The Relationship of Hemorrhoids to Portal Hypertension*

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Jacobs DM, Bubrick MP, Onstad GR, Hitchcock CR. The relationship of hemorrhoids to portal hypertension. Dis Colon Rectum 1980;23:567-569. Records of 188 patients with documented portal hypertension were reviewed to determine the incidence of hemorrhoids as well as bleeding complications associated with this condition. The incidence of hemorrhoids among these patients was not increased compared to the normal population. Six of the patients with portal hypertension did, however, bleed massively from hemorrhoids. Elevated portal venous pressure is an important factor in those patients having severe hemorrhoidal bleeding. The presence of coagulation defects may also be of considerable importance. [Key words: Hemorrhoidal disease: Hypertension, portal]

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CONSIDERABLE CONTROVERSY exists over the relationship of hemorrhoids to portal hypertension. Nesselrod[1] has stated that hemorrhoids are not related to portal hypertension as the hemorrhoidal plexus is decompressed by the hypogastric venous system. Goligher[2] *et al.* contend that hemorrhoids can, on occasion, be causally related to portal hypertension. In order to help clarify these conflicting concepts a study was made of hemorrhoids complicating portal hypertension at the Hennepin County Medical Center.

Patients and Methods

Records were reviewed of 188 patients treated for portal hypertension at the Hennepin County Medical Center from January 1973 through December 1979. The presence of esophageal varices was used to document the existence of portal hypertension (Table 1). Esophageal varices were diagnosed endoscopically in 157 patients (84 per cent), surgically in 10 patients (5 per cent), and at autopsy in 21 patients (11 per cent).

Clinical Characteristics of the Study Group: The average age was 50 years, with a range of 23 to 82 years. There were 125 men (67 per cent) and 63 women (33 per cent). Etiology of the liver disease was Laennec's cirrhosis in 174 patients (93 per cent), postnecrotic cirrhosis in eight patients (4 per cent) and a variety of other causes in six patients (Table 2). Clotting studies as measured by prolongation of prothrombin time to greater than 2 seconds above control or elevation of the partial thromboplastin time to greater than 40 seconds, were abnormal in 99 patients (53 per cent). At the end of the study period 100 patients (54 per cent) had died and most of these deaths (83 per cent) were related to the underlying liver disease.

Clinical Characteristics of Patients with Hemorrhoids: Fifty-two patients had clinically significant hemorrhoids documented by history and examination (27.6 per cent). This group was similar to the entire group in age, sex, distribution, type of liver disease, and degree of clotting abnormality.

The hemorrhoids were classified as mild if there was no significant prolapse (31 patients), moderate if manifested by mucosal prolapse (6 patients), and severe if acutely thrombosed and unreducibly prolapsed (1 patient). In 14 cases the severity could not be determined (Table 3).

Bleeding was reported by 37 of the patients with hemorrhoids (71 per cent); this bleeding could be documented in the hospital in only 23 patients (44 per cent). Six of these 23 patients bled massively in the hospital requiring transfusion of an average of 6.8 units of blood per patient. Among these six patients requiring transfusion, hemorrhoids were classified as mild in four patients and moderate in two patients (Table 4).

The incidence of coagulation defects was 52 per cent among the 23 patients with documented hemorrhoidal bleeding and 52 per cent for the entire study group. The incidence of coagulation defects for the subset of patients who required transfusion, surgery, or both was 82 per cent (Table 5).

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TABLE 1. *Documentation of Portal Hypertension by Demonstration of Esophageal Varices*

Method	Patients	
	Number	Per Cent
Endoscopy	157	84
Surgery	10	5
Autopsy	21	11
	188	

TABLE 2. *Underlying Liver Disease*

	Patients	
	Number	Per Cent
Laennac's cirrhosis	174	93
Postnecrotic cirrhosis	8	4
Retroperitoneal fibrosis	1	3
Primary biliary cirrhosis	1	
Hepatoma	1	
Metastatic cancer	1	
Polycystic	1	
Indeterminate	1	
	188	

TABLE 3. *Hemorrhoids in Patients With Portal Hypertension*

Classification	Number of Patients
Mild	31
Moderate	6
Severe	1
Indeterminate	14
	52

Treatment

The basic approach to these patients was conservative. Bed rest, fluid replacement, correction of coagulation defects and stool-softening agents controlled bleeding in most of the patients. Blood transfusions were given to all six of the patients bleeding massively. Two of these patients also required surgical intervention; one patient had a single quadrant hemorrhoidectomy and the other had a three quadrant banding. Among the remaining 17 patients, seven received elective local treatment of their hemorrhoids; three of these patients had banding procedures and four patients had hemorrhoidectomies. Two of the hemorrhoidectomies were single quadrant excisions.

Results

None of the patients had recurrence of rectal bleeding following banding or hemorrhoidectomy (Table 6). One patient died from a massive esophageal variceal hemorrhage seven days after hemorrhoidectomy.

Of the 29 patients with hemorrhoids and no documented bleeding history, nine subsequently underwent portacaval decompression. None of these patients had late hemorrhoidal bleeding.

Of the 23 patients with hemorrhoidal bleeding, six had subsequent portacaval decompression. No patient in this group had significant postshunt hemorrhoidal bleeding.

Discussion

The major theories of the etiology of hemorrhoids usually propose anal submucosal varicosities, redundant anal mucosa, or some form of vascular hyperplasia as the anatomic structure we call a hemorrhoid.[3] The predisposing conditions are many and include dietary habits, sedentary life style, bowel habits, and pregnancy.[2]

The relationship of portal hypertension to hemorrhoids has been especially controversial. Nesselrod[1] has stated that hemorrhoids are not related to portal hypertension as the hemorrhoidal plexus is decompressed by the hypogastric venous system. Taylor[4] demonstrated that pressure equal to or greater than portal pressure exists in the hemorrhoidal venous plexus and he emphasized the distinct clinical absence of hemorrhoidal bleeding among cirrhotic patients. However, Goligher *et al.*[2] and others[5] have stated that there is an etiologic relationship between portal hypertension and hemorrhoids.

Our present study agrees with Hunt's[6] finding and indicates that the incidence of hemorrhoids in patients with portal hypertension is not increased above that expected in the population at large. We report an incidence of 28 per cent which is actually low compared to the incidence of 52 per cent that Buie[7] reported from a large series of patients examined proctoscopically at the Mayo Clinic.

The frequency of hemorrhoids in patients with portal hypertension is less important clinically than the demonstration of a subset of patients with portal hypertension who tend to bleed massively from their hemorrhoids. This level of bleeding is far greater than that seen in the noncirrhotic population. Given the propensity for serious bleeding, the anatomic relationship of the hemorrhoids to the portal venous system becomes important. The venous drainage of the hemorrhoids is, in part, through the superior hemorrhoidal system and this system is under increased pressure in patients with portal hypertension. Coagulation abnormalities undoubtedly play an im-

TABLE 4. *Bleeding in Patients With Hemorrhoids and Portal Hypertension*

	Patients	
	Number	Per Cent
Nonbleeding hemorrhoids	29	56
Bleeding hemorrhoids	23*	44
Massive bleeding	6	11

* Includes only documented cases of bleeding.

portant role in this bleeding. It is our feeling, however, that the underlying liver disease in portal hypertension contributes greatly to this inordinate bleeding problem.

Because of the propensity of hemorrhoids to bleed excessively in the presence of portal hypertension we have been reluctant to perform any procedures that might lead to slough of anorectal tissue with resultant raw surfaces. We have also found conservative measures to be adequate for most patients. These measures include bed rest, fluid replacement, correction of coagulation defects with fresh frozen plasma and vitamin K, stool softeners or bulk-forming agents, and periodic use of blood transfusion as indicated. We have not used injection therapy or cryotherapy in these patients partly because of concerns for mucosal slough.

When conservative therapy has failed we have chosen either banding or hemorrhoidectomy as local treatment. Of particular value from the point of view of safety is the limited one-quadrant hemorrhoidectomy which we now favor as the initial operative procedure of choice for patients with portal hypertension who continue to bleed. This procedure offers the best control of bleeding at the time of the procedure and has a minimal potential for slough of necrotic tissue. The safety of this procedure lies in the fact that the wound can be resutured in the event of late bleeding in the postoperative period. Such resuture may be difficult or impossible in the presence of multiple suture lines from a formal three-quadrant hemorrhoidectomy. Following the one-quadrant hemorrhoidectomy additional quadrants may be staged if bleeding persists from other sites. To date we have encountered no significant bleeding in any of our patients following this procedure.

In view of the proposed relationship between hemorrhoids and portal hypertension it is noteworthy that none of the 15 patients who subsequently underwent portacaval shunting procedures had further rectal bleeding. This may be a significant relationship. If portal hypertension is related to the severity of bleeding in these patients, an adequate decompression should also decrease the severity if not the incidence of bleeding. This finding is especially important for the patient in whom bleeding cannot be controlled with conservative measures and local therapy alone. In such an instance portacaval decompression may be lifesaving.

TABLE 5. *Coagulation Defects**

	Number of Patients	Coagulation Defects	
		Number	Per Cent
Entire study group	188	98	52
Patients with bleeding hemorrhoids	23	12	52
Patients requiring surgery, transfusion or both	11	9	82

* Prothrombin times >2 sec above control or PTT >40 sec.

TABLE 6. *Results of Treatment and Follow-up*

	Number of Patients
Patients having nonbleeding hemorrhoids	29
Subsequent portacaval decompression for esophageal varices	9
Late bleeding	0
Patients having bleeding hemorrhoids	23
Local therapy (banding, hemorrhoidectomy)	9
Late bleeding	0
Subsequent portacaval decompression for esophageal varices	6
Late bleeding	0

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