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Isolated Splenic Vein Thrombosis

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Isolated splenic vein thrombosis is being recognized more frequently as a complication of pancreatic disease and as the cause of gastrointestinal hemorrhage in patients without liver disease. The increased incidence reflects advances in diagnostic radiology and a higher index of suspicion for the diagnosis.

Splenic vein thrombosis should be suspected in: (a) a patient with a history of pancreatitis and gastrointestinal blood loss; (b) a patient with splenomegaly in the absence of portal hypertension, cirrhosis, or hematologic disease; and (c) in the setting of isolated gastric varices.

Celiac angiography has replaced splenoportography as the definitive diagnostic tool for splenic vein thrombosis and is indicated prior to operation for suspected portal hypertension or for complications of pancreatitis. The importance of making the diagnosis lies in differentiating the lesion from the more common case of hepatic and extrahepatic portal hypertension. The treatment of choice is splenectomy.

The incidence of isolated splenic vein thrombosis has increased over the past 15 years. This rise is due to an increased awareness of the problem and consequently to earlier diagnosis, rather than to a change in the natural history of diseases associated with splenic vein thrombosis.

In earlier literature, conditions such as splenic anemia and Banti's syndrome were described. Descriptions of this syndrome may in fact have been clinical descriptions of individuals with splenic vein thrombosis [1]. Without the technical ability to confirm the diagnosis, its actual incidence was unknown. Postmortem descriptions of splenic vein thrombosis were reported in 1920, and in 1939 a review of the syndrome and description of its pathophysiological features were published [2, 3]. Few cases were reported until after Sutton's review of 54 cases from the English literature between 1900 and 1968 [4]. Since then, at least 144 additional cases have been reported in the United States, and a similar number can be found in the foreign literature [5–40].

The increase in reported cases since 1968 reflects advances in diagnostic radiology and a higher index of suspicion for this entity. The importance of making the diagnosis has remained critical in the treatment of portal hypertension, gastric varices, gastrointestinal hemorrhage, and pancreatic disease.

A review of the case reports since Sutton's article in 1968 establishes the association between pancreatic disease and splenic vein thrombosis, illustrates the improved methods of diagnosis and treatment, and documents the expected long-term outcome.

Methods and Material

One hundred and forty-four cases of isolated splenic vein thrombosis were reported between 1969 and 1984 in the English literature. Included in the reports with sufficient patient data were 61 males and 29 females, ranging from 1 to 70 years of age, with an average age of 44 years.

The diagnosis of splenic vein thrombosis was frequently unsuspected, and in several cases had been misdiagnosed during previous hospitalizations. Patients presented to a physician because of recurrent gastrointestinal blood loss in 65 (45%) cases manifested by anemia, hematemesis, or melena, and with recurrent abdominal pain in 37 (26%) cases. Splenomegaly was documented in 46 cases: 32 on clinical examination and 14 either at

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laparotomy, during angiography, computed tomography, liver/spleen scan, or intravenous pyelography.

The etiology of the thrombosis was most often related to pancreatic disease. Pancreatitis characterized as chronic, acute, traumatic, or hereditary, or cases associated with a pseudocyst represented 81 (56%) of 144 cases. Five cases of acute pancreatitis were specifically noted. The second largest category of pancreatic disease causing splenic vein thrombosis was carcinoma. The microscopic diagnosis was rarely mentioned; however, there were 3 islet cell, 1 adenocarcinoma, and 1 cystadenoma, in addition to 8 other pancreatic malignancies.

Iatrogenic causes included splenectomy, umbilical vein catheterization, Warren-Zeppa shunt, and partial gastrectomy. Miscellaneous causes included splenic artery aneurysm, gastric ulcer, retroperitoneal fibrosis, retroperitoneal Hodgkin's disease, retroperitoneal liposarcoma, peripancreatic lymphoma, pancreatic transplantation, and "idiopathic" thrombosis.

Fifty-one patients were evaluated for gastrointestinal blood loss; 25 had gastric varices, 4 esophageal varices, and 22 gastroesophageal varices. In addition, there were 22 patients (13 with gastric and 9 with gastroesophageal varices) reported without a history of gastrointestinal blood loss. Of the 73 patients with varices, 68 (93%) were associated with pancreatic disease.

Approximately 72 upper gastrointestinal barium studies were done. The diagnosis of gastric or esophageal varices was correctly made in 31 of these studies, suggested in 20, and missed in 21. Similarly, gastroscopic studies were done in 45 patients. The diagnosis of gastric or esophageal varices was correctly made in 17, suggestive in 10, and missed in 18. Angiography and splenoportography were successful at diagnosing varices 100% of the time.

The diagnosis of splenic vein thrombosis was documented by either celiac or superior mesenteric artery angiography in 88 cases and splenoportography in 14.

The treatment for splenic vein thrombosis included splenectomy in 79 of the 85 patients who underwent an operation. Additional procedures were performed based on the patients' diagnosis. A distal pancreatectomy was done in 10, pseudocyst drainage in 7 (3 externally; 4 internally, 2 by cystogastrostomy and 2 by cystojejunostomy), a total pancreatectomy in 4, partial gastrectomy in 3, and a gastrotomy for gastric variceal ligation in 5 patients.

Postoperative follow-up varied from 5 weeks to 14 years. The majority of patients (92%) who had had a splenectomy were alive and well without recurrent gastrointestinal hemorrhage after a mean follow-up period of 11 months. There were 6 deaths after splenectomy, 1 due to hepatic failure, 1 to a pancreatic tumor diagnosed 12 months after operation, 1 to extensive retroperitoneal fibrosis, and 3 to complications associated with pseudocyst.

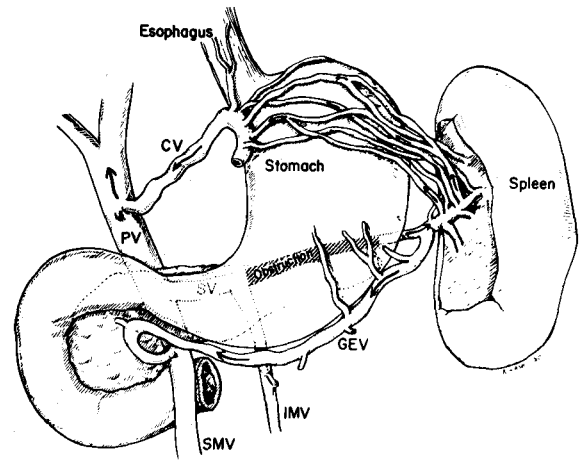

Fig. 1. Splenic vein thrombosis causing left-sided portal hypertension. Note the splenomegaly, gastric varices, and dilatation of short gastric, gastroepiploic (GEV), and coronary veins (CV). The portal vein (PV), superior mesenteric vein (SMV), and inferior mesenteric vein (IMV) are patent. Previously published in the American Journal of Surgery [21]. Used by permission.

Discussion

The pathogenesis of splenic vein thrombosis relates to its anatomic location (Fig. 1). The splenic vein, approximately 0.5 cm in diameter and 12 cm long, lies inferior to the splenic artery and directly posterior to the pancreas, extending along its tail and neck. The splenic vein ends in a 90° turn to empty into the portal vein. Because of its location, any type of pancreatic disease is likely to involve the splenic vein. In addition to the relationship of the vein with the pancreas, there are closely approximated pancreaticolienal lymph nodes which accompany the splenic artery as it runs along the posterior surface of the pancreas, just superior to the splenic vein. Lymph nodes along the left border of the lesser sac are directly inferior to the pancreas and splenic vein, and when involved with retroperitoneal disease, contribute to the occurrence of splenic vein thrombosis [34, 41, 42].

Thrombosis of the splenic vein develops by 1 of 2 methods [5]. Intrinsic damage to the intima of the splenic vein occurs as a result of neoplastic and inflammatory disease or surgical manipulation. Extrinsic damage occurs by compression of the vein secondary to edema, cellular infiltration, and fibrosis. The result in either case is stasis of blood flow and eventually partial to complete occlusion.

The natural history suggests that the greater the duration, frequency, or extent of pancreatic disease, the more likely that the splenic vein circulation will be permanently compromised. Surprisingly, associated portal vein thrombosis is a relatively rare occurrence and is evident as a preterminal event in 2 clinical situations: (a) secondary to suppurative portal pyelophlebitis during the septic stages of necrotizing pancreatitis, and (b) when an underlying carcinoma in the head of the pancreas is the cause of the pancreatitis.

In Sutton's review of splenic vein thrombosis, the most common cause was pancreatic cancer, noted in 13 (35%) of 54 cases, followed by pancreatitis in only 6 cases. In our review, pancreatitis, diagnosed by biopsy or at operation, was more common, occurring in 87 (60%) of 144 cases. It should be noted that very few patients were being evaluated for a presumed exacerbation of pancreatitis when the diagnosis of splenic vein thrombosis was made. Four patients were noted to have had only a single previous episode of pancreatitis prior to the diagnosis [21]. The majority of patients gave a history of recurrent abdominal pain and previous hospitalizations for pancreatitis. This suggests that repeated episodes of indolent pancreatic inflammation may lead to splenic vein thrombosis.

The overall incidence of splenic vein thrombosis in patients with pancreatitis is unknown. It is clear, however, that splenic vein thrombosis may occur in any phase of pancreatic inflammation. Both the thrombotic process and the pancreatitis may be occult and the first clinical manifestation may be acute or chronic gastrointestinal blood loss from the varices. There have been several studies that indicate that the splenic vein is abnormal in 9 to 90% of patients with pancreatitis. Rosch and Herfort conducted splenoportography in 95 patients with chronic pancreatitis, and found 85 with slight to moderate splenic vein deformation, and 4 with advanced occlusion and collateral flow [43]. Leger reported that 54% of patients with surgically proven pancreatitis had abnormal splenoportograms and 24% complete occlusion of the splenic vein [44]. Lemaitre [45] reported an 8.5% incidence in a similar group, and Rignault [46] found 9 of 20 patients with chronic pancreatitis to have splenic vein thrombosis after splenoportography. Recently, Little and Moossa reported 16 cases of splenic vein occlusion in 92 patients with acute or chronic pancreatitis [21]. These studies and the present review suggest a strong association between pancreatitis and splenic vein thrombosis.

Tumors of the pancreas are less frequently associated with splenic vein thrombosis. Unlike an inflammatory process, pancreatic tumors tend to be localized. In this review there were 13 cases of pancreatic cancer including 3 islet-cell tumors [5, 8, 35]. Islet-cell tumors have a slow growth rate and are rarely diagnosed as mass lesions disturbing normal anatomic structures. Islet-cell tumors are more likely to be recognized by excessive secretion of a peptide hormone before they lead to splenic vein thrombosis. A nonfunctioning islet-cell tumor, as reported by Wolf and associates [35], is more likely to cause venous involvement because of the delay in diagnosis and subsequent increase in size. Localizing small islet-cell tumors can frequently be a problem, but they can usually be excised, and patient prognosis is good. In contrast, splenic vein occlusion secondary to pancreatic adenocarcinoma is highly suggestive of extrapancreatic extension, an unresectable lesion and a poor prognosis.

Iatrogenic causes of splenic vein thrombosis are rarely reported. There were 5 cases of splenic vein thrombosis after splenectomy [37]. Details of the patient histories were not available. No additional reports of this complication were found in the pediatric or trauma literature, or in follow-up of the 76 patients treated with splenectomy in this review.

There were 3 reports of splenic vein thrombosis following Warren-Zeppa's distal splenorenal shunt. This illustrates the importance of considering splenic vein thrombosis in patients who continue to have gastrointestinal bleeding postoperatively [26]. In the senior author's experience with 2 additional cases, such massive relentless variceal hemorrhage following thrombosed Warren-Zeppa shunt necessitated emergency splenectomy under difficult conditions. This was followed, at the same operation, by a standard portocaval shunt.

Selective venous catheterization has rarely been associated with splenic vein thrombosis. There are no reported complications of this type after percutaneous venous sampling. The 3 reported cases after umbilical vein catheterization illustrate that it can occur after only 24 hours of catheter use and with delayed side effects [33]. The authors suggest that the catheter initially causes a direct intimal lesion that is further predisposed to infection by decreased blood flow and the presence of a foreign body.

After splenic vein thrombosis occurs, the preferential route of venous flow is toward the portal vein. The hepatoportal or portoportal pathway begins with the short gastric veins which cross the fundus of the stomach, drain into the left and right gastric or coronary vein, and end in the portal circulation. Increased flow across the short gastric veins creates a local form of extrahepatic portal hypertension, sometimes referred to as left-sided or "sinistral" portal hypertension. Eventually, gastric varices develop along the greater curvature and fundus of the stomach [36] (Fig. 2).

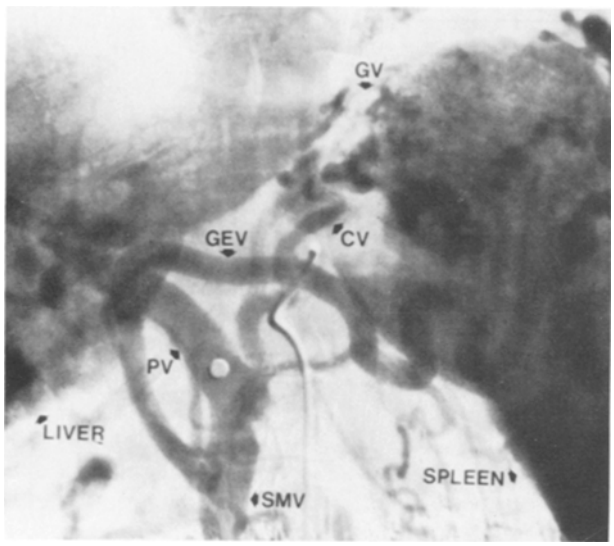

Fig. 2. Venous phase on selective celiac axis angiogram. The splenic vein is not visualized, indicating splenic vein occlusion. Gastric varices (GV), a dilated coronary vein (CV), and a dilated gastroepiploic vein (GEV) are present. The portal vein (PV) and superior mesenteric vein (SMV) are normal. Previously published in the American Journal of Surgery [21]. Used by permission.

The extragastric component of the collateral pathway is represented by the left gastroepiploic vein. Its path runs through the omental branches to the right gastroepiploic vein and finally to the superior mesenteric or portal vein. Dilatation of the gastroepiploic veins is thus another common feature associated with splenic vein thrombosis.

There are 2 additional venous pathways described in the literature. The first is referred to as the hepatofugal or portasystemic pathway and also involves the left gastroepiploic vein. The vein drains across the omental branches of the left colic vein to drain into the inferior mesenteric vein. Hemorrhage from colonic varices in patients with sinistral portal hypertension secondary to pancreatitis is very rare but has been reported [39]. The second pathway, described by Leger, drains along the diaphragmatic and intercostal veins to the caval venous system.

The most frequent complication associated with splenic vein occlusion is gastrointestinal hemorrhage. Recurrent hematemesis, melena, and anemia are manifestations of the significant blood loss from bleeding gastric and esophageal varices. Isolated gastric varices are a sensitive indicator of splenic vein occlusion but are one of the later and more difficult signs to diagnose. The ability to see varices by upper gastrointestinal studies or gastroscopy is suboptimal and often delays the diagnosis of splenic vein thrombosis [24].

The radiologic characteristics of these lesions have been well described. When outlined by barium, they appear as thick, tortuous mucosal folds, filling defects, or distorted mucosal configurations over the greater curvature extending toward the cardia [23]. The correct diagnosis, however, often requires differentiating them from gastric neoplasms [27, 47].

Esophageal varices, usually secondary to portal hypertension of hepatic origin, also develop after splenic vein thrombosis. In the present review, 22 (46%) cases of gastroesophageal varices and 4(1%) of isolated esophageal varices were reported. Esophageal varices presumably develop when hepatofugal collaterals via the short gastric veins are inadequate to decompress the leftsided hypertension. Anatomically, they are likely to develop if the coronary vein inserts into the splenic vein, reportedly occuring in 17% of cases, instead of the portal vein [21, 32].

After percutaneous splenoportography was popularized in 1951 by Leger, physicians were able to diagnose splenic vein thrombosis preoperatively. This procedure is rarely used today. Angiography is the preferred method for making the diagnosis and is a safer, more controlled procedure in a patient who often may have associated coagulation abnormalities and elevated portal or systemic pressures [36]. Angiography outlines the location of obstruction and route of decompression. The celiac axis injection provides access to surruounding arterial beds and abnormal hepatic and pancreatic neoplastic vasculature, as well as for venous phase studies. The angiographic diagnosis is confirmed when the splenic vein fails to opacify on the venous phase of the splenic artery injection. Also seen is a diffuse blush of venous collaterals in the splenic hilum and often a display of dilated gastroepiploic and short gastric veins.

Ultrasonography has recently been advocated as an inexpensive, noninvasive means of early recognition of splenic vein thrombosis. Results of the studies, however, have suggested that its use may be limited to the diagnosis of portal hypertension and portal vein thrombosis. The size and location of the splenic vein make it less optimal for ultrasonographic visualization. In the emergency work-up of gastrointestinal hemorrhage, if extrahepatic portal obstruction is suspected, an ultrasound scan demonstrating a normal portal and splenic vein eliminates the diagnosis [48, 49].

The treatment for splenic vein thrombosis is splenectomy. Removal of the spleen decreases the venous outflow through the collateral circulation and decompresses the associated varices to prevent further hemorrhage. In most situations, an additional procedure is done simultaneously to treat the associated pathological conditions and prevent fur-

ther complications. Partial pancreatectomy and internal pseudocyst drainage procedures are frequently done in patients with a pseudocyst at the time of splenectomy. The preferred drainage procedure is a cystojejunostomy Roux-en-Y over a cystogastrostomy to avoid precipitating bleeding from transected gastric varices [15, 21, 30].

In the emergency situation, it has been advocated that gastric varices be oversewn through a gastrotomy [32, 50]. This procedure was done in only 5 patients because the majority of patients were not actively bleeding at the time of operation [35]. There is a single case of a patient who was returned to the operating room for suture and ligation of gastric varices after splenectomy.

Transcatheter splenic artery embolization in unstable patients has been suggested by Jones and associates to produce a "nonsurgical splenectomy" [15]. The associated complication of splenic abscess and the inability to treat the pancreatic disease directly make it a less than optimal alternative to splenectomy.

The results after splenectomy have been good. In the present review, 92% of patients had not had another episode of gastrointestinal blood loss after a mean follow-up of 11 months. Postoperative complications occurred in only 6 patients and were secondary to extensive hepatic or pancreatic disease. In 3 of these patients, there was a delay in surgical intervention because of the patients' poor condition.

The risk of splenectomy and of postoperative sepsis are issues that must be considered in the patient with splenic vein thrombosis. Whether preoperative innoculation with pneumovaccine is indicated as a routine prophylaxis is uncertain at present. When the diagnosis is made incidentally or at the time of operation for complications of pancreatitis, an elective splenectomy might be considered. For this reason, we suggest routine preoperative angiography prior to operation for portal hypertension and pancreatic disease. With this information, the likelihood of gastrointestinal hemorrhage secondary to splenic vein thrombosis can be assessed and a treatment plan developed.

Conclusion

Isolated splenic vein thrombosis is being recognized more frequently as a complication of pancreatic disease and as a precursor to gastrointestinal variceal hemorrhage. The importance of differentiating the lesion from the usual case of extrahepatic and hepatic portal hypertension lies in choosing the most appropriate corrective surgical procedure.

The diagnosis of splenic vein thrombosis should be suspected and ruled out in the following situations: (a) a patient with a history of pancreatitis who has bled from esophageal or gastric varices; (b) splenomegaly in the absence of portal hypertension, cirrhosis, or a hematologic disorder; (c) gastric varices on gastroscopy or barium studies; (d) collateral veins in the left upper abdomen during the vascular phase of rapid sequence intravenous pyelography [15]; and (e) a patient scheduled for an elective operation for a complication of pancreatitis.

Résumé

La thrombose isolée de la veine splénique est reconnue avec une plus grande fréquence qu'elle complique une affection pancréatique ou qu'elle soit la cause d'une hémorragie digestive chez des malades dont le foie est normal. Ce fait reflète les progrès dans le diagnostic radiologique et dans la connaissance de sa relative fréquence.

La thrombose de la veine splénique doit être envisagée: (a) chez un malade qui présente une histoire de pancréatite ou d'une hémorragie gastro-intestinale; (b) chez un sujet porteur d'une splénomégalie en l'absence d'hypertension portale, de cirrhose ou de maladie hématologique; et (c) en présence de varices gastriques isolées.

L'angiographie coeliaque a pris la place de la splénoportographie pour porter le diagnostic; elle est indiquée avant toute opération pour hypertension portale ou pancréatite compliquée. Cette exploration capitale permet de distinguer la thrombose de la veine splénique de causes plus courantes à l'origine de l'hypertension portale d'origine hépatique ou extrahépatique. Le traitement de choix est la splénectomie.

Resumen

La trombosis aislada de la vena esplénica viene siendo reconocida con mayor frecuencia como consecuencia de enfermedad pancreática y como causa de hemorragia gastrointestinal en pacientes libres de enfermedad hepática. La aumentada incidencia es un reflejo de los avances en radiología y de un más alto índice de sospecha en el proceso diagnóstico.

Se debe sospechar la presencia de trombosis de la vena esplénica en: (a) un paciente con historia de pancreatitis y de sangrado gastrointestinal; (b) un paciente con esplenomegalia en ausencia de hipertensión portal, cirrosis o enfermedad hematológica, y (c) en presencia de várices gástricas aisladas.

La angiografía celiaca ha suplantado a la esplenoportografía como método definitivo de

diagnóstico en casos de trombosis de la vena esplénica, y se halla indicado antes de la operación por sospecha de hipertensión portal y por complicaciones de la pancreatitis. La importancia de hacer el diagnóstico reside en diferenciar esta lesión de otras causas más comunes de hipertensión portal hepática y extrahepática. El tratamiento de elección es la esplenectomía.

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