Portal Thrombosis in Cirrhotics

A Retrospective Analysis

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The development of thrombotic obstruction in the portal bed of cirrhotic patients presents special problems in diagnosis and treatment. In the cirrhotic population treated for portal hypertension at our Surgical Department during the period 1967–1983 (512 patients), the incidence of thrombosis in the portal bed was 16.6% (85 patients). Bleeding was the main presenting symptom (70/85), with a mean of four episodes prior to treatment. Careful angiographic studies and intraoperative evaluation are fundamental steps to determine the exact anatomical involvement, the presence of recanalized veins or fresh occluding clots, and the applicability of shunt procedures. A massive portosplenomesenteric involvement often associated with poor surgical possibilities was found in 19 patients (22.3%). The presence of partially recanalized veins and fresh occluding clot suitable for disobliterative techniques prior to shunt was found in 16 patients, and out of 73 operated patients a total of 55 shunt procedures could be performed. Fifty-three patients who bled from varices could be followed up to 5 years: 39 underwent a shunt procedure with a 51.2% 5-year survival rate, while only one of 14 nonshunted or nonoperated survived up to 3 years, and a lethal bleeding was the cause of death in all but one. Disobliterative techniques (Fogarty thrombectomy and endovenectomy of intimal fibrotic thickenings) prior to shunting provided a good long-term patency rate with a 50% protection from lethal bleeding recurrences. Nonshunt procedures and the extensive involvement of the portal bed are associated with bad prognosis. Also, endoscopic sclerotherapy, attempted in patients with massive thrombosis, could not prevent recurrences and death from bleeding. Despite a 30% failure rate in our study, shunting surgery should be considered the only therapeutical possibility of preventing further thrombotic recurrences and consequent life threatening bleeding episodes.

THROMBOSIS in the portal venous system is the main underlying lesion of extrahepatic portal hypertension. Differing from the childhood condition, in which it represents the frequent evolution of portal vein injury or neonatal omphalitis[1,2] and leads to portal cavernomatous transformation, in adults it is quoted as a rare cause of bleeding gastroesophageal varices.[3–5] The etiology of portal vein thrombosis often remains obscure in any age groups,[6] but the association with liver cirrhosis is well known; nevertheless, it does not seem fully investigated. Patients affected from portal hypertension because of liver cirrhosis should be considered prone to the development of thrombotic complication due to the low flow state in the portal bed and to the presence of periportal lymphangitis and fibrosis. Lack of information from large series, necessity of a careful angiographic evaluation, and misleading interpretations when partially recanalized veins or diverting portal flows are present make it difficult to determine a reliable incidence rate in cirrhotics. Moreover, this disorder, particularly when the portal bed is widely involved, provides difficult technical problems in the management of bleeding varices. In the present study we describe our experience with a large group of patients who developed portal thrombosis during the evolution of liver cirrhosis.

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Materials and Methods

From 1967 to 1983, 547 patients affected from portal hypertension were treated at our Department of Surgery. A thrombotic obstruction of the portal vein and/or its radicles was found in 85 of 512 cirrhotic patients during the preoperative assessment or at the operating table, accounting for a 16.6% rate. Sixty-five were men and 20 were women, ranging in age from 19 to 70 years (mean: 47.7 years). The main presenting symptom was bleeding from ruptured gastroesophageal varices (70/85), with a mean of four episodes prior to our observation (range: 1–10). Gastroesophageal varices were highly suspected as the cause of bleeding by GI roentgenograms and successful

TABLE 1. *Presenting Symptoms in 85 Cirrhotic Patients with Portal Bed Thrombosis*

	Number of Patients	%
Bleeding esophageal varices	70	82.3
Splenomegaly and/or hepatomegaly	5	5.8
Ascites	4	4.7
Others	6	7.2

TABLE 2. *Etiology of Cirrhosis and Child Classification in 85 Patients*

Etiology of Cirrhosis	Number of Patients	Child Classification		
		A	B	C
Alcoholic	30	—	26	4
Posthepatic	28	3	22	3
Idiopathic	27	5	18	4

tamponade by Sengstaken tube in the first years of the present study or directly confirmed by endoscopy thereafter. In Table 1 the causes that led to hospitalization are listed. Pathology of cirrhosis and Child classification are presented in Table 2. Angiographic evaluation included splenoportography, ileoportography, and celiacomesenteric arteriography.

On the basis of preoperative angiographic studies and intraoperative findings, the patients were retrospectively divided into four groups according to whether the portal vein alone was involved by thrombotic obstruction (Fig. 1), the portal vein with both splenic and mesenteric radicles (Fig. 2), or the portal vein with one radicle (Fig. 3), or one radicle alone (Fig. 4) (see Table 3 and Fig. 5). In 63 cases, varying degrees of recanalization have been observed, while in the remaining 22 an occluding clot was present. In seven patients, the occluding clot was recent. In the other 15, the portal vein was a thick fibrotic tendon with numerous collateralizing vessels.

Surgical Management

Bleeding from ruptured gastroesophageal varices was the indication for operation in 61 patients, ascites in two, and hypersplenism in 10, for a total of 73 operated patients. In 12 patients, surgery was not performed either because of patient's reluctance, poor surgical risk, or technical considerations. Portosystemic shunt could be performed in 29 patients. In 14 of them, removal of fresh thrombi or intimal fibrotic thickenings was accomplished before shunting. In 26 patients, the anatomy and pathology of the thrombotic obstruction prevented the use of the portal vein, but a shunt could be performed at the splenic (after thrombectomy or endovenectomy in two) or mesenteric level. In the latter case, a prosthesis interposition technique has been adopted (Dacron® 12 mm).

Splenectomy was performed early in our experience in 10 patients, portoazygos disconnection in three patients, while another kind of nonshunt procedure was performed in five further cases (see Table 4). In four of five disconnected patients, thrombotic obstruction was radicular (3 patients) and portoradicular (1 patient).

Results

The overall incidence of portal thrombosis in our cirrhotic patients was 16.6%. A high rate of widespread involvement of the portal vein and its radicles was found (45.8%), and the portal vein was unavailable for shunting in 62.7% of cases.

Our data do not permit correlations between cirrhosis that is postalcoholic or posthepatitic in nature and thrombosis development, but nine of 12 patients with thrombosis of the splenic vein alone and a free portal trunk were alcoholics, suggesting an associated pathogenetic role for chronic pancreas inflammation.

Liver carcinoma was found in two patients at autopsy, while in another 53 patients there was no clinical or autopsy evidence of neoplastic liver changes. Fresh occluding thrombi (7 patients) and partial recanalization of previ-

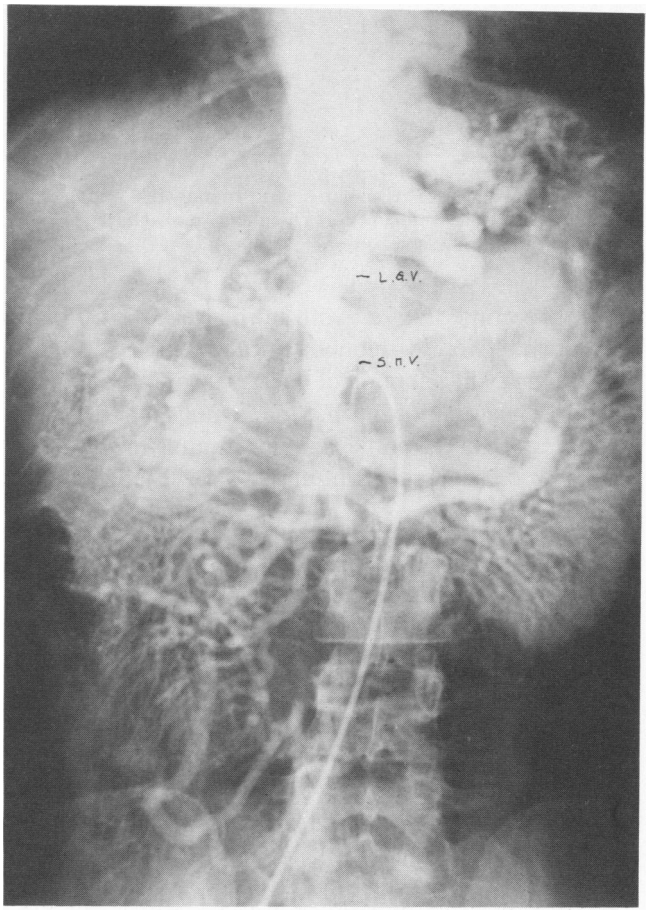

FIG. 1. Venous phase of superior mesenteric arteriography: thrombotic obstruction of portal vein and backflow into left gastric vein.

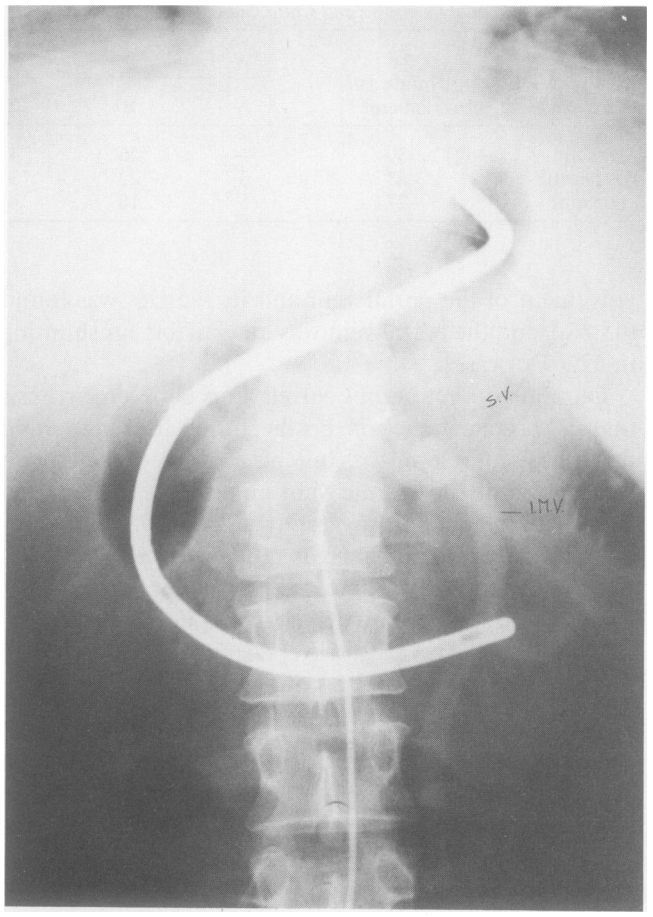

FIG. 2. Venous phase of celiacomesenteric arteriography: nonfilling of portal, distal splenic, and superior mesenteric veins. Diversion of the flow in the inferior mesenteric vein.

ously thrombosed vessels with intimal fibrotic remnants (63 patients) were a frequent intraoperative finding. In this situation, the adoption of Fogarty thrombectomy and/or endovenectomy of intimal thickenings made possible the utilization of the portal vein for shunting in 14 patients otherwise considered unshuntable on the angiographic basis.

The operative mortality was 8.2%. In Fig. 6 and Table 5, the clinical evolution of 53 patients up to 5 years from a group of 70 who bled from ruptured varices is represented. Thirty-nine patients underwent a shunt procedure, while 14 could not be shunted or were not operated at all. Only one patient in the second group survived up to 3 years; in all but one, the cause of death was bleeding recurrences.

In the shunted group, a survival rate of 51.2% had been observed at 5 years, but 30.7% of them died because of bleeding recurrences. Satisfying figures of survival should be noted in those patients in which a previous thrombectomy and/or endovenectomy made available the portal vein for shunting: five of 10 were alive at 5 years with a patent anastomosis and no further bleedings (see Table 5). The group of patients with extensive thrombosis involving the portal vein and both radicles provided poor results: 13 of 19 could in no way be shunted and a 69.3% mortality rate has been observed in the first year. But even in this setting the construction of a mesentericocaval shunt allowed a long-term survival in two patients. Shunted alcoholic patients in this study did not show a different pattern of survival if compared to the nonalcoholic patients. Five of 11 were alive at 5 years (45.4%) with a 27.1% rate of death from bleeding but a higher (18.1%) rate of death due to hepatic failure (7.1% rate in the nonalcoholic group). However, a segmental localization of the thrombotic obstruction limited to one radicle and a free portal trunk, the most favorable clinical setting, was more frequently represented in the alcoholic group.

Encephalopathy or behavioral changes were not objectively investigated other than on clinical grounds. However, their incidence does not seem different from that

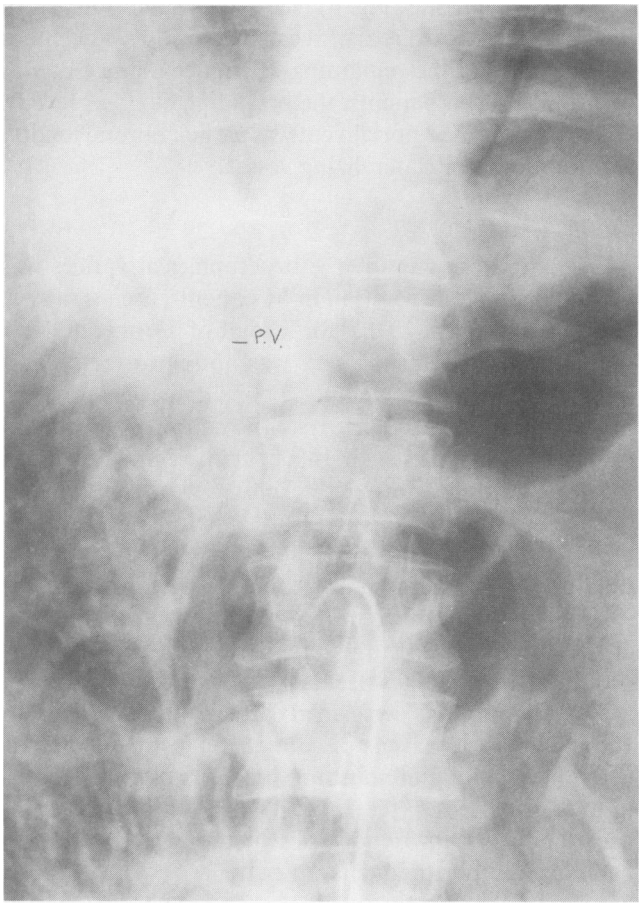

FIG. 3. Venous phase of superior mesenteric arteriography: nonfilling of the superior mesenteric vein and partial recanalization of the portal vein.

observed in our operated cirrhotic population without thrombotic obstruction.[7]

Discussion

Portal thrombosis is generally quoted as a rare cause of adult onset bleeding from gastroesophageal varices; however, it does not seem fully investigated when associated to liver disease. Sherlock stated that thrombosis in the portal vein is a rare complication of cirrhosis and that it is usually due to invasion by primary carcinoma.[8] Galambos in his monograph about cirrhosis does not mention this disorder.[9] Few studies published in the English literature in the last 20 years about extrahepatic portal obstruction described patients with associated liver cirrhosis.[5,10–12] As reported elsewhere, our experience is quite different.[13] Of 547 patients affected by portal hypertension, we faced portal thrombosis and its related problems in 140. In 85 of them, the thrombotic complication occurred in the evolution of an intrahepatic block due to liver cirrhosis, accounting for a 16.6% rate.

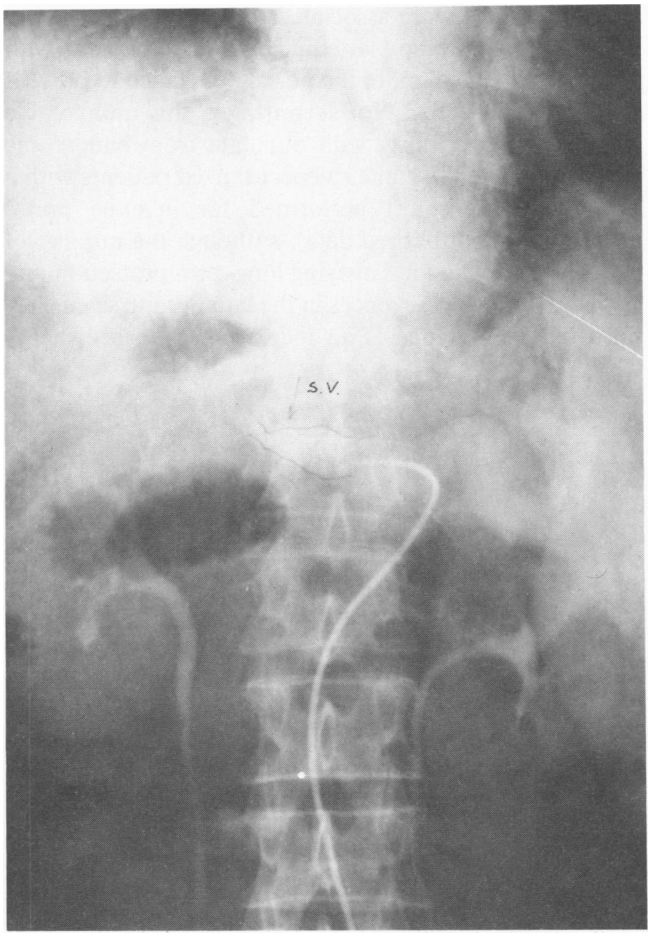

FIG. 4. Venous phase of celiac arteriography: thrombotic obstruction of the splenic vein at the confluence with portal vein.

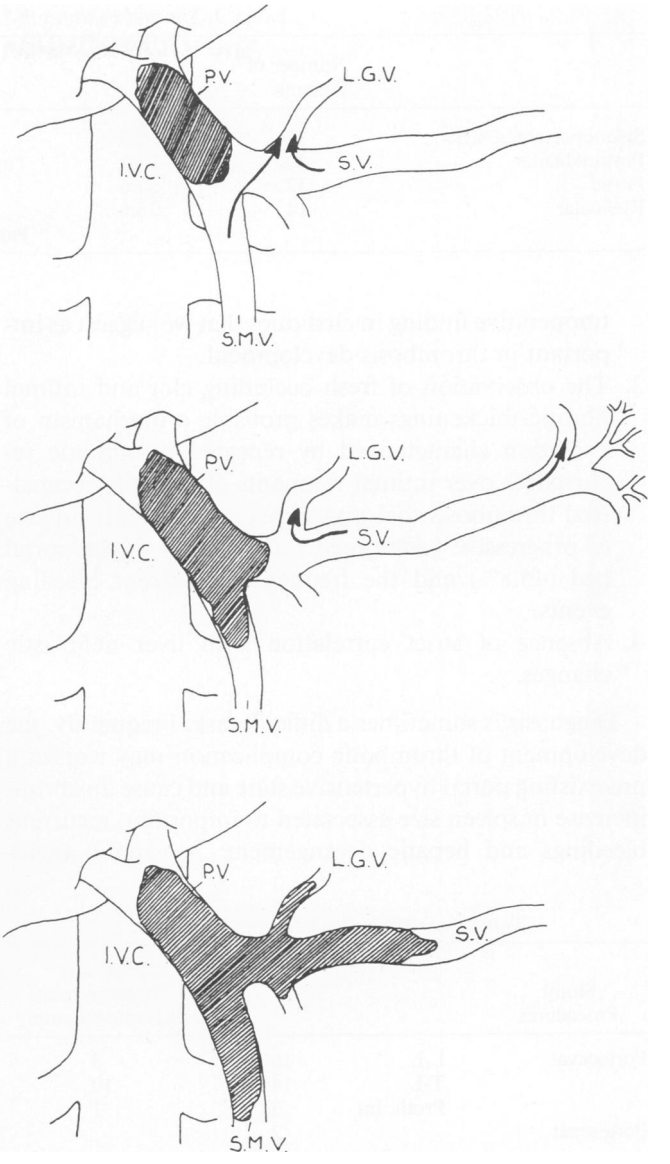

FIG. 5. Schematic illustration of different anatomical involvements of thrombotic obstruction in the portal system.

It is widely accepted that the low flow state in the portal bed is the main predisposing condition to thrombus formation. According to our retrospective analysis, other correlating factors seem possible.

1. The etiology of cirrhosis while posthepatitic or alcoholic in nature does not seem to have an important role, but the involvement of the splenic vein alone was frequently associated with alcoholic cirrhosis (9/12) and with the intraoperative finding of an enlarged sclerotic pancreas, supporting a possible role for chronic pancreas inflammation.
2. Periportal lymphangitis and fibrosis is a frequent in-

TABLE 3. *Site and Features of Thrombotic Obstruction in 85 Patients*

	Number of Patients	%				
Splenoportomesentric	19	22.3				
Portoradicular	20	23.5	Totally occluding thrombosis	22	Old age clot	15
Portal	32	37.6			Fresh clot	7
Radicular	14	16.6				
			Partially recanalized thrombosis	63		

traoperative finding in cirrhotics that we suggest as important in thrombosis development.

3. The observation of fresh occluding clot and intimal fibrotic thickenings makes probable a mechanism of evolution characterized by repeated thrombotic recurrences over intimal remnants of partially recanalized thrombosis that may explain our high incidence of progressive widespread involvement of the portal bed (45.8%) and the frequently recurrent bleeding events.
4. Absence of strict correlation with liver neoplastic changes.

Diagnosis is sometimes a difficult task. Frequently, the development of thrombotic complication may worsen a pre-existing portal hypertensive state and cause an abrupt increase in spleen size associated to important, recurrent bleedings and hepatic derangement. A careful angiographic evaluation is mandatory, but misleading interpretations are possible when a partially recanalized vein is present and the intimal thickenings remain unnoticed or when important diversion of portal flow into collateral circulation causes the apparent nonfilling of the portal vein. Therefore, intraoperative evaluation represents a fundamental step in order to know the exact extension of the thrombotic obstruction, to detect fresh occluding clot or intimal fibrotic remnants suitable of disobliterative techniques, and to search carefully the optimal vein for shunting. According to our data, surgery should be considered the treatment of choice because medical therapy and tamponade seem associated to bad prognosis. Also, injection sclerotherapy, attempted in three patients with extensive involvement of the portal bed, failed to prevent death from bleeding. Not surprisingly, this unfavorable result compares poorly with our data from endoscopic sclerosis adopted in some splenectomized patients with a previous failed shunt performed for juvenile portal thrombosis (unpublished data). Although the number of patients is limited, the satisfying long-term protection rate against bleeding recurrences in the latter group seems justified by the presence of an increasing pattern of hepa-

TABLE 4. *Surgical Procedures in 73 Patients*

Shunt Procedures			Previous Thrombectomy or Endovenectomy
Portocaval	L-L	10	3
	T-L	14	10
	Proth. Int.	3	1
Portorenal		2	
		29	
Splenorenal	Central	7	
	Distal	2	
	L-L	4	1
	L-L + Prot.	2	1
	L-T	1	
Splenocaval		1	
		17	
Mesentericocaval with Prothesis		9	
		9	
Total		55	16
Other procedures			
Splenectomy		10	
Portalazjgos disconnection		3	
Crile		1	
Left gastric vein ligature and splenectomy		2	
Peritoneovenous shunt		1	
Hepatic decortication		1	
Total		18	

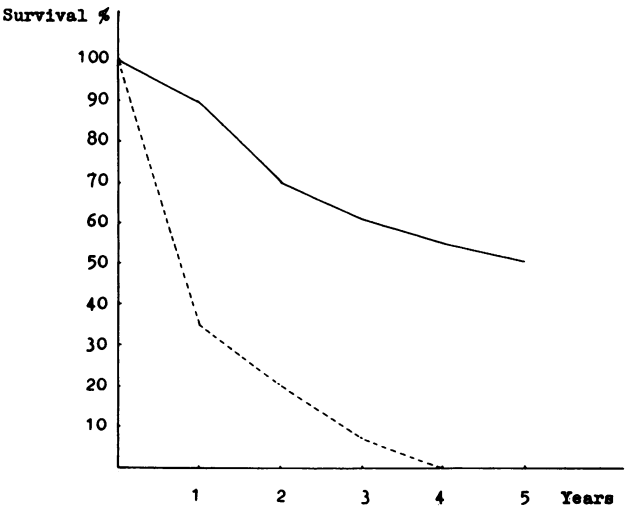

FIG. 6. Five years' survival rate of 53 patients who bled from ruptured varices. —— Shunted patients (39 patients); ----- Nonshunted or nonoperated patients (14 patients).

TABLE 5. *Five Years' Clinical Evolution of 53 Bleeding Patients*

Treatment	Number of Patients	Survival after 3 Years		Survival after 5 Years		Death from Bleeding		Death from Hepatic Failure		Death from Other Causes
		Number of Patients	%	Number of Patients	%					
Portosystemic shunt	9	6	66.6	5	55.5	2	22.2	2	22.2	—
Portosystemic shunt + thromb. or endovenectomy	10	6	60.0	5	50.0	3	30.0	1	10.0	1
Splenorenal shunt	13	8	61.5	6	46.1	5	38.4	1	7.7	1
Mesentericocaval shunt	7	4	57.1	4	57.1	2	28.5	—	—	1
Total	39	24	61.5	20	51.2	12	30.7	4	10.2	3
Nonshunt procedure or nonoperated	14	1	—	—	—	13	92.8	—	—	1

topetal collateral vessels. On the contrary, this really favorable possibility is greatly compromised when an intrahepatic block is associated to portal thrombosis. Despite technical difficulties, every effort should be made to perform a shunting procedure. In this regard, the satisfying figures provided by portocaval shunt performed after Fogarty thrombectomy and endovenectomy, a technique rarely reported previously,[14] should be noted: five of 10 have been protected from bleeding by a patent anastomosis for up to 5 years. The adoption of a mesocaval shunt with prosthesis interposition, even though incorrect in theory, becomes a justified risk if the rationale is to decrease in any way the portal pressure gradient and other shunting procedures are technically unpracticable. When prosthetic grafts are employed, we use long-term anticoagulation therapy. Patients with portal thrombosis associated with liver cirrhosis should be considered for unique problems in surgical treatment and a particular subset of portal hypertension patients with a poor prognosis.

In the present series the anatomopathological features of the thrombotic obstruction provide a 21.2% rate of unshuntable patients at high risk of lethal bleeding recurrence within a short period of time, even if a disconnective procedure has been performed. But if an increase of flow velocity in the portal bed may be created by a patent anastomosis, prevention of further thrombotic recurrences and consequent bleeding events is possible. Despite our 5-year 30.7% rate of recurrent lethal bleeding, shunt procedures should be considered the only valuable therapeutical method in cirrhotic patients who developed a thrombotic obstruction in the portal bed.

References

1. Alvarez F, Bernard O, Brunelle F, et al. Portal obstruction in children. I. Clinical investigation and hemorrhagic risk. J Pediatr 1983; 103:696–702.
2. Fonkalsrud EW, Myers NA, Robinson MJ. Management of extrahepatic portal hypertension in children. Am Surg 1974; 180:487–491.
3. Maddrey WC, Sen Gupta KP, Basu Mallik KC, Iber FL. Extrahepatic obstruction of the portal venous system. Surg Gynecol Obstet 1968; 989–998.
4. Turcotte JG, Child CG. Idiopathic extrahepatic portal hypertension in adults. Am J Surg 1972; 123:35–42.
5. Ottinger LW, Austen WG. A study of 136 patients with mesenteric infarction. Surg Gynecol Obstet 1967; 124:251–261.
6. Price JB. Extrahepatic portal hypertension. *In* Rutherford RB, ed. Vascular Surgery. Philadelphia: WB Saunders, 1984; 1070–1077.
7. Marni A, Trojsi C, Belli L. Distal splenorenal shunt. Am J Surg 1981; 142:372–376.
8. Sherlock S. Diseases of the Liver and Biliary System. Oxford: Blackwell Scientific Publications, 1981.
9. Galambos JT. Cirrhosis. Philadelphia: WB Saunders, 1979.
10. Yale CE, Crummy AB. Splenic vein thrombosis and bleeding esophageal varices. JAMA 1971; 217:317–320.
11. Grauer SE, Schwartz SI. Extrahepatic portal hypertension. Ann Surg 1979; 189:566–574.
12. Sack G, Aldrete JS. Primary mesenteric venous thrombosis. Surg Gynecol Obstet 1982; 154:205–208.
13. Belli L, Puttini M, Marni A. Extrahepatic portal obstruction. J Cardiovasc Surg 1980; 21:439–448.
14. Bergentz SE, Ericsson B, Hedner U, Leandoer L. Thrombosis in the superior mesenteric and portal veins: report of a case treated with thrombectomy. Surgery 1974; 76:286–290.