A Late Presentation of COVID-19 Induced Bowel Ischemia

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Introduction

- COVID-19 produces a systemic inflammatory response.
- Literature documents cases of acute bowel ischemia via superior mesenteric thrombosis.
- Lack of data on chronic systemic effects of COVID-19 infection.
- We report a case of late presentation of bowel ischemia and stricture in a patient with a history of COVID-19.

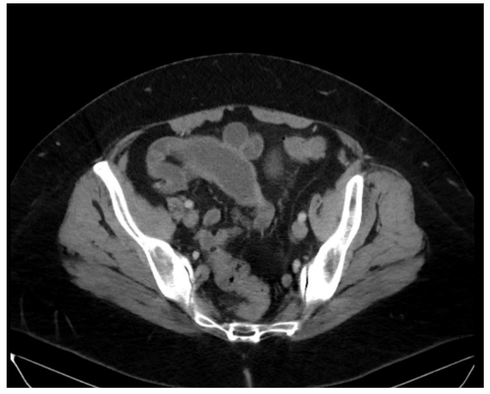
Case Presentation

•55 YO G3P3 w/ Asymptomatic COVID+

•PMH: Uterine Fibroids

•PSxH: BTL (2004) & RATH w/ BSO (2015)

•FH: Ovarian Cancer (Mother)



Clinical Course

•**June 2020**: Asymptomatic COVID-19+

•**July-August 2020**: Melena without abdominal pain

•**Winter 2020**: Upper EGD reveals hiatal hernia

•**April 2021**: Acholic Stools → Colonoscopy reveals diverticulosis

•**June 2021**: Admitted with crampy epigastric/RUQ abdominal pain with associated N/V. CT scan of abdomen and pelvis demonstrated cholelithiasis and distended gallbladder. Patient then underwent uneventful laparoscopic cholecystectomy. Pathology revealed mild chronic cholecystitis with cholelithiasis. Patient was discharged several days after cholecystectomy feeling better.

•**July 2021**: Admitted to acute care facility 3x for recurrent SBO with non-operative management.

•**July 30 2021**: Upper and Lower Endoscopy revealed no abnormalities.

•**August 1 2021**: Discharged with no symptoms

•**August 6 2021**: Given Dummy Capsule. Serial abdominal XRays revealed failure of capsule to traverse small bowel and enter the colon.

•**August 10 2021**: Patient admitted with PO intolerance, crampy abdominal pain, and emesis. Abdominal CT confirms partial SBO.

•**August 10 2021**: Patient underwent laparoscopic small bowel resection with functional entero-enterostomy. Grossly, at time of surgical intervention it appeared patient had benign isolated small bowel stricture. Pathology revealed a 1.5 X 1.5 cm benign ulceration with active inflammation.

•**August 14 2021**: Discharged home. She has resolved her primary persistent symptoms of crampy abdominal pain, weight loss, and PO intolerance.

Pathophysiology

• A COVID-19—induced hyperinflammatory and hypercoagulable state is characterized by activation of leukocytes and endothelial cells.

• Elevated inflammatory markers: Erythrocyte Sedimentation Rate, C-Reactive Protein, IL-2, IL-6, IL-8, and IL-10.

• *Disseminated Intravascular Coagulation*: overactivation of platelets and coagulation factors with subsequent thrombosis– elevated D-Dimer and Fibrin Degradation Products.

• Inflammation and Hyper-Coagulation contribute to thrombosis in the gastrointestinal vasculature.

• Acute thrombosis does not allow sufficient time for collateral circulation to develop, causing ischemia and infarction of gastric tissue.

• Ischemia causes chronic transmural inflammation with subsequent sub-mucosal and sub-serosal fibrosis – thus forming a stricture. Fibrotic bowel does not undergo peristalsis and inhibits passage of intraluminal contents.

• In another scenario, ischemic tissue can form ulcerations, which have the potential to perforate and form intra-intestinal fistulas with adjacent bowel and subsequent obstruction.

Conclusions

•COVID-19 can be responsible for both acute and chronic embolic and thrombotic events.

•Clinicians need to maintain a high index of suspicion in patients with a history of COVID-19 for bowel ischemia.