ORIGINAL RESEARCH

Association between Portal Hypertensive Gastropathy with Different Grades of Liver Dysfunction in a Tertiary Hospital: A Cross-sectional Study

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Abstract

Background: Controversy exists regarding how and to what extent portal hypertensive gastropathy (PHG) varies with different grades of liver dysfunction. Our aims and objectives were to assess the correlation of PHG with different grades of liver dysfunction.

Materials and methods: Seventy (70) patients diagnosed with chronic liver disease (CLD) with gastrointestinal bleeding admitted to the Department of Medicine, R. G. Kar Medical College and Hospital, Kolkata were considered as the study group in this retrospective study design. Portal hypertension (HTN) was confirmed by endoscopic features and their history, clinical, hematological, and biochemical data. Patients with systemic disorders, such as diabetes, renal failure, septicemia, and congestive cardiac failure were excluded from the study. Endoscopically, it was found that 35 patients had congestive gastropathy (CG), while another 35 patients without gastropathy served as controls. The Child's Pugh scoring system was applied to assess the severity of liver dysfunction.

Results: The prevalence of esophageal varices and CG were frequently observed in the patients with portal HTN in this study. Congestive gastropathy is more commonly located in the fundus and body (74.28%). Mild gastropathy (57.14%) is more commonly encountered than severe gastropathy (42.85%). The number of patients with bleeding episodes is found to be more in patients with PHG [34(97.14%)] compared with non-PHG group [28(80%)]. In patients with mild PHG, child's grade A was found in 2 (10%) cases, grade B in 13 (65%) cases, and grade C in 5 (25%) cases. In patients with severe PHG, child's grade A was found in 2(13.3%), grade B in 4 (26.6%), and grade C in 9 (60%) cases.

Conclusion: Severe PHG was associated more with grade C liver disease (severe grade liver dysfunction). The correlation between severity of PHG and liver dysfunction was found to be significant. More studies are needed to enable one to predict which patients will develop PHG and which will bleed from it.

Keywords: Ectasia, Endoscopy, Gastric varix, Liver dysfunction, Portal hypertension.

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Introduction

The portal system is framed with the veins which drain blood from the alimentary tract, spleen, gall bladder, and pancreas. Because of the lack of valves in the portal venous system, a retrograde transmission of increased pressure occurs due to the resistance developed at any level between the right heart and splanchnic vessels.[1–3] The normal pressure of the portal vein is around 7 mm of Hg. Greater than 12 mm of Hg is considered portal hypertension (HTN).

Portal HTN can be assessed clinically, radiologically, endoscopically (as graded of varices), and direct assessment of the pressure. The difference between wedge hepatic venous pressure (WHVP) and free hepatic venous pressure (FHVP) is known as the hepatic venous pressure gradient (HVPG), which is also termed as the portal (sinusoidal) pressure. These are measured by balloon catheter. In our study, portal HTN was assessed clinically, radiologically, and endoscopically (by measuring the grade of varices).[3–6]

Not only the discrete varices but also a variety of intestinal mucosal changes owing to the microcirculation abnormalities are characteristically seen in the chronic portal HTN. Some gastric mucosal changes, known as portal hypertensive gastropathy (PHG) are associated with cirrhosis and other factors. In one such study, the PHG is classified as mild which is characterized by fine pink speckling or a mosaic pattern, whereas severe lesions are denoted by discrete red spots or diffuse hemorrhages.

These are found maximally in the fundus and may involve any part of the stomach. These small polygonal areas look like a mosaic pattern encompassed by a depressed and pale-yellow border. The risk of bleeding is highly predicted if the lesions appear to be red

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Table 1: Child–Pugh classification

	Numerical points		
Parameter	*1*	*2*	*3*
Ascites	Absent	Mild	Moderate-to-severe
Encephalopathy	None	Grade I–II	Grade III–IV
Serum bilirubin (mg/dL)	<2	2–3	>3
Serum albumin (gm/dL)	>3.5	2.8–3.5	<2.8
Prothrombin time (seconds increased over control)/I.N.R.	1–3/1.7	4–6/1.7–2.3	>6/>2.3
	Child–Pugh class		
I.N.R. = International normalized ratio	*A*	*B*	*C*
Numerical score	5–6	7–9	10–15

Class – A: Well compensated disease, Class – B: Significant functional compromise, Class – C: Decompensated disease

or cherry red spots.[7,8] The black-brown spots are developed due to intra-mucosal hemorrhage. The main histological features are vascular ectasia within the mucosa. Some studies report that after sclerotherapy such changes in the gastric mucosa may be increased, however controversy exists. This PHG (portal hypertensive gastropathy) can be exempted only after the reduction of portal pressure. Portal hypertensive gastropathy accounts for 1–8% of primary upper gastrointestinal bleeding and 30–60% of secondary acute or chronic bleeding in the first 12 months, mainly after sclerosing therapy of varices. Congestive gastropathy has been classified into four stages depending on the appearance of gastric mucosa.

These alterations occur more predominantly in the fundus of stomach, and are caused by venous and capillary ectasia as well as by arteriovenous shunts. Another gastric mucosal change known as gastric antral vascular ectasia (GAVE) is denoted by enhanced arteriovenous communication among the dilated precapillaries, veins and the muscularis mucosa that produces increased gastric mucosal perfusion.[9,10] Gastric antral vascular ectasia is seen mainly in portal HTN, also seen in cases of scleroderma, Syndrome: (C- Calcinosis, R- Raynaud's phenomenon, E- Esophageal dysfunction, S- Sclerodactyly, T- Telangiectasia) (CREST) syndrome etc. This GAVE must be differentiated from PHG, and that is not related to portal HTN directly rather influenced by dysfunction of liver. The severity of liver dysfunction in cirrhosis are assessed by Child's criteria. The three variables such as size of lesion, presence of red signs and liver dysfunction are the best predictors of bleeding. Portal hypertensive gastropathy alters with grades of liver dysfunction but controversy exists.

Although the most common bleeding complication due to portal HTN is variceal bleeding, but congestive gastropathy (CG) are developed by many patients due to venous HTN. The mucosal bleeding are the most common characteristics rather than the brisk hemorrhage, and that is typical of a variceal bleeding source. Moreover, PHG is a variable pathologic condition; that is, it remains unchanged in 29% cases, worsened or improved in 25% cases, spontaneous resolution occurs within 3–6 months in 55% cases. 5 years survival without PHG is 85%, with mild PHG, it is 72% and with severe PHG it is 46%. Chances of re-bleeding in PHG are 56% in 12 months period.

The enhancing resistance to portal circulation is the fundamental hemodynamic abnormality of portal HTN. This may be mechanical owing to architectural disturbances and nodularity or due to obstruction of the portal vein in cirrhosis. The collagenosis of the space of Disse, swelling of hepatocytes, and the resistance exerted by portosystemic collaterals vessels are other intrahepatic factors. The dynamic increase in intrahepatic vascular resistance is also observed.[11–13]

In this context, the goal of the present study was to substantiate the fact that the various grades of liver dysfunction (assessed by Child's Pugh score) have different influences in producing PHG. Better knowledge of those may aid in preventing the bleeding episode.

Materials and Methods

The study was carried out among inpatients of the Medicine ward and Gastroenterology outpatient department (OPD) of a tertiary care hospital R. G. Kar Medical College, Kolkata from August 2016 to July 2017. Seventy (70) patients diagnosed chronic liver disease (CLD) with gastrointestinal bleeding or swelling of abdomen or other features were considered as the study group. The portal HTN in the patients with CLD was confirmed by endoscopic features and their history, clinical, hematological and biochemical profile. Patients with systemic disorders, such as diabetes, renal failure, septicemia, and congestive cardiac failure were excluded from this study to minimize the possibility of pathophysiological and clinical changes in PHG, as well as to minimize the error in endoscopic findings in PHG.

A full medical history was taken and complete clinical examination was done maintaining a proforma. The clinical diagnosis of portal HTN was based on findings such as enlarged or shrunken, firm liver with irregular surface mainly left lobe palpable in most of the case along with splenomegaly, prominent abdominal wall collateral or past history of hematemesis and melaena.

Complete blood count with erythrocyte sedimentation rate (ESR), blood glucose (fasting), urea, creatinine, liver function test (LFT) with prothrombin time (PT) and I.N.R, Ascitic fluid study (cell type, cell count, protein, glucose including gram's stain) were done in the Pathology and Biochemistry department using standard method and Auto analyzer.

Ultrasonography (USG) of abdomen was done in the radiology department to measure the portal vein diameter, hepatic and splenic size, hepatic echotexture, ascites. The severities of liver dysfunction were assessed by applying the Child's Pugh criteria (Table 1).

Upper gastrointestinal endoscopy was done by a well experienced gastroenterologist in the gastroenterology department

Table 2: Distribution of patients according to Child's Pugh classification in relation to severity of PHG

	Child's Pugh class (n = 35)						
	A		*B*		*C*		
Type of PHG	*No*	*%*	*No*	*%*	*No*	*%*	*Total*
Mild PHG (*n* = 20)	2	10.0	13	65.0	5	25.0	20
Severe PHG (*n* = 15)	2	13.3	4	26.6	9	60.0	15
Total	4		17		14		35

Table 3: Association of Child's grade B and grade C with severity of PHG.

	Child's grade B		*Child's grade C*			
Type of PHG (n = 31)	No	%	No	%	*Total*	*Chi–square results*
Severe PHG	4	30.7	9	69.2	13	$\chi^2 = 5.24$
Mild PHG	13	72.2	5	27.8	18	*p*-value = 0.02
Total	17		14		31	

Table 4: Test for significance in child's grade A and grade C with severity of PHG

	Child's grade A		*Child's grade C*			
Type of PHG (n = 18)	No	%	No	%	*Total*	*Chi–square results*
Mild PHG	2	28.6	5	71.4	7	$\chi^2 = 0.27$
Severe PHG	2	18.1	9	81.8	11	*p*-value = 0.6
Total	4		14		18	

by using flexible video-endoscopy (Olympus GIF-100) after overnight fasting using only topical anesthetic (2% lignocaine viscous solution) applied to the mouth and throat to search varix, esophagitis, CG and ulcer. The whole procedure was explained to the patient. Congestive gastropathy was classified into four stages depending on the endoscopic appearance of gastric mucosa.

- Stage I: Superficial redness on the surface of the gastric mucosa.
- Stage II: White reticular form with separate areas of prominent, pink, and edematous mucosa.
- Stage III: Cherry red spots.
- Stage IV: Diffuse bleeding.

In the study, the severity of gastropathy was classified as (a) Mild (include stage I and stage II) and (b) Severe (include stage III and stage IV). The categorical variables were summarized as number and percentage. Descriptive statistics were performed and frequency distribution tables were constructed to show the distribution of the data. Further analyses were carried out using Chi-square tests with Yates' continuity correction wherever applicable to find out the association of the levels of gastropathy and stages of liver dysfunction. The data entry and analyses were done using latest versions of Microsoft Excel and SPSS.

Results

The esophageal varices were identified in 35 cases (100%) in patients of PHG group and in 34 cases (97.14%) with non-PHG group. Red color signs were found in 6 (17.14%) cases in patients with PHG and in 1 (2.85%) case in patients without PHG. In the patients with PHG, mild gastropathy were found in 20 (57.14%) cases and severe gastropathy in 15 (42.85%) cases. In 5 (14.28%) cases, the gastropathy was found in antrum, 26 (74.28%) cases involved fundus and body and in 4 (11.42%) cases it was diffuse. No gastropathy were seen in patients without PHG. The child's grade A was found in 4 (11.43%) cases with PHG and 9 (25.71%) cases without PHG. Child's grade B has been found in 17 (48.57%) cases with PHG and in 22 (62.85%) cases without PHG. Child's grade C has been found in 14 (40.0%) cases with PHG and in 4 (11.42%) cases without PHG.

Table 2 presents that child's grade A was found in 2 (10.0%), grade B in 13 (65.0%) and grade C in 5 (25.0%) cases with mild PHG. Child's grade A was found in 2 (13.3%), grade B in 4 (26.6%), and grade C in 9 (60.0%) cases with severe PHG.

Table 3 depicts the associations between the severity of PHG and Child Pugh's grades B and C. It was noted that severe grades of liver dysfunction are associated more strongly with severe grades of PHG with a significant *p*-value ($p = 0.02$) whereas lower grades of liver dysfunction do not have a significant association with severe PHG with a p-value of 0.6 as given in Table 4.

Discussion

In present days, gastro-esophageal endoscopic characteristics of portal HTN have the predictive value for bleeding risk and such endoscopic features also help to identify patients for prophylactic management. In our study, esophageal varices are found in all cases with PHG and 34 (97.14%) cases without PHG.

In the year 1986, Burroughs et al.[14] recognized the importance of determining the prevalence of endoscopic signs. Beppu et al.[15] regarded red color signs (RCS) as microtelangiectasia or small dilated vessels and found it in 76% of cases. Cales et al.[16] observed a similar prevalence of RCS. Kleber et al. in a retrospective study observed RCS in 22% of cases. In our study, red color signs (RCS)

were observed in 6 (17.14%) cases in patients with PHG and in 1 (2.85%) case without PHG.

In patients with portal HTN, gastric mucosal changes are frequently encountered. The term congestive gastropathy (CG) was coined by McCormack et al.[17–19] Portal hypertensive gastropathy is defined by congestive changes in the gastric mucosa due to increased portal pressure and was first described about 20 years back. Mosaic pattern of gastric mucosa (mild PHG or CG) was observed by Papazian et al.[20] in 94% of their cases. The prevalence of PHG in portal hypertensive patients varies from 4 to 98% (the mean prevalence is 53%).[21] The mild PHG is more prevalent, and encountered in 20–57% of patients (the mean prevalence is 49%); severe PHG is found in 7–41% of patients (mean prevalence 49%). The abnormalities of the mucosal found in PHG are typically seen from proximal to antral part of the stomach, and most commonly encountered in the fundus.[22] McCormack et al.[17] reported CG in 51% of cases of portal HTN. He observed that out of 51% cases, 56.9% have mild CG and 43.1% have severe CG. He found CG more commonly in the region of fundus and body of the stomach. Nilius R observed CG more prominently in the fundus. In our study, PHG was seen in 35 cases out of 70 cases among which 20 (57.14%) cases were mild and 15 (42.85%) cases were severe. In 5 (14.28%) cases, PHG was seen in antrum, 26 (74.28%) cases involved fundus and body and in 4 (11.42%) cases, it was diffuse. Our current findings in our study are more or less consistent with the above study results.

Several investigators have evaluated the sensitivity as well as the specificity of PHG (especially congestive gastropathy) lesions in the finding of portal HTN.[9,22] Their evaluated reports show consistently high specificity (93–100%) toward mosaic pattern (snake skin appearance) but controversy is present for the sensitivity of this sign. Several authors[23–25] have mentioned the prevalence of this sign is about 30–40%. This diversity of observation may be interpreted by variability among the study groups or by incorrect inter-observer agreement.

In our study, a prepyloric ulcer was found in 1 case, an ulcer in D1 in three cases. Fassio E et al.[26] in his study observed that in 13.8% of cases, the cause of upper gastrointestinal bleeding was peptic ulcer. The previous studies mentioned that prevalence of peptic ulcer disease (PUD) in known cirrhotic patients was found to be about 20% and 30% of hemorrhage in cirrhotic cases was found secondary to peptic ulcer.[27] Sutton FM[28] studied 222 cases with esophageal varices and found that only varices in 65% cases, varices and CG in 15% cases and gastric ulcer in 4% of cases were responsible for upper gastrointestinal bleeding. Endoscopic sclerotherapy-induced PHG is more common irrespective of the cause of portal HTN. It is explained by the large number of collateral circulations that must be adapted at the cardia, owing to occlusion of the varices after sclerotherapy.

It is more often asked that "is portal HTN the only determinant for the evolvement of PHG?" This seems unlikely because the development of PHG does not occur in all patients with increasing portal pressure or obliteration of the varices. From the different studies, it is evident that there is no difference in variceal pressure among all patients with PHG and non-PHG groups. Therefore, not only the pressure, but other relevant conditions like progression of the portosystemic shunts, gastric microcirculation variability among the individuals, and ectopic varices may be possible determinant factors in the development of PHG in a patient. The development of PHG greatly depends on the stage and severity of the liver dysfunction. The incidence of PHG is significantly higher in patients with Child's C liver disease compared with patients having child's A or B disease. This higher incidence of PHG among patients having a child's class C disease probably contributed by some humoral substances, the neuronal influences, or some other unknown factors.

Conclusion

Though there is little correlation between the severity of portal HTN and PHG, it is clearly concluded that a chronically elevated portal pressure is a prerequisite to develop such disorder. The severity of PHG was correlated with the severity of the liver disease, that is, severe PHG was observed more in child's grade C disease. Thus, other than portal HTN other contributing factors may also be responsible for the pathogenesis of PHG. The number of patients with bleeding episodes is found to be more in patients with PHG [34 (97.14%)] than in patients without PHG [28 (80%)]. Therefore, more studies are required for the prediction about which group of patients will develop PHG and which patients may bleed from it.

Take Home Message

- Some contributing factors may also be responsible for the pathogenesis of PHG other than portal pressure.
- Severe PHG is associated more with a severe grade liver dysfunction (Grade C liver disease).
- More research is required for the prediction about which group of patients will develop PHG and who may bleed from it.

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