Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

General Medicine

Portal Hypertensive Gastropathy and its Association among the Patients with Decompensated Chronic Liver Disease

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DOI: 10.36347/sjams.2023.v11i05.015 | **Received:** 08.03.2023 | **Accepted:** 25.04.2023 | **Published:** 18.05.2023

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Abstract **Original Research Article**

Background: Although portal hypertension remains the crucial factor for the development of PHG, other factors should be considered in the development and progression of this condition like hepatic function, oesophageal varices, type of hepatitis virus and hypersplenism as evidenced by haematological variables (Anemia, leucopenia and thrombocytopenia). ***Aim & Objectives:*** To study the frequency of portal hypertensive gastropathy and its correlation with haematological and biochemical parameters and other endoscopic findings. ***Materials and methods:*** A prospective, hospital based, time bound study of 100 patients, admitted with decompensated chronic liver disease (diagnosed based on the evidence of sonography criteria) in a tertiary care center. Patients aged > 18 years and above admitted in SDM medical college and hospital were considered, demographic data, physical examination was done, basic investigations, hematological, biochemical (renal function test and liver function test) , coagulation profile, serology were evaluated. Upper gastrointestinal endoscopy procedure was done and data was collected in a pre designed performa. ***Results:*** The frequency of PHG in our study was 64%.Presence of PHG had significant association with SBP, DBP, haemoglobin, TLC. We also found significant association between PHG and grade 1 and 3 of esophageal varices, gastric varices and erosive gastritis. ***Conclusion*:** The frequency of PHG was 64% in the studied group and the presence of PHG correlates with presence of esophageal varices, erosive gastritis and gastric varices suggesting a common pathophysiology.

Keywords: Portal hypertensive gastropathy, Esophageal varices, Cirrhosis, Child-Pugh score.

INTRODUCTION

Portal hypertensive gastropathy refers to changes in the mucosa of the stomach in patients with portal hypertension by far the most common cause of this is cirrhosis of the liver. The prevalence of PHG in cirrhotic patients has been reported to be variable, ranging between 11% to 98%, while the incidence varies from 25% to 50%. Portal hypertensive gastropathy is defined as presence of mosaic-like pattern or a diffuse, erythematous, and reticular cobblestone pattern of stomach mucosa with or without superimposed red punctate lesions >2 mm in diameter and a depressed white border is the distinctive appearance of portal hypertensive gastropathy. Esophagogastroduodenoscopy (EGD) findings are used to diagnose portal hypertensive gastropathy (PHG) in patients. Portal hypertensive gastropathy (PHG) is a common finding in patients with cirrhosis of liver with portal hypertension, which in recent years has been recognized as a cause of acute or insidious gastrointestinal bleeding in these patients. With this background, we aimed to study the frequency of portal hypertensive gastropathy and its correlation with haematological and biochemical parameters and other endoscopic findings.

A mosaic-like pattern or a diffuse, erythematous, and reticular cobblestone pattern of stomach mucosa with or without superimposed red punctate lesions, >2 mm in diameter, and a depressed white border is the distinctive appearance of portal hypertensive gastropathy, which is defined by this [1-3]. Esophagogastroduodenoscopy (EGD) findings are

used to diagnose portal hypertensive gastropathy (PHG) in patients [4].

The prevalence of PHG in cirrhotic patients has been reported to be variable, ranging between 11% and 98%, while the incidence varies from 25% to 50% [5, 6]. The pathogenesis of portal hypertensive gastropathy may be related to both congestion and hyperemia in the stomach. Patients with PHG are often asymptomatic and are diagnosed when endoscopy is performed for other reasons. Hence it should be suspected in patients who have risk factor for developing portal hypertension.

Portal hypertensive gastropathy is typically diagnosed endoscopically, it characteristically appears as a fine, white, reticular, pattern with backgroud pinkish mucosa giving a snake skin pattern. These patients need to be treated with primary prophylaxsis with non-selective beta blockers.

Portal hypertensive gastropathy (PHG) is a rather common finding in patients with liver cirrhosis and portal hypertension, which in recent years has been recognized as a cause of acute or insidious gastrointestinal bleeding in these subjects.[5] In the last two decades, portal hypertensive gastropathy (PHG) has emerged as a new entity in various devastating complications of chronic liver disease.

According to certain theories, PHG is a dynamic disorder that can not only get better or perhaps go away entirely, but also get worse from mild to severe [5]. This finding suggests that although portal hypertension is still a major factor in the development of PHG, other factors, such as hepatic function, oesophageal varices, the type of hepatitis virus, and hypersplenism as shown by haematological variables, should be taken into account in the onset and progression of this condition (anemia, leucopenia and thrombocytopenia).

With this background, we aimed to study the frequency of portal hypertensive gastropathy and its correlation with haematological and biochemical parameters and other endoscopic findings.

Aims and Objectives

To study the frequency of portal hypertensive gastropathy and its correlation with haematological and biochemical parameters and other endoscopic findings.

Inclusion Criteria:

- All patients aged >18years age of either gender, diagnosed to have cirrhosis of liver with portal hypertension admitted in Medicine wards and icu. S.D.M College of Medical Sciences and Hospital, Sattur, Dharwad 580009.
- Patients willing to participate in the study

Exclusion Criteria:

- Portal hypertension requiring emergency management
- History of trauma
- Non-cirrhotic portal hypertension
- Portal hypertension with mass per abdomen

MATERIAL AND METHODS

Ours is a Prospective study. Data were collected from medical records of patients admitted to our hospital from 20/02/2021 to 20/02/2022 in wards and ICU after the Institutional Ethical Clearance. One hundred patients, admitted with decompensated chronic liver disease (diagnosed based on the evidence of sonography criteria) were enrolled. We disqualified patients with severe renal or cardiac impairment occlusion, previous history of chronic small-bowel disease, history of recent or current intake of NSAIDs or vasoactive drugs. A consistent protocol was applied to all of the patients, which included taking a thorough demographic details, clinical history, medical history and performing systemic examinations. Complete history comprised contemporary history as well as demographic information (name, age, gender, address etiology of PHT, and previous history of upper gastrointestinal bleeding and any endoscopic intervention), laboratory (complete blood count, liver function tests, and prothrombin time and renal function tests), radiological (abdominal ultrasonography), endoscopic and histological characteristics.The severity of cirrhosis was classified according to the Child-Pugh criteria. Portal hypertensive gastropathy was graded according to McCormack.[3]PHE changes were graded according to De Palma *et al*., [4] which included mucosal inflammatory-like abnormalities (fold thickening, edema, erythema, granularity and friability) and vascular lesions (cherry red spots, telangiectasia, or angiodysplasia-like lesions, and varices).The Statistical Package for Social Sciences (SPSS), 21 version, was used to analyze the collected data. Student's t-tests for quantitative variables and χ^2 test for categorical variables were used to assess the associations. All significance tests used a 5% level of significance.

Ethical Committee:

Permissions – taken from the Institutional Ethics Committee and medical records department (Ref: SDMCMS&H/IEC2021, Date: 19/02/2021).

RESULTS

There were 100 patients whose data were collected. Demographic variables of patients is shown in Table1. Out of 100 patients, 12 patients (12%) were in the age group of 20–35 years, 42 patients (42%) seen were in the age group of 36–50 years, 26 patients (26%) were in the age group of 51-65 years, 15 patients(15%) were in the age group of 66-80 years and 5 patients(5%

) were in the age group of 81-95 years, and consisting 82 male patients (82%) and 18 female patients (18%).

Clinical characteristics of the patients are shown in Table.2. Among 100 patients ,61 patients(61%) were classified as Child-Pugh class C ,29 patients(29%) were classified as Child-Pugh class B, 10 patients(10%) were classified as Child-Pugh class C. Upper GI endoscopy is done in all the 100 patients, 14 patients(14%) had normal endoscopy,86 patients(86%) had abnormal endoscopy findings. among them 64% had PHG, 33% had grade 2 varices, 28% had grade 1 varices, 22% had grade 3 varices, 21% had erosive gastritis, 15% had gastric varices, 14% had duodenitis and 6% had hiatus hernia. Association of clinical and laboratory parameters with PHG is shown in Table 3. It was observed that presence of lower blood pressure recordings (SBP- 107±15, DBP- 68 ±10), low haemoglobin (SD- 8.2 ± 1.9), leukocyte count (SD- 10157 ± 5398) have strong association with PHG with P value<0.05 . Association of upper GI endoscopic features with PHGis shown in Table 4. It was observed that the presence of Grade 1 (35.9%) and Grade 3 (29.7%) esophageal varices, gastric varices (20.3%) and erosive gastritis (12.5%)have strong association with PHG with P value<0.05.

Table 1: Demographic parameters of study participants

Demographic variables		**Frequency**
Age	**20 to 35 years**	12%
	36 to 50 years	42%
	51 to 65 years	26%
	66 to 80 years	15%
	81 to 95 years	5%
Gender	**Males**	82%
	Females	18%

Table 2: Clinical characteristics in study subjects

	Clinical characteristics		Frequency
Child Pugh class	A		10%
	B		29%
	C		61%
Upper GI endoscopy	NORMAL	14	14%
	ABNORMAL	86	86%
	AMONG 86 PATIENTS		
	Esophageal varices	Grade 1	28%
		Grade 2	33%
		Grade 3	22%
	Gastric varices		15%
	PHG		64%
	Erosive gastritis		21%
	Hiatus hernia		6%
	Duodenitis		14%

Table 3: Association of clinical and laboratory parameters with PHG

Clinical and laboratory parameters		**PHG**		**p-value**
		Present (n=64)	**Absent (n=36)**	
Clinical	**SBP (mmHg)**	107 ± 15	115 ± 16	**0.011**
	DBP(mmHg)	68 ± 10	73 ± 12	**0.014**
Complete blood count	**Haemoglobin (gm/dl)**	8.2 ± 1.9	9.4 ± 1.9	**0.003**
	TLC (/cumm)	10157 ± 5398	7876 ± 3101	**0.022**
	Platelets (/cumm)	1.27 ± 0.69	1.4 ± 1.09	0.467
	PT	23.43 ± 8.20	22.29 ± 6.52	0.476
	INR	2.07 ± 1.19	2.05 ± 0.71	0.927
Renal function tests	**Serum Urea (mg/dl)**	46.3 ± 27.2	38.6 ± 21.3	0.147
	Serum Creatine (mg/dl)	1.54 ± 0.95	1.44 ±1.11	0.627
	Total Protein (mg/dl)	6.13 ± 0.88	6.45 ± 0.78	0.073
Liver function tests	**Albumin (mg/dl)**	2.01 ± 0.59	2.23 ± 0.63	0.081
	Total Bilirubin (mg/dl)	5.66 ± 7.14	4.28 ± 4.89	0.303
	AST (IU/L)	90 ± 82	82 ± 58	0.620
	ALT(IU/L)	51 ± 50	44 ± 21	0.390
	ALP (IU/L)	131 ± 62	138 ± 126	0.707

Table 4: Association of upper GI endoscopic features with PHG

Upper GI endoscopic features		PHG		p-value
		Present (n=64)	Absent (n=36)	
Esophageal varices	**Grade 1**	23 (35.9%)	5 (13.9%)	**0.018**
	Grade 2	25 (39.1%)	8 (22.2%)	0.086
	Grade 3	19 (29.7%)	3 (8.3%)	**0.013**
Gastric varices		13 (20.3%)	2 (5.6%)	**0.047**
Erosive gastritis		8 (12.5%)	13 (36.1%)	**0.005**
Hiatus hernia		4 (6.3%)	2 (5.6%)	0.888
Duodenitis		8 (12.5%)	6 (16.7%)	0.564

DISCUSSION

PHG has been recently recognized as an important complication of cirrhosis with portal hypertension. It is more frequently observed in patients with more severe liver disease and in patients with cirrhosis who have had previous endoscopic treatment with sclerotherapy or endoscopic variceal ligation [1, 2, 5-7].

The frequency of PHG in our study was 64%, which was comparable to studies by Abbasi *et al*., [8] (79.27%), Kumar *et al*., [9] (55%), Tiwari *et al*., [10] (66.6%) and Ahmed *et al*., [11] (83.3%). The prevalence of PHG is varied in literature and it has been in between 16% to 100% in patients with cirrhosis [12].

We observed no association of liver function tests with presence of PHG. This observation was similar to study by Primignani *et al*., [1], Merkel *et al*., [13] Abbasi *et al*., [8].

Presence of PHG was associated with oesophageal varices grade. Our results regarding this relationship were in agreement with other researchers [11, 14], who also found this significant relationship between oesophageal varices and PHG. However, Tiwari et al., [14] did not find this association.

Researchers Parikh *et al*., [15], Kumar *et al*., [9], Bayraktar *et al*., [16], Pan *et al*., and Primignani *et al*., [1] found a strong correlation between the grade of varices and the presence and severity of PHG. On the other hand, PHG and the grade of varices were not related in any way, according to Gupta *et al*., [17], Dong *et al*., [18], Iwao *et al*., [19], and Yang *et al*., [20].

There could be a number of reasons for the discrepancies in the studies' findings. Secondly, there is interobserver variation because PHG is an objective diagnosis given during esophagogastroduodenoscopy. Varied classifications for categorising the severity of PHG can also be the reason of variation. For the same reason, esophageal varices also fall under these categories. The duration of the disease, usage of beta-blockers, or variceal ligation have all been linked to the severity of PHG [9, 17].

CONCLUSION

The frequency of portal hypertensive gastropathy is 64% and the presence of PHG correlates with presence of esophageal varices, erosive gastritis and gastric varices suggesting a common pathophysiology. PHG has statistical association with anemia as it can cause acute and chronic bleeding leading to iron deficiency anemia.PHG has no statistical significance with biochemical parameters.

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