ORIGINAL ARTICLE

FREQUENCY OF RECTAL VARICES IN PATIENTS WITH CIRRHOSIS

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ABSTRACT

Objective: To document the frequency of rectal varices in patients with cirrhosis of liver and compare it with that of oesophageal varices in liver and to compare the frequency of rectal varices with non-cirrhotic controls.

Design: A cross-sectional analytical survey.

Place and Duration of Study: The study was conducted in the medical wards of Civil Hospital, Karachi from August 2000 to July 2001.

Patients and Methods: All patients of confirmed cirrhosis of liver, presenting during the study period, were selected for initial workup. On the basis of upper gastrointestinal (GI) endoscopy, patients were segregated into those with oesophageal varices (Group-A) and those without them (Group-B). A matched control group (Group-C) was added, which consisted of patients of irritable bowel syndrome (IBS) who underwent sigmoidoscopic/colonoscopic examination during the study period. Fiberoptic sigmoidoscopy was done in all selected patients. Statistical analysis for continuous variables was done by student's 't' test while non-continuous variables were analyzed by Mann-Whitney-U test.

Results: A total of 104 patients (males 61; females 43) were included. Hepatic encephalopathy grade was significantly lower in Group-B ($p < 0.0001$). Grade-I varices were seen in 13 patients, Grade-II in 38 and Grade-III in 33 patients of Group-A. Rectal varices were present in 59.9% of patients in Group-A as compared to Group-B in which no one had them ($p<0.0001$).

Conclusion: Rectal varices are common in patients of portal hypertention.

KEY WORDS: *Portal hypertention. Rectal varices. Cirrhosis. Haemorrhoids. Colonopathy.*

INTRODUCTION

Cirrhosis of liver is a disease with many complications. In Pakistan it is mainly due to viral infection especially due to hepatitis B and C, although other causes like alcohol and metabolic diseases are also identified. In Pakistan the anti-HBc prevalence is reported to be at 31% and the prevalence of hepatitis C has been reported to be at 6.5%.[1] Portal hypertension is an important complication of cirrhosis and manifests as ascites, edema, esophageal varices and rectal varices. Variceal hemorrhage from esophagus is a life-threatening complication of portal hypertension with 30% mortality in the first bleed and with early re-bleeding occurring in 30-50% of patients.[2]

Although much work has been done on the upper GI complications of portal hypertension, the data on lower GI complications of portal hypertension is scanty. These complications include rectal and colonic varices, non-specific inflammatory changes, mucosal thickening and vasculopathy.[3-5] The diagnosis of rectal varices could be missed and can sometimes be fatal.[6] The cited frequency of rectal varices in cirrhosis varies from 44% to 89%.[7-10] The exact prevalence and significance of these lesions, their relationship to the severity of the liver disease, and their association with gastric mucosal changes in our area is, however, not known. The current study was designed to document the frequency of rectal varices in patients presenting with cirrhosis of liver and to correlate it with the presence of esophageal varices.

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PATIENTS AND METHODS

This cross-sectional survey was conducted in the Medical Wards of Civil Hospital, Karachi from August 2000 to July 2001. All patients of confirmed cirrhosis of liver, presenting during the study period, were selected for initial workup.

Exclusion criteria were co-existing cardiac disease, co-existing chronic obstructive airway disease, prior total colectomy, hepatic malignancy, fulminant hepatic failure or hepatic encephalopathy grade-IV patients, who have been operated for hemorrhoids and patients in whom porto-systemic shunt surgery had been done.

Findings of all the patients were entered on a proforma. In all patients, after obtaining an informed consent, upper GI endoscopy and sigmoidoscopy was done. Grading of varices was done according to Conn *et al*.[11] Rectal varices were defined as veins originating > 4 cm above the anal verge, clearly distinct from hemorrhoids, not contiguous with the anal columns and or pectinate line.[12] Hemorrhoids were defined as large venous structures at or just proximal to anus and were subdivided into internal and external hemorrhoids.[13] On sigmoidoscopy, rectal varices appeared as either discrete veins or secular swellings that were blue/slate grey in color, while hemorrhoids appeared as purple, well vascularised mucosa, prolapsing into the proctoscope.[14] Hepatic encephalopathy (HE) is a potentially reversible, complex neuropsychiatric syndrome, characterized by disturbances in consciousness and behaviour, personality changes, fluctuating neurological signs and asterixis and distinctive electro-encephalographic changes, which occurs secondary to chronic liver disease.[15] Grading of HE is given in Table I.

On the basis of upper GI endoscopy, patients were segregated into those with esophageal varices (Group-A), and those with-

out them (Group-B). A matched control group (Group-C) was added, which consisted of patients of irritable bowel syndrome (IBS), who underwent sigmoidoscopy/colonoscopy during the study period in our unit. Presence of rectal varices among the groups was compared. All selected patients underwent standard proctoscopic and flexible sigmoidoscopic examination by a consultant gastroenterologist. Statistical analysis for continuous variables was done by student's 't' test, while non-continuous variables were analyzed by Mann-Whitney-U test and X^2 test.

Results

A total of 104 patients fulfilling the selection criteria were included. These included 61 (58.7%) males and 43 (41.3%) females. Mean age ±SD of males was 48.5 ±11.5 years and that of females was 51.3 ±9.3 years. The difference of age in gender was not significant on student's 't' test (p = 0.19; 95% CI –6.94 to 1.47).

Table I: Grading of hepatic encephalopathy*,[1,35]

Grade	Level of consciousness	Personality and intellect	Neurological abnormalities	EEG abnormalities
0	Normal	No abnormality	Nil	Nil
Sub-clinical	Normal	No abnormality	Nil except impaired psychometric testing	Nil
1	Inverted sleep pattern; restless	Forgetful, mild confusion, agitation, irritability	Tremor, apraxia, incoordination, impaired hand writing	Slowing 5-cps triphasic waves
2	Lethargic; slow response	Disorientation for time, amnesia, decreased inhibitions, inappropriate behaviour	Asterixis, dysarthria, ataxia, hypoactive reflexes	Slowing triphasic waves
3	Somnolent but arousable; confused	Disorientation as regards place, aggresive behaviour	Asterixis, hyperactive reflexes; Babinski's sign; muscle rigidity	Slowing triphasic waves
4	Coma unarousable	Nil	Decerebrate	Slow 2-3 cps delta activity

*Adapted from: Gitlin N. Hepatic encephalopathy In: Zakim D, Doyer TD, (edi). Hepatology text book of liver diseases. 3rd ed. Philadelphia: *W B Saunder* 1996; **1**: 605-17.

History of jaundice was present in 85 (81.7%) of patients; hematemesis was present in 48 (46.2%), melena in 73 (70.2%), pedal edema in 68 (65.4%), clinical jaundice in 59 (56.7%), ascites in 82 (78.8%) and spleen was palpable in 93 (89.4%). Details are given in Table II.

Upper GI endoscopy was done within a week of admission of all the selected patients and was classified into two groups on the basis of presence of varices. Patients with varices were allocated to Group-A and those without it to Group-B. On this basis Group-A comprised of 84 patients and Group-B had 20 patients. Statistically significant differences between the two groups were found in age, hemoglobin levels, platelet counts, ALT, alkaline phosphatase, serum globulins and ascitic fluid albumin levels (Table III).

Table II: Clinical findings according to gender.

	Male (%)	Female (%)	Total (%)
History of jaundice	50 (48.1)	35 (33.7)	85 (81.7)
Hematemesis	33 (31.7)	15 (14.4)	48 (46.2)
Malena	41 (39.4)	32 (30.8)	73 (70.2)
Pedal edema	37 (35.6)	31(29.8)	68 (65.4)
Clinical jaundice	31 (29.8)	28 (26.9)	59 (56.7)
Ascites	44 (42.3)	38 (36.5)	82 (78.8)
Distended abdominal veins	41 (39.4)	22 (21.2)	63 (60.6)
Palpable spleen	55 (52.9)	38 (36.5)	93 (89.4)

Table III: Mean values with standard deviation and statistical signifi--cance of different variables according to groups.*

Variables	Groups	Mean	Std. Dev.	p-Value§
Age (years)	Group-A	48.6	10.5	0.03
	Group-B	54.1	10.4	
Haemoglobin (g/dl)	Group-A	10.9	2.2	0.0001
	Group-B	8.6	1.7	
TLC (x10^3/mm^3)	Group-A	7.1	3.2	0.2
	Group-B	6.3	2.9	
Platelets (x10^9/mm^3)	Group-A	112.7	55.9	0.01
	Group-B	149.0	65.9	
BUN (mg/dl)	Group-A	15.8	9.7	0.2
	Group-B	12.9	9.4	
Bilirubin (mg/dl)	Group-A	2.8	2.3	0.6
	Group-B	3.1	3.8	
ALT (IU)	Group-A	40.8	25.2	0.003
	Group-B	69.5	71.9	
Alk. Phosp. (IU)	Group-A	168.3	90.9	0.0001
	Group-B	294.7	105.4	
S. Albumin (g/dl)	Group-A	2.6	0.7	0.5
	Group-B	2.7	0.5	
S. Globin (g/dl)	Group-A	4.0	0.9	0.0001
	Group-B	3.1	0.1	
PT difference with control (sec.)	Group-A	5.2	2.6	0.6
	Group-B	5.6	1.9	
Portal vein diameter (mm)	Group-A	12.5	1.2	0.1
	Group-B	12.2	0.4	
Ascitic fluid albumin (g/dl)	Group-A	0.3	0.4	0.03
	Group-B	0.6	0.5	
Ascitic fluid TLC (mm^3)	Group-A	353.2	654.1	0.6
	Group-B	419.0	308.4	

** Group-A (n=84), Group-B (n=20)*
§ Statistically Significance p < 0.05

In Group-A, 24 (28.6%) patients were not in hepatic encephalopathy, while Grade-I HE was present in 14 (16.7%), Grade-II HE was present in 26 (31.0%) and Grade-III HE was present in 20 (23.8%) of patients.

In Group-B 16 (80%) of patients were not in HE while only 4 (20%) patients were in Grade-I HE. None of the patients in Group-B was having Grade-II or III HE. Patients in Group-B were having statistically significant lower HE Grade on Chi-square testing (p < 0.0001) Table IV.

All patients were tested for the hepatitis B and C. In Group-A, hepatitis B surface antigen was present in 19(22.6%), HCV

Table IV: Grading of hepatic encephalopathy, viral markers and sig - moidoscopic findings according to the groups.*

	Hepatic encephalopathy			
	Absent	Grade-I	Grade-II	Grade-III
Group-A	24 (28.6)	14 (16.7)	26 (31.0)	20 (23.8)
Group-B	16 (80.0)	4 (20.0)	0	0
	Hep B	Hep C	Hep B+C	Indeterminate
Group-A	19 (22.6)	46 (54.8)	12 (14.3)	7 (8.3)
Group-B	14 (70.0)	0	2 (10.0)	4 (20.0)
	Rectal varices		Haemorrhoids	
	Present	Absent	Present	Absent
Group-A	50 (59.5)	34 (40.5)	7 (8.3)	77 (91.7)
Group-B	0	20 (100.0)	2 (10.0)	18 (90.0)

** Group-A (n=84), Group-B (n=20).*

antibodies were detected in 46 (54.8%), 12 (14.3%) patients were harbouring both HBV and HCV, while in 7 (8.3%) both viruses tested negative. In Group-B hepatitis B was positive in 14 (70%), both hepatitis B+C were present in 2(10%) and both viruses were negative in 4 (20%) of patients (p<0.0001). Details are given in Table IV.

As only Group-A was having patients with oesophageal varices according to our grouping criteria, the results of only Group-A is presented. Grade-I varices were seen in 13 patients, Grade-II in 38 and Grade-III in 33 patients.

Sigmoidoscopic findings in Group-A revealed that rectal varices were present in 50 (59.5%) patients and were absent in 34 (40.5%) patients. In Group-B none of the 20 patients had rectal varices (p< 0.0001). Haemorrhoids in Group-A were present in 7(8.3%) and 2(10%) patients of Group-B which was non-significant (p= 0.6). A group of 63 patients who underwent sigmoidoscopy for workup of irritable bowel syndrome in our unit, during the study period, were included as a control Group-C. The frequency of haemorrhoids in this group was 5(7.9%) and none of them had rectal varices. Thus, there is no significant difference between the frequencies of haemorrhoids in the three groups. Details are given in Table IV.

Discussion

The current study showed that patients with oesophageal varices (Group-A) had significantly higher frequency of rectal varices as compared to those who did not had oesophageal varices (Group-B). It was due to the increase in portal pressure in patients of Group-A. Moreover the frequency of haemorrhoids was not affected by the presence or absence of oesophageal varices. Also there was no significant difference in frequency of haemorrhoids from that of control. A study from India has reported the frequency of rectal varices at 37% in patients with portal hypertension and none of the established parameters, e.g., aetiology of portal hypertension, child's class, oesophageal variceal eradication by sclerotherapy or band ligation, history of variceal bleeding, grade of oesophageal varices, presence of portal hypertensive gastropathy or gastric varices as predictive of the occurrence of colorectal varices.[16] Individuals with portal hypertension were reported to be more predisposed to develop colitis-like abnormalities and mucosal vascular lesions.[17-19] Exacerbation of hepatic dysfunction has no significant effect on increase in bleeding from rectal varices.[20]

Apart from these, many changes in the wall of blood vessels of colon have been described in patients of portal hypertension. It has been shown that cirrhotic patients have a significantly higher mean diameter of vessels in all three layers. Qualitatively, increased number of small vessels and prominent branching were noted, especially in the superficial and intermediate layers. Tortuous, thick-walled vessels, suggesting arterialisation of venules, were present in some cases.[21] Some researchers have pointed out that although the frequency of colorectal varices is increased in patients of cirrhosis; there is no significant increase in hepatic venous pressure gradient or glucagon as compared to controls showing that they are not causative factors.[22]

The prevalence of haemorrhage from rectal varices is significantly increased in rectal varices of more advanced form, and the prevalence is also significantly higher in patients with positive "red colour" sign.[20] Endoscopic injection with N-butyl-2-cyanoacrylate (Histoacryl), performed over the recto sigmoid varices effectively controls the bleeding from the rectal varices.[23] The transjugular intrahepatic porto-systemic shunting (TIPS) is also an effective modality in the therapy of cirrhotic patients with bleeding stomal or anorectal varices unresponsive to conservative management.[24-26] There were some apprehensions that treatment of oesophageal varices by sclerotherapy or band ligation may increase the risk of developing rectal varices. However, obliteration of oesophageal varices does not affect the prevalence of haemorrhoids, anorectal varices or portal hypertensive colopathy.[27-30] Some newer investigations are also being utilized for early detection of rectal varices. Endoscopic rectal ultrasound has been found to be superior in detecting early as well as florid changes in rectum.[31-32] Even transvaginal ultrasound is also very sensitive in detecting rectal and pararectal varices.[33] The researchers say pelvic CT scans of patients with portal hypertension can yield further information about the presence and extent of pararectal venous collaterals, which may be of particular importance in those patients requiring pelvic surgery. The presence of pararectal varices on CT and the diameter of the IMV do not correlate with the presence of rectal varices on colonoscopy. Decompression of portal hypertension by rectal and pararectal varices does not result in a decreased incidence of oesophageal varices.[34] Portal circulation, in particular the contribution of the inferior mesenteric vein, can be evaluated in a relatively non-invasive way by per rectal portal scintigraphy.[35] By this technique the rectal portal shunt index could be calculated which is useful in predicting the prognosis.[36]

Conclusion

Rectal varices are common in patients with portal hypertension. There is no significant difference in the occurrence of haemorrhoids in patients with or without portal hypertension and normal controls. In patients with portal hypertension, with lower GI bleeding, the possibility of rectal varices should be considered.

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