Significance of an Enlarged Splenic Artery in Patients with Bleeding Varices

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THE results of recent angiographic and isotopic studies in patients with splenomegaly support the view that an abnormally large arterial inflow into the spleen may be an important component of portal circulatory congestion.[4, 11, 15, 16] When cirrhosis, a blood dyscrasia or extrahepatic portal vein obstruction is the underlying disorder, increase in diameter of the splenic artery often accompanies splenomegaly and the abnormal diameter of the splenic artery then correlates closely with increased total spleen blood flow.[1, 2, 3] The association between size of the spleen and diameter of the splenic artery is not absolute, however, as this latter may be within normal limits even in patients with massive splenomegaly; on the other hand, an abnormally large splenic artery is rarely observed in the absence of splenomegaly.

The high incidence of continued or recurrent variceal hemorrhage following splenectomy or splenic artery ligation has tended to obscure the fact that these maneuvers have been successful in about 30% of all patients with bleeding varices.[8, 14] Why these relatively simple alternatives to portacaval shunt have been effective in some but not in all patients is unknown but it is possible that abnormally high inflow into the portal system via the splenic artery could characterize the group of patients who respond best. Based on these considerations an attempt was made to assess the prognostic significance of an enlarged splenic artery in patients undergoing splenic artery ligation and/or splenectomy for bleeding esophageal varices. A review of the records of 12 consecutive patients including seven with an enlarged splenic artery forms the basis for this report.

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Clinical Data

From January 1967 to April 1971 12 adult patients underwent splenic artery ligation and or splenectomy for bleeding esophageal varices on the surgical services of either Bellevue or University Hospital.

Group 1. Enlarged Splenic Artery

Seven patients, five men and two women ranging in age from 48 to 69 years entered the hospital bleeding massively from varices and were found to have splenomegaly as well as an enlarged splenic artery.

A history of prolonged alcoholism was obtained in four patients while three others presented with various underlying disorders as follows: myeloid metaplasia, biliary cirrhosis secondary to carcinomatous obstruction of the common bile duct and thrombotic occlusion of the extrahepatic portal vein. In each of the four patients with histories of alcoholism chemical tests of liver function as well as biopsy sections disclosed evidence of longstanding hepatic cirrhosis. Scans of the liver and spleen following the administration of TE 99 sulfur colloid showed decreased hepatic and increased splenic uptake in three of the four; in one of these three patients a significant reduction in circulating formed blood elements was also found (hypersplenism). Al-

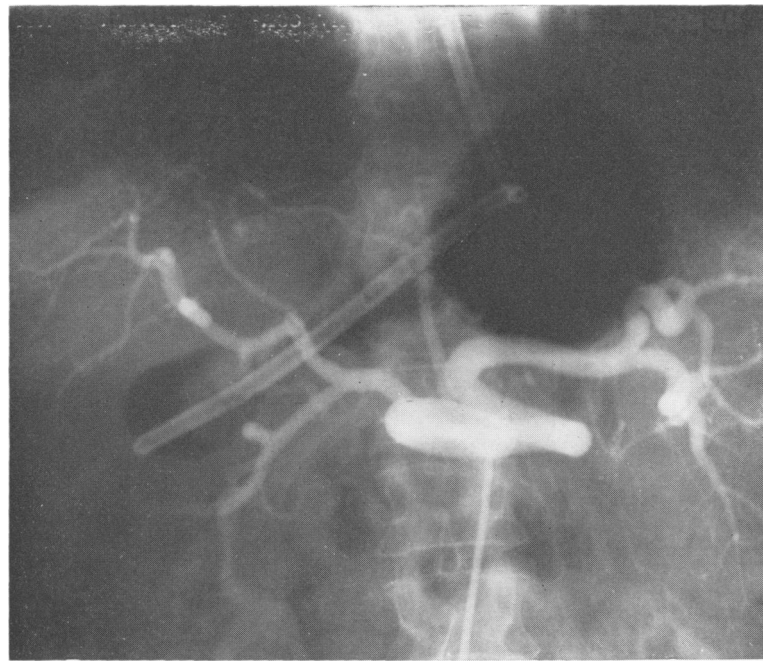

FIG. 1A. Celiac angiograms, one day before operation, of patient F. G. who underwent ligation of the splenic artery.

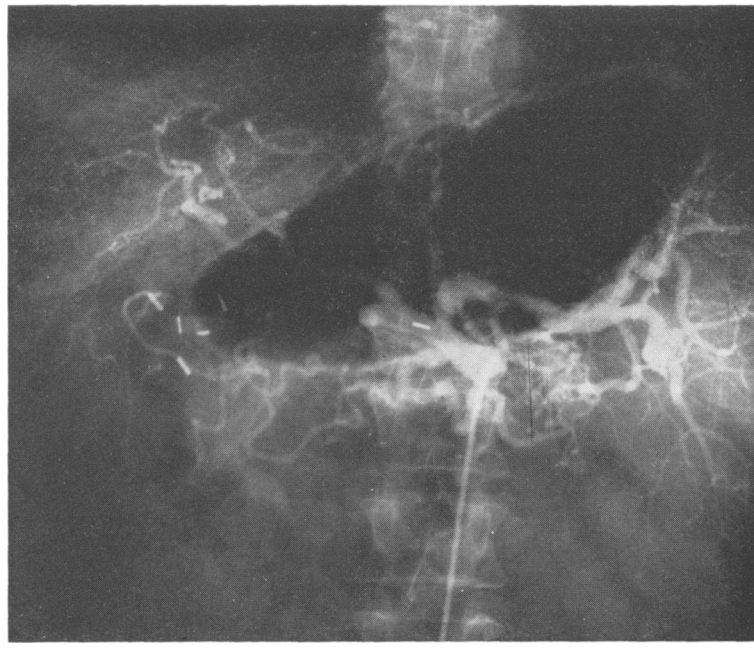

FIG. 1B. One year later. The point of surgical ligation can be seen just to the right of the radiopaque clip overlying the spinal column. Contrast material seems to be entering the splenic artery just distal to the point of ligation via two collateral channels from the right gastroepiploic artery.

bumin levels of the seven patients ranged from 2.7 to 4.2 Gm./100 ml. and bilirubin from 0.2 to 10 mg./100 ml. Large amounts of ascitic fluid were present in two patients and smaller amounts in one. Variceal hemorrhage required transfusion of 1½ to 4 liters of blood and then ceased with the help of balloon tamponade or following the administration of pitressin via the superior mesenteric artery in each patient with two exceptions who were operated upon while still bleeding.

In six patients celiac angiography confirmed the presence of an enlarged spleen and with one exception demonstrated that the splenic and portal veins were patent. The one exception occurred in patient E. N. in whom the portal vein was not visualized and who was found at operation to have a thrombus occluding the vein at the hilus of the liver. Maximal diameter of the splenic artery in six pateints undergoing celiac angiography ranged from 9 mm to 1.5 cm. at its widest point and averaged 1.2 cm. (normal 8 mm. or less).[6, 7, 9, 10] Circulation time from the splenic artery to the hilar portal vein ranged from 8 to 10 seconds and averaged 9.1 seconds. In one patient not undergoing preoperative angiography a splenic artery 1 cm. in diameter was found at operation.

At operation four patients underwent splenic artery ligation and three splenectomy. Portal pressures ranged from 14 to 40 cm. of water and averaged 33 cm. Operative occlusion of the splenic artery resulted in visible decrease in size of the spleen in all seven patients and decrease in elevated portal pressure averaging 8 cm. of water in the five patients in whom this was measured.

Postoperative courses of all seven patients were marked by absence of any life threatening complications and all left the hospital in good condition. Recurrent variceal bleeding has not been observed in any of these seven patients. Four patients are alive and have been free of ascites, encephalopathy and recurrent variceal hemorrhage for up to 4 years, the longest period of observation to date. F. D., the patient with myeloid metaplasia underwent suprapubic prostatectomy 1 year after splenectomy and died several weeks later of congestive heart failure. A second patient, E. M., died with widespread metastases 2 years after splenic artery ligation without recurrence of variceal bleeding. L. Z., the only patient with cirrhosis who is not alive died 6 weeks postoperatively in hepatic coma.

After an interval of 1 year celiac angiography was repeated in two of the patients who had previously undergone splenic artery ligation (Fig. 1). In both cases contrast material entered the splenic artery via collaterals from the gastroepiploic and pancreatic arteries just distal to the site of ligation and distal portions of the splenic artery as well as intrasplenic branches were visibly smaller than diameters observed preoperatively. Pertinent data from these seven patients are presented in Table 1.

Group 2. Normal Splenic Artery

Five patients, three men and two women ranging in age from 38 to 64 years entered the hospital bleeding massively from varices. Splenomegaly was present in three and celiac angiography performed preoperatively in two of these disclosed a normal size splenic artery. At operation three other patients also were found to have a splenic artery of normal size.

A history of prolonged alcoholism was obtained in four patients in whom chemical tests of liver function as well as biopsy sections disclosed evidence of longstanding cirrhosis. In addition to cirrhosis one of these four patients was found at operation to have a multicentric hepatoma. The remaining patient presented with extrahepatic biliary obstruction secondary to carcinoma of the head of the pancreas and evidence of longstanding intrahepatic bile stasis. A scan of the liver and spleen following the administration of TE 99 sulfur colloid in the three patients with splenomegaly disclosed decreased hepatic and increased splenic uptake. No evidence of hypersplenism was found in any of these patients. Albumin levels ranged from 2 to 4.1 Gm./100 ml. and bilirubin from 4.6 to 12 Mg./100 ml. Large amounts of ascitic fluid were present in three patients and ascites was absent in the other two. Variceal hemorrhage required transfusion of 2–8 liters of blood and then ceased in three of the five patients with the help of balloon tamponade or

intra-arterial pitressin. The other two patients underwent laparotomy while still bleeding. Portal pressures ranged from 19 to 47 cm. of water with a mean of 31. These pressures did not change significantly following splenectomy or splenic artery ligation. Variceal bleeding either continued or resumed postoperatively in all five patients and with one exception all died in the postoperative period. The exception was a patient in whom postsplenectomy variceal bleeding was controlled by thoracic duct cannulation and who subsequently underwent a thoracic duct to jugular vein shunt. Although this shunt became obstructed after 4 months variceal bleeding did not recur and the patient remained alive and well for 4 years. Pertinent data from these five patients is shown in Table 2.

TABLE 1. *Data from Patients with Enlarged Splenic Artery*

Pt.	Age	Sex	Diagnosis	Trans-fusion (liters)	Alb. (Gm./100 ml.)	Bil. (mg./100 ml.)	As-cites	Spl. Art. (mm.)	Circ. Time Splen. Art. to Hilar Port. V. (sec.)	Portal Pressure (cm. H_2O) Pre.	Portal Pressure (cm. H_2O) Post.	Sple-nectomy	Splenic Artery Lig.	Course	Follow-Up in Months
F. D.	69	M	Myeloid metaplasia	1½	4.1	0.2	0	13	10	33	26–13	✓		Alive and well	12
L. N.	66	M	Cirrhosis	2	4.2	0.2	0	11	8.5	40	34	✓		Alive and well	48
S. O.	65	M	Cirrhosis	4	2.9	2.4	0	9	8	34	26	✓		Alive and well	24
L. N.	55	M	P. V. thrombosis	2	3.5	0.2	2+	11		37	25		✓	Alive and well	22
F. G.	49	M	Cirrhosis	3	4.2	10	3+	15	10		29		✓	Alive and well	24
L. Z.	68	F	Cirrhosis	4	2.7	4	2+	9		39	34		✓	Died in hepatic coma	1½
E. M.	48	F	Ca. pancreas	3	3.4	0.5	0	10		14	14		✓	Died of metastatic cancer	26

Discussion

In this small series of 12 patients with bleeding varices including ten with splenomegaly, splenic artery ligation and or the splenectomy was effective in controlling variceal hemorrhage in only the seven patients in whom splenomegaly was combined with an enlarged splenic artery. This suggests that preoperative celiac angiography in patients with bleeding varices and splenomegaly may enable the surgeon to select for splenectomy or splenic artery ligation those most likely to benefit from these procedures. As a dilated splenic artery signifies that high inflow into the spleen and portal system is a significant component of the hemodynamic derangement, ligation of the splenic artery and/or splenectomy are rational alternatives to portacaval shunt in such patients.

Elevated portal pressures fell following occlusion of the enlarged splenic artery in all five patients in whom the pressure was measured before and after this maneuver. In contrast, in the three patients without an enlarged splenic artery levels of portal pressure remained unchanged following arterial ligation. These observations support

TABLE 2. *Data from Patients without an Enlarged Splenic Artery*

Pt.	Age	Sex	Diagnosis	Trans-fusion (liters)	Alb. (Gm./100 ml.)	Bil. (mg./100 ml.)	As-cites	Spl. Art. (mm.)	Portal Pressure (Gm. H_2O) Pre.	Post.	Sple-nectomy	Splenic Art. Liga-tion	Course
J. R.	55	F	Cirrhosis	2	3.5	7.2	4+	L 8	47	47	√		Rebled from varices 2 weeks postop. and died
K. K.	64	F	Cirrhosis	6	2.5	4.6	4+	L 8	34	36	√		Continued to bleed from varices and died
R. S.	59	M	Ca. pancreas	3	2.4	5.2	2+	L 8	19			√	Continued to bleed from varices and died
R. R.	57	M	Cirrhosis-hepatoma	6	2.0	6.0	4+	6	37	35	√		Continued to bleed from varices and died
T. L.	38	M	Cirrhosis	8	4.1	12	0	L 8	20 – 25		√		Post splenectomy hemorrhage from varices stopped by thoracic duct cannulation Alive and well 4 years

the view that in patients with splenomegaly and an enlarged splenic artery increased arterial inflow into the portal system may be the dominant alteration in the pathogenesis of portal hypertension. In four patients with an enlarged splenic artery and marked portal hypertension, the fact that circulation time from splenic artery to hilar portal vein overaged only 9.1 seconds, a value well within normal limits conforms to this view. In such patients an explanation of portal hypertension based predominantly on impairment to portal flow no longer seems adequate.

Johnson *et al.*[5] compared the average size of the splenic artery in nine patients with cirrhosis and bleeding varices with that of 30 control patients and found a larger average size in the former group. Whether or not any of these nine patients had splenomegaly was not stated. The average diameter found in the cirrhotic patients, 8.6 mm. is, however, one that would be considered almost within normal limits (8 mm.) by other investigators. This discrepancy may be the reason these authors found no correlation between the angiographic findings and the results of splenectomy.

When applied to all patients with splenomegaly without regard to the size of the splenic artery, splenic artery ligation and/or splenectomy have been followed by recurrent or continued bleeding in at least half the reported cases.[8, 12] Moreover the results are even worse in children but available data do not clarify the reason for this. Shumacker [13] for example reported that of ten children with splenomegaly and bleeding varices treated by splenectomy only one survived, a patient in whom splenectomy had been deferred until age 32. It seems reasonable to speculate that when results of angiographic studies in children with bleeding varices and splenomegaly become available the vascular alterations will prove to be somewhat different from that seen in the adult.

Although elevated portal pressure declined in all five patients in whom this was measured, an immediate return to normal levels was never observed. In the patient with myeloid metaplasia, however, wedged hepatic vein pressure did return to normal after an interval of 2 weeks, a decrease of 12 cm. from the level obtained immediately after splenectomy. High inflow through either the superior mesenteric or hepatic arteries may at times be a factor which limits the extent which portal pressure falls following interruption of flow in a large splenic artery. In this particular patient the superior mesenteric artery was 1.5 cm. in diameter or 0.2 cm. wider than the splenic artery. The fact that portal pressure fell and bleeding varices did not recur in this as well as the other six patients suggests that abnormal inflow via the splenic artery may be the pivotal factor even when flow through the mesenteric and/or hepatic arteries is increased.

Angiographic evidence of resumption of flow in a previously ligated splenic artery observed in two patients described here, has not been reported previously. While this observation could support the selection of splenectomy rather than splenic artery ligation alone, there may be advantages in preserving the spleen as an important site of spontaneous portal systemic shunts. Moreover more distal ligation of the splenic artery, closer to the hilus of the spleen, might decrease the likelihood of resumption of flow through collaterals.

Summary

The significance of an enlarged splenic artery was considered in 12 patients who underwent splenic artery ligation and/or splenectomy for bleeding varices. Seven patients with splenomegaly and an enlarged splenic artery all survived and left the hospital without recurrent hemorrhage. Five comparable patients without an enlarged splenic artery including three with splenomegaly all continued to bleed or rebled and died early in postoperative periods. The results suggest that when an enlarged splenic artery can be identified by celiac angiography or at operation in patients with bleeding varices, splenic artery ligation and/or splenectomy may be reasonable alternatives to portacaval shunt.

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