Al-Azhar International Medical Journal

Volume 3 | Issue 8 Article 5

8-1-2022

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How to Cite This Article

Ibrahim, Randa SalahEldin; Abd Elmoety, Amr Aly; Baddour, Nahed; Salem, Perihan; and Metawea, Marwa (2022) "Spectrum of gastric Lesions in Portal Hypertension and Its Association with H. Pylori," *Al-Azhar International Medical Journal*: Vol. 3: Iss. 8, Article 5.
DOI: https://doi.org/10.21608/aimj.2022.134893.1932

OPEN AIMJ ORIGINAL ARTICLE

Internal Medicine

Spectrum of Gastric Lesions in Portal Hypertension and Its Association with H. Pylori

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Received *for publication May 04, 2022;* ***Accepted*** *August 28, 2022;* ***Published online*** *August 28, 2022.*

doi: 10.21608/aimj.2022.134893.1932

Citation: *Randa S. , Amr A. and Nahed M.et al. Spectrum of Gastric Lesions in Portal Hypertension and Its Association with H. Pylori. AIMJ. 2022; Vol.3-Issue8 : 27-31.*

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ABSTRACT

Background: Upper endoscopic screening of cirrhotic patients reveals several gastric findings besides portal hypertensive gastropathy (PHG). These findings contribute to more than 20% of upper gastro-intestinal bleeding. The role of Helicobacter pylori (H pylori) and other lesions associated with PHG is debatable.

Aim of the work: To describe the spectrum of lesions in the gastric mucosa in patients with portal hypertension, and to correlate the presence of H pylori with the risk of bleeding and serum gastrin level.

Patients and Methods: The study included fifty patients who met the inclusion criteria. Upper endoscopy was performed on all of them, and biopsies were taken from the stomach and were examined by histopathology.

Result: Among the 50 patients, 20% had non-variceal bleeding. White light endoscopy (WLE)'s most common documented finding was PHG. According to histopathology, each of the biopsies obtained yielded multiple histopathological findings H. pylori was found in 88% of the patients, followed by Gastric antral vascular ectasia (GAVE) (56%) then PHG and gastritis (54 %). Only H. pylori correlated with the presence of PHG.

Conclusion: Biopsy is recommended in cirrhotic patients during endoscopy to tailor treatment accordingly. In PHG, H. pylori treatment is recommended to decrease chances of bleeding.

Keywords: *Portal Hypertension; Portal Hypertensive Gastropathy; Histopathology; Endoscopy; H. pylori.*

Disclosure: *The authors have no financial interest to declare in relation to the content of this article. The Article Processing Charge was paid for by the authors.*

Authorship: *All authors have a substantial contribution to the article.*

INTRODUCTION

Portal hypertension is responsible to multiple problems associated with chronic liver disease like upper gastro-intestinal hemorrhage. In cirrhotic patients, variceal bleeding accounts for more than 70% of incidents of bleeding, and portal hypertensive gastropathy (PHG) is primarily responsible for persistent blood loss and anemia. [1,2]

Endoscopic screening of patients revealed a significant frequency of other pathologies besides varices and PHG like Gastric antral vascular ectasia (GAVE), peptic ulcer, erosions, gastritis and polyps. [3] These lesions represent another important source of bleeding. [4]

The emergence of gastro-duodenal lesions in portal hypertension is thought to be caused by several causes. Mucosal barrier weakening produced by portal hypertension-related mucosal hemodynamic alterations and exposure to noxious substances can be described as two key contributors. [5]

Helicobacter pylori (H. pylori), a spiral gram-negative bacteria that is resistant to gastric juice, infects roughly 50 -70 % of the world population, with the highest frequency in low-income areas. It was found to exacerbate local gastric inflammatory response through release of inflammatory cytokines resulting in a wide range of gastric pathologies. The link between H. pylori and the pathogenesis of PHG and risk of bleeding in cirrhotic patients is debatable.[6,7] Also, the acidity of the stomach in cirrhotic patient with PHG differs than normal healthy stomach, [8] which makes it more vulnerable to have H pylori.

Thus, our aim was to describe gastric mucosal lesions as a source of bleeding in patients with portal hypertension, and to study the correlation of H. pylori with the risk of bleeding and serum gastrin level.

PATIENTS AND METHODS

The study was approved by the Ethics Committee of Alexandria University Faculty of Medicine with number 0201288. An informed consent was taken from all patients, in accordance with the principles of the Declaration of Helsinki (revision of Edinburgh, 2000).

A cross-sectional observational study where all patients were recruited from Alexandria Main University Hospital, Hepatobiliary department between December 2019 and July 2020. 50 patients were included in the study. The inclusion criteria were patients with liver cirrhosis regardless the etiology, and with portal hypertension based on history, clinical examination, and laboratory investigations, and after exclusion of patients with prior history of proton pump inhibitor, antibiotic, or nonsteroidal drug intake within the past month. History and number of attacks of upper gastrointestinal bleeding was documented. Laboratory investigations included complete blood picture, complete liver profile and serum gastrin level were measured. Portal hypertension was confirmed by ultrasonography presenting any of signs of portal hypertension like splenomegaly, ascites, and dilated portal vein.

White light upper endoscopy (WLE) was done to all patients to assess for antral lesions in addition to assessment for the presence and grading of varices and PHG. Two biopsies were obtained from each of the body and antrum of the stomach, biopsies were obtained from the erosions and nearby mucosa. All biopsies were fixed in paraffin then prepared and stained with hematoxylin and eosin. H pylori was diagnosed upon histopathological examination. Then, the patients were divided into two groups: H pylori group and non-H pylori group.

Statistical analysis

Data were analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). Qualitative data were described using number and percent. Quantitative data were described using range, mean and standard deviation. Chi-square test for categorical variables, to compare between different categories. Fisher's Exact or Monte Carlo correction for chi-square when more than 20% of the cells have expected count less than 5. Mann Whitney test for abnormally distributed quantitative variables, to compare between two studied groups. Significance of the obtained results was judged at the 5% level.

RESULTS

Descriptive data

The mean age of the studied population was (57.60 ± 7.63) years. Thirty-two (64%) patients were males vs 18 (36%) females. Patients underwent for endoscopy for different purposes. Twenty-five patients (50%) had history of upper gastrointestinal bleeding. The clinical signs found were documented.

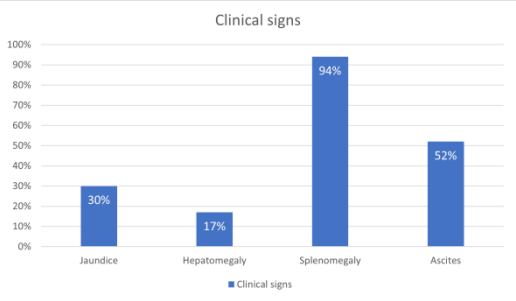

Fig. 1: Distribution of clinical findings found in the studied patients

Endoscopic data

During endoscopy, the highest documented finding was PHG (47/50); 48% (24/50) of them had mild PHG, 46% (23/50) had severe PHG. This was followed by multiple antral erosions in 46% (23/50) of patients, and 28% had single antral erosion.

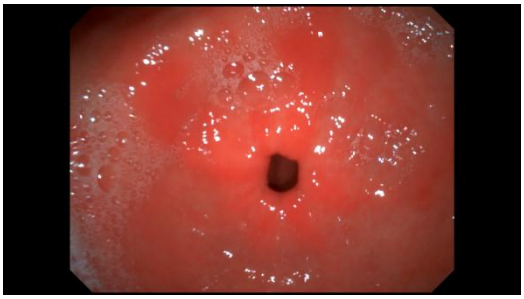

Fig. 2: Endoscopic view of the antrum of a patient with H pylori showing multiple lesions in the antrum.

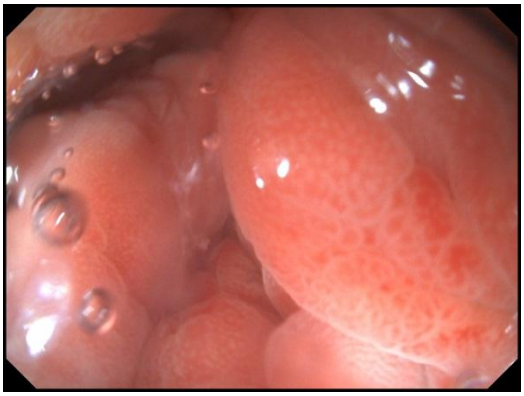

Fig. 3: Endoscopic view of the body of a patient with H pylori showing portal hypertensive gastropathy with signs of H pylori in the biopsy.

According to varices, 3/50 patients had grade 1 esophageal varices (EV), 8 patients had varices grade 2 and most of the patients (28/50) had grade 3 EV. There were no documented gastric varices in the studied population.

Histopathological findings

Eighty-eight percent of patients had H. pylori either as a sole pathology or in association with other findings. Other than H pylori, GAVE had the highest prevalence with 56% (28/50). This was followed by gastritis, 54% (27/50). Gastritis was furtherly

categorized into 28% (14/27) reactive gastritis, 18% (9/27) allergic gastritis and 4% eosinophilic gastritis. PHG was diagnosed in 52 % (27/50) of cases. Dysplasia, and vascular malformation each was reported in 4% (2/50) of the cases.

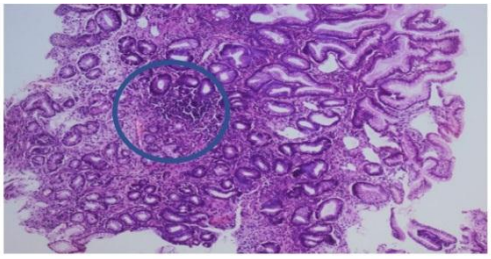

Fig. 4: A case with PHG, evidence of H pylori related gastritis was seen in antral biopsies. This was seen in the form of a band like infiltrate of chronic nonspecific inflammatory cells in the upper layers of the mucosa composed of lymphocytes, histiocytes, plasma cells and a few eosinophils

H. pylori

H. pylori group had lower hemoglobin level, platelet count and fasting gastrin level when compared to non-H. pylori patients, but this was not statistically significant, p 0.808, 0.676, and 0.296 respectively. Although the number of attacks of hematemesis was higher in H. Pylori group, it was statistically insignificant, p= 0.070.

Laboratory parameters	H.pylori		Test of sig.	P
	No (n = 6)	Yes (n = 44)		
Hematemesis				
Median (Min. –Max.)	0 (0 –1)	1 (0 –8)	U= 76.0	0.070
Hemoglobin				
Mean ±SD.	9.83 ±1.61	9.66 ±1.64	t= 0.244	0.808
Median (Min. –Max.)	9.45 (7.9 –12.6)	9.2 (7 –13.5)		
Platelets				
Mean ±SD.	113.33 ±37.2	114.64 ±61.70	U= 118.0	0.676
Median (Min. –Max.)	106 (62 –165)	99.5 (37 –363)		
WBCs				
Mean ±SD.	3.76 ±2.66	5.45 ±3.15	U = 67.0	0.052
Median (Min. –Max.)	2.79 (1.6 –8.98)	4.3 (1.53 –18.1)		
Gastrin				
Mean ±SD.	258.52 ±294.96	149.71 ±138.01	U = 97.0	0.296
Median (Min. –Max.)	151 (40.1 –846)	117 (29.5 –736)		

t: Student t-test **U:** Mann Whitney test **p:** p value for comparing between the studied categories

Table 1: The comparison between clinical and laboratory parameters of H pylori and non-H pylori group.

H. pylori was higher in PHG (42/47, 89.3%), when diagnosed by WLE, than non-PHG group. H. pylori correlated with the presence of PHG, p= 0.041. There was no correlation between grade of varices and H. pylori, p=1.00.

	H.pylori		c^2	P
	No (n = 6)	Yes (n = 44)		
Endoscopic findings				
Varices				
No EV	1 (16.7%)	10 (22.7%)	1.009	^{MC}p= 1.000
Grade 1	0 (0%)	3(6.8%)		
Grade 2	1 (16.7%)	7 (15.9%)		
Grade 3	4 (66.7%)	24 (54.5%)		
PHG				
No	1 (16.7%)	0 (0%)	6.420*	^{MC}p= 0.041*
Mild	1 (16.7%)	23 (54.8%)		
Severe	4 (66.7%)	19 (45.2%)		
Other#	0 (0%)	2 (4.5%)		
Pathological findings				
GAVE	4 (66.7%)	24 (54.5%)	0.315	^{FE}p=0.570
Dysplasia	1 (16.7%)	1 (2.3%)	2.849	^{FE}p=0.228
Allergic	2 (33.3%)	7 (15.9%)	1.086	^{FE}p=0.293
Reactive	1 (16.7%)	13 (29.5%)	0.434	^{FE}p=0.490
Eosinophilic	1 (16.7%)	3 (6.8%)	0.696	^{FE}p=0.452
Vascular Malformations	0 (0%)	2 (4.5%)	0.284	^{FE}p=1.000

Table 2: Correlation between H pylori and non-H pylori group of between different endoscopic and pathologic findings

H. pylori was found in 85.7 % (24/28) of cases of GAVE, when diagnosed by histopathology. As regards gastritis, 85.2% (23/27) of total cases of gastritis had H. pylori 14.8 % of them had allergic gastritis, 37% reactive and 7.4% eosinophilic gastritis. All cases of vascular malformation had H. pylori (2/2) and one case had dysplasia and H. pylori (1/2).

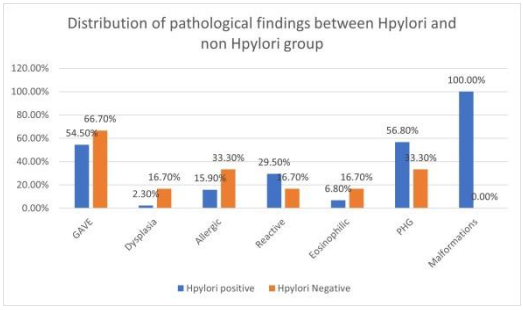

Fig. 5: shows the distribution of gastric lesions between H pylori and non-H pylori group

DISCUSSION

Hyperdynamic circulation associated with portal hypertension results in increased blood flow of the gastrointestinal tract. In addition, there is impairment of gastric mucosa microcirculation paralyzing local mucosal defense mechanisms. Eventually, the gastric mucosa becomes more prone to injury by noxious agents such as non-steroidal anti-inflammatory drugs and H. pylori, ending up with erosions, ulcers, and bleeding. [5,9]

In our study, we reported the spectrum of lesions of gastric mucosa by endoscopy and histopathology in

patients with portal hypertension and correlate these findings to upper gastrointestinal (GI) bleeding. WLE is the diagnostic modality for gastric mucosal lesions. But, in the presence of portal hypertension, many pathologies can affect the gastric mucosa. Their differential diagnosis is wide, and they could have the same endoscopic features. PHG was the most frequent finding, being found in 94% of cases by endoscopy. These results were consistent with the results demonstrated by several studies, that reported prevalence of PHG > 90%, [9,10] and more than reported by Tiwari et al. [11] This was followed by 74 % antral erosions, on histopathology basis, there was overlap between the different findings, giving up to three pathologies in the same patient.

We reported 88% of cases of H. pylori by histopathology. Chaudhary et al, reported 70.4 % of cases of H. pylori by rapid urease test in patients with portal hypertension, while Voulgaris et al, and Puri et al reported 54% and 67% respectively. [7,12,13] The high prevalence reported by our study could be explained by the high prevalence of H. pylori in Egypt. [14] The variation in reported prevalence of H. pylori by the different studies may be also related to the differences in the used diagnostic modalities as well as the differences between the socio-economic status of studied populations.

Non variceal bleeding was reported in 20 % of cases (10/50) in our study. All these cases had H pylori, 8/10 had GAVE and 7/10 of cases had gastritis. This demonstrates that there is no single player responsible for upper GI bleeding.

In literature, the relation of PHG and H. pylori is controversial. In our study, we demonstrated high prevalence of H. pylori in PHG group 89.4% vs 66.7% in non PHG by endoscopy. Furthermore, a positive correlation was found between H. pylori and the presence of PHG, p=0.041. Similar results were reported by *Eldessouky et al.* [15] PHG is associated with decreased protective prostaglandins, high pH, due to reduced acid secretion, and thinner mucous layer than the normal gastric mucosa. All these factors weaken the gastric mucosa rendering it more susceptible to H. pylori infection. [16] In contrast, Puri et al. did not demonstrate any relation between H. pylori and PHG severity. [13]

We reported also, low fasting gastrin level in H. pylori patients (149.71 ± 138.01 ng/dl) vs (258.52 ± 294.96 ng/dl) in non-H. pylori patients. On the contrary, Liu et al reported high levels of gastrin with H. pylori infection. [17] This could be explained by the difference in the studied population, as the patients in their study were non cirrhotic.

Although, there was no correlation between H. pylori and upper GI bleeding, the number of attacks were higher in H. pylori group than in non-H. pylori. There was no correlation between H. pylori infection and esophageal varices, p=1.00. Jun et al demonstrated that there was no correlation between H pylori infection and bleeding from esophageal varices. [18] Hypoacidity caused by gastric mucosal atrophy associated with H. pylori could have a protective effect on variceal bleeding. [19]

CONCLUSION

More than one factor contributes to mucosal changes seen on endoscopy in cirrhotic patients. Each of these factors should be tackled while managing these patients to lower the chances of upper GI bleeding and deterioration of the liver condition. As some of the patients are proven to have dysplastic changes, gastric biopsy during endoscopy is still recommended. The presence H pylori is correlated with the presence of PHG.

Finally, the limitation of our study was the small sample size due to the COVID19 pandemic that prevent us from recruiting more patients. So, further larger studies are needed to study the effect of portal hypertension changes on H. pylori infection and its virulence. We also recommend the assessment of these different lesions found by virtual chromoendoscopy.

Conflict of interest : none

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