



A STUDY ON PORTAL HYPERTENSIVE COLOPATHY IN PATIENTS WITH CIRRHOSIS OF LIVER

Thiyagarajan G., Sabarinathan R., Ratnakar K., Pugazhendhi T., Premkumar K., Kani Shaik M., Murali A., Lavkush P Tiwari

Institute of Medical Gastroenterology, Rajiv Gandhi Government General Hospital and Madras Medical College, Chennai, India

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ABSTRACT

Aim: In patients with cirrhosis of liver with portal hypertension, one of the important cause for lower gastrointestinal bleed is portal hypertensive colopathy. In this study, we evaluated the prevalence of colopathy in patients with cirrhosis of liver and factors associated with it.

Methods: We evaluated the liver function test, ascitic fluid analysis, complete blood count, prothrombin time and child pugh class with colonoscopic findings in 25 patients over the period of 6 months. All patients underwent upper gastrointestinal endoscopy and full length colonoscopy to observe changes due to portal hypertension.

Results: We found portal hypertensive colopathy in 17 (68%) patients. Among these patients solitary vascular ectasia in 9 (36%), diffuse vascular ectasia in 10 (40%), redness in 7 (28%) and rectal varix in 1 (4%) were noted. There was no significant relationship between Child-Turcotte-Pugh class (CTP) and prevalence of portal hypertensive colopathy. Colonic mucosal changes were significantly associated with presence of esophageal varices and portal hypertensive gastropathy. Laboratory parameters such as platelet count, bilirubin level, International normalised ratio, serum albumin level and serum alanine aminotransferase (ALT) level were not related to presence of portal hypertensive colopathy. High serum ascities albumin gradient (SAAG) ascites in cirrhotic patients was associated with occurrence of portal hypertensive colopathy.

Conclusion: Portal hypertensive colopathy should be considered in the differential diagnosis of lower GI bleed in the clinical scenario of chronic liver disease. Patients with esophageal varix, portal hypertensive gastropathy, high SAAG ascites, and CTP class B and C should be submitted for full length colonoscopic examination.

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INTRODUCTION

Cirrhosis of liver is one of the major cause of portal hypertension which results in mucosal and hemodynamic changes in almost entire gastrointestinal (GI) tract.¹ Esophageal varices, portal hypertensive gastropathy (PHTG) and colonic mucosal changes occurs due to portal hypertension (PHT). These complications are the most common causes of gastrointestinal haemorrhage leading to severe and life-threatening outcomes ending up in high mortality.^{2,3} In India, Portal hypertensive changes in lower gastrointestinal tract due to cirrhosis is less explored, except for one study done on prevalence of colonic and other changes due to portal hypertensive colopathy (PHC).⁴ The colorectal mucosal lesions in patients with liver cirrhosis were reported as portal hypertensive colopathy which was one of the important causes of lower gastrointestinal tract bleeding.^{5,6}

The prevalence of PHC and factors influencing it are not well-established in literature particularly in South Indian population. So in this study, we analysed the prevalence of PHC in patients with liver cirrhosis, factors associated with it and influence of various laboratory markers.

MATERIAL AND METHODS

Patients aged more than 18 years with cirrhosis of liver who gave consent for the study were included. Liver cirrhosis was established by physical signs, laboratory workup and features of ultrasonography of abdomen. Routine laboratory work up was performed in all patients. All were submitted for upper gastrointestinal endoscopy and colonoscopy to observe for features of portal hypertension. Along with this, physical signs and symptoms were noted. Patients with inflammatory bowel disease, hemorrhoids, fissure in ano, patients on NSAID, hemodynamically unstable, anticoagulants, undergoing radiotherapy were excluded. We prospectively enrolled 40 patients aged more than 18 years with cirrhosis of liver from January 2016 to June 2016. 15 patients were

*Corresponding author: Thiyagarajan G

Institute of Medical Gastroenterology, India City/District: Chennai

excluded from the study (8 patients didn't give the consent, 3 had hemorrhoids, 2 had fissure in ano, 2 were on anticoagulants). Finally 25 patients were included in the study. All patients underwent upper GI endoscopy under conscious sedation to evaluate the presence of esophageal varices, portal hypertensive gastropathy and gastric varices. Severity of liver disease was assessed based on Child-Pugh's classification. Ascitic fluid analysis was done and SAAG was calculated wherever necessary. All patients underwent a total colonoscopy under conscious sedation after preparation with polyethylene glycol electrolyte solution. Portal hypertensive colopathy was diagnosed by the presence of vascular ectasia, redness and blue vein. Vascular ectasia is of two types: solitary vascular ectasia and diffuse (multiple) vascular ectasia. Type, size, number and location of this vascular lesions were noted.

Statistical Analyses

Numerical data was expressed as mean±SD. Statistical analysis were made using chi square test for comparing categorical variables, while student unpaired 't' test was done to compare difference in mean between both groups. P value less than 0.05 was considered significant.

RESULTS

In our study the median age was 51.12 ± 8.79 years. 23 (92%) patients were male. Ascites was present in 22 (88%) patients. Jaundice was seen in 17 (68%). Bleeding per rectum was noted in 4 (16%) subjects of which 3 (75%) had PHC. The clinical characteristics of the cirrhotic patients with or without PHC are shown in Table 1. In our study, comparison of the etiology of the liver cirrhosis in patients with and without PHC revealed no statistically significant difference between groups (P value-0.149).

in 10 (40%), redness (Fig. 3) in 7 (28%) and rectal varix (Fig. 4) in 1 (4%) patients (Table 3).



Figure 1 Colonoscopic image showing solitary vascular ectasia in transverse colon (black arrow)

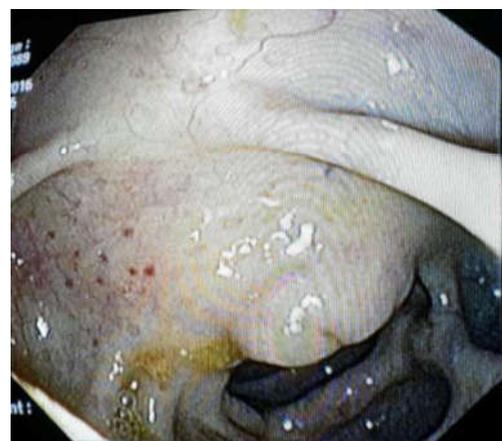


Figure 2 Colonoscopic image showing diffuse vascular ectasia in caecum

Table 1 Clinical characteristi

	Total cases (n=25)	PHC Positive (n=17)	PHC Negative (n=8)	P value	Significance
Age (years)	51.12 ± 8.79	50.70 ± 9.6	52.00 ± 7.2	0.739	NS
Sex(Male:Female)	23:02	16:07	02:01	0.569	NS
Pedal edema	10 (40%)	6 (35.2%)	4 (50%)	0.484	NS
Jaundice	17 (68%)	13 (76.4%)	4 (50%)	0.186	NS
Bleeding per rectum	4 (16%)	3 (17.6%)	1 (12.5%)	0.468	NS
Splenomegaly	9 (36%)	8 (47.0%)	1(12.5%)	0.093	NS
Hepatomegaly	6 (24%)	4 (23.5%)	2 (25.0%)	0.936	NS
Ascites	22 (88%)	15 (88.2%)	7 (87.5%)	0.133	NS
Anemia	16 (64%)	11 (64.7%)	5 (62.5%)	0.915	NS
Portal hypertension in upper GI endoscopy	19 (76%)	15 (88.2%)	4 (50.0%)	0.037	Significant
Portal hypertension in portal venous doppler study	18 (72%)	17 (100%)	1 (12.5%)	0	Significant

PHC= Portal hypertensive colopathy; NS=Not significant

The most common etiology in both the groups was alcohol followed by cryptogenic. Of note both HBV and HCV infection related cirrhosis were present in 2 (8%) patients respectively. PHC was present in all 4 cases. Features of PHT in upper GI endoscopy findings were seen in 19 patients. Among those most common finding was esophageal varix 12 (63.1%). Esophageal varices and PHTG were related to occurrence of PHC. While other clinical findings such as pedal edema, jaundice, hepatomegaly, splenomegaly, ascites, bleeding per rectum, anemia were not significantly related to the occurrence of PHC. Among 25 patients PHC was present in 17 patients (68%), in which solitary vascular ectasia (Fig. 1) was observed in 9 (36%), diffuse vascular ectasia (Fig. 2)

It signifies that two or more lesions were present in one person. Patients were classified into three groups based on Child-Pugh classification (Table 4). There were 3 (12%), 13 (52%) and 9 (36%) patients confined to Child Pugh class A, B and C respectively. Out of which PHC was positive in 1 (6%), 8 (47%), 8 (47%) of patients with Child Pugh class A, B and C respectively. Overall 22 (88%) patients were presented with combined class B and C, among them 16 (94%) had PHC. There existed a linear correlation between number of patients with PHC and severity of Child-Pugh class, as evidenced by increase in number of patients with PHC associated with the combined B and C class. But there was no statistically significant difference between the groups (P value of 0.17).

Table 2 Etiology of liver cirrhosis was compared with PHC

Etiology	PHC Present	PHC Absent	Total
Alcohol	12 (48%)	5 (20%)	17 (68%)
Cryptogenic	1 (4%)	3 (12%)	4 (16%)
HBV infection	2 (8%)	0	2 (8%)
HCV infection	2 (8%)	0	2 (8%)
Total	17 (68%)	8 (32%)	25 (100%)

P = 0.149 (Non significant)

HBV= Hepatitis B virus; HCV= Hepatitis C virus; PHC= Portal hypertensive colopathy; NS=Not significant



Figure 3 Colonoscopic image showing redness in sigmoid colon

Table 3 Colonoscopically detected number of various lesions in patients with portal hypertensive colopathy

Site	Findings	Number of lesion
Caecum	Diffuse vascular ectasia	2 (8%)
Transverse colon	Solitary vascular ectasia	1 (4%)
	Redness	1 (4%)
Ascending colon	Diffuse vascular ectasia	1 (4%)
Splenic flexure	Solitary vascular ectasia	1 (4%)
	Redness	1 (4%)
Descending colon	Solitary vascular ectasia	1 (4%)
	Diffuse vascular ectasia	4 (16%)
	Solitary vascular ectasia	5 (20%)
Sigmoid colon	Diffuse vascular ectasia	3 (12%)
	Redness	5 (20%)
	Solitary vascular ectasia	1 (4%)
Rectum	Varix	1 (4%)
	Solitary vascular ectasia	9 (36%)
	Diffuse vascular ectasia	10 (40%)
Total number of lesions	Redness	7 (28%)
	Rectal varix	1 (4%)

Table 4 Child Pugh class correlation with PHC

Child Pugh class	Total cases (n=25)	PHC +VE (n=17)	PHC -VE (n=8)
Class A	3 (12%)	1 (6%)	2 (25%)
Class B	13 (52%)	8 (47%)	5 (62.5%)
Class C	9 (36%)	8 (47%)	1 (12.5%)
Class (B + C)	22 (88%)	16 (94%)	6 (75%)
P value		0.17	
Significance		NS	

PHC= Portal hypertensive colopathy; NS=Not significant

The laboratory data of these patients are shown in Table 5. The parameter which significantly associated with the presence of PHC was high SAAG (P value 0.028). Other parameters like platelet count, bilirubin, ALT, AST, serum albumin were analysed with presence of PHC. Though there

was difference in mean among these parameters, it was not statistically significant.



Figure 4 Colonoscopic image showing rectal varix

DISCUSSION

In our study, portal hypertensive colopathy was seen in 68% (17/25) of the patients with cirrhosis of liver. Many previous studies showed a prevalence of 50-84% of colopathy in patients who had cirrhosis of liver and portal hypertension.⁸⁻¹⁰ Our study also had similar prevalence of colopathy. In our study, portal hypertensive colopathy was found in different locations, recto sigmoid colon was most common location with 60% of patients had lesion in that area, 20% in the descending colon, 8% in splenic flexure, 8% in the transverse colon, 4% in the ascending colon and 8% in the cecum.

Solitary vascular ectasia 5 (20%) was found predominantly in the rectosigmoid colon. Diffuse vascular ectasias were found predominantly in the descending colon 4 (16%).

Redness 5(20%) was mostly found in sigmoid colon. In our study these findings signifies that PHC was predominantly present in the left side of the colon particularly in rectosigmoid colon.

Except for one study¹¹ where there was significant association between child pugh class and presence of colopathy, in rest of the studies there was no significant relationship between worsening of child pugh class in correlation with colopathy. Though there was difference in number of patients in each group in class B and C, it was not statistically significant similar to many previous studies. This may be due to low sample size of our study and by improving the number of patients we may get a statistically significant association.

Clinical features of liver cirrhosis like jaundice, ascites, pedal edema, bleeding PR did not have any statistically significant impact on incidence of colopathy. But there was a significant association between portal hypertension and colonic mucosal changes with a P value of 0.000, which proves that cirrhotic patients with increase in portal pressure develop PHC. Contrary to one prior study¹² it was observed in our study that colonic mucosal changes associated with upper GI endoscopy findings such as esophageal varices and PHTG with a statistically significant P value of 0.037. The positive association was due to portal hypertension which is the common denominator for mucosal changes of whole GI tract in cirrhosis of liver.

Table 5 Laboratory parameters correlation with PHC

Parameters	Total(n=25)	PHC Positive (n=17)	PHC Negative (n=8)	P value	Significance
Haemoglobin (mg/dL)	9.11±2.08	9.16±1.94	9.0±2.49	0.869	NS
TWC (cells/μL)	6972±2482	7117±2962	6662±951	0.678	NS
Platelet count (cells/μL)	92440±55303	97823±64182	81000±29110	0.49	NS
Total bilirubin (mg/dL)	4.93±5.5	6.05±6.36	2.55±1.57	0.142	NS
Direct bilirubin (mg/dL)	2.67±3.22	3.27±3.78	1.4±0.48	0.18	NS
Indirect bilirubin (mg/dL)	2.26±2.62	2.78±2.95	1.15±1.25	0.151	NS
Total protein (mg/dL)	5.71±1.54	5.78±1.68	5.55±1.29	0.729	NS
Serum albumin (mg/dL)	2.9±0.57	2.78±0.56	3.15±0.55	0.142	NS
ALT (IU/L)	53.76±31.70	50.76±32.87	60.12±30.10	0.503	NS
AST (IU/L)	65.80±50.26	62.94±50.84	71.87±51.88	0.688	NS
INR	1.58±0.34	1.58±0.35	1.58±0.33	0.961	NS
Ascitic fluid-protein (mg/dL)	1.5±1.42	0.77±1.43	1.84±1.32	0.078	NS
Ascitic fluid-albumin (mg/dL)	1.26±0.87	0.81±1.13	1.47±0.70	0.086	NS
SAAG	1.11±0.81	1.35±0.69	0.60±0.86	0.028	Significant

PHC= Portal hypertensive colopathy; NS=Not significant; TWC=Total white cell count; ALT=Alanine aminotransferase; AST=Aspartate aminotransferase; PT=Prothrombin time; INR= International normalised ratio
Values are expressed as mean ± SD

Though colonic mucosal changes (94.1%) were more frequent in the higher class of CTP scoring system (combined class B&C), where as these components individually (biochemical-bilirubin, prothrombin time, albumin & clinical-ascites) were not correlated with occurrence of colopathy.

In our study serum ascites albumin gradient (SAAG) was one important factor related to colopathy. There was positive relationship between high SAAG to that of colonic changes due to portal hypertension with a statistically significant P value of 0.028. While there was no significant relationship between presence of colopathy with ascitic fluid protein and albumin.

Among 4 patients with bleeding per rectum 1 patient had active oozing from vascular ectasia in descending colon. Patient was submitted for argon plasma coagulation and haemostasis was achieved. Other patients with features of PHT were treated with non selective beta blocker (propranolol).

Some limitations of our study are small sample size, no head to head comparison between among individuals with different CTP score and not measuring hepatic venous pressure gradient (HVPG).

CONCLUSION

Portal colopathy should be considered in the differential diagnosis of lower GI bleed in the clinical scenario of chronic liver disease. Patients with esophageal varix, PHTG, high SAAG ascites, and CTP class B and C should be submitted for full length colonoscopic examination.

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