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Prevalence of Portal Hypertensive Colopathy in Patients with Cirrhosis in A Tertiary Care Hospital

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Abstract
Introduction: *Cirrhosis is defined by the World Health Organization (WHO) as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules. In cirrhosis, normal liver is replaced by fibrotic tissue and regenerative nodules leading to progressive loss of liver function. Cirrhosis is an important cause of mortality and morbidity. The clinical presentation of cirrhosis is variable depending on the etiology and whether the hepatocellular or portal hypertension predominates.*
Materials and Methods: *This study was conducted in the Department of Gastroenterology, Kurnool Medical College and Govt General Hospital from January 2018 to December 2018. In this study we evaluated the liver function and colonoscopic findings in 60 patients with liver cirrhosis. Liver cirrhosis was confirmed by histology or by compatible physical findings, laboratory data, and radiographic features. The etiologies of cirrhosis are shown in Table 1. The main cause of liver cirrhosis was post-viral hepatitis (68%) related to hepatitis B (6%) or C (62%) infection. In addition, 13 patients (27%) had hepatocellular carcinoma.*
Results: *In our cirrhotic patients, the primary indications for colonoscopy included a positive fecal occult blood test in 16 (34%), melena in 11 (23%), iron deficiency anemia in 5 (10%), diarrhea in 2 (4%), abdominal pain in 2 (4%), high level of serum CEA in 2 (4%), and screening in5 (10%) patients. Overall portal hypertensive colopathy was present in 44 patients (73.33%), whereas vascular ectasia was observed in 17 (36%), diffuse vascular ectasia in 20 (42%), redness in 10 (21%) and blue vein in 6 (12%) patients.*
Conclusion: *As the Child-Pugh class worsens and platelet count decreases, the prevalence of portal hypertensive colopathy increases in patients with liver cirrhosis. A colonoscopic examination in patients with liver cirrhosis is indicated, especially those with worsening Child-Pugh class and/or decreasing platelet count, to prevent complications such as lower gastrointestinal bleeding.*
Key Words: *Cirrhosis, portal hypertension, colonoscopy, portal hypertensive colopathy.*

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I. Introduction

Cirrhosis is defined by the World Health Organization (WHO) as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules. In cirrhosis, normal liver is replaced by fibrotic tissue and regenerative nodules leading to progressive loss of liver function. Cirrhosis is an important cause of mortality and morbidity. The clinical presentation of cirrhosis is variable depending on the etiology and whether the hepatocellular or portal hypertension predominates. The diagnosis of cirrhosis is based on the clinical features, laboratory investigations, radiologic features and histology. Cirrhosis can be asymptomatic or present with complications like ascites, spontaneous bacterial peritonitis (SBP), Hepatorenal Syndrome (HRS), variceal haemorrhage, hepatic encephalopathy and Hepatocellular Carcinoma (HCC). In the West, predominant aetiology is alcohol and NASH. In developing countries along with alcohol viral hepatitis B and C are still common causes of cirrhosis. Other rare causes of cirrhosis are Wilson's disease, Haemochromatosis, primary biliary cirrhosis, primary sclerosing cholangitis and Alpha-1-antitrypsin deficiency. The profile of cirrhosis may vary with different age and ethnic groups, geographical, social and aetiological factors. This study was conducted in a tertiary care hospital of Karnataka to determine the aetiology and clinical profiles of patients with cirrhosis of liver.

II. Materials And Methods

This study was conducted in the department of gastroenterology, Kurnool Medical College and Govt General Hospital from January 2018 to December 2018. In this study we evaluated the liver function and colonoscopic findings in 60 patients with liver cirrhosis. Liver cirrhosis was confirmed by histology or by compatible physical findings, laboratory data, and radiographic features. The etiologies of cirrhosis are shown in

Table 1. The main cause of liver cirrhosis was post-viral hepatitis (68%) related to hepatitis B (6%) or C (62%) infection. In addition, 13patients (27%) had hepatocellular carcinoma.

An upper gastrointestinal endoscopy was performed in all patients to evaluate the presence of esophageal varices, cardiac varices, and congestive gastropathy. Liver disease severity was assessed according to Child-Pugh's classification. All patients underwent a full colonoscopy with anti-cholinergic drugs, after preparation with polyethylene glycol electrolyte solution. Noted changes in colonic mucosa included the number, size, and location of vascular lesions. Portal hypertensive colopathy was defined endoscopically inpatients with vascular ectasia, redness, and blue vein. Vascular ectasia was further classified in to two types: type 1, solitary vascular ectasia; and type 2, diffuse vascular ectasia. Results from multiple, independent observers were compiled to determine the prevalence of portal hypertensive colopathy.

Statistical analyses: Data are expressed as mean ± SD. Statistical comparisons were made with the $\chi 2$ test. A P value less than 0.05 was considered significant.

III. Results

S.No	Diagnosis	Patients (n)
1	HCV	35
2	HBV	5
3	Non B non C	6
4	Alcohol	7
5	AIH	4
6	PBC	3

Table 1: Etiology of liver cirrhosis

Characteristics	PHC Positive N=44	PHC Negative N=16
Age (years)	63.6±5.8	60.23±9.5
Sex (M:F)	32:12	9:7
Child-Pugh (A:B+C)	19:25	10:6
PHG		
None	31	8
Mild	6	4
Severe	7	4
Ascites	4 (9.09)	2 (12.5)
Splenomegaly	25 (56.81)	6 (37.5)
Alcohol	6 (13.63)	6 (37.5)
HCC	9 (20.45)	9 (56.25)
EG Varices	19 (43.18)	6 (37.5.63)

Table 2: Patient Demographic Characteristics

Characteristics	PHC Positive N=44	PHC Negative N=16
Prothrombin Time	70.1±14.5	81.3±18.6
ALT (IU/L)	52.6±32.2	53.2±41.6
Platelet (*10^4/mm^3)	8.2±4.2	11.2±4.7
Serum Albumin (g/dl)	3.3±0.5	3.2±0.4
Total bilirubin (mg/dl)	1.54±0.76	1.65±0.65
Cholinesterase (pH)	0.43±0.25	0.54±0.21
ICGR15	29.65±14.34	25±8.5

Table 3: Lab data of 60 Patients

In our cirrhotic patients, the primary indications for colonoscopy included a positive fecal occult blood test in 16 (34%), melena in 11 (23%), iron deficiency anemia in 5 (10%), diarrhea in 2 (4%), abdominal pain in 2 (4%), high level of serum CEA in 2 (4%), and screening in5 (10%) patients. Overall portal hypertensive colopathy was present in 31 patients (66%), whereas vascular ectasia was observed in 17 (36%), diffuse vascular ectasia in 20 (42%), redness in 10 (21%) and blue vein in 6 (12%) patients. The clinical characteristics of the cirrhotic patients with or without portal hypertensive colopathy are shown in Table 2. As the Child-Pugh class worsened, the prevalence of portal hypertensive colopathy increased. Child-Pugh class B and C were significantly associated with portal hypertensive colopathy. Portal hypertensive gastropathy, esophageal varices, hepatocellular carcinoma, and presence of ascites were not related to occurrence of portal hypertensive colopathy. The laboratory data of these patients are shown in Table 3.Platelet count but not serum ALT level was significantly associated with portal hypertensive colopathy. The prothrombin time as well as serum albumin and total bilirubin levels were not related to the occurrence of portal hypertensive colopathy.

IV. Discussion

In the present study, portal hypertensive colopathy was present in 73.33% (44/60) of the cirrhotic patients. Several studies have described the colonic findings associated with cirrhosis and portal hypertension was observed in 50-84% patients with liver cirrhosis. Histologic examination of rectal mucosal lesions in patients with liver cirrhosis revealed dilatation of blood vessels in the mucosa, increased lymphocytes and plasma cells in the lamina propria, and edema of the mucosa. In our study, portal hypertensive colopathy was found in 23% of patients in recto sigmoid colon, 11% in the descending colon, 24%in the transverse colon, 23% in the ascending colon and16% in the cecum. We classified the portal hypertensive colopathy into four types. Solitary vascular ectasias were found predominantly in the transverse and ascending colon (55%). Diffuse vascular ectasias were found predominantly in the right side colon (45%). Redness was found in the overall colon and blue vein in the rectum. In contrast to prior studies. Our data demonstrated that the prevalence of portal hypertensive colopathy increased with worsening Child- Pugh class. The association with Child-Pugh class may be a result of increased hemodynamic dysfunction in those with more advanced liver disease. A decreased platelet count was also related to occurrence of portal hypertensive colopathy, which to our knowledge has not been reported previously. It is possible that the concurrent low platelet count and portal hypertensive colopathy may be due to a relatively high number of patients with post-viral infection in this Japanese cohort as post-viral liver cirrhosis decreases platelet count more than alcoholic liver cirrhosis does.

V. Conclusion

As the Child-Pugh class worsens and platelet count decreases, the prevalence of portal hypertensive colopathy increases in patients with liver cirrhosis. A colonoscopic examination in patients with liver cirrhosis is indicated, especially those with worsening Child-Pugh class and/or decreasing platelet count, to prevent complications such as lower gastrointestinal bleeding.

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