A Study of Colonoscopic Findings in Cirrhotic Patients with Portal Hypertension

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ABSTRACT

Introduction: It has been noticed that persons with cirrhosis have great amount of colonic lesions. So study was planned to study the spectrum and frequency of colonic lesions in patients with portal hypertension due to cirrhosis, to assess whether the presence of portal hypertension related colonic lesions correlates with CTP and MELD scores and to study the association between upper GI findings of portal hypertension such as esophageal varices, gastric varices, portal hypertensive gastropathy and colonic lesions.

Material and Methods: This cross sectional study was performed for a period of one year. In this study, 100 patients with cirrhosis of liver and 50 age and sex matched controls were enrolled if they met the inclusion criteria.

Results: Portal hypertension related colonic lesions were noted in 59% of patients. Prevalence of portal hypertension related colonic lesions increased with worsening liver function as ascertained by higher CTP and MELD scores. Presence of esophageal varices and prior history of endoscopic variceal ligation correlated with occurrence of rectal varices. There was no association between ascites, splenomegaly, gastric varices, portal hypertensive gastropathy and the presence of colonic lesions. Serum bilirubin and prothrombin time were significantly higher in patients with portal hypertension related colonic lesions. Low serum albumin and decreasing platelet count correlated with presence and frequency of portal hypertension related colonic lesions.

Conclusion: Cirrhotic patients with portal hypertension have significantly higher frequency of colonic lesions as compared to controls. The frequency of portal hypertension related colonic lesions increases with worsening CTP and MELD scores.

Keywords: Cirrhosis, Portal hypertension, Child-turcotte-Pugh score

INTRODUCTION

Cirrhosis, a final pathway for a wide variety of chronic liver diseases, is 14th most common cause of death worldwide.[1] The clinical course of patients with cirrhosis is often complicated by a number of complications that are independent of the etiology of the underlying liver disease. These include portal hypertension and its consequences including variceal bleed, ascites, hepatic encephalopathy, hepatorenal syndrome, hepatopulmonary syndrome, portopulmonary hypertension; cirrhotic cardiomyopathy, hepatic osteodystrophy, endocrine dysfunction and hepatocellular carcinoma. Portal hypertension is defined as the elevation of hepatic venous pressure gradient (HVPG) above 5mmHg. Portal hypertension and its complications are the leading causes of death and liver transplantation, in patients with cirrhosis.[2] Portal hypertension causes hemodynamic and mucosal changes in the entire gastrointestinal (GI) tract. The various portal hypertension related lesions in the upper GI tract include gastroesophageal varices, portal hypertensive gastropathy, gastric antral vascular ectasia. These have been studied extensively. Similarly portal hypertension also produces vascular changes throughout the colon. The portal hypertension related changes in the colon include portal hypertensive colopathy (PHC), colorectal varices and haemorrhoids.[3,4] Portal hypertensive colopathy (PHC) is characterized by erythema of the colonic mucosa, vascular lesions including cherry-red spots, arterial spider like lesions or angiodysplasia-like lesions.[5] The prevalence for PHC ranges from 25 to 70%. Bleeding from PHC is estimated to be upto 9%. There is no universally accepted classification system for grading the severity of mucosal abnormalities in patients with PHC. Rectal varices are described as dilated veins that originate more than 4 cm above the anal verge, not contiguous with the anal columns and do not prolapse into the proctoscope. The prevalence of rectal varices ranges from 7% to 44% in various studies and the bleeding from the varices is seen in upto 8%. Similarly prevalence of haemorrhoids in cirrhotic patients range from 22 to 89%. This makes comparisons between studies challenging. Although colorectal lesions are a source of acute and chronic bleeding, they have received little attention in the literature. Also the variability of the results of previous studies does not allow us to define with any certainty the prevalence of these lesions. These discrepancies between various studies may be because of imprecise terminology, lack of uniform endoscopic descriptions, inter-observer variability. Portal hypertension related colonic lesions has been reported to be associated with a lower platelet count,[6] an increasing severity of cirrhosis (Child grade), large esophageal varices, gastric varices, higher portal pressure. Colonoscopy readily identifies the portal hypertension related colonic lesions. It is generally considered safe in cirrhotic patients and doesn't worsen the clinical state. Some previous studies suggest that colonoscopic examination is needed in these patients, especially those with worsening Child-Pugh class and decreasing platelet count, to prevent complications, such as lower gastrointestinal bleeding.

MATERIAL AND METHODS

This cross sectional study was performed from December 2013 to November 2015 at Department of Gastroenterology, Osmania General Hospital. This study was approved by the ethical committee of the hospital. All patients with cirrhosis evaluated in the Department of Gastroenterology were enrolled in this study if they met the inclusion criteria. Informed consent was

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taken from all the subjects.

Inclusion criteria: Patients with cirrhosis of liver, age >18 years.

Exclusion Criteria: Age <18 years, patients with hepatic encephalopathy, very sick patients (shock, inotropic support, ARDS, ventilatory support).

The diagnosis of cirrhosis with portal hypertension was based on characteristic findings including physical stigmata of cirrhosis, liver function tests, prothrombin time, ultrasonographic findings (like nodular liver surface, coarse echotexture of liver parenchyma, splenomegaly etc), upper gastrointestinal endoscopy findings (varices and portal hypertensive gastropathy).

100 patients with cirrhosis of liver were included in the study. Detailed medical history was taken from all patients and they underwent complete physical examination, standard laboratory tests including complete blood picture, biochemical tests of liver and kidney function, serum electrolytes, ultrasound of the abdomen and ascitic fluid analysis, upper GI endoscopy. Special investigations such as Serum ceruloplasmin, 24 Hour urinary copper, Serum copper, Serum Ig G, ANA, SMA, anti-LKM1, Serum α_1-anti trypsin levels, Transferrin saturation, Serum Ferritin, Serum Iron Anti- mitochondrial antibody (AMA) when required. For assessment of severity, cirrhotic patients were divided into A, B and C classes according to Child Pugh criteria. They were also divided into 4 MELD groups (as per UNOS) as follows: Group 1 – MELD ≤10, Group 2 – MELD- 11-18, Group 3- 19-24, Group 4- MELD >24.

All patients with ascites were classified according to international ascites club as follows Group 1 – Mild, Group 2 -Moderate, Group 3- Severe. In upper G I endoscopy oesophageal varices were classified into three grades namely Grade I-Varices may be small and straight. Grade II-Tortuous and occupying less than one third of the esophageal lumen Grade III-Large and occupying more than one third of the esophageal lumen Colonoscopy was done using Olympus CV-150 series colonoscope (Olympus corporation, Tokyo, Japan). Conscious sedation with midazolam or propofol was provided when requested. The following lesions were identified in cirrhotic patients with portal hypertension: Portal hypertensive colopathy: characterized by erythema of the colonic mucosa with or without mosaic pattern, vascular lesions including cherry-red spots (flat or slightly elevated red lesion less than 10 mm in diameter), arterial spider like lesions (central arteriole with radiating vessels which blanches on pressure from biopsy forceps), or angiodysplasia-like lesions. Rectal varices were defined as dilated vessels noted 4cms above the anal verge and which do not proplapse into proctoscope. Haemorrhoids: internal or external.

A group of 50 age and sex matched persons undergoing colonoscopy for irritable bowel syndrome were taken as controls. They also underwent complete physical examination, standard laboratory tests including complete blood picture, biochemical tests of liver and kidney function, serum electrolytes, ultrasound of the abdomen, upper GI endoscopy.

RESULTS

In this study, 100 patients with cirrhosis of liver and 50 age and sex matched controls were included.

In the present study it was observed that the mean age of cases was 44.2 years compared to 45.6 years in controls. There was no statistically significant difference in the mean age between cases and controls. Among the cases 81% were male and 19% were female. Among the controls 78% were male and 22% were female. There was no statistically significant difference in distribution of patients based on gender between cases and controls.

Association with all the clinical findings with severity of Child Pugh class are significant ie P-value <0.05 only, association between rectal varices and severity of Child Pugh class is insignificant(P->0.05)

Age group in years	Cases		Controls	
	Number	%	number	%
20-30 yrs	9	9	5	10
31-40 yrs	29	29	14	28
41-50 yrs	36	36	12	24
51-60 yrs	19	19	14	28
61-70 yrs	7	7	5	10
Mean ± SD	44.2 ± 10.4		45.6 ± 11.6	
t-value	0.771			
p value	value p value = 0.442			
Sex				
Females	19	19	11	22
Males	81	81	39	78
Chi square 0.188, p value 0.665				

Table-1: Distribution of patients based on Age

Findings	Present	Absent	P-value
Association between PHC and severity of Child Pugh class			
Class –A	2	16	0.03
Class-B	11	26	
Class-C	20	25	
Association between rectal varices and severity of Child Pugh class			
Class –A	0	18	0.204
Class-B	4	33	
Class-C	7	38	
Association between hemorrhoids and severity of Child Pugh class			
Class –A	3	15	0.002
Class-B	16	21	
Class-C	29	16	
Association between PHC and MELD score			
<10	0	18	<0.001
11 – 18	19	43	
19 – 24	9	5	
>24	5	1	
Association between rectal varices and severity of MELD score			
<10	0	18	<0.001
11 – 18	2	60	
19 – 24	7	7	
>24	2	4	
Association between hemorrhoids and severity of MELD score			
<10	2	16	<0.001
11 – 18	31	31	
19 – 24	11	3	
>24	4	2	

Table-2: Association of findings in present study

Findings	Present	Absent	P-value
Association between PHC and Endoscopic variceal Ligation			
Present	15	25	0.43
Absent	18	42	
Association between rectal varices and endoscopic variceal ligation			
Present	8	32	0.014
Absent	3	57	
Association between hemorrhoids and endoscopic variceal ligation			
Present	19	21	0.93
Absent	29	31	
Association between PHC and gastric varices			
Present	9	14	0.47
Absent	24	53	
Association between rectal varices and gastric varices			
Present	4	19	0.264
Absent	7	70	
Association between hemorrhoids and gastric varices			
Present	13	10	0.351
Absent	35	42	
Association between PHC and portal hypertensive gastropathy			
Present	22	45	0.96
Absent	11	22	
Association between rectal varices and portal hypertensive gastropathy			
Present	8	59	0.66
Absent	3	30	
Association between hemorrhoidsvand portal hypertensive gastropathy			
Present	28	39	0.076
Absent	20	13	

Table-3: Association between clinical finding and gastric findings

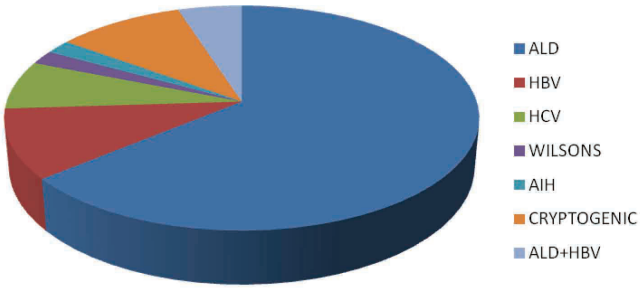

Figure-1: Distribution of cases based on etiology

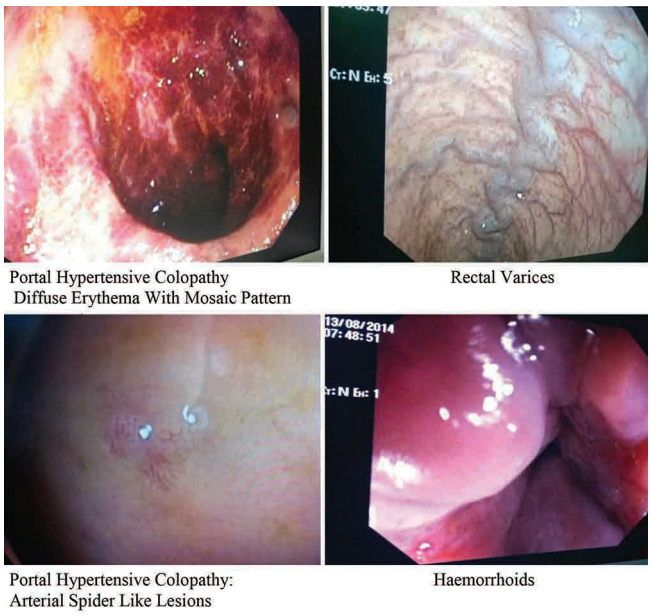

Figure-2: Colonoscopic pictures in the study.

DISCUSSION

Cirrhosis, at present is considered as a dynamic and potentially reversible disease. It consists of two stages, compensated and decompensated cirrhosis, each with a distinct prognosis and different predictors of survival. The development of portal hypertension is a hallmark in the history of cirrhosis, and its progression parallels that of the disease.

It is generally considered safe in cirrhotic patients and doesn't worsen the clinical state.

In the present study, the mean age of patients was 44.2 years compared to 45.6 years in controls. There was no significant difference in the mean age between cases and controls. Among the cirrhotics included in the study 36% belonged to the age group of 41 – 50 years, 29% belonged to the age group of 31 – 40 years. Among the controls 28% belonged to the age group of 51-60 years and 31-40 years each, 24% belonged to the age group 41-50 years. Of the 100 patients of cirrhosis included in the study 81% were male and 19% were female. Among the controls 78% were male and 22% were female. There was no statistically significant difference in distribution of patients based on gender between cases and controls. The most common cause of cirrhosis in the present study was alcoholic liver disease, seen in 64 % of cases followed by hepatitis B infection in 10% of the cases, Hepatitis C infection in 7%, alcohol with co-existent hepatitis B infection in 5%, Wilsons disease in 2%, autoimmune hepatitis in 2% and the cause could not be determined in 10% of the cases. In the study by Diaz-Sanchez et.al, the main cause of cirrhosis was alcohol consumption (45.5%) followed by hepatitis C virus infection (31.8%). In the study by Ito et.al, the main cause of liver cirrhosis was post-viral hepatitis (68%) related to hepatitis B (6%) or C (62%) infection. For the cirrhotic patients included in the study, child-turcotte-pugh(CTP) was calculated and the patients were divided into Child-pugh class A,B,C. In the present study 18% of patients belonged to class A, 37% belonged to class B and 45% to class C. The patients were also categorised based on MELD score into 4 groups. In the present study it was observed that 62 % of cases had a MELD score between 11 – 18 followed by, 18 % cases had a MELD score of < 10, 14% had score between 19 – 24 and 6 % above 24.

Among the cases included in the present study, colonoscopy was abnormal i.e. revealed one or more of the portal hypertension related lesions (portal hypertensive colopathy, rectal varices and haemorrhoids) in a total of 59% patients. Of these 25% patients had portal hypertensive colopathy with haemorrhoids, 19% patients had only haemorrhoids, 5% had rectal varices with haemorrhoids, 5% had portal hypertensive colopathy only, 3% had rectal varices with haemorrhoids, 3% had only rectal varices. Among controls 18(36%) had abnormal findings on colonoscopy. On comparing the frequency of abnormal colonoscopy findings between cases and controls, the cirrhosis had statistically significant (p= 0.007) higher frequency of abnormal findings on colonoscopy. Also haemorrhoids were found in 48% of cases as

compared to 22% of controls, which was statistically significant (p=0.002). In the present study portal hypertensive colopathy(PHC) was seen in 33% of the cases. This is similar to the studies by Ghoshal UC et al,[7] Bini EJ et al,[8] who reported a frequency of 36.6% an 38% respectively. However there is wide variation in the reported frequency of PHC in different studies. In studies by Diaz-sanchez A et al,[9] Weismuller TJ et al,[10] PHC was seen 23.9% and 24.3% of cases respectively which is lower than the present study. Zaman A et al,[11] reported a prevalence as low as 3%, whereas Salama ZA et al,[12] Jeong IB et al,[13] Bresci et al,[14] Ito K et al[6] reported prevalences of 45.7%, 45.8%, 54%, 66% respectively which is higher than the present study. This wide variation in the prevalence of PHC may be due to lack of lack of a clear classification system, lack of consensus on the endoscopic appearance of PHC and interobserver variability. Rectal varices were forund in 11% of cases. This in accordance with reported prevalence of 14.3% by Salama ZA et al[12] and 12% by Ito K et al.[6]

In the studies by Diaz-Sanchez et al[9] and Zaman A et al[11] rectal varices were seen in 7.6% and 7%. However, Ghoshal UC et al,[7] Misra SP[15] et.al reported higher rates, 31.7% and 40% respectively. Also haemorrhoids were seen in 48% of the cases in present study, similar to the study by Diaz-Sanchez et al,[9] who reported haemorrhoids in 52.3%. There is statistically significant (p value=0.002) difference in the frequency of haemorrhoids between cases and controls with higher frequency among cases. However Jeong IB et al,[13] Ghoshal UC et al,[7] Zaman A et al[11] reported lower rates, 25%, 21.9%, 21% respectively. This discrepancy may be attributed to lack of clear grading system, different aetiologies of liver disease and variation in severity of liver disease of the cases included. Bleeding per rectum was noted in 12% of cases in the present study, of which 3% presented with severe bleeding requiring blood transfusion. All the patients who presented with severe bleeding per rectum had rectal varices. Also majority of the presenting with bleeding per rectum had haemorrhoids. In the previous studies, Bresci et al,[14] reported a lower GI bleeding rate of 6%. Salama ZA et al,[12] reported a bleeding rate of 20%, mostly from haemorrhoids. Ghoshal UC et al[14] reported that detection of colorectal varices but not PHC was associated with haematochezia. In the study by Ganguly S et al,[16] overt bleeding per rectum was seen in 4%of patients with colopathy and 8% of the patients with rectal varices. In the study by Ito K et.al, the primary indications for colonoscopy were faecal occult blood test positive in 34% and anaemia in 10%. This lower incidence of bleeding from rectal varices may be related to rectal varices having thicker walls and are less superficial than those in the lower esophagus.[4]

Cirrhosis is characterized by the presence of extensive fibrosis and numerous regenerative nodules replacing the normal liver parenchyma. The mechanism of portal hypertension in cirrhosis is increased intrahepatic resistance due to fibrosis with concomitant increase in portal flow due to haemodynamic changes such as splanchnic vasodilatation. With advanced liver disease there is worsening of fibrosis and haemodynamic dysfunction leading to higher portal pressure.

In the present study, of the 33 patients with portal hypertensive colopathy, 61% were from Child-Pugh class C, 33% from Child-Pugh class B and 6% from Child –Pugh class A. On analysis there is a statistically significant (p=0.03) relation between the severity of liver disease and presence of PHC. Also 63.6% of the cases with rectal varices were from Child-Pugh class C, 36.4% from Child-Pugh class B and none of the cases from Child-Pugh class A had rectal varices. Of the cases with haemorrhoids, 60.4% were from Child-Pugh class C, 33.6% from Child-pugh class B and 6.2% were from Child-Pugh class A. The relation between presence of haemorrhoids and severity of liver disease was statistically significant (p value=0.002). Similarly Ito et al,[6] demonstrated that the prevalence of portal hypertensive colopathy increased with worsening Child Pugh class. Gad YZ et.al and Salama ZA et al,[18] also demonstrated increase in the prevalence of portal hypertension related colonic lesions with increasing severity of liver disease. The increase in the prevalence of portal hypertension related colonic lesions with advanced Child-Pugh class may be a result of increase in portal pressure due to increasing fibrosis coupled with worsening haemodynamic dysfunction associated with advanced liver disease. Higher portal pressure as measured by HVPG has been reported to correlate with the presence of portal hypertensive colopathy.[9] MELD score is the most accepted prognostic scoring system for allocation of organs for liver transplantation. It accurately predicts survival in patients in with decompensated cirrhosis. When the cases with PHC were categorised based on MELD score, 15.2 % of cases with portal hypertensive colopathy had MELD score > 24 compared to 1.5 % in patients with no portal hypertensive colopathy. There was statistically significant relation between presence of portal hypertensive colopathy and increasing MELD score. $p < 0.001$. Also, 18.2 % of cases with rectal varices had MELD score > 24 compared to 4.5 % in patients with no rectal varices. The association between rectal varices and increasing MELD score was statistically significant ($p < 0.001$). Of the patients with haemorrhoids, 8.3 % had MELD score > 24 compared to 3.8 % in patients with no haemorrhoids. There was high statistical significance between presence of hemorrhoids and MELD score. p=0.001. This relation between portal hypertension related colonic lesions and MELD score may be explained by worsening of fibrosis leading to increase in portal pressure and worsening haemodynamic dysfunction associated with high MELD score suggestive of advanced liver disease. Similarly Jeong IB et al[13] demonstrated a statistically significant correlation between the prevalence of PHC and increasing MELD score.

Ascites is the most common complication of cirrhosis, and 60% of patients with compensated cirrhosis develop ascites within 10 years during the course of their disease. Ascites develops when the HVPG increases more than 12mm of Hg and when it is more than 16mm of Hg, ascites becomes refractory. When the presence of portal hypertension associated colonic lesions was compared with severity of ascites statistically significant relation was not demonstrated in the present study (p>0.05). Similarly Ito et al,[16] Diaz-sanchez et al[9] and Ghoshal et al[7] also reported no relation between the presence and severity of ascites with occurrence of portal hypertension related colonic lesions. Splenomegaly is present in 50% of cirrhotics with portal hypertension. In the present study splenomegaly was noted in 54.5% of the cases with PHC, 72.7% of those with rectal varices and 56.2% of those with haemorrhoids. On comparing with the cases who did not have portal hypertension related colonic lesions, there was no statistically significant (p>0.05) relation

between the presence of splenomegaly and portal hypertension related colonic lesions. Similarly Ito et al[16] did not find any correlation between the presence of splenomegaly and portal hypertension related colonic lesions. Esophageal varices are the most common site for the formation of portosystemic collaterals in cirrhotic patients with portal hypertension.

In the present study when the association between the presence of PHC and grade of esophageal varices was evaluated, there was no statistically (p=0.07) significant relation. Similarly when the association between presence of haemorrhoids and esophageal varices was analysed, it was found that there was no statistically significant relation (p=0.35). This finding is in accordance with the previous studies. Diaz-Sanchez et al,[9] Ito K et al,[6] Ghoshal UC et al,[7] found no association between the presence of PHC and esophageal varices. In the present study it was observed that 54.5 % patients with rectal varices had grade III esophageal varices compared to 21.3 % patients without rectal varices. There was a statistical significance between presence of rectal varices and esophageal varices (p= 0.03). Hosking et al[18] noted rectal varices in 19% of patients with cirrhosis without esophageal varices, 39% in patients with esophageal varices without history of bleeding, and 59% in patients with esophageal varices and history of bleeding. Similarly Gad YZ et al[17] demonstrated a significant relation between the presence of esophageal varices and rectal varices. Varices do not develop until a minimal threshold HVPG of 10mmHg is reached. Rectal varices are collaterals between superior rectal veins which drain into the inferior mesenteric system and the middle inferior rectal veins which drain into the iliac veins and are one of the most common site for ectopic varices.

The presence of portal hypertension related colonic lesions has been reported to correlate with portal pressure. In patients with a prior history of endoscopic variceal ligation for esophageal varices, PHC was noted in 45.5%. There was no statistically significant relation between prior history of EVL and occurrence of PHC. Also, in the cases with haemorrhoids prior history of endoscopic variceal ligation was noted in 39.6%. Similarly Jeong Ib et al[13] noted no significant relation between prior history of EVL and presence of PHC or haemorrhoids. In the present study, it was observed that among the 11 patients with rectal varices 72.7 % had prior history of endoscopic variceal ligation compared to 36% patients without rectal varices. There was a statistical significance between presence of rectal varices and prior history of esophageal variceal ligation (p=0.019). This finding is similar to the study by GadYZ et al.[17] A large study conducted in Japan by Watanabe et.al reported that 95% of patients with rectal varices had a history of esophageal varices and 87% of these patients had previously undergone endoscopic variceal obliteration for esophageal varices. The indication for endoscopic band ligation of esophageal varices are large varices(>5mm) and variceal haemorrhage. Variceal haemorrhage occurs only when hepatic venous pressure gradient is more than 12mm of Hg. The mechanism of formation of rectal varices after treatment of esophageal varices is thought to be the result of obliteration of supplying vessels such as the left gastric, posterior gastric and short gastric veins leading to development of collateral vessels of the inferior mesenteric venous system and thus the formation of rectal varices.

In the present study there is no statistically significant relation between the presence of gastric varices and any of the portal hypertension related colonic lesions (PHC, rectal varices and haemorrhoids), p>0.05. Similarly, the studies by Ghoshal UC et al,[7] Diaz-Sanchez et al,[9] Salama ZA et al[12] noted no association between gastric varices and portal hypertension related colonic lesions. Portal hypertensive gastropathy was noted in 66.7% of the cases with PHC, 72.7% of those with rectal varices and 58.3% of those with haemorrhoids. On analysis there was no statistically significant association between portal hypertensive gastropathy and portal hypertension related colonic lesions (p value>0.05). this finding is in accordance with studies by Ito et al[6] and Diaz-Sanchez et al.[9]

The mean serum bilirubin of cases with portal hypertension related lesions was 2.9 ± 1.5mg/dl and for the cases with no portal hypertension related colonic lesions it was 1.59 ± 0.62mg/dl. On comparing the means between the two groups, serum bilirubin was significantly higher in the cases who had portal hypertension related colonic lesions (p value<0.001). Serum bilirubin is a component of both CTP score and MELD score. Development of jaundice is considered as a sign of decompensation in cirrhosis. Decompensation is negligible in patients with compensated cirrhosis with an HVPG < 10 mm Hg, whereas it reaches 40% at 4 years in patients with an HVPG 10 mmHg.

The mean prothrombin time in cases with portal hypertension related colonic lesions was 18.89 ± 3.12sec and for the cases with no portal hypertension related colonic lesions it was 16.5 ± 2.03sec. On analysis, prothrombin cases with portal hypertension related colonic lesions had higher prothrombin time (p value <0.001). Prothrombin time is a measure of hepatic synthetic function. Prothrombin time with INR is a component of both CTP and MELD scores. High values of bilirubin and prothrombin time are associated with high CTP and MELD scores suggestive of advanced liver disease. As discussed above advanced liver disease is associated with increased frequency of portal hypertension related colonic lesions due to higher portal pressures and worsening haemodynamic dysfunction. Serum albumin is a test of synthetic function of liver. It is a component of child-turcotte-pugh score. Lower levels of serum albumin are associated with advanced liver disease. The mean value of serum albumin among patients with portal hypertension related colonic lesions was 2.57 ± 0.53g/dl compared to a mean albumin value of 2.85 ± 0.56g/dl among patients without portal hypertension related colonic lesions. On statistical analysis serum albumin was significantly lower in the cases who had portal hypertension related colonic lesions (p value=0.012). Thrombocytopenia (platelet count <150,000/microL) is a common complication in patients with cirrhosis. It has been observed in up to 76% of patients.[19] Declining platelet count may be one of the earliest signs of portal hypertension in cirrhosis. The degree of thrombocytopenia has been shown to be a useful prognostic marker in cirrhotic patients. Possible causes of thrombocytopenia in cirrhosis include splenic sequestration of platelets, suppression of platelet production in the bone marrow, and decreased activity of the hematopoietic growth factor thrombopoietin (TPO).[20-21]

The mean platelet count among cases with portal hypertension related colonic lesions was 1.12 ± 0.19×10^5/µl and it was 1.5 ± 0.42×10^5/µl in cases without portal hypertension related

colonic lesions. On comparing the means between these two groups, platelet count was significantly lower in the cases with portal hypertension related colonic lesions. Ito et al,[6] reported that count was related to occurrence of portal hypertensive colopathy. Similarly Gad YZ et al[17] and Jeong IB et al[13] reported a statistically significant relation between decreased platelet count and portal hypertension related colonic lesions. The relation between platelet count and portal hypertension related colonic lesions may be explained by the association of low platelet count with advanced fibrosis. Advanced fibrosis leads to high portal pressures due to increased intrahepatic resistance. Presence PHC has been demonstrated to correlate with portal pressure.

CONCLUSION

Cirrhotic patients with portal hypertension have significantly higher frequency of colonic lesions as compared to controls. The frequency of portal hypertension related colonic lesions increases with increase in the severity of liver disease as ascertained by Child-turcotte-Pugh score. Portal hypertension related colonic lesions are more frequent in cirrhotic patients with higher MELD score. There is no association between the severity of ascites and the presence of portal hypertension related colonic lesions. Presence of splenomegaly does not correlate with the presence colonic lesions due to portal hypertension. There is a statistically significant correlation between the presence of rectal varices and the size of esophageal varices. Prior history of Endoscopic variceal ligation of esophageal varices is associated with increase in the occurrence of rectal varices. Presence of gastric varices and portal hypertensive gastropathy did not correlated with the presence of portal hypertension related colonic lesions. Serum bilirubin and prothrombin time were significantly more in patients with portal hypertension related colonic lesions. Lower serum albumin and platelet count correlate with presence and frequency of portal hypertension related colonic lesions.

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