

## Esophageal Varices

Esophageal varices occur when the submucosal veins in the lower esophagus become engorged. This is usually caused by portal hypertension in patients with cirrhosis, but any cause of portal hypertension (e.g., thrombus) can lead to the formation of esophageal varices. Increased portal pressures result in the shunting of the blood from the left gastric veins to the esophageal veins. If the veins become too enlarged and rupture, it can lead to hematemesis, melena, or even hypotension. IV fluids, antibiotics, octreotide, and endoscopic procedures, such as band ligation, sclerotherapy, and balloon tamponade are the mainstay treatment options for acute stabilization. Nonselective beta-blockers can be used for secondary prophylaxis, and transjugular intrahepatic portosystemic shunting (TIPS) can be attempted in refractory cases.



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### Pathophysiology

#### Cirrhosis

##### [C-Roses-On-Liver](#)

Cirrhosis leads to portal hypertension and subsequent formation of esophageal varices (EV). Complications of EV (e.g., severe bleeding) are common causes of death in patients with cirrhosis.

#### Portal Hypertension

##### [Portal Hiker-BP](#)

Portal hypertension is defined as increased pressure in the portal vein. It is usually secondary to other medical conditions such as cirrhosis, portal vein thrombosis, schistosomiasis, or right heart failure. Basically, anything that leads to congestion and increased pressure within portal veins can cause portal hypertension, which often leads to the formation of esophageal varices.

#### Left Gastric Vein and Esophageal Veins

##### [Left Gastric Vein and Sarcophagus-Veins](#)

Portal hypertension promotes shunting of blood through portosystemic collateral pathways. The esophageal venous system is one of those pathways, and shunting of blood from the left gastric vein to esophageal veins drives the formation of submucosal esophageal varices.

### Clinical Features

#### Hematemesis

##### [Blood in Vomit](#)

Hematemesis is vomiting blood, and is one of the possible manifestations of esophageal varices.

#### Melena

##### [Black Intestine-stool](#)

Profuse GI bleeding caused by the rupture of esophageal varices may result in melena, which refers to dark, tarry stool and is caused by oxidation of hemoglobin as the blood passes through the GI tract. If the bleeding is vigorous and transit time in the intestines is short, the blood may not have time for oxidation, and patients may develop hematochezia, which refers to bright, red-colored stools.

## Hypotension

### Hippo-BP

Massive venous bleeding from esophageal varices often leads to hypotension, which can cause hypoperfusion of vital organs (e.g., brain, kidneys) and eventually death.

## Acute Management

### IV Fluids

#### IV Fluid Acute-angle

The acute management of esophageal varices starts with IV fluids, typically isotonic solutions, to raise/stabilize blood pressure and prevent end-organ hypoperfusion.

### Antibiotics

#### ABX-guy

Patients with variceal bleeding should be given prophylactic antibiotics, preferably before endoscopy. IV broad-spectrum antibiotics such as ceftriaxone are typically used for this purpose. The goal is to complete a total of seven days of antibiotic therapy. Patients who are discharged before seven days are transitioned to oral ciprofloxacin to complete the seven-day course.

### Octreotide

#### Octo-tree-ride

IV octreotide is often given for the management of acute variceal hemorrhage on the basis that it reduces portal venous pressure via splanchnic vasoconstriction and thus decreases shunting of the blood into the submucosal veins.

### Endoscopic Band Ligation or Sclerotherapy

#### Line-gate and Skull-arrow-laser

Endoscopic band ligation or sclerotherapy may be used to reduce bleeding of varices in patients with cirrhosis. Ligation is done by placing an O-ring around the base of the enlarged vein, and sclerotherapy involves injecting a sclerosing agent into the base of the enlarged vein.

### Balloon Tamponade

#### Balloon Tampon

Balloon tamponade mechanically compresses the varices, controlling hemorrhage. This procedure involves two balloons with three lumens, one for the gastric balloon, one for the esophageal and one for gastric aspiration (Sengstaken-Blakemore tube).

## Prophylaxis

### Nonselective Beta Blockers

#### Nonselective Beta-fish with LOL Blocks and Purple-axes

Nonselective beta-blockers (e.g., nadolol, propranolol) block the adrenergic dilatory tone in mesenteric arterioles, resulting in the unopposed alpha-adrenergic activity, which leads to vasoconstriction. This decreases blood flow from the systemic circulation into the portal circulation and thus reduces pressure in the portal system. Beta-blockers are used for prophylaxis of esophageal variceal bleeding.

### Transjugular Intrahepatic Portosystemic Shunt (TIPS)

#### Q-TIPS with Shunt

TIPS, or transjugular intrahepatic portosystemic shunt, is a nonsurgical procedure that creates a shunt. This shunt connects systemic and portal venous systems, which redirects portal blood flow to reduce venous pressure and decrease portosystemic shunting. This is done by puncturing the wall of the hepatic vein and using a catheter to connect it to the portal vein. This procedure is contraindicated in those with severe hepatic encephalopathy since it may shunt excessive ammonia toxins to cerebral circulation.