Gastric Varices

Abdul Aleem MD
Lehigh Valley Health Network, Abdul.Aleem@lvhn.org

Hiral N. Shah MD
Lehigh Valley Health Network, hiral_n.shah@lvhn.org

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Aleem A, Shah H.

Continuing Education Activity

Gastric varices are dilated portosystemic collateral blood vessels that develop as a complication of portal hypertension or extrahepatic portal vein obstruction. Based on the location and relation to the esophagus, gastric varices are further classified into gastroesophageal varices isolated gastric varices. Acute bleeding from gastric varices is life-threatening and is associated with higher mortality compared to esophageal variceal bleeding. This activity reviews the etiology, pathophysiology, diagnosis, management of gastric varices and highlights the interprofessional team's role in evaluating and managing patients with this condition.

Objectives:

- Describe the etiology and pathophysiology of gastric varices.
- Summarize the diagnostic evaluation of gastric varices.
- Describe the management of gastric varices,
- Review interprofessional team strategies for improving care coordination and communication to improve outcomes in patients with gastric varices.

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Introduction

Gastric varices are dilated submucosal collateral veins that develop in the setting of portal hypertension due to any etiology with or without cirrhosis.[1] Compared to esophageal varices, gastric varices are less common occurring in approximately 20% of cirrhotic patients. However, gastric varices have a higher propensity to bleed severely and are often associated with poor patient outcomes.[2][3] Based on their stomach location, gastric varices are classified as gastroesophageal varices (GOV) and isolated gastric varices (IGV).

Among all the different classifications available to describe gastric varices, Sarin et al.'s classification are the most commonly used. Gastroesophageal varices are extensions of esophageal varices and are termed as GOV type 1(GOV1) when they extend below the gastroesophageal junction along the lesser curvature and GOV type 2(GOV2) when they extend into the fundus of the stomach. Isolated gastric varices (IGV) located in the fundus of the stomach are termed IGV type 1(IGV1) or commonly referred to as fundal varices. IGV type 2(IGV2) are ectopic varices located anywhere in the stomach. GOV1 represents almost 75% of all gastric varices, followed by GOV2, 21%, IGV1 less than 2%, and IGV2, which comprises 4%.[4]

Etiology

Gastric varices are commonly encountered in cirrhotic patients as a result of portal hypertension. However, gastric varices can occur in patients with noncirrhotic portal hypertension and are considered distinct with a different etiology, pathophysiology, and management strategy compared to gastric varices secondary to portal hypertension.[5]

Causes of Cirrhosis Related Portal Hypertension

- Alcohol
- Viral hepatitis (HBV, HCV)
- Non-alcoholic steatohepatitis
- Autoimmune hepatitis
- Primary biliary cholangitis (PBC)
- Primary sclerosing cholangitis
- Hereditary hemochromatosis
- Wilson disease
- Alpha-1 antitrypsin deficiency

Causes of Noncirrhotic Portal Hypertension (NCPH)

- Extrahepatic portal venous obstruction (EHPVO)
- Portal vein thrombosis

- Splenic vein thrombosis (SVT) due to chronic pancreatitis, pancreatic pseudocysts, pancreatic neoplasm
- Infiltrative diseases-macrocytosis, amyloidosis
- Congenital hepatic fibrosis
- Schistosomiasis
- Noncirrhotic portal fibrosis
- Drug-induced
- Hepatic venous outflow tract obstruction (HVOTO)
- Granulomatous-sarcoidosis
- IVC obstruction
- Constrictive pericarditis
- Severe right-sided heart failure

Epidemiology

The exact prevalence of gastric varices is unknown. Two single-center prospective studies involving more than 500 patients with both cirrhotic and non-cirrhotic hypertension have estimated the prevalence of gastric varices to be between 15 to 20%.[6][7][8] With the advent of endoscopic ultrasound, prevalence rates as high as 55% to 78% have been reported.[9] The incidence of bleeding from gastric varices is estimated to be at 10% to 30 % in patients with underlying portal hypertension.

Pathophysiology

Portal hypertension is the hemodynamic dysfunction commonly associated with liver cirrhosis and a clinical manifestation of noncirrhotic hepatic and extrahepatic disorders.[10] As mentioned earlier, portal hypertension is the primary determinant implicated in developing gastric varices. Normally, the portal vein receives blood from the coronary vein, splenic vein, superior mesenteric vein, and the normal hepatic venous pressure gradient (HVPG), representing the difference between the wedged hepatic vein pressure and the free hepatic venous pressures, ranges from 3 to 5 mmHg.[11]

The pathophysiology of portal hypertension in cirrhotic patients is explained by various factors that include decreased tone of the hepatic vasculature from endothelial dysfunction due to disequilibrium between vasoconstrictors and vasodilators and increased hepatic resistance to portal blood flow from cellular damage, fibrosis, and regenerative nodular formation.[12] When the HVPG increases beyond a certain threshold level, usually ≥ 10 mmHg, portal hypertension becomes clinically significant, resulting in complications such as ascites, development of gastroesophageal varices, hepatic encephalopathy, and functional renal failure.[10]

The ensuing portal hypertension also results in spontaneous portosystemic collateral shunts between the splenic vein and the left renal vein, commonly referred to as gastro-renal shunts. These collateral shunts mediated by angiogenic factors such as vascular endothelial growth factor (VEGF) and VEGFR-2 are commonly seen in approximately 85% of patients with gastric varices.[13][14][15] Approximately 15% of patients with GVs have various portosystemic venous pathways, such as the left inferior phrenic, pericardiac, and azygous-hemiazygos veins. Additionally, the portal venous blood flow is aggravated by a state of hyperdynamic circulation due to splanchnic vasodilatation from increased production of endogenous vasodilators (nitric oxide, prostacyclin, and TNF) and increased cardiac output.[16]

The increased pressure and portal blood flow in the portosystemic circulation due to the above factors lead to gastric varices causing elevated intra-variceal pressure and wall tension, increasing the risk of variceal rupture leading to a life-threatening GI bleed.[17] HVPG > 20mmHg is associated with increased rebleeding and severity of variceal bleeding, translating into increased mortality.[18] The incidence of gastric variceal bleeding is based on the location of gastric varices, size of varices, the severity of liver failure based on Child-Pugh score, presence of variceal red spots on endoscopic evaluation, and presence of hepatocellular carcinoma.[19][20]

Alternatively, gastric varices can also occur in the absence of cirrhosis and normal HVPG due to segmental portal hypertension from any obstruction in the splenic vein, such as thrombosis and stenosis sequela of pancreatic pathology (pancreatitis, pancreatic pseudocysts, or neoplasms).[21][8] This obstruction in the splenic vein causes shunting of the blood resulting in the development of spleno-portal collaterals, which causes increased pressure in the submucosal veins of the fundus resulting in the formation of isolated gastric varices. Isolated gastric varices secondary to splenic vein thrombosis are considered distinct from gastric varices secondary to cirrhosis.[22]

History and Physical

History

In patients with a new diagnosis of cirrhosis of unknown etiology, history of alcohol use (onset, quantity, and frequency of use), illicit drug use, and family history of liver disorders should be elicited. In patients with decompensated cirrhosis of known etiology, history should be elicited regarding prior or ongoing variceal bleeding (hematemesis, melena, hematochezia), ascites (swelling, discomfort, fullness, lower extremity swelling, dyspnea, orthopnea), hepatic encephalopathy (confusion, excessive sleepiness).In patients without cirrhosis with incidental findings of gastric varices, history should be obtained about any personal history of thrombophilia or family history of thrombophilia and history of pancreatic disorders.

Physical Exam

A general examination to assess vital signs that include heart rate, blood pressure, respiratory rate, and oxygen saturation should be performed on all patients at baseline. A focused systemic examination evaluating clinical signs specifically related to cirrhosis and portal hypertension should be performed as described below**.**

Ocular

- Scleral icterus

Neck

- Elevated jugular venous pulse (right-sided heart failure)

Pulmonary

- Pleural effusion (hepatohydrothorax)

Abdominal

- Ascites (abdominal distension, fullness of flanks, fluid thrill, puddle sign, shifting dullness, abdominal tenderness)
- Splenomegaly
- Periumbilical collateral circulation (caput-medusae)

Skin

- Spider angiomata

Musculoskeletal

- Edema
- Icteric skin
- Asterixis
- Palmar erythema
- Dupuytren contracture
- Terry nails

Genitourinary

- Testicular atrophy
- Scrotal edema

Neurological

- Overt and covert encephalopathy
- Asterixis

Miscellaneous

- Gynecomastia
- Fetor hepaticus

Clinical Features

- Unlike esophageal varices, gastric varices uncommonly present with acute variceal bleeding and are routinely discovered on screening EGD for varices in patients with portal hypertension.

- The main risk factors for bleeding are the severity of liver dysfunction, location and size of the varices, and presence of bleeding stigmata. [19][20]
- Patients present with acute gastric variceal bleeding are critically ill on presentation as gastric variceal bleeding tends to be more severe and life-threatening.
- GOV2 subtype of gastric varices bleed more frequently than GOV1 and carry the worst prognosis.
- Alternatively, the patients with segmental portal hypertension secondary to splenic vein thrombosis in pancreatic disorders are diagnosed with gastric varices incidentally based on cross-sectional imaging.

Evaluation

Patients with a new diagnosis of cirrhosis should undergo a complete laboratory workup to ascertain the etiology of cirrhosis. In patients with decompensated cirrhosis presenting with acute variceal bleeding, initial blood work with a complete blood count, comprehensive hepatitis panel that includes liver and renal function panel, coagulation profile, arterial blood gas analysis should be performed.

- **Diagnostic esophagogastroduodenoscopy (EGD)** should be performed to assess and stratify gastric varices in patients with decompensated cirrhosis or incidental findings of gastric varices on cross-sectional imaging in patients without cirrhosis.
- **Endoscopic ultrasound (EUS)** is useful for better characterization of gastric varices, and also its therapeutic potential in the management of gastric varices has gained momentum in recent years.
- Noninvasive diagnostic methods such as **transient elastography** can be used to assess the severity of fibrosis in patient's or at risk of developing clinically significant portal vein hypertension (CSPH)
- Invasive diagnostic methods such as **HVPG measurement** can be used to diagnose CSPH and predict the development of gastric varices and estimate the risk of variceal bleeding.
- Imaging modalities such as **ultrasound of the liver, CT, or MRI** can be used make a diagnosis of cirrhosis and also to rule out hepatocellular carcinoma (HCC)
- **Hepatic Doppler sonography, CT or MRI angiography, venous phase celiac arteriography** can be used to assess the patency of the portal and splenic veins to rule out thrombosis.
- **Liver biopsy** should be considered in patients to assess the severity of fibrosis and to ascertain etiology in cirrhotic patients with negative laboratory workup.

Treatment / Management

The optimal management of gastric varices remains unclear due to the lack of high-quality large clinical trials. However, there is consensus that the management of GOV1 varices is similar to that of esophageal varices. Nevertheless, based on the 2016 AASLD guidelines, the management of gastric varices (GOV2/IGV1) depends on the acuity of presentation and is classified into three categories: primary prophylaxis, management of acute variceal bleeding, and secondary prophylaxis.[2]

- **Primary Prophylaxis**
    - Beta-blockers have been used as a long-term prophylactic treatment for portal hypertension. The 2016 AASLD guidelines recommend non-selective beta-blockers (NSBBs) therapy such as propranolol or nadolol as primary prophylaxis of acute variceal hemorrhage from gastric varices, especially GOV2 or IGV1 that have not bled. The primary prophylaxis of GOV1 varices is similar to that of esophageal varices.[2]
- **Management of Acute Gastric Variceal Bleeding**
    - The most crucial step in managing patients presenting with acute gastric variceal bleed is admitting the patient to the ICU for initial resuscitation with packed red blood cells (PRBCs) transfusion and respiratory status protection.
    - PRBCs transfusion should be limited to a target hemoglobin of approximately 8 g/dL
    - Associated coagulopathy and thrombocytopenia should be corrected.
    - The patient should be maintained on empiric broad-spectrum antibiotics, especially in patients with cirrhosis, to decrease the risk of bacterial infections, which is associated with increased rates of mortality.
    - The role of vasoactive agents such as octreotide, vasopressin, terlipressin, and somatostatin in gastric variceal bleeding management is not clear. Nevertheless, they are still utilized because of their safety and potential benefits.
    - Urgent upper endoscopy must be performed for diagnosis and treatment.
- **Endoscopic Therapies**

Data regarding the role of endoscopic therapy for the treatment of bleeding gastric varices is limited. However, endoscopic hemostasis is the mainstay in the management of patients presenting with acute gastric variceal bleeding. Current available endoscopic interventions include endoscopic injection sclerotherapy, tissue adhesives such as cyanoacrylate-based tissue adhesives, thrombin, and endoscopic variceal ligation (EVL).[23]

- **Endoscopic injection sclerotherapy (EIS)** with sclerosing agents such as alcohol, polidocanol, or tetradecyl sulfate is associated with increased complications and rebleeding rates. It hence is not recommended in the management of acute gastric variceal bleeding, especially GOV2.[24][25]
- **Direct injection of thrombin or fibrin glue** appears to be a promising therapeutic intervention, but their role has not been adequately evaluated in managing acute gastric variceal bleeding.
- **Cyanoacrylate (CYA) glue** is a class of synthetic glue mixed with lipiodol, an oily contrast agent before injection into varix that rapidly polymerizes into a hard-acrylic plastic upon exposure to water or blood, leading to hemostasis.[24] Current American Society for Gastrointestinal Endoscopy (ASGE) guidelines recommend an evaluation with diagnostic endoscopy and treatment of gastric variceal hemorrhage with gastric variceal obturation (GVO) with the employment of a cyanoacrylate-based compound for the treatment of acute gastric variceal hemorrhage by endoscopists familiar with this technique. Otherwise, endoscopic variceal ligation (EVL) is recommended, especially GOV1 and small GOV2.[26] A meta-analysis study by Qiao et al. reported higher rate of active bleeding control (OR, 4.44; 95% CI, 1.14-17.30;P =0.032) ,lower rebleeding rate (OR, 0.33; 95% CI, 0.18- 0.60; P =0.0004) and lower gastric varices recurrence rate (RR, 0.26; 95% CI, 0.11-0.61; P =0.002) in patients treated with cyanoacrylate injection compared to patients who were treated with EVL.[27]
- **Endoscopic Ultrasound(EUS)-guided therapy**: EUS-guided CYA glue injection that precisely identifies gastric varix by color Doppler and delivers the CYA into varix leading to variceal obliteration and EUS guided coiling that utilizes the insertion of micro-coils into varix leading to variceal obliteration are emerging techniques that have been employed in the management of gastric varices. The benefit of the EUS guided approach is that it precisely identifies the actual degree of variceal obliteration. A single-center retrospective study involving 152 patients with high-risk gastric fundal varices treated with EUS-guided combined coil and CYA glue injection reported that the combination therapy was highly effective for hemostasis in active bleeding and also when used for primary and secondary bleeding prophylaxis of gastric varices. A multicenter meta-analysis by McCarty et al. concluded that EUS-guided therapies were safe, effective, and technically feasible for the treatment of GV. This study also noted that among different types of EUS-guided therapies, EUS combination therapy with coil embolization + CYA injection is a preferred approach for managing GV over EUS-based monotherapy (coil alone or CYA alone).[28]

- ***TIPS and BRTO*:** If acute gastric variceal bleeding is not amenable to be controlled with endoscopic intervention, then transjugular intrahepatic portosystemic shunt (TIPS) or Balloon occluded retrograde transvenous obliteration (BRTO) should be performed

    - **TIPS** is an invasive technique that involves creating an intrahepatic connection between the hepatic and the portal venous system resulting in the diversion of the portal venous flow into the systemic circulation, thereby reducing portal pressure. It is the treatment of choice in acute gastric variceal bleeding, especially GOV2 and IGV1. Randomized control trials have shown that TIPS has been more effective than cyanoacrylate injection in preventing rebleeding from gastric varices, with similar survival rates and frequency of associated complications.[29]
    - **BRTO** has emerged as a safe and clinically effective treatment option in managing gastric variceal bleeding and should be considered an alternative option, especially if patients with associated refractory hepatic encephalopathy. BRTO is an invasive technique that involves the retrograde injection of a sclerosing agent into gastric varix after occluding the portosystemic shunt with an occlusion balloon under fluoroscopic guidance. However, the performance of BRTO requires the presence of a spontaneous portosystemic shunt such as a gastro-renal shunt or a gastro-caval which is present in 60 to 85 % of cases and should be predetermined by computed tomography or magnetic resonance imaging prior to the performance of this procedure. A meta-analysis of 24 studies involving 1,016 patients by Park et al. reported the technical success rate for BRTO is 96.4%, and the clinical success rate was 97.3%.[30] Retrospective studies comparing TIPS with BRTO have reported that the active bleeding control rate, rebleeding rate, and complication rate were better with BRTO compared to TIPS.[31][32]

- ***Balloon Tamponade***

    - Although data regarding the use of balloon tamponade in gastric variceal bleeding is limited, balloon tamponade with Sengstaken-Blakemore tube or Lintone-Nachlas tube should be considered in patients with massive acute gastric variceal not amenable to endoscopic intervention as a bridge to more definite treatments.

- ***Surgical Approach***

    - With the advent of newer minimally invasive and improved diagnostic and therapeutic techniques via endoscopy and interventional radiology, the role of surgery in managing acute gastric variceal bleeding is limited and should be considered a last resort when the above measures fail. However, splenectomy or splenic artery embolization should be considered a means of definitive therapy in

patients presenting with acute gastric variceal bleeding secondary to sinistral or left-sided portal hypertension caused by splenic vein thrombosis (SVT). Splenectomy eliminates collateral venous flow and prevents future gastric variceal bleeding secondary to splenic vein thrombosis.[22]

- **Secondary Prophylaxis**
    - Nonselective beta-blockers in combination with endoscopy evaluation therapy (EVL or cyanoacrylate injection) are recommended as first-line therapy to prevent rebleeding in patients who recover from GOV1 hemorrhage.[2]
    - TIPS or BRTO are indicated as first-line therapy in patients of recover from gastric variceal hemorrhage secondary to GOV2 or IGV1 type of gastric varices.
    - EUS guided cyanoacrylate glue injection, or EUS guided coil embolization, is a secondary prophylactic option for patients in whom the performance of TIPS or BRTO is not technically feasible.

Differential Diagnosis

Differential diagnosis of gastric varices manifesting as hematemesis includes common etiologies of upper GI hemorrhage:

- Peptic ulcer disease (PUD)
- Esophageal varices
- Acute gastric erosions
- Ulcerated submucosal lesions
- Malignant gastric ulcers
- Dieulafoy's lesion
- Mallory-Weiss tear
- Portal hypertensive gastropathy (PHG)
- Gastric antral vascular ectasia (GAVE)

However, in the absence of bleeding and noted on upper endoscopy, the differential diagnosis includes the following:

- Epithelial lesions (lipomas, duplication cysts, pancreatic rests)
- Carcinoid tumor
- Lymphangiomas
- Gastric lymphoma
- Gastrointestinal stromal tumor (GIST)
- Inflammatory gastric polyp

Prognosis

Although gastric varices and portal hypertension an incidental acute gastric variceal bleeding is lower than esophageal variceal bleeding, gastric varices have a high propensity to bleed severely. They are often associated with higher mortality than esophageal variceal bleeding. The overall prognosis is dependent on the severity of the underlying disease and commonly used prognostic model such as Child-Pugh score and Model for end-stage liver disease (MELD) score is useful to predict the severity of underlying liver disease and also are effective in estimating the risk of mortality.

Complications

Spontaneous rupture of gastric varices can result in

- Aspiration
- Acute blood loss anemia
- Hemorrhagic shock
- Multiorgan failure
- Increased susceptibility to bacterial infections
- Death

Enhancing Healthcare Team Outcomes

Management of gastric varices and their resulting complications requires an interprofessional team approach consisting of gastroenterologist, hepatologist, internist, intensivist, critical care nurse, pharmacist, respiratory therapist, blood bank team, endoscopy technician, and interventional radiologist. The patient's hemodynamics and blood work should be monitored closely, and there should be close communication between the caring nurse and the ordering physician. The pharmacy team should make all critical drugs available, and the blood bank should mobilize blood products as needed. The gastroenterology team should discuss the case with the interventional radiology team well in advance, especially in patients presenting with acute gastric variceal hemorrhage in anticipation of TIPS or BRTO. If specialized interventional radiologic techniques such as TIPS or BRTO are unavailable, supportive therapy should be provided, and the patient should be transferred to a facility where these procedures are available. Such a holistic team approach goes a long way in improving patient outcomes. [Level 5]

Review Questions

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Publication Details

Author Information

Authors

Abdul Aleem[1]; Hiral Shah[2].

Affiliations

[1] Lehigh Valley Health Network, Allentown, USA

[2] Lehigh Valley Health Network

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