

POSTTRAUMATIC LEFT-SIDED PORTAL HYPERTENSION MANIFESTED WITH BLEEDING FUNDAL VARICES

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POSTTRAUMATIC LEFT-SIDED PORTAL HYPERTENSION MANIFESTED WITH BLEEDING FUNDAL VARICES (Abstract): Splenic vein obstruction (usually due to thrombosis) induces left-sided portal hypertension and isolated fundal varices in patients with unaffected liver parenchyma and permeable portal vein. Pancreatic etiology is among the most frequent for splenic vein thrombosis. Hemorrhage from isolated fundal varices is a life-threatening situation and an unusual endoscopic finding. Isolated splenic vein thrombosis with gastric varices is rare and represents one of the few curable syndromes inducing portal hypertension. The treatment for this situation is controversial, various options being described in the literature: endoscopic injection hemostasis, interventional radiology techniques and surgery. We herein describe the clinical case of a 50-year old patient with bleeding fundal varices, which proved to be attributable to a blunt abdominal trauma thirty six years previously, successfully managed by stapling fundectomy with splenectomy.

KEY WORDS: ISOLATED FUNDAL VARICES; HEMORRHAGE; FUNDECTOMY; SPLENECTOMY

SHORT TITLE: Hemorrhagic fundal varices

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INTRODUCTION

Hemorrhagic isolated gastric varices (IGV) are a life-threatening complication induced by isolated splenic vein thrombosis (ISVT) and represent an unusual endoscopic finding. The leading etiology for the sinister portal hypertension (SPH) is the pancreatic diseases [1]; less common causes are renal diseases, iatrogenic splenic vein lesions, blunt abdominal trauma [2-4].

ISVT must be considered in all the patients with upper gastrointestinal bleeding and normal liver function, permeable portal vein and splenomegaly. This is one of the few curable syndromes inducing portal hypertension.

We describe herein a case admitted in our department with bleeding IGV

successfully managed by stapling fundectomy with splenectomy.

CASE PRESENTATION

A 50-year-old male patient referred to our department for massive upper gastrointestinal bleeding. The patient did not have any past medical history except for a blunt abdominal trauma from a motorcycle accident thirty six years ago managed non-operatively. Physical exam including lung, heart, and liver was unrevealing; the heart rate was 98/minute and blood pressure was 100/60 mmHg. No splenomegaly was detected. His hemoglobin level was 66 g/L, red blood cells of 2.4×10^{12} /L, Ht of 22%; white blood cell count was 9.3×10^9 /L and platelet count was 250×10^9 /L. Biochemical

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tests (including liver enzymes) were normal except for a minor hypoproteinemia.

An upper gastrointestinal endoscopy revealed no esophageal varices and isolated bleeding gastric varices IGV, considered as F3 according to Sarin SK classification [4], covered with a huge blood clot. Since endoscopic mini-loop hemostasis was unfeasible in this patient, surgical management was decided. During surgery huge subserosal fundal varices and a 1.5 cm in diameter short gastric vein were observed (Fig. 1) and no liver cirrhosis or pancreatic pathology.

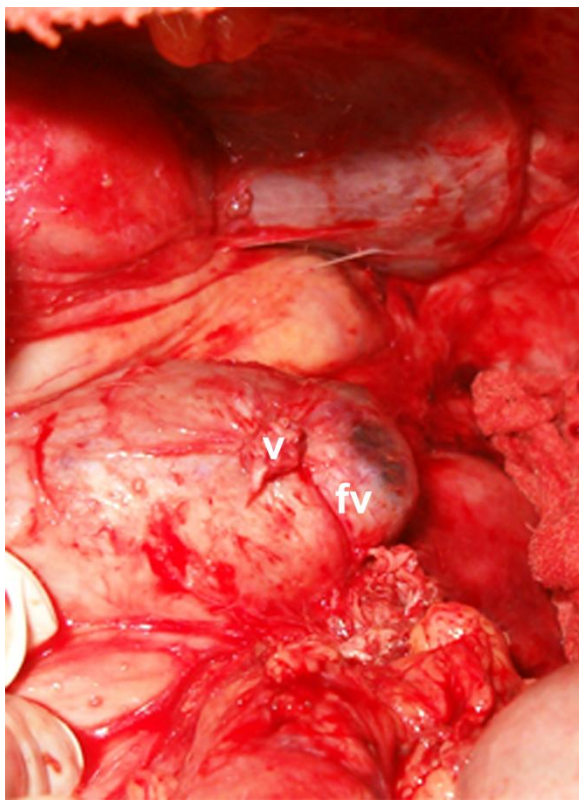


Fig. 1 Subserosal fundal varices (fv) and ligated short gastric vein (v)

DISCUSSION

Isolated impairment of the blood flow through the splenic vein, usually caused by thrombosis, induces SPH. Initially described by Greenwald and Wasch in 1939 [5] this condition is defined as localized portal hypertension manifested with gastric varices, permeable portal vein and normal liver function tests [3]. The incidence of left-sided portal hypertension is less than 5% of all the

Devascularisation on the greater gastric curvature, splenectomy and stapling fundectomy using a Proximate® linear stapler 60 (Ethicon Endo-surgery inc. USA) have been performed (Fig. 2).

Microscopic examination of the resected specimen revealed submucosal and subserosal located gastric varices.

The postoperative follow-up was uneventful and the patient discharged 11 days after surgery. During a 4 month follow-up period the patient is free of disease recurrence.

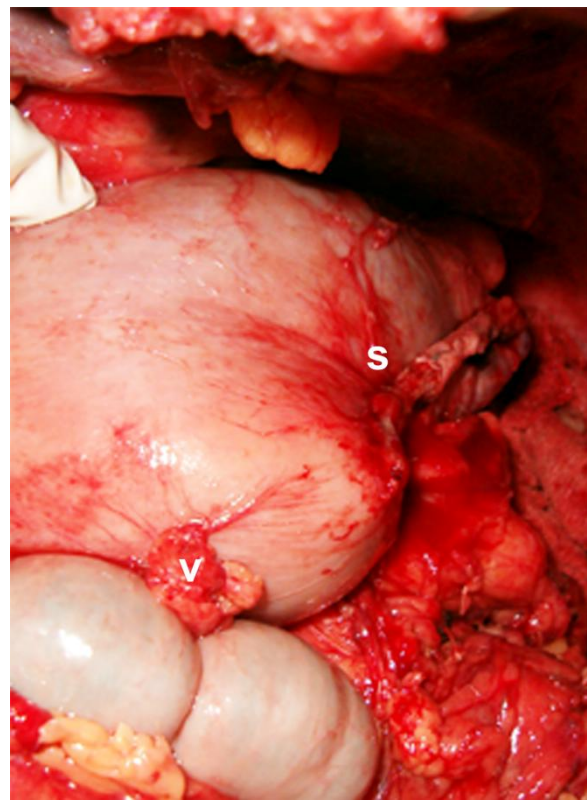


Fig. 2 Stapling line after fundectomy (s) and ligated short gastric vein (v)

patients with portal hypertension, up to date being described about 450 cases [3,6]. The main etiologies are pancreatic disorders (acute or chronic pancreatitis as well as pancreatic neoplasms) [6,7]. Various disorders other than pancreatic diseases can cause ISVT: umbilical vein catheterization; partial gastrectomy; distal splenorenal shunt; splenectomy; selective venous catheterization; metastatic carcinoma; lymphoma; retroperitoneal liposarcoma;

renal cancer; gastric cancer; colon cancer; retroperitoneal fibrosis; splenic artery aneurysms; gastric ulcer; hepatportal sclerosis; hereditary thrombocytopenia; myeloproliferative disorders; protein S deficiency; systemic lupus erythematosus; renal abscess; tuberculous adenitis; retroperitoneal abscess; benign renal cysts [8,9]. However, these are rare and have a wide spectrum in terms of their mechanisms of splenic vein obstruction. Due to the fact of blunt abdominal trauma history, as well as lack of other possible etiologies for gastric varices we assumed that the most possible etiological factor for SPH as a result of splenic vein thrombosis in our patient was the blunt abdominal trauma.

The pathophysiologic mechanism of venous thrombosis in intraabdominal veins after trauma is multifactorial; the most commonly suspected mechanisms are endothelial injury of the venous wall secondary to shear force and portal system venous stasis secondary to compression by perivenous hematoma [10].

Gastrointestinal bleeding associated with ISVT is due to varices that usually develop in short gastric and left gastroepiploic veins [1].

Most commonly, SPH is asymptomatic and is diagnosed incidentally. In symptomatic cases, the first clinical manifestation of SPH is generally acute or chronic upper gastrointestinal bleeding usually from a ruptured gastric varix, and rarely from esophageal or colonic varices [11].

There are many options for the nonoperative treatment of gastric variceal bleeding, such as, balloon-occluded retrograde transvenous obliteration (BRTO), endoscopic detachable snare, endoscopic band ligation and endoscopic sclerotherapy using cyanoacrylate glue [12-15]. However, bleeding control as well as preventing recurrent hemorrhage with these methods is difficult, thus some authors advocate surgical treatment to be the gold-standard procedure for bleeding fundal varices: paraesophageal perigastric devascularization

with fundectomy for gastric fundal varices [16,17]. In our patient endoscopic hemostasis was unfeasible due to the huge blood clot covering the fundus of the stomach, thus surgical hemostasis was considered. Since no signs of esophageal varices were present on initial upper endoscopy and no signs of liver cirrhosis were diagnosed intraoperatively, we considered periesophagogastric devascularization on lesser curvature unnecessary, thus the surgical management was limited to greater curvature devascularisation, stapling fundal resection and splenectomy. Generally, splenectomy is considered the procedure of choice in the management of hemorrhage due to isolated splenic vein thrombosis [9], but we agree with others that fundectomy is mandatory in order to remove all lesions that bears the intramural and extramural gastric varices [17].

CONCLUSION

The sinister portal hypertension with hemorrhagic fundal varices is a rare but life-threatening condition, and this clinical situation must be suspected in all the patients with upper gastrointestinal bleeding and normal liver tests. The treatment of choice for bleeding fundal varices is considered to be the fundal resection and splenectomy, this clinical entity being among the few curable syndromes inducing portal hypertension.

CONFLICT OF INTERESTS

None to declare.

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