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# Helicobacter pylori and portal hypertensive gastropathy diagnosed by narrow band imaging (NBI): The role of its eradication

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**Abstract:**

***Background and Aim**: Portal hypertensive gastropathy (PHG) is a common endoscopic finding in patients with portal hypertension. The pathophysiology of this condition is not obviously understood. Although portal hypertension remains the crucial trigger for the development of PHG, other factors could be responsible for the progression of this condition. The aim of this study was to evaluate the prevalence of helicobacter pylori (H. pylori) infection among cirrhotic patients with PHG diagnosed by using narrow band imaging (NBI) system and to asses the role of its eradication in improvement of PHG.*

***Patients and Methods**: This study included 120 consecutive patients with HCV-related liver cirrhosis. All patients were subjected to an upper gastrointestinal endoscopy using NBI technique and histopathologic testing of H. pylori. The diagnosis and the severity of PHG were assesed on doing endoscopy. Child-Pugh score was*

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*calculated to assess the severity of liver cirrhosis. Concomitant nonbismuth quadruple eradication therapy was given to H. pylori postive patients with PHG. One month later after treatment, a second endoscopic assesment of PHG was done to those with confirmed eradicated H. pylori infection patients (using H. pylori stool antigen test).*

***Results:*** *PHG was detected in 78 patients with overall prevalence 65%. Out of those 78 patients, 36 had mild PHG (46.15 %) and 42 had severe PHG (53.85 %). A total of 90 patients out of 120 confirmed to have H. pylori infection with overall prevalence of 75 %. The prevalence of H. pylori was higher among those with severe PHG (36 out of 42; 85.7%) rather than those with mild PHG (24 out of 36; 66.7%) and without PHG (30 out of 42; 71.4%) (p=0.12). On re-assesment of H. pylori associated PHG cured cases (n=42) by a second endoscopic examination; the number of patients with mild PHG decreased from 18 to 12 patients, as 6 of them became without PHG, however none of patients with severe PHG showed any improvement (n=24) (p=0.014)*

***Conclusion:*** *There may be a minor role for H. pylori infection in PHG due to HCV-related liver cirrhosis and eradication of H. pylori may improve mild but not severe PHG.*

**Key words**: Portal hypertensive gastropathy, Narrow band imaging, helicobacter pylori, liver cirrhosis

## INTRODUCTION:

Patients with liver cirrhosis are at high risk of gastrointestinal bleeding, commonly from gastroesophageal varices. However, portal hypertensive gastropathy (PHG) is another cause of bleeding in those patients. It leads to chronic occult blood loss [1].

Portal hypertensive gastropathy occurs as a complication of cirrhotic or non-cirrhotic portal hypertension. PHG may present with acute massive or insidious bleeding. Endoscopically, abnormality of the gastric mucosa is classically

described as a mosaic-like pattern that resembles the skin of a snake, with or without red spots [2]. Moreover, it seemed that the color of the mucosa was due to the degree of capillary dilatation, while the degree of red spots was due to the amount of intramucosal hemorrhage [3].

The narrow band imaging (NBI) is an endoscopic imaging technique for the enhanced visualization of mucosal microscopic structure and capillaries of the superficial mucosal layer, depending on changing the spectral features of the illumination. NBI obtains its images by using narrower bands of red, blue and green filters (R/B/G), which are different from conventional red, blue and green filters [4]. The depth of penetration into the mucosa depends on the wave length used superficial for blue band and deep for red band and intermediate for green band [5]. Thus, lesions could be identified by changes in color and irregularity of mucosal surface [6].

Combining NBI with magnifying system allow very clear images of the capillaries of the mucosal surface and microvascular architecture of the gastric mucosa in patients with liver cirrhosis [7].

Infection by helicobacter pylori (H. pylori) is highly prevalent, especially in low socioeconomic developing countries [8]. It is responsible for lesions like gastroduodenal erosions and ulcers. In patients with liver cirrhosis, their prevalence is controversial, as well as the association with PHG [9].

The endoscopic findings of an H. pylori affected stomach include erythema, erosions, antral nodularity, thickening of gastric mucosal folds, and visible submucosal vessels. However, these findings have low sensitivity and specificity for diagnosis [10]. Recently, Taiwanese endoscopists performed a study using close-up observation between the endoscopic tip and the gastric mucosa and found the "mosaic pattern" in the corpus mucosa. This method is a more sensitive and specific way to

determine H. pylori infection status [11]. They classified gastric mucosal patterns into two categories [normal regular arrangement of collecting venules (RAC) and abnormal mosaic pattern]. However, the classification was insufficient to predict all H. pylori infections. Another used four categories: a normal RAC and three abnormal patterns including mosaic-like appearance (type A), diffuse homogenous redness (type B), and untypical pattern (type C) to predict a H. pylori-infected stomach [12].

Conventional white light endoscopy correlates poorly with histopathological findings of H. pylori-induced gastritis [13]. Some studies using magnifying endoscopy have shown that endoscopic features are associated with histopathological findings related to H. pylori infection [4]. Successful eradication of H. pylori dramatically changes the histopathological findings of gastritis. Recently, changes of magnifying endoscopic features with NBI were investigated during H. pylori eradication [14].

Thus, studying the association of H. pylori with PHG could be useful for better understanding of the pathogenesis of PHG, then eradication of H. pylori should be beneficial in the management of PHG.

We performed this study to verify the prevalence of H. pylori infection among HCV- cirrhotic patients with PHG diagnosed by using the magnifying NBI system and evalute the role of its eradication in improvement of PHG.

## PATIENTS AND METHODS:

One hundred and twenty (120) consecutive patients with HCV related liver cirrhosis were enrolled in the study, recruited from those attending the Endoscopy unit of Ain Shams University hospital. The study was performed in the period between June

2015 and March 2016. An informed consent was obtained from each patient.

Patients with hepatocellular carcinoma, gastric surgery, peptic ulcer or gastric malignancy, recent variceal bleeding (within 2 weeks), and patients using beta blockers, nitrates, nonsteroidal anti-inflammatory drugs, proton pump inhibitors, antibiotics (up to 1 month) or a prior H. pylori eradication therapy were excluded from the study.

A thorough medical history and full clinical examination were applied in all participants. A complete blood count, liver and renal functions were performed for all. An abdominal ultrasound (Toshiba real-time scanner instrument with a 3.5 MHz convex transducer) was done. The diagnosis of liver cirrhosis was based on clinical, biochemichal and radiological findings. HCV antibodies were detected using Microparticle Enzyme Immunoassay (AxSYM, third generation assay, Abbott Laboratories, IL, USA). The severity of liver disease was assessed using Child-pugh classification.

All patients were endoscopically evaluated by the NBI technique using Pentax EPK-i video processor which provides a spectacular image with an unrivaled of 1.25 megapixels which is approximately 50% higher than any other endoscopy using a sophisticated software that enhances the image area providing detailed imaging of mucosa topography and vascularity. Regarding endoscopic NBI picture of PHG, Mosaic like pattern and reddening mucosa (equivalent to mild PHG by Baveno classification) [15] appeared as extended and swollen gastric pits with various degrees of dilated and convoluted capillaries surrounding the gastric pits **(Figure 1)** , while intra-mucosal hemorrhage around capillaries (equivalent to severe PHG) **(Figure 2)** [16]. OV were classified as small (small straight), medium (tortuous occupying less than 1/3 the lumen) , and large (coil shaped occupying more than 1/3 the lumen) [17].

Biopsy specimens were taken from the gastric antrum, body and incisura (2-1-2). Paraffin embedded sections were prepared from the specimens. The specimens were then dewaxed and taken to water and then incubated in 2% Giemsa solution in distilled water for 30 minutes at room temperature. After rinsing in tap water the sections were quickly dehydrated through ethanol solutions before being cleared with xylene and mounted in DPX (a mixture of distyrene, a plasticizer, dissolved in toluene-xylene). Under light microscopy, curved, bent, pole-like, spiral, and fusiform bacteria were accepted as H. pylori.

Patients with PHG who were positive for H. pylori received concomitant nonbismuth quadruple eradication therapy (esmoprazole 40 mg BID, amoxicillin 1000 mg BID, metronidazole 500 mg BID, and clarithromycin 500 mg BID for 14 days). Results of successful eradication were diagnosed by H. pylori stool antigen (HPSA) using rapid strip HPSA test (a rapid immunoassay test) done 4 weeks after receiving eradication therapy and they were off PPI and antibiotics . A second endoscopic examination was carried out for those with negative HPSA to re-assess PHG .

**Statistical analysis:** The collected data was revised, coded, tabulated and introduced to a PC using Statistical package for Social Science **(**IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.**).** Quantitative parametric variables are expressed as mean and SD. Qualitative variables are expressed as frequencies and percent. **Student t** Test was used to compare a continuous variable between two study groups. **Chi-square** was used to examine the relationship between Categorical variables. **Wilcoxon signed rank** test was used to the statistical significance of the difference of PHG before and after h.pylori treatment. P-value < 0.05 was considered statistically significant.

## RESULTS:

The ages of the studied patients ranged between 39-68 years with mean of 54 ± 7.85 years. 78 subjects (65 %) were males, while 42 (35%) were females. 42 (35%) were smokers. Regarding the presence of oesophageal varices, 36 (30%) had no varices, 36 (30%) had small varices, 18 (15%) had medium varices, and 30 (25%) had large varices. According to Child Pugh classification, 12 patients were classified as Child A (10%), 66 as Child B (55%) and 42 as Child C (35%).
Laboratory data of all the studied patients is shown in table **(1)**.

On doing upper GI endoscopy, PHG was found in 78 patients (65%). Out of those 78 patients, 36 had mild PHG (46.15%) and 42 had severe PHG (53.85%). Comparison between patients with PHG (n=78) and those without PHG (n=42) as regards clinical data is shown in table **(2)**. Also, comparison between the 2 groups as regards laboratory data is shown in table **(3)**. There was no statistical significant difference as regards sex, smoking, Child Pugh classification, variceal presence and grading ($P$>0.05). The mean age of patients with PHG was 52.3 ± 6.2 years compared to 54.8 ± 8.5 years in those without PHG (p= 0.001). comparison of clinical data between patients with mild PHG and those with severe PHG is summarized in table **(4)**. There was a statistical significant difference between patients with mild PHG (n=36) and patients with severe PHG (n= 42) regading age, sex, smoking and Child Pugh classification (p<0.05). Yet, no association between O.V and the severity of PHG could be noticed (p>0.05)

H. pylori infection was confirmed in 90 patients out of 120 with overall prevalence of 75%, with a higher prevalence among males than females (92.3%vs. 42.9%; p=0.0001). Besides, a significant difference between the 2 groups was

noticed as regards variceal presence and grading (p=0.004); however, no significant difference was detected regarding age, smoking, Child Pugh classification (p>0.05) **(Table 5).** Comparison between patients with and without H. pylori infection is shown in table **(6).**

A total of 60 patients out of 78 with PHG had H. pylori infection (76.9%) **(Table 7)**; including 24 with mild PHG, and 36 with severe PHG . The prevalence of H. pylori was higher among those with severe PHG (36 out of 42; 85.7%) rather than those with mild PHG (24 out of 36; 66.7%) and without PHG (30 out of 42; 71.4%) (p=0.12). **(Table 8, Figure 3)**

Eradication of H. pylori was confirmed in 42 patients out of 60 with PHG, 4 weeks after treatment, with a successful eradication rate 70%; including 18 patients with mild PHG and 24 with severe PHG. On re-evaluation of H.pylori associated PHG cured cases (n=42) by a second endoscopic examination; none of patients with severe PHG showed any improvement (n=24), however the number of patients with mild PHG decreased from 18 to 12 patients, as 6 of them became without PHG (p=0.014) **(Table 9 , Figure 4).**

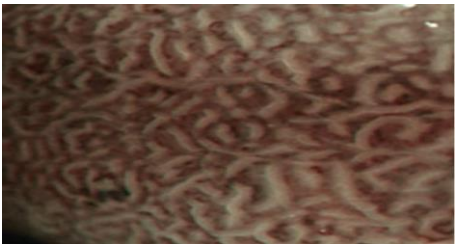

**Figure (1): Picture of mild PHG by NBI**

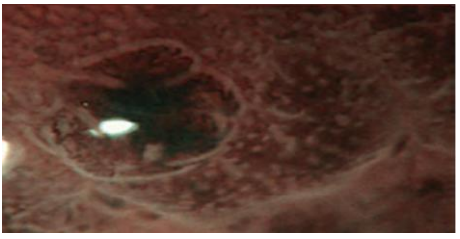

**Figure (2): Picture of severe PHG by NBI**

**Table (1): Laboratory data among all the studied patients**

| Laboratory parameter | Mean | ±SD | Minimum | Maximum |
|---|---|---|---|---|
| **WBCS (x $10^3$/$mm^3$)** | 8.05 | 4.95 | 2.7 | 20 |
| **PLT(x $10^3$/$mm^3$)** | 117.1 | 58.33 | 44 | 230 |
| **HB (gm/dl)** | 9.95 | 1.36 | 8 | 12 |
| **ALT(IU/L)** | 31.75 | 14.37 | 12 | 81 |
| **AST(IU/L)** | 62.2 | 38.23 | 26 | 200 |
| **Bilirubin (mg/dl)** | 1.92 | 0.91 | 0.5 | 3.9 |
| **Albumin (gm/dl)** | 2.3 | 0.46 | 1.7 | 3.5 |
| **Protein (gm/dl)** | 6.29 | 0.64 | 5 | 8 |
| **INR** | 1.58 | 0.35 | 1.1 | 2.5 |
| **Creatinine (mg/dl)** | 1.55 | 1 | 0.5 | 4 |

**Table (2)**: **Comparison between patients with and without PHG regarding clinical data**

| Parameter | | PHG before ttt | | | | P |
|---|---|---|---|---|---|---|
| | | Patients without PHG (n= 42) | | Patients with PHG (n= 78) | | |
| **Age** | | 52.3 ± 6.2 | | 54.8 ± 8.5 | | 0.001 |
| **Sex** | *Male* | 24 | 30.8% | 54 | 69.2% | 0.185 |
| | *Female* | 18 | 42.9% | 24 | 57.1% | |
| **Smoking** | *Yes* | 18 | 42.9% | 24 | 57.1% | 0.185 |
| | *No* | 24 | 30.8% | 54 | 69.2% | |
| **Child classification** | *A* | 6 | 50% | 6 | 50% | 0.131 |
| | *B* | 18 | 27.3% | 48 | 72.7% | |
| | *C* | 18 | 42.9% | 24 | 57.1% | |
| **Varices** | *No* | 18 | 50% | 18 | 50% | 0.086 |
| | *Small* | 12 | 33.3% | 24 | 66.7% | |
| | *Medium* | 6 | 33.3% | 12 | 66.7% | |
| | *Large* | 6 | 20% | 24 | 80% | |

**Table (3)**: **Comparison between patients with and without PHG regarding laboratory data**

| | PHG before ttt | | | | P |
|---|---|---|---|---|---|
| | Patients without PHG (n= 42) | | Patients with PHG (n= 78) | | |
| | Mean | ±SD | Mean | ±SD | |
| **WBCs (x $10^3$/$mm^3$)** | 9 | 5.2 | 6.2 | 3.8 | 0.001 |
| **PLT(x $10^3$/$mm^3$)** | 136.5 | 61.3 | 81 | 27.7 | 0.001 |
| **Hb (gm/dl)** | 10.2 | 1.2 | 9.6 | 1.5 | 0.036 |
| **ALT(IU/L)** | 34.4 | 10.4 | 30.3 | 16 | 0.135 |
| **AST(IU/L)** | 83.1 | 53.8 | 50.9 | 18.7 | < 0.001 |
| **Bilirubin (mg/dl)** | 1.7 | 0.9 | 2.4 | 0.7 | < 0.001 |
| **Albumin (gm/dl)** | 2.4 | 0.5 | 2.1 | .*40* | 0.001 |

| | | | | | |
|---|---|---|---|---|---|
| **T. Proteins (gm/dl)** | 6.1 | 0.4 | 6.4 | 0.7 | 0.031 |
| **INR** | 1.7 | 0.4 | 1.5 | 0.3 | 0.001 |
| **Creatinine (mg/dl)** | 1.2 | 0.5 | 1.7 | 1.2 | 0.001 |

**Table (4): Comparison between patients with mild and severe PHG as regards clinical data**

| | | PHG before ttt | | | | |
|---|---|---|---|---|---|---|
| | | **Mild (n=36)** | | **Severe (n=42)** | | **P** |
| **Age (years)** | | 58.8 ± 8.5 | | 51.4 ± 7 | | 0.001 |
| **Sex** | Male | 18 | 23.1% | 36 | 46.2% | 0.001 |
| | Female | 18 | 42.9% | 6 | 14.3% | |
| **Smoking** | Yes | 6 | 14.3% | 18 | 42.9% | 0.012 |
| | No | 30 | 38.5% | 24 | 30.8% | |
| **Child classification** | A | 6 | 50% | 0 | 0% | 0.001 |
| | B | 12 | 18.2% | 36 | 54.5% | |
| | C | 18 | 42.9% | 6 | 14.3% | |
| **Varices** | No | 6 | 16.7% | 12 | 33.3% | 0.671 |
| | Small | 12 | 33.3% | 12 | 33.3% | |
| | Medium | 6 | 33.3% | 6 | 33.3% | |
| | Large | 12 | 40% | 12 | 40% | |

**Table (5): Relationship between H. pylori and clinical data**

| | | H. pylori | | | | |
|---|---|---|---|---|---|---|
| | | **Negative (n=30)** | | **Positive (n=90)** | | **P** |
| **Age** | | 55.4 ± 6.5 | | 53.5 ± 8.2 | | 0.244 |
| **Sex** | *Male* | 6 | 7.7% | 72 | 92.3% | 0.0001 |
| | *Female* | 24 | 57.1% | 18 | 42.9% | |
| **Smoking** | *Yes* | 12 | 28.6% | 30 | 71.4% | 0.507 |
| | *No* | 18 | 23.1% | 60 | 76.9% | |
| **DM** | *Yes* | 12 | 28.6% | 30 | 71.4% | 0.507 |
| | *No* | 18 | 23.1% | 60 | 76.9% | |
| **HTN** | *Yes* | 12 | 33.3% | 24 | 66.7% | 0.168 |
| | *No* | 18 | 21.4% | 66 | 78.6% | |
| **Child classification** | *A* | 0 | 0% | 12 | 100.0% | 0.107 |
| | *B* | 18 | 27.3% | 48 | 72.7% | |
| | *C* | 12 | 28.6% | 30 | 71.4% | |
| **Varices** | *No* | 12 | 33.3% | 24 | 66.7% | 0.004 |
| | *Small* | 12 | 33.3% | 24 | 66.7% | |
| | *Medium* | 6 | 33.3% | 12 | 66.7% | |

**Table (6): Relationship between H. pylori and laboratory data**

| | H. pylori | | | | P |
|---|---|---|---|---|---|
| | Negative (n=30) | | Positive (n=90) | | |
| | Mean | ±SD | Mean | ±SD | |
| WBCs (x $10^3$/$mm^3$) | 6.6 | 3.3 | 8.5 | 5.3 | 0.02 |
| PLT (x $10^3$/$mm^3$) | 102 | 58.6 | 122.1 | 57.7 | 0.1 |
| Hb (gm/dl) | 10.2 | 1 | 9.9 | 1.5 | 0.17 |
| ALT (U/L) | 31.0 | 11.5 | 32 | 15.3 | 0.74 |
| AST (U/L) | 65.4 | 25.7 | 61.1 | 41.6 | 0.6 |
| Bilirubin (mg/dl) | 2.9 | 0.8 | 1.6 | 0.7 | 0.001 |
| Albumin (gm/dl) | 2.3 | 0.6 | 2.3 | 0.4 | 0.81 |
| T. Proteins (gm/dl) | 6.6 | 0.8 | 6.2 | 0.6 | 0.003 |
| INR | 1.6 | 0.1 | 1.6 | 0.4 | 0.54 |
| Creatinine (mg/dl) | 1.8 | 0.8 | 1.5 | 1.1 | 0.17 |

**Table (7): Relationship between H. pylori and PHG**

| | | PHG | | | | P |
|---|---|---|---|---|---|---|
| | | Negative (n=42) | | Positive (n=78) | | |
| | | N | % | n | % | |
| H. pylori | Negative | 12 | 28.6% | 18 | 23.1% | 0.507 |
| | Positive | 30 | 71.4% | 60 | 76.9% | |

**Table (8): Relation between PHG degree and H.pylori at baseline**

| | | PHG | | | | | | P |
|---|---|---|---|---|---|---|---|---|
| | | No PHG (n=42) | | Mild PHG (n=36) | | Severe PHG (n=42) | | |
| | | n | % | n | % | n | % | |
| H. pylori | Negative | 12 | 28.6% | 12 | 33.3% | 6 | 14.3% | 0.123 |
| | Positive | 30 | 71.4% | 24 | 66.7% | 36 | 85.7% | |

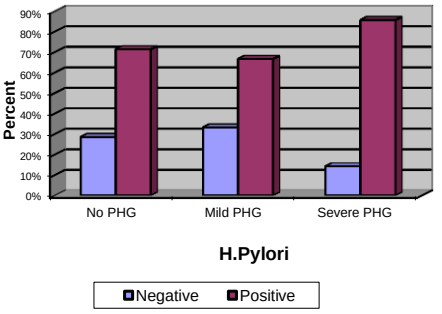


**Figure (3): Relation between PHG degree and H.pylori at baseline**

**Table (9): Comparison between PHG grade before and after 2nd endoscope among H.pylori cured cases (n=42)**

| | Before 2nd endoscopy | | After 2nd Endoscopy | | P |
|---|---|---|---|---|---|
| | n | % | n | % | |
| **No PHG** | 0 | 0% | 6 | 14.3% | 0.014 |
| **Mild PHG** | 18 | 42.9% | 12 | 28.6% | |
| **Severe PHG** | 24 | 57.1% | 24 | 57.1% | |

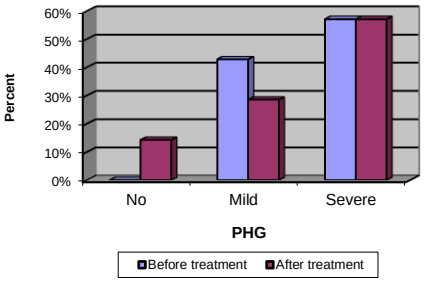


**Figure (4): Comparison between PHG grade before and after 2nd endoscope among H.pylori cured cases**

## DISCUSSION:

Portal hypertension characterized by venous and capillary ectasia in the mucosa and submucosa due to hemodynamic changes that lead to congestion with a change in gastric mucosal blood flow, which in turn leads to activation of cytokines, growth factors, and hormones that perpetuate this hyperdynamic gastric circulation [18]. PHG occurs as a complication of cirrhotic as well as non cirrhotic portal hypertension.

The routine method for the diagnosis of PHG is white light endoscopy (WLE). Recently, NBI can diagnose the subtle and flat mucosal GI lesions which are often missed or remain uncharacterized on WLE. Since the sub-epithelial capillaries of stomach have minimum diameter of 8 µm, combining

magnification endoscopy with NBI has been studied for detailed examination of capillary patterns in stomach [19].

PHG was found in 78 patients out of 120 patients with liver cirrhosis included in the present study (65%). In previuos studies, the prevalence of PHG in cirrhotic patients varies between 20% and 98% [20]. This great variation may be attributed to the study of different populations and variable patient selection, in addition to different interpretation of endoscopic lesions.

Only the age of the patients that showed a significant difference regarding the presence of PHG (p=0.001); besides, a relatively younger age was noticed in those with severe PHG (p=0.001). Male gender dominanted in severe PHG (p=0.001). Other clinical parameters, including Child Pugh class and the presence or the severity of varices, showed insignificant relation with the presence of PHG. Some previous studies have demonstrated a higher prevalence of PHG in patients with advanced liver disease, esophageal varices [21], however, these results may be inconclusive and not reaching significance. Our findings correlate well with those of Pan et al. [22] who found that the presence of PHG is not affected either by the severity of liver disease or by the presence or the grade of varices.

The pathogenesis of PHG is still not fully understood. The pre-eminent pathogenetic factor seems to be an increase of the portal pressure. Several investigators have evaluated the effect of H. pylori on PHG with controversial results. To fill the knowledge gap in this area, we aimed at exploring the prevalence of H. pylori in patients with PHG that could help identifying the pathogenesis of PHG.

In the present study, a high prevalence of H. pylori was reported among all the studied patients (90 patients out of 120) with overall prevalence of 75%, a figure that is higher than that of Abbas et al. (62.1%) [23]. In a study from south India, 16 of the 37 patients with cirrhosis were positive for H. pylori

(43.24%) [24]. Also, we noticed male predominance among H. pylori patients (92.3%vs. 42.9%; p=0.0001).

Furthermore, H. pylori infection was more prevalent among patients with PHG but the difference did not reach significance (76.9% vs. 71.4%; p=0.507); thus our study concluded that the role of H. pylori in the pathogenesis of PHG is minor. Numerous studies have demonstrated that H. pylori infection is not associated with PHG [25-28]. Contrariwise, Sathar et al. [29] reported an association between H. pylori infection and PHG in cirrhotic patients, but some limitations were noticed in their study, including low specificity and low sensitivity of H. pylori serology in cirrhotic patients [30, 31], potential selection bias, and underreporting of H. pylori seroprevalence when considering that the study was performed in India where H. pylori prevalence is extremely high in the general population [32].

On discriminating PHG into mild and severe; the prevalence of H. pylori was higher among patients with severe PHG (36/42, 85.7%) rather than mild PHG (24/36, 66.7%); a finding that goes with Sathar et al. [29] where seroprevalence rate in cirrhotics with severe PHG (19/24, 79.2%) compared to those with mild PHG (12/46, 26.1%). But, this finding was claimed as its prevalence in literature was found to range widely from 23% to 79% and from 22% to 81% in cirrhotics with mild and severe PHG, respectively [33]. Consequently, this finding is not reliable, and a selection bias cannot be ruled out. In addition, in a previous study, the H. pylori status was 52%, 22%, and 0% in patients with mild, moderate, and severe gastropathy, respectively, indicating an inverse relatioinship of severity of PHG with H. pylori colonization and this explained by severe congestive gastropathy make the gastric mucosa not suitable for colonization of H. pylori and this may be related to decreased synthesis of urea by the unhealthy gastric mucosa[24].

An explanation for increased prevalence of H. pylori among severe PHG in our study that some factors like increased inducible nitric oxide synthase expression resulting in high reactive oxygen species, impairment of gastric mucosal defence due to PHG in cirrhotic patients might increase virulence of H. pylori to produce a synergistic effect between H. pylori and PHG. Furthermore, colonization with H. pylori strains result in gastric inflammatory response, including interleukin-8, tumor necrosis factor-α, which may be associated with the sequence of events leading to PHG [29].

Eradication of H. pylori was confirmed in 42 patients out of 60 with PHG, 4 weeks after treatment, with a successful eradication rate 70%. On re-evaluation of H. pylori associated PHG cured cases by a second endoscopic examination; none of patients with severe PHG showed any improvement, however the number of patients with mild PHG decreased from 18 to 12 patients, as 6 of them became without PHG with significant difference regarding mild PHG improvement before and after H.pylori eradication ($p= 0.014$).

There was a diverse of contradiction about the role of H. pylori eradication in cirrhotic patients. In past, many studies reported that no need for its routine eradication in cirrhotic patients [34-36]; while Sathar and co-worker [29] found that there was significant association between H. pylori infection and PHG in cirrhotic patients which is also related to severity of PHG. Thus, H. pylori needs to be eradicated in cirrhotic patients with PHG.

In conclusion, there may be a minor role for H. pylori in the pathogenesis of PHG in cirrhotic patients. A significant difference regarding mild PHG improvement before and after H. pylori eradication was also noticed. Further prospective studies on large number of patients are warranted to show whether routine eradication of H. pylori may benefit the

treatment of PHG especially severe portal gastropathy in cirrhosis.

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