Significance of Splenomegaly in Patients with Hepatic Cirrhosis and Bleeding Esophageal Varices

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IN patients with hepatic cirrhosis splenomegaly is usually considered to be the result of portal stasis, a concept which underlies the convenient clinical term "congestive splenomegaly."[9, 22] Viewed in this light there is little reason to attach special significance to the finding of splenomegaly in patients who have bleeding esophageal varices.

Information from experiments in animals suggests, however, that this simple mechanical explanation for splenomegaly may be inadequate.[13, 21] Interruption of splenic venous drainage for example is not followed by sustained splenomegaly.[24] In addition, chemically induced injury to the liver results in alterations in size and function of the spleen even when it is transplanted out of the portal system.[2] Based on an assumption that the origin of splenomegaly in cirrhosis is still obscure, an attempt was made to assess the incidence, relationship to portal pressure, significance and course of splenomegaly in patients undergoing portacaval shunt for bleeding esophageal varices. A review of the records of 16 patients including eight who had splenomegaly forms the basis for this report.

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Clinical Data

From January 1964 through December 1968, 16 patients with hepatic cirrhosis underwent portacaval shunt for bleeding esophageal varices on the surgical service of Bellevue Hospital.

Group I: Without Splenomegaly

Eight patients without splenomegaly, three women and five men ranging in age from 40 to 53 years (mean 46) entered the hospital bleeding massively from varices and required amounts of blood which varied from 3½ to ten liters. Except for one patient with postnecrotic cirrhosis, all had histories of prolonged alcoholism. Chemical tests of liver function as well as biopsy sections disclosed evidence of long-standing cirrhosis in each patient.

Albumin levels ranged from 2.8 to 4 Gm. /100 ml. and bilirubin from 1.7 to 9.7 mg. /100 ml. Large amounts of ascitic fluid were present in three and a small amount appeared transiently in a fourth. Variceal bleeding stopped spontaneously or with the help of balloon tamponade in all except one patient in whom hemorrhage stopped immediately after thoracic duct cannulation.

Each patient underwent an end-to-side portacaval shunt after cessation of hemorrhage. At operation portal pressures ranged

TABLE 1. *Data from Patients without Splenomegaly*

Pt.	Age	Sex	Transfusion (liters)	T.D.	Alb. (Gm./100 ml.)	Bil. (mg./100 ml.)	Ascites	Portal Pressure (cm. H_2O) Pre	Post	Course
A. L.	51	F	3½	0	2.9	8	4+	47	16	Coma followed by cardiac arrest—died on 4th post-op. day
S. H.	40	M	6	0	3.2	9.7	4+	45	9	Atelectasis plus pneumonia—died on 28th post-op. day
J. M.	42	M	3½	0	2.9	3.2	0			Cardiac arrest—died on 3rd post-op. day
M. B.	41	F	5	0	2.8	2.5	4+	45	20	Sepsis plus coma—died on 21st post-op. day
M. R.	43	M	10	v	4	5.7	0	50		Hepatic failure—died on 5th post-op. day
J. C.	47	M	7	0	3.3	10	0	39		Congestive heart failure—died on 5th post-op. day
E. V.	53	M	3½	0	3.2	4.7	2+			Hemorrhage from liver biopsy—died on 12th post-op. day
H. M.	52	F	3½	0	3.1	3.1	0	47	31	Alive at 53 months-severe encephalopathy

Note: T.D. = Thoracic duct cannulation to control hemorrhage.

from 30 to 50 cm. of water with a mean of 45. All survived operation but with the exception of one patient all died in the early postoperative period. The causes of death are listed in Table 1. The sole survivor was readmitted to the hospital 53 months postoperatively with severe post-shunt encephalopathy. Pertinent data from this group of patients are presented in Table 1.

Group II: Splenomegaly

Eight patients, seven men and one woman ranging in age from 14 to 63 years (mean 45) had enlarged spleens. With the exception of one patient whose hematocrit was normal on admission, each one entered the hospital bleeding massively and required amounts of blood which varied from 2½ to seven liters. Apart from the youngest patient who had histologic evidence of postnecrotic cirrhosis with a history of jaundice six years earlier, all of these patients as those in Group 1, had long histories of alcoholism with markedly deranged chemical tests of liver function and liver biopsies which disclosed evidence of longstanding cirrhosis.

Albumin levels ranged from 2.9 to 4.4 Gm./100 ml. and bilirubin from 1.7 to 4.6 mg./100 ml. None of these patients had ascites.

Based on platelet counts which ranged from ten to 129,000 plus varying degrees of pancytopenia five of the eight patients were considered to have hypersplenism in addition to splenomegaly. Pre-operative x-ray visualization of the portal and splenic venous system in three such patients by splenoportography or celiac angiography disclosed patent splenic and portal veins.

Variceal hemorrhage stopped spontaneously or with the help of a balloon tamponade in every patient except two and in these hemorrhage ceased immediately after thoracic duct cannulation. Following cessa-

tion of bleeding each patient underwent an end-to-side portacaval shunt. At operation portal pressures ranged from 19 to 49 cm. of water with a mean of 37. Operative occlusion of the portal vein disclosed findings consistent with hepatopedal flow in five of six such studies.

The postoperative courses of these eight patients were marked by the absence of any life threatening complications and all left the hospital in good condition. To date all patients have been free of ascites, encephalopathy or recurrent variceal hemorrhage. Data obtained at the time of follow-up visits indicate that splenomegaly has persisted in seven patients from two to 28 months while in one the spleen receded under the costal margin 9 months postoperatively. Although platelet counts have not returned to normal levels in any of the five patients with hypersplenism, splenectomy has not been required. Postoperative studies with chrome 51 labeled red cells in three patients with hypersplenism showed abnormal retention in the spleen in only one. X-ray visualization of the portal system by splenoportography in one and superior mesenteric arteriography in another confirmed patency of the portacaval shunt.

Data from these eight patients are presented in Table 2.

Discussion

The incidence of palpable splenomegaly in patients with hepatic cirrhosis varies in published reports from 32 to 42%.[5, 17] Among 50 patients with cirrhosis who died with bleeding esophageal varices, the spleen weighed more than 350 Gm. (suggesting that it was palpable) in 28.[10] Demonstration of a palpable spleen in eight of the 16 patients reported here therefore conforms with the experience of others.

With one exception, nine patients who survived portacaval shunts were characterized by the presence of splenomegaly and in these, levels of albumin tended to be higher and bilirubin lower than in those without splenomegaly. There was no correlation between splenomegaly and the types of hepatic disease, ages of the patients or severity of variceal hemorrhages. Although these findings indicate that splenomegaly may help distinguish better risk patients, the available data do not clarify the basis for this relationship. On the other hand, the fact that splenic artery inflow is abnormally increased in patients with cirrhosis and splenomegaly is confirmed by the results of numerous recent angiographic and isotopic studies.[14, 15, 23] Such a derangement would favor the development of hemorrhage from varices even in the absence of the impaired transhepatic portal flow associated with terminal cirrhosis.

The partial success of splenectomy alone in patients with bleeding varices also bears on the questions of abnormally increased arterial inflow.[16] If, as is generally assumed, 40% of portal vein flow normally derives from the splenic artery,[20] a much greater proportion of portal flow would derive from this source in the presence of splenomegaly. In such patients therefore, ligation of the splenic artery with preservation of the spleen and its surrounding collateral circulation might be a rational alternative to portacaval shunt.

Persistence of splenomegaly or hypersplenism or both in the patients reported here plus an additional patient who underwent portacaval shunt but who did not bleed from varices, conforms to the findings described earlier by others.[11, 12, 18, 21] Although splenectomy is usually not required to deal with alterations in formed blood elements which persist after portacaval shunt, it is clear that the shunt cannot be regarded as a "physiological splenectomy."[4] Any explanation that long-standing fibrotic changes in the spleen pre-

TABLE 2. *Data from Patients with Splenomegaly*

Pt.	Age	Sex	Transfusions (liters)	T.D.	Alb. (Gm./100 ml.)	Bil. (mg./100 ml.)	Ascites	Portal Pressure (cm. H_2O) Pre.	Occl.	Post	Splenomegaly Pre.	Post	Platelets $\times 10^3$ Pre.	Post	Course	Follow-up in months
Y. E.	14	M	7	v	3.3	4.6	0	42	30/49	25	3+	0	10	70	Alive and well	31
C. F.	40	M	3½	0	2.9	1.6	0	40		29	2+	3+	40	69	Alive and well	2
T. H.	63	M	2½	0	3.1	4.3	0				3+	1+	40		Alive and well	19
G. C.	46	M	6½	0	3.5	4.1	0	39	37/25		2+	2+	50	99	Alive and well	10
E. R.	48	M	2	0	3.7	0.8	0	49	38/48	10	2+	2+	129	129	Alive and well	18
G.V.	50	M	0	0	4.4	2	0	31	/44	16	1+	1+	n.l.	n.l.	Alive and well	24
B. R.	48	F	2½	0	3.5	3.5	0	41	20/	23	1+	1+	n.l.	n.l.	Alive and well	28
E. P.	46	M	4½	v	4.1	0.7	0	19	15/29	12	1+	1+	n.l.	n.l.	Alive and well	26

Note: T.D. = Thoracic duct cannulation to control hemorrhage. Post-op. splenomegaly and platelets refers to time of last follow-up visit.

vent its return to normal size after portacaval shunt is countered by the fact that reduction in spleen size occurs within minutes of interrupting flow in the splenic artery even in patients with splenomegaly of many years' duration.

Although all the patients reported here entered the hospital with bleeding varices, deranged tests of liver function, longstanding disease and elevated levels of portal pressure, splenomegaly was absent in 50%. Portal pressures tended to be somewhat higher in fact in patients without splenomegaly. Considered together with the fact that alterations in size and function of the spleen persisted after successful portacaval shunt, these findings provide little support for the concept of portal stasis as the dominant factor in the pathogenesis of splenomegaly and hypersplenism.

Although the mechanism responsible for splenomegaly is still obscure two alternative views can be considered. First, a reciprocal relationship exists between reticuloendothelial activity in the liver and in the spleen.[7] Portacaval shunt, for example by diverting portal inflow, is followed by marked decrease in hepatic and increase in splenic RE-activity.[1, 6] Reduction in hepatic RE-activity probably secondary to impaired transhepatic portal blood flow also occurs in patients with cirrhosis and results in a corresponding increase in splenic RE-activity.[8] It seems reasonable to speculate that this sustained increase in splenic function would lead in turn to splenomegaly and augment arterial flow.

A second possible mechanism is based on primary alterations in the arterial circulation of the spleen. A congenital or traumatic arteriovenous fistula of the splenic vessels, in the absence of hepatic disease, results in splenomegaly and hypersplenism.[3, 19] AV shunting in the spleen may also develop in patients with cirrhosis and result in a similar derangement.[24]

Application of the term "congestive splenomegaly" to patients with cirrhosis implies the presence of information which is, in fact, lacking. Whether the abnormal load on the spleen is functional or arterial, it is almost certainly not venous alone. When examined in the light of current knowledge, explanations of splenomegaly based on "the variable amount of portal hypertension" [22] no longer seem adequate.

Summary

The significance of splenomegaly with hepatic cirrhosis and bleeding esophageal varices was considered in patients upon whom portacaval shunt operations were performed. Eight patients without splenomegaly all did poorly after portacaval shunts. Seven died early in postoperative periods. Eight comparable patients who had splenomegaly all survived and improved.

It is suggested that the term "congestive splenomegaly" is an over simplification that the spleen enlarges in the presence of a cirrhotic liver for reasons other than resistance to venous outflow; that either increased splenic arterial flow or increase in reticuloendothelial activity in the spleen are implicated in enlargement of the organ.

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