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Original Research Article

Portal Hypertensive Gastropathy and Colopathy in Cirrhotics with Anaemia

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Conflict of interest: Nil

Abstract

Background: This study was to document portal hypertensive gastropathy (PHG) and portal hypertensive colopathy (PHC) in a given cirrhotic with anaemia.

Methods: 87 patients participated in this hospital-based prospective cross-sectional study, which was carried out from January 2016 to July 2017 at the Department of Gastroenterology, M.S. Ramaiah Medical College, MSRIT, Bengaluru. The patients underwent a thorough clinical evaluation, history, and physical examination, Biochemical investigations like CBC, LFT, PT-INR,Peripheral smear, Serum iron studies, Vitamin B12 levels,Viral markers-HBsAG and Anti-HCV, etiological workup for the underlying cirrhosis was done, USG abdomen was performed to confirm liver cirrhosis, all patients underwent Upper GI endoscopy and colonoscopy. CTP and MELD score were calculated in all patients. The study was approved by the institutional ethics committee and the participants provided written informed consent.

Results: There were 68 males and 19 female patients, the mean age was 53.6 years. Mild anaemia was present in 29.9% of cases, moderate anaemia in 49.4%, and severe anaemia in 20.7% of cases. On upper GI endoscopy, mild PHG was present in 67.8%, severe PHG in 31% and GAVE was present in 2.3%. On colonoscopic examination, portal hypertensive colopathy was present in 26.4% and rectal varices in 10.3%. Mild PHG and severe PHG were statistically significantly correlated with the CTP score ($p = 0.006/0.003$). The correlation between PHC and severity of underlying liver disease (CTP status/MELD score) was not statistically significant ($p = 0.062/0.431$). The correlation between Hb and PHC was statistically significant ($p = 0.001$). There was a difference in the Hb values between the patients with or without PHC (8.4±1.6 gm/dl vs. 9.9±1.8 gm/dl, $p = 0.001$). There was a difference in the Hb values between the patients with or without severe PHG (8.3±1.6 gm/dl vs. 10±1.7 gm/dl, $p < 0.001$). The correlation between PHG and PHC was statistically significant ($p < 0.001$). PHC was present in 73.9% of patients with severe PHG, only 26.1% of patients with mild PHG had PHC.

Conclusion: Mild PHG and severe PHG were statistically significantly correlated with the CTP score. The presence of severe PHG on upper gi endoscopy was associated with severe anaemia (Hb < 8gm/dl) and the presence of PHC on colonoscopy was associated with lower Hb (PHC absent Hb-9.9±1.8 gm/dl, PHC present Hb-8.4±1.6 gm/dl). The correlation between the increasing severity of PHG and the presence of PHC was statistically significant.

Keywords: Portal Hypertensive Gastropathy (PHG), Portal Hypertensive Colopathy (PHC), Anaemia.

Introduction

Any type of chronic liver disease might lead to the significant complication of PHT (portal hypertension). One of the most important medical effects of portal hypertension in people with cirrhosis is the development of GAVE (Gastric Antral Vascular Ectasia), PHG (Portal Hypertensive Gastropathy),gastroesophageal varices, ectopic varices and Portal hypertensive colopathy. In individuals with portal hypertension, these vascular lesions are thought to be a major cause of acute upper gastrointestinal bleeding, chronic blood loss and anaemia. In patients with portal hypertension, it has been noted in recent years that the entire gastrointestinal tract, including the stomach, is involved, as the venous drainage occurs through the portal venous system. There have been descriptions of involvement in the colon, small intestine, and duodenum. However, the upper gastrointestinal tract and colon in patients with portal hypertension have only been the subject of a small amount of research.

PHG(Portal hypertensive gastropathy) and PHC (Portal Hypertensive Colopathy) are important clinically because they lead to acute GI bleeding and/or chronic blood loss. PHG is defined as the mucosal changes in the gastric mucosa of patients with portal hypertension, the primary pathologic change is characterized by vascular ectasia. PHG is recognized endoscopically as a mosaic-like pattern called snake skin mucosa with or without red spots. PHC is the term used to describe colorectal mucosal lesions seen in patients with portal hypertension. Even though it's unclear how much of a clinical impact these lesions have on patients with portal hypertension, they are believed to be significant causes of lower gastrointestinal bleeding. Multiple vascular-looking lesions (telangiectasias, cherry red patches, and angiodysplasia-like lesions), colitis-like abnormalities (granularity, erythema, oedema, friability), colorectal varices, or a combination of these findings are among the poorly characterised colonoscopic features of PHC. There is confusion surrounding the clinical significance and diagnostic criteria for this condition. Uncertain terminology, inconsistent endoscopic descriptions, inconsistency between observers, and a lack of distinctive histopathologic traits could all contribute to this. It is commonly recognized that Acute esophageal variceal bleeding is managed mainly by EVL (Endoscopic Variceal Band Ligation) and previously by EST (Endoscopic Sclerotherapy). Most studies have shown that individuals who have undergone EVL or EST have a higher risk of PHG. PHG and PHC result in chronic gastrointestinal blood loss and iron deficiency anaemia in cirrhotics. In this study, we evaluated the prevalence of portal hypertensive gastropathy and portal hypertensive colopathy in patients with chronic liver disease with anaemia.

Aims and Objectives

- To document portal hypertensive gastropathy in a given cirrhotic with anaemia
- To document portal hypertensive colopathy in a given cirrhotic with anaemia.
- To correlate portal hypertensive gastropathy or portal hypertensive colopathy with the severity of liver disease
- To correlate portal hypertensive gastropathy or portal hypertensive colopathy with the severity of anaemia

Materials & Methods

87 patients participated in this hospital-based prospective cross-sectional study, which was carried out from January 2016 to July 2017 at the Department of Gastroenterology, M. S. Ramaiah Medical College, MSRIT, Bengaluru. The patients underwent a thorough clinical evaluation, history, and physical examination, Biochemical investigations like CBC, LFT, PT-INR, Peripheral smear, Serum iron studies, Vitamin B12 levels, Viral markers-HBsAG and Anti-HCV, etiological workup for the underlying cirrhosis was done, USG abdomen was performed to confirm liver cirrhosis, all patients underwent Upper GI endoscopy and colonoscopy. CTP and MELD score were calculated in all patients. The study was approved by the institutional ethics committee and the participants provided written informed consent.

Inclusion Criteria

- Age above 18 years
- Anaemia with chronic liver disease (Hb<12.9 gm/dl)
- Anaemia with imaging evidence of chronic liver disease
- Anaemia with laboratory evidence of cirrhosis (low albumin, A: G reversal, low platelet count, elevated PT/INR)
- Anaemia with a fibroscan value >14 Kpa
- Anaemia with a liver biopsy evidence of cirrhosis

Exclusion Criteria

- EHPVO (Extra Hepatic Portal Vein Obstruction)
- Acute liver cell failure
- Patients with known hepatocellular carcinoma or extrahepatic malignancy
- Patients with active GI bleeding
- Patients with documented recent GI bleeds (< 30 days)

Statistical Methods

This work has used both descriptive and inferential statistical analysis. Results for categorical measurements are provided in numbers (%), whereas results for continuous measurements are displayed as mean±SD (min-max). At the five percent significance level, significance is evaluated. The following data-related assumptions are made: 1. A normal distribution is required for dependent variables. 2. Random samples taken from the population and independent cases for each sample

To determine the relevance of study parameters between three or more patient groups, an ANOVA (Analysis of Variance) has been used. The significance of research parameters on a continuous scale between two groups (inter group analysis) on metric parameters has been determined using the student t-test (two-tailed, independent).

Fisher or Chi-square The importance of research parameters on a categorical scale between two or more groups has been determined using an exact test in a non-parametric setting for qualitative data analysis. Fisher's exact test was used when cell samples were very small.

Significant figures

+ Suggestive significance (P-value: 0.05<P<0.10)

* Moderately significant (P value:0.01<P ≤0.05)

** Strongly significant (P value : P≤0.01)

Statistical Software: The statistical software, namely SPSS 18.0 and R environment version 3.2.2 were used for the analysis of the data and Microsoft Word and Excel were used to generate tables.

Results

Table 1: Association of Mild PHG and Severe PHG in Relation to MELD Score

		MELD Score				
	<10 (n=38)	10-18 (n=35)	19-24 (n=9)	>24 (n=5)	Total (n=87)	P-Value
Mild PHG						
□No	8(21.1%)	12(34.3%)	4(44.4%)	4(80%)	28(32.2%)	0.042*
□Yes	30(78.9%)	23(65.7%)	5(55.6%)	1(20%)	59(67.8%)	
Severe PHG						
□No	31(81.6%)	23(65.7%)	5(55.6%)	1(20%)	60(69%)	0.023*
□Yes	7(18.4%)	12(34.3%)	4(44.4%)	4(80%)	27(31%)	
Chi-Square/Fisher Exact Test						

In our study, mild and severe PHG were correlated with the MELD score. In patients with a MELD score between 10 and 18, 65.7% had mild PHG. 80% of patients with a MELD score >24 had severe PHG.

Table 2: Comparison of Clinical Variables According to MELD Score

Variables	MELD Score				Total	P-Value
	<10	10-18	19-24	>24		
Age in years	53.58± 11.84	54.69± 9.73	50.56±16.4	52.00±12.02	53.62±11.45	0.795
Haemoglo-bin (g/dl)	10.14± 1.80	8.96± 1.87	8.84±1.26	10.00±1.71	9.52±1.85	0.025*
TLC	6270.21± 2423.45	7865.37± 4988.60	11111.11± 5044.71	10168.00± 3276.82	7636.74± 4208.86	0.006**
Platelet Count	119447.47± 62153.80	101257.18± 60343.24	129000.00± 74188.95	127600.00± 120964.04	113586.26± 66389.09	0.537
Serum Iron	32.71± 18.39	30.40±28.17	19.00±7.75	20.20±9.23	29.64±22.14	0.292
TIBC	355.32± 79.58	370.69± 93.44	386.89±121.81	342.40±166.03	364.02±94.72	0.741
Ferritin	101.38± 287.04	110.86± 253.18	65.78±116.05	113.80±173.96	102.22±252.24	0.972
Trasferrin	22.49± 12.63	21.01± 25.84	16.44±13.27	15.20±11.95	20.85±18.99	0.757
Vita-minB12	468.65± 215.80	578.48± 412.45	828.78±557.32	517.20±143.45	552.88±358.58	0.051+
PT INR	1.15± 0.07	1.48± 0.25	2.03±0.69	2.51±0.75	1.45±0.49	<0.001**

The association between haematological variables and age with the MELD score was not statistically significant (p > 0.01), except for PT/INR

Table 3: Association of Mild PHG and Severe PHG in Relation to CTP Score

PHG	CTP			Total (N = 87)	P-Value
	A (N = 41)	B (N = 35)	C (N = 11)		
Mild PHG					
□No	7 (17.1%)	14 (40%)	7 (63.6%)	28 (32.2%)	0.006**
□Yes	34 (82.9%)	21 (60%)	4 (36.4%)	59 (67.8%)	
Severe PHG					
□No	35 (85.4%)	21 (60%)	4 (36.4%)	60 (69%)	0.003**
□Yes	6 (14.6%)	14 (40%)	7 (63.6%)	27 (31%)	
Chi-Square/Fisher Exact Test					

Mild PHG and severe PHG were significantly associated with the CTP score (p = 0.006/0.003). Out of 11 patients with CTP-C status, mild PHG was present in 4 (36.4%) and severe PHG in 7 (63.6%).

In 35 patients with CTP-B status, mild PHG was present in 21 (60%) and severe PHG in 14 (40%). CTP-A status was present in 41 patients, 34 (84.9%) patients had mild PHG and 6 (14.6%) had severe PHG with worsening of liver disease.

Table 4

Haemoglobin (g/dl)	PHC		Total
	No	Yes	
<8	7(10.9%)	11(47.8%)	18(20.7%)
8-10.9	34(53.1%)	9(39.1%)	43(49.4%)
11-12.9	23(35.9%)	3(13%)	26(29.9%)
Total	64(100%)	23(100%)	87(100%)
Haemoglobin Levels According to Incidence of PHC			
Haemoglobin (g/dl)	**PHG**		**Total**
	Mild	**Severe**	
<8	6(10%)	12(44.4%)	18(20.7%)
8-10.9	30(50%)	13(48.1%)	43(49.4%)
11-12.9	24(40%)	2(7.4%)	26(29.9%)
Total	60(100%)	27(100%)	87(100%)
Haemoglobin (g/dl) Levels According to Incidence of PHG			
P<0.001**, Significant, Chi-Square Test			

We observed a difference in the Hb values between the patients with or without severe PHG (8.3±1.6 gm/dl vs. 10±1.7 gm/dl, p < 0.001)

Table 5: PHG in Association with PHC

PHG	PHC		Total
	No	Yes	
Mild	54(84.4%)	6(26.1%)	60(69%)
Severe	10(15.6%)	17(73.9%)	27(31%)
Total	64(100%)	23(100%)	87(100%)
P<0.001**, Significant, Chi-Square Test			

The association between PHG and PHC was statistically significant (p < 0.001). PHC was present in 73.9% of patients with severe PHG, only 26.1% of patients with mild PHG had PHC.

Discussion

Aetiology of Underlying Cirrhosis

In our study, ethanol was the most common aetiology of cirrhosis in 50 (57.5%) patients, followed by non-alcoholic steatohepatitis in 19 (21.8%) patients, chronic hepatitis B in 11 (12.6%), hepatitis C in 4 (4.6%), AIH in 2 (2.3%) and cryptogenic in 1 (1%)

The majority of patients with cirrhosis usually have underlying portal hypertension and then develop portal hypertensive gastropathy and portal hypertensive colopathy; ultimately, these patients have acute or chronic gastrointestinal blood loss resulting in anaemia.

Cirrhosis Aetiology and PHG/PHC

In our study, we did not find a relationship between the underlying etiology of cirrhosis and PHG/PHC frequency or severity. There were no appreciable variations in the aetiology of cirrhosis between patients without PHG and people with mild or severe PHG in endoscopic research conducted by Iwao et al. [1] involving 47 patients with histologically established cirrhosis.

Prevalence of PHG in Cirrhosis with Anaemia

According to reports, the incidence of PHG in cirrhotic individuals varies from 25% to 50%, whereas the prevalence ranges from 11% to 98%.

In our study, PHG was present in 86 out of 87 patients (98. 8%). The prevalence of mild portal hypertensive gastropathy was 67.8% and severe portal hypertensive gastropathy was 31%.

This wide variability likely reflects variability in classification criteria, interpretation of endoscopic lesions, study populations, and the natural history of PHG. [2,3,4]

PHG and Liver Disease Severity

According to our study, in patients with cirrhosis and anaemia, a CTP (Child turcott pugh) score of >9 and a MELD (Model for end stage liver disease) score of >12 were substantially linked to the

existence and severity of PHG. In our study, the association between mild and severe PHG and the CTP score was statistically significant (p-value = 0.006/0.003).

Of the 11 individuals with CTP-C, 7 (63.6%) had severe PHG. PHG was shown to be 87% more common in patients with Child-Pugh stage C than in those with Child-Pugh stage A, according to Sarin et al.'s research. [5] Only Child-Pugh stage C was found to be independently linked with PHG (OR = 2.68; 95% CI: 1.16-6.20, P = 0.021) in another investigation. [6] In a study of 222 patients with cirrhosis, 48 patients developed PHG. Merli et al. [7] found that the presence of oesophageal varices and Child-Pugh stage B or C were independent risk factors for developing PHG.

In our study, the severity of PHG was statistically significantly correlated with the MELD score. Out of 5 patients who had MELD >24, 80% had severe PHG. Comparable to a recent study by Young et al. showing that a MELD score of 11.3±3.5 predicts severe PHG. The mean MELD score in patients without PHG was 7.6 ± 1.7, in patients with mild PHG it was 10.2 ± 4.0, and in patients with severe PHG it was 11.3 ± 3.5; P < 0.001), and there was a significant correlation between the MELD score and PHG severity in another study by Kim et al.[8]

Similar to this, Pan et al. study [9] found that the development of PHG is less affected by the presence or absence of gastric varices and the severity of cirrhosis (Child-Pugh grade). Additionally, Abbas et al. [10] were unable to discover a relationship between the severity of PHG and the Child-Pugh and MELD scores. Thus, opinions regarding the connection between PHG and liver function are still divided.

PHG–Grade of Varices

In our study, the severity of PHG was associated with an increasing grade of oesophageal varices (p < 0.01).

Out of 34 patients with large oesophageal varices, 21 (61.8%) had severe PHG and 32.8% had mild PHG.

PHG prevalence was significantly higher in patients with oesophageal varices [80 of 104 patients, 77%] than in patients without oesophageal varices [51 of 84 patients, 61%; P = 0.007] in the NIEC study, which included 188 of 373 patients with cirrhosis who were not receiving variceal sclerotherapy. Additionally, PHG prevalence increased significantly with increasing variceal size ($\chi 2 = 13.2$, P < 0.0003). [11] Among 230 cirrhotic patients, Gupta et al. [12] found no correlation between the extent of oesophageal varices and the prevalence of PHG. Comparably, Bellis et al.'s research of 59 cirrhosis patients [13] revealed a non-significant trend towards more severe PHG in those with big vs. minor varices. According to Abbasi et al. [14] there was a strong correlation (r = 0.46; P < 0.001) between the size of the oesophagus and the incidence of PHGs in 217 individuals with cirrhosis.

Profile of Anaemia in Cirrhosis

We classified the anaemia in cirrhosis into mild (11-12.9 gm/dl), moderate (8-10.9 gm/dl) and severe (< 8 gm/dl)

20.7% had severe anaemia, 49.4% had moderate anaemia and 29.9% had mild anaemia. The severity of anaemia was significantly associated with PHG and PHC (p-value < 0.01). 47.8% of patients with severe anaemia had PHC.

The severity of anaemia was not significantly associated with the MELD score or CTP score (p-value > 0.05).

It was reported that about 10% of gastropathy causes anaemia due to chronic blood loss, 2.5% of patients experience acute bleeding, and the mortality rate related to acute bleeding reaches 12.5%.

18 patients had severe anaemia (Hb<8 gm%); PHC was present in 47.8%; mild PHG in 10%; and severe PHG in 44.4%.

43 patients had moderate anaemia (Hb=8-10.9gm%), PHC was present in 39.1% and mild PHG in 50% and severe PHG in 48.1%

26 patients had mild anaemia (Hb-11-12.9gm%), PHC-13%, mild PHG-40%, and severe PHG in 7.4%; vitamin B12 deficiency in 2.3%, anaemia of chronic disease in 37.9% and iron deficiency anaemia in 59.77%.

Anaemia is one of the most important consequences of chronic bleeding from PH-related lesions. We observed differences in haemoglobin values between patients with and without PHC. We also observed a statistically significant correlation between the presence of anaemia and the severity of portal hypertensive gastropathy.

PHC in Cirrhotics with Anaemia

The study found that 26.4% of participants had portal hypertensive colonopathy. In 60 patients with cirrhosis and portal hypertension, Misra et al., prospective study [15] found that 57% of them had portal hypertensive colonopathy.

Comparison of Prevalence of PHC in Various Published Studies

Prevalence of Rectal Varix

The incidence of rectal varix was 10.3% in our investigation. Tam et al. found that 16% of 75 patients with cirrhosis and portal hypertension (of whom more than 80% were positive for HBV or HCV) had rectal varix in their prospective analysis. Another prospective study by Ghosal et al. [16]

examined the lower gastrointestinal tract colonoscopically in 41 patients with cirrhosis and portal hypertension. The results showed that 36% of these patients had rectal varix. Additionally, they found that the occurrence of haematochezia is correlated with the presence of rectal varix rather than portal hypertensive colopathy, and that no parameter, including CTP class and oesophageal variceal eradication by EST with or without EVL, predicted the occurrence of either condition. Interestingly, Goenka et al. found that up to 89% of 75 patients with cirrhosis and portal hypertension had rectal varix in another prospective investigation. [17]

Comparison of Prevalence of Rectal Varix in Various Published Studies

Prevalence of Haemorrhoids

Haemorrhoids were present in 58.6% of the participants in our study. In the adult population, haemorrhoids affect 10% to 25% of people. [18] In order to assess the prevalence of haemorrhoids, a prospective study conducted by Bresci et al. [19] included 85 patients with cirrhosis and portal hypertension who did not have any other important diseases. The results showed that the prevalence of haemorrhoids was surprisingly high, reaching up to 70%.

Comparison of Prevalence of Haemorrhoids in Various Published Series

The decreased frequency of rectal varices and haemorrhoids in our investigation compared to the other published series could be attributed to variations in the patient populations examined, interobserver variability among endoscopists, or variations in the rationale for colonoscopy. In contrast to our investigation, which included patients with cirrhosis and portal hypertension but no overtly lower gastrointestinal blood loss, the majority of the aforementioned studies have performed colonoscopies on patients who had overtly lower gastrointestinal symptoms.

Correlation of PHC and Severity of Liver Disease

In the present study of 23 patients with portal hypertensive colopathy, Child Pugh-A was 26.1%, Child Pugh-B was 56.5% and Child Pugh-C was 17.4%. In our study, the relationship between the presence of PHC and CTP status was not statistically significant ($p = 0.062$). There was no increase in the prevalence of PHC with worsening CTP status, this was similar to the studies published by Kozarek et al., [20] Ganguly et al., [21] and Bresci et al.

In our study, the relationship between the presence of portal hypertensive colopathy and an increasing MELD score was not statistically significant ($p = 0.431$). Of the 23 patients who had portal hypertensive colopathy, 47.8% had a MELD score between 10 and 18 and only 8.7% had a MELD score >24.

PHC and oesophageal Variceal Grading Correlation

In our study, 69.5% of patients with PHC had large oesophageal varices, 26.1% had small oesophageal varices, and 4.3% had obliterated varices. Thus, our research showed that the likelihood of having PHC rises as oesophageal varices grow in size, a finding that was statistically significant (p-value < 0.01). Except for a single prospective study by Misra et al. that demonstrated a higher incidence of PHC in patients with larger oesophageal varices, nearly all previously published studies (N.EL. Kady et al., [22] Keiichi et al., and Ghosalet al.) that evaluated the relationship between PHC and oesophageal variceal grading revealed no relationship at all.

PHC and Site Predilection

Similar to the findings of Misra et al. study, more than 50% of the patients in our investigation had major left-sided colonic involvement. On the other hand, 74% of the patients in a retrospective analysis by Bini et al. [23] had diffuse colonic involvement. Right colonic involvements were more common than left colonic involvements in a different prospective research study conducted by Kozarek et al.

Colonic Varices

Colic varices were not present in any of the patients in either group in our investigation. In line with our research, Misra et al. and Naveau et al. [24] similarly reported that in their cohort of 50 and 100 patients, respectively, no patients developed colonic varices. In a different investigation, just one of the fifty patients was found to have colonic varices by Ganguly et al.

PHG Relationship to PHC

In our study, out of 23 patients who had portal hypertensive colopathy, 26.1% had mild PHG and severe PHG was present in 73.9%. In our study, the relationship between the increasing severity of PHG and the presence of PHC was statistically significant (p-value < 0.01).

In a study done by Yamakado et al. [25] PHC is significantly associated with PHG, out of 27 patients with PHC, PHG was present in 24 patients ($p = 0.0230$). In contrast, Ito K [26] et al. showed that the correlation between PHC and the presence of PHG was not statistically significant (P=NS).

Conclusion

The prevalence of portal hypertensive gastropathy in cirrhotics with anaemia in our study was 98.8%, mild PHG in 67.8% and severe PHG in 31%. Mild PHG and severe PHG were both significantly linked to the CTP score. The prevalence of severe PHG

rose as liver function decreased and liver disease got worse. The prevalence of portal hypertensive colopathy and rectal varices in our study was 26.4% and 10.3% respectively. The profiles of anaemia in cirrhotics in our study were iron deficiency anaemia in 59.7%, anaemia of chronic disease in 37.9% and vitamin B12 deficiency anaemia in 2.3%. In our study, the presence of severe PHG on endoscopy was associated with severe anaemia (Hb<8 gm/dl). In our study, the presence of PHC was associated with lower Hb (no PHC, Hb-9.9±1.8 gm/dl, PHC present, Hb-8.4±1.6 gm/dl). In our study, the relationship between the increasing severity of PHG and the presence of PHC was statistically significant.

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