Correlation of Portal Hypertensive Gastropathy with Helicobacter Pylori Infection, Liver Dysfunction, Hypersplenism and Oesophageal Varices

OSAMA MOHAMAD HAMMAD, M.D. 1; MOHAMAD AHMAD ABU-SEIF, M.D. 2; MAGDY ASHOUR, M.D. 3 and TAMER HIFNAWY, M.D. 4

The Departments of Tropical Medicine 1; Internal Medicine 2, Faculty of Medicine Benisuef University; Pathology 3, Military Academy and Public Health 4, Faculty of Medicine, Benisuef University.

Abstract

Background: Portal hypertensive gastropathy (PHG) is a common endoscopic finding in patients with portal hypertension. The pathophysiology of this condition is not clearly understood. Although portal hypertension remains the crucial trigger for the development of PHG, other factors should be considered in the progression of this condition.

Study Aims: The aim of this study was to investigate the role of H. pylori infection, liver function, hypersplenism and oesophageal varices in development and severity of PHG.

Patients and Methods: This case control study included 60 cases with liver cirrhosis and PHG and 30 healthy individuals with non ulcer dyspepsia as a control group. Among patients group, liver cirrhosis was diagnosed by clinical, biochemical, sonographic and/or histopathological criteria of cirrhosis. After having the informed consent signed from all study subjects for participation in the study, all patients were subjected to upper gastrointestinal (GIT) endoscopy to assess the grades of oesophageal varices and PHG and four antral biopsies were taken for detection of H. pylori infection by histopathological examination. The grade of liver dysfunction was assessed according to Child-Pugh scoring system and grade of hypersplenism was assessed by reduction of white blood cells (WBCs), haemoglobin (HGB) and platelets (PLT). In control group, upper GIT endoscopy was done to them and four antral biopsies were taken for histopathological examination for detection of H. pylori infection. Individuals of control group and positive for H.pylori infection were subjected to triple therapy (clarithromycin, amoxicillin and omeprazole) for one week to eradicate this infection.

Results: There was statistically insignificant difference between the prevalence of H. pylori infection in PHG patients (70%) and control group (63.3%). The prevalence of H. pylori infection was statistically lower in patients with severe PHG (45.5%) than those with mild PHG (84.2%). There was statistically significant correlation between grade of PHG and grade of liver dysfunction, hypersplenism and oesophageal varices.

Conclusion: Our data showed that the H. pylori infection did not correlate with PHG, but severe PHG was significantly associated with less H. pylori infection. There was significant correlation between grade of severity of PHG and grade of liver dysfunction, hypersplenism and oesophageal varices.

Key Words: Portal hypertensive gastropathy (PHG) – Helicobacter pylori infection – Liver dysfunction – Hypersplenism – Oesophageal varices.

Introduction

THE term portal hypertensive gastropathy (PHG) refers to the mosaic pattern, congestion and oedema of the mucosa with or without red spots seen endoscopically in patients with portal hypertension [1]. It is a common endoscopic finding in patients with portal hypertension and is the cause of one out of five bleeding episodes in these patients [2]. The pathophysiology of this condition is not clearly understood. It has been suggested that PHG is a dynamic condition, which may not only worsen from mild to severe, but also improve and even disappears completely [3]. This finding suggests that although portal hypertension remains the crucial trigger for the development of PHG, other factors should be considered in the progression of this condition [4].

The correlation of PHG and Helicobacter pylori (H. pylori) infection, liver function, hypersplenism and oesophageal varices is still an issue of controversy [4-6].

The prevention and cure of PHG is important in decreasing upper gastrointestinal haemorrhage in patients with liver cirrhosis and this may be achieved with understanding the interrelated factors of PHG incidence and development. This study was aimed to investigate the role of Helicobacter pylori infection, liver function, hypersplenism and oesophageal varices in development and severity of hypertensive gastropathy.
**Patients and Methods**

A case control study technique was used. The study took place in Benisuif University Hospital in Benisuif Governorate. In the period between March 2007 to August 2008.

**Inclusion criteria:**
- Age between 18-70 years.
- Both sexes.
- Urban-rural residency.

**Exclusion criteria:**
- Emergency cases.
- Those with diminished mental capacity.
- Refusal of participation.
- Patients with non-cirrhotic portal hypertension, portal vein thrombosis, treatment with beta blockers or nitrates, previous endoscopic treatment of varices (sclero-therapy or band ligation) or previous surgical portosystemic shunt or TIPS.

**I- Cases:**

During the one year study period, sixty patients with liver cirrhosis and congestive gastropathy were enrolled in this study. They were 38 males and 22 females with mean age 49±9.5 years. Liver cirrhosis was diagnosed by clinical, biochemical, sonographic and/or histopathological criteria [7]. The degree of PHG was assessed according to the third Baveno International Consensus Workshop [8] and classified as mild when mosaic-like pattern (MLP) without redness of areola was present and severe when the MLP was superimposed by red signs.

**Diagnosis of Helicobacter pylori:**

Four antral biopsies were taken from each patient for the diagnosis of H. pylori infection. The biopsies were stained by Giemsa stain and examined for detection the presence of H. pylori.

**Determination of severity of liver disease:**

The severity of liver disease was classified according to Pugh's modification of Child's scoring system [9].

**Determination of degree of hypersplenism:**

Hypersplenism was assessed by the reduction of WBC, HGB and PLT (Table 1). Three to 4 scores were defined for mild hypersplenism, 5 to 6 scores for moderate and 7 to 9 scores for severe [6].

**II- Control group:**

Consists of 30 healthy subjects suffering of dyspepsia. Upper gastrointestinal endoscopy had been done to them and no peptic ulceration was detected in them. Four antral biopsies were taken for detection of H. pylori infection by histopathological examination. They were 21 males and 9 females with mean age 38±8.5 years. Individuals of control group and positive for H.pylori infection were subjected to triple therapy (clarithromycin, amoxicillin and omeprazole) for one week to eradicate this infection.

An informed consent was taken from patients and individuals of control group to participate in this study.

**Statistical analysis:**

Double data entry was done and data checks and cleaning were performed. The data base was converted to SPSS version 15.0 to perform descriptive analysis. All variables were treated as categorical variables. Chi Square test was used. The threshold of significance was fixed at the 5% level.

**Results**

**H. pylori infection rate in patients with PHG and control group:**

Forty two (70%) patients were positive for H. pylori infection and 19 (63.3%) individuals of control group were positive for H. pylori infection. There was insignificant difference of H. pylori infection in PHG group and control group (Table 2).
Association between degree of PHG and H. pylori infection:

Out of 38 patients with mild PHG, 32 (84.2%) were positive for H. pylori infection and out of 22 patients with severe PHG, 10 (45.5%) were positive for H. pylori infection. There was significant correlation between severe PHG and low H. pylori infection (Table 2).

Table (2): Association between H. pylori infection and presence and degree of PHG.

<table>
<thead>
<tr>
<th>H. pylori infection</th>
<th>+ve</th>
<th>–ve</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with PHG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. = 60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group No. = 30</td>
<td>19 (63.3%)</td>
<td>11 (36.7%)</td>
<td>0.523 (NS)</td>
</tr>
<tr>
<td>Patient with Mild PHG No. = 38</td>
<td>32 (84.2%)</td>
<td>6 (15.8%)</td>
<td>0.002**</td>
</tr>
<tr>
<td>Patients with severe PHG No. = 22</td>
<td>10 (45.5%)</td>
<td>12 (54.5%)</td>
<td></td>
</tr>
</tbody>
</table>

\( \chi^2 = 0.407 \quad p = 0.523 \) (NS), \( \chi^2 = 9.966 \quad p = 0.002**. \)

Fig. (1): Association between degree of PHG and H. Pylori infection among cases.

Association between PHG grading and severity of liver disease:

There was statistical significant correlation between the grade of PHG and degree of severity of liver disease (Table 3).

Table (3): Association between PHG grading and severity of liver disease.

<table>
<thead>
<tr>
<th>Grade of PHG</th>
<th>Child-Pugh grade</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild grade</td>
<td></td>
<td>22 (57.9%)</td>
<td>6 (15.8%)</td>
<td>10 (26.3%)</td>
<td>0.01*</td>
</tr>
<tr>
<td>No. = 38</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe grade</td>
<td>No. = 22</td>
<td>4 (18.2%)</td>
<td>8 (36.4%)</td>
<td>10 (45.5%)</td>
<td></td>
</tr>
</tbody>
</table>

Total \( 26 (43.3%) \quad 14 (23.3%) \quad 20 (33.3%) \)

\( \chi^2 = 9.1 \quad p = 0.01 \)

Discussion

Portal hypertensive gastropathy (PHG) is a pathological process in the gastric mucosa that develops due to portal hypertension and bears a high risk of haemorrhage due to gastric mucosal capillary dilatation. This mucosal congestion makes the mucosa more susceptible to damage from other agents as biles, aspirin and H. pylori and reduce the capacity to repair damage [11]. Reports on whether H. pylori infection can be a risk factor in
the development of PHG are conflicting. Yang et al. [12] found that H. pylori colonization of the stomach of cirrhotic patients was likely to be contributed to the development of PHG. Abdel-Hafez et al. [13] found positive correlation between H. pylori and severity of gastritis in portal hypertensive patients. They concluded that, the gastric changes resulting from the vascular and hormonal changes that could be produced by portal hypertension may facilitate colonization with H. pylori. This may enhance gastritis and bleeding.

On the other hand many studies concluded that the role of H. pylori infection in the pathogenesis of PHG is unlikely and that there was no need for its routine eradication in cirrhotics [5,14,15].

In this study, there was insignificant correlation between H. pylori infection and PHG and this in agreement with the previous studies [4,14,15].

In the present study, there was significant correlation between the degree of severity of PHG and low prevalence of H. pylori infection. Severe congestive gastropathy make the gastric mucosa not suitable for colonization of H. pylori and this may be related by decreased synthesis of urea by the unhealthy gastric mucosa. This result was also concluded by Bahnacy et al. [14], Batmanabane et al. [15] and Dong et al. [16].

In the present study, there was significant correlation between grade of congestive gastropathy and degree of severity of liver disease. This is similar to other studies that found that the prevalence and severity of PHG were correlated with severity of liver disease [17-19]. The more severely affected liver, the less metabolization of the vasodilator material leading to more congestion of gastric mucosa and the increase of grade of PHG. Ascites in patients with grade B and C liver function may press the portal vein and make the congestion of the gastric mucosa worse. Hepatocellular inflammation causes liver congestion and swelling, impedes portal vein vessels, then increases portal vein pressure and leads to gastroenteric mucosal congestion, hypoxia and edema [16,19].

Controversially, others found that the degree of liver dysfunction was not correlated with the severity of PHG in patients with cirrhosis [6,7,20]. So there is still no consensus on the relationship between liver function and PHG.

Hypersplenia exactly reflects the existence of portal hypertension. Research about hypersplenia and PHG is inadequate. Pan et al. [6] found that there was a significant correlation between the severity of PHG and hypersplenism. They explained their results by that: first, the congested spleen enables gastric mucosa blood to clot by increasing the blood flow in the portal vein and second, the decrease of platelets makes the stop of gastric mucosal haemorrhage difficult, the lowering of leucocytes results in easy infection of gastric mucosa and the reduction of erythrocyte decrease the oxygen supply of gastric mucosa.

In this study there was statistically significant correlation between the grade of PHG and degree of hypersplenism.

Esophageal varices exactly reflect the presence of portal hypertension. But the correlation between oesophageal varices and PHG is obscure [20-21]. Diverging the portal vein blood has some preventive effect on PHG [16] and there was a statistically insignificant correlation between grade of esophageal varices and grade of PHG [4,16,24].

In this study there was significant correlation between grade of oesophageal varices and grade of PHG and this agrees with other researchers results [7,23,24]. This can be explained by that both conditions are related to portal hypertension.

In conclusion, this one of the few studies evaluating the relationship between PHG and H. pylori infection, liver function, hypersplenism and oesophageal varices varies which an issue of controversy. Our data showed that the H. pylori infection did not correlate with PHG, but severe PHG was significantly associated with less H. pylori infection. There was significant correlation between grade of severity of PHG and grades of liver dysfunction, hypersplenism and oesophageal varices.

References