Isolated gastric varices secondary to a pancreatic pseudocyst. A case report

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Case Report

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Background: It is described a case of isolated gastric varices secondary to a pancreatic pseudocyst in a 27-year-old female with a history of acute pancreatitis. The purpose of this study is the docmentation of a rare complication of extrahepatic portal hypertension, caused by obstruction of the splenic vein due to a pancreatic pseudocyst.

The patient presented hematemesis and melena, indicative of upper gastrointestinal bleeding. Diagnostic tests: including liver ultrasound and contrast-enhanced computed tomography, revealed left portal hypertension, splenomegaly, and collateral circulation in the form of gastric varices. Isolated varices were confirmed by endoscopy in the gastric fundus. During initial management, octreotide was administered, successfully controlling the bleeding without further complications. This case underscores that complications of pancreatitis, such as pseudocysts, can lead to splenic thrombosis, segmental portal hypertension, and the development of gastric varices. Splenectomy, in cases of bleeding varices associated with splenic obstruction, is the definitive treatment to prevent recurrence.

Key words: Gastric varices, Pancreatic pseudocyst.

Gastric varices (GV) are abnormally dilated venous vessels located within the gastric wall, resulting from increased blood flow and elevated pressure in the portal venous system. Although traditionally recognized in the context of portal hypertension secondary to liver cirrhosis, GV can also be observed in non-cirrhotic patients. In such cases, the development of GV is primarily attributed to thrombosis of the splenoportal venous axis. This thrombosis disrupts normal venous drainage, leading to localized hypertension and collateral formation within the gastric circulation.

Among the most common causes of splenoportal venous thrombosis are pancreatic pathologies, including chronic pancreatitis, acute pancreatitis, pancreatic pseudocysts, and pancreatic neoplasms. These conditions often result in extrinsic compression or direct invasion of the splenic or portal veins, ultimately leading to obstruction and thrombosis. The clinical implications of GV in non-cirrhotic patients differ significantly from those in cirrhotic individuals, necessitating distinct diagnostic approaches and management strategies.

The Sarin classification is the most widely used endoscopic classification system for gastric varices (Figure 1). They are divided into gastroesophageal varices, which extend from esophageal varices, and isolated gastric varices. Gastroesophageal varices are subdivided into GOV1 75% of cases and extend inferiorly along the lesser curvature, and GOV2 21% of cases and extend superiorly to the cardia and fondus. GOV1 has a vascular supply similar to that of esophageal varices, arising from the left gastric vein, whereas GOV2 has a vascular supply from the posterior gastric veins and short gastric veins. Isolated gastric varices are subdivided into IGV1 (less than 2%), located in the cardia and fondus, and IGV2 (4%), located in the gastric body and antrum. IGV1 behaves similarly to GOV2, whereas IGV2 is more often associated with splenic vein thrombosis and non-cirrhotic portal hypertension (1-2).

The Saad-Caldwell classification organizes GVs according to their afferent and efferent vasculature. Saad-Caldwell type 1 varices are located in the cardia and derive their blood supply from the left gastric vein (called the right-sided portal circulation) and correlate with GOV1. Type 2 varices are cardiofundus varices that derive their blood supply from the posterior gastric veins and short gastric veins (called the left-sided portal circulation) and correlate with IGV1 or GOV2. Type 3 varices are cardiofundus varices that derive their blood supply from the left gastric vein, as well as the posterior gastric veins and short gastric veins, and correlate with GOV2 or IGV1. Type 4 varices are similar to types 2 and 3 varices, but with the presence of splenic vein thrombosis. These groups are subdivided according to the absence of a draining gastrorenal shunt (types 1a, 2a, 3a, 4a) or its presence (types 1b, 2b, 3b, 4b) (1-2).

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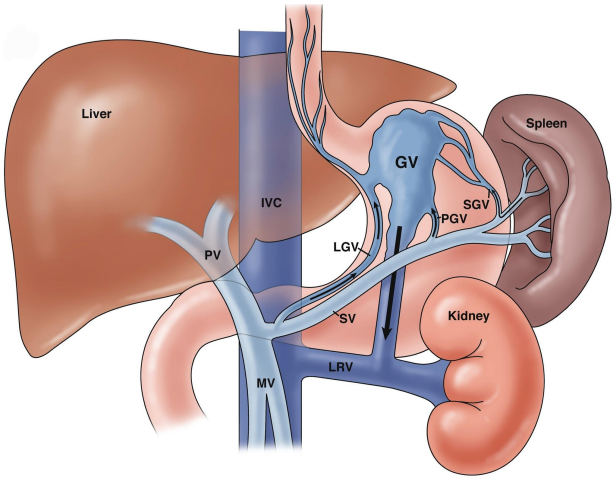

Figure 1. Vascular anatomy of gastric varices (6). GV, gastric varices; IVC, inferior vena cava; LGV, left gastric vein; LRV, left renal vein; MV, mesenteric vein; PGV, posterior gastric vein; PV, portal vein; SGV, short gastric veins; SV, splenic vein.

Patients with uncomplicated varices do not present symptoms related to the varices themselves. The main associated complication is ruptura causing generally severe gastrointestinal bleeding manifested as bloody vomiting. The estimated risks of bleeding at one year, three years, and five years after diagnosis is 16%, 36%, and 44%, respectively. Mortality associated with bleeding from gastric varices is higher than with bleeding from esophageal varices, and can reach levels of up to 30%. This condition is ussually diagnosed by upper gastrointestinal endoscopy, although varices can also be detected on radiological tests, such as computed tomography or magnetic resonance imaging. Endoscopic ultrasound with Doppler imaging can be very useful for differential diagnosis between varices and other non-venous pathologies of the stomach wall (1-2).

Initial management of hemorrhage due to GV includes general supportive and resuscitative measures, and treatment with vasoactive drugs such as somatostatin or terlipressin. Treatment options for the acute episode are diverse; It us posible to distinguish between direct methods, using endoscopy or endoscopic ultrasound, and indirect methods such as transjugular intrahepatic portosystemic shunt (TIPS) or other interventional radiology techniques. Currently, the recommended treatment is endoscopic injection of cyanoacrylate, and in cases of treatment failure, TIPS is used (1).

Case report

A 27-year-old female patient with a history of acute pancreatitis 5 years prior and a complication of a pancreatic pseudocyst in the tail portion of the pancreas 1 year prior. An endoscopy was performed 8 months ago for upper gastrointestinal bleeding, which revealed no esophageal varices but isolated gastric varices (IGV1) (Figure 2). One month prior, presented upper gastrointestinal bleeding with hematemesis and melena. A diagnostic approach was performed with an ultrasound of the liver and biliary tract, revealing normal liver dimensions with no nodular or cystic lesions, no intra- or extrahepatic biliary dilation, and surgical absence of a gallbladder. Changes in the splenoportal axis suggested portal hypertension, a 16-mm portal vein with slow hepatopetal flow, and the presence of collateral circulation. The patient had moderate splenomegaly, a spleen measuring 158 mm longitudinally, and an enlarged spleen of 8 mm. In addition, a simple and contrast-enhanced computed tomography of the abdomen was requested, which reported 2 cystic images at the head level of 18 and 28

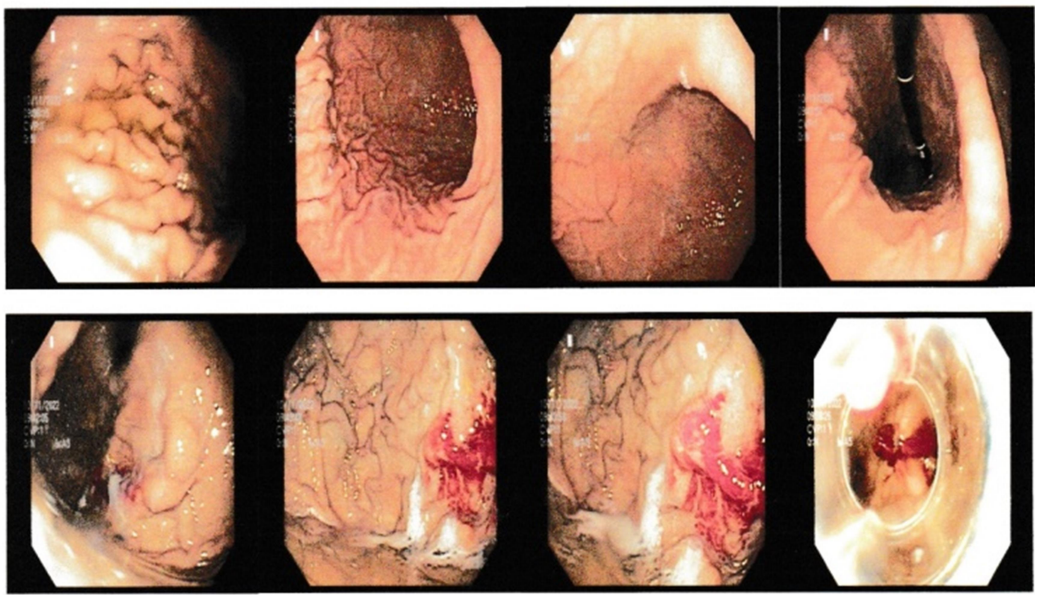

Figure 2. Upper endoscopy: The patient's isolated gastric varices with traces of bleeding can be seen.

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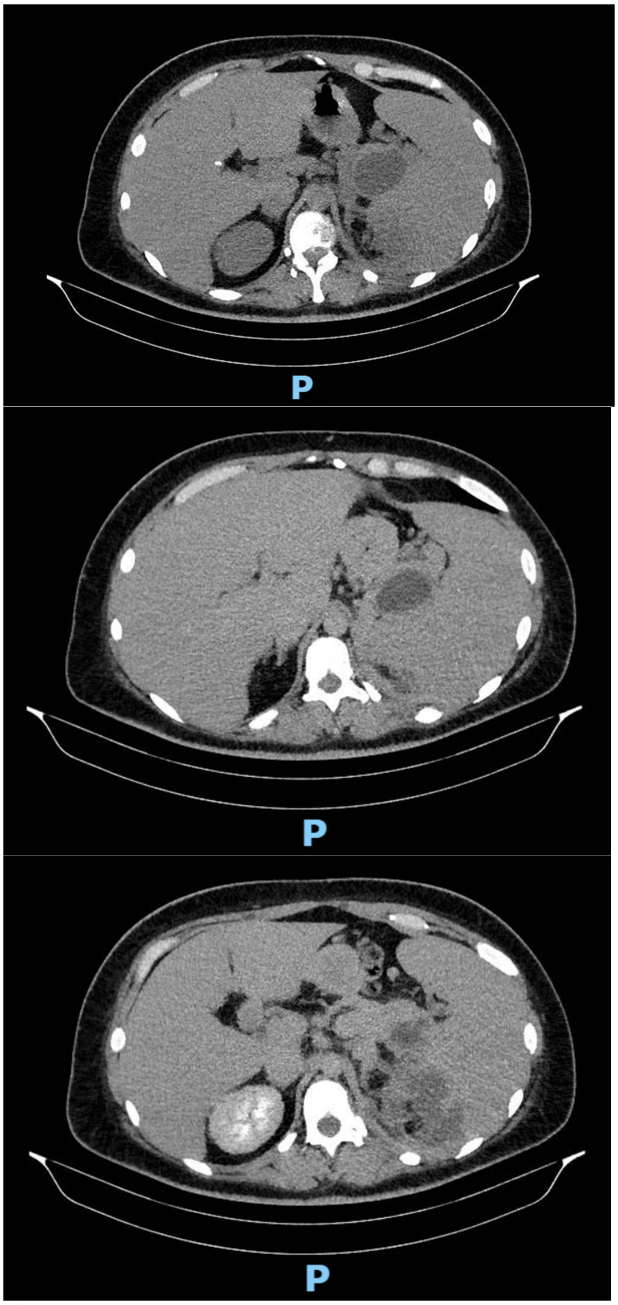

Figure 3. Plain and contrast-enhanced computed tomography of the abdomen: splenomegaly and pancreatic pseudocyst can be seen.

mm, and at the tail level 57 mm. The spleen was enlarged and measured 160 mm in relation to the splenomegaly, and the liver was normal. Reinforcement of the gastric mucosa was seen at the level of the greater curvature to rule out an inflammatory process at this level. Multiple varicose gastro-splenic venous structures were observed. During his stay, he was treated with octreotide with satisfactory results and no other data on bleeding.

Discussion

Although rare, splenic vein obstruction represents a distinct and clinically relevant cause of extrahepatic portal hypertension, accounting for approximately 5% of its cases. In turn, extrahepatic portal hypertension comprises about 5% of all cases of portal hypertension. Emerging evidence highlights the role of complications from acute pancreatitis, such as pancreatic pseudocysts, in the pathogenesis of splenic vein thrombosis and the development of segmental or left-sided portal hypertension. In these cases, obstruction of the splenic vein impedes normal venous outflow from the spleen, leading to redirection of blood through low-pressure collateral pathways. These collateral vessels include the short gastric veins, which drain into the coronary vein, and the left gastroepiploic vein, which empties into the superior mesenteric vein. The resulting increase in flow through the short gastric veins leads to localized venous hypertension and the subsequent formation of gastric varices, particularly along the greater curvature and the fundus of the stomach. This pathophysiological mechanism underscores the importance of recognizing pancreatic disease and splenic vein thrombosis as potential non-cirrhotic causes of gastric varices and left-sided portal hypertension.

Conclusion

Patient who, following an episode of acute pancreatitis, developed a pancreatic pseudocyst compressing the splenic veins, causing splenomegaly with the development of collateral circulation in the form of gastric varices, evidenced by endoscopy in the fundus secondary to left portal hypertension. Two years after the endoscopy, the patient developed an episode of gastrointestinal bleeding, which resolved with octreotide.

The evaluation of a non-liver-positive patient with gastrointestinal bleeding due to varices should be aimed at diagnosing possible causes of extrahepatic portal hypertension.

In patients with isolated splenic vein obstruction and bleeding varices, the treatment of choice is splenectomy. Removal of the spleen reduces venous flow from the collaterals, thus preventing further bleeding. It provides a cure rate in over 90%.

Conflicts of interests

The authors have no financial or personal relationships that could inappropriately influence or bias the content of this article.

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