Clinicopathological Evaluation Of Portal Hypertensive Colopathy In Cirrhotic Patients

Malarvizhi Murugesan¹, Jamila Alagarsamy², S.Selvi³, ArcotRajeswaran Venkateswaran⁴ And M.S.Revathi⁵.

¹(Assistant Professor, Department Of Medical Gastroenterology, Stanley Medical College, TheTamilnadudr.Mgr Medical University,India) ²(Professor, Department Of Pathology, Stanley Medical College, The Tamilnadudr.Mgr Medical University,India) ³(Assistant Professor, Department Of Pathology, Stanley Medical College, The Tamilnadudr.Mgr Medical University,India)

^{4,5}(Professor, Department Of Medical Gastroenterology, Stanley Medical College,

The Tamilnadudr.Mgr Medical University,India)

Abstract

Aim: The aim of this study was to evaluate the colonoscopy findings in Patients with Cirrhosis Portal Hypertension and Histopathologic correlation of those colonoscopy lesions. The prevalence of portal hypertensive colopathy was evaluated, the lesions were biopsied and specimen sent for Histopathologic examination. The prevalence of Portal Hypertensive colopathy were correlated with severity of Liver Disease.

Methodology: Cases of Cirrhosis with Portal Hypertension presenting to the Medical Gastroenterology Department, 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 specimenwere 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.

Though the histopathologic evaluation had characteristiclesions, which correlated with Endoscopic findings.it did not correlate with the severity of Liver Disease.

Hence acolonoscopy examination is indicated, especially those with worsening Child- Pugh class and or decreasing platelet count but a routine Histopathological examination is not indicated. Other incidental findings in colonoscopy and histopathology was also made out during the study.

Keywords: Cirrhosis, Portal hypertensive Colopathy.

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]Liver cirrhosis causes multiple complications such as portal hypertension, hepatic encephalopathy, hepatorenal syndrome, Hepato-cellular carcinoma (HCC) and liver failure. In patients with liver cirrhosis and portal hypertension, 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 colonoscopy findings, histological examination of colonoscopic biopsy and laboratory data in 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 hypertensive colopathy into four types. Type 1 is the presence of redness in colonic wall, type 2 is the presence of blue vein, type 3 is the presence of solitary vascular ectasia[Fig.1] and type 4 is the presence of diffuse vascular ectasia.[Fig.2]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 +/- 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 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.

IV. 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 1991in 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% patientswith 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 24 patients with Malena, among them 4 four patients had active oozing form lesions. 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.

V. Conclusion

Lower gastrointestinal bleeding is the major complication of portal hypertensive colopathy. In patients with liver cirrhosis, the prevalence of PHC was higher in Cirrhosis due to Alcohol. The Prevalence of Portal Hypertensive colopathy increases with worsening of Child-Pugh class and in Patients with low 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. However these characteristic lesions though correlated with the endoscopic findings, did not show any association with clinical severity of the disease, This is a descriptive study to show the Histological characteristics of Endoscopic lesions in Portal Hypertensive colopathy, routine Histological Examination of these lesions are not indicated.

References

- Schuppan D, Afdhal NH. Liver cirrhosis. Lancet. 2008;371:838-851. [1].
- [2]. Viggiano TR, Gostout CJ. Portal hypertensive intestinalvasculopathy: a review of the clinical, endoscopic, and histopathologic features. Am J Gastroenterol 1992; 87: 944-954
- [3]. Arendt T, Barten M, Lakner V, Arendt R. Diffuse gastric antral vascular ectasia: cause of chronic gastrointestinal blood loss. Endoscopy 1987; 19: 218-220
- Gostout CJ, Viggiano TR, Balm RK. Acute gastrointestinal bleeding from portal hypertensive gastropathy: prevalence and clinical [4]. features. Am J Gastroenterol 1993; 88: 2030-2033
- Kozarek RA, Botoman VA, Bredfeldt JE, Roach JM, Patterson DJ, Ball TJ. Portal colopathy: prospective study of colonoscopy in [5]. patients with portal hypertension. Gastroenterology 1991; 101: 1192-1197
- Naveau S, Bedossa P, Poynard T, Mory B, Chaput JC. Portal hypertensive colopathy. A new entity. Dig Dis Sci 1991; 36: 1774-[6]. 1781
- Tam TN, NG WW, Lee SD. Colonic mucosal changes in patients with liver cirrhosis. GastrointestEndosc 1995; 42: 408-412 [7].
- Bini EJ, Lascarides CE, Micale PL, Weinshel EH. Mucosal abnormalities of the colon in patients with portal hypertension: an [8]. endoscopic study GastrointestEndosc 2000; 52: 511-516
- [9]. Weinshel E, Chen W, Falkenstein DB, Kessler R, Raicht RF.Hemorrhoids or rectal varices: defining the cause of massive rectal hemorrhage in patients with portal hypertension.Gastroenterology 1986; 90: 744-747
- [10]. Wang M, Desigan G, Dunn D. Endoscopic sclerotherapy for bleeding rectal varices: a case report. Am J Gastroenterol 1985; 80: 779-780
- Wilson SE, Stone RT, Christie JP, Passaro E. Massive lower gastrointestinal bleeding from intestinal varices. Arch Surg 1979; 114: [11]. 1158-1161
- [12]. Misra SP, Dwivedi M, Misra V. Prevalence and factors influencing hemorrhoids, anorectal varices, and colopathy in patients with portal hypertension. Endoscopy 1996; 28: 340-345
- [13]. Bresci G, Gambardella L, Parisi G, Federici G, Bertini M, Rindi G, Metrangolo S, Tumino E, Bertoni M, Cagno MC, Capria A. Colonic disease in cirrhotic patients with portal hypertension: an endoscopic and clinical evaluation. J ClinGastroenterol 1998; 26: 222-227.
- [14]. Yamakado S, Kanazawa H, Kobayashi M. Portal hypertensive colopathy: endoscopic findings and the relation to portal pressure. Intern Med 1995; 34: 153-157.
- [15]. Goenka MK, Kochhar R, Nagi B, Mehta SK. Rectosigmoid varices and other mucosal changes in patients with portal hypertension. Am J Gastroenterol 1991; 86: 1185-118.

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		РНС	PHC Negative	P Value			
		Positive	n =39%				
		n=61%					
1	ALT (IU/L)	58±31	59±42	Non Significant			
2	Platelet x (10 ⁴ /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

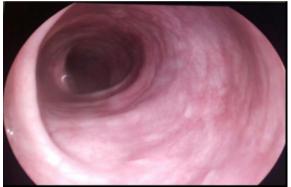
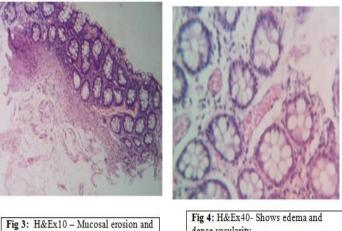


Fig 2: Endoscopic Findings- Diffuse vascular ectasia



vascularity and lymphocytic infiltrate

dense vasularity

