in non-bleeding group. There was statistically significant difference between studied groups as regard to ascites in ultrasound. There was increase severe ascites in bleeding group but increase moderate ascites in non-bleeders group (Table 3). There was statistically significant difference between the studied groups as regard serum bilirubin and serum albumin (Table 4).

DISCUSSION

Cirrhosis of liver is a major problem in the world and is characterized by extensive fibrosis. Portal hypertension is a complication of cirrhosis and can lead to the development of gastrointestinal varices. **(5)**

Helicobacter pylori (H. pylori) are a worldwide gastrointestinal infection. there is a significant association between H. pylori infection and severity of gastropathy due to portal hypertension, chronic stomach upset and frequent

upper gastrointestinal bleeding. [6]

The aim of our study was to investigate H. pylori infection among patients with liver cirrhosis and its correlation with the degree of liver injury according to Child–Pugh classification and the stage of esophageal varices and its bleeding.

This prospective case control study included 150 patients with chronic liver diseases and portal hypertension. They were recruited and assessed for eligibility from at Kasr AL-ainy Hospitals and EL Fayoum general Hospital and they were divided into three groups; A) bleeding group that included 50 patients complaining of haematemesis as a result of bleeding oesophageal varices (Ovs) or gastric, B) non-bleeders group that included 50 patients attending to endoscopy unit for prophylactic treatment of OVs C) a control group that included 50 patients with chronic liver disease without Ovs.

Regarding the demographic characteristics of the studied groups, our results indicated that the mean age of the non-bleeding, bleeding and control groups were 45.2±2.15; 48.65±3.2 and 53.25± 1.2 respectively, with no statistically significant difference among the studied groups (P value=0.354). There were no significant difference between the studied groups regarding genders (P value = 0.922). This study was a case control study with age and gender matched study groups to improve study efficiency by improving precision and avoiding the confounding effects of both age and gender in the statistical analysis.[7]

Regarding the severity of liver cirrhosis assessed by child-pugh class, our results indicated a statistically significant increase of category A in control group (100%) and increase of category C

among the bleeding group while there were increase in category B among the non-bleeding group (P = 0 001). A study by **Kumar *et al.*** [8]indicated that the prevalence of large esophageal varices in patients with advanced child-Pugh class was higher than that in patients with child-Pugh class A. Moreover, **Cherian *et al.*** [9]also found that child-Pugh class B/C emerged as significant predictors for the presence of esophageal varices.

Among the bleeding group, regarding the child stage, our result indicated that 46% of patients were stage B and 54% were stage C, moreover regarding the OVs grade, our result indicated that ١٦% of patients were OV grade II, ٤٠% were OV grade III and 46% were grade VI. Such finding was in agreement with **a** previous study by **Gunda *et al.*** [10] indicated that liver cirrhosis of Child-Pugh B/C was reported as an independent predictor of esophageal varices.

Among the bleeding group, regarding the prevalence of ascites, our results indicated that 72% of the cases have moderate to severe ascites and 28% has mild ascites. We observed a significant increase moderate and severe ascites among the bleeding group in comparison with the non-bleeding group regarding the grade of ascites (P value=0.003).

Such finding was in agreement with **Gunda *et al.*** [10] that indicated high prevalence of ascites (94.3%) among patients with esophageal varices and liver cirrhosis. A previous study by **Hassanin *et al.*** [11] indicated that 53% of patients with cirrhotic liver have moderate to severe grade ascites.

Additionally, **Kumar *et al.*** [8]study indicated that the prevalence of ascites among cirrhotic patients with grade III and IV esophageal varices was 78.26%

while the prevalence of ascites among cirrhotic patients with grade I and II esophageal varices was 62.96%.

Our result indicated that no statistically significant differences in the prevalence of H. pylori infection among the studied groups (P value = 0.121). The frequency of H. pylori infection in the non-bleeding, bleeding and control groups was 56%, 36% and 50% respectively. Such findings were in agreement with **Eid *et al.*** [12]study that indicated no significant correlation between H. pylori infection and the severity of liver cirrhosis. **Pogorzelska *et al.*** [2]indicated that the overall prevalence of H. pylori infection in cirrhotic patients ranged from 35.1% to 70.6%. This discrepancy is perhaps related to the different investigational tools used for the diagnosis of H. pylori infection.

A recent retrospective study by **Guo *et al.*** [13] indicated that patients who failed the initial H pylori eradication have an increased risk of upper gastrointestinal bleeding compared to patients who responded to the initial therapy and early retreatment within 3 months should be considered. On the other hand, our result was in disagreement with **Elsebaey *et al.*** [14] that indicated significant increase of the prevalence of H. pylori among the bleeding group in comparison with non-bleeding group.

Among the bleeding group, regarding the biochemical markers of varices, our results indicated that the mean of serum bilirubin was 2.94 with range from 1 to 4 and mean of serum albumin was 2.39 mg/dL with range from 1.4 to 3.5 mg/dL. Such findings were in agreement with Mohamed *et al.* [15]that indicated that most of patients with cirrhosis presented with upper gastrointestinal bleeding have albumin levels less than 3.5 mg/dL and bilirubin level higher than 2 mg/dL. A previous study by Budiyasa *et al.*

[16]indicated that in esophageal varices patients, the serum albumin level ranged between 1.10-3.60 mg/dL, the average value was 2.21 ± 0.451 mg/dL and that serum albumin level can predict the presence and the degree of EV in patients with liver cirrhosis.

Our results indicated a statistically significant difference regarding the albumin and bilirubin levels among the studied groups (P value = 0.001). The mean albumin level among the non-bleeding, bleeding and control groups was 3.67± 4.08; 2.39± 0.63 and 4.64± 0.56 respectively. The mean bilirubin level was 2.06± 0.84; 2.94± 1.14 and 1.11± 0.11 respectively. A previous study indicated that the mean level of bilirubin in patients with chronic liver diseases and H. pylori infection was 2.7± 0.5260 mg/dl that is consistent with our results. [17] On the other hand, our result was in disagreement with Gunda *et al.* [10]that indicated no significant difference in serum albumin levels between those with and without esophageal varices.

Conclusion

H. pylori infection is not a risk factor for bleeding from esophageal varices. Child-Pugh class B/C emerged as significant predictor for the presence of esophageal varices.

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