Etiologic Factors and Consequences of Splenic Vein Obstruction

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SPLENIC VEIN obstruction is an infrequently recognized and infrequently reported intra-abdominal vascular problem. It is usually grouped with portal hypertension, rarely being considered a separate entity. Thus, Sutton,[4] in 1968, found only 53 cases of isolated splenic vein obstruction in the English literature. No cases of primary splenic vein obstruction have been reported; it is always secondary to or a complication of localized upper abdominal disease or a generalized systemic process. The course of that primary disease may be profoundly altered, however, by this complication, which often is manifest by gastrointestinal bleeding. When bleeding occurs, splenectomy is the procedure of choice, combined with the appropriate treatment of the primary disease. In this report we will present a summary of eight cases of splenic vein obstruction.

Clinical Material

Between 1955 and 1971, eight patients were seen at the North Carolina Baptist Hospital with surgical, pathologic, or angiographic evidence of isolated splenic vein obstruction. Six of these cases were observed within the last 2 years. The data from the eight cases are presented in Table 1.

Clinical Histories: Of the three patients presenting with massive hematemesis, melena or both, two had undergone, at another hospital, an operative procedure that had unsuccessfully controlled the bleeding, and had been transferred immediately to our hospital for further treatment.

Laboratory Findings: Anemia was present only in the patients with hematemesis and melena and could be accounted for by the blood loss. Bone marrow studies were done on two patients: the marrow was slightly hyperplastic in one and hypoplastic in the other. None of the eight patients had clearcut evidence of hypersplenism.

Angiography: Celiac axis angiography was done in the six patients seen after angiography had become a common diagnostic procedure. Splenoportography was done in one of the six as well. Representative angiograms are shown in Figures 1 and 2.

Diagnoses: Disease of the pancreas was primary in all of these cases (Table 1). The diagnosis was clearcut in six; in the two cases diagnosed as nonspecific pancreatitis, a mass containing multiple small cystic spaces was found at operation. The mass incorporated the pancreas and the splenic vein, and there were huge, dilated, tortuous and thin-walled veins about the stomach, omentum and splenic pedicle. The pathologist did not believe that the material submitted for diagnosis was clearly a cystadenoma.

Results: One patient died. The remaining seven patients were discharged and have remained well, with the exception of the two with carcinoma.

Discussion

Splenic vein obstruction may be caused by thrombosis, benign or malignant tumors, inflammation, or fibrosis.[5] The course of the splenic vein along the superior-posterior surface of the pancreas, where it often forms a groove in the pancreas makes it susceptible to entrapment by any disease involving the pancreatic body and tail. Obstruction of this vein becomes life-threatening only when bleeding into the gastrointestinal tract or the peritoneal cavity occurs. Rupture of the subserosal venous collateral circulation with intraperitoneal hemorrhage is a particu-

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larly treacherous complication, and anyone observing, during celiotomy, how these vessels bleed at the slightest touch must wonder why this complication does not occur more often. The stomach is the most common site of bleeding, however, since there the thin-walled, dilated, submucosal collateral vessels are subject to corrosion by acidic peptic juice. When such hemorrhage is the manifestation of splenic vein obstruction, we believe that splenectomy alone is the procedure of choice and that it can be expected to control both intragastric and intraperitoneal bleeding. Any other surgical maneuver is directed at the primary disease causing the obstruction.

When splenic vein obstruction is present without complicating hemorrhage, the therapy should be directed toward the primary disease, and splenectomy should be done or not, depending on the surgeon's assessment of the case. It is of interest that Arner,[1] using follow-up or serial splenoportography observed re-opening of the splenic vein in one patient 17 months after the initial roentgenographic diagnosis had been made. Whether this is a regular phenomenon is not known.

The development of abdominal angiography has greatly enhanced our understanding of this complication of upper abdominal disease. The detection of six cases in the last two years, all diagnosed by angiography, contrasts sharply with the two cases seen in the previous 15 years when diagnosis depended on observation at operation or autopsy. The two angiographic methods of observing the splenic venous circulation are complementary. Celiac axis injection is the more versatile, and allows study of other arterial beds; the venous phase must be included in the study if all information is to be obtained. Although splenoportography yields better definition of the splenic venous bed when clarification of detail is essential to the proposed treatment, the hazard to the patient is increased by splenic puncture.

The development of angiography as a diagnostic tool has allowed clarification of the course of the collateral circulation in isolated splenic vein obstruction. In the six patients in this series in whom angiography was done, the blood draining from the spleen returned to the portal vein by one of three routes: 1) retrograde flow through the short gastric veins, across the fundus of the stomach to the left and right gastric or coronary vein, and thence to the portal vein; 2) retrograde flow through the left gastroepiploic vein, across omental branches to the branches of the right gastroepiploic vein, and thence to the superior mesenteric or portal vein; 3) less commonly, retrograde flow through the left gastroepiploic vein to omental branches or branches of the left colic vein, and thence to the inferior mesenteric vein. Leger[2] has demonstrated a fourth route, via the diaphragmatic and intercostal branches back to the caval system. If the left

TABLE 1. *Summary of Clinical Data from Eight Patients with Splenic Vein Obstruction*

Pt #	Sex	Age (yrs)	Date of Admission	Presenting Problem	Splenomegaly	Hypersplenism	Angiography	Diagnosis	Operation	Result
1	F	37	7/31/55	G. I. Bleeding	yes	no	no	Pseudocyst of pancreas	Splenectomy, Pancreatic cystjejunostomy	Expired 1 wk. p.o., Peritonitis
2	M	41	2/10/59	Abdominal pain	no	no	no	Acute pancreatitis Intraperitoneal hemorrhage	Splenectomy, Evac. clot, Biop. omentum	Recovered
3	F	45	5/4/71	G. I. Bleeding	yes	?	yes	Cystadenoma of pancreas	Splenectomy, Distal pancreatectomy	Recovered
4	M	55	10/25/71	Abdominal pain	no	no	yes	Ca pancreas	Expl. celiotomy, Biop. liver	Recovered
5	F	23	1/14/72	G. I. Bleeding	yes	no	yes	Fibrosis, Cyst formation, Mild Inflammation	Splenectomy, Distal pancreatectomy	Recovered
6	F	27	2/7/72	Mass, abdominal pain	yes	no	yes	Chronic nonspecific pancreatitis and abscess	Splenectomy, Distal pancreatectomy	Recovered
7	F	41	2/23/72	Mass, abdominal pain	yes	no	yes	Ca pancreas	Expl. celiotomy, Biopsies, Cholecystojejunostomy	Recovered
8	F	25	3/23/72	Mass, abdominal pain	no	no	yes	Pseudocyst of pancreas	Cystogastrostomy	Recovered

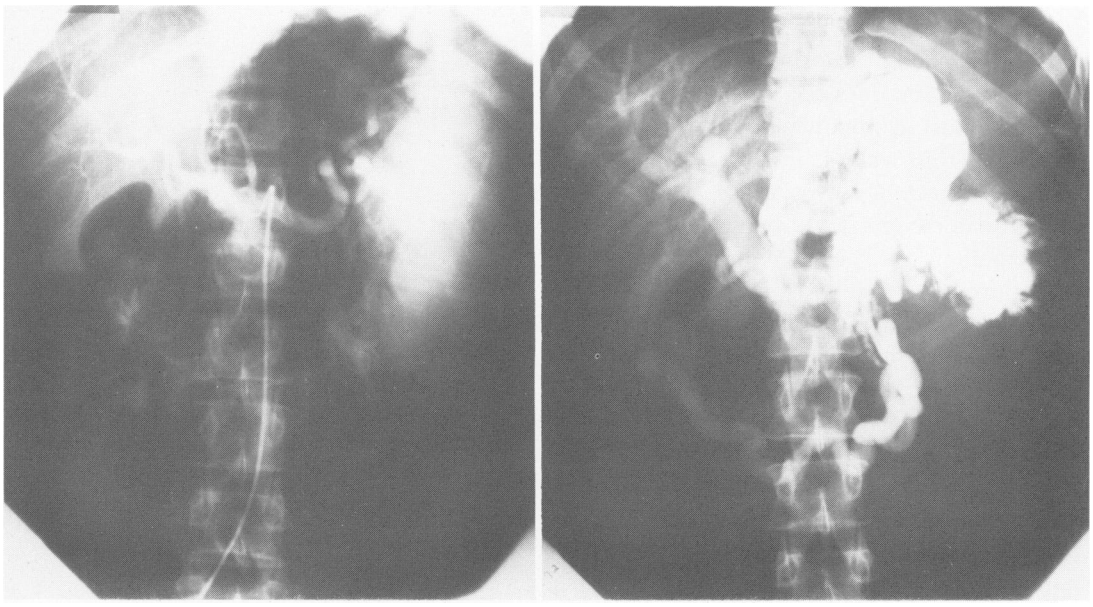

FIG. 1. Celiac axis angiogram (left) and splenoportogram (right) from Case 5. On the left, the arterial phase clearly delineates the tortuous splenic artery and the enlarged spleen. The splenic vein did not fill on any of the films in this sequence. On the right, this frame clearly outlines the dilated tortuous collaterals over the fundus of the stomach filling the coronary and portal veins. The left and right gastroepiploic veins also act as collaterals.

gastric or coronary venous system is open and available for drainage, the esophageal plexus is apparently rarely involved, and esophageal varices rarely develop. That there may be other pathways is certainly probable since the upper abdomen is more generously supplied with collateral arterial and venous circulation than any other part of the body.[3]

It is of interest that none of these patients had clearcut evidence of hypersplenism.

Summary

Eight patients with isolated splenic vein obstruction have been seen and treated at the North Carolina Baptist Hospital over the past 17 years. From a review of these patients, the following conclusions are suggested.

Splenic vein obstruction may be caused by a variety of pathologic conditions. All in these patients were related to the pancreas. Splenic vein obstruction is rarely if ever a primary disease.

Splenic vein obstruction may produce intragastric or intraperitoneal bleeding that may be life-threatening. Splenectomy is then the procedure of choice.

Splenic vein obstruction may produce no signs or symptoms.

Splenic vein obstruction usually results in the development of collateral pathways to the portal vein through the gastric or gastroepiploic veins.

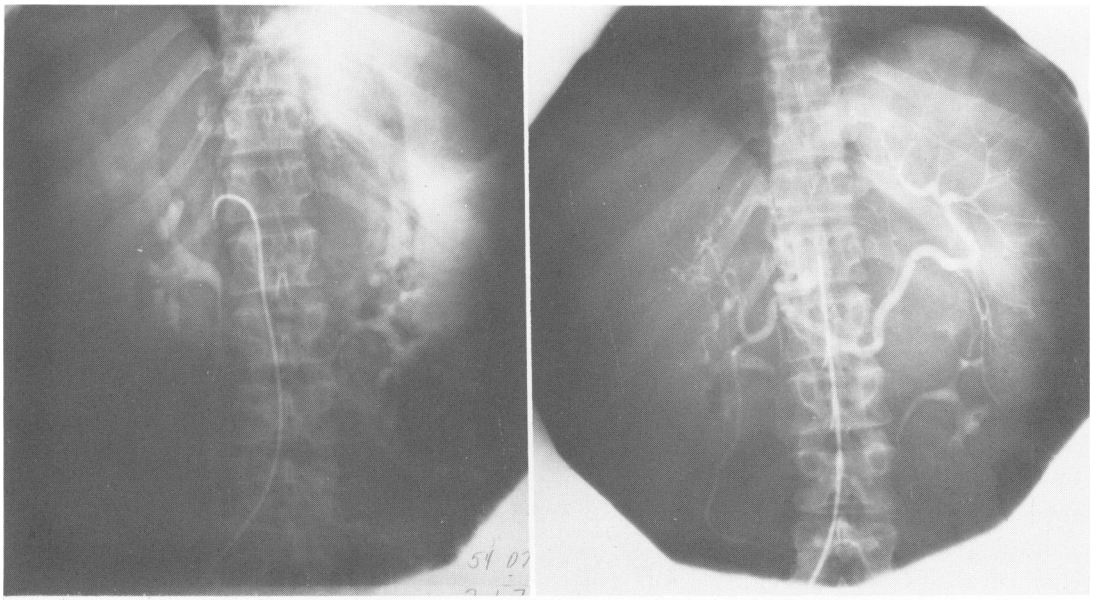

FIG. 2. Two frames in the series of celiac axis angiograms from Case 6. On the right, the arterial phase shows the large spleen and on the left, the venous phase delineates dilated tortuous veins filling retrogradely to the epiploics, omental, left colic, and then to the inferior mesenteric vein. The portal vein is faintly outlined in the right upper quadrant above the right renal pelvis.

Celiac axis arteriogram and splenoportography are the only reliable means of diagnosing this complication other than operation or autopsy.

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Discussion

Professor J. Philip Sandblom (Lausanne, Switzerland): I was very interested in commenting on this beautifully presented series of a disorder that has seemed to be very rare. I agree with the authors that it might be occurring more often than has been thought.

The reason for my opinion is that 10 years ago we went through our material of portal hypertension in over 200 patients. By way of splenoportography we found four patients with splenic vein obstruction.

Three of them bled from ruptured esophageal varices. Only one of the four had pancreatic disease as primary cause; the three others were unexplained. They might have been of a different kind than the ones presented. Their symptoms of pain, hemorrhage, and short-lasting ascites recurred several times.

All were treated by splenectomy, and none rebled. One patient died because of recurrent thrombosis that continued up into the mesenteric vein, and a large intestinal resection was performed.

Another point of interest is the source of hemorrhage in portal hypertension. It has been thought that these patients generally bleed from ruptured varices in the esophagus or the fundus of the stomach. If the bleeding source is hemorrhagic gastritis, this has not been thought to be related directly to their portal hypertension. Patients may bleed from many sources in portal hypertension, including the esophagus, the gastric mucosa, and even the biliary tract. Twice I found hemorrhage into the gallbladder.

I think the primary cause is the portal hypertension, through the congestion of the mucosa which secondarily succumbs to the deleterious effects of the gastric juice in hyperacidity and drug abuse. The series that the authors here reported so beautifully is also an example of the fact that the hemorrhage in portal hemorrhage is not necessarily due to burst varicosities of the esophagus.

Dr. Atef Salam (Atlanta): During the last 18 months we saw five patients with splenic vein thrombosis, three of whom presented with upper GI bleeding. All of them had previous vagectomy and pyloroplasty, although the cause of bleeding was not recognized at the time of surgery.

[Slide] The workup of these patients should include liver catheterization, direct or indirect splenoportography and superior mesenteric angiography. This slide demonstrates the most characteristic angiographic feature of this disease, namely a tortuous, dilated gastroepiploic vein.

The diagnosis may not be made preoperatively if it is not suspected or if the studies could not be done because of severe bleeding. The correct diagnosis can be made at the time of operation if the surgeon is familiar with the operative findings characteristic of this disease. Dilated gastroepiploic veins and gastric varices in absence of any evidence of portal hypertension in the superior mesenteric venous bed should raise the possibility of splenic vein thrombosis. The diagnosis can be confirmed by threading a catheter through one of the branches of the superior mesenteric vein into the portal vein. [Slide] In contrast to patients with generalized portal hypertension, portal pressure and liver perfusion with portal blood remain normal in patients with isolated thrombosis of the splenic vein. Contrast material injected directly into the portal vein would not opacify the coronary vein of the variceal plexus since the direction of blood flow in these vessels is toward the portal vein.

Finally, I would like to raise two questions regarding the operative management of pancreatic pseudocyst associated with splenic vein thrombosis. Splenectomy in these patients will leave the cyst in free communication with the rest of the peritoneal cavity. I wonder whether Dr. Johnston would agree to staging the operation, namely the cyst is internally drained first and splenectomy is delayed to a later date. The second question is: "What would be the method of choice for internal drainage of the pseudocyst in presence of splenic vein thrombosis?" Dr. Zeppa and Dr. Smith in their excellent presentations indicated that bleeding is sometimes encountered postoperatively following cystogastrostomy. The risk of this complication is even greater in presence of splenic vein thrombosis because of the increased vascularity of the stomach wall associated with this disease. For this reason, we prefer cystojejunostomy for internal drainage of pancreatic pseudocyst in such patients.

Dr. Richard T. Myers (Closing): We are aware of the great work Professor Sandblom has done in this area, and thank him for emphasizing the points that were important to us; namely, surprise at the infrequency with which we have encountered it in recent years is probably due more to the increasing incidence of pancreatic disease, rather than our diagnostic acumen.

We would certainly agree that this is a form of localized portal hypertension, and that one must consider the possibility of bleeding from the gastric mucosa as well as the esophageal varices.

Dr. Salam, we thank you for your comments. It's an interesting parallel to the increasing frequency in Winston-Salem and Atlanta. We would agree that the diagnosis cannot be made preoperatively as to the primary etiologic factors concerned.

So far as your posed question, I think in the absence of bleeding from the pseudocyst we would certainly agree that the procedure should be staged.

Finally, in the interest of time, I would like to simply underline and emphasize one of the points; namely, the use of angiography in upper GI bleeding. I think this paper would tend to—at least, to me—indicate the value of looking at the venous phase as well as the arterial phase. We tend to become very enthralled at what is going on within the lumen of the bowel, and not the venous phase of the angiography. This has been a great boon to the preoperative diagnosis and management of gastrointestinal bleeding, and I think, properly extended and utilized, it can be of great value in uncovering some of the obscure causes which we are now missing.