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Case Report

Treatment Strategy for Gastric Variceal Bleeding Caused by Sinistral Portal Hypertension Based on Hemodynamic Characteristics: A Case Report

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Abstract

Gastric varices secondary to sinistral portal hypertension (SPH) are a rare cause of upper gastrointestinal (GI) hemorrhage. Endoscopic histoacryl injection is widely used for endoscopic hemostasis in portal hypertension (PH) and is considered a rescue treatment for massive acute GI bleeding. However, it is associated with a high rate of recurrence. Herein, we report a case of repeated pancreatitis-induced SPH that led to refractory gastric variceal (GV) bleeding, ultimately treated by splenectomy. From a hemodynamic perspective, this case explored treatment strategies for GV bleeding caused by SPH and provided insights for the clinical diagnosis and treatment of affected patients.

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Keywords ● Sinistral portal hypertension ● Gastric varices ● Gastrointestinal hemorrhage ● Hemodynamics ● Treatments

What's Known

- Gastric varices resulting from sinistral portal hypertension are a rare cause of upper gastrointestinal bleeding. Current treatment options include endoscopic therapy, splenectomy, and partial splenic artery embolization.

What's New

- From the perspective of hemodynamics, this case report explored the treatment strategies for gastric variceal bleeding caused by regional portal hypertension. Splenectomy eliminates the pathological condition of venous hypertension localizing the gastrosplenic region and decreases blood flow through the collateral circulation, thus immediately controlling gastric variceal bleeding.

Introduction

Sinistral portal hypertension (SPH), also referred to as regional, segmental, or left-sided portal hypertension (PH), is a pathological condition of splanchnic hypertension localized to the gastrosplenic region. It is primarily attributed to obstruction of the splenic vein outflow pathway, such as splenic vein thrombosis or external compression, which ultimately results in elevated venous pressure and the formation of collateral circulation to reroute blood from the gastrosplenic region.[1] SPH is a completely curable form of PH characterized by normal liver function, isolated gastric varices (with or without esophageal varices), and/or splenomegaly.[2] In most cases, SPH is often found incidentally during investigation. Recently, it has been reported that patients with SPH are at increased risk of life-threatening gastrointestinal (GI) hemorrhage, with reported incidence rates ranging from 4% to 17%.[1-3] Previous studies demonstrated that endoscopic histoacryl injection is the first-line treatment for gastric variceal (GV) bleeding caused by PH.[2-4] It exhibits excellent hemostatic effects and definite curative effects. For control of acute GV hemorrhage, endoscopic histoacryl injection is a very effective treatment modality. However, the efficacy of endoscopic variceal treatment is often questioned, and it may increase the risk of recurrent bleeding.[4, 5] Given the failure of endoscopic intervention to achieve long-term hemostatic control, splenectomy was ultimately performed as a definitive surgical management strategy in clinical practice.[5, 6] From a hemodynamic perspective,

this case report investigated therapeutic approaches for GV bleeding resulting from SPH, offering insights into the clinical diagnosis and management of affected patients.

Case Presentation

A 31-year-old woman was referred to the General Hospital of Western Theater Command (Chengdu, China) for evaluation and management of upper GI hemorrhage in November 2024. Approximately 10 months before admission, the patient experienced hematemesis without an identifiable cause. Endoscopy revealed that the gastric mucosa was elevated with fundal varices without active bleeding. Nine days prior to admission, the aforementioned symptoms recurred. She was discharged after conservative treatment for both episodes. Four hours before admission, the patient experienced four episodes of hematemesis with gastric contents, with an estimated total volume of approximately 450-500 mL.

Over the past decade, the patient experienced multiple episodes of recurrent pancreatitis, all of which resolved with conservative management. She had a documented history of type 2 diabetes mellitus and hyperlipidemia. There was no known family history of genetic disorders, and she denied any history of adverse lifestyle habits.

After admission, physical examination revealed findings consistent with anemia; no other significant abnormalities were detected, and there was no evidence of splenomegaly. Blood examinations revealed the following: red blood cell (RBC) count: 2.28×10^12/L, hemoglobin (Hb) concentration: 59 g/L, platelet (Pl) count: 138×10^9/L, and triglyceride level: 3.2 mmol/L.

Admission hepatitis serology, tumor marker levels, and coagulation profiles were negative. Emergency gastroscopy confirmed the presence of multiple varices with associated bloodstains located in the fundus and body of the stomach (figure 1). Abdominal computed tomography (CT) venography revealed multiple tortuous and thickened vascular structures within the perigastric region, consistent with varicose veins.

Blood transfusion, acid suppression, vasoconstriction, fluid replacement, and blood glucose control were also initiated. On the 2nd day after admission, the patient received an endoscopic injection of tissue adhesive (B·Braun, Germany) for the gastric fundus varices (figure 2). On the 5th day after admission, the patient developed a fever with a peak temperature of 39.1 °C, which resolved following anti-infective therapy and physical cooling. Serial monitoring revealed rising hemoglobin levels, normalization of stool color, and no evidence of active bleeding. On the 8th day of hospitalization, 320-slice CT portal venography revealed poor visualization of the splenic vein and mild stenosis of the portal vein in the region adjacent to the superior mesenteric vein. In addition, tortuous and dilated vessels were observed in the hepatogastric space, gastric fundus, splenic hilum, and abdomen, suggesting the presence of potential collateral circulation (figure 3).

On the 9th day of hospitalization, portal vein puncture angiography was performed to further evaluate the anatomical configuration of the portal vein branches, the existence of gastric fundal varices, and the condition of associated collateral circulation. During the procedure, the guidewire could not be advanced into the splenic vein branches; therefore, embolization of collateral circulation was not carried out.

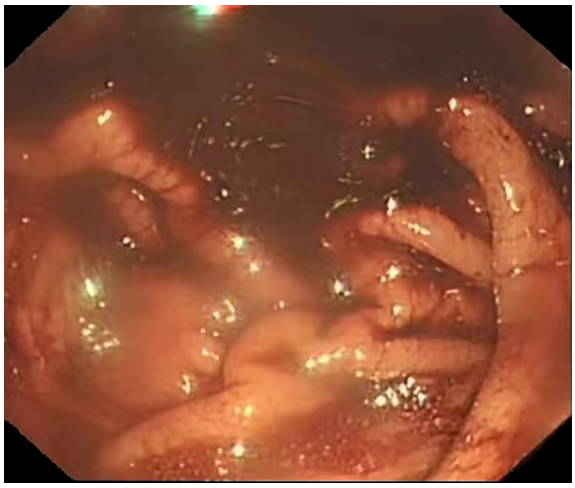

Figure 1: Gastroscopy confirms the presence of gastric varices with evidence of active hemorrhage.

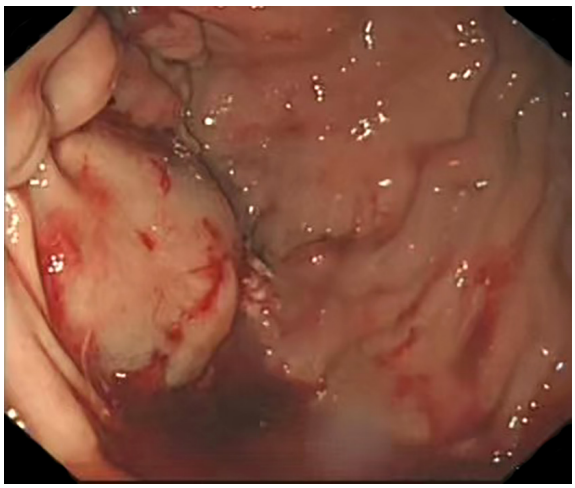

Figure 2: Endoscopic injection of tissue adhesive was performed to achieve hemostasis for gastric variceal bleeding.

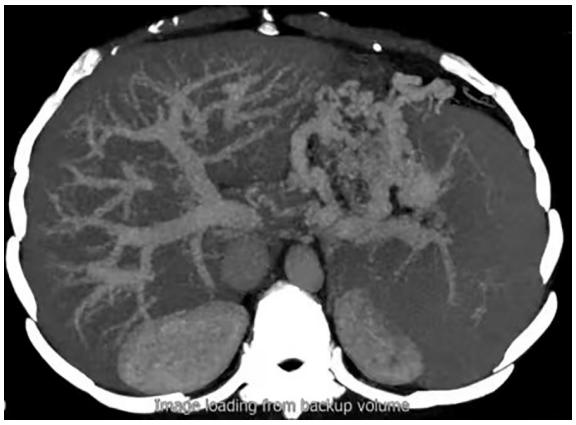

Figure 3: The 320-slice computed tomography (CT) portal venography reveals dilated, tortuous vessels in the hepatogastric space, gastric fundus, splenic hilum, and abdominal cavity, indicating collateral circulation.

Intraoperative portal venous pressure, measured using a pressure transducer, was found to be normal at approximately 15 cmH_2O (normal value: 13-24 cmH_2O). Accordingly, placement of a portal vein stent was not indicated.

On the 10th day of hospitalization, the patient presented with hematemesis, expelling approximately 100 mL of dark-red gastric contents. Blood pressure was recorded at 85/50 mmHg, consistent with hypotension. An urgent hemoglobin measurement revealed a level of 55 g/L. Given the failure of endoscopic histoacryl injection to achieve hemostasis, laparoscopic splenectomy was performed in the surgical department. Intraoperative findings revealed mild splenomegaly with tortuous and dilated vessels at the splenic hilum. The pancreas exhibited a firm parenchymal texture, suggestive of chronic inflammatory changes. Postoperative histopathological examination confirmed chronic congestive splenomegaly (figure 4). No episodes of recurrent bleeding were observed during the postoperative period, and hemoglobin levels showed a progressive increase compared to preoperative values. Following confirmation

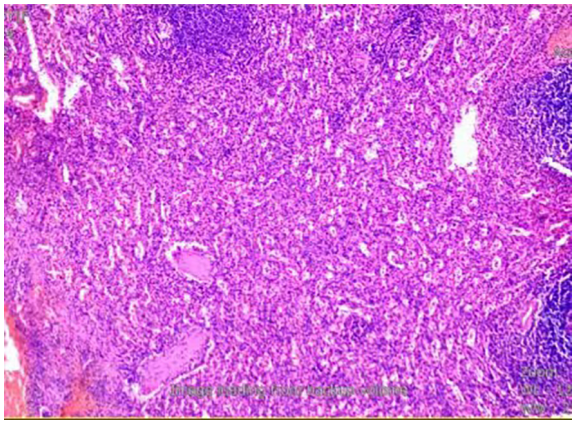

Figure 4: Postoperative biopsy confirms chronic congestive splenomegaly.

of clinical stability, the patient was discharged. We concluded that the patient's GV hemorrhage was secondary to chronic pancreatitis–induced regional (pancreatogenic) portal hypertension (PH). During the 6-month follow-up period, no further episodes of gastrointestinal bleeding were reported.

Written informed consent was obtained from the patient prior to publication, including consent for the use of clinical data and any accompanying images.

Discussion

The splenic vein is a relatively large, non-tortuous vessel formed by five or six tributaries from the spleen. It courses inferior to the splenic artery and, following its exit from the splenic hilum, runs posterior to the body and tail of the pancreas.[1-3] Anatomically, the close spatial relationship between the splenic vein and the posterior aspect of the pancreatic tail predisposes to a high incidence of splenic vein obstruction in the setting of pancreatic disorders, while the splenic artery remains largely unaffected.[3] Injury from persistent pancreatic inflammation and external compression from pancreatic necrosis or pseudocyst leads to stenosis, thrombosis, or occlusion of the splenic vein. Pancreatic disease obstructs splenic vein flow, and the resulting increase in pressure within the splenic venous system is transmitted through the anastomoses between the splenic vein and the gastric or gastroepiploic veins, leading to gastric varices.[4-6] In contrast to generalized PH, liver function tests and portal vein pressure were within normal limits in patients with SPH.[3, 4] The expected flow direction in the portal vein is hepatopetal.

Under normal physiological conditions, the splenic artery constitutes the principal arterial supply to the spleen. Blood enters the spleen via the splenic hilum and ultimately drains into the portal vein through the splenic vein. Blood from the gastrosplenic region drains into the splenic hilum through both the short gastric veins and posterior gastric veins, flowing into the portal vein together with the splenic vein. Occlusion of the splenic vein with concomitant uninterrupted blood flow through the splenic artery leads to increased pressure in the splenic venous system. Under these circumstances, to preserve splenic perfusion, the body compensates by dilating and hypertrophying the splenic artery, thereby increasing arterial blood flow.[4, 7] Thus, a left-sided malignant hyperdynamic circulation is established, further increasing pressure in the splenic hilum region. When the pressure within the splenic hilar region exceeds that in

the gastric fundal venous system, retrograde blood flow develops through both short gastric and posterior gastric veins into the gastric fundus, ultimately contributing to the formation of gastric varices.[4-6, 8] When the pressure in the gastric fundus vein exceeds that in the portal vein, the gastric coronary vein dilates, and blood refluxes into the portal vein through this vessel, resulting in portal vein enlargement. Compared with gastric fundal varices caused by PH due to liver cirrhosis, those caused by SPH have higher blood flow pressure and relatively fewer shunt channels. Consequently, the varicose veins associated with SPH exhibit rapid progression and substantial bleeding volumes.

Current guidelines recommend endoscopic therapy as first-line treatment for bleeding gastric varices.[8] Endoscopic therapeutic options for gastric variceal bleeding include band ligation, cyanoacrylate (CYA) injection, and thrombin. The strongest clinical evidence supports the use of CYA injection, which is recommended as first-line endoscopic therapy in both the American Association for the Study of Liver Diseases guidelines and the Baveno V consensus.[7, 9] For control of acute gastric variceal bleeding, endoscopic CYA glue injection is a highly effective therapeutic modality. Some studies demonstrated that endoscopic injection of CYA was an effective and safe treatment for GV bleeding and obliteration, particularly in localized cases.[9, 10] Indeed, the efficacy of endoscopic variceal treatment is often questioned and may increase the risk of recurrent bleeding since it can increase the pressure of the collaterals after blocking the outflow tract of splenic blood.[7, 9-11] A study comparing endoscopic tissue adhesive injection for bleeding from gastric fundal varices ruptured due to PH showed that both short-term and long-term treatment outcomes in patients with SPH were significantly worse than those in patients with cirrhosis-induced PH.[7] Patients with SPH have complicated bleeding GVs, and the risk of death is relatively higher when recurrent GV hemorrhage occurs. This finding was associated with the malignant hypermetabolic hemodynamic cycle of SPH, as mentioned previously.[4, 11] Given that the blood supply from the splenic artery continuously increases, the pressure within the gastric fundal varices rises accordingly. SPH is characterized by a relatively short duration, fewer collateral circulations, and limited shunt pathways.[7] Endoscopic injection of tissue adhesive effectively occludes the primary shunt pathways in these patients, thereby leading to a subsequent increase in pressure within the splenic hilar region. Consequently, the hemostatic efficacy of the procedure was significantly compromised. It is exclusively indicated for life-threatening hemorrhage secondary to rupture of gastric fundal varices caused by SPH.[10, 11] Furthermore, conventional endoscopic NBC injections might lead to severe complications, including renal or pulmonary thromboembolism, fever, intense pain caused by intraperitoneal injection, mucosal necrosis at the injection site, and GI bleeding.[8-10, 12] Despite its numerous limitations, endoscopic tissue adhesive therapy enables rapid hemostasis and is therefore retained as a first-line therapeutic option for gastric fundal variceal bleeding. However, this is typically a temporary measure. In cases of suboptimal therapeutic efficacy, further interventions targeting the splenic artery and vein should be considered based on the underlying etiology and hemodynamic alterations to achieve curative intent.[9-11]

The occurrence of variceal bleeding secondary to splenic vein thrombosis is a potentially life-threatening event. Splenectomy is a traditional management for GI hemorrhage from SPH. Splenectomy eliminates the venous hypertension localized to the gastrosplenic region and decreases collateral blood flow, thus immediately controlling GV bleeding.[8, 10, 11] Splenectomy is the treatment of choice for symptomatic patients suffering from recurrent variceal hemorrhage. In such patients, splenectomy corrects potentially life-threatening bleeding, reduces risks related to multiple blood transfusions, prevents hypersplenism, and might reduce or eliminate future bleeding episodes.[10, 11] Postoperatively, regular monitoring of platelet dynamics is indicated to prevent thrombosis, with simultaneous emphasis on infection prophylaxis. For patients who are not surgical candidates, partial splenic artery embolization might be considered as an alternative to reduce splenic arterial inflow and ameliorate PH.[13] However, this procedure might be associated with potential complications such as splenic infarction, splenic hematoma, and ectopic embolization.[13, 14] In addition, since the portal pressure and liver function are often normal in these patients, portosystemic shunting should not be considered in SPH.[1, 6, 8]

Regrettably, subsequent follow-up was not possible as the patient was lost to follow-up in the later stage.

Conclusion

SPH complicated by GI bleeding is a complex and clinically challenging condition. Although various therapeutic options are available, they require individualized treatment strategies. A

comprehensive assessment of the patient's underlying pancreatic pathology, overall clinical status, and hemodynamic changes in the portal venous system is essential for guiding the selection of the most appropriate therapeutic strategy.

Authors' Contribution

J.Z**:** Collection and assembly of data, data curation, formal analysis, writing the original draft. S.T and J.Z: Conceptualization, supervision, review, and 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.

Declaration of AI

The authors declare that no artificial intelligence (AI) tools were used in the preparation of this manuscript.

Conflict of Interest: None declared.

References

1 Zheng K, Guo X, Feng J, Bai Z, Shao X, Yi F, et al. Gastrointestinal Bleeding due to Pancreatic Disease-Related Portal Hypertension. Gastroenterol Res Pract. 2020;2020:3825186. doi: 10.1155/2020/3825186. PubMed PMID: 32308674; PubMed Central PMCID: PMC7140141.

2 Zheng J, Luo G, Deng MM. Clinical Analysis of Sinistral Portal Hypertension. Iran J Med Sci. 2024;49:487-93. doi: 10.30476/ijms.2023.99570.3164. PubMed PMID: 39205819; PubMed Central PMCID: PMC11347591.

3 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.

4 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: PMC6482754.

5 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.

6 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: PMC6699198.

7 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.

8 Liu M, Wei N, Song Y. Splenectomy versus non-splenectomy for gastrointestinal bleeding from left-sided portal hypertension: a systematic review and meta-analysis. Ther Adv Gastroenterol. 2024;17:17562848241234501. doi: 10.1177/17562848241234501. PubMed PMID: 38450104; PubMed Central PMCID: PMC10916482.

9 Yang J, Zeng Y, Zhang JW. Modified endoscopic ultrasound-guided selective N-butyl-2-cyanoacrylate injections for gastric variceal hemorrhage in left-sided portal hypertension: A case report. World J Clin Cases. 2022;10:6254-60. doi: 10.12998/wjcc.v10.i18.6254. PubMed PMID: 35949826; PubMed Central PMCID: PMC9254179.

10 Patrono D, Benvenga R, Moro F, Rossato D, Romagnoli R, Salizzoni M. Left-sided portal hypertension: Successful management by laparoscopic splenectomy following splenic artery embolization. Int J Surg Case Rep. 2014;5:652-5. doi: 10.1016/j.ijscr.2014.03.010. PubMed PMID: 25194596; PubMed Central PMCID: PMC4189059.

11 Tang SH, Zeng WZ, He QW, Qin JP, Wu XL, Wang T, et al. Repeated pancreatitis-induced splenic vein thrombosis leads to intractable gastric variceal bleeding: A case report and review. World J Clin Cases. 2015;3:920-5. doi: 10.12998/wjcc.v3.i10.920. PubMed PMID: 26488031; PubMed Central PMCID: PMC4607813.

12 Spaander MC, Darwish Murad S, van Buuren HR, Hansen BE, Kuipers EJ, Janssen HL. Endoscopic treatment of esophagogastric variceal bleeding in patients with noncirrhotic extrahepatic portal vein thrombosis: a long-term follow-up study. Gastrointest Endosc. 2008;67:821-7. doi: 10.1016/j.

gie.2007.08.023. PubMed PMID: 18206153.
13 Liu J, Meng J, Yang M, Zhou C, Yang C, Huang S, et al. Two-step complete splenic artery embolization for the management of symptomatic sinistral portal hypertension. Scand J Gastroenterol. 2022;57:78-84. doi: 10.1080/00365521.2021.1983641. PubMed PMID: 34581650.
14 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: PMC9463960.