

Anorectal varices – their frequency in cirrhotic and non-cirrhotic portal hypertension

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Abstract

Anorectal varices in portal hypertension have been little studied. Seventy eight per cent of 72 patients with portal hypertension had anorectal varices shown at flexible sigmoidoscopy. Significantly more patients with non-cirrhotic portal hypertension had these varices than patients with cirrhosis (89% v 56%, $p < 0.01$).

Portal hypertension leads to the development of collaterals between the portal and systemic circulations, namely at the gastroesophageal junction, anal region, falciform ligament, and areas where the abdominal organs are in contact with retroperitoneal tissues. These collateral vessels may not develop to the same degree since the amount of blood flowing through them may differ. Because of this, varices at one region may be small and those at another large. Hosking *et al* recently showed a frequency of anorectal varices of 44% in their group of cirrhotic patients.¹ We have previously reported the frequency of anorectal varices in a small number of patients with portal hypertension from our centre.² Since we have an appreciable proportion of patients with non-cirrhotic portal hypertension we investigated the frequency of anorectal varices in this group of patients and compared it with the value found in patients with cirrhosis.

Patients and methods

We studied 72 consecutive patients with portal hypertension. Twenty five patients had cirrhosis proved at biopsy (mean (SD) age 44.5 (10.8) years) and 47 had non-cirrhotic portal hypertension (mean (SD) age 20.9 (8.44) years). The aetiology of cirrhosis in the 25 patients with this disorder was alcohol in 16, hepatitis B virus in four, and cryptogenic in five. The non-cirrhotic

group included 37 patients with extrahepatic portal venous obstruction (mean (SD) age 18.8 (6.8) years) diagnosed by splenoportovenography and 10 patients with non-cirrhotic portal fibrosis (mean (SD) age 28.6 (9.6) years) diagnosed by splenoportovenography and liver biopsy according to the criteria laid down by Indian Council of Medical Research.³ All these patients were assessed clinically for any upper or lower gastrointestinal bleeding.

Fifty five (76%) of the 72 patients had presented with an upper gastrointestinal bleed in the past and were thus put on a sclerotherapy programme. Only one patient presented with bleeding per rectum.

All patients were assessed for oesophageal varices with an upper gastrointestinal endoscope (Olympus GIF X Q) and anorectal or colonic varices (limited to the rectum, sigmoid, and descending colon) with a flexible sigmoidoscope (Olympus CF, PIOS). Colonic preparation for sigmoidoscopy was done by giving 200 ml of 20% mannitol orally three to four hours before the sigmoidoscopic examination. Flexible sigmoidoscopy was undertaken once the patient developed loose clear stools.

Anorectal varices were diagnosed if bluish or grey distended tortuous or sacular veins were seen above the anal margin and extending into the rectum. They were described as small or large if their diameter was less or more than 5 mm respectively. Similarly, oesophageal varices were said to be small (grade I-II) or large (grade III-IV) if their diameter was less or more than 5 mm respectively.

The anorectal varices were shown to a second observer and an independent assessment was made.

Results

Anorectal varices were observed in 56 (77.7%) of

TABLE I Frequency (%) of anorectal varices, oesophageal varices, and upper gastrointestinal (UGI) bleeding in patients with non-cirrhotic portal hypertension (NCPH) and cirrhosis (C)

	Non-cirrhotic patients			Cirrhotic patients (n=25)	Statistical significance (p)			
	EHPO (n=37)	NCPF (n=10)	NCPH (n=47)		NCPH v C	EHPO v C	NCPF v EHPO	NCPH v C
Anorectal varices:								
Large	16 (43)	4 (40)	20 (42)	4 (16)	<0.05	<0.05	NS	NS
Small	19 (51)	3 (30)	22 (47)	10 (40)	NS	NS	NS	NS
Large+small	35 (94)	7 (70)	42 (89)	14 (56)	<0.01	<0.01	NS	NS
Absent	2 (5)	3 (30)	5 (10)	11 (44)	<0.01	<0.01	NS	NS
Oesophageal varices:								
Large	34 (92)	9 (90)	43 (91)	17 (68)	<0.05	<0.05	NS	NS
Small	3 (8)	1 (10)	4 (8)	6 (24)	NS	NS	NS	NS
Large+small	37 (100)	10 (100)	47 (100)	23 (92)	NS	NS	NS	NS
Absent	0	0	0	2 (8)	NS	NS	NS	NS
UGI bleed:								
Present	35 (95)	7 (70)	42 (89)	13 (52)	<0.01	<0.01	NS	NS
Absent	2 (5)	3 (30)	5 (10)	12 (48)	<0.01	<0.01	NS	NS

EHPO=extrahepatic portal venous obstruction; NCPF=non-cirrhotic portal fibrosis; NS=not significant.

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TABLE II Correlation of anorectal varices with upper gastrointestinal (GI) bleeding

Anorectal varices	Upper GI bleed (no (%))		p
	Present	Absent	
Large	21 (38)	3 (17.6)	NS
Small	26 (38)	6 (35.2)	NS
Large+small	47 (85.4)	9 (53)	<0.02
Absent	8 (14.5)	8 (47)	<0.02

the 72 patients with portal hypertension. Forty two (89.3%) of the 47 patients with non-cirrhotic portal hypertension had anorectal varices compared with 14 (56%) of the 25 patients with cirrhosis ($p < 0.01$). Anorectal varices were also significantly more common in extrahepatic portal venous obstruction compared with cirrhosis ($p < 0.01$) (Table I). Large anorectal varices were seen in 24 patients (16 extrahepatic portal venous obstruction, four non-cirrhotic portal fibrosis, and four cirrhosis) and small in 32 patients (19 extrahepatic portal venous obstruction, three non-cirrhotic portal fibrosis, 10 cirrhosis). Significantly more patients with non-cirrhotic portal hypertension and extrahepatic portal venous obstruction had large anorectal varices compared with cirrhosis patients ($p < 0.05$). Upper gastrointestinal bleeding as a presenting feature was also significantly more common in non-cirrhotic portal hypertension and extrahepatic portal venous obstruction compared with cirrhosis (Table I). Of the 55 patients who presented with an upper gastrointestinal bleed, 47 (85.4%) had evidence of anorectal varices, which was significantly higher than in patients without bleeding ($p < 0.02$) (Table II).

Oesophageal varices were detected in 70 (97%) of the 72 patients (Table I). Large oesophageal varices were significantly more common in patients with non-cirrhotic portal hypertension and extrahepatic portal venous obstruction than in cirrhotic patients. Although more patients with large oesophageal varices had large anorectal varices compared with those patients with small oesophageal varices, the difference was not statistically significant.

There was no significant difference in the frequency of anorectal varices, oesophageal varices, and upper gastrointestinal bleeding when patients with non-cirrhotic portal fibrosis were compared with extrahepatic portal venous obstruction and cirrhosis.

None of the patients studied had evidence of varices in the sigmoid or descending colon.

Discussion

Portal hypertension in most patients results in the development of oesophagogastric varices which are associated with massive upper gastrointestinal bleeds. Rectal varices, on the other hand, constitute another collateral pathway, which helps in decompressing the portal system into the systemic circulation through the superior middle and inferior haemorrhoidal veins.^{4,6} They are not usually associated with appreciable morbidity.

Our finding of anorectal varices in 78% of patients with portal hypertension is a significantly higher percentage than the 44% reported

by Hosking *et al.*¹ This difference is related to the different population of patients studied in the two series. Two thirds of our patients were non-cirrhotic and a third were cirrhotic whereas all their patients had cirrhosis.¹ Our cirrhotic patients had similar frequency of anorectal varices to that reported by Hosking *et al.*¹ Indeed, we found a significantly higher incidence of varices in patients with non-cirrhotic portal hypertension (especially extrahepatic portal venous obstruction) than in cirrhotic patients. This is not surprising since patients with non-cirrhotic portal hypertension, present with complications of portal hypertension alone, which are much greater and of longer standing – for example upper gastrointestinal bleed – than those found in patients with cirrhosis, where other complications of liver disease such as ascites and encephalopathy are likely to precipitate a presentation. Though patients with non-cirrhotic portal hypertension differed significantly from patients with cirrhosis in terms of presence of anorectal varices, oesophageal varices, and upper gastrointestinal bleeding as shown in Table I, this difference was mainly contributed by patients with extrahepatic portal venous obstruction. However, the number of patients with non-cirrhotic portal hypertension in this study was not great enough to make a categorical statement on whether these patients behave more like those with extrahepatic portal venous obstruction or cirrhosis. It has also been observed by other authors that ectopic varices are more commonly observed in patients with portal venous obstruction than in those with cirrhosis.⁷

Unlike oesophageal varices, anorectal varices rarely bleed. Bleeding anorectal varices have been reported by Wilson *et al* in two of 309 patients with oesophageal varices,⁸ while McCormack observed this in four of 112 patients with portal hypertension.⁹ In a larger series by Johnson, rectal varices were reported in five of their 1100 patients,¹⁰ while Hosking *et al* observed bleeding anorectal varices in only two of 100 patients with portal hypertension.¹ In our series, only one patient with extrahepatic portal venous obstruction bled from anorectal varices.

Our findings agree with those of Hosking *et al* that anorectal varices reflect a later stage in the development of portal hypertension.¹ This is supported by the fact that patients with large oesophageal varices have more frequent anorectal varices than patients with small or no oesophageal varices. Moreover, the incidence of anorectal varices was significantly higher in patients who had an upper gastrointestinal bleed, which, as a complication, occurs later in the course of portal hypertension.

Anorectal varices are best shown by endoscopy, since it is a direct means of establishing a diagnosis. We preferred to use a flexible sigmoidoscope in assessing anorectal varices, since patients tolerate it better and the bowel lumen can be distended with air, allowing a better view of the rectum than with rigid sigmoidoscope.¹¹ We believe that the flexible sigmoidoscope is as sensitive as the proctoscope in showing anorectal varices, although internal piles that are not anorectal varices may be missed.¹²

The incidence of colonic varices in portal hypertension has not been reported previously. Only 24 cases of bleeding colonic varices from the sigmoid or descending colon have been reported.⁷ We did not find sigmoid or descending colon varices in any of the 72 patients we studied.

In conclusion, in our series significantly more patients with non-cirrhotic portal hypertension had anorectal varices, compared with patients with cirrhosis. Even if the varices are large, however, they do not usually bleed.

- 1 Hosking SW, Johnson AG, Smart HL, Triger DR. Anorectal varices, haemorrhoids and portal hypertension. *Lancet* 1989; i: 349–52.
- 2 Chawla YK, Dilawari JB. Anorectal varices haemorrhoids and portal hypertension. *Lancet* 1989; i: 725.

- 3 Workshop on non-cirrhotic portal fibrosis. Indian Council of Medical Research, New Delhi: 1969: 36.
- 4 Edwards FA. Functional anatomy of portosystemic communications. *Arch Intern Med* 1951; 88: 137–54.
- 5 Britton RC. Influence of porto-systemic collateral patterns and distribution of varices on results of surgical treatment of bleeding esophageal varices. *Surgery* 1963; 53: 567–74.
- 6 Fleming RJ, Seeman WB. Roentgenographic demonstration of unusual extra esophageal varices. *Am J Roentgenol* 1968; 103: 281–90.
- 7 Lebrech D, Benhamau JP. Ectopic varices in portal hypertension. *Clin Gastroenterol* 1985; 14: 105–21.
- 8 Wilson SE, Stone RT, Christie JP, Panaro E. Massive lower gastrointestinal bleeding from intestinal varices. *Arch Surg* 1979; 114: 1158–61.
- 9 McCormack TT, Barkley HR, Simms JM, Johnson AG. Rectal varices are not piles. *Br J Surg* 1984; 74: 163.
- 10 Johnson K, Bardin J, Orloff MJ. Massive bleeding from hemorrhoidal varices in portal hypertension. *JAMA* 1980; 244: 2084–5.
- 11 Bohlman TW, Katon RM, Lipshutz GR, McCool MF, Smith FW, Clifford SM. Fibroptic pansigmoidoscopy. *Gastroenterology* 1977; 72: 644–9.
- 12 Bernstein WC. What are hemorrhoids and what is their relationship to the portal system? *Dis Colon Rectum* 1983; 26: 829–34.