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Left-sided portal hypertension revisited

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ABSTRACT

Background: Splenic vein obstruction can lead to left-sided portal hypertension, which is a rare segmental portal hypertension condition, often caused by inflammatory or neoplastic disease of the pancreas. Today, adequate study by cross-sectional imaging, e.g., ultrasound and computed tomography, allows the identification of particular features of the venous collateral pathways that favor the development of gastroesophageal varices.

Methods: A retrospective study of 15 cases of left-sided portal hypertension secondary to complete thrombosis of the splenic vein was performed, with special attention to the morphodynamic conditions predicting the development of gastroesophageal varices.

Results: In patients with left-sided portal hypertension, gastroesophageal varices were greatly favored by two conditions: collateral pathways directed to the gastric fundus and hypertension in the left gastric vein. This last condition typically occurs when the left gastric vein inflows into an already obstructed splenic trunk, or in the case of concomitant portal hypertension. On the contrary, patients with left-sided portal hypertension and collaterals connected with the left renal or adrenal veins have minor risk of gastroesophageal varices.

Conclusions: In every case of left-sided portal hypertension, upper digestive endoscopy and close follow-up are recommended. Besides, computed tomography can demonstrate particular conditions directly favoring gastroesophageal varices, and aid in selection of the appropriate therapeutic decisions.

Key words: *Left-sided portal hypertension, splenic vein thrombosis, portal hypertension*

Introduction

Left-sided portal hypertension (LSPH) is a particular hemodynamic condition involving the region of the splenic vein. This rare syndrome can be considered a typical example of segmental portal hypertension and it is often secondary to neoplastic or inflammatory pathology of the pancreas.

Thrombosis of the splenic vein and subsequent development of collateral systems are the two distinctive features of LSPH.

Today, the renewed interest in LSPH has been promoted by modern cross-sectional imaging tools, e.g., color Doppler ultrasound (US) and contrast-enhanced thin section (2.5 mm) angio computed tomography (CT), which allow precise study of the entire portal venous system [1-4]. The aim of our retrospective study was to analyze the different morphodynamic and clinical aspects of this disease.

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Anatomo-functional features

Several pathophysiological and clinical aspects of LSPH are correlated with the anatomy of the splenic venous system.

The splenic vein is the main channel of venous drainage of the spleen and the upper gastric greater curvature. Important collaterals stem from its proximal portion, close to the splenic hilum: the short gastric veins, linked to the venous apparatus of the stomach and the left gastroepiploic vein, connect with the portal vein. Other spleen venous collaterals are represented by the splenorenal and splenoadrenal veins, which join with the inferior vena cava system.

The middle portion of the splenic vein receives other tributaries: the pancreatic collaterals, and sometimes, in an anatomical variant, the left gastric vein. The inferior mesenteric vein usually inflows to the most distal part of the splenic vein and it is rarely involved in the collateral pathways.

Patients and Methods

We reviewed radiological images and clinical reports of 15 cases of LSPH observed between 2009 and 2013.

The inclusion criteria were: age between 25 and 65 years; normal liver biochemical tests, including transaminases, and cholestasis and hepatic synthetic function indices; and careful morphological study of the abdomen by US and CT proving complete obstruction of the splenic vein, but normal morphology of the liver and the portomesenteric venous trunk.

The exclusion criteria were: only partial obstruction of the splenic vein; pre-existing diseases of the liver, spleen, portomesenteric trunk, upper digestive tract other than the pancreas, retroperitoneum and retroperitoneal organs; and any previous abdominal surgery.

We selected a group of 15 cases comprising 8 men and 7 women aged 47–65 years. In these patients, splenic vein thrombosis was secondary to chronic pancreatitis (4 cases), pancreatic pseudocyst (3 cases), pancreatic body cancer (4 cases) and primary prothrombotic state (4 cases).

Results

Different morphodynamic conditions could be identified.

In 9 cases (60%), the splenic venous outflow was

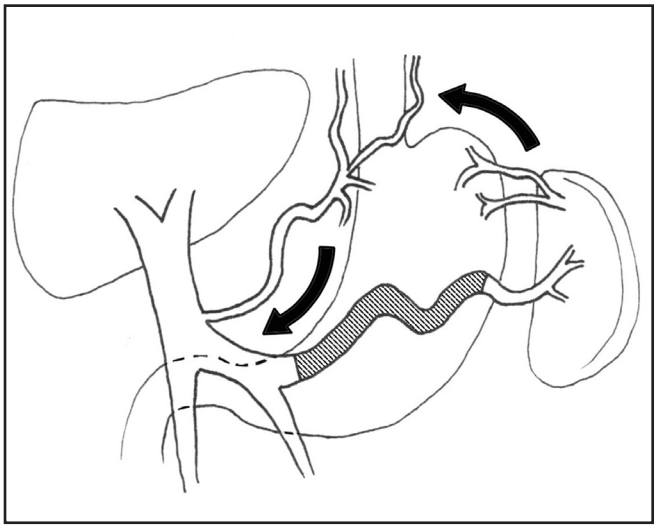

Figure 1. The short gastric veins direct the splenic outflow toward the gastric fundus; the left gastric vein supplies adequate drainage into the portal trunk.

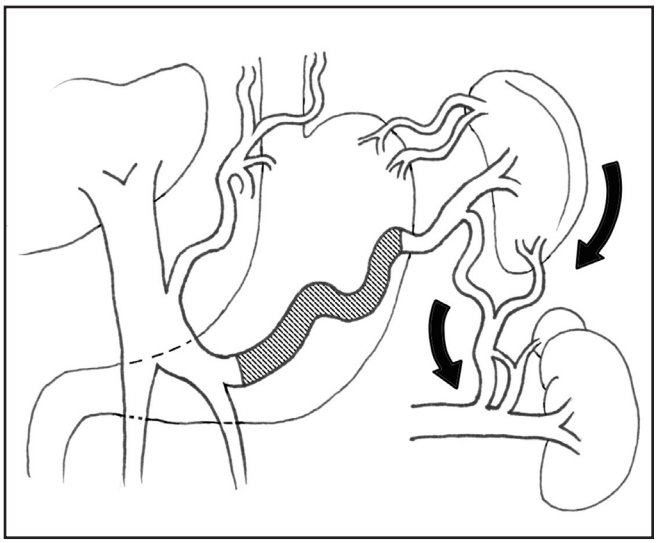

Figure 2. The splenic venous outflow is mainly directed to the left adrenal and renal veins.

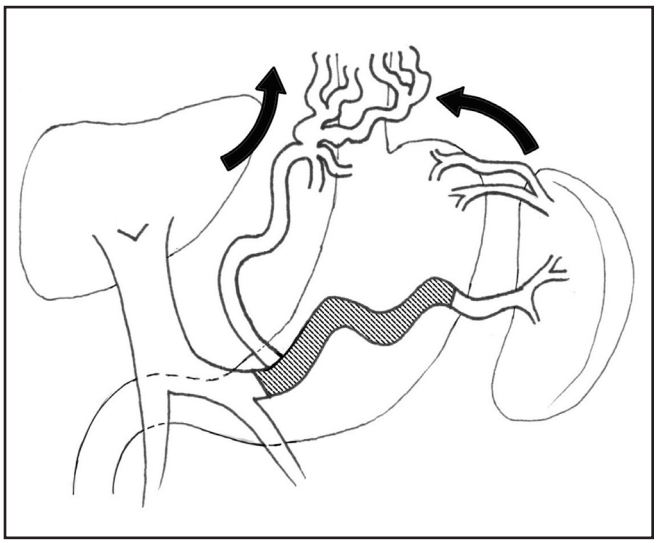

Figure 3. The left gastric vein, flowing into an obstructed splenic vein, is congested, and it favors the development of gastroesophageal varices.

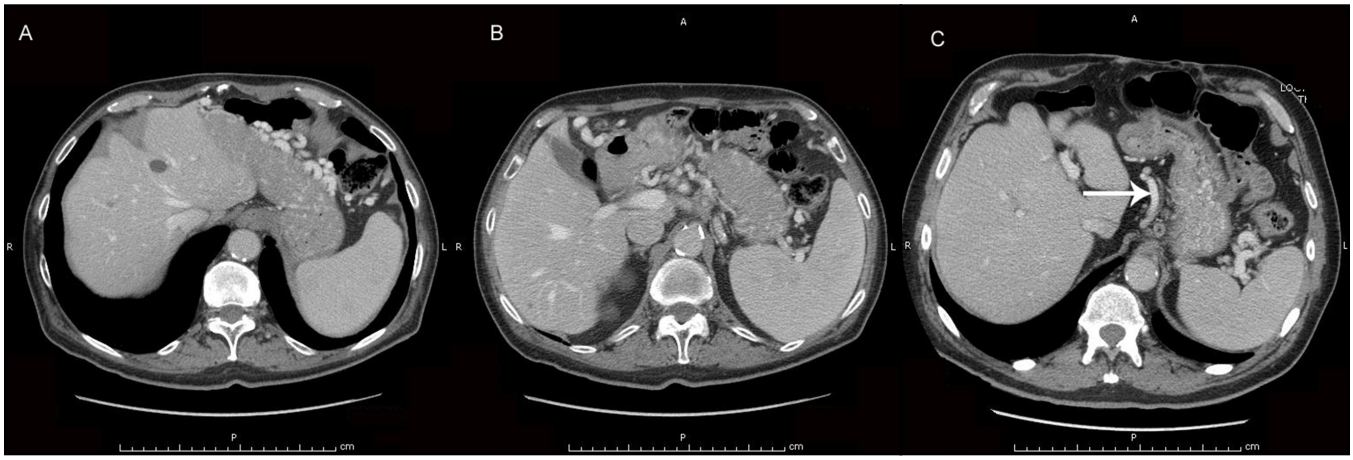

Figure 4. CT scans demonstrating different gastric venous collateral pathways. **A:** Congestion of the gastroepiploic veins; **B:** Retrogastric venous collaterals; **C:** A dilated left gastric vein (arrow).

directed toward the gastric fundus by the short gastric veins, and then to the portal system through the left gastric vein (Figure 1). There was no association with splenomegaly or gastroesophageal varices, confirming hemodynamic equilibrium. The primary disease was chronic pancreatitis in 3 cases, pancreatic pseudocysts in 2 cases, tumor of the pancreatic body in 2 cases and prothrombotic state with thrombosis of the femoroiliac venous trunks in 2 cases.

In 3 patients (20%), 1 with chronic pancreatitis, 1 with pancreatic pseudocysts, and 1 with prothrombotic state, the collateral venous system involved the splenorenal or splenoadrenal collaterals, but not the gastroesophageal compartment. There was no association with gastroesophageal varices (Figure 2).

In the remaining 3 cases (20%), 2 with pancreatic body cancer and 1 with pro-thrombotic state, the collateral pathways developed toward the gastric fundus through the short gastric veins. The left gastric vein, inflowing in an anatomical variant into a completely thrombosed splenic trunk, appeared congested and surrounded by many collaterals, blocking adequate venous drainage from the stomach. In this last group, we found LSPH was associated with mild splenomegaly, a mean volume increase of 20% and gastroesophageal varices, as demonstrated by CT and by upper digestive endoscopy (Figure 3).

In all cases, the diagnosis of LSPH was incidental during study of the underlying disease, considering mild splenomegaly can remain asymptomatic.

We observed no dilatation of the inferior mesenteric vein or ectopic abdominal varices.

Discussion

We merely included gastric and esophageal varices as a unique complication of LSPH, although recognizing that isolated gastric varices are typical of this disease [2].

CT allows detailed study of the entire portal venous system, including the splenic vein and its collaterals, with the only limitation being the thinner branches. Therefore, if an obstructed splenic vein is discovered, adequate study of its collaterals by cross-sectional imaging is essential (Figure 4) [5,6]. US does not have an equivalent value to CT, as results are sometimes influenced by transient hemodynamic conditions and interobserver variations.

The splenic vein receives its main blood supply from the spleen and collaterals located in its proximal portion. Therefore, thrombosis, although initial but interrupting the blood flow, easily spreads along the vein by the mechanism of upstream congestion and downstream stasis.

A direct equivalence relation between complete obstruction of the splenic vein and secondary gastroesophageal varices cannot always be verified because of the different features of the collateral pathway. We determined two main anatomo-functional conditions that prevent the development of gastroesophageal varices.

First, the splenic venous outflow is directed through the short gastric veins toward the gastric fundus, then arrives at the portal system through the left gastric vein (Figure 1).

Second, the main collateral pathway of the splenic vein develops toward the renal and adrenal veins, linked

with the inferior vena cava system (Figure 2). This last condition is favored by adhesions between the spleen and the retroperitoneum and by splenomegaly.

On the contrary, the onset of gastroesophageal varices is facilitated by a collateral splenic pathway directed towards the gastric fundus and by its subsequent congestion. This typically happens when the left gastric vein inflows, in an anatomical variant, into an obstructed splenic vein, or when there is association with a portal hypertension condition (Figure 3) [7-11]. This last hemodynamic situation can be more correctly recognized as a complication of generalized portal hypertension.

We must remember that the spleen collaterals can also involve mainly the gastroepiploic and transverse colonic veins [12,13]. This condition, not observed in our series, is more common after pancreaticoduodenectomy and spleen-preserving pancreatectomy when performed with splenic vein ligation. It can also cause early postoperative bleeding [14-20].

The abovementioned anatomo-functional algorithms do not exempt patients, in the case of demonstrated LSPH, from upper digestive endoscopy and close follow-up [8-14].

Moreover, in patients with bleeding gastroesophageal varices, especially without signs of liver disease, LSPH has to be considered in the differential diagnosis, and a CT study is mandatory [21].

Conclusions

LSPH is a clinical syndrome with different aspects and possible complications, especially gastroesophageal varices. Currently, in absence of specific clinical signs, it can also be easily diagnosed by radiological imaging, and it can complicate any abdominal surgery [22-24].

In the case of LSPH, different therapeutic strategies are possible today: splenic artery embolization, splenectomy and splenectomy plus gastroesophageal devascularization. Detailed study of the hemodynamic conditions associated with this syndrome is necessary for appropriate treatment.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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