

Splenic vein thrombosis in a patient with chronic pancreatitis

S Nasiri, J Khurgami, K Donboli

Citation

S Nasiri, J Khurgami, K Donboli. *Splenic vein thrombosis in a patient with chronic pancreatitis*. The Internet Journal of Surgery. 2007 Volume 16 Number 2.

Abstract

The most common cause of isolated thrombosis of the splenic vein is chronic pancreatitis caused by perivenous inflammation¹. Although splenic vein thrombosis (SVT) has been reported in up to 45% of patients with chronic pancreatitis, most patients with SVT remain asymptomatic⁷. In those patients with gastrointestinal bleeding secondary to esophageal or gastric varices, the diagnostic test of choice to assess for the presence of SVT is late-phase celiac angiography. Splenectomy effectively eliminates the collateral outflow and is the treatment of choice⁴.

CASE REPORT

A 23-year-old male complaining of upper gastrointestinal bleeding in the form of melena and episodes of massive hematemesis was admitted in the surgical ward of Shariati Hospital. He had episodes of melena and hematemesis since 4 months ago. He complained of abdominal pain right before hematemesis. He did not have any complaints of abdominal pain, GI bleeding, nausea and vomiting or weight loss before this. He was admitted in the gastrointestinal ward 20 days ago for upper GI bleeding management, in upper endoscopy there was severe gastritis with positive H. pylori test and no obvious bleeding source. At admission he had a supine blood pressure of 90/55mmHg and an upright blood pressure of 75/50mmHg with a pulse rate of 92/min. Baseline laboratory investigations revealed: White blood cell count (WBC) 4500/mm³, hemoglobin (Hb) 7.2g/L, platelets 178000/mm³, amylase 112U/L, prothrombin time (PT) 14 sec. with INR 1.3, creatinin 1mg/L, blood urea nitrogen (BUN) 18mg/L, alanin transferase (ALT) 10U/L, aspartate transferase (AST) 11U/L, alkaline phosphatase (Alk. Ph.) 90U/L, bilirubin total 0.9mg/dl and direct 0.3mg/dl, and fasting blood sugar (FBS) 90mg/dl. In emergency upper endoscopy there was oozing of blood in the fundus with no obvious lesion. After stabilization he underwent diagnostic studies.

In plain abdominal X-ray there were calcifications in the pancreatic region (figure 1). In abdominal ultrasonography there was an echogenic center (gallstone) in the gallbladder neck with normal billiary tract and mild ascites.

In abdominal and pelvic CT scan with IV and oral contrast there were mild splenomegaly and hyperdense regions in the pancreas as calcifications (Figure 2). Endoscopic ultrasonography of the gastric fundus was performed and there were fundal varices.

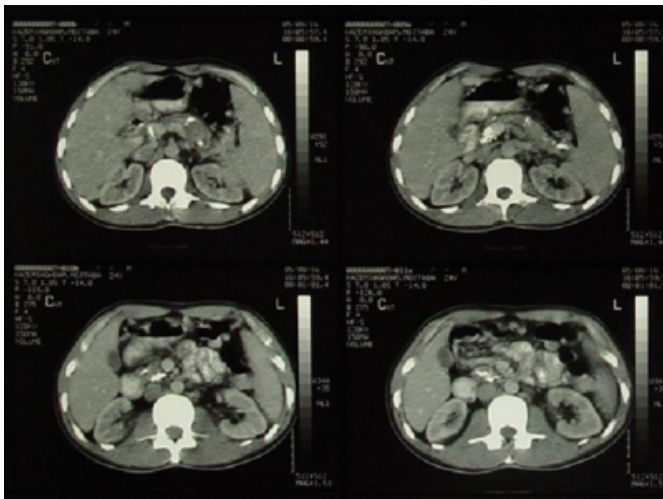
Figure 1

Figure 1: Plain abdominal X-ray.



Figure 2

Figure 2: Abdominal CT scan with pancreatic calcification.

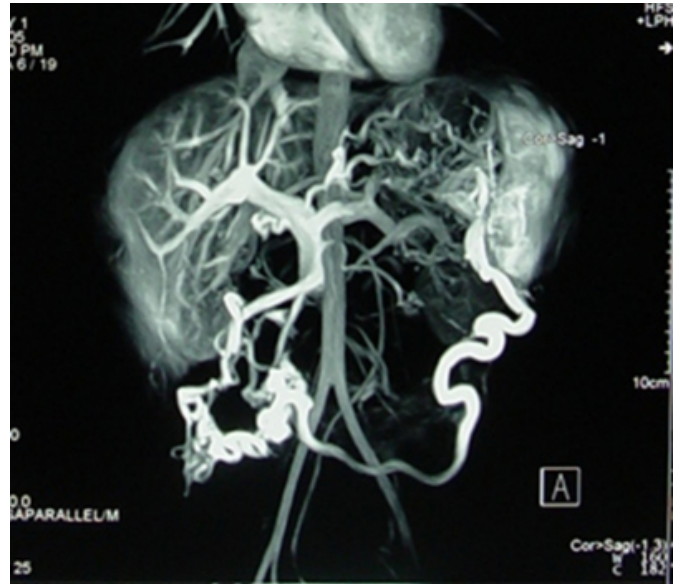


With a probable diagnosis of splenic vein thrombosis, Doppler ultrasonography was performed and there was splenic vein thrombosis up to the communication of splenic vein and superior mesenteric vein, with patent portal vein and numerous collaterals around the spleen. These findings

were confirmed in magnetic resonance venography (MRV) (figure 3). The patient underwent splenectomy with a good post-operative period with no complications and was discharged on post-operative day 4 with good general condition.

Figure 3

Figure 3: MR venography with splenic vein thrombosis.



DISCUSSION

Sinistral, or left-sided, portal hypertension caused by splenic vein thrombosis (SVT) can result in massive gastrointestinal (GI) bleeding from esophageal or gastric varices or hypertensive gastropathy. Isolated SVT develops most often in patients with acute or chronic pancreatitis¹, or pancreatic carcinoma². Unlike patients with generalized portal hypertension, most patients with SVT are asymptomatic and have normal hepatic function. Splenic vein thrombosis should be suspected in the following groups of patients: (1) those with a history of pancreatitis and newly diagnosed GI bleeding, (2) patients with splenomegaly but no portal hypertension, cirrhosis, or hematologic disease, and (3) individuals with isolated gastric varices³. There are many other causes of GI bleeding in patients with chronic pancreatitis, including pseudoaneurysms, pseudocysts, hemosuccus pancreaticus, peptic ulcer disease, gastritis, and Mallory-Weiss tears.

Splenic vein thrombosis in acute or chronic pancreatitis results from perivenous inflammation caused by the anatomic location of the splenic vein along the entire posterior aspect of the pancreatic tail, where it lies in direct contact with the peripancreatic inflammatory tissue. The

exact mechanism of thrombosis is likely multifactorial, including both intrinsic endothelial damage from inflammatory or neoplastic processes and extrinsic damage secondary to venous compression from fibrosis, adjacent pseudocysts, or edema¹. Obstruction of the splenic vein may also be caused by enlarged retroperitoneal lymph nodes or by pancreatic or perisplenic nodes that are located near the splenic artery, superior to the splenic vein. These nodes lie adjacent to the pancreas and the splenic vein and compress the splenic vein when involved in an inflammatory or neoplastic process³.

Although early reports found that the most common cause of SVT was pancreatic carcinoma⁴, more recent reviews have discovered acute or chronic pancreatitis, particularly in the caudal pancreas, to be the probable cause of SVT in the majority of cases^{1,3,5}. The relatively lower incidence of SVT in patients with pancreatic carcinoma as compared to pancreatitis may be secondary to its well-localized nature, in contrast to the diffuse inflammation of pancreatitis; pancreatitis is the initiating event in approximately 60%^{3,6} of patients, although the diagnosis of SVT in these cases is not always made during the acute attack. This suggests that repeated indolent episodes of pancreatitis result in SVT. Other causes of SVT include adenopathy from metastatic carcinoma, lymphoma, and iatrogenic causes after surgery such as splenectomy, partial gastrectomy, and distal splenorenal shunt^{3,5,6}.

Because removal of the spleen eliminates venous collateral outflow and thereby decompresses surrounding varices, splenectomy is the treatment of choice for isolated SVT⁴. In patients with acutely bleeding varices from isolated SVT, urgent splenectomy should be performed, both because of the lack of other effective options and because isolated gastric varices have a greater potential for exsanguinating hemorrhage than esophageal varices⁷.

CORRESPONDENCE TO

Dr. Shirzad Nasiri General Surgery Ward, Shariati Hospital, North Kargar St., Tehran, Iran. E-mail: nasiri@razi.tums.ac.ir Tel: +98-2184902450

References

1. Bernades P, Baitis A, Levy P, et al. Splenic portal venous obstruction in chronic pancreatitis. *Dig. Sci.* 1992;37:340-346
2. Leger L, Lenriot JP, Lemaigra G. L'hypertension et la stase portales segmentaires dans les pancreatites chroniques. A propos de 126 cas examinés par splenoportographie et spleno-manometrie. *J.Chir.* 1968;95:599-608
3. Moosa AR, Gadd MA, Isolated splenic vein thrombosis. *World J. Surg.* 1985;9:348-390
4. Sutton JP, Yarborough DY, Richards JT. Isolated splenic vein occlusion. *Arch. Surg.* 1970;100:623-630
5. Evans GRD, Yellin AE, Weaver FA, et al. Sinistral (left-sided) portal hypertension. *Am. Surg.* 1990;56:758-763
6. Madsen MS, Petersen TH, Sommer H. Segmental portal hypertension. *Ann. Surg.* 1996;204:72-77
7. Sharon M. Weber, Layton F. Rikkers. Splenic vein thrombosis and gastrointestinal bleeding in chronic pancreatitis. *World J. Surg.* 2003;27:1271-1274

Author Information

Shirzad Nasiri, MD

General Surgery Ward, Shariati Hospital

Jamak Khurgami, MD

General Surgery Ward, Shariati Hospital

Kianoush Donboli, MD

General Surgery Ward, Shariati Hospital