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An endoscopic study on portal hypertensive colopathy in cirrhotic patients

Dr Hadi Meeran Hussain
Consultant Physician PAF Hospital, Lahore
Corresponding author email: drhadihussain234@gmail.com

Dr Nadeem Fazal
Head of Department of Medicine, Combined Military Hospital, Lahore

Dr Mohummad Farooq
Head of Department of Medicine, PAF Hospital, Lahore

Dr Amir Habib
Department of Gastroenterology, CMH Lahore

***Abstract*---**Background and Aim: Liver Cirrhosis patients are more prone to lower gastrointestinal (GI) bleeding hypothetically caused by portal hypertensive colopathy (PHC) and anorectal varices (ARVs). The present study aimed to determine the incidence of colopathic lesions and investigate their association with cirrhosis severity and portal hypertensive colopathy manifestation. Patients and Methods: This cross-sectional study was carried out on 80 liver cirrhosis patients in the Department of Gastrointestinal, CMH Hospital Lahore from January 2021 to December 2022. Individuals aged >18 years with liver cirrhosis were enrolled. Each individual underwent laboratory, clinical, and ultrasonographic examinations. Upper gastrointestinal endoscopy was used to detect the PHC. Based on the colopathic lesions severity, patients were categorized into three grades: grade I (colonic mucosa), grade II (mucosa with mosaic-like pattern), and grade III (angiodysplasia-like colon lesions). SPSS version 27 was used for data analysis. Results: The overall mean age was 48.62±5.6 years with an age range 19 to 70 years. Of the total patients, there were 60 (75%) male and 20 (25%) females. Child-Pugh classification of patients were as follows: Class A 16 (20%), Class B 29 (36.3%), and Class C 35 (43.7%). Portal hypertensive gastropathy was diagnosed in 72 (90%) cases. About 56 (70%) patients had esophageal varices and the most prevalent esophageal varices was grade 3. Of the total 68 (85%) cases of diagnosed colopathic lesions, the incidence of grade 1, grade 2, and grade 3 was 21 (30.9%), 29 (42.6%), and 18 (26.5%) respectively. Conclusion: The present study found that colopathic lesions are

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common in cirrhosis patients. Endoscopic therapy for varices increases the likelihood of acquiring greater stages of colopathy. The latter might be used to predict a poor outcome in people with HCV cirrhosis.

***Keywords*---**cirrhotic patients, portal hypertensive colopathy, endoscopy.

Introduction

Cirrhosis is a chronic liver disorders and 14th leading cause of mortality worldwide [1]. Portal hypertension and related complications such as hepatorenal syndrome, ascites, portopulmonary hypertension, hepatocellular cancer, variceal hemorrhage, and endocrine dysfunction etc. are various underlying causes of cirrhosis. Portal hypertension is defined as the exceeding of hepatic venous pressure gradient (HVPG) beyond 5 mm Hg. Portal hypertension associated complications are the leading cause for majority of liver transplantation and higher rate of mortality [2]. Portal hypertension affects the whole gastrointestinal (GI) system, causing hemodynamic and mucosal alterations [3, 4]. The upper GI tract lesions associated with portal hypertension include gastroesophageal varices, portal hypertensive gastropathy, and gastric antral vascular ectasia [5]. Additionally, mucosal and hemodynamic variations are considered to be caused by portal hypertension throughout the gastrointestinal tract, culminating in gastropathy and colopathy [6]. These two complications might leads to gastrointestinal hemorrhage [7]. However, it has been advised that cirrhotic patients to have colonoscopy in order to examine portal hypertension-related lesions in the colorectal area [8].

Esophagus varices, portal hypertensive gastropathy (PHG), and stomach are widely characterized by individual portal hypertension and liver cirrhosis [9]. These are the most prevalent causes of upper GI hemorrhage [10]. The incidence of ARV and PHC in liver cirrhosis patients were 40% being the prevalent cause of lower GI hemorrhage [11]. Based on Capsule endoscopic data, occult blood is caused by portal hypertensive enteropathy [12]. Despite numerous investigations over the last two decades, there are still many discrepancies about the prevalence and diagnostic criteria of colopathy [13]. There is paucity of data regarding PHC in cirrhosis patients posing significant challenges of managing these syndrome in HCV-related cirrhotic patients. Therefore, the present study aimed to determine the prevalence of colopathic lesions in cirrhotic patients and lesions severity associated with to portal hypertension.

Methodology

This cross-sectional study was carried out on 80 liver cirrhosis patients in the Department of Gastrointestinal, CMH Hospital Lahore from January 2021 to December 2022. Individuals aged >18 years with liver cirrhosis were enrolled. Each individual underwent laboratory, clinical, and ultrasonographic examinations. Upper gastrointestinal endoscopy was used to detect the PHC. Based on the colopathic lesions severity, patients were categorized into three

grades: grade I (colonic mucosa), grade II (mucosa with mosaic-like pattern), and grade III (angiodysplasia-like colon lesions). The presence of grade II hepatic encephalopathy, various etiologies of liver disease, and hemodynamic instability were the exclusion criteria. Child–Pugh's categorization into Class A, B, and C was used for the assessment of liver disease severity. Each patient's venous blood was tested for liver function tests (LFT), CBC, and fasting blood glucose. An upper gastrointestinal endoscopy was then conducted to determine the existence and grade of portal hypertensive gastropathy and esophageal varices.

SPSS version 27 was used for data analysis. The frequency was determined for qualitative data, and the 2-test was used to compare groups with high-degree colopathy to those without (or with just grade 1). Mean and standard deviation were determined for quantitative data, and one-way analysis of variance was used to compare groups when applicable. P values less than 0.05 were deemed significant.

Results

The overall mean age was 48.62±5.6 years with an age range 19 to 70 years. Of the total patients, there were 60 (75%) male and 20 (25%) females. Child-Pugh classification of patients were as follows: Class A 16 (20%), Class B 29 (36.3%), and Class C 35 (43.7%). Portal hypertensive gastropathy was diagnosed in 72 (90%) cases. About 56 (70%) patients had esophageal varices and the most prevalent esophageal varices was grade 3. Of the total 68 (85%) cases of diagnosed colopathic lesions, the incidence of grade 1, grade 2, and grade 3 was 21 (30.9%), 29 (42.6%), and 18 (26.5%) respectively. Patient's clinical details and laboratory data are shown in Table-I. Table-II represents the ultrasound and endoscopic findings. The incidence of colopathic lesion's grades are shown in Figure-1. Table-III represents the comparison of portal hypertensive colopathy different grades with liver disease severity and portal hypertension indicators.

Table-I Patient's clinical details and laboratory data

Variables	Value (mean ± SD)
Age (years)	48.62±5.6
Gender N (%) Male Females	 60 (75) 20 (25)
Classification of Child-Pugh class Class A Class B Class C	 16 (20) 29 (36.3) 35 (43.7)
Laboratory data Hemoglobin (g/dl) ALT Platelets (×10^3/mm^3) AST Bilirubin (mg/dl) Creatinine (mg/dl) Fasting blood glucose (mg/dl)	 12.46±1.79 42.92±17.62 88.57±41.69 47.26 ±23.13 2±0.92 0.85±0.42 106.0±31.21

Table-II ultrasound and endoscopic findings

Parameters	Value N (%)
Ascites N (%) Absent Mild Moderate	 17 (21.3) 29 (36.3) 34 (42.5)
Splenic size (mean ± SD)	14.89±2.42
PVD	12.54±1.89
PV velocity	12.47±2.56
Esophageal varices grading Grade 1 Grade 2 Grade 3 Grade 4	 5 (8.9) 15 (26.8) 23 (41.1) 13 (23.2)
PHC Grading Grade 1 Grade 2 Grade 3	 23 (31.9) 31 (43.1) 18 (25)

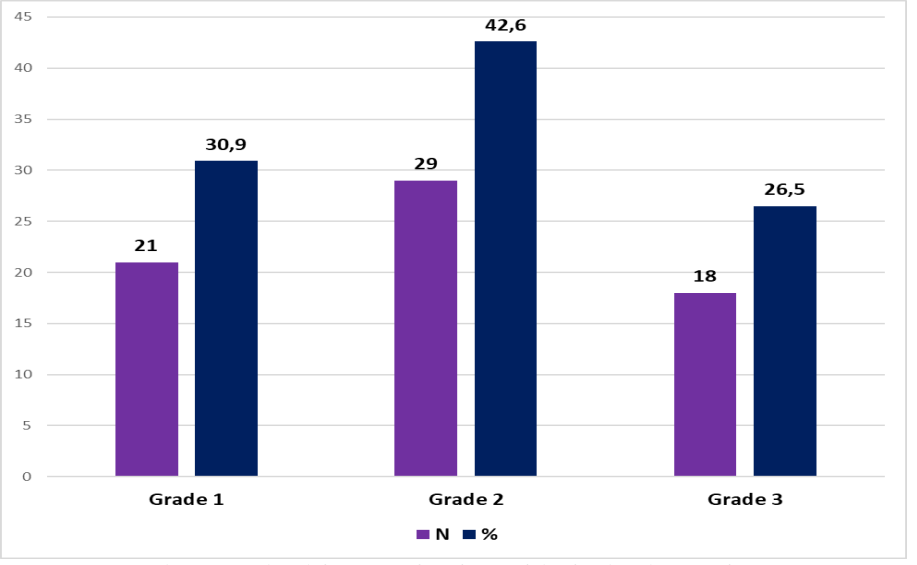

Figure-1 incidence of colopathic lesion's grades

Table-III comparison of portal hypertensive colopathy different grades with liver disease severity and portal hypertension indicators

Indicators	Absent (N=8)	PHC Grade 1 (N=23)	PHC grade 2 (N=31)	PHC grade 3 (N=18)	P-value
Splenic size (cm)	13.12±1.49	14.04±2.73	15.19±1.69	16.17±2.64	0.031
PVD (mm)	11.89±2.12	12.56±1.82	13.38±1.52	14.21±2.59	0.10
PV velocity (cm/s)	14.59±2.32	13.26±2.29	11.69±1.72	12.19±3.18	0.05
Platelets ($\times 10^3/mm^3$)	143.58±43.49	112.79±45.62	69.89±19.59	70.89±19.59	<0.001

Discussion

The present study mainly focused on the endoscopic study of portal hypertensive colopathy in cirrhotic patients and found that colopathic lesions are common in cirrhosis patients. Endoscopic therapy for varices increases the likelihood of acquiring greater stages of colopathy. The normal liver parenchyma replaced by various regenerating nodules and severe fibrosis are used to distinguished cirrhosis. The portal hypertension mechanism increased the intrahepatic resistance in cirrhosis patients due to fibrosis, with a portal flow simultaneous increase due to haemodynamic alterations. With severe liver illness, fibrosis and haemodynamic dysfunction intensify, resulting in greater portal pressure. The recent study predicted that the significant incidence of colopathic lesions (85%) in HCV-induced cirrhosis patients. According to published research, the incidence of colopathy among cirrhotic patients ranges from 25% to more than 80% [14, 15]. For the examination of acute lower GI bleeding, the significant choice for diagnosis with lower complication is Colonoscopy [16]. Upper GI hemorrhage is five times more common than rectal bleeding [17]. Suspected rectal bleeding can be effectively assessed by inquiry tool availability, bleeding severity, and mainly rely on expertise [18]. Hemodynamic issues such as PHC and colonic varices are caused by portal hypertension through gastrointestinal tract [19].

A previous study associated the increased portal pressure with colopathy [20]. Another study reported an association between colopathy, lower platelet count, and severe esophageal varices [21]. The hypertensive colopathy was related with lower gastrointestinal hemorrhage severity by Achim et al [22] who used Tran's jugular intrahepatic portosystemic shunt for immediate cease of bleeding. Hajjar et al [23] combined all the complications associated with portal hypertension including the enteropathy of portal hypertensive entity.

Our findings show that individuals who have received EVL or EST for esophageal varices had higher incidence of PHC. An earlier study reported that PHG increased incidence after EST causing complete variceal obliteration due to blood flow redistribution increases the mosaic pattern, vascular ectasias, and gastric mucosa seen on endoscopy pattern [24]. In contrast, some investigations showed no link between esophageal varices and colopathy [25, 26]. El-Toukhy et al. [27] found hemorrhoids in 25.3% cirrhotic patients, attributing to liver disease and portal hypertension severity, whereas Demetiou et al [28] reported that PHC endoscopic characteristic was hemorrhoids. All of them revealed increased portal hypertension. It is unclear if this reflects a direct relationship between the severity of the latter and the existence of liver cirrhosis patients.

Previous research on the relationship between the liver disease severity and colopathy produced inconsistent results [29]. Numerous studies corroborated this link [30]. Others, on the other hand, have been unable to confirm such a link [31]. Several of the latter trials contained a substantial number of individuals with minor liver disease, which appears to have influenced their findings. In the present study, Child-Pugh score was used to relate the advanced grading of colopathy with liver disease severity. As a result, poorer hepatic functions are likely to be accompanied with increased hemodynamic dysfunction and, as a result, colopathic lesions [32].

The current study found that patients who had previously had endoscopic care of esophageal varices had greater rates of colopathy. The blood flow via the portal system is redistributed as a result of the variceal obliteration. As a result, the pressure in the colonic mucosa rises, causing colopathic lesions to occur [33]. There are no standardized therapy recommendations for colopathic lesions. Octreotide or shunt operations may provide future hope for symptomatic individuals with these lesions [34].

Conclusion

The present study found that colopathic lesions are common in cirrhosis patients. Endoscopic therapy for varices increases the likelihood of acquiring greater stages of colopathy. The latter might be used to predict a poor outcome in people with HCV cirrhosis.

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