emDocs

The EM Educator Series

The EM Educator Series: The Sick Patient with Cirrhosis

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Cases:

#1: A 43-year-old male with past history of cirrhosis presents after several episodes of hematemesis. He is hypotensive at 73/42 mm Hg and HR 124 bpm.

#2: A 51-year-old female with a history of alcohol abuse and cirrhosis presents with abdominal pain and fever. You detect a fluid wave on exam with shifting dullness, and she is diffusely tender.

Questions for Learners:

1) Are there typical lab and imaging findings in patients with cirrhosis?
2) How do you organize the resuscitation in the sick patient with cirrhosis? What are your priorities?
3) How do you manage the airway in the patient with severe hematemesis?
4) What about the patient with severe GI bleeding and cirrhosis?
5) What infectious disease considerations are there? What about spontaneous bacterial peritonitis?
6) What causes hepatic encephalopathy? What are alternative etiologies for altered mental status in these patients? How do you treat hepatic encephalopathy?
7) What do you need to consider regarding liver disease and the coagulation pathway?
8) Pearls & Pitfalls

Suggested Resources:

- ✓ Articles:
  - emDOCs – Approach to the Sick Cirrhotic Patient
  - emDOCs – Spontaneous Bacterial Peritonitis – Pearls & Pitfalls
  - emDOCs – EM@3AM: Hepatic Encephalopathy
  - PulmCrit – Coagulopathy management in the bleeding cirrhotic: Seven pearls and one crazy idea
  - R.E.B.E.L. EM – Should You Give Albumin in Spontaneous Bacterial Peritonitis (SBP)?
  - First 10 EM – Management of the Massive GI Bleed
- ✓ Podcast/Vodcast:
  - EMCrit – EMCrit Podcast 5 – Intubating the Critical GI Bleeder
  - EMCrit – Podcast 196 – Having a Vomit SALAD with Dr. Jim DuCanto – Airway Management Techniques during Massive Regurgitation, Emesis, or Bleeding
  - CORE EM – Episode 24.0 – Hepatic Encephalopathy
  - EM Cases – Episode 101 GI Bleed Emergencies Part 1
  - EM Cases – Episode 102 GI Bleed Emergencies Part 2

Answers for Learners:

1) Are there typical lab and imaging findings in patients with cirrhosis?

Labs:

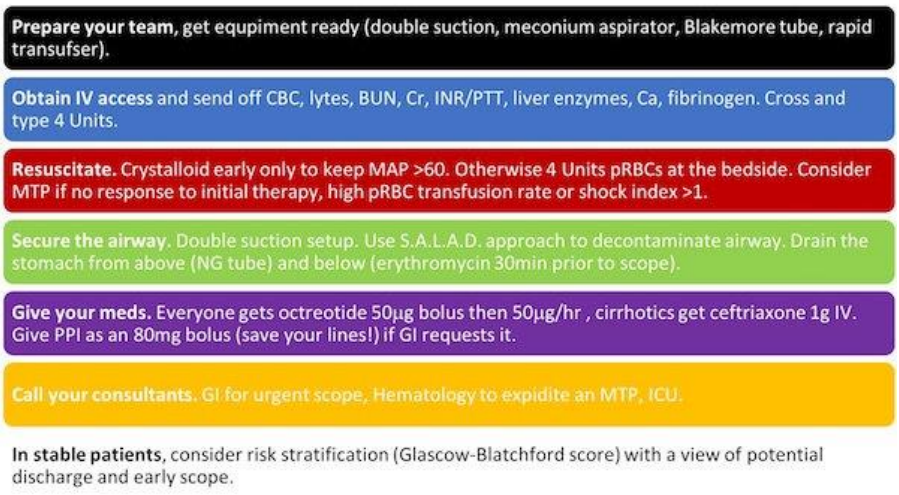
- CBC: Anemia, thrombocytopenia
- BMP: Hyponatremia, AKI, Elevated BUN
- Transaminitis
- Coags: Elevated INR
- Elevated ammonia

Imaging:

- Nodular liver
- Ascites
- Portal or splenic vein thrombosis
- Varicies (gastric / esophageal)

2) How do you organize the resuscitation in the sick patient with cirrhosis? What are your priorities?

- Ensure that the patient is responsive. If the patient is unresponsive and has no pulse, begin Advanced Cardiac Life Support (ACLS) measures.
- Ensure that the patient is protecting his or her airway. If the patient has massive hematemesis or shows poor mentation, have a low threshold to initiate advanced airway measures (see below).
- Check the lungs and breath sounds for equal chest rise and to evaluate for other abnormal breath sounds.
- Intravenous access is critically important in these patients, and ensuring bilateral large bore IV access is essential. If you cannot obtain IV access, then acquire an intraosseous (IO) line. In the initial resuscitation stages, IV and IO access are better than central access.
- For circulation, repeat VS and BP readings liberally. If the patient is hypotensive and not bleeding, an IV fluid bolus may help. Adequate perfusion should be measured by clinical factors such as mental status, capillary refill, pulse pressure, HR, pulse strength, urine output and mean arterial pressure (MAP). Using more than one clinical factor is best. If the patient is bleeding, start a transfusion with 1:1:1 ratio of pRBC:FFP:platelets. These patients often are coagulopathic. Replacing fibrinogen (if less than 100mg/dL) with cryoprecipitate can also be helpful. Tranexamic acid (TXA) may also be useful.
- Ensure you adequately expose the patient and look for signs of infection.
- Complete a quick neurologic exam (gross cranial nerve, motor, sensory, and cerebellar exams).
- Always check an initial blood glucose, as these patients are often hypoglycemic.



3) How do you manage the airway in the patient with severe hematemesis?
4) What about the patient with severe GI bleeding and cirrhosis?

Intubating these patients can be difficult. The concepts of no desaturation (NO DESAT) and delayed sequence intubation (DSI) can be beneficial in decreasing morbidity and mortality. Pre-oxygenating and de-nitrogenating the lungs prior to intubation will provide an oxygen reservoir prior to intubation. This is particularly helpful in patients like the cirrhotic patient, who may desaturate more quickly during intubation. To perform DSI, place the patient on supplemental oxygen by NC. If needed, add a facemask with 15 liters (L) O2. If the patient continues to demonstrate low oxygen levels, non-invasive positive pressure ventilation (NIPPV) may be considered. However, use caution and avoid NIPPV in patients with active hematemesis. Ketamine is a useful medication to administer during DSI as it helps reduce patient discomfort and agitation, allowing for adequate pre-oxygenation and intubation. Using ketamine will also allow you to place a nasogastric tube, which will decrease aspiration risk and clear the stomach of any present blood. A NG tube should only be used to clear the stomach of potential aspiration material and not to diagnose an upper gastrointestinal (GI) bleed.

EMCrit (www.emcrit.org) provides an excellent summary on intubating patients with GI bleeding. This can work with any sick cirrhotic patient. The steps include:

(1) Empty the stomach using a NG tube, and administer metoclopramide 10mg IV.

(2) Intubate the patient with the head of the bed elevated to 45 degrees. Have suction ready.

(3) Ensure adequate pre-oxygenation. Without pre-oxygenation, these patients may rapidly desaturate once medications are provided.

(4) Use smaller doses of the induction and sedation medications. Many of these patients will already be hypotensive and/or altered and thus, will require lower doses of sedation medication (s).

(5) On the other hand, use higher doses of the paralytic medication given during induction and intubation. Paralytic medications such as rocuronium will also augment lower esophageal tone.

(6) Maximize your equipment and have backup techniques and tools ready. These include a videoscope, direct laryngoscope, bougie, laryngeal mask airway (LMA), suction set up, and a meconium aspirator.

(7) If you fail the first attempt, bag slowly and gently, and consider placing an LMA.

(8) If the patient vomits, place him or her in Trendelenburg position to keep emesis contents out of the lungs.

(9) The meconium aspirator can be attached to the endotracheal tube (ETT) for improved suction if your baseline suction device is weaker.

(10) Expect the patient to aspirate with the intubation and be prepared for massive systemic inflammatory response (SIRS).

5) What infectious disease considerations are there? What about spontaneous bacterial peritonitis?

Complications of cirrhosis most commonly include infection but also may include aspiration, hematemesis, encephalopathy and renal failure. The administration of antibiotics will dramatically decrease mortality and even recurrent bleeding risk. Common infections include urinary tract infections (UTI) (29%), SBP (23%), and respiratory infections (11%). Administering antibiotics such as ceftriaxone 1g or cefotaxime 2g is essential, as up to 20% of patients with cirrhosis will have an infection at time of admission. In addition, up to half of patients with cirrhosis will develop infection over the course of hospitalization. These patients are also at high risk for aspiration. Although it is controversial as to whether endotracheal intubation increases risk of aspiration, it should be completed to protect the airway in the setting of severe hematemesis. Placement of a NG tube can assist in decompressing the stomach and clearing blood. These patients are also at increased risk of encephalopathy and renal failure with variceal bleeding (these will be covered later).

SBP is defined as an infection of ascitic fluid. Most cases are due to E. coli and/or Klebsiella. SBP is usually a sign of end-stage liver disease. Classic signs of this disease include fever (most common), abdominal pain, and altered mental status. Abdominal pain and altered mental status are often subtle, and because these patients are baseline hypothermic, a definition for fever in these patients should be 37.8oC. On exam, any abdominal tenderness, increase in ascites, or hypotension should raise one's suspicion for SBP. Other signs and symptoms include diarrhea, ileus, hypothermia, acidosis, and azotemia. If the patient has a temperature greater than 37.8oC, abdominal pain/tenderness, change in mental status, or ascitic neutrophil (PMN) count greater than 250 cells/mm3, then perform a paracentesis and start antibiotics.6,26-30 Each hour in delay of paracentesis increases mortality by 3.3%.

SBP is diagnosed with an ascitic fluid PMN count ≥250 cells/mm3, > 1000 white blood cells (WBC), positive culture results, and exclusion of any secondary cause (such as a surgical infection). Send the ascitic fluid for cell count/differential, culture, Gram stain, albumin, glucose, protein, LDH, amylase, and bilirubin. Send at least 20 cc of ascitic fluid in two separate blood culture bottles, which can increase yield by 25%.

Once cultures are obtained, start antibiotics immediately (either cefotaxime 2g IV or ceftriaxone 1g IV). Cefotaxime 2g IV every 8 hours provides high antibiotic levels in the ascitic fluid. Renal failure develops in up to 40% of patients with SBP, and this can be decreased with administration of IV albumin infusion at 1.5g/kg.

There has been literature on utilizing urine dipstick to quickly diagnose SBP if the dipstick returns positive for leukocyte esterase and/or nitrates. However, sensitivities vary from 31% to 100%, with specificities of 81% to 100%. This may help rule in the diagnosis of SBP, but it cannot be used to rule out disease. A dipstick device designed specifically for diagnosing infection of ascitic fluid is currently in development

Other tests of peritoneal fluid have shown promise in diagnosing SBP. An ascites lactate level of greater than 25mg/dL has demonstrated sensitivity and specificity approaching 100%. Along with ascitic fluid neutrophils, a peritoneal fluid pH < 7.35 has also shown promise in diagnosing SBP. Lactoferrin (an iron binding protein present in PMNs) presence in ascitic fluid has been shown in one study to have a sensitivity of 96% and specificity of 87%. However, this test requires further validation.

6) What causes hepatic encephalopathy? What are alternative etiologies for altered mental status in these patients? How do you treat hepatic encephalopathy?

This is primarily a diagnosis of exclusion that occurs in patients with severe liver disease, primarily due to nitrogen waste accumulation and increased metabolism of ammonia to glutamine in the CNS. It occurs with increased nitrogen load, decreased toxin clearance, and alteration in neurotransmitters.

Precipitants include GI bleed, high dietary protein, infection (SBP, UTI), low potassium, alkalosis, constipation, dehydration, vomiting, opioids, benzodiazepines, alcohol, and others.

Hepatic encephopathy is a diagnosis of exclusion. Consider intracerebral hemorrhage/subdural hematoma, hypoglycemia, hyper/hyponatremia, renal failure, sepsis/SBP, intoxication/overdose, Wernicke-Korsakoff syndrome, etc.

Identify and correct precipitating cause(s) thorough history, exam, labs, and imaging as indicated to evaluate for GI bleed, infection (SBP), hypoglycemia, intravascular hypovolemia, renal failure (hepato-renal syndrome), electrolyte derangement, hepatocellular CA, or hepatic/portal vein thrombosis.

Treat underlying cause and lower serum NH3: Lactulose for acute portal systemic encephalopathy: 20-30g (30-45cc) every hour to bowel movement, reduce to TID/QID, and titrate to 2-3 soft stools QD. Rifaximin for those intolerant of lactulose or failing to improve after 48 hours of therapy (off-label: 400mg q 8hrs for 5-10 days) can be used, as can neomycin (associated with nephrotoxicity and ototoxicity). Treat hypokalemia, which contributes to renal ammonia production.

Other treatments include polyethylene glycol, flumazenil, L-carnitine, acarbose, sodium benzoate, melatonin, serotonin antagonists, and opioid antagonists.

7) What do you need to consider regarding liver disease and the coagulation pathway?

If the patient is bleeding, start a transfusion with 1:1:1 ratio of pRBC:FFP:platelets. These patients often are coagulopathic. Replacing fibrinogen (if less than 100mg/dL) with cryoprecipitate can also be helpful. Tranexamic acid (TXA) may also be useful.

Although I am not certain that a balanced transfusion is necessary, GI bleed patients will often have an elevated INR, and early empiric administration of FFP with the packed cells definitely makes some sense.

If the patient is on warfarin, I would give the 4 factor prothrombin complex concentrate complex that we stock immediately. (Dworzynski 2012) If the patient is on one of the novel anticoagulants – well, they do wear off eventually. For renal failure patients, a dose of DDAVP (0.4mcg/kg IV) is reasonable. I am not aware of any quality evidence for tranexamic acid (1gram IV), but extrapolating from what we know about other bleeding conditions, I think it is reasonable to give.