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Original Article

# *Helicobacter pylori* Infection in Patients with Portal Hypertensive Gastropathy Owing to Liver Cirrhosis: Prevalence and Relation with Severity of Gastropathy

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ABSTRACT

**Background and Aim:** *Helicobacter pylori* is a major human pathogen. Its role in the pathogenesis of portal hypertensive gastropathy (PHG) is debated. The aim of this study was to evaluate the prevalence of this infection in patients with portal hypertension due to liver cirrhosis and its relation with severity of gastropathy. **Patients and Methods:** Sixty consecutive patients with liver cirrhosis were enrolled in the study. All patients were subjected to an upper gastrointestinal endoscopy (UGIE), and rapid urease testing for *H. pylori* was performed. The diagnosis and severity of PHG was evaluated on UGIE. Child-Turcotte-Pugh (CTP) and model for end-stage liver disease (MELD) scores were calculated to assess the severity of liver cirrhosis. **Results:** *H. pylori* infection was reported in 33 patients with overall prevalence 55%. The presence of *H. pylori* was observed in 26 (67%) cirrhotic patients with PHG compared to 7 (33%) cirrhotic patients without PHG. The risk estimate showed a significant association between *H. pylori* and PHG in cirrhotic patients ($P$ = 0.0133, odds ratio [OR]: 4.00, 95% confidence interval [CI]: 1.298–12.325). Out of the 26 patients with PHG and *H.* pylori infection, 17 had severe PHG (65.3%) and 9 had mild PHG (34.6%) whereas 4 patients had severe PHG (30.8%) and 9 had mild PHG (69.2%%) in the group of *H. pylori*-negative patients. The difference was statistically significant ($P$ = 0.04, OR: 4.25, 95% CI: 1.0188–17.729). Of the 39 patients with PHG, 21 (53.85%) had severe PHG and 18 (46.15%) had mild PHG. No significant relation was found between *H. pylori* infection and severity of liver cirrhosis as regards CTP score ($P$ = 0.76) and MELD score ($P$ = 0.56). **Conclusion:** Our results showed a significant association between *H. pylori* infection and the occurrence and also the severity of gastropathy in patients with liver cirrhosis. Yet, the severity of liver cirrhosis itself did not correlate with *H. pylori* or the severity of gastropathy.

**KEYWORDS:** *Helicobacter pylori, liver cirrhosis, portal hypertensive gastropathy*

## INTRODUCTION

Cirrhosis is a major health problem with high incidence and prevalence worldwide. It is associated with alterations in gastrointestinal mucosa, with increased risk for peptic ulcer disease.[1]

Portal hypertensive gastropathy (PHG) is the change in the gastric mucosa of patients with portal hypertension, defined as the presence of mucosal friability and dilated blood vessels in the mucosal surface.[2] Endoscopically, gastric mucosa is classically described as a mosaic-like pattern that resembles snake skin, with or without red

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spots.[3] Histopathologic features include vascular ectasia of the mucosal and submucosal veins and capillaries.[4] The pathogenesis of PHG is not completely understood and is likely to be complicated. However, evidence suggests that portal hypertension is a key factor, where elevated portal pressure can induce changes of local hemodynamics, thus causing congestion in the stomach. These changes may then activate cytokines and growth factors, such as tumor necrosis factor-α (TNF-α), which activate endothelial nitric oxide synthase and endothelin 1. Nitric oxide induces hyperdynamic circulation and peroxynitrite overproduction which, together with endothelin overproduction, may cause damage of gastric mucosa. When combined with the characteristics of impaired mucosal defense and healing, these factors may together produce PHG in patients with portal hypertension.[5]

The prevalence of PHG varies widely; frequencies from 4% to 98% have been recorded in studies of patients with portal hypertension.[6] The prevalence of PHG is shown to be closely associated with the severity of cirrhosis assessed by Child-Turcotte-Pugh (CTP) classification, being more common in Child-Pugh C than in Child-Pugh A patients.[7]

*Helicobacter pylori* is a major etiological factor of peptic ulcer disease, which is frequently encountered in patients with liver cirrhosis.[8] Colonization of the gastric mucosa by *H. Pylori* might have an indirect role in PHG as colonization is, at least theoretically, associated with inflammation. *H. pylori* virulence factors induce the production of pro-inflammatory cytokines such as TNF-α which affect mucosal inflammation.[9] Several investigators have evaluated the effect of *H. pylori* on liver cirrhosis and PHG with controversial results. Some reports have shown a higher seroprevalence and a synergistic effect of *H. pylori* on liver cirrhosis and PHG. However, most studies have not found any correlation between *H. pylori* and PHG.[10]

A meta-analysis by Vergara *et al*.,[11] which included seven studies that assessed the prevalence of *H. pylori* infection and endoscopic lesions associated with cirrhosis, concluded that infection by *H. pylori* was present in 60.7% of the patients with increased risk of developing peptic ulcer. However, in another study, Batmanabane *et al*.[12] from India concluded that PHG does not provide a favorable environment for colonization by *H. pylori*, suggesting no contribution of the bacteria in the pathogenesis of PHG.

Thus, knowledge of the prevalence of *H. Pylori* infection in cirrhotic patients and the study of its association with PHG could be useful for better understanding of the pathogenesis of PHG. We performed this study to evaluate the prevalence of *H. pylori* infection among cirrhotic patients with PHG and to correlate the severity of liver disease and PHG with *H. pylori*.

## Patients and Methods

This study was conducted in the Department of Medical Gastroenterology, SMS Medical College, Jaipur, from January 2016 to January 2017, after the approval from the ethical committee. An informed consent was obtained from each patient.

A total of sixty consecutive patients of liver cirrhosis were enrolled in this study [Figure 1]. Patients with active peptic ulcer disease, primary or secondary malignancy, past gastric surgery, recent injection sclerotherapy, or band ligation for esophageal or gastric varices within 4 weeks, patients on nonsteroidal anti-inflammatory drugs or proton pump inhibitors, and those who underwent eradication therapy for *H. pylori* in the past 2 months were excluded from our study.

All patients who were admitted to the hospital, detailed history taking, thorough clinical examination, routine laboratory investigations, and abdominal ultrasound were done. The diagnosis of liver cirrhosis was based on clinical, biochemical, and radiological findings. The detection of hepatitis C virus (HCV) antibodies, hepatitis B virus surface antigen, and detailed ethanol intake history was done in all patients. Upper gastrointestinal endoscopy (UGIE) was performed for all patients to verify the presence of PHG, assess its severity, and assess the presence of esophageal varices (EV) or fundal varices. Baveno classification was used to assess the severity of PHG. PHG scoring system proposed by Baveno III Consensus Workshop [Table 1] was used to assess PHG.[13]

*H. pylori* infection was assessed by rapid urease test. UGIE done with four biopsies taken from the antrum (within 2 cm from the pylorus) and four biopsies taken from the gastric body (greater curvature side of the midbody). Out of four biopsy specimens, two from the antrum and two from the gastric body were used to identify *H. pylori*. This detection method is based on a

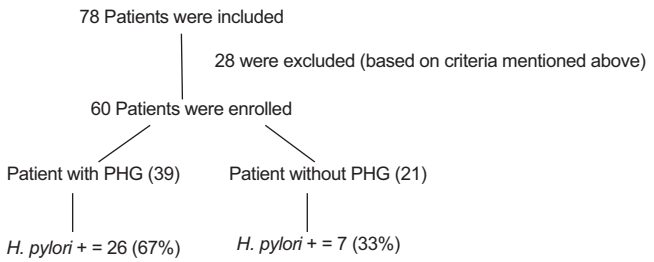


**Figure 1:** Flowchart

**Table 1: Portal hypertensive gastropathy scoring system proposed by Baveno III consensus workshop**

| Parameters | Score |
|---|---|
| Mucosal mosaic pattern | |
| Mild | 1 |
| Severe | 2 |
| Red markings | |
| Isolated | 1 |
| Confluent | 2 |
| Gastric antral vascular ectasia | |
| Absent | 1 |
| Present | 2 |

Mild portal hypertensive gastropathy≤3. Severe portal hypertensive gastropathy≥4.

**Table 2: Modified Child-Pugh classification of the severity of liver disease**

| | Points assigned | | |
|---|---|---|---|
| Parameters | 1 | 2 | 3 |
| Albumin (g/dl) | >3.5 | 2.8–3.5 | <2.8 |
| Bilirubin (mg/dl) | <2 | 2–3 | >3 |
| INR | <1.7 | 1.7–2.3 | >2.3 |
| Ascites | None | Slight–moderate | Tense |
| Encephalopathy | 0 | 1–2 | 3–4 |

A total Child–Turcotte–Pugh score of 5–6 is considered class A, 7–9 is class B, and 10–15 is class C. INR, International normalized ratio.

coloring, which is due to bacterial urease activity. The presence of *H. pylori* was indicated by a red coloring within 2–5 h.

The severity of liver disease was assessed using CTP classification [Table 2] and model for end-stage liver disease (MELD). The MELD score is calculated as follows: MELD score = 10 × [0.957 × ln(creatinine)] + [0.378 × ln (bilirubin)] + [1.12 × ln(INR)] + 6.43. It was calculated by an online calculator of the United Network for Organ Sharing (http://www.unos.org).

### Statistical analysis

Statistical analysis was made with the SPSS Statistics versions 18.0 IBM SPSS (NY, IBM Corp). Quantitative variables are expressed as mean and standard deviation. Qualitative variables are expressed as frequencies and percentages. Student's *t*-test was used to compare a continuous variable between two study groups. Chi-square and Fisher's exact test was used to examine the relationship between categorical variables. The odds ratio (OR) was used to investigate the strength of the associations. The confidence interval (CI) was considered as 95%. $P < 0.05$ was considered statistically significant.

## Results

A total of sixty adult patients with established liver cirrhosis (clinically, laboratory, and radiologically) were enrolled in the present study. They were 39 males (65%) and 21 females (35%) (mean age 55.75 ± 5.95 years).

According to CTP classification, 9 patients were classified as Child A (15%), 30 as Child B (50%), and 21 as Child C (35%). *H. pylori* infection was reported in 33 out of 60 patients with overall prevalence 55%. On UGIE, PHG was found in 39 patients (65%). Out of those 39 patients, 18 had mild PHG (46.15%) and 21 had severe PHG (53.85%). The mean age of patients with PHG was 58.3 ± 6.4 years compared to 53.2 ± 5.5 years in those without PHG ($P = 0.003$).

*H. pylori* infection was more prevalent among patients with PHG than those without PHG (67% vs. 33%; OR: 4.00, 95% CI: 1.298–12.325; $P = 0.0133$). Other clinical characteristics and endoscopic findings of patients with and without PHG are summarized in Table 3.

More importantly, out of the 26 patients with PHG and *H. pylori* infection, 17 had severe PHG (65.3%), and 9 had mild PHG (34.6%). On the other hand, only four patients had severe PHG (30.8%) and nine had mild PHG (69.2%) out of 13 patients who were *H. pylori* negative (OR: 4.25, 95% CI: 1.0188–17.729; $P = 0.04$) [Table 4]. Insignificant relation was found between *H. pylori* infection and severity of liver cirrhosis as regards CTP score ($P = 0.76$) and MELD score ($P = 0.56$) [Table 5].

## Discussion

PHG is not the most common cause of significant upper gastrointestinal tract bleeding in patients with portal hypertension, but bleeding is the most important complication of this disease. The incidence of acute upper gastrointestinal tract bleeding from PHG varies widely (2%–12%).[14] It is reported that 10% of PHGs cause anemia because of the chronic blood loss and 2.5% of patients experienced acute bleeding.[2]

The pathogenesis of PHG is likely to be complicated, but PHG seems to occur because of portal hypertension and alteration in gastric microcirculation, which produce mucosal surface hypoxia[15,16] and affect epithelial cell integrity, probably mediated through local factors such as overproduction of nitric oxide, oxygen-free radicals, endothelin-1, TNF-α, and prostaglandins.[17]

However, there are other factors associated with the presence and severity of PHG. These include prior

**Table 3: Clinical and endoscopic characteristics of patients with and without portal hypertensive gastropathy**

| Patient characteristics | Patients with PHG (*n*=39) | Patients without PHG (*n*=21) | *P* |
|---|---|---|---|
| Age (years) | 58.3±6.4 | 53.2±5.5 | 0.003 |
| Gender | | | |
| Male | 24 (61.5) | 15 (71.4) | 0.44 |
| Female | 15 (38.5) | 6 (28.6) | |
| Child-Pugh score | 8.6±1.2 | 8.5±1.6 | 0.78 |
| MELD score | 19.2±5.1 | 18.4±4.9 | 0.56 |
| Esophageal varices, *n* (%) | | | |
| No | 12 (30.8) | 6 (28.6) | 0.82 |
| Small | 12 (30.8) | 6 (28.6) | |
| Medium | 6 (15.3) | 2 (9.5) | |
| Large | 9 (230) | 7 (33.4) | |
| Patients with *H. pylori*, *n* (%) | 26 (67) | 7 (33) | 0.0133 |

PHG=Portal hypertensive gastropathy, MELD=Model for end-stage liver disease, *H. pylori=Helicobacter pylori*

**Table 4: Relation between *Helicobacter pylori* infection and the severity of portal hypertensive gastropathy**

| Severity of PHG | *H. pylori*-positive patients (*n*=26) | *H. pylori*-negative patients (*n*=13) | *P* |
|---|---|---|---|
| Mild PHG, *n* (%) | 9 (34.6) | 9 (69.2) | 0.04 |
| Severe PHG, *n* (%) | 17 (65.3) | 4 (30.8) | |

PHG=Portal hypertensive gastropathy, *H. pylori=Helicobacter pylori*

**Table 5: Relation between *Helicobacter pylori* infection and the severity of liver cirrhosis**

| Severity of liver disease | *H. pylori*-positive patients (*n*=33) | *H. pylori*-negative patients (*n*=27) | *P* |
|---|---|---|---|
| Child-Pugh score | 8.5±1.3 | 8.4±1.2 | 0.76 |
| MELD score | 18.0±3.5 | 18.5±3.0 | 0.56 |

MELD=Model for end-stage liver disease, *H. pylori=Helicobacter pylori*

treatment of EV, etiology of portal hypertension (cirrhotic vs. noncirrhotic), severity of primary liver disease, and *H. pylori* infection.[18]

Colonization of the gastric mucosa by *H. Pylori* might have an indirect role in PHG as colonization is, at least theoretically, associated with inflammation. *H. pylori* virulence factors induce the production of pro-inflammatory cytokines such as TNF-α, which enhance mucosal inflammation.[9]

A Chinese study by Yang *et al*. investigated the possible association between *H. pylori* infection and PHG in cirrhotic patients and suggested that *H. pylori* colonization of the stomach of cirrhotic patients was likely to be contributory to the pathogenesis of PHG.[19] In contrast, Balan *et al*. reported detection of *H. pylori* in 40% of cirrhotic patients, a figure identical to the prevalence of the organism in the general population. They concluded that *H. pylori* infection is unlikely to be an important factor in the pathogenesis of PHG.[20] A meta-analysis of seven studies by Vergara *et al*.[11] that assessed the prevalence of *H. pylori* infection and endoscopic lesions associated with cirrhosis and concluded that infection by *H. pylori* was present in 60.7% of the patients with increased risk of developing peptic ulcer. In another study of 37 patients, Batmanabane *et al*.[12] found an overall prevalence of *H. pylori* in 43% of patients with PHG and a decline in *H. pylori* positivity with increasing severity of PHG. They concluded that there was no contribution of the bacteria to the pathogenesis of PHG. Although this did not reach significance due to the limited number of patients, it suggests that the gastric mucosa in portal hypertension might not provide a hospitable environment for the colonization of *H. pylori*, especially when there is severe hemorrhagic congestion and edema of the mucosa.

On the contrary, Arafa *et al*.[21] found that each of *H. pylori* and PHG independently increased inducible nitric oxide synthase (iNOS) in gastric mucosa of cirrhotic patients. However, its role in the development of PHG is still conflicting. In our study, the overall prevalence of *H. pylori* in all patients with liver cirrhosis was 55%, a figure comparable to that of Abbas *et al*.[22] who found a prevalence of 62.1% and Safwat *et al*.[23] who found prevalence of 60%. Yet, a lower seroprevalence (35.7%) was reported by Sathar *et al*.[24] This discrepancy could be attributed to the different tools of *H. pylori* diagnosis as they depend on anti-*H. Pylori* IgG serology.

On investigating the relation between *H. pylori* and PHG in cirrhotic patients, we found a higher prevalence of the infection among patients with rather than those without PHG (67% vs. 33%, $P = 0.0133$); in addition, a significant association was found between *H. pylori* and PHG as an independent risk factor (OR: 4.00, 95% CI: 1.298–12.325; $P = 0.0133$). Similarly, the recent study of Sathar *et al*.[24] showed a significant association between *H. pylori* and PHG (OR: 2.134, 95% CI: 1.052–4.327; $P = 0.034$) and by Safwat *et al*.[23] (OR: 4.12, 95% CI: 1.191–14.252; $P = 0.025$). It has been reported that *H. pylori* increase obviously in cases with portal hypertension, thus may play a role in the development of PHG. On the contrary, other studies suggested that *H. pylori* infection was unlikely to contribute in the pathogenesis of PHG.[20,25] The socioeconomic status of the studied patients may have an impact on this difference. In addition, it has been postulated that PHG does not provide an adequate environment for *H. pylori* colonization, and therefore, this organism does not add significantly to the occurrence of PHG.[12]

Moreover, in the current study, out of the 26 patients with PHG and *H. pylori* infection, 17 (65.3%) had severe PHG, whereas only 4 (30.8%) out of 13 *H. pylori*-negative patients had severe PHG (OR: 4.25, 95% CI: 1.0188–17.729; $P = 0.04$), reflecting a significant relation between the infection and severity of PHG. While other studies showed no correlation with PHG severity,[18,22,26] our results were similar to Sathar *et al*.[24] and Safwat *et al*.,[23] who noticed a significant relation between *H. pylori* and severity of PHG ($P < 0.001$). In another study,[27] *H. pylori* was supposed to be one of the factors important for regulation of gastric mucosal capillary network function and structure, i.e., morphometric changes of gastric mucosa.

In view of the association between *H. pylori* and the severity of liver cirrhosis, several investigators have evidenced no relation with the advancement of liver disease.[28-30] Moreover, Kim *et al*.[29] noticed that the prevalence of *H. pylori* infection declines as the CTP score increases ($P < 0.001$). Similarly, in our study, no significant correlation was found between *H. pylori* and the degree of severity of liver cirrhosis regarding both CTP and MELD scores ($P > 0.05$). On the contrary, El-Masry *et al*.[31] revealed that the prevalence of *H. pylori* infection in HCV-infected patients was increased very significantly ($P = 0.003$) with increasing MELD and also CTP score ($P = 0.04$).

This study suggests that the gastric mucosa in cirrhosis might provide a hospitable environment for the colonization of *H. pylori*, especially when there is severe hemorrhagic congestion and edema of the mucosa. Factors such as increased iNOS expression resulting in high reactive oxygen species, impairment of gastric mucosal defense 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 results in gastric inflammatory response, including interleukin-8 and TNF-α, which may be associated with the sequence of events leading to PHG.

As this is a retrospective case–control study, further prospective studies with a large number of patients are needed to validate the association of PHG with *H. pylori* infection. One of the major limitations of the study is the absence of histological data, not only for the diagnosis of *H. Pylori* infection but also for the differentiation of lesions attributed to *H. pylori* gastritis from lesions due to PHG.

## Conclusion

Our results reflect a significant association between *H. pylori* infection and the occurrence and also the severity of PHG in patients with liver cirrhosis. Yet, the severity of liver cirrhosis itself did not correlate with *H. pylori* or the severity of PHG. Thus, whether eradication therapy is beneficial or not in patients with PHG has to be explored in the future studies.

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### Conflicts of interest

There are no conflicts of interest.

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