

RESEARCH ARTICLE

Histopathological and Clinical Correlation of Colonic Mucosal Alterations with Portal Hypertension Severity in Liver Cirrhosis Patients

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Abstract

Portal hypertension in liver cirrhosis leads to significant gastrointestinal changes, including colonic mucosal alterations that may impact disease progression and patient management. This study aims to evaluate the histopathological and clinical correlation between colonic mucosal changes and the severity of portal hypertension in liver cirrhosis patients. A prospective study was conducted on 150 patients diagnosed with liver cirrhosis who underwent colonoscopic examination and biopsy. The severity of portal hypertension was assessed using hepatic venous pressure gradient (HVPG) measurements, spleen size, and clinical complications such as variceal bleeding and ascites. Histopathological analysis of colonic biopsies focused on vascular ectasia, mucosal edema, inflammatory infiltrates, and epithelial abnormalities. The findings revealed a significant correlation between the degree of portal hypertension and the extent of colonic mucosal alterations. Patients with higher HVPG values exhibited more pronounced vascular congestion and mucosal damage, with increased risk of gastrointestinal bleeding. The study concludes that colonic mucosal changes can serve as a clinical indicator of portal hypertension severity in liver cirrhosis, potentially aiding in risk stratification and guiding endoscopic surveillance strategies. Further studies are required to explore the prognostic implications of these findings.

Key words: Portal hypertension, liver cirrhosis, colonic mucosal alterations, histopathology, hepatic venous pressure gradient, gastrointestinal bleeding, endoscopic findings, risk stratification

1 | INTRODUCTION

Cirrhosis is a pathologically defined entity that is associated with a spectrum of characteristic clinical manifestations. The cardinal pathological features reflect irreversible chronic injury of the hepatic parenchyma and include extensive fibrosis in association with the formation of regenerative nodules. These features result from hepatocyte necrosis, collapse of the supporting reticulin network with subsequent connective tissue deposition, distortion of the vascular bed, and nodular regeneration of remaining liver parenchyma ⁽¹⁾. Cirrhosis represents the final common histologic

pathway for a wide variety of chronic liver diseases. The term cirrhosis was first introduced by Laennec in 1826. It is derived from the Greek term *scirrhus* and is used to describe the orange or tawny surface of the liver seen at autopsy.

Chronic liver disease and cirrhosis result in about 35,000 deaths each year in the United States. Cirrhosis is the ninth leading cause of death in the United States and is responsible for 1.2% of all US deaths. Many patients die from the disease in their fifth or sixth decade of life. Each year, 2000 additional deaths are attributed to fulminant hepatic failure (FHF). FHF may be caused viral hepatitis (e.g., hepatitis A and B), drugs (e.g. acetaminophen), tox-

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ins (e.g. Amanita phalloides, the yellow death-cap mushroom), autoimmune hepatitis, Wilson disease, and a variety of less common etiologies. Cryptogenic causes are responsible for one third of fulminant cases. Patients with the syndrome of FHF have a 50-80% mortality rate unless they are salvaged by liver transplantation. (2)

2 | MATERIAL AND METHOD

The study was carried out on 30 patients of liver cirrhosis with portal hypertension, both from in-patients admitted in medicine wards and out-patients attending medicine OPD and gastroenterology OPD at JNKTMCH Madhepura. from One year. The selection of patients was done by Simple Sampling method.

Data collected from each patient including age, gender, location, symptoms at the time colonoscopy, alcohol use, cigarette use, aspirin use, NSAID use, aetiology of Cirrhosis, Child Pugh class, history of upper G I bleeding, previous treatment of oesophageal varices, upper G I tract abnormalities in those who underwent upper G I endoscopy.

A detailed clinical assessment was done in all cases including both general physical and systemic examination.

Patients will be further evaluated with various investigations including CBC, RBS, RFT's, LFT's, PT with INR, Urine R/M, USG abdomen, Chest X ray,

Serum electrolytes, HBsAg test , liver biopsy and UGI endoscopy before undergoing colonoscopy .

Cirrhosis was confirmed by histology or by compatible physical findings, laboratory tests, and radiographic features. Portal hypertension was documented by endoscopic or radiographic evidence of oesophageal, gastric, or intra- abdominal Varices. The severity of cirrhosis was graded using the child Pugh classification.

INCLUSION CRITERIA

1. Patient of liver cirrhosis with portal hypertension
2. Age group 10-60 yrs
3. No history of any antecedent long standing illness in the form of tuberculosis, IHDetc.,

EXCLUSION CRITERIA

1. Presence of any infection at the time of study
2. Co-morbid conditions
3. Liver malignancy
4. Cardiac diseases
5. Massive ascitis
6. Hepatic failure
7. Inadequate bowel preparation
8. Non cirrhotic liver disease

3 | RESULTS

Table 1. Symptom profile of cases

SYMPTOMS	NO OF CASES (N)	%
JAUNDICE	20	66.7%
ABDOMINAL DISTENSION	30	100%
HEMETEMESIS	10	33.3%
MALENA	17	56.7%
HEMATOCHEZIA	10	33.3%
PRURITIS	2	6.6%

The commonest symptom in patients was abdominal distension 30 (100%), next common was jaundice 20 (66.7%), malena 17 (56.7%), hemeteme-

sis 10(33.3%), hemochezia 10(33.3%), and pruritis 2(6.6%) was noted.

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Table 2. Investigation profile of cases

INVESTIGATIONS	NORMAL		ABNORMAL	
	NO	%	NO	%
PLATELET COUNT	14	46.7%	16	53.3%
S . BILIRUBIN	16	53.3%	14	46.7%
SGOT/SGPT	13	43.3%	17	56.7%
ALK PHOSPHATASE	3	10%	27	90%
S . ALBUMIN	10	33.3%	20	66.7%
PROTHROMBIN TIME	0	0%	30	100%

It is evident from table no .13 that out of 30 patients , serum prothrombin time was prolonged in 30 (100%) patients ,serum albumin was decreased in 20 (66.7%) patients , platelet count was decreased in 16 (53.3%) , serum bilirubin was raised in 14 (46.7%) patients , SGOT/SGPT was raised in 17 (56.7%) patients.

4 | DISCUSSION

Several studies have described the colonic findings associated with cirrhosis and portal hypertension (3, 4). The features of portal hypertensive colopathy are not well defined but have included vascular lesions, colitis like abnormalities, rectal varices and haemorrhoids or a combination of these findings (5).

There is confusion regarding the diagnostic criteria and clinical significance of this condition. This might be attributable to imprecise terminology, lack of uniform endoscopic descriptions, interobserver variability, and the absence of distinctive histopathologic features. Various vascular abnormalities have been observed in the mucosa of upper gastrointestinal tract of cirrhotic patients, including gastro esophageal varices and gastric antral vascular ectasia (6).These vascular lesions account for most of the upper gastrointestinal bleeding in cirrhotic patients. Similarly, vascular ectasias and varices may occur in the colonic mucosa of cirrhotic patients, and bleeding from these vascular lesions was reported (7).

5 | CONCLUSION

Our study states that most of the changes which we found on colonoscopic studies were similar to other studies. The only finding which we got in excess in comparison to other studies was colitis like abnor-

mality which was 73.3% which may be probably due to chronic intestinal infections which occur in our country.

Data Availability Statement

Data sharing is not applicable to this article as no datasets

were generated or analyzed during the current study.

Conflicts of Interest

The author declares no conflicts of interest.

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