Original Article

Frequency and Severity of Portal Hypertensive Gastropathy in Cirrhosis

Poudyal S, Sharma S, Khadga PK, Pathak R, Jha A, Shrestha R[1]

Department of Gastroenterology, Tribhuvan University Teaching Hospital, Institute of Medicine

[1]Department of Internal medicine, Tribhuvan University Teaching Hospital, Institute of Medicine

Correspondance to: Dr Sagar Poudyal

Email: drsagarpoudyal@gmail.com

Abstract

Introduction: Portal hypertensive gastropathy (PHG) is a common endoscopic finding in patients with Cirrhosis. The pathophysiology of PHG was not clearly understood. It was thought that Portal Hypertension was an important triggering factor for the development of PHG but the other factor must be considered for its progression. It is one of the clinically important gastric mucosal lesions because it may cause acute and chronic gastrointestinal blood loss leading to anemia. The aim of our study is to determine the frequency and severity of PHG in patient with Cirrhosis.

Methods: This was a Cross-sectional observational study involving 61 consecutive Cirrhotic Patients, who attended Upper GI endoscopy were enrolled in this study as per inclusion and exclusion criteria in the Department of Gastroenterology Tribhuvan University Institute of Medicine. Child-Pugh's score and MELD score was determined at the entry to determine the severity of liver disease. Data regarding clinical and laboratory investigations were collected. Variceal size was measured endoscopically and the severity of PHG was graded according to Mac Cormack Classification.

Results: Out of total 61 patients, the frequency of PHG was found to be 47.5% among them where 44.2% patients had mild PHG and 3.3% patients having severe PHG. There were 29.5% patients in child pugh A, 39.3% in child pugh B and 31.2% in child pugh C. During analysis insignificant relation was found between the PHG with Child pugh score (ρ=0.4) and MELD score (ρ=0.7).When PHG frequency was related to alcohol intake the relations were statistically not significant. There was no association found between portal hypertensive gastropathy with esophageal varices and gender of the patient.

Conclusion: Our data showed that the frequency and the severity of PHG are not influenced by the Gender of the patient, etiology and severity of cirrhosis or by presence of esophageal varices

Key words: Cirrhosis, Portal hypertensive gastropathy, esophageal varices.

Introduction

Cirrhosis is a common health problem with high incidence and prevalence according to the world literature. It is associated with alterations in the gastrointestinal mucosa, with high risk for peptic ulcer disease.[1] The term PHG (portal hypertensive gastropathy) refers to the mosaic-like pattern, congestion and edematous mucosa with or without red spots seen endoscopically in patients with portal hypertension.[2] It is a common endoscopic finding in patients with portal hypertension and is the cause of around one out of five bleeding episodes in these patients.[3]

PHG is thought to be due to venous congestion and gastric mucosal capillary dilation as a result of portal hypertension. The exact pathogenesis of PHG is still poorly understood.[4]

PHG is one of the clinically important gastric mucosal pathology because it may cause acute or chronic gastrointestinal blood loss leading to anemia. The prevalence of PHG varies widely; frequencies from 4%

to 98% have been recorded in studies of patients with portal hypertension[5]. The pathogenesis of this disorder is however controversial and not clearly understood. It has been assumed that PHG is a dynamic condition, which may not only worsen from mild to severe, but also improve or even disappears completely.[4]

Several pathophysiological mechanisms have been studied for PHG. They include increased serum gastrin[6] leading to increased acid secretion, alteration in blood flow[7, 8] Decreased secretion of prostaglandin in the gastric mucosa[9] and the presence of Helicobacter pylori (H. pylori) infection.[10]

Portal hypertension is the important trigger factor in development of PHG with improvement of PHG following shunt procedures supporting such an association.[7] However, several studies have reported that the natural history of PHG is also influenced by the severity of liver disease,[8] and also with presence and size of gastroesophageal varices, and previous variceal eradication by endoscopic variceal sclerotherapy or banding.[4] The present study was undertaken to assess the frequency and severity of PHG in Nepalese patient with Cirrhosis. In addition, we have attempted to determine the role of severity of liver disease, etiology of portal hypertension and presence of gastric varices, on the occurrence of PHG.

Methods

All patients admitted in Department of Gastroenterology at TUTH with Cirrhosis and related complications during the study period. The total of 61 consecutive cirrhotic patients with liver Cirrhosis who were attending for diagnostic endoscopy at our institute was enrolled in this study. The study was performed between June 2016 and August 2016 according to the ethical standards approved by (IRB) Institutional Review Board of Research department of Institute of Medicine. The informed and written consent was obtained from each patient. Patient with hepatocellular carcinoma (HCC), history of GI surgery, recent history of acute variceal bleeding and previous history of band ligations, were excluded from the study.

Baseline assessment included a thorough medical history and full clinical examination. A complete panel of laboratory studies, including complete blood count, liver and renal functions were performed for all patients. An abdominal ultrasound was also done. The diagnosis of liver cirrhosis was based on clinical, radiological, liver function test (prothrombin time and serum albumin), and presence of esophageal or fundal varices.

As causative factors for liver cirrhosis, chronic hepatitis B was diagnosed in cases with detectable hepatitis B surface antigen, and chronic hepatitis C was diagnosed in patients positive for anti-HCV antibody and positive HCV RNA as causative factors for liver cirrhosis, Patients who had ingested alcohol daily at a dose of > 80gm for more than 10 years in the absence of other causative factors such as evidence of a viral infection were classified as having alcoholic liver cirrhosis.

Ascites was classified according to the International Ascites Club.[11] Hepatic Encephalopathy(HE) was graded according to West Haven Criteria.[12] Upper GI endoscopy (UGIE) was performed for all patients (Pentax EG-290Kp Videoscope) to verify the presence of PHG, to assess its severity and to assess the presence of esophageal (EV) or fundal varices.

Esophageal varices were graded from grade I to grade IV using classification of Paquet.[13] The severity of PHG was graded according to Mc Cormack's classification into two classes: mild and severe.[14] Mild PHG comprises snake-skin or mosaic pattern or fine pink speckling, and severe PHG comprises cherry red spots with or without spontaneous bleeding.

Gastric antral vascular ectasia (GAVE) was diagnosed by the presence of flat or slightly raised, red stripe-like lesions radiating from the pylorus to the antrum and body of the stomach.

The severity of liver disease was assessed using Child-pugh classification based on patients' clinical and laboratory data (ascites, HE, serum albumin and bilirubin, and prothrombin time) and also Model of End stage live disease (MELD) score. The MELD score was calculated according to original formula proposed by the Mayo clinic group[1], where MELD score = $3.8 \times \log$ (serum bilirubin) + $11.2 \times \log$ (INR) + $9.6 \times \log$ (serum creatinine).

Statistical analyses

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Association of PHG with different variables including patient characteristics and complications

of cirrhosis was tested using ANOVA for continuous variables and chi-square test for nominal variables. *P* values of less than 0.05 were considered statistically significant. Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) release 21 for Windows (SPSS, Inc., Chicago, IL). Microsoft word and Excel have been used to generate tables and graph.

Results

The present study included 61 in patients with liver cirrhosis who were admitted in Department of Gastroenterology at Tribhuvan University, Institute of Medicine (TU, IOM) the data was collected during the period of June 2016 to August 2016.

Out of 61 cirrhotic patients majority 79.5% were males and 29.5% were females. The mean age of cirrhotic patients were 51.49±11.3 years with range 20-76years. Among the total Cirrhotic patient, 83.6% were alcoholic and 16.4% were nonalcoholic (6.6% were Nonalcoholic steatohepatitis, 4.9% were Chronic Hepatitis B, 3.3% were Chronic Hepatitis C, 1.6% were Hepatitis C, , 1.6% was Cryptogenic) as shown in figure 1.

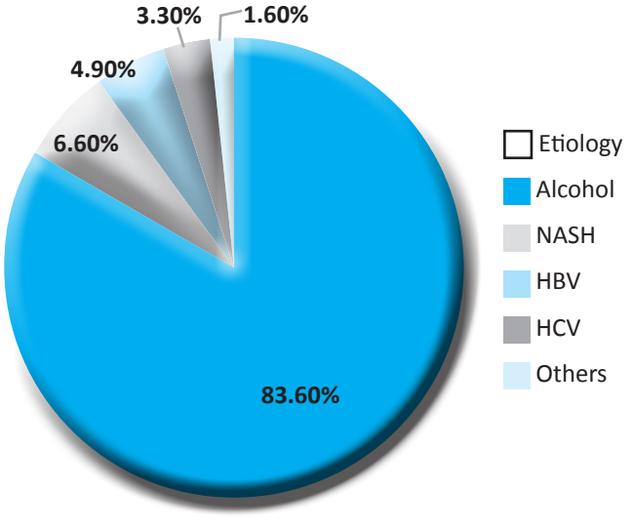

Fig 1. Distribution of Cirrhosis by risk factors

Among total 61 patients, 44.2% had mild PHG and of these 37 % were in Child A, 37% in Child B and 7 (26%) in child C (Fig above).out of 61 patients only 2 had severe PHG, of them 1(50%) patient in child A and 1(50%) patient in child B.

The mean Child-Pugh score was 8.10 ± 2.3 (range, 5-13); the mean MELD score was 15.93±5.9 (range, 6-32), there were 18(29.5%) in child A, 24(39.3%) in child B and 19 (31.1%) of patient in Child C. During endoscopy of 61 patients 75.4% patients had esophageal varices 24.6% had no esophageal varices, among the total esophageal varices 29.5% had grade I, 24.6% had grade II, 13.1% had grade III and 8.2% had grade IV varices.

Among cirrhotic patient 3.3% patient had Gastroesophageal varix (GOVI) and 1.6 % patient had isolated gastric varix (IGVI). Out of the total 61 Cirrhotic patients, 47.5% patients were found to have PHG where 44.2% patients had mild PHG and 3.3% had severe PHG. In the study 52.4% had ascites where mild ascites were 16.4%, moderate were 24.6% and severe were 11.4% as shown in table 1.

Table 1: Basic Characteristics of the patients

Parameter	Frequency
Age	51±11.31year(Mean)
Gender	
Male	43(70.5%)
Female	18(29.5%)
Causes of Cirrhosis	
Alcohol	51(83.6%)
NASH	4(6.6%)
Hepatitis B Virus	3(4.9%)
Hepaitis C Virus	2(3.3%)
Cryptogenic	1(1.6%)
Child Pugh SCORE	8.10±2.29 (Mean)
Child-A	18(29.5%)
Child- B	24(39.3%)
Child- C	19(31.2%)
MELD SCORE	15.90±5.88
PHG	
Mild	27(44.2%)
Severe	2(3.3%)
Absent	32(52.5%)
Esophageal Varices	
Grade I	18(29.5%)
Grade II	15(24.6%)
GradeIII	8(13.1%)
Grade IV	5(8.2%)
Ascites	
Minimal	10(16.4%)
Moderate	15(24.6%)
Severe (Tense)	7(11.5%)
Absent	29(47.5%)

Insignificant relation was found between the PHG and Child pugh score (p=0.44). Similarly no significant relation found between PHG and MELD score (p=0.78).

When PHG frequency was related to alcohol intake, 28 (55%) of the patients with alcohol intake had PHG as opposed to 4 (40%) patients who did not consume alcohol. The relations were statistically not significant (ρ=0.4) as shown in Table 2.

Table 2: Correlation of PHG with Child pugh score, MELD Score, esophageal Varices and etiology of Cirrhosis

Parameters		PHG Positive	PHG Negative	ρ value
Child-Pugh Score	<8	13	15	**0.446**
	≥8	19	14	
MELD Score	<12	10	8	**0.786**
	≥12	12	21	
Esophageal Varices	absent	9	6	**0.562**
	present	23	23	
Etiology of Cirrhosis	alcoholic	28	23	**0.496**
	nonalcoholic	4	6	

There was no association found between portal hypertensive gastropathy and esophageal varices (p=0.24). Also there was no significant association found between PHG and gender of patient. Insignificant relation was found between severity of PHG and MELD Score (ρ.0.05) Insignificant relation was found between severity of PHG and Child Class (ρ=0.4) (figure 2)

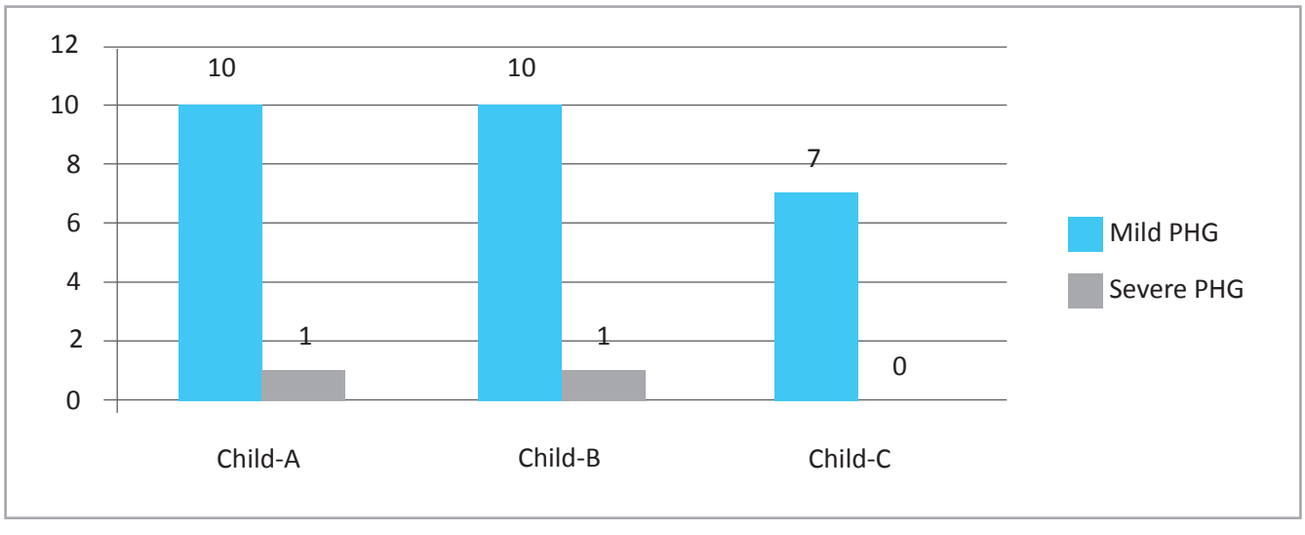

Fig 2: Severity of PHG according to Child Pugh score (Class)

Discussions

In recent years, gastric mucosal lesions have been recognized as an important complication of cirrhosis with portal hypertension. A variety of terms including erosive gastritis, acute gastric erosions, and vascular ectasia have been postulated to describe this pathology. Because the pathological change is characterized by vascular ectasia rather than mucosal inflammation, the term congestive gastropathy was coined by McCormack et al.[3] Now a days, PHG is the preferred term.

Portal hypertensive gastropathy is the change in gastric mucosa in patients with portal hypertension, where mucosal friability and dilated blood vessels are present in the gastric mucosal surface.[15] It was reported that about 10%

of Gastropathy causes anemia due to the chronic blood loss, 2.5% of patients experienced acute bleeding, and mortality rate related to acute bleeding reaches 12.5%

According to the literature overall prevalence of PHG ranges between 20% and 98% in patients with cirrhosis of liver.[2] There may be a higher prevalence among patients with high Child-Pugh scores patient with esophageal Varices or a history of variceal treatment.[16]

The frequency of PHG in our study was found to be 48% among them 3.27% patients having severe portal hypertensive gastropathy. This discrepancy may be related to selection of patient with different etiologies of cirrhosis and interobserver variability. This high frequency of PHG and severity is supported by other study.[4]

It has been previously shown that the overall prevalence of PHG was higher in patients in Child Pugh class B than in patients in class A and C, and that the prevalence of severe gastropathy was lowest in patients in class C[17] as with previous studies the frequency of PHG in our study was higher in Child class B followed by child class A and C but it was not statistically significant (ρ=0.4).

The pathogenesis of PHG is still complex, and many controversies exist. Until now, a variety of etiological factors have been investigated to illustrate the pathogenesis of PHG. Many factors including splanchnic blood flow, humoral factors, local disturbances in the regulation of vascular tone, and portal pressure have been examined to explain the underlying mechanisms.[5, 18] It was postulated that PHG develops as a result of vascular congestion induced by blockade of gastric blood drainage rather than by hyperemia.[19]

In our study the etiology of cirrhosis did not influence the development of portal hypertensive gastropathy (ρ=0.4). Similarly PHG was not influenced by the gender of cirrhotic patient. (ρ=0.4). In our study though 75% of patient had coexisting esophageal varices and none of our patient had history of band ligation, but there was no statistically significant relation found between the development of varices and portal hypertensive gastropathy (ρ>0.5).

Several studies have shown that PHG is aggravated by the sclerotherapy and banding therapy of esophageal varices,[20-24] but long-term follow-up studies by indicate Hou et al found that that changes in the severity of PHG after variceal sclerotherapy are reversible.[24] The result of study published by Sarin et al,[16] who found that PHG developing after variceal eradication is often transitory and less severe, while, in the case of pre-existing PHG, endoscopic therapy for varices could worsen the PHG, with a likelihood of bleeding.

In our study CTP score of > 8 and a MELD score of > 12 was not significantly associated with presence and severity of PHG in patients with cirrhosis; both these scores being reflective of severity of liver cirrhosis. Study by Dong et al. noted that 68% of their patients with PHG had liver cirrhosis, with 27% of patients with severe PHG being classified as CTP class C and only 7% classified as class A.[25] Similarly, a recently published study by Young et al. demonstrated that the MELD score of 11.3±3.5 is a predictor of severe PHG.[4] In our study, we did not find any significant relation between Child pugh score, MELD score, the presence or grade of esophageal varices and the occurrence or severity of PHG (ρ value >0.05). Similarly in the study by Pan et al.[26] development of PHG is less influenced either by the severity of cirrhosis (Child-Pugh grade) and or by the presence or non presence of gastric varices furthermore, Abbas et al[27] could not find any correlation of Child-Pugh and MELD scores with the severity of PHG. So there is still no consensus on the relationship between liver function and PHG.

Conclusion

Our data showed that the frequency and the severity of PHG are not influenced by the etiology of liver cirrhosis, severity of cirrhosis or by the size of varices. There were several limitations of our study being observational study and small number of patient the results of this study cannot be projected for the large group of general population. Hence further large prospective studies are required to better understand the pathogenesis of the gastric lesions in patients with portal hypertension.

Conflicts of interest: None declared

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