Journal of Community Hospital Internal Medicine Perspectives

Volume 13 | Issue 4 Article 6

2023

# A Rare Case of Pancolonic Ischemia Complicated by Fusobacterium Necrophorum Bacteremia: A Review of Colonic Ischemia for Internists

Alaina Chodoff
*Department of Internal Medicine, Greater Baltimore Medical Center, Towson, Maryland*,
achodoff@gbmc.org

Diana Whitehead
*Department of Gastrointestinal Disease, Director of Inflammatory Bowel Disease, Greater Baltimore Medical Center, Towson, Maryland*

Beryl Otieno
*Department of Internal Medicine, Chief Resident, Greater Baltimore Medical Center, Towson, Maryland*

Joseph Fuscaldo
*Department of Internal Medicine, Medical Director of Quality Improvement, Greater Baltimore Medical Center, Towson, Maryland*



## Recommended Citation

Chodoff, Alaina; Whitehead, Diana; Otieno, Beryl; and Fuscaldo, Joseph (2023) "A Rare Case of Pancolonic Ischemia Complicated by Fusobacterium Necrophorum Bacteremia: A Review of Colonic Ischemia for Internists," *Journal of Community Hospital Internal Medicine Perspectives*: Vol. 13: Iss. 4, Article 6.
DOI: 10.55729/2000-9666.1197
Available at: https://scholarlycommons.gbmc.org/jchimp/vol13/iss4/6

CASE REPORT

# A Rare Case of Pancolonic Ischemia Complicated by *Fusobacterium Necrophorum* Bacteremia: A Review of Colonic Ischemia for Internists

Alaina Chodoff [a,*], Diana Whitehead [b], Beryl Otieno [c], Joseph Fuscaldo [d]

[a] Department of Internal Medicine, Greater Baltimore Medical Center, Towson, MD, USA
[b] Department of Gastrointestinal Disease, Director of Inflammatory Bowel Disease, Greater Baltimore Medical Center, Towson, MD, USA
[c] Department of Internal Medicine, Chief Resident, Greater Baltimore Medical Center, Towson, MD, USA
[d] Department of Internal Medicine, Medical Director of Quality Improvement, Greater Baltimore Medical Center, Towson, MD, USA

**Abstract**

**The varied clinical presentation and objective findings associated with colonic ischemia (CI) overlap with multiple disease processes. A high index of suspicion is critical for timely diagnosis and prognostication to avoid delays in treatment. We present a case that highlights the challenges of diagnosing CI and the high morbidity associated with severe disease. Case report: A sixty-four-year-old female presented to our community hospital with acute onset abdominal pain, nausea, and diarrhea, complicated by septic shock. She was initially given a diagnosis of infectious colitis until a colonoscopy was performed revealing extensive pancolonic ulcerations. Histopathological features on biopsy were most consistent with colonic ischemia. Blood cultures grew *Fusobacterium Necrophorum*. Surgery was avoided due to the high morbidity and mortality of performing a total colectomy and ileostomy. The patient continued to struggle with abdominal pain, diarrhea, and hematochezia, consistent with continuous CI, leading to recurrent hospitalizations.**

## 1. Introduction

Ischemic bowel disease comprises a heterogenous group of disorders that can be divided into mesenteric ischemia and colonic ischemia (CI). CI is the most frequent form of intestinal ischemia with an incidence of 22.9/10,0000 persons-year.[1] CI is a disease with variable clinical presentation that correlates with the anatomic distribution. The most common symptoms are abdominal pain (87%), rectal bleeding (84%) and diarrhea (56%).[2] Disease severity depends on the location and length of colon involved, the duration of compromised blood flow, and the underlying degree of stenosis to the culprit vessels. Patient risk factors for severe disease must also be considered and these include age, male gender, chronic kidney disease, chronic obstructive pulmonary disease, hepatitis C positivity, history of cancer and warfarin use at the time of diagnosis.[3] We describe a rare presentation of pancolonic ischemia, highlighting some of the challenges associated with treating severe ischemic bowel disease.

## 2. Case presentation

Ms. W is a sixty-four-year-old woman with a history of hypertension, hypothyroidism, tobacco use disorder, takotsubo cardiomyopathy with recovered ejection fraction and stroke who presented with acute abdominal pain preceded by two? 2 days of nausea and vomiting. On examination, the patient appeared acutely ill with dry mucous membranes and periumbilical abdominal tenderness without peritoneal signs. The temperature was 36.4 °C, the blood pressure 65/34 mmHg, the heart rate 78 bpm, and the oxygen saturation 96% breathing ambient air. The results of laboratory studies revealed a hemoglobin of 12.0 g/dL, white blood cell count of 19,890 μL, lactic acid level of 3.4 mmol/L. Serum chemistry results

Received 24 January 2023; revised 1 April 2023; accepted 6 April 2023.
Available online 29 June 2023

* Corresponding author.
E-mail address: achodoff@gbmc.org (A. Chodoff).

https://doi.org/10.55729/2000-9666.1197

were notable for a creatinine level of 2.65 mg/dL from a baseline of 0.8 mg/dL, BUN 36 mg/dL, CO2 22mEq/L. Within a few hours of arrival, the patient developed voluminous non-bloody diarrhea. Computed tomography (CT) of the abdomen and pelvis without contrast revealed generalized colonic thickening with no obvious transition point. Moderate fluid distension of multiple loops of small bowel, the ascending colon, transverse colon, and splenic flexure was noted. Concern for stricture in the region of the proximal descending colon was suggested. Stool tests returned negative for *Clostridium Difficile* toxin. The fecal occult blood test was positive.

A presumptive diagnosis of septic shock was made secondary to infectious colitis. Blood cultures were pending at that time. Ms W received aggressive fluid resuscitation and broad spectrum intravenous antibiotic coverage with piperacillin-tazobactam. Her clinical condition required admission to the Intensive Care Unit for vasopressor support. Colorectal surgery and gastroenterology services both recommended continuing supportive care to achieve hemodynamic stability prior to performing a colonoscopy. On the second day of hospitalization, renal function improved, and a repeat CT abdomen and pelvis was performed with IV contrast. The repeat scan was notable for pancolonic thickening with more marked involvement of the left side. (See image below) Additional studies at this time resulted, including a fecal calprotectin level of 1190 μg/mg (reference range, <50), positive fecal lactoferrin, normal stool culture and blood cultures growing gram variable rods. Inflammatory markers were markedly elevated including the erythrocyte sediment rate of 105 mm/h, C-reactive protein level of 24 mg/dL, and D-dimer >20 μg/mlFEU.

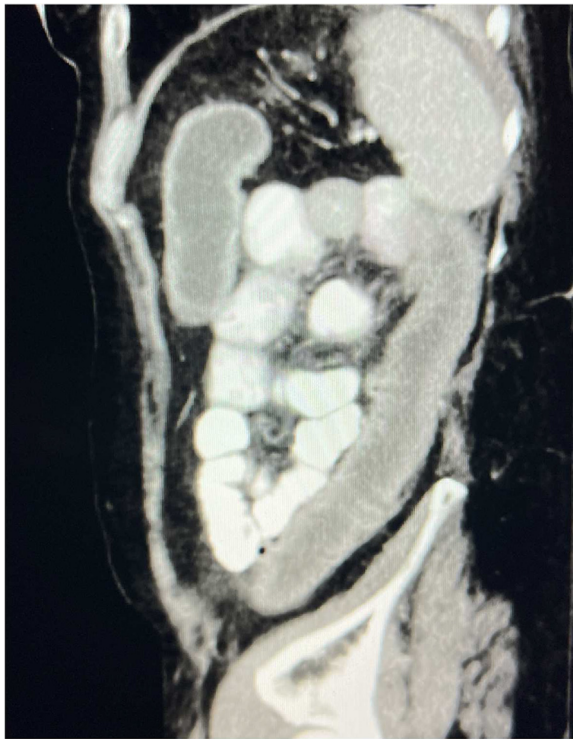

*Fig. 1. Severe wall thickening and peri-colonic stranding involving the descending colon.*

On the third day of hospitalization, the patient was downgraded to the general medical floor. A colonoscopy was performed. Findings of the procedure included scattered severe mucosal changes characterized by altered vascularity, congestion, friability, granularity, mucus, and deep ulcerations throughout the ascending, transverse, descending and sigmoid colon, consistent with severe ischemic colitis. Endoscopic biopsies confirmed pancolonic mucosal ulcerations, most severe in the transverse and descending colon, with rectal sparing. Cytomegalovirus immunostaining was negative. MRI angiography was negative for high grade stenosis of the celiac, proximal superior mesenteric or proximal inferior mesenteric arteries.

The patient's diarrhea resolved, but her abdominal pain, nausea and vomiting continued. On the seventh day of hospitalization, blood cultures returned positive for *Fusobacterium Necrophorum*. The patient remained on piperacillin-tazobactam. Consultation with colorectal surgery recommended against surgery due to the high overall morbidity and mortality of performing a total colectomy and ileostomy. Ms. W was discharged three weeks after admission as her bowel movements were normalizing and she was tolerating a low residue diet.

The patient was readmitted to a different hospital within just one week of discharge with recurrent bloody diarrhea complicated by vancomycin-resistant *Enterococcus* bacteremia. Ms. W was stabilized and discharged with a plan for follow up with the Gastroenterology team, but again returned to our hospital with abdominal pain, voluminous diarrhea, and severe anemia. Repeat CT angiogram of the abdomen and pelvis revealed persistent diffuse colonic thickening without pneumatosis or vascular occlusion. Antineutrophil cytoplasmic antibodies testing was negative. The patient's ongoing burden of symptoms and anemia were attributed to persistent ischemic bowel disease. Surgical consultation again recommended deferring surgery and monitoring for symptom improvement with a plan to repeat a colonoscopy shortly after discharge.

Ms. W ultimately sought a second opinion closer to her home. Repeat colonoscopy performed four 4 months after her initial presentation revealed deep and serpentine ulcerations in a continuous and circumferential pattern involving the transverse and

sigmoid colon sparing the rectum. Chart review revealed that she was diagnosed with IBD and prescribed steroids with plans to transition to biologic therapy. The patient died a few months later at home, but details are not available to our team. No autopsy was performed.

## 3. Discussion

In contrast to mesenteric ischemia, which most commonly arises from embolic disease, CI is the result of inadequate intestinal blood flow in 95% of cases.[4] The colon receives less blood flow compared with the rest of the GI tract and is thus more vulnerable to ischemia.[4,5] Adults older than age sixty-five are particularly susceptible to CI due to the increase in vascular resistance and increased susceptibility to hypoperfusion in the colonic microvasculature.[6] Comorbid atherosclerotic cardiovascular disease (ASCVD) is a leading risk factor for CI. Patients will commonly have at least one major risk factor for ASCVD including hypertension, type 2 diabetes, tobacco use disorder, congestive heart failure, chronic obstructive pulmonary disease, or ESRD.[5-7]

CI can be challenging to diagnose because of the nonspecific presenting symptoms and variable clinical presentation. The left colon is affected in 75% of cases and frequently occurs in the watershed areas of the colon.[8,9] The splenic flexure or "Griffith's point", describes the point between the territories formed by the middle colic branch of the superior mesenteric artery (SMA) and the right colic branch of the inferior mesenteric artery (IMA). The rectosigmoid or Sudeck's point occurs at the junction between the last sigmoid branch and the superior rectal branch of the IMA. The rectum is uncommonly involved due to dual blood supply from the splanchnic and systemic arterial systems.[10]

Isolated right colon ischemia (IRCI) occurs in about 10% of cases and carries a significantly worse prognosis.[11] Compared to segmental CI, pancolonic ischemia is rare and is more likely to occur in the setting of severe sepsis with prolonged hypotension.[12] Presumably, the SMA and IMA circulations are both affected. Pancolonic ischemia is associated with a mortality rate upwards of 21% and frequently requires surgical intervention.[12] Similar to IRCI, emergent surgical consultation is required along with initiation of broad-spectrum antibiotics.[3]

As demonstrated by our case, CI is not a straightforward diagnosis. No laboratory test or imaging modality finding is specific to CI. Low hemoglobin, hypoalbuminemia, elevated LDH and CPK may help differentiate more severe disease.[13-15] Abdominal X-ray is usually normal, although rarely may show "thumbprinting" due to submucosal edema/hemorrhage of the bowel wall.[16] A CT scan of the abdomen and pelvis with oral and IV contrast is the image study of choice to search for CI. In patients with IRCI, CT angiography should also be done to exclude acute mesenteric ischemia.[17] Findings suggestive of CI on CT scan include segmental wall thickening, peri-colonic fat stranding, thumbprinting and ascites. If infectious colitis remains on the differential, stool culture studies should be sent to evaluate for invasive bacteria, including *Escherichia coli O157:H7*, *Salmonella*, and *Shigella* species.[18] *Clostridium Difficile* colitis should especially be considered in patients who have recently been hospitalized or used antibiotics.[18]

A colonoscopy with biopsy within 48 h of hospitalization is the gold standard diagnostic study. Colonoscopy frequently shows nonspecific signs of inflammation, including segmental erythema, friability, superficial ulcerations, edematous colonic mucosa, and scattered hemorrhagic erosions.[19] The "single-stripe sign," a single linear ulcer running along the longitudinal axis of the colon, is less commonly visualized but is highly specific for CI.[20] Histologic findings include focal crypt dropped out, lamina propria and submucosal hemorrhage and edema, hemosiderin laden macrophages in the submucosa, fibrin thrombi in capillaries with infiltration of neutrophils, erosion, and granulation tissue hyperplasia.[21] Necrosis and ghost cells i.e., preserved cellular outline with empty cellular content are pathognomonic, but infrequently seen.[9] In gangrenous CI, the colon may appear black or grey green with severe epithelial loss.[9]

Despite the above work-up, CI can be difficult to differentiate from inflammatory bowel disease and infectious colitis. On colonoscopy, the segmental distribution and abrupt transition between injured and non-injured mucosa, supports ischemia over inflammatory bowel disease, although this can be more difficult to detect in severe cases presenting with pancolitis and transmural infarction.[22] In addition, because the rectum receives its blood supply from three distinct arterial branches, the rectum is classically spared in a colonic ischemic event. Patients who develop chronic CI may subsequently be misdiagnosed as having inflammatory bowel disease. The incidence of chronic CI is quite variable across studies, ranging from 0 to 25%.[3] Glucocorticoids are not recommended for the treatment of CI, except in the presence of vasculitis. Patients with CI will respond poorly to immunosuppressive therapy and have an increased risk of perforation on steroids.[23]

Although most cases of CI are transient and spontaneous recovery is expected with supportive management, severe injury caused by transmural necrosis of the bowel can lead to gangrenous colitis (10–20%) and fulminate universal colitis (1%).[24-26] Patients with mild to moderate CI should be managed conservatively with bowel rest and intravenous fluid to improve colonic perfusion. Prophylactic antibiotics are warranted for moderate-severe disease. Conditions predisposing to colonic hypoperfusion should be promptly evaluated and managed including heart failure, severe anemia, hypovolemia, and septic shock. Surgical intervention is required for cases of severe CI when signs of necrotic bowel are present. Patients with severe disease that do not undergo surgery require close clinical follow up to monitor for ongoing symptoms, protein-losing colopathy or recurrent bouts of sepsis that would subsequently warrant surgery.[3] Failure of nonsurgical management is more likely when the patient presents with sepsis, non-bloody diarrhea, bilateral or right-sided disease distribution.[3] Postoperative mortality rates for CI remain high for patients who require surgery due to the severity of the underlying disease and the frequency of postoperative complications. In a large systemic review of the management of CI, surgery was required in 19.6% of patients in the included studies, of whom 39.3% died.[27]

## 4. Conclusion

Our case demonstrates the challenges of recognizing, triaging, and managing CI. Front line clinicians need to be familiar with the spectrum of presentation, involved anatomy, and treatment options. Supportive care is the main treatment modality for mild-moderate disease. Close monitoring for surgical intervention is warranted for patients who present with severe disease. There is a need for improved diagnostics and treatment modalities to reduce the morbidity associated with this disease. Creation and dissemination of revised expert consensus guidelines and management algorithms would be helpful. For now, clinician knowledge, concern, and high index of suspicion appear to be the best tools available.

## Conflict of interest

No conflict of interest.

## References


1. Yadav S, Dave M, Varayil JE, et al. A population-based study of incidence, risk factors, clinical spectrum, and outcomes of ischemic colitis. *Clin Gastroenterol Hepatol*. 2015 Apr;13(4):731–738. https://doi.org/10.1016/j.cgh.2014.07.061.
2. Longstreth GF, Yao JF. Epidemiology, clinical features, high-risk factors, and outcome of acute large bowel ischemia. *Clin Gastroenterol Hepatol*. 2009 Oct;7(10):1075–1080. https://doi.org/10.1016/j.cgh.2009.05.026.
3. Brandt LJ, Feuerstadt P, Longstreth G, et al. ACG clinical guideline: epidemiology, risk factors, patterns of presentation, diagnosis, and management of colon ischemia (CI). *Am J Gastroenterol*. 2015;110(1):18–44. https://doi.org/10.1038/ajg.2014.395.
4. Haglund U, Bulkley GB, Granger DN, et al. On the pathophysiology of intestinal ischemic injury. Clinical review. *Acta Chir Scand*. 1987;153(5–6):321–324.
5. Demetriou G, Nassar A, Subramonia S. The pathophysiology, presentation and management of ischaemic colitis: a systematic review. *World J Surg*. 2020;44:927–938. https://doi.org/10.1007/s00268-019-05248-9.
6. Iacobellis F, Narese D, Berritto D, et al. Large bowel ischemia/infarction: how to recognize it and make differential diagnosis? A review. *Diagnostics*. 2021 Jun;11(6):998. https://doi.org/10.3390/diagnostics11060998.
7. Iacobellis F, Narese D, Berritto D, et al. Large bowel ischemia/infarction: how to recognize it and make differential diagnosis? A review. *Diagnostics*. 2021 Jun;11(6):998. https://doi.org/10.3390/diagnostics11060998.
8. Gandhi SK, Hanson MM, Vernava AM, et al. Investigation and management of ischemic colitis. *Cleve Clin J Med*. 2003;70(11):928–930. https://doi.org/10.3949/ccjm.70.11.920, 920–1, 925–6.
9. Ahmed M. Ischemic Bowel Disease in 2021. *World J Gastroenterol*. 2021;27(29):4746–4762. https://doi.org/10.2748/wjg.v27.i29.4746.
10. Washington C, Carmichael JC. Management of ischemic colitis. *Clin Colon Rectal Surg*. 2012;25(4):228–235. https://doi.org/10.1055/s-0032-1329534.
11. Brandt LJ, Boley SJ. Colonic ischemia. *Surg Clin*. 1992;72:203–229.
12. Brandt LJ, Feuerstadt P, Santolaria S, et al. Anatomic patterns, patient characteristics, and clinical outcomes in ischemic colitis: a study of 313 cases supported by histology. *Am J Gastroenterol*. 2010;105(10):2245–2252. https://doi.org/10.1038/ajg.2010.217.
13. Montoro MA, Brandt LJ, Santolaria S, et al. Clinical patterns and outcomes of ischaemic colitis: results of the Working Group for the Study of Ischaemic Colitis in Spain (CIE study). *Scand J Gastroenterol*. 2011;46(2):236–246. https://doi.org/10.3109/00365521.2010.525794.
14. Mosele M, Cardin F, Inelmen EM, et al. Ischemic colitis in the elderly: predictors of the disease and prognostic factors to negative outcome. *Scand J Gastroenterol*. 2010;45(4):428–433. https://doi.org/10.3109/00365520903513225.
15. Huguier M, Barrier A, Boelle P, et al. Ischemic colitis. *Am J Surg*. 2006;192(5):679–684. https://doi.org/10.1016/j.amjsurg.2005.09.018.
16. Trotter JM, Hunt L, Peter MB. Ischaemic colitis. *BMJ*. 2016;22:355. https://doi.org/10.1136/bmj.i6600. i6600.
17. Elder K, Lashner BA, Solaiman FA. Clinical approach to colonic ischemia. *Cleve Clin J Med*. 2009;76(7):401–409. https://doi.org/10.3949/ccjm.76a.08089.
18. Maimone A, De Ceglie A, Siersema PD, et al. Colon ischemia: a comprehensive review. *Clin Res Hepatol Gastroenterol*. 2021 Nov;45(6), 101592. https://doi.org/10.1016/j.clinre.2020.101592.
19. Zou X, Cao J, Yao Y, et al. Endoscopic findings and clinicopathologic characteristics of ischemic colitis: a report of 85 cases. *Dig Dis Sci*. 2008;54:2009–2015. https://doi.org/10.1007/s10620-008-0579-1.
20. Zuckerman GR, Prakash C, Merriman RB, et al. The colon single-stripe sign and its relationship to ischemic colitis. *Am J Gastroenterol*. 2003;98(9):2018–2022. https://doi.org/10.1111/j.1572-0241.2003.07633.x.
21. Cerilli LA, Greenson JK. The differential diagnosis of colitis in endoscopic biopsy specimens: a review article. *Arch Pathol Lab*

*Med*. 2012;136(8):854–864. https://doi.org/10.5858/arpa.2012-0205-RA.
22. Galandiuk S, Morpurgo E, et al. Colon and rectum. In: Townsend CM, ed. *Sabiston textbook of surgery*. 21st ed. Elsevier, Inc; 2022:1320–1400, 2022.
23. Theodoropoulou A, Koutroubakis IE. Ischemic colitis: clinical practice in diagnosis and treatment. *World J Gastroenterol*. 2008;14(48):7302–7308. https://doi.org/10.3748/wjg.14.7302.
24. Añón R, Boscá MM, Sanchiz V, et al. Factors predicting poor prognosis in ischemic colitis. *World J Gastroenterol*. 2006;12(30):4875–4878. https://doi.org/10.3748/wjg.v12.i30.4875.
25. Baixauli J, Kiran RP, Delaney CP. Investigation and management of ischemic colitis. *Cleve Clin J Med*. 2003 Nov;70(11):920–921. https://doi.org/10.3949/ccjm.70.11.920, 925–6, 928-921.
26. Paterno F, McGillicuddy EA, Schuster KM, et al. Ischemic colitis: risk factors for eventual surgery. *Am J Surg*. 2010 Nov;200(5):646–650. https://doi.org/10.1016/j.amjsurg.2010.07.005.
27. O'Neill S, Yalamarthi S. Systematic review of the management of ischaemic colitis. *Colorectal Dis*. 2012 Nov;14(11):751–763. https://doi.org/10.1111/j.1463-1318.2012.03171.