All Bleeding Stops...Eventually

DALHOUSIE UNIVERSITY Inspiring Minds

Simon Field, MBBCh, CCFP(EM), FCFP; and Melissa Keith, BSc

David's Case

David, 54-years-old, presented to the ED after he was found collapsed in the washroom of an outpatient clinic surrounded by a large amount of hematemesis.

The patient had a history of ongoing alcohol abuse, Hepatitis C, liver cirrhosis and portal hypertension. He had recently sustained a metacarpal fracture and was being followed up in an outpatient clinic for this injury.

Upon examination, the patient was found to be in hemorrhagic shock with BP in the 40s and a heart rate of 38 bpm.

Urgent endotracheal intubation was performed and resuscitation with large volumes of IV fluids was commenced. The patient was given fresh blood, frozen plasma, pantoprazole and octreotide.

Read on for more on David.

1. What are the causes of gastric varices?

Portal hypertension is one of the main causes of gastric varices. The hepatic venous pressure gradient (HVPG), the pressure gradient between the portal vein and inferior vena cava, must be > 12 mmHg (normal: 3 mmHg to 6 mmHg) for variceal formation. This often develops as a complication of thrombus or other venous obstruction, liver cirrhosis (alcohol or hepatitis related), congestive heart failure, schistosomiasis or congenital hepatic fibrosis. Liver cirrhosis is the most common cause of portal hypertension, accounting for 90% of all cases.

2. When should gastric varices be suspected clinically?

- Continued alcohol use
- Presence of ascites
- Poor liver function (*i.e.*, cirrhosis, advanced Child-Pugh score, elevated Model for End-Stage Liver Disease [MELD] score)

When a diagnosis of liver cirrhosis is made, screening esophagogastroduodenoscopy (EGD) should be done to check for varices. The elastic properties of the vessel, pressure gradient and red whale sign (cherry-red hematocystic spots on endoscopic examination) are all predictors of varices and likelihood of hemorrhage.

3. How are gastric varices classified?

Gastric varices are classified mainly based on location. Gastroesphoageal varices are continuous with the

David's case cont'd

After stabilization and normalization of vital signs, gastroscopy was performed by gastroenterology in the ED and revealed gastric varicosities in both the antrum and fundus of the stomach. Sclerotherapy was performed but bleeding persisted.

After failed transjugular intrahepatic portosystemic shunt (TIPS) and Blackmore procedures, the patient was taken to the operating room for a splenectomy and gastric devascularization.

esophagus along the greater (type I) and lesser (type II) curvatures of the stomach. Isolated gastric varices occur in the fundus (type I) or in the body, antrum, or duodenal bulb (type II).

4. *What are the complications of gastric varices?*

One of the most serious complications of gastric varices is rupture. Rupture of gastric varices is associated with a mortality rate of 25% to 55%. The most important predictor of hemorrhage is the size of the varices—large varices are classified as > 5 mm in diameter.

5. *How are gastric varices managed?*

The major goal of treatment is prophylaxis. Pharmacologically, non-selective β-blockers are used to decrease BP and cardiac output, therefore decreasing portal venous pressure. These have been shown to reduce the risk of hemorrhage from 25% to 15% in patients with medium to large sized varices. Prophylactic use of β-blockers is not recommended for patients with small (< 5 mm diameter) varices. In addition, many studies show that continued alcohol use with alcoholic liver disease is associated with increased mortality and recurrent variceal bleeding, though this remains controversial.

Acute hemorrhage is managed by blood volume replacement, drug therapy (vasoactive drugs such as terlipressin, prophylactic antibiotics) and endoscopic evaluation of the varices. Prophylactic antibiotics, such as oral norfloxacin, are important to prevent bacterial infections, reduce incidence of early rebleeding and improve survival. Preventing hypovolemia via volume replacement can help prevent complications such as acute renal failure.

Current treatments available for gastric varices include gastrorenal shunt surgery, endoscopic injection sclerotherapy and transjugular intrahepatic portosystemic shunting (TIPS). In the TIPS procedure, a stent is placed between the portal and hepatic veins, reducing portal pressures. If this fails, or is not indicated, then devascularization may be preformed in addition to a splenectomy, as seen in this case. Dx

Take-home message

- Patients presenting with well-compensated liver cirrhosis should have variceal screening via esophagogastroduodenoscopy every 2 to 3 years, or 1 to 2 years if small varices are present. Screening should be done annually if the cirrhosis is not well controlled
- Mortality from gastric varices can be as high as 50% in those with a history of cirrhosis
- Variceal rupture is the most serious complication of gastric varices. Hemorrhage from gastric varices can be prevented by decreasing the hepatic venous pressure gradient to < 12 mmHg, which can be achieved with pharmacological prophylaxis in suitable candidates
- First-line treatment for gastric varices is endoscopic tissue adhesive injection in an attempt to arrest bleeding immediately. Endoscopic sclerotherapy and balloon tamponade are other options to consider should injection fail, although ballon tamponade is a very temporary solution and should not be considered a final treatment

Resources
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Dr. Field is an Assistant Professor, Department of Emergency Medicine, Dalhousie University; and an Emergency Physician, QEII Health Sciences Centre and IWK Health Centre, Halifax, Nova Scotia.

Ms. Keith is a Second Year Medical Student, QEII Health Sciences Centre and Dalhousie University, Halifax, Nova Scotia.

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Return undeliverable Canadian addresses to:
STA Communications Inc.
955 boulevard St-Jean, Suite 306
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