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Gastric Varices Secondary to Splenic Vein Occlusion due to Pancreatic Diseases

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Abstract

Gastric varices that arise secondary to splenic vein occlusion can result in hypersplenism or gastrointestinal hemorrhaging. This article review gastric varices secondary to splenic vein occlusion due to pancreatic diseases with regard to causes of the condition, as well as diagnostic and therapeutic approaches.

Diagnosis of gastric varices is made following esophago-gastro-duodenoscopy, and splenic vein occlusions are diagnosed from enhanced computed tomographic scans in almost all cases. Specific findings of gastric varices secondary to splenic vein occlusion are based on endoscopic ultrasonographic color flow images of gastric variceal flow that clearly depicted round cardiac and fundal regions at the center, with varices expanding to the curvatura ventriculi major of the gastric body.

Several treatment options for gastric variceal bleeding secondary to splenic vein occlusion have been proposed. Splenectomy, which decompresses the short gastric vein by cutting off inflow, has generally been considered the best treatment of choice in such condition. Endoscopic injection sclerotherapy using cyanoacrylate, is useful in the treatment of bleeding gastric varices due to splenic vein occlusion. As patients with splenic vein occlusion have normal portal pressure and normal hepatic function, portal systemic shunting is not indicated. Splenic arterial embolization, which reduces blood flow through the splenic parenchyma, is another effective method of controlling bleeding from gastric varices secondary to splenic vein occlusion. Treatment of gastric varices secondary to splenic vein occlusion is directed to the underlying pancreatic diseases.

Keywords: Gastric varices; Splenic vein occlusion; Pancreatic disease endoscopic ultrasonography; Color doppler; Left-sided portal hypertension

Abbreviations: EGD: Esophago-Gastro-Duodenoscopy; EUS: Endoscopic Ultrasonography; ECDUS: Endoscopic Color Doppler Ultrasonography; ECDUS: Endoscopic Color Doppler Ultrasonography; CT: Computed Tomography; EIS: Endoscopic Injection Sclerotherapy

Introduction

Gastric variceal hemorrhage is a common complication of portal hypertension and is associated with higher rates of morbidity and mortality than hemorrhage of esophageal varices [1]. Although hemodynamic studies of gastric varices are employed worldwide [2-4], splenic vein occlusion is often clinically silent, presenting no obvious symptoms. However, gastric varices secondary to splenic vein occlusion can cause hypersplenism or gastrointestinal hemorrhaging (left-sided portal hypertension) [5-8].

In this article, we review the diagnostic and therapeutic approaches, for left-sided portal hypertension due to pancreatic diseases.

Pathophysiology/Symptoms of Left-sided Portal Hypertension

Splenic vein occlusion results in left-sided portal hypertension (characterized by gastric varices, splenomegaly and normal liver function) [9-11] that is secondary to various diseases [12-14]. The majority of splenic vein occlusions are the result of pancreatic diseases, including acute and chronic pancreatitis or pancreatic tumor. Bertina reported that Von Leiden factor (FVL) deficiency has been reported in 2% to 30% of patients with portal vein thrombosis [15]. Moreover, the other articles found that FVL deficiency was also highly associated with splenic vein thrombosis [16,17]. As other causes, advanced left renal cancer and myeloproliferative disease are recognized. There is a strong association between pancreatic diseases and splenic vein occlusion because of the splenic vein, s anatomy. Sutton et al. reported that 35% of their cases of isolated splenic vein occlusion were caused by tumors and only 17% by pancreatitis [9]. On the other hand, Sakorafas et al. found that pancreatitis was the etiology in 87 (60%) of 144 cases, while pancreatic malignancy was detected in only [13] (9%) of the patients [7], and recent reviews have clarified acute or chronic pancreatitis to be the probable cause of splenic vein thrombosis in the majority of cases [18]. There is a possible pathogenetic mechanism implying the increased intra-abdominal pressure leading to compression of the splenic vein in the case of pancreatic pseudocyst [19,20].

Due to splenic vein occlusion, splenic venous flow must drain into collateral veins (short gastric vein and left gastroepiploic vein). Then, the increased blood flow dilates the submucosal veins of stomach, resulting in gastric varices (Figure 1). Because blood drainage is diverted by the coronary vein into the patent portal system, the presence of gastric varices without esophageal varices is a very specific sign of splenic vein occlusion [17]. This condition, which is commonly silent clinically, can cause hypersplenism or gastrointestinal hemorrhage.

Although splenic vein occlusion is commonly silent clinically,

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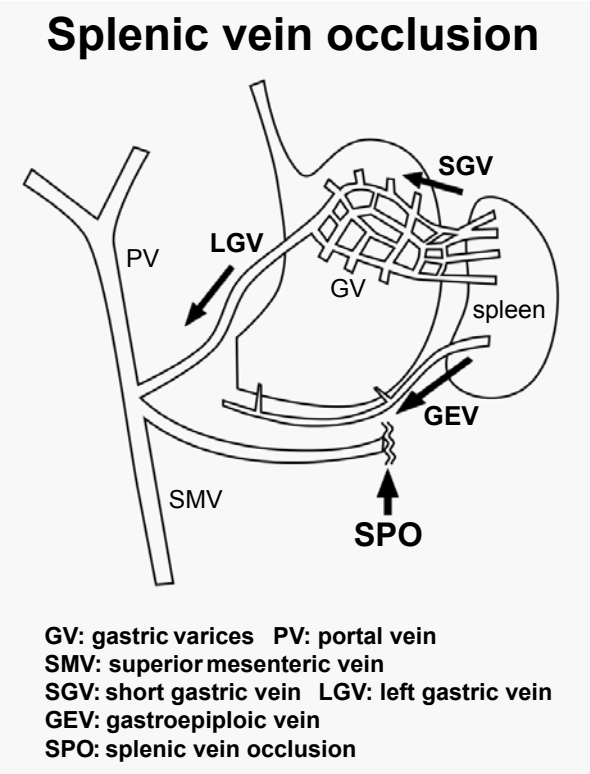

Figure 1: Hemodynamics of splenic vein occlusion.

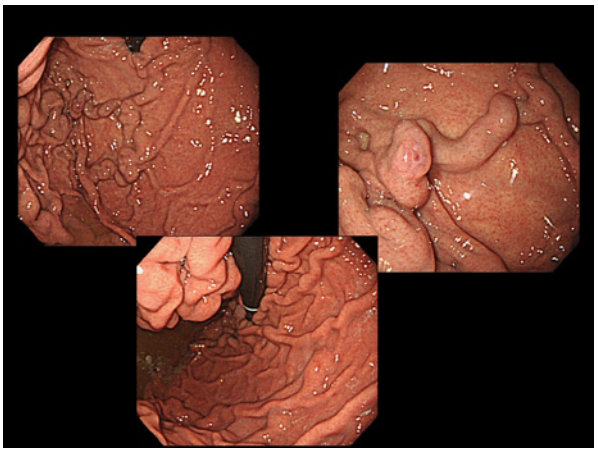

Figure 2: Endoscopic findings are gastric varices secondary to splenic vein occlusion after acute pancreatitis.

this condition may cause hypersplenism or gastrointestinal bleeding due to gastric varices. In previous studies of splenic vein occlusion, gastrointestinal bleeding has been reported to occur in 16-78% of patients [8,9,13,21-24]. Sutton et al. [9] found 53 cases of such occlusion, and reported a 64% incidence of upper gastrointestinal bleeding. Itzchak and Glickman [24] noted gastrointestinal hemorrhaging in in only 3 of 19 patients with splenic vein occlusion. Sarin et al. [22] studied the prevalence of gastric varices in 568 patients with portal hypertension, and they reported that 7 of 9 patients (78%) with gastric varices due to splenic vein occlusion had a history of previous variceal bleeding. Due to recent improvements in cross-sectional imaging, Heider et al. reported gastric variceal bleeding from pancreatitis-induced splenic vein thrombosis (with minimal symptoms) in only 4% of patients [25].

Diagnosis of Gastric Varices Secondary to Left-sided Portal Hypertension

Gastric varices have been diagnosed by esophago-gastro-duodenoscopy (EGD), a useful modality for observing gastric varices of a certain size and extent (Figure 2). EGD is usually the initial investigation in patients with portal hypertension for the purpose of distinguishing between gastric varices and gastric folds, and it has a very sensitive predictive value for variceal hemorrhage [26]. However, there are few cases of red color-positive gastric varices and it is difficult to diagnose a high risk for bleeding of gastric varices. Still further, EGD is a limited modality for detecting gastric varices, given how deep the submucosal or extramural collateral veins of gastric varices are.

Endoscopic ultrasonography (EUS) has become a useful modality for the diagnosis for esophagogastric varices, and it is considered most useful in evaluating gastric varices [27-29]. In a previous report, Sato et al. described the utility of endoscopic color Doppler ultrasonography (ECDUS) in patients with gastric varices [30]. Relative to EUS, ECDUS color images of blood flow in vessels allow for detailed sonographic visualization of vessels and evaluation of vascular blood flow for the diagnosis of gastric varices [31,32].

Endoscopic evidence is not sufficient to distinguish between gastric varices due to splenic vein occlusion or the gastric fold. Additional images resulting from ECDUS color analysis of gastric variceal flow clearly depicted a round fundal region at the center, with varices that expanded to the curvatura ventriculi major of the gastric body on gastric varices due to splenic vein occlusion (Figures 3a and 3b) [23]. Variceal location of ordinary gastric varices was classified as fundal (located far from the cardiac orifice) and cardiac and fundal (located between the cardiac orifice and the fundus), however, there was no case with varices expanding to the curvatura ventriculi major of the gastric body with ECDUS findings of ordinary gastric varices [30]. These data provide specific findings that can function as hallmarks of gastric varices due to splenic vein occlusion. The sensitivity of ECDUS in identifying gastric varices due to splenic vein occlusion should be considered for patients with high clinical suspicion [33].

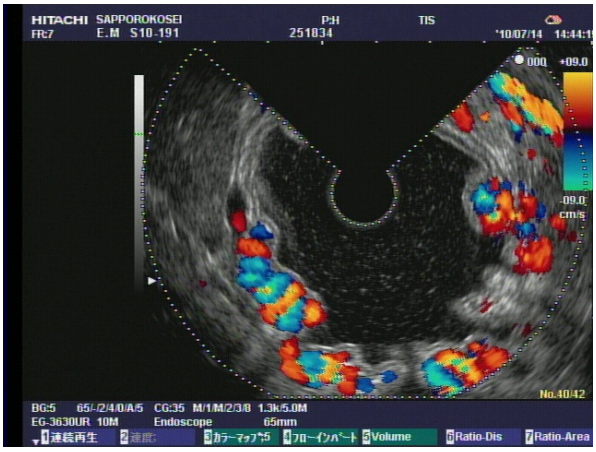

Figure 3a: Endoscopic color Doppler ultrasonography color flow images of gastric variceal flow clearly depicted round cardiac and fundal regions at the center, with varices expanding to the curvatura ventriculi major of the gastric body.

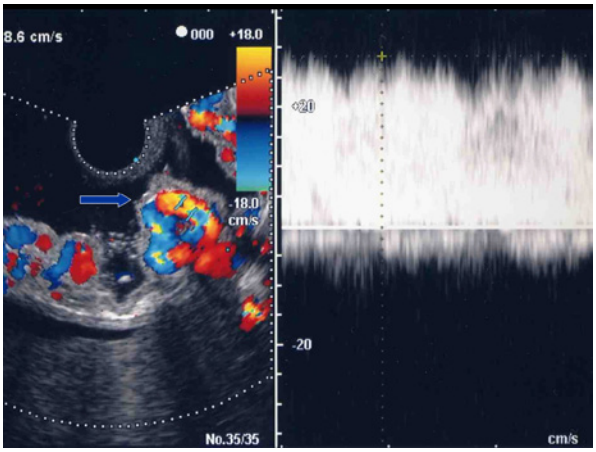

Figure 3b: Fast-Fourier transform analysis showed a continuous wave.

Percutaneous ultrasonographic examinations and color flow imaging are used as a preliminary, non-invasive method for splenic vein occlusion. However, those methods may be less accurate in assessing splenic vein patency because of the anatomic location of splenic vein, and in addition, may be unsuccessful when the acoustic window is not available for evaluation of whole portal venous system [34,35]. Contrast-enhanced computerized tomography (CT) portography can demonstrate the portal venous system in a short time [36,37]. However, it uses ionizing radiation and requires a large amount of iodinated contrast material.

Magnetic resonance (MR) angiography is a useful noninvasive method for the assessment of the portal venous system, and it appears to be a more accurate diagnostic procedure than Doppler ultrasonography and CT [38,39].

Venous phase angiography accurately presents the location of the splenic vein obstruction and collateral veins. Diagnosis of splenic vein occlusion is confirmed on angiography when venous collaterals in the splenic hilum and dilated short gastric veins and gastroepiploic veins are seen [40].

The diagnosis of left-sided portal hypertension complicating chronic pancreatitis is based on clinical presentation, preoperative endoscopic and radiographic imaging, and operative findings [13,41], and Evans et al. mentioned that splanchnic angiography is necessary for accurate diagnosis of left-sided portal hypertension [42]. The incidence of left-sided portal hypertension does not correlate to the severity of pancreatitis and may be a result of a mild or subclinical episode of pancreatitis [13,21].

Treatments of Gastric Varices Secondary to Left-sided Portal Hypertension

Several treatment options for gastric variceal bleeding secondary to splenic vein occlusion have been proposed. Splenectomy, which decompresses the short gastric vein by cutting off inflow, has generally been considered the best treatment of choice in such condition [9,11,13,41,42]. Due to the absence of malignant diseases in our patients, splenectomy was a useful treatment for left-sided portal hypertension. However, it is often difficult to determine whether surgical therapy should be undertaken in patients with advanced disease such as pancreatic carcinoma.

Endoscopic injection sclerotherapy (EIS) is more difficult to perform for gastric varices than for esophageal varices, and several reports have demonstrated that EIS of cyanoacrylate is an effective and safe treatment for gastric variceal bleeding [43-45]. Cyanoacrylate polymerizes immediately on contact with blood, resulting in rapid hemostasis. Major complications of the procedure include ulceration and recurrent bleeding, although more serious complications, including embolization to the brain [46], portal vein [47], lung [48,49], and spleen [50,51] have also been reported. Other complications include bacteremia [52] and abscesses [53]. However, it is now the first-choice treatment worldwide for obliteration of bleeding gastric varices. EIS using cyanoacrylate, is useful in the treatment of bleeding gastric varices due to splenic vein occlusion [23,54]. As patients with splenic vein occlusion have normal portal pressure and normal hepatic function, portal systemic shunting is not indicated. Splenic arterial embolization has been used in the treatment of hypersplenism. It is performed by transcatheter deposition of gianturco coils, autologous clots, or absorbable gelatin sponge [55]. Splenic arterial embolization, which reduces blood flow through the splenic parenchyma, is another effective method of controlling bleeding from gastric varices secondary to splenic vein occlusion [13,56-58]. Although this technique can be complicated by pain, splenic abscess or septicemia; splenic arterial embolization is an attractive alternative treatment for gastric varices secondary to splenic vein occlusion in patients at high surgical risk. The role of the splenic arterial embolization should be further analyzed for this condition.

Treatment for this condition should be performed corresponding to the underlying pancreatic diseases. The prognosis of gastric varices secondary to left-sided portal hypertension mainly depends on the underlying pancreatic disease. Most cases of pancreatic carcinoma have a shorter life expectancy and the incidence of gastric variceal bleeding is very low in that short life. When active bleeding is present unresponsive to conservative management, operation or endoscopic therapy should be considered on quickly. There is no consensus on the treatment of asymptomatic patients.

Conclusions

1. ECDUS color flow images of gastric variceal flow clearly depicted round cardiac and fundal regions at the center, with varices expanding to the curvatura ventriculi major of the gastric body as the specific findings of gastric varices secondary to splenic vein occlusion.

2. At present, although there is no consensus on the treatment of gastric varices secondary to splenic vein occlusion, we suggest that it would be directed to the underlying pancreatic diseases.

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