Hi Joan, when you submit this form, the owner will be able to see your name and email address.

1. Patient Type *
   - Adult (>18yo or older)
   - Pediatric (<18yo)

2. Clinical Scenario: *

   Your patient presents for the 3rd time in a month with sudden intense abdominal pain, an elevated lactate, and diaphoretic in appearance. His pain is out of proportion to his exam, in which his abdomen is soft, NTNI. He gets his 3rd CTA of the abdomen for mesenteric ischemia, which is again negative. Symptoms and lactate improve with fluids. The internist admitting the patient suggests he likely has NOMI "non-occlusive mesenteric ischemia.”

3. Clinical Question: *

   How is NOMI diagnosed and treated, what can be done for this patient?

4. Summary/Assessment: *

   Non-occlusive mesenteric ischemia "NOMI" is the cause of 20-30% of acute mesenteric ischemia, and is a diagnosis of exclusion when there is no evidence of embolism, dissection, or strangulation. It is defined as insufficient oxygen delivery despite normal vasculature. It is found in patients with heart failure, peripheral arterial disease, septic shock, vasoconstrictive medications including digoxin, cocaine, recent cardiac surgery, and dialysis. This patient had prior bypass and PAD. It can be found in ICU patients requiring high doses of vasopressors. NOMI occurs with splenectomy vasoconstriction or vasospasm of the intestinal vessels. The patient will present with acute abdominal pain out of proportion to exam. NOMI can have a high mortality (70-80%) due to difficulty in diagnosing the disease. CT scan is the preferred immediate imaging technique, which should demonstrate narrowing or reduced vascular filling. It can be followed with selective angiography. Some studies show selective mesenteric arteriography can relieve spasm resulting in a missed diagnosis. This imaging technique also allows for local injection of a vasodilator if needed (either papaverine or nitroglycerin). General treatment includes reversing the low flow state with IVF resuscitation, and treatment of the underlying cause-heart failure, sepsis, removal of vasoconstricting medications. IVF resuscitation is the initial management of NOMI. Interestingly, the literature differs in how critical care and gastroenterology describe this disease. Critical care literature regards it as "very common" and it is included in multi-organ failure of severe disease. It is a complication of the patient’s cause of critical illness other sepsis, surgery, etc. It is thought to lead to "weakness" of the bowel wall leading to translocation of bacteria. Enteral nutrition of these patients may help improve ischemia. Gastroenterologists consider it a rare initial presentation of acute mesenteric infarction most likely attributed to different drugs or renal disease, where the patient presents with abdominal pain first. Colonoscopy is recommended as the gold standard for early presentations.

5. References: *

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1. Patient Type *
   - Adult (18yo or older)
   - Pediatric (<18yo)

2. Clinical Scenario: *

   GI was a 76 y/o M, w/his/o COPD s/p trach, RA, NSTEMI, HTN, and prior bleeding PUD while on anticoagulation, who p/w 2 days worsening weakness and lightheadedness, and dark stools. His PBT was positive, and his Hgb was 6.1. He was typed & crossed for blood, and GI was consulted to see him after admission. The GI fellow asked if we had given IV PPI, octreotide, ceftriaxone.

3. Clinical Question: *

   What is the utility of these treatments in presumed non-variceal UGIB?

4. Summary/Assessment: *

   First, acid suppression. Intuitively, it makes sense that acid suppression therapy would be beneficial in UGIB, due to either ulcers or varices. However, a Cochrane review of IV PPI bx for UGIB prior to endoscopy found no significant differences in the clinically-important outcomes of mortality, rebleeding, or need for surgery. In fact, the National Institute for Health and Care Excellence (NICE) in the UK directly states: “Do not offer acid suppression drugs (proton pump inhibitors or H2-receptor antagonists) before endoscopy to patients with suspected non-variceal upper gastrointestinal bleeding.” In contrast, several high-quality RCTs have found that IV PPI bx + endoscopy does significantly reduce rate of rebleeding, need for further transfusion, and hospital LOS. (However, the trials showing the greatest benefit to IV PPI bx in PUD were conducted in Asia, and there actually trends toward harm in European studies.) Next, octreotide. Somatostatin or its analogue octreotide are routinely used in variceal UGIB, although the data supporting their use is lacking (a Cochrane review of their use found no benefit to mortality or need for transfusion). Regarding nonvariceal UGIB, there was a meta-analysis of somatostatin/octreotide vs H2 blockers or placebo, published in 1997. The study authors found an overall modest effect on the outcomes of “ongoing bleeding” or “rebleeding”, which seemed most pronounced for patients in whom endoscopy would be delayed or unavailable. There was no significant effect on need for surgery or transfusion requirement. Lastly, antibiotics. The routine use of antibiotics for variceal UGIB is fairly well-established. Up to 20% of patients admitted with variceal bleeds have a bacterial infection on admit, and up to 50% will develop such an infection during their stay. These patients have increased mortality. The NNT, citing the Cochrane review of antibiotics given for variceal bleeding, lists an NNT of 22 for mortality, and NNT 4 for prevention of infection. However, I could find no studies directly investigating the use of antibiotics for nonvariceal, nonmircotic UGIB. Take-home: There is limited data to support the use of commonly-used pre-endoscopy IV medications in nonvariceal UGIB. The best data support the use of a restrictive transfusion strategy (Hgb greater than 7), and early (within 24 hours) endoscopy for actively-bleeding lesions.

5. References: *