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Original Article

Relation between Helicobacter pylori infection and severity of portal hypertensive gastropathy in cirrhotic patients

Hepatology and gastroenterology

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ABSTRACT

Background: Cirrhosis is a great health problem worldwide and is associated with many complications. Portal hypertensive gastropathy (PHG) is one of its most common complications and in some patients may lead to gastrointestinal bleeding. The relationship between Helicobacter pylori (H. pylori) infection and PHG is not clearly identified.

Objective: This work aimed to study a possible association between H. pylori infection and PHG and to correlate the severity of PHG with H. pylori infection in cirrhotic patients.

Methodology: A cross sectional comparative study included 90 cirrhotic patients, who were classified into 3 groups. G1: included 30 age and sex-matched cirrhotic patients with no PHG. G2: included 30 age and sex-matched cirrhotic patients with mild PHG. G3: included 30 age and sex-matched cirrhotic patients with severe PHG. Endoscopic examination was done to evaluate the presence and severity of portal hypertensive gastropathy and for diagnosis of H. pylori infection.

Results: H. pylori infection by histopathology was detected in 61 studied patients (67.8%). H. pylori infection was more prevalent among cirrhotic patients with PHG (78.3%) than patients without PHG (46.7%) (P=0.0029). H. pylori infection was detected in 26(86.7%), 21(70.0%), 14(46.7%) in patients with severe, mild and no PHG respectively. H. pylori infection was more prevalent in patients with severe PHG relative to patients with no PHG (p-value = 0.001).

Conclusion: The current study showed a significant relation between H. pylori infection and PHG in cirrhotic patients and it might have a role in the pathogenesis of severe PHG.

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Key words: Cirrhosis, H. pylori, portal hypertensive gastropathy.

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INTRODUCTION

Cirrhosis is a common health problem globally. It is associated with many changes in the mucosa of the gastrointestinal tract, with higher risk for peptic ulcer disease [1]. Portal hypertensive gastropathy (PHG) complicates portal hypertension either cirrhotic or non-cirrhotic. The mucosa of the stomach in PHG has specific endoscopic appearance described as a mosaic-like pattern like a snakeskin, with or without red spots [2]. H. pylori is a gram- negative bacterium colonizes the mucosa of the stomach. It is the main cause of peptic ulcer disease and may be associated with gastric cancer or mucosa associated lymphoid tissue lymphoma (MALT) [3]. H. pylori DNA in some patients with chronic hepatic disease could be detected in liver tissue, suggesting that coinfection with H. pylori may aggravate the liver condition [4]. Internationally, histopathology is the accepted gold standard for the diagnosis of H. pylori infection. H. pylori stool antigen can be used for monitoring of H. pylori treatment [5].

The prevalence of H. pylori in patients with liver cirrhosis, and its association with PHG are controversial [6]. Puri *et al*. showed a significant relation between presence and severity of gastropathy and H. pylori infection in patients with liver cirrhosis. The presence of H. pylori was observed in 26 (67%) cirrhotic patients with PHG compared to 7 (33%) cirrhotic patients without PHG. Hemorrhagic congestion and edema of the gastric mucosa in cirrhosis might provide a suitable environment for the colonization of H. pylori [7]. The

virulence of H. pylori is increased in cirrhotic patients and exaggerates the effect of PHG on the gastric mucosa. This is due to increased inducible nitric oxide synthase (iNOS) expression and impairment of gastric mucosal defense in cirrhotic patients with PHG [8]. More studies are needed to confirm the association of H. pylori infection with PHG [7]. If H. pylori infection is found to have a role in the pathogenesis of PHG, treatment of H. pylori will be essential to decrease complications of PHG. This work aimed to study a possible association between H. pylori infection and PHG and to correlate the severity of PHG with H. pylori infection in cirrhotic patients.

PATIENTS AND METHODS

The study was approved by the ethical committee at the department of Hepatology, Gastroenterology, and Infectious diseases, El-Hussein University hospital, and at Al-Azhar faculty of Medicine. Cross sectional comparative study was conducted on 90 cirrhotic patients. They were-classified into 3 groups according to scoring system proposed by Baveno III consensus workshop as reported by De Franchis [9]. G1: included 30 age and sex-matched cirrhotic patients with no PHG (as control group). G2: included 30 age and sex-matched cirrhotic patients with mild PHG. G3: included 30 age and sex-matched cirrhotic patients with severe PHG.

Patients with peptic ulcer found in upper gastrointestinal endoscopy, history of gastric surgery, previous therapy for eradication of H. pylori, patients who had primary or secondary hepatic malignancies, history of previous band ligation or sclerotherapy for esophageal varices, acute variceal bleeding in the previous 2 weeks, patients with thrombosis of the portal, supra hepatic veins or inferior vena cava, patients who had an active infection, and those who were on beta blockers, nitrates, NSAIDS, PPI, or H_2 receptor antagonists in the last 4 weeks were excluded from the study.

Study population was subjected to 1) Full history taking. 2) Thorough clinical examination. 3) Laboratory investigations including: CBC (Cell dyn ruby 34685BG, spectra group), liver biochemical profile: AST, ALT, serum albumin, total and direct bilirubin (Integra chemical analyzer using Roche kits), INR (Biolabo diagnostics, Solea 100 using Biolabo prothrombin kit), renal function tests: serum creatinine and blood urea (Integra chemical analyzer using Roche kits), HCV-Ab testing using 3rd generation ELISA and HBsAg (Chromate immunological analyzer by using Genlab adaltis kits) and Alphafetoprotein (Cobas 411 using Roche kits). 4) Abdominal ultrasound: for diagnosis of cirrhosis, hepatic focal lesions, portal vein diameter and patency, splenomegaly, splenic vein diameter and ascites. 5) Endoscopic examination: to evaluate the presence and grade of esophageal varices as well as portal hypertensive gastropathy and to assess its severity. Six images were selected, consisting of two for the gastric body, two for the antrum and two for the gastric fundus. At each endoscopy, the severity of portal hypertensive gastropathy was scored using Baveno III consensus workshop. 6) Diagnosis of H. pylori infection: through endoscopy and biopsy for histopathology which is the gold standard for the diagnosis of H. pylori. Biopsies were obtained from all parts of the stomach since the colonization of H. pylori might be spotty.

Statistical analysis: Data analysis was done using SPSS software version 21. $\chi 2$ -test was used for comparison of categorical data. Quantitative data were expressed in the form of mean ±SD and independent t- test was used for comparison between groups. Probability level (P) was assumed significant if it was less than 0.05.

RESULTS

A total of 90 patients with liver cirrhosis were enrolled in the study divided into 3 groups. 30 patients with cirrhosis and no endoscopic diagnosis of PHG, 30 patients with cirrhosis and endoscopic diagnosis of mild PHG, as well as 30 patients with cirrhosis and endoscopic diagnosis of severe PHG. The mean age of patients with no PHG was 51.96±5.78 years and 60% were men, the mean age of patients with mild PHG was 51.30±4.94 and 63.3% were men whereas the mean age of patients with severe PHG was 50.56±4.49 years and 60% were men. There was no significant difference in age, sex and smoking distribution in the studied groups (p value > 0.05) (table 1).

Child score was assessed in studied groups. Cirrhotic patients with no PHG were classified as 23 patients with Child A score (76.7%), 7 patients with child score B (23.3%) while none had Child C score (0%) with overall mean Child scoring of 5.83±1.56. Patients with PHG was classified as patients with Child A score 17 (56.7%) and 11 (36.7%), patients with Child B score 13 (43.3%) and 0 (0%), patients with Child C score 0(0%) and 19 (63.3%) with overall mean Child scoring of 6.50±1.83 and 9.10±3.23 in patients with mild and severe PHG, respectively. There was statistically significant difference in Child score between patients with severe PHG relative to patients with no and mild PHG (p-value < 0.001) (table 1 and figure 1). Dilated splenic vein (>9mm), dilated portal vein (>14mm) and moderate ascites were detected by ultrasonography in 14(46.7%), 18(60%) and 5(16.7%) in patients having mild PHG and were detected in 21(70.0%), 23(76.7%) and 7(23.3%) in patients having severe PHG with statistically significant difference relative to patients with no PHG (p-value < 0.05) (table 1).

The study showed that serum albumin was decreased while INR, serum bilirubin and AFP were increased in patients having severe PHG with statistically significant difference relative to no and mild PHG groups (p-value < 0.05) (table 2). As regard to the etiology of cirrhosis, 78 of studied cirrhotic patients were due to chronic HCV

infection (86.7%) while only 12 patients were due to chronic HBV infection (13.3%). Chronic HCV infection was detected in 26(86.7%), 25(83.3%), 27(90.0%) in patients with no, mild and severe PHG respectively. Chronic HBV infection was detected in 4(13.3%), 5(16.7%), 3(10.0%) in no, mild and severe PHG groups respectively without statistically significant difference (p value > 0.05) (table 2).

H. pylori infection was detected in 61 studied patients (67.8%). There was a significant relation between H. pylori infection and PHG. Results showed that H. pylori infection was more prevalent among cirrhotic patients with PHG (78.3%) than patients without PHG (46.7%) (P=0.0029) (Table 3). H. pylori infection was detected in 26(86.7%), 21(70.0%), 14(46.7%) in patients with severe, mild and no PHG respectively. H. pylori infection had statistically significant difference in patients with severe PHG relative to patients with no PHG (p-value = 0.001) (table 4).

Endoscopic examination of cases revealed that esophageal varices were detected in 34 studied patients (37.8%). There was a significant relation between PHG and development of esophageal varices. Results showed that esophageal varices were more prevalent among cirrhotic patients with PHG (48.33%) than patients without PHG (16.7%) (P=0.005) (Table 5). Esophageal varices were detected in 5(16.7%), 12(40%), 17(56.7%) in patients with no, mild and severe PHG respectively with statistically significant difference in patients with severe and mild PHG relative to patients with no PHG (p-value <0.05) (table 6).

Table (1): The baseline characteristics of 90 patients who were enrolled in the study

Variables		No PHG (n=30)	Mild PHG (n=30)	Severe PHG (n=30)	P1	P2	P3
Age (years)	Mean ± SD	51.96±5.78	51.30±4.94	50.56±4.49	0.633	0.299	0.550
	Min-Max	43.00-60.00	44.00-59.00	43.00-60.00			
Sex	Male	18 (60%)	19 (63.3%)	18 (60%)	0.791	1.0	0.791
	Female	12 (40%)	11 (36.7%)	12 (40%)			
Smokers		5 (16.7%)	6 (20%)	6 (20%)	0.739	0.739	1.0
Child score							
A		23 (76.7%)	17 (56.7%)	11 (36.7%)	0.10	0.002*	0.121
B		7 (23.3%)	13 (43.3%)	0 (0%)	0.10	0.005*	0.001*
C		0(0%)	0 (0%)	19 (63.3%)	-	0.001*	0.001*
Mean ± SD		5.83±1.56	6.50±1.83	9.10±3.23	0.134	0.001*	0.001*
Splenomegaly (≥13cm)		24(80.0%)	15(50.0%)	19(63.3%)	0.038*	0.240	0.297
Dilated splenic vein (>9mm)		4(13.3%)	14(46.7%)	21(70.0%)	0.005*	0.001*	0.067
Hilar collaterals		9(30.0%)	12(40.0%)	14(46.7%)	0.417	0.184	0.602
Dilated portal vein (>14mm)		8(26.7%)	18(60.0%)	23(76.7%)	0.028*	0.001*	0.20
Ascites							
No		17(56.7%)	17(56.7%)	14(46.7%)	1.0	0.438	0.438
Mild		13(43.3%)	8(26.7%)	5(16.7%)	0.175	0.024*	0.347
Moderate		0 (0%)	5(16.7%)	7(23.3%)	0.019*	0.005*	0.518
Severe		0 (0%)	0 (0.0%)	4 (13.3%)	-	0.038*	0.038*

P1: p-value of mild PHG relative to no PHG, P2: p-value of severe PHG relative to no PHG, P3: p-value of severe PHG relative to mild PHG., * p-value <0.05.

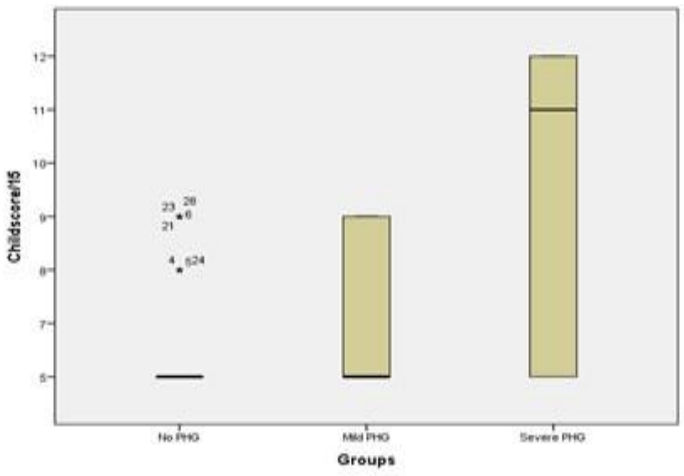

Figure (1): Child score among studied groups

Table (2): Laboratory data among studied groups

Laboratory data		No PHG (n=30)	Mild PHG (n=30)	Severe PHG (n=30)	P1	P2	P3
Serum Albumin (3.5 -5 gm/dl)		3.98±0.69	3.77±0.66	2.75±0.99	0.236	0.001*	0.001*
INR		0.86±0.21	1.35±0.12	1.65±0.27	0.001*	0.001*	0.001*
Total Bilirubin (1.2 mg/dl)		1.80±0.79	2.36±1.29	3.55±2.20	0.047*	0.001*	0.014*
ALT (≤50 U/L)		41.63±5.67	41.70±5.16	43.86±5.88	0.962	0.140	0.135
AST (≤50 U/L)		41.80±4.98	40.20±4.27	43.30±3.30	0.187	0.175	0.003*
Serum creatinine (0.7-1.4 mg/dl)		0.87±0.25	0.77±0.10	0.90±0.29	0.053	0.611	0.02*
Blood Urea (12-20 mg/dl)		35.90±4.07	33.93±4.17	36.03±5.07	0.07	0.911	0.085
AFP		40.46±10.45	41.06±10.71	48.96±14.4	0.827	0.011*	0.019*
HBsAg	Positive	4(13.3%)	5(16.7%)	3(10.0%)	0.739	0.488	1.0
	Negative	26(86.7%)	25(83.3%)	27(90.0%)			
HCV Ab	Positive	26(86.7%)	25(83.3%)	27(90.0%)	1.0	1.0	0.706
	Negative	4(13.3%)	5(16.7%)	3(10.0%)			

P1: p-value of mild PHG relative to no PHG, P2: p-value of severe PHG relative to no PHG, P3: p-value of severe PHG relative to mild PHG.
AFP: Alpha-fetoprotein, * p-value <0.05

Table (3): Relation between Helicobacter pylori infection and PHG in cirrhotic patients

Variables	No PHG (n=30)	PHG (n=60)	P-value
H. pylori positive	14 (46.7%)	47(78.3%)	0.0029
H. pylori negative	16(53.3%)	13(21.7%)	

Table (4): Relation between Helicobacter pylori infection and severity of PHG in cirrhotic patients

Variables	No PHG (n=30)	Mild PHG (n=30)	Severe PHG (n=30)	P1	P2	P3
H. pylori positive	14 (46.7%)	21(70.0%)	26(86.7%)	0.067	0.001*	0.117
H. pylori negative	16(53.3%)	9(30.0%)	4(13.3%)			

P1: p-value of mild PHG relative to no PHG, P2: p-value of severe PHG relative to no PHG, P3: p-value of severe PHG relative to mild PHG.
* p-value <0.05

Table (5): Relation between esophageal varices and PHG in cirrhotic patients.

Variables	No PHG (n=30)	PHG (n=60)	P - value
No OV	25(83.3%)	31(51.67%)	0.003
OV	5(16.7%)	29(48.33%)	

OV: esophageal varices.

Table (6): Relation between esophageal varices grades and severity of PHG among studied groups.

Variables	No PHG (n=30)	Mild PHG (n=30)	Severe PHG (n=30)	χ2	P1	P2	P3
	n (%)	n (%)	n (%)				
No OV	25(83.3%)	18(60.0%)	13(43.3%)	11.96	0.039*	0.002*	0.409
Grade I-II OV	5(16.7%)	7(23.3%)	11(36.7%)				
Grade III-IV OV	0(0%)	5(16.7%)	6(20.0%)				

P1: p-value of mild PHG relative to no PHG, P2: p-value of severe PHG relative to no PH, P3: p-value of severe PHG relative to mild PHG
OV: esophageal varices, * p-value <0.05

DISCUSSION

H. pylori is a microaerophile, Gram-negative bacillus, resistant to the activity of gastric juice. It is a pathogen of the stomach [10]. H. pylori lives mainly in the prepyloric part of the stomach specifically on the surface of epithelial cells of mucous membrane. Infection with these bacteria is one of the most common in the world. In highly developed countries, 50% of the population is infected, whereas in developing countries the proportion is about 90% [11]. PHG is an important cause of morbidity in patients with cirrhotic or non-cirrhotic portal hypertension [12]. In patients with PHG, dilated blood vessels and mucosal friability are present in the mucosal surface. PHG develops because of portal hypertension which leads to increase gastric blood flow and congestion of mucosal and submucosal blood vessels [13]. PHG leads to decreased local mucosal defense and decreased mucous secretion so the mucosa becomes weak and susceptible to injurious agents like non-steroidal anti-inflammatory drugs and H. pylori colonization [14]. The incidence of H. pylori infection among patients with liver cirrhosis and HCV infection increases with more pronounced liver failure [15].

The present study was done to investigate the association between H. pylori infection and the severity of portal hypertensive gastropathy in patients with liver cirrhosis. This study showed that there was no significant difference in age, sex and smoking distribution in studied groups because the sample was cross-matched (p-value > 0.05). In the present study, there was no significant difference between studied groups in distribution of diabetes and hypertension. This agrees with Puri *et al.* [7] who detected that there was no statistically significant association between PHG and fasting blood sugar. Upper gastrointestinal tract bleeding from PHG varies widely (2-12%) [16]. It is reported that ~10% of PHGs cause anemia due to chronic blood loss and 2.5% of patients experienced acute bleeding [13].

In the current study upper gastrointestinal tract bleeding was common in severe PHG group (56.7%) relative to (53.3% and 36.7% in mild & no PHG groups respectively) (p-value < 0.05). GI bleeding is the only known clinically relevant complication of PHG. PHG is responsible for < 1% of upper GI bleeding in the general population, and for about 8% of non-variceal upper GI bleeding in patients with liver disease. The reported frequency of acute upper GI bleeding in patients with PHG ranges in incidence from 2%-20% [12]. Numerous studies reported that PHG is correlated with liver disease severity [17]. The current study showed that signs of liver disease such as jaundice, lower limb edema, spider nevi, palmer erythema, flapping tremors, epigastric tenderness, splenomegaly, and ascites were common in severe PHG group (50%, 50%, 50%, 46.7%, 36.7%, 53.3% and 63.3% respectively) than patients having no or mild PHG. Jaundice, flapping tremors and ascites only had statistically significant difference relative to no PHG group (p-value < 0.05). This agreed with other studies reported a significant relation between PHG and jaundice & splenic size [18, 19]. Moreover, there were other studies showed a significant relation between severe PHG and development of lower limb edema, flapping tremors, palmer erythema, spider nevi and epigastric tenderness [20].

This study showed that Child score was higher in severe PHG group (9.10±3.23) relative to mild PHG (6.50±1.83) and no PHG (5.83±1.56) groups (p-value < 0.001). This agrees with several studies as Kim (2008) [21] who concluded that there was significant association between PHG, and liver disease severity assessed by Child score. Moreover, several studies reported that PHG is correlated with liver disease severity measured by Child-Pugh stage. Merli *et al.* [16] showed that Child-Pugh class B or C was correlated with the progression from mild to severe PHG. Moreover, De Lisi *et al.* [22] reported a significantly higher prevalence of PHG in

Child-Pugh stages B or C, as compared to stage A. On the contrary, other studies showed no significant differences between severity of PHG regarding to Child score [7, 17, 23].

The current study showed statistically significant dilated splenic vein (>9mm), dilated portal vein (>14mm) and moderate ascites in patients having mild and severe PHG relative to no PHG group (p-value < 0.05). This agrees with studies done by Zardi *et al.* [17] and Elsakaty *et al.* [24]. In contrast to the study of Abbas [25] who showed lower portal vein diameter in patients with PHG. Also, results of this study agreed with other studies who concluded that ascites was more significant in PHG patients in comparison to patients without PHG [24, 26].

This study showed that Hb concentration and RBCs count were significantly decreased in patients having severe PHG relative to no and mild PHG groups (p-value < 0.001). Patients with PHG may suffer from an acute GIT bleeding or may commonly have an iron deficiency anemia due to chronic blood loss from the congested and friable mucosa [27].

This study showed that serum albumin was decreased while INR, serum bilirubin and AFP were increased in patients having severe PHG with statistically significant difference relative to no and mild PHG groups (p-value < 0.05). Several studies showed that lower serum albumin, prolongation of INR and high total serum bilirubin were observed in severe PHG [19, 21, 28]. On the contrary, Puri *et al.* [7] showed that there was no statistically significant association between PHG and serum albumin, INR and total serum bilirubin. These different results could be attributed to different etiologies of liver cirrhosis in these studies.

This study showed that cirrhosis in studied patients was due to chronic HCV infection 78(86.7%) while in only 12 patients was due to chronic HBV infection (13.3%). This attributed to high prevalence of HCV infection (75%) among Egyptian patients followed by hepatitis B (18%) and other causes of cirrhosis (7%) [28, 29]. Wang *et al.* [30] found a strong association between chronic hepatitis C and H. pylori infection.

This study showed that H. pylori infection was detected in 61(67.8%) cirrhotic patients. There was significant association between H. pylori infection and PHG (78.3% in patients with PHG versus 46.7% in patients without PHG) (P=0.0029). This agrees with studies done by Safwat *et al.*[28], Sathar *et al.* [29] and Eid *et al.* [31]. These studies suggested that gastric mucosa in cirrhosis might provide a hospitable environment for the colonization of H. pylori, due to severe hemorrhagic congestion and edema of the mucosa. In contrast to Pan *et al.* [32] who concluded that H. pylori infection are unlikely to be involved in the pathogenesis of PHG.

The current study showed significant association between H. pylori infection and severity of PHG (86.7% in sever PHG versus 46.7% in patients with no PHG) (p-value = 0.001). This agrees with studies done by Safwat *et al.* [28], Sathar *et al.* [29] and El-Nashar *et al.* [33]. They also noticed a positive correlation between H. pylori infection and severity of PHG. In contrast, Eid *et al.* [31] did not find a significant correlation between H. pylori infection and the severity of PHG or the severity of liver cirrhosis.

In this study, esophageal varices were detected in 34(37.8%) studied cirrhotic patients and there was a significant correlation between PHG and development of esophageal varices (48.33% in patients with PHG versus 16.7% in patients with no PHG) (P=0.005). Several studies reported a significant relation between PHG and presence and size of esophageal varices [23-26]. On the other hand, some studies showed that there was no significant relation between PHG and severity of esophageal varices and their size [32, 34].

CONCLUSION

The current study showed a significant relation between H. pylori infection and PHG in cirrhotic patients (P=0.0029) and between H. pylori infection and severity of PHG in cirrhotic patients (p-value=0.001). H. pylori might have a role in the pathogenesis of severe PHG. Eradication of H. pylori infection in cirrhotic patients with PHG will be beneficial to decrease complications of PHG. Periodic scanning of cirrhotic patients with PHG for possible H. pylori infection should be encouraged.

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الملخص العربي

العلاقة بين العدوى بالميكروب الحلزونى وشدة اعتلال المعدة نتيجة ارتفاع ضغط الدم في الوريد البابى في مرضى التليف الكبدى

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ملخص البحث:

الخلفية: إن تليف الكبد يمثل مشكلة صحية كبيرة في جميع أنحاء العالم ويرتبط بالعديد من المضاعفات. واعتلال المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى هي واحدة من المضاعفات الأكثر شيوعا، وفي بعض المرضى قد يؤدي إلى نزيف من الجهاز الهضمي. والعلاقة بين العدوى بالميكروب الحلزونى وشدة اعتلال المعدة نتيجة ارتفاع ضغط الدم في الوريد البابى لم تحدد بوضوح.

الهدف: والهدف من هذه الدراسة هو دراسة اى ارتباط محتمل بين العدوى بالميكروب الحلزونى واعتلال المعدة

نتيجة ارتفاع ضغط الدم في الوريد البابى وربط شدة اعتلال المعدة بالعدوى بالميكروب الحلزونى في مرضى التليف الكبدى.

الطرق: وشملت الدراسة مقارنة 90 مريضا من مرضى التليف الكبدى تم تصنيفهم إلى 3 مجموعات. المجموعة الأولى: شملت 30 مريضا من مرضى التليف الكبدى مطابقين فى العمر والجنس مع عدم وجود اعتلال فى المعدة نتيجة ارتفاع ضغط الدم في الوريد البابى. المجموعة الثانية: شملت 30 مريضا من مرضى التليف الكبدى مطابقين فى العمر والجنس مع وجود اعتلال بسيط فى المعدة نتيجة ارتفاع ضغط الدم في الوريد البابى. المجموعة الثالثة: شملت 30 مريضا من مرضى التليف الكبدى مطابقين فى العمر والجنس مع وجود اعتلال شديد فى المعدة نتيجة ارتفاع ضغط الدم في الوريد البابى. تم إجراء الفحص بالمنظار لتقييم وجود وشدة اعتلال المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى ولتشخيص العدوى بالميكروب الحلزونى.

النتائج: تم الكشف عن العدوى بالميكروب الحلزونى عن طريق فحص الأنسجة في 61 مريضا (67.8٪). وكانت العدوى بالميكروب الحلزونى أكثر انتشاراً بين المرضى المصابين باعتلال المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى (78.3%) من المرضى الذين لا يعانون من اعتلال فى المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى (64.7%)(القيمة الإحصائية=0.0029). وتم تشخيص العدوى بالميكروب الحلزونى فى 26 مريضا (86.7%)، 21 مريضا (70.0%)، 14 مريضا (46.7%) من المرضى الذين لا يعانون والذين يعانون من اعتلال بسيط والذين يعانون من اعتلال شديد فى المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى على التوالى. وكانت العدوى بالميكروب الحلزونى أكثر انتشارا في المرضى الذين يعانون من اعتلال شديد فى المعدة مقارنة بالمرضى الذين لا يعانون من اعتلال فى المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى (القيمة الإحصائية=0.001).

الإستنتاجات: وأظهرت الدراسة وجود علاقة كبيرة بين العدوى بالميكروب الحلزونى واعتلال المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى في مرضى التليف الكبدى وأنه قد يكون لها دور في التسبب في الاعتلال الشديد فى المعدة نتيجة ارتفاع ضغط الدم فى الوريد البابى.

الكلمات المفتاحية: التليف الكبدى، الميكروب الحلزونى، اعتلال المعدة نتيجة ارتفاع ضغط الدم في الوريد البابى.

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