

Anorectal Varices in Liver Cirrhosis

ALI GABALLA, M.D.; HANAA EL DEGWI, M.D;
MAZEN NAGA, M.D.; NADIA EL FIKI, M.D.
and HUSSEIN KHAYRI, M.D.

*The Internal Medicine and Surgical Departments,
Faculty of Medicine, Cairo University.*

Abstract

The study comprised 135 patients with liver cirrhosis and portal hypertension. They were classified into three groups: Group A comprising 55 patients that had no history of esophageal bleeding, Group B comprising 70 patients that had undergone endoscopic injection for bleeding esophageal varices and Group C comprising 10 patients that had undergone splenectomy vasoligation. The prevalence of hemorrhoids and anorectal varices has been studied in the three groups. Only anorectal varices showed significant increase in their incidence after esophageal sclerotherapy ($p < 0.005$), but both hemorrhoids and anorectal varices, increase significantly with the increase in severity of liver disease ($p < 0.001$). Significant increase of hemorrhoids and anorectal varices was found with the increase in grading of esophageal varices with tendency to more significant increase in frequency of anorectal varices ($\chi^2 = 9.3 p < 0.01$). Anorectal varices also showed significant increase with the increase in the number of sessions of sclerotherapy ($\chi^2 = 9.3 p = 5.601 p = 0.07$). Finally it was found that, in all three groups, the presence or absence of hemorrhoids has no relation to the incidence of anorectal varices ($\chi^2 = 1.3 p < 0.05$).

Introduction

ANORECTAL varices are portosystemic collaterals which develop in patients with portal hypertension and can occur in the rectum, anal canal and/or external anal margin [1]. The etiology of and relation

between anorectal varices and hemorrhoids are the subject of much debate [2] Thompson [3] defined hemorrhoids as vascular cushions that had no direct connection to the portal vein. Although anorectal varices and hemorrhoids were believed by

Goligher [4] to be one and the same disorder, the concept of being separate entities has been enunciated in the work of Goenka et al in 1991 [5]. In fact it is very important to distinguish between rectal variceal bleeding and bleeding from hemorrhoids when they co-exist because their treatment differs, and resection of varices mistaken as hemorrhoids can be disastrous [6]. In the present study we aimed to establish the prevalence of anorectal varices and hemorrhoids in patients with liver cirrhosis, and to correlate their presence with the grading of esophageal varices and with the severity of liver disease. The effect of different therapeutic modalities viz sclerotherapy and devascularization on the incidence of anorectal varices and hemorrhoids was also studied.

Material and Methods

One hundred and thirty five patients with portal hypertension presenting to the gastrointestinal endoscopy and surgical departments of Kasr El Eini Hospital were included in the study. They were 47 females and 88 males with a mean age of 40.56 ± 8.6 . All patients had hepatic cirrhosis and portal hypertension. They were evaluated clinically and biochemically.

Clinical details of the patients included history of upper or lower gastrointestinal bleeding. Treatment received for gastrointestinal bleeding or anorectal disease was recorded.

Laboratory investigations included full blood picture, liver function tests, coagu-

lation profile and abdominal ultrasonography. After establishment of these parameters, patients were classified in accordance with Pugh's modification of Child's classification to assess the severity of their liver disease [7]. During upper gastrointestinal endoscopy, the presence of esophageal varices and their grading [8] as well as the gastric mucosal changes were recorded. Proctosigmoidoscopic examination was done for all patients after anal inspection and per-rectal examination using the fiberoptic sigmoidoscopy. Anorectal varices were identified using the criteria of Hosking et al [1] outlined in table (1).

Combined Peri and intra variceal injection technique was used for bleeding esophageal varices. It aimed at achieving sclerosis of the varices and the inner esophageal wall [9]. Ethanolamine oleate was the sclerosant used in all patients. The treatment was repeated at weekly intervals until the varices were largely obliterated. To achieve this 3 to 6 sessions were necessary.

Results

Patients were classified into 3 groups:

Group A: 55 patients (22 females & 33 males) mean age 40 ± 8.1 yrs. They had no history of upper gastrointestinal bleeding but all had esophageal varices on endoscopy.

Group B: 70 patients (23 females & 47 males) - mean age 40.52 ± 8.96 years. They underwent endoscopic injection

sclerotherapy for bleeding esophageal varices.

Group C: 10 patients (2 females & 8

males) mean 32.9 ± 4.77 years. They underwent splenectomy vasoligation. The results of all patients were shown in tables (2, 3, 4, 5 &) and Fig (1).

Table (1): Criteria for Diagnosing Lower Gastrointestinal Varices.

Site of varix	Method of examination	Features
External anal	Inspection	Perianal swelling, compressible, which refills 2 seconds of pressure release
Internal anal	Sigmoidoscopy	Blue / Slate-grey coloured veins or saccular swellings
Colorectal	Sigmoidoscopy	Dilated tortuous vessels.

Table (2): The Prevalence of Hemorrhoids and Anorectal Varices in the three Groups of Patients.

Vascular Lesions	Group A 55 pts	<i>p</i>	Group B 70 pts	<i>p</i>	Group C 10 pts	<i>p</i>
Hemorrhoids	Nb 22		24		4	
	% 40	> 0.05	34.29	> 0.05	40	> 0.05
			42		5	
Anorectal Varices	Nb 26	< 0.005		> 0.05		> 0.05
	% 47.27	Sig	60		50	
Free	Nb 7		4		1	
	% 12.73		5.71		10	

Table (3): The Relation of Hemorrhoids and Anorectal Varices in all Patients to Modified Child's grade of Liver Disease.

Vascular Lesions		Grade A	Grade B & C	<i>p</i>
Hemorrhoids	Nb	11	39	< 0.001
	%	22	78	Sig
Anorectal Varices	Nb	14	58	< 0.001
	%	19.44	80.56	Sig

Table (4): Relation of Hemorrhoids and Anorectal Varices to Grades of Esophageal Varices.

Vascular Lesions		Grade I Varices 5 pts	Grade II Varices 17 pts	Grade III Varices 19 pts	Grade IV Varices 14 pts
Hemorrhoids	Nb	1	5	7	9
	%	20	29.41	36.84	64.29
Anorectal Varices	Nb	0	4	10	12
	%	0	23.35	52.63	85.71

$$x^2 = 9.3$$

$p < 0.01$ Sig.

Table (5): Relation of Hemorrhoids and Anorectal Varices to Number of Sclerotherapy Sessions.

Vascular Lesions		Sessions 14 pts	4 Sessions 34 pts	5 Sessions 16 pts	6 Sessions 6 pts
Hemorrhoids	Nb	5	4	3	2
	%	35.71	41.18	18.75	33.3
Anorectal Varices	Nb	7	19	10	5
	%	50	55.88	62.5	83.3

$$x^2 = 5.601$$

$p < 0.01$ Sig.

Table (6) The Prevalence of Anorectal Varices in Patients with and without Hemorrhoids Disease.

Patients With hemorrhoids	Vascular		Grade A	Grade B	Grade C
	lesions		22 pts	24 pts	4 pts
	Prevalence	Nb	12	16	1
	of anorectal varices	%	54.54	66.67	25
Patients Without hemorrhoids	Vascular		Grade A	Grade B	Grade C
	lesions		33 pts	46 pts	6 pts
	Prevalence	Nb	14	25	4
	of anorectal varices	%	42.42	54.35	66.66

$$x^2 = 5.601$$

$p > 0.01$ Non Sig.

Discussion

Few data are available as to the prevalence of colonic disease in cirrhotics. The prevalence of rectal varices and hemorrhoids in cirrhotic subjects is controversial [10]. Although Jacobs et al [11] report 0% and 28% prevalences for rectal varices and hemorrhoids respectively, Hosking et al in 1989 [1] report 44% and 63% prevalences of these two lesions. Other authors report rectal varices in 3% of cirrhotic patients [12] compared with 0.07% of the general population [13]. Rabinovitz et al in 1990 [10] reported 3.6% and 25.2% for rectal varices and hemorrhoids respectively.

In the present study the prevalence of hemorrhoids was 40% in patients with no

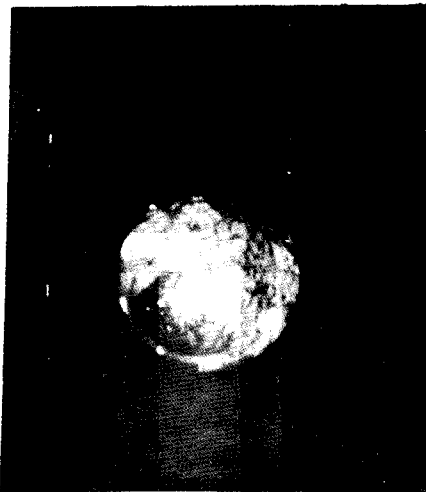


Fig. 1: Colorectal varices seen by flexible sigmoidoscopy.

history of upper gastrointestinal hemorrhage (group A), 34.29% in patients who underwent injection sclerotherapy (group B) and 40% in patients who underwent splenectomy vasoligation (group C). Regarding the prevalence of anorectal varices, it was 47.27% in group A, 60% in group B & 50% in group C (Table 2). These results denote higher prevalence of anorectal varices in patients with obliterated esophageal varices following sclerotherapy ($p < 0.005$). Hosking et al [1] and later Goenka et al [5] found no difference in the incidence of anorectal varices in patients with freshly diagnosed esophageal varices and those whose varices had been obliterated by sclerotherapy. On the other hand Foutch and Sivak [14] and later Keane and Brilton in 1986 [15] reported the occurrence of massive bleeding from colorectal varices after endoscopic injection sclerosis of esophageal varices. They postulated the hypothesis that lower GIT varices are more prevalent and may bleed after esophageal variceal sclerotherapy. Our results support this hypothesis.

The results of our study also showed significant increase in the prevalence of both hemorrhoids and anorectal varices with the increase in the severity of liver disease ($p < 0.001$) as shown in table (3). These results are different from those of Rabinovitz et al [10] who showed a significant trend for the presence of hemorrhoids but not for anorectal varices with the increasing degrees of disease severity.

Other investigators [1, 16] showed that the prevalence of anorectal varices had no relation to Child's Pugh's grading.

In the present study all patients had liver cirrhosis and portal hypertension. The pathogenesis of hemorrhoids and/or rectal varices would seem to be dependent on the venous drainage of the rectum and anal canal. Varices occur where portosystemic anastomosis are present, however, colonic varices are less common than esophageal varices [1, 17]. The relationship between portal hypertension, esophageal varices, rectal varices and hemorrhoids has been widely studied. Doberneck and Janovski [18] and later Izsak and Finlay [19] reported that portal hypertension was found in three fourths of cases with rectal varices, however, he showed no association between the presence or absence of rectal varices and the degree of portal hypertension. Also Goenka et al. [5] found no correlation between anorectal varices and esophageal variceal sore, portal congestive gastropathy or history of esophageal variceal bleeding. Wang et al. [16] obtained the same results. In contrast Hosking et al. [1] showed that the prevalence of anorectal varices increased with the length of history of portal hypertension. They stated that in the presence of portal hypertension, esophageal varices usually but not invariably develop before anorectal varices while hemorrhoids occur in the absence or presence of esophageal varices. The results of our study showed that the prevalence of

hemorrhoids and anorectal varices increased significantly with the esophageal score with tendency to more significant increase in anorectal varices than in hemorrhoids ($x^2 = 9.3$ & p value 0.01) as shown in table 4.

When we correlated the hemorrhoids and anorectal varices to the number of sclerotherapy sessions we found that there is significant increase of the anorectal varices with the increase in the number of session with x^2 value of 5.601 & p value of 0.07 (as shown in table 5). This indicates that the severity of esophageal varices and the completion of ablation is accompanied with higher incidence of anorectal varices. This can be explained on hemodynamic changes in the portal bed following obliteration of the varices. Fergali and Kamel [20] studied these hemodynamic changes after sclerotherapy by duplex sonography and reported an increased in size of portal vein, splenic vein and superior mesenteric vein with increase cross section, diminished velocity and reverse flow in the superior mesenteric vein. This is supported by the study on the pathogenesis of hemodynamic changes in the portal bed that was suggested by Khayri in 1966 [21]. He postulated the possibility of the shift of the portal blood from gastroesophageal communication to other sites of the portosystemic collaterals. On the other hand the group of patients studied after surgical ablation (group C) had the incidence of their hemorrhoids and anorectal varices not

changing significantly ($p < 0.05$ table 1) as compared to patients of group A who had no obliteration of their esophageal varices.

Accurate differentiation between anorectal varices and hemorrhoids is essential for proper treatment. The results of our study showed that whether the patient is bleeding (group B-C) or not (group A) the incidence of anorectal varices did not differ significantly in patients having hemorrhoids from those not having hemorrhoids $x^2 = 1.3$ P value > 0.05 (table 6) i.e. presence or absence of hemorrhoids had no relation to the prevalence of anorectal varices. Bleeding from anorectal varices although infrequent, has been reported by many authors [22, 23, 6 & 5] and it is very important to distinguish between rectal variceal bleeding and bleeding from hemorrhoids when they co-exist. Banding, submucosal injection cryosurgery and resection are the modalities of treatment used for hemorrhoids while underrunning sutures [6], sclerotherapy [22] and portosystemic shunts [24] are used for anorectal varices. Hosking and Johnson [6] showed that resection of varices mistaken as hemorrhoids can occasionally be disastrous. Recently, Heaton et al in 1992 [25] showed that injection sclerotherapy is satisfactory for symptomatic anorectal varices.

According to the above findings we can conclude that awareness of the prevalence of anorectal varices is important in patients with liver cirrhosis. Their presence

can be a useful bedside examination to support variceal etiology for upper gastrointestinal hemorrhage. The relationship to esophageal varices, sclerotherapy parenchymal liver disease and possible coexistence with hemorrhoids may alert the clinician to the proper diagnosis in cirrhotic patients especially those presenting with gastrointestinal hemorrhage.

However, there remains two important questions:

- (a) The possible variation in the pathogenesis of hemorrhoids and anorectal varices in portal hypertension.
- (b) The low incidence of bleeding from anorectal varices as compared to the high incidence of bleeding from esophageal varices, possibly due to anatomical variation of the mucosa of both sites, alkalinity versus acidity (erosive theory) respectively or periodicity of the motility of the rectum versus continuous motility even at the rest of the esophagus.

References

1. HOSKING S.W., SMART HI, JOHNSON A. G, & TRIGER D.R.: Anorectal varice, hemorrhoids and portal hypertension. *Lancet*, 1-349-52, 1989
2. WATTS G.T.: Piles are caused by varicose veins in the anal canal. *Lancet*. 1-144-45, 1986.
3. THOMSON W. H.: The nature of hemorrhoids. *Br. J. Surg.*, 62: 542-52, 1975.
4. GOLIGHER J.C.: *Surgery of the anus, rectum and colon* 5th ed. London: Balliere Tindall, 98-99, 1985.
5. GOENKA. M. K, KOCHHAR R, NAGI B & MEHTA S.K.: Rectosigmoid Varices and other mucosal changes in patients with portal hypertension. *Am. J. Gastroent* 9-1185-89, 1991.
6. HOWKING S.E. & HOHNSON A.G.: Bleeding anorectal varices - a misunderstood condition. *Surgery*, 104-70-73, 1988.
7. PUGH RNH, MURRAY - LYON IM, DARWSON JL PIETRONI MC, WILLIAMS R.: Transection of the esophagus for bleeding esophageal varices. *Br. J. Surg.*, 60: 646-649, 1973.
8. BUSET, M., DES MAREZ, B., BAIZE M., BOURGEOIS N. and CREMER M.: Bleeding esophagogastric varices: An endoscopic Study. *Am. J. Gastroenterod.*, 82, No. 3: 241-244, 1987.
9. SOEHENDRA, N., DE HEER K., KEMPENERS J., and RUNGE M.: Sclerotherapy of esophageal varices: acute arrest of gastrointestinal haemorrhage or long-term therapy. *Endoscopy*, 15, 136, 1983.
10. RABINOVITZ M, SCHADE R. R., DINDZANS VJ, BELLE S, THIEL D & GOVALER J.: Colonic disease in cirrhosis. *Gastroenterology*, 99-195-99, 1990.
11. JACOBS D.M., BUBPICK MP, ONSTAD

- G, HITCHCOCK R.: The relationship of hemorrhoids to portal hypertension. *Dis. Colon Rectum*, 23: 567-59, 1980.
12. BRILTON R. C.: Influence of portal systemic collateral patterns and distribution of varices on results of surgical treatment of bleeding esophageal varices. *Surgery*, 53: 567-574, 1963.
13. FELDMAN M, SMITH VM, WARNER CG.: Varices of the colon. Report of three cases *J.A.M.A.*, 179: 729-30, 1962.
14. FOUTCH PC and SIVAK MV.: Colonic variceal hemorrhage after endoscopic injection sclerosis of esophageal varices: A report of three case. *Am. J. Gastroenterol.*, 79: 756-60, 1985.
15. KEANE RM and BRILTON DC.: Massive bleeding from rectal varices following repeated injection sclerotherapy of esophageal varices. *Br. J. Surg.*, 73:120, 1986.
16. WANG TP, LEE FY, TSAI Y T, LEE SD WANG S. S., HSIA H. C. et al.: Relationship of portal pressure, anorectal varices and hemorrhoids in cirrhotic patients. *J. Hepatol.*, 15 (1-2) 170-3, 1992.
17. CHAWLA Y, DILAWARI J. B.: Anorectal varices, their frequency in cirrhotic and non cirrhotic portal hypertension. *Gut*, 32 (3) 309-11, 1991.
18. DOBERNECK RC and JANOVSKI NA. Isolated bleeding from colonic varices in patients with liver disease. *Am. J. Dig. Dis.* 15: 834-841, 1970.
19. IZSAK E. M. and FINLAY J.M.: Colonic Varices. Three case reports and review of the literature. *Am J. Gastroenterol.*, 73: 131-136, 1980.
20. FERGALI A and KAMEL N.: Effect of sclerotherapy on the portal circulation using Duplex scanning in portal hypertension. *Monufia Medical Journal*, Vol. 2-3 189-194, 1992.
21. KHAYRI M.: Splenotomy devascularisation operation for the treatment hepatic fibrosis. *Kasr El Eini J. surg.*, 7:37-42, 1966.
22. WANG M., DESIGAN G, DUNN D.: Endoscopic sclerotherapy for bleeding rectal varices. A case report. *Am. J. Gastroenterol.*, 80: 779-80, 1985.
23. WELNSHEL E, CHEN W, FAL KENSTEIN D. B. et al.: Hemorrhoids or rectal varices: defining the cause of massive hemorrhage e portal hypertension. *Gastroenterology*, 90: 744-47, 1986.
24. Mc CORMACK TT, BAILEY H. R., SIMMS J. M. et al.: Rectal varices are not piles. *Br. J. Surg.*, 71: 163, 1984.
25. HEATON ND, DAVENPORT M, HOWARD E.R: Symptomatic hemorrhoids and anorectal varices in children with portal hypertension *J. Pediatr. Surg.*, 27(7) 833-35, 1992.