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CASE REPORT

Repeated pancreatitis-induced splenic vein thrombosis leads to intractable gastric variceal bleeding: A case report and review

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Abstract

Gastric varices (GV) are one of the most common complications for patients with portal hypertension. Currently, histoacryl injection is recommended as the initial treatment for bleeding of GV, and this injection has been confirmed to be highly effective for most patients in many studies. However, this treatment might be ineffective for some types of GV, such as splenic vein thrombosis-related localized portal hypertension (also called left-sided, sinistral, or regional portal hypertension). Herein, we report a case of repeated pancreatitis-induced complete splenic vein thrombosis that led to intractable gastric variceal bleeding, which was treated by splenectomy. We present detailed radiological and pathological data and blood rheology analysis (the splenic artery - after a short gastric vein or stomach vein - gastric coronary vein - portal vein). The pathophysiology can be explained by the abnormal direction of blood flow in this patient. To our knowledge, this is the first reported case for which detailed pathology and blood rheology data are available.

Key words: Splenic vein thrombosis; Intractable gastric variceal bleeding; Recurrent pancreatitis; Review

Core tip: Here, we report a case in which chronic

pancreatitis-induced complete splenic vein thrombosis led to intractable gastric variceal bleeding, which is effectly treated by splenectomy. We have provided details regarding the imaging and pathology data, and we describe the hemodynamic characteristics. Then, we reviewed the disease onset and treatment methods, which may provide a reference for the clinical diagnosis and treatment of similar patients.

Tang SH, Zeng WZ, He QW, Qin JP, Wu XL, Wang T, Wang Z, He X, Zhou XL, Fan QS, Jiang MD. Repeated pancreatitis-induced splenic vein thrombosis leads to intractable gastric variceal bleeding: A case report and review. *World J Clin Cases* 2015; 3(10): 920-925 Available from: URL: http://www.wjgnet.com/2307-8960/full/v3/i10/920.htm DOI: http://dx.doi.org/10.12998/wjcc.v3.i10.920

INTRODUCTION

Gastric varices (GV) are among the most common complications affecting patients with portal hypertension, which has a mortality rate that can reach as high as 20% within 6 wk[1]. Currently, histoacryl injection is recommended as the initial treatment for bleeding GV, and this approach has been confirmed to be highly effective for most patients in many studies[2-5]. However, this treatment might be ineffective for some types of GV, such as splenic vein thrombosis-related localized portal hypertension (also called left-sided sinistral or regional portal hypertension). Herein, we report a case of recurrent pancreatitis-induced complete splenic vein thrombosis that led to intractable gastric variceal bleeding, which was treated by splenectomy. We present detailed radiological and pathological data and blood rheology analysis results (splenic artery - after a short gastric vein or stomach vein - gastric coronary vein - portal vein). The pathophysiology can be explained by the abnormal direction of blood flow in this patient. To our knowledge, this is the first reported case for which detailed pathology and blood rheology data are available.

CASE REPORT

A 58-year-old man was admitted to our hospital due to recurrent melena lasting for over a month and vomiting lasting for two hours. His past history revealed a history of heavy drinking of at least 200 g daily that exceeded 30 years; however, approximately 7 years before, his alcohol consumption had decreased. Over the past 7 years, he had experienced recurrent pancreatitis five times, and all incidences resolved. Approximately one month prior to admission, this patient began to experience melena with no obvious cause. Endoscopy showed that the gastric mucosa was elevated with fundal varices without active bleeding. After conservative treatment, the melena became intermittent. Then, another endoscopic examination revealed severe GV, and the patient received five histoacryl injections. Subsequently, he experienced intermittent melena and vomited approximately 200 mL of blood. Physical examination showed anemia, splenomegaly spanning three ribs across the liver, and active bowel sounds (7/min). Blood examinations revealed the following: Red blood cell, 2.98×10^{12}/L; hemoglobin concentration, 67 g/L; and platelet count, 90×10^{9}/L. Both liver and kidney functions were normal. Abdominal enhanced computed tomography (CT) showed cirrhosis and an enlarged portal vein.

The patient was diagnosed with alcoholic cirrhosis, portal hypertension, splenomegaly and GV. Then, emergency endoscopy revealed bleeding GV, and a second histoacryl injection treatment was performed. However, this patient was also experiencing intermittent vomiting, which had become more frequent because the histoacryl injection did not effectively stop the bleeding from fundus varices. Emergency transjugular intrahepatic portosystemic shunt placement was performed as a hemostatic treatment. Portal vein puncture was successful, and portal vein radiography showed an enlarged portal vein; however, the splenic vein and gastric coronary vein were not imaged (Figure 1). Then, another abdominal enhanced CT and portal systemic vascular reconstruction were performed. The enhanced CT scan revealed an enlarged portal vein from the origin of the gastric coronary vein and an enlarged and circuitous gastric coronary vein (Figure 2A). The splenic vein did not show any flow signals in the portal venous phase (Figure 2B). The portal systemic vascular reconstruction image did not show the splenic vein or spleen signals. These data indicated that the intractable gastric variceal bleeding was not induced by alcoholic cirrhosis or portal hypertension but rather by regional portal hypertension promoted by complete splenic vein thrombosis after recurrent pancreatitis.

Taking into account the poor general condition of the patient, splenic artery embolization could have led to serious complications. Therefore, laparotomy was performed for splenectomy. After opening the abdomen, normal liver size, color and texture were observed. During surgery, we found adhesions of the spleen to organs and tissues, such as the stomach, transverse colon and kidney. Approximately two hours were spent separating the extensive adhesions.

After separation of the surrounding tissues and ligation of the splenic artery and short gastric vessels, we successfully removed the spleen and found that the pancreas was very hard to the touch. After anatomical resection of the spleen, we found that the splenic vein was completely blocked by thrombosis (Figure 3A), and the pathology results further confirmed splenic vein thrombosis (Figure 3B). One month after splenectomy, endoscopic examination revealed that the fundal varices had markedly reduced, and ultrasound examination revealed a normal-sized portal vein.

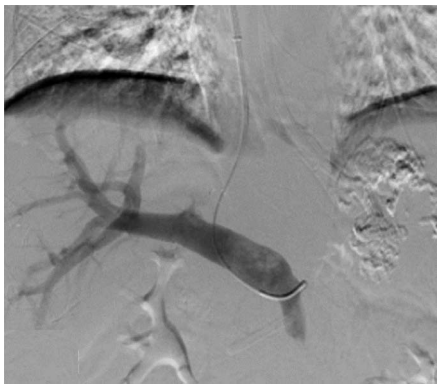

Figure 1 Direct portal vein radiography shows an enlarged portal vein; however, the splenic vein and gastric coronary vein could not be imaged.

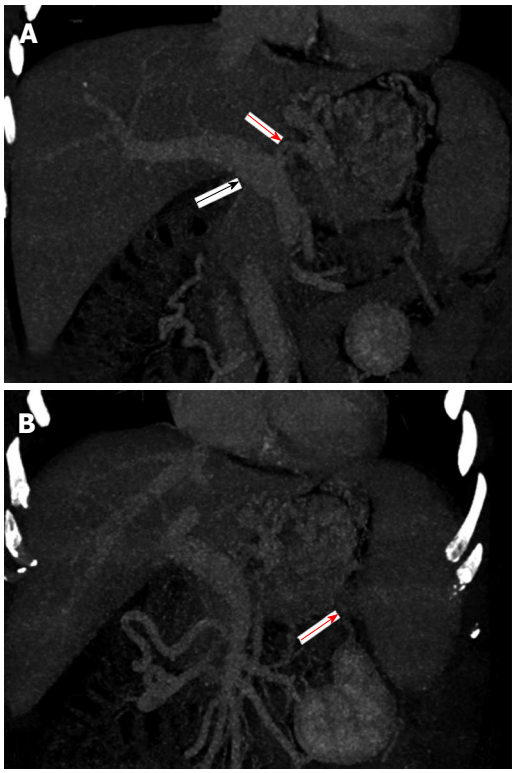

Figure 2 Enhanced computed tomography scan. A: An enlarged portal vein from the origin of the gastric coronary vein and an enlarged, circuitous gastric coronary vein; B: Splenic vein flow signals in the portal venous phase are absent.

DISCUSSION

Gastric variceal bleeding due to regional portal hypertension with splenic vein thrombosis is a severe, life-threatening condition, which is very difficult to control[6]. Patients with splenic vein thrombosis-induced GV, who usually have normal hepatic function, are unlike those with generalized portal hypertension[7], and their mortality risk is higher than that of patients with variceal hemorrhage due to other causes[6,8]. A previous study has shown that as many as 37 different specific etiologies lead to splenic vein thrombosis[9], the most common of which is pancreatitis[10]. The rate of splenic vein thrombosis is 7% to 20% in patients who have previously suffered from pancreatitis[11]. Splenic vein thrombosis induced by pancreatitis was first reported by Hirschfeldt[12]. Other causes of this disease include myeloproliferative neoplasm[13,14], gastrointestinal, pancreatic and hepatobiliary cancers, liver cirrhosis[15], abdominal compression and vibration[16], pancreatic exocrine cancer[17], factors secondary to splenic metastatic cancer[18], minimally invasive distal pancreatectomy[19], and splenic laceration[20].

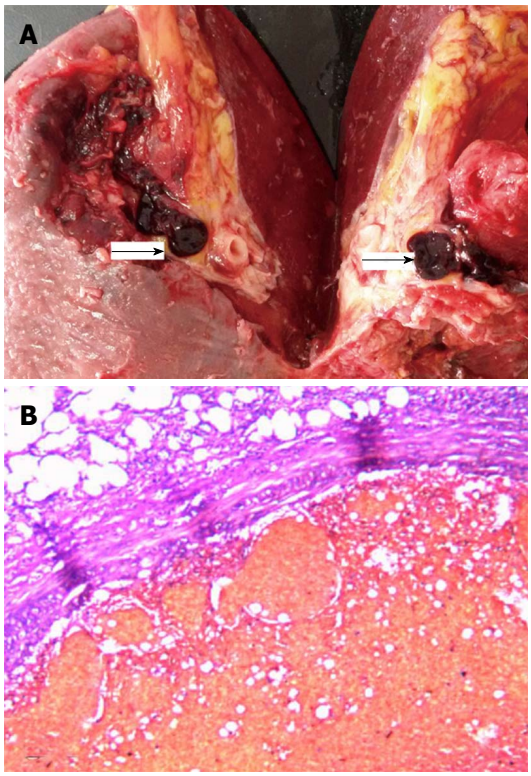

Figure 3 Anatomical resection of the spleen and hematoxylin-eosin staining staining. A: The splenic vein is completely blocked by thrombosis; B: The splenic vein is completely filled by thrombosis.

The splenic vein originates in a large and non-tortuous vessel from the spleen, lies inferior to the splenic artery, and runs behind the pancreatic body and tail. Therefore, the splenic vein endothelium can be damaged by inflammation in the nearby pancreatitis, which can induce splenic vein thrombosis and obstruction. Since the first report of splenic vein thrombosis induced by pancreatitis in 1920[12], five types of pancreatitis have been identified, including chronic, acute, familial, traumatic and autoimmune pancreatitis (Table 1), the most common of which is chronic pancreatitis[11,21-24]. Recently, we have reported a patient with chronic pancreatitis-associated splenic vein thrombosis caused by regional portal hypertension who was treated by partial splenic artery embolization[25]. Acute pancreatitis has been reported to be another common cause of splenic vein thrombosis[23,26,27]. In addition, familial[28], traumatic[29] and autoimmune[30] pancreatitis-induced splenic vein thrombosis and GV have been reported.

Table 1 Etiologies of pancreatitis-induced splenic vein thrombosis

Chronic pancreatitis
Longstreth *et al*[21], 1971
Little *et al*[22], 1981
Moossa *et al*[23], 1985
Bernades *et al*[24], 1992
Heider *et al*[11], 2004
Tang *et al*[25], 2015
Acute pancreatitis
Moossa *et al*[23], 1985
Madsen *et al*[26], 1986
Rogers *et al*[27], 1989
Familial pancreatitis
McElroy *et al*[28], 1972
Traumatic pancreatitis
Salam *et al*[29], 1973
Autoimmune pancreatitis
Ishikawa *et al*[30], 2012

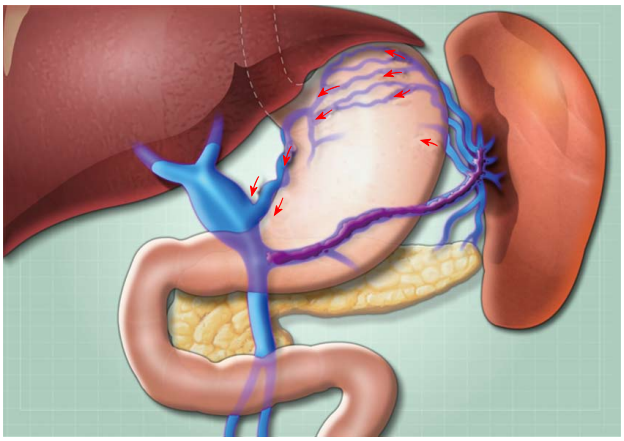

Figure 4 A schematic diagram of the pathophysiological and blood flow changes in this patient.

Herein, we report a case of pancreatitis-induced complete splenic vein thrombosis that led to intractable gastric variceal bleeding. This patient was first misdiagnosed with alcoholic cirrhosis-induced portal hypertension. After direct portal venography and portal vein reconstruction, the patient was finally diagnosed with regional portal hypertension induced by complete splenic vein thrombosis after pancreatitis. Normally, blood flows through the splenic artery and short gastric vein from the fundus back to the portal vein. After passing through the spleen, blood flows through the splenic vein[31]. However, when the splenic vein is completely blocked, splenic artery blood cannot flow back through the splenic vein, which causes the spleen to become congested and enlarged. Blood must reflux to the gastric fundus vein through the short gastric vein, which results in a significant increase in gastric fundus pressure, varices, and reflux to the vena cava through the stomach, the renal vein shunt and other branches. When the pressure of the gastric fundus vein is higher than that of the portal vein, the gastric coronary vein will become enlarged, and blood will reflux to the portal vein through the gastric coronary vein, inducing portal vein enlargement (Figure 4). Therefore, these blood rheology findings explain all of the symptoms, signs, laboratory test results and imaging data of the patient.

Antithrombotic therapy has been recommended for venous thromboembolic disease[32-34]. An institutional (Mayo clinic) database search has revealed that a total of 2454 patients were diagnosed with acute pancreatitis from January 1996 to December 2006, with splenic vein thrombosis noted in 45 (1.8%) patients, and the use of oral anticoagulation was considered to be reasonably safe in these patients[35]. However, for chronic pancreatitis, the incidence of splenic vein thrombosis can reach 20% to 40%[36-38]. For complete splenic vein thrombosis patients, antithrombotic therapy may aggravate the risk of bleeding due to fundal varices. Therefore, splenic artery embolization is one of the best treatments for bleeding GV induced by splenic vein thrombosis[14,39-42]. However, "post-embolization syndrome" is a common side effect experienced after splenic artery embolization and includes abdominal pain, fever, vomiting, and purulent infection depending on the arterial embolism size and the patient's condition. Another study has suggested that transjugular endovascular recanalization of the splenic vein is a safe and effective therapeutic option in patients with regional portal hypertension and is not associated with an increased risk of procedure-related complications[43]. As the condition of the patient in the present report was poor due to massive blood loss, we chose splenectomy *via* laparotomy, which was successful.

This paper describes a case of chronic pancreatitis-induced complete splenic vein thrombosis, which led to intractable gastric variceal bleeding. We have provided details regarding the imaging and pathology data and have described the hemodynamic characteristics. In addition, we have reviewed the disease onset and treatment methods, which may provide a reference for the clinical diagnosis and treatment of similar patients.

COMMENTS

Case characteristics

A 58-year-old man with recurrent melena lasting for over a month and vomiting lasting for 2 h.

Clinical diagnosis

Chronic pancreatitis-induced complete splenic vein thrombosis led to intractable gastric variceal bleeding.

Laboratory diagnosis

Red blood cell, 2.98 × 10^{12}/L; hemoglobin concentration, 67 g/L; and platelet count, 90 × 10^{9} /L.

Imaging diagnosis

Enhanced computed tomography scan revealed an enlarged portal vein from the origin of the gastric coronary vein and an enlarged and circuitous gastric coronary vein. The splenic vein did not show any flow signals in the portal venous phase.

Treatment
Laparotomy was performed for splenectomy.

Experiences and lessons
This is the first reported case for which detailed pathology and blood rheology data are available.

Peer-review
A very interesting paper.

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