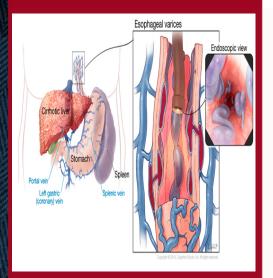
JUNE 2015 VOLUME #2 ISSUE 21

EM CASE OF THE WEEK

BROWARD HEALTH MEDICAL CENTER DEPARTMENT OF EMERGENCY MEDICINE

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Hematemesis is an ominous sign, especially in the patient with underlying portal hypertension and cirrhosis. The emergency physician must be aware of the most likely causes and be prepared for aggressive resuscitation as these patients can quickly go into hemorrhagic shock.

EM CASE OF THE WEEK

EM Case of the Month is a monthly "pop quiz" for ED staff. The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.



Upper GI bleeds in the Emergent Setting

A 45 year old male with a PMH of alcholic cirrhosis signs into the ED with 2 days of vomiting blood, subjective fever, fatigue, abdominal pain and black tarry stools. His vital signs are T 98.8, HR 110, RR 18, BP 94/60, O2 sat 98%. Physical exam is significant for jaundice, ascites and abdominal distention. Which of the following is the most likely cause of the patient's bleed?

- A. Peptic Ulcer
- B. Mallory Weiss tear
- C. Boerhaave's Syndrome
- D. Esophageal variceal hemorrhage
- E. Gastric Cancer



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Take Home Points

- The most important primary intervention for a patient with acute variceal hemorrhage is volume!
- Gold standard of diagnosis is endoscopy
- Octreotide is used for active variceal bleeding
- Non-selective beta blockers are used for bleeding prophylaxis in patients with esophageal varices without bleeding

Upper GI bleeds in the Emergent Setting

The correct answer is D. While all of these choices can cause upper GI bleeding, the history of alcohol abuse should raise your suspicion for Boerhaave's and variceal hemorhage. The history of cirrhosis along with the physical exam of jaundice and ascites should help sway your choice to variceal hemorrhage.

Discussion:

The most common cause of portal hypertension of is cirrhosis. The damaged tissue of the liver obstructs blood flow. This backed up blood has to go somewhere so it begins to use collateral circulation to pool this blood and venous vessels around the esophagus become tortuous and engorge into varices or abnormally dilated vessels. Varices appear to be the cause of upper GI bleeding in up to 90% of patients with cirrhosis. The most important predictor of hemorrhage is the size of varices; the largest varices are at highest risk of bleeding. Another predictor is Hepatic Vein Pressure Gradient greater than 10mmHg.

Signs and Symptoms:

Nonbleeding varices are generally asymptomatic. Once varices are bleeding, patients present with hematemesis, passage of black or bloody stools, lightheadedness, or decreased urination. Associated signs of variceal hemorrhage include decompensated liver function manifested as jaundice, hepatic encephalopathy, worsened or newonset ascites. Physical examination will likely reveal hypotension, pallor and signs of chronic liver disease such as spider angiomatas, palmar erythema, gynecomastia, or splenomegaly. A rectal examination should be performed on all patients without obvious bleeding. A black tarry stool on the gloved finger suggests an upper gastrointestinal source, and further workup needs to be pursued.

For a list of educational lectures, grand rounds, workshops, and didactics please visit http://www.BrowardER.com and click on the "Conference" link. All are welcome to attend!

JUNE 2015 VOLUME #2 ISSUE 21

Diagnosis:

The gold standard for the diagnosis of varices is esophagogastroduodenoscopy (EGD). It is generally recommended that patients with cirrhosis undergo elective endoscopic screening for varices at the time of diagnosis and periodically thereafter if no or small varices are detected.

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Small varices are those less than 5mm and large varices are those greater than 5mm. Small varices should be followed with repeat EGDs. Another procedure that is currently being studied for screening for varices is esophageal capsule endoscopy. Pilot studies suggest it is safe and well tolerated. The diagnosis of variceal hemorrhage is made when endoscopy shows one of the following: active bleeding from a varix, clots overlying a varix, or absence of another potential source of bleeding.

Treatment

Cirrhosis and Acute episode of hemorrhage

- Start two large bore IVs for intravascular volume support and send a type and crossmatch for blood products in case necessary
- Be prepared to intubate patient as aspiration of blood occurs frequently
- Administer antibiotics clinical trials have proven that this decreases rate of infection and early rebleeding
- Somatostatin/Octreotide causes splanchnic vasoconstriction. The advantage of octreotide is that it can stop variceal hemorrhage in up to 80% of patients and is nearly devoid of side effects.
- Endoscopic variceal ligation effective 80-90% of the time
- TIPS procedure if disease is severe

Cirrhosis and Medium/Large vessels without bleeding

- High risk of hemorrhage non-selective B-blockers (NSBB) or endoscopic variceal ligation (EVL) are indicated
- Not at high risk of hemorrhage NSBB is preferred. EVL should be used in patients with contraindications to NSBB. If using NSBB, follow up EGD is not necessary

Why non-selective B-blockers? NSBB (propanolol, nadolol) are thought to help treat varices because they provide several desired effects:

- O Decrease cardiac output via B1-receptor blockade
- Unopposed alpha vasoconstriction leads to arteriolar splanchnic vasoconstriction
- Less blood inflow to portal system

► Cirrhosis and small varices without bleeding

- If small risk of hemorrhage, non-selective b-blockers should be used for the prevention of first variceal hemorrhage
- If EGD determines there is no risk of bleeding, NSBB can be used but there is no supporting data
- If no B-blockers are given, a repeat EGD in 2 years is recommended

▶ Cirrhosis without varices

• If the absence of varices, there is no evidence to support the use of non-selective B-Blockers

This week's case was written by Anuj Gupta. Anuj is a 4th year medical student from NSU-COM. Anuj rotated at Broward Health North in March 2015.