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Original Article

Clinical Analysis of Sinistral Portal Hypertension

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Abstract

Background: Sinistral portal hypertension (SPH) is the only type of portal hypertension that is entirely curable. However, it can easily cause varicose veins in the esophagus and/or stomach, as well as upper gastrointestinal hemorrhage. This study aimed to investigate the clinical characteristics and treatments of sinistral portal hypertension.

Methods: All patients with pancreatic disease were included in this retrospective cohort study at the Affiliated Hospital of Southwest Medical University (Luzhou, China) from September 2019 to September 2021. The required information including the patient's demographics, serum laboratory indicators, imaging and endoscopy examinations, and clinical features were gathered and evaluated. The results were expressed as numbers and percentages.

Results: Out of the 830 patients with pancreatic diseases, 61 (7.3%) developed SPH. The most common cause of SPH was acute pancreatitis (80.3%), followed by chronic pancreatitis (11.5%). The splenic vein was the most frequently affected vein in patients (45/61, 73.8%). The findings of the contrast-enhanced computed tomography (CECT) indicated that 51 cases (83.6%) had gastric fundal-body varices, and three cases had combined gastric and esophageal varices. In the perigastric collateral channel formation, gastroepiploic varices (43/61, 70.5%) most frequently occurred in patients with SPH. Splenomegaly was a prevalent manifestation in SPH patients (45.9%). Five cases had gastrointestinal variceal hemorrhage.

Conclusion: SPH was associated with the patency of the splenic vein and the formation of distinctive perigastric collateral veins. Surgery and/or endoscopic treatment were recommended, particularly for patients who have experienced a significant amount of gastrointestinal bleeding and have failed conservative treatment.

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Keywords ● Pancreatic diseases ● Sinistral portal hypertension ● Varicose veins

What's Known

- Some studies have explained the main causes and several treatments of sinistral portal hypertension.

What's New

- We found that SPH was associated with the patency of the splenic vein and the development of distinctive perigastric collateral veins. Surgery and/or endoscopic treatment were recommended, particularly for patients who have experienced a significant amount of gastrointestinal bleeding and have not responded to conservative treatment.

Introduction

Sinistral portal hypertension (SPH), also known as regional, segmental, or splenoportal hypertension, is a type of splanchnic hypertension localized in the left-sided gastrosplenic region. It is frequently caused by stenosis, obstruction, or thrombosis of the portosplenomesenteric veins (primarily the splenic vein).[1-3] SPH is a form of extrahepatic portal hypertension (EPH) that arises in the absence of liver cirrhosis and accounts for less than 5% of all

patients with portal hypertension.[1, 2]

SPH is a completely curable portal hypertension characterized by normal liver function, isolated gastric varices (with or without esophageal varices), and/or splenomegaly.[3, 4] It is a rare clinical syndrome mostly caused by chronic pancreatitis (CP), acute pancreatitis (AP), and pancreatic carcinoma.[5] Anatomically, the splenic vein is located posterior to the pancreas and in direct contact with it. Persistent pancreatic inflammation, along with external compression from pancreatic necrosis or pseudocyst, causes stenosis, thrombosis, or splenic vein occlusion.[1, 2] In most cases, SPH is a rare condition, with most patients being asymptomatic and hence disregarded by clinicians. Clinical findings frequently occurred incidentally on radiological imaging.[1-3] However, patients with SPH are at risk of gastrointestinal hemorrhage caused by esophageal and gastric varices, which is frequently severe and potentially fatal. It is estimated to occur in 4-17% of cases.[6, 7] Therefore, the accurate diagnosis and effective control of SPH should be considered to avoid lethal complications. The present retrospective cohort study aimed to investigate the clinical characteristics and treatments of SPH in individuals with pancreatic disorders.

Patients and Methods

This retrospective single-center study was conducted in the Affiliated Hospital of Southwest Medical University, Luzhou, China, from September 2019 to September 2021. Patients who met the following criteria were excluded: being under the age of 18 and pregnant, end-stage chronic disease or severe immune system disorders or other systemic tumors, chronic liver disease and cirrhosis, history of gastric, splenic, or pancreatic surgery,[5] taking anticoagulants before admission or other coagulopathy disorders, incomplete data. In accordance with the international guidelines, all study patients received standard medical treatment, including fluid therapy, pain control, nutritional support, infection complication prevention, and intensive care management. The study was approved by the Ethics Board of the Affiliated Hospital of Southwest Medical University (code: KY2023026).

Diagnosis and Definitions

The diagnostic criteria for SPH included, the presence of collateral vessels detected by contrast-enhanced computed tomography (CECT) or gastric fundal varices found by endoscopy, with or without the presence of splenomegaly or hypersplenism, exclusion of other causes of portal hypertension, such as idiopathic portal hypertension or hepatic cirrhosis.[1, 4] The presence of collateral vessels refers to disproportionate increases in the caliber and number of vessels at any location. Each type of varix was defined as follows: 1) gastroepiploic vein varices involved draining along the great curve of the stomach, beneath the gastric antrum, or at its junction with the gastrocolic truck and were considered abnormal when measuring greater than 6 mm in diameter, 2) gastric coronary vein varices involved draining in the gastrohepatic ligament when the serpiginous and tubular vessels in the gastrohepatic ligament measured greater than 6 mm in size, 3) short gastric varices were considered to be present when the diameter of the vessel was greater than 5 mm between the spleen and stomach, away from the splenic hilum, 4) if the diameter of the gastric fundal vein located in the gastric fundal wall was greater than 5 mm, varices were indicated, and 5) esophageal varices had a diameter of 4 mm or more in the distal esophagus.[3, 5]

The portal venous system, including portal, splenic, and superior mesenteric veins, was evaluated for the degree of vascular patency (normal, stenosis, occlusion), and the presence of thrombosis. A normal vessel was defined as having no constriction, with its lumen completely filled with contrast medium during enhancement. Venous stenosis was defined as a more than 50% decrease in the caliber of the lumen. Venous occlusion was defined as complete occlusion of blood vessels with no blood flow.[3, 5] Thrombosis was defined as a filling defect within the lumen of the vessel on contrast-enhanced images.[8]

Statistical Analysis

All statistical analyses were performed using SPSS software version 25.0 (SPSS Inc., Chicago, Illinois, USA). The analyzed data were presented as numbers and percentages.

Results

Demographic Characteristics, Serum Laboratory Indicators, Clinical Features, and Treatment

This study recruited 830 patients with pancreatic disease who met the inclusion criteria which was stated previously. Among 830 patients, 61 patients developed SPH. The demographic characteristics, serum laboratory indicators, and clinical features are presented in tables 1 and 2. Of the patients, 49 (80.3%) patients were men, and 12 (19.7%) patients were women.

Table 1: Demographic characteristics, serum laboratory indicators, and clinical features

Demographic characteristics		Sinistral portal hypertension
Sex	Male (%)	49 (80.3%)
	Female (%)	12 (19.7%)
Age (year)		49.21±13.994
The etiology of pancreatic disease	Acute pancreatitis	49 (80.3%)
	Chronic pancreatitis	7 (11.5%)
	Pancreatic tumor	5 (8.2%)
The severity of acute pancreatitis	Mild-AP	4 (6.6%)
	Moderate-AP	6 (9.8%)
	Severe-AP	39 (63.9%)
The etiology of primary disease	Biliary	22 (36.1%)
	Alcohol	3 (4.9%)
	Hyperlipidemia	13 (21.3%)
	Mixed factors	3 (4.9%)
	Idiopathic	8 (13.1%)
Smoking status (%)		36 (59.0%)
Drinking status (%)		38 (62.3%)
Serum laboratory indicators	PLT (×10^9/L)	215.8 (137-361)
	ALT (U/L)	37.6 (14.4-42.5)
	AST (U/L)	46.3 (17.1-56.5)
	TBIL (umol /L)	20.2 (10.2-20.6)
	DBIL (umol /L)	8.8 (2.9-9.3)
	Cr (umol /L)	74.8 (51.2-75.6)
	FIB (s)	5.5 (3.6-6.6)
	D-D (ng /mL)	5.9 (2.0-7.8)
	FDP (g /L)	22.8 (6.5-60.7)
Thrombocytopenia (%)		5 (8.2%)
Unspecific abdominal pain (%)		33 (54.1%)
Gastrointestinal bleeding (%)		5 (8.2%)
Treatment of patients with bleeding	Surgery	2 (3.1%)
	Medical treatment	3 (4.9%)

PLT: Platelet; ALT: Alanine transaminase; ASL: Aspartate transaminase; TBIL: Total bilirubin; DBIL: Direct bilirubin; Cr: Creatinine; FIB: Fibrinogen; D-Di: D-dimer; FDP: Fibrin degradation products

The mean age of the patients was 49.21±13.994 years. The most common cause of SPH was acute pancreatitis, followed by chronic pancreatitis and pancreatic malignancy. The liver and kidney functions of the patients were basically normal. Five patients developed thrombocytopenia during hospitalization. Splenomegaly was a common manifestation in SPH patients (45.9%, 28/61), while only 2 (3.3%) cases with hypersplenism were presented in the present study.

Patients with SPH were complicated with pancreatic parenchymal necrosis and necrosis was mainly located in the body-tail (32, 52.5%) of the pancreas. Application of necrotic tissue elimination was performed in three patients. Only four patients with SPH underwent drainage of pancreatic pseudocysts, and one received pancreatic duct-jejunum Roux-en-Y anastomosis. There was no perioperative mortality and no life-threatening complications.

Patients with SPH developed pancreatic parenchymal necrosis, which was primarily localized in the pancreas' body tail (32, 52.5%). Necrotic tissue removal was used on three patients. Only four SPH patients underwent pancreatic pseudocyst drainage, and one had pancreatic duct-jejunum Roux-en-Y anastomosis. There was no perioperative mortality or life-threatening complications.

Among the patients with SPH, 33 (54.1%) experienced unspecific abdominal pain, and 5 (8.2%) developed gastrointestinal variceal bleeding. Melena was the most common symptom, occurring in all patients with gastrointestinal hemorrhage, while two (40%) patients presented with both melena and hematemesis. One patient underwent splenectomy, and one took splenectomy combined with paraesophagogastric devascularization. The other three patients received conservative medical treatment. The bleeding was stopped in all of these patients at discharge.

Among the patients with SPH, 33 (54.1%) reported unspecific stomach pain, whereas 5 (8.2%) developed gastrointestinal variceal hemorrhage. Melena was the most prevalent symptom, affecting all patients

Table 2: Imaging features on contrast-enhanced computed tomography

Imaging features on CECT			Sinistral portal hypertension
Portosplenomesenteric vein (%)	Patency of splenic vein	Normal	16 (26.2%)
		Stenosis	41 (67.2%)
		Occlusion	4 (6.6%)
		Splenic vein thrombosis	8 (13.1%)
	Patency of portal vein	Normal	42 (68.9%)
		Stenosis	18 (29.5%)
		Occlusion	1 (1.6%)
		Splenic vein thrombosis	8 (13.1%)
	Patency of SMV	Normal	50 (82.0%)
		Stenosis	10 (16.4%)
		Occlusion	1 (1.6%)
		Splenic vein thrombosis	3 (5.0%)
Main varicose veins (%)	Gastric fundal - body vein		51 (83.6%)
	Splenic hilum vein		25 (41.0%)
	Combined gastric and esophageal vein		3 (4.9%)
	Gastric coronary vein, GCV		22 (36.1%)
	Gastric short veins, GSV		40 (65.6%)
	Gastroepiploic vein, GEV		43 (70.5%)
	Gastric fundic plexu		23 (37.7%)
Type of pancreatic parenchyma necrosis (%)	Head		4 (6.6%)
	Neck		3 (5.0%)
	Body-tail		32 (52.5%)
Pancreatic pseudocyst (%)			22 (36.1%)
Splenomegaly (%)			28 (45.9%)
Splenic infarction (%)			3 (5.0%)
Hypersplenism (%)			2 (3.3%)

with gastrointestinal bleeding, with two (40%) individuals experiencing both melena and hematemesis. One patient got a splenectomy, while another had a splenectomy paired with paraesophagogastric devascularization. The remaining three patients underwent conservative medical care.

Features and Distribution of Varices

The gastric fundal body and the splenic hilum were the most commonly detected collateral vessels on CECT (figure 1). Three (3/61, 5.0%) patients presented with combined gastric and lower esophageal varices. Moreover, gastroepiploic varices (43/61, 70.5%), gastric short varices (40/61, 65.6%), gastric coronary varices (22/61, 36.1%), and gastric fundal plexu varices (23/61, 37.7%) were found in patients with SPH (table 2).

During hospitalization, 34 patients with SPH underwent endoscopy. 18 (29.5%) cases of isolated varices were found in these patients (figure 2), and only one patient experienced both esophageal and gastric varices (table 3).

Portosplenomesenteric Veins and Their Association with SPH

SPH was associated with the patency of portosplenomesenteric veins, with the splenic vein being the most common abnormal vein.

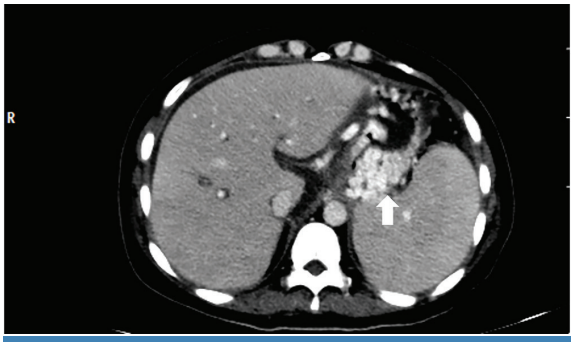

Figure 1: Contrast-enhanced computed tomography (CECT) image demonstrated splenomegaly and fundic varices.

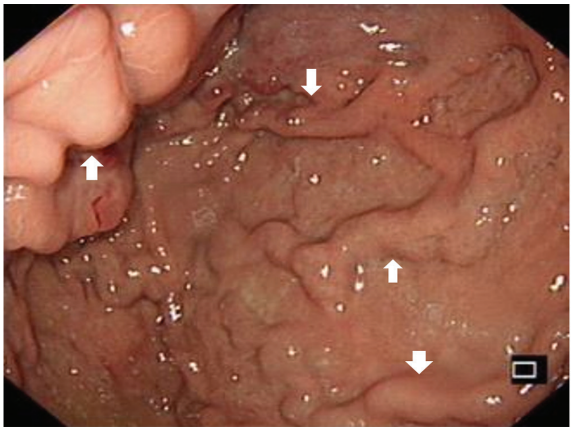

Figure 2: The varices at the fundus of the stomach were as shown on gastrointestinal endoscopy.

Table 3: Endoscopic material during hospitalization

Variables	Endoscopic material	Sinistral portal hypertension
	No varices (%)	15 (24.6%)
	Esophageal varices (%)	0 (0%)
	Gastric varices (%)	18 (29.5%)
	Esophageal varices+Gastric varices (%)	1 (1.6%)

In this retrospective study, patients with SPH were complicated with stenosis (41, 67.2%) and occlusion (4, 6.6%) of splenic vein, as well as splenic venous thrombosis (8, 13.1%). Abnormalities were also detected in the portal vein (stenosis, 29.5%; occlusion, 1.6%; thrombosis, 13.1%) and superior mesenteric vein (stenosis, 16.4%; occlusion, 1.6%; thrombosis, 5.0%) in SPH patients (table 2, figure 3).

Discussion

In the present study, acute pancreatitis was the most frequently observed pathology (80.3%, 49/61) leading to SPH, which was consistent with the findings of previous studies.[3, 7, 9] The splenic vein was vulnerable to pancreatic lesions due to its close anatomical course along the upper surface of the pancreas. Pancreatic necrosis was primarily located in the body-tail of the pancreas, where the splenic veins were more vulnerable to chronic inflammation and compression.[7] Blood flow through the splenic vein might be blocked due to thrombosis formation, injury from persistent pancreatic inflammation, or a nearby mass effect.[1, 2, 10] It causes venous hypertension, which is transmitted to the left side of the portal system through the anastomoses between the splenic vein and the gastric vein, or gastroepiploic vein.[3, 5, 9]

Yu and others demonstrated that SPH might be confined to either the splenic venous or superior mesenteric branch or might involve the whole splenomesentericoportal axis. Moreover, they reported that occlusion and stenosis of the splenic vein were independent risk factors for SPH.[5] The present study similarly showed that SPH might be associated with the patency of portosplenomesenteric veins. In patients with thrombosis or narrowing of the splenic vein, the blood flows through the splenic vein is blocked, which could lead to SPH manifesting as venous hypertension in collateral pathways such as the gastroepiploic vein, the short gastric vein, the coronary vein, and veins located in the upper half of the stomach.[1, 2, 5] The present study found that the gastroepiploic varices (70.5%) were most common in patients with SPH, followed by the short gastric vein varices (65.6%) on CECT. This effect might be explained by anatomical factors, as the gastroepiploic and short gastric veins are

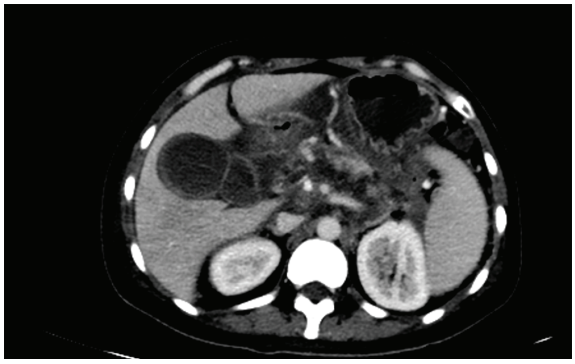

Figure 3: The splenic vein was indistinct on contrast-enhanced computed tomography (CECT).

closer to the splenic helium than other gastric veins. Patients with SPH primarily had collateral pathways that mainly occurred through the gastroepiploic vein into the superior mesenteric vein. However, some patients had collateral pathways that led from the gastric coronary vein into the portal vein.[1, 2, 11] Our findings were consistent with previous research.[3, 5] SPH causes mainly isolated fundic varices and mostly without lower esophageal varices. The combination of gastric and esophageal varices occurs only when the gastric coronary vein drains distally in the splenic vein obstruction. It differs from the pathogenesis and treatment of cirrhosis-related portal hypertension. In portal hypertension secondary to hepatic cirrhosis, gastric and esophageal varices are the most prevalent collateral veins, and they mostly flow into the source veins via the splenic and portal veins.[3, 12]

SPH is a rare cause of gastrointestinal bleeding and has the characteristics of splenomegaly, isolated gastric varices, and normal hepatic function. It was reported that many patients with SPH were asymptomatic or had unspecific abdominal pain.[1-3] In patients with SPH, splenomegaly, a sign of chronic portal hypertension, is frequently seen. Although its exact mechanisms are not fully understood, they are associated with venous congestion and increased splenic arterial flow.[1, 2] The incidence of gastrointestinal bleeding was reported to be variable, ranging from 1.92%-18%.[4, 8, 13] In a previous study with a relatively long follow-up period, 3.8% of 53 patients experienced bleeding.[13] In the present study, 18 (29.5%) patients developed gastric

fundal vein varices, and only one patient had esophageal and gastric varices on endoscopy. Five patients (8.2%) developed gastrointestinal variceal bleeding presenting mostly with melena. One patient underwent splenectomy, and one took splenectomy combined with paraesophagogastric devascularization. The other three patients received conservative medical treatment. The bleeding was controlled efficiently in all of these patients. The present guidelines recommend endoscopic therapy as the first-line treatment for bleeding gastric varices. Endoscopic therapeutic options for gastric variceal bleeding include band ligation, cyanoacrylate (CY A) injection, and thrombin.[1, 14] Moreover, management of SPH involves surgical correction of the underlying causes, such as pancreatic neoplasms or cysts, combined with splenectomy to reduce the arterial flow into the left portal system. Surgery should be performed as soon as possible in patients with active bleeding and those who did not respond to conservative treatment. Splenectomy is the treatment of choice.[15] Removal of the spleen decreases venous outflow through the collateral circulation and decompresses the associated varices to prevent further hemorrhage.[5, 16, 17] Splenic arterial embolization could be used as a pretreatment to splenectomy to reduce blood flow to the spleen and might be attempted in children who are not candidates for splenectomy or stent implantation.[10, 16, 18] The management of asymptomatic patients is more controversial than that of symptomatic ones. While some recommended splenectomy as a preventative approach, others did not report any significant benefit of this procedure in survival.[1, 16, 19] However, more evidence suggested that cautious waiting was a reasonable course of treatment for asymptomatic individuals.[2, 15, 16, 18, 20]

The present study had several limitations. First, it was a single-center study with a relatively small sample size. Second, the study did not include all follow-up data, which made it impossible to observe the dynamic changes for SPH. Third, since the present study was retrospective, care should be taken in interpreting the results, and they should be validated in prospective studies.

Conclusion

SPH was associated with the patency of the splenic vein and the formation of distinctive perigastric collateral veins. Surgery and /or endoscopic treatment were recommended, especially for patients who had numerous episodes of gastrointestinal bleeding and did not respond to conservative treatment.

Authors' Contribution

J.Zh: Data collection and assembly, data curation, formal analysis, and drafting; L.G; Mm.D: Study concept, supervision, review & editing; All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Conflicts of Interest: None declared.

References

1 Pereira P, Peixoto A. Left-Sided Portal Hypertension: A Clinical Challenge. GE Port J Gastroenterol. 2015;22:231-3. doi: 10.1016/j.jpge.2015.10.001. PubMed PMID: 28868414; PubMed Central PMCID: PMCPMC5579977.

2 Fernandes A, Almeida N, Ferreira AM, Casela A, Gomes D, Portela F, et al. Left-Sided Portal Hypertension: A Sinister Entity. GE Port J Gastroenterol. 2015;22:234-9. doi: 10.1016/j.jpge.2015.09.006. PubMed PMID: 28868415; PubMed Central PMCID: PMCPMC5579986.

3 Xie CL, Wu CQ, Chen Y, Chen TW, Xue HD, Jin ZY, et al. Sinistral Portal Hypertension in Acute Pancreatitis: A Magnetic Resonance Imaging Study. Pancreas. 2019;48:187-92. doi: 10.1097/MPA.0000000000001242. PubMed PMID: 30629031.

4 Li H, Yang Z, Tian F. Clinical Characteristics and Risk Factors for Sinistral Portal Hypertension Associated with Moderate and Severe Acute Pancreatitis: A Seven-Year Single-Center Retrospective Study. Med Sci Monit. 2019;25:5969-76. doi: 10.12659/MSM.916192. PubMed PMID: 31400275; PubMed Central PMCID: PMCPMC6699198.

5 Yu C, Ding L, Jiang M, Liao Q, Huang X, Lei Y, et al. Dynamic Changes and Nomogram Prediction for Sinistral Portal Hypertension in Moderate and Severe Acute Pancreatitis. Front Med (Lausanne). 2022;9:875263. doi: 10.3389/fmed.2022.875263. PubMed PMID: 35721067; PubMed Central PMCID: PMCPMC9198833.

6 Liu Q, Song Y, Xu X, Jin Z, Duan W, Zhou N. Management of bleeding gastric varices in patients with sinistral portal hypertension. Dig Dis Sci. 2014;59:1625-9. doi: 10.1007/s10620-014-3048-z. PubMed PMID:

24500452.
7 Ru N, He CH, Ren XL, Chen JY, Yu FF, Yan ZJ, et al. Risk factors for sinistral portal hypertension and related variceal bleeding in patients with chronic pancreatitis. J Dig Dis. 2020;21:468-74. doi: 10.1111/1751-2980.12916. PubMed PMID: 32584511.
8 Harris S, Nadkarni NA, Naina HV, Vege SS. Splanchnic vein thrombosis in acute pancreatitis: a single-center experience. Pancreas. 2013;42:1251-4. doi: 10.1097/MPA.0b013e3182968ff5. PubMed PMID: 24152951.
9 Wang YL, Zhang HW, Lin F. Computed tomography combined with gastroscopy for assessment of pancreatic segmental portal hypertension. World J Clin Cases. 2022;10:8568-77. doi: 10.12998/wjcc.v10.i24.8568. PubMed PMID: 36157801; PubMed Central PMCID: PMCPMC9453378.
10 Abraham M, Doshi S, Asfari MM, Yap JEL, Bowers HG. Isolated Gastric Variceal Hemorrhage Secondary to Idiopathic Sinistral Portal Hypertension. Cureus. 2021;13:e16165. doi: 10.7759/cureus.16165. PubMed PMID: 34367775; PubMed Central PMCID: PMCPMC8336330.
11 Pandey V, Patil M, Patel R, Chaubal A, Ingle M, Shukla A. Prevalence of splenic vein thrombosis and risk of gastrointestinal bleeding in chronic pancreatitis patients attending a tertiary hospital in western India. J Family Med Prim Care. 2019;8:818-22. doi: 10.4103/jfmpc.jfmpc_414_18. PubMed PMID: 31041207; PubMed Central PMCID: PMCPMC6482754.
12 Zhou HY, Chen TW, Zhang XM, Wang LY, Zhou L, Dong GL, et al. The diameter of the originating vein determines esophageal and gastric fundic varices in portal hypertension secondary to posthepatitic cirrhosis. Clinics (Sao Paulo). 2012;67:609-14. doi: 10.6061/clinics/2012(06)11. PubMed PMID: 22760900; PubMed Central PMCID: PMCPMC3370313.
13 Zhou J, Ke L, Yang D, Chen Y, Li G, Tong Z, et al. Predicting the clinical manifestations in necrotizing acute pancreatitis patients with splanchnic vein thrombosis. Pancreatology. 2016;16:973-8. doi: 10.1016/j.pan.2016.10.001. PubMed PMID: 27727096.
14 de Franchis R, Baveno VF. Revising consensus in portal hypertension: report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2010;53:762-8. doi: 10.1016/j.jhep.2010.06.004. PubMed PMID: 20638742.
15 Loftus JP, Nagorney DM, Ilstrup D, Kunselman AR. Sinistral portal hypertension. Splenectomy or expectant management. Ann Surg. 1993;217:35-40. doi: 10.1097/00000658-199301000-00007. PubMed PMID: 8424698; PubMed Central PMCID: PMCPMC1242731.
16 Gautam AD, Sanket, Agarwal A, Yadav RR. Emergent Management of Gastric Variceal Bleed in the Setting of Acute Pancreatitis-Related Sinistral Hypertension With Partial Splenic Embolization: A Series of Two Cases. Cureus. 2022;14:e29002. doi: 10.7759/cureus.29002. PubMed PMID: 36112972; PubMed Central PMCID: PMCPMC9463960.
17 Wang L, Liu GJ, Chen YX, Dong HP, Wang LX. Sinistral portal hypertension: clinical features and surgical treatment of chronic splenic vein occlusion. Med Princ Pract. 2012;21:20-3. doi: 10.1159/000329888. PubMed PMID: 22024761.
18 Paramythiotis D, Papavramidis TS, Giavroglou K, Potsi S, Girtovitis F, Michalopoulos A, et al. Massive variceal bleeding secondary to splenic vein thrombosis successfully treated with splenic artery embolization: a case report. J Med Case Rep. 2010;4:139. doi: 10.1186/1752-1947-4-139. PubMed PMID: 20482817; PubMed Central PMCID: PMCPMC2890017.
19 Makowiec F, Riediger H, Emmrich J, Kroger J, Hopt UT, Adam U. Prophylactic splenectomy for splenic vein thrombosis in patients undergoing resection for chronic pancreatitis. Zentralbl Chir. 2004;129:191-5. doi: 10.1055/s-2004-822782. PubMed PMID: 15237324.
20 Sarin SK, Jain AK, Jain M, Gupta R. A randomized controlled trial of cyanoacrylate versus alcohol injection in patients with isolated fundic varices. Am J Gastroenterol. 2002;97:1010-5. doi: 10.1111/j.1572-0241.2002.05622.x. PubMed PMID: 12003381.