

## A Study on Portal Hypertensive Colopathy in Cirrhotic Patients

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### Abstract

**Aim:** The aim of this study was to evaluate the prevalence of Portal Hypertensive Colopathy in Patients with Cirrhosis Portal Hypertension and Clinical Correlation with the severity of the Liver Disease using Child Pugh's score.

**Methodology:** This Study was done in Department of Medical Gastroenterology in Government Stanley Medical college. Cases of cirrhosis with Portal Hypertension presenting to the medical gastroenterology department both Outpatients and Inpatients were included in the study. Symptomatic Patients were subjected to White Light Colonoscopy. Lesions characteristic of Portal Hypertensive Colopathy were studied. Those lesions were biopsied and specimen were subjected to Histopathological Examination.

**Results:** In a study of 100 cases of cirrhosis, portal hypertensive colopathy was present in 61 patients (61%), including solitary vascular ectasia in 30 patients (30%) and diffuse vascular ectasia in 41 patients (41%). Histological study revealed a marked dilatation in the microcirculation at the level of the ascending colon and rectum. The Child - Pugh class was correlated with a severity of portal hypertensive colopathy. Platelet count and history of Alcohol intake was significantly associated with portal hypertensive colopathy.

**Conclusion:** A history of alcohol intake was the main cause of Liver Cirrhosis. As the Child - Pugh class worsens and platelet count decreases, the prevalence of portal hypertensive colopathy increases in patients with Liver Cirrhosis. Histopathologic Evaluation correlated with the findings. A colonoscopic examination is indicated, in symptomatic patients especially those with worsening Child- Pugh class and or decreasing platelet count. Other incidental findings in colonoscopy and histopathology was also made out during the study.

**Keywords:** Cirrhosis, Portal hypertensive Colopathy

Date of Submission: 30 -04-2017

Date of acceptance: 09-09-2017

### I. Introduction

Liver Cirrhosis is an important cause of morbidity and mortality in India. The commonest causes of liver cirrhosis are alcohol intake, viral infections, Non-Alcoholic fatty Liver Disease (NAFLD), autoimmune hepatitis, inherited diseases and other rare etiologies.[1] Government Stanley Medical College being the only Government Centre approved for Liver Transplantation has enumerable Patients attending the Outpatient and Inpatient department. Liver Cirrhosis causes multiple complications such as portal hypertension, Hepatic encephalopathy, Hepatorenal syndrome, Hepato-cellular carcinoma (HCC) and Liver failure. Among which Upper and Lower Gastrointestinal Bleeding are important causes of Morbidity and Mortality. Patients with Liver Cirrhosis and Portal Hypertension, are screened for Esophageal and Gastric Varices, Portal hypertensive Gastropathy are well established and are the commonest cause of gastrointestinal bleeding in cirrhotic patients.[2-4] However, the prevalence and clinical importance of portal hypertensive colopathy (PHC) in patients with liver cirrhosis is not well established. In this study, we evaluate the prevalence, histological features and clinical significance of portal hypertensive colopathy in patients with Liver Cirrhosis.

### II. Materials And Methods

We evaluated the Patients for their symptoms, Laboratory investigations were done, Imaging was Done, The patients were assessed for the Liver disease based on Child Pugh Score. All symptomatic Patients

with Lower GI Bleeding, Anemia were subjected to Colonoscopy. A Colonoscopy was done after obtaining consent from the patient. The Colonoscopic findings were documented, these lesions were Biopsied for Histopathological Examination, The Study included 100 patients with Liver Cirrhosis over a period of 2 years in the Department of Medical Gastroenterology, Government Stanley Medical College Hospital, Chennai. Patients with Extra-hepatic portal venous hypertension, Non-cirrhotic portal fibrosis and severe coagulopathy were excluded from the study.

Liver Cirrhosis and Portal hypertension was confirmed by compatible physical findings, laboratory data and radiological features. An upper gastrointestinal endoscopy was done in all patients to evaluate esophageal and gastric varices and portal hypertensive Gastropathy. All patients underwent full colonoscopy and biopsy of colonic lesion was done in patients with vascular changes in colonic mucosa. The number, size, location of vascular lesion was recorded. Portal hypertensive colopathy was defined as presence of redness, blue vein, solitary or diffuse vascular ectasia in colonoscopy of patients with liver cirrhosis and portal hypertension. Histo-pathological sections studied from the colonic biopsy revealed mucosa with superficial erosions and sub mucosa shows dilated blood vessels, edema and chronic inflammatory cells composed of lymphocytes and plasma cells. [Fig.3,4]

### **III. Statistical Analysis**

Data are expressed as mean  $\pm$  SD. Statistical comparisons were made with Chi square test. A p-value of less than 0.05 was considered significant.

### **IV. Results**

The primary indication for colonoscopy in our patients with Liver Cirrhosis are Lower GI bleeding in 12 patients, positive Fecal occult blood test in 36, melena in 24, Iron deficiency anemia in 11, abdominal pain in 6, diarrhea in 5 and screening in 18 patients. The histopathological changes noted in the patients were colopathy associated with normal mucosa in 54, mucosal erosion in 41, hyperplastic polyp [Fig.5] in 4 and adenomatous polyp [Fig.6] in 1 patient. The clinical details of our 100 cirrhotic patients with or without portal hypertensive colopathy are shown in [table-1]. The prevalence of portal hypertensive colopathy increases with worsening of Child-Pugh class was noted. A statistically significant association was noted between alcohol intake and the presence of portal hypertensive colopathy. The laboratory data of our patients are shown in the [table-2]. Only platelet count was significantly associated with portal hypertensive colopathy. Total bilirubin, serum albumin, ALT levels were not related to the development of portal hypertensive colopathy.

### **V. Discussion**

Portal Hypertensive Colopathy (PHC) is a condition that describes the pathologic changes and mucosal abnormalities observed in the large intestine of patients with portal hypertension. The term Portal Hypertensive Colopathy (PHC) was first used by Naveau et al in 1991 in a study reporting the colonic vascular ectasia and rectal varices. Endoscopic abnormalities described in patients with portal hypertension ranges from vascular ectasias, anorectal or colonic varices, [5,6] hemorrhoids and non-specific inflammatory changes. The prevalence of PHC in patients with cirrhosis varies from 25% to 85% in various studies. [7-11] In the present study, portal hypertensive colopathy was observed in 61% (61/100 patients). Portal hypertensive colopathy was found in caecum and Ascending colon in 14 patients, transverse colon in 12, Descending and Sigmoid colon in 10 and rectum in 64 patients.

We classified the PHC into 4 types. Type-1 or redness in colonic wall was noted in 15% patients with PHC, Type-2 or blue vein in 14%, type-3 or Solitary vascular ectasia in 30% and type-4 or diffuse vascular ectasia in 41%. Redness was observed all over colon whereas blue vein was commonly noted in rectum. Solitary vascular ectasia were found predominantly in caecum and transverse colon where as diffuse vascular ectasia in left sided colon. The histopathological changes observed in the colonic biopsy were dilated and congested capillaries, capillaries with thickened wall were seen in significantly higher number of sections from patients with PHC than those without PHC. The other histological changes seen in the biopsies are edema, increased mononuclear cell infiltration and fibromuscular proliferation in the lamina propria. These histologic changes showed no correlation with the clinical or endoscopic findings of PHC. The dilatation of blood vessels in the mucosa, increased lymphocytes and plasma cells in the lamina propria and edema of mucosa are features suggestive of colopathy. The main pathologic change in PHC observed in several studies is colonic mucosal capillary dilation.

Similar to prior studies, we observed the prevalence of PHC was commonly associated with Child-Pugh class. This association is explained by the result of increased hyperkinetic circulation observed in patients with worsening Child-Pugh class and advanced liver disease. In contrast to previous other studies, we noted an interesting association between alcohol intake and PHC [12-15] which to our knowledge has not been reported previously. This may be possibly due to relatively high number of patients with alcoholic cirrhosis in our cohort

and selection bias. Like other studies, we also noted a significant association between decreasing platelet count and PHC. Total bilirubin, Serum Albumin, ALT levels, Ascites, Splenomegaly were not related to the appearance of PHC. Portal hypertensive colopathy is not a distinct entity, rather, a local manifestation of portal hypertension. Bleeding from PHC varies from 0 to 9%. In our study, we had 10 patients with Bleeding, 24 patients with melena. In spite of endotherapy at the same sitting for these patients, bleeding recurred. All four belong to Child class B, with history of alcohol intake and low platelet count. Hence, we advise colonoscopy in patients with worsening Child-Pugh score, alcohol etiology, and decreasing platelet count.

### VI. Conclusion

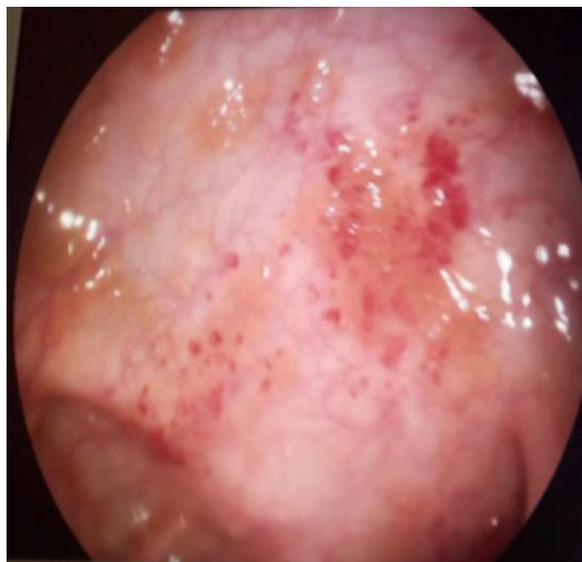
Lower gastrointestinal bleeding is the major complication of Portal hypertensive colopathy. In patients with liver cirrhosis, the prevalence of PHC increases with alcohol intake, worsening Child-Pugh class and decreasing platelet count. Hence a colonoscopic examination is indicated in these patients to prevent complications like lower gastrointestinal bleeding. Major histologic changes seen in colonic biopsies of patients with PHC such as dilated tortuous mucosal capillaries with wall thickening, edema of lamina propria and mild chronic inflammatory infiltrate does not show any association with clinical or endoscopic features. Hence a routine Histopathological examination is not necessary unless indicated.

**Table 1**

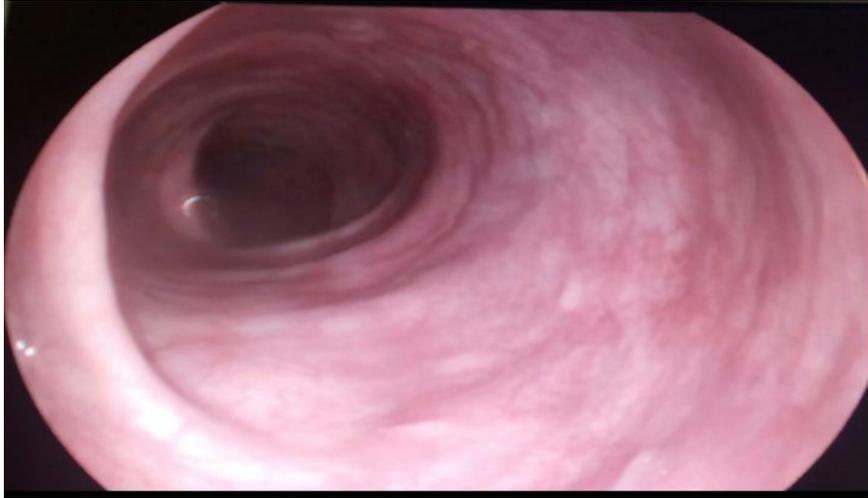
Characteristics of 100 patients				
S.NO		PHC Positive n=61%	PHC Negative n =39%	P Value
1	Age (year)	59 ±7	57±9	Non Significant
2	Sex (M:F)	47:19	21:18	Non Significant
3	Child Pugh Score (A:B+C)	21:40	24:5	<0.001 Significant
4	PHG None Mild Severe	46 10 4	20 10 2	Non Significant
5	Ascites	11	2	Non Significant
6	Splenomegaly	48	22	Non Significant
7	Alcohol	61	26	<0.001 Significant
8	HCC	24	10	Non Significant

**Table 2**

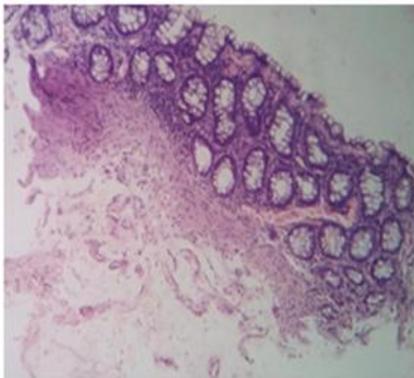
Laboratory Data				
S.NO		PHC Positive n=61%	PHC Negative n =39%	P Value
1	ALT (IU/L)	58±31	59±42	Non Significant
2	Platelet x (10 <sup>9</sup> /mm)	8±4	12±5	<0.05 Significant
3	S.Albumin	3.2±0.5	3.8±0.5	Non Significant
4	Total Bilirubin	1.5±0.9	1.0 ±0.7	Non Significant



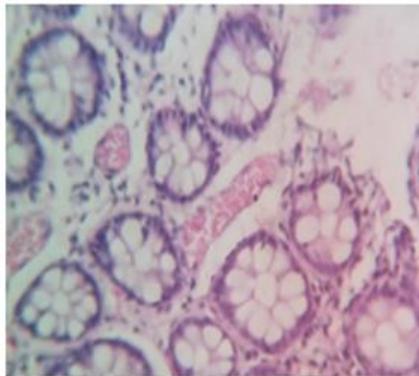
**Fig. 1:** Endoscopic Findings- Solitary vascular ectasia



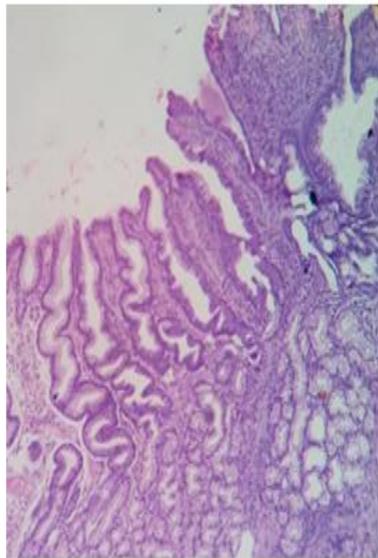
**Fig 2:** Endoscopic Findings- Diffuse vascular ectasia



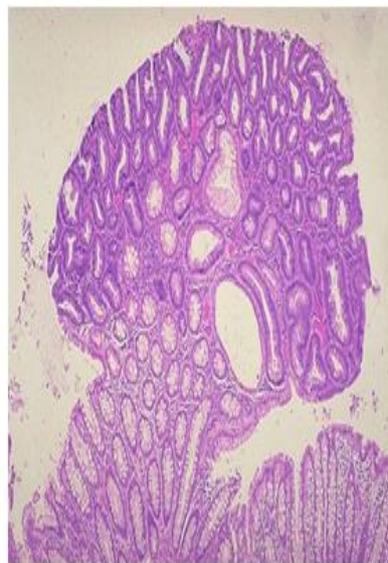
**Fig 3:** H&E x10 – Mucosal erosion and vascularity and lymphocytic infiltrate



**Fig 4:** H&E x40- Shows edema and dense vascularity



**Fig5:** H&E x10 – Hyperplastic polyp



**Fig 6:** H&E x10 – Adenomatous polyp

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\*Murugesan Malarvizhi. "A Study on Portal Hypertensive Colopathy in Cirrhotic Patients."  
*IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)* 16.9 (2017): 90-94