CASE REPORT

WILEY

A case of colonic obstruction combined with ischemic colitis

Szemein Gan¹ | Yee Kent Liew¹ | Sohil Pothiawala²

¹Department of Emergency Medicine, Sengkang General Hospital, Singapore, Singapore ²Department of Emergency Medicine, Woodlands Health Campus, Singapore, Singapore

Correspondence

Szemein Gan, Department of Emergency Medicine, Sengkang General Hospital, Singapore, Singapore. Email: szemein.gan@mohh.com.sg

Keywords: chronic constipation, ischemic bowel, stercoral colitis

1 | INTRODUCTION

The prevalence of chronic constipation increases with age, with up to 26% of women and 16% of men who were 65 years or older self-reporting to have constipation.¹ Constipation is usually not severe and is treated with a combination of nonpharmacologic and pharmacologic medications. If left untreated, chronic constipation can lead to complications, such as fecal impaction, overflow diarrhea, hemorrhoids, and rarely inflammation, obstruction, and even ischemia of the bowel.

Stercoral colitis is an inflammatory condition of the bowel associated with chronic constipation. Ischemic bowel resulting from stercoral colitis is extremely rare; and only few cases have been reported to date. This case describes the diagnostic challenge of ischemic colitis from fecal impaction and colonic obstruction, which is a serious life-threatening complication of chronic constipation especially in elderly patients.

2 | CASE REPORT

A 72-year-old man presented with abdominal discomfort, vomiting, and constipation for 3 days. He had a medical history of diabetes, hypertension, and hyperlipidemia and was discharged from the hospital the day before after admission to the hospital for recurrent falls. On examination, he was afebrile, with a heart rate of 106 beats per minute, and blood pressure of 91/61 mm Hg. Abdominal examination revealed mild epigastric tenderness. His blood tests showed a normal white cell count of $6.10 \times 109/L$. Blood gas analysis showed metabolic acidosis with pH of 7.244, sodium bicarbonate level of 15.1 mmol/L, and serum lactate of 12 mmol/L. His serum amylase was 812 U/L (normal range 38-149 U/L). He was provisionally diagnosed to have acute pancreatitis with high anion gap metabolic acidosis and was resuscitated with intravenous crystalloids. Over the next few hours, he developed guarding over the epigastrium and periumbilical region. Repeat blood investigations showed rising serum lactate of 19.8 mmol/L despite fluid resuscitation. Computed tomography (CT) mesenteric angiogram showed bowel distension with multiple gas-fluid levels and associated rectosigmoid fecal impaction with wall thickening, which were suggestive of stercoral colitis (Figures 1 and 2). There was slight reduced opacification of the left colic and sigmoidal branches of the inferior mesenteric artery relative to its terminal branch, but there was no evidence of bowel ischemia or pancreatitis.

His lactic acidosis continued to worsen and he eventually developed septic shock. Due to the likelihood of ischemic bowel, he underwent urgent diagnostic colonoscopy (Figure 3), which revealed patchy transmural and mucosal ischemia of the rectum and the colon, extending up to the cecum. He underwent total proctocolectomy with end ileostomy and was transferred to the intensive care unit. Histopathology showed patches of mucosal ulceration, acute inflammation throughout the intestinal wall with acute serositis, and ischemic changes in the entire colon. Postoperatively, he developed oliguric Kidney Disease Improving Global Outcomes (KDIGO) stage 2 acute kidney injury secondary to septic shock, which required 3 days of continuous renal replacement therapy. He also developed small bowel obstruction. His condition stabilized after a month and he was eventually transferred to the community hospital for rehabilitation.

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made. © 2021 The Authors. Aging Medicine published by Beijing Hospital and John Wiley & Sons Australia, Ltd.



FIGURE 1 CT scan of the abdomen showing a large amount of feces in the anterior descending recto-sigmoid colon with wall thickening (red arrow) and surrounding fat stranding (yellow arrow)



FIGURE 2 CT scan of the abdomen showing large bowel distention with multiple gas-fluid levels

3 | DISCUSSION

Constipation and fecal impaction are frequently seen in the elderly. The common complications from fecal impaction include overflow fecal incontinence, acute urinary retention, and urinary tract infections. Complications, such as bowel obstruction from large fecaloma, colonic ischemia, and perforation from stercoral colitis, have been rarely reported in the literature.

Stercoral colitis is a life-threatening inflammatory bowel condition as a result of fecal impaction. Its exact incidence is not known, with an estimated postmortem stercoral ulcer incidence ranging from 0.04% to 2.3%.² Bedridden nursing home patients, diabetics, psychiatric patients on antipsychotics, and palliative patients on long-term opiates are more susceptible to chronic constipation and stercoral colitis.

The hard, stagnated fecal material leads to focal compression on the colonic wall and vasculature, leading to decreased blood flow, which eventually results in ulceration and pressure necrosis of that segment of the bowel wall. The ulceration typically occurs in areas adjacent to the fecaloma, especially on the antimesenteric side of the bowel, which is more vulnerable to ischemia. If the fecal impaction is not treated promptly, the focal pressure necrosis will eventually lead to bowel perforation and subsequent high risk of sepsis and mortality. The majority of stercoral ulcerations are found in the rectosigmoid colon, which has the narrowest colonic diameter and poor blood supply.³ Rarely, the vasculature of an entire segment of the bowel is affected. Involvement of greater than 40 cms of the colon and intestinal perforation are the highest predictors of mortality.⁴ A recent case report has described extensive ischemic colitis proximal to the site of fecal impaction from stercoral colitis.⁵

Clinical presentation of stercoral colitis can vary from vague diffuse abdominal pain to acute abdomen with peritonism. It can be confused with diverticulitis, which is relatively more common in the elderly patients. Our patient with epigastric tenderness had an elevated serum amylase leading to an initial suspicion of pancreatitis. Asymptomatic hyperamylasemia is seen in patients with inflammatory bowel disease. Eight percent of patients with Crohn's disease had significantly high serum amylase but it was not associated with pancreatitis.⁶ Hyperamylasemia is also seen in cases of increased bowel wall permeation, such as in bowel perforation or ischemia. The proposed mechanism for hyperamylasemia in bowel ischemia is the transperitoneal absorption of spilled bowel fluid, which contains amylase, into the lymphatics and systemic circulation.⁷ Serum amylase also has a prognostic value in these cases, with higher serum amylase correlating with higher mortality.⁷

Early diagnosis of patients with stercoral colitis can be challenging, with only about 10% of patients diagnosed before surgery.⁸ Laboratory results are nonspecific and they include leukocytosis and elevated acute phase reactant proteins. Elevated lactate and anion gap metabolic acidosis in patients with stercoral colitis should raise suspicion for bowel ischemia or perforation. As physical examination and laboratory findings are nonspecific, imaging is key in making the diagnosis of stercoral colitis. A CT scan of the abdomen and pelvis is the most sensitive and specific, and aids in diagnosis. The common findings include presence of fecaloma in the colon, thickening of colon wall, and pericolonic fat stranding.⁹ There can be diffuse bowel wall edema from increased intraluminal pressure, which appears as a mucosal discontinuity on CT imaging. This distinguishes stercoral colitis from uncomplicated fecal impaction, where the colonic wall is typically thin. Additional findings like dense mucosa, perfusion defects, ascites, and abnormal gas predicts its severity.¹⁰ Our patient's CT scan showed large amounts of fecal material distending the rectosigmoid colon, along with wall thickening and surrounding mild fat stranding, which were suggestive of stercoral colitis.

Once the diagnosis of stercoral colitis is confirmed, prompt initiation of appropriate therapy helps in reducing morbidity as well as mortality from its associated complications, like bowel perforation and peritonitis. Not all patients with stercoral colitis require surgery, with up to 50% of uncomplicated stercoral colitis managed conservatively using bowel cleansing, enema, and fecal disimpaction.⁹ The





FIGURE 3 Endoscopic findings of dusky colonic mucosa

best way to prevent stercoral colitis is to prevent constipation and fecal impaction. Early diagnosis of fecal impaction should prompt aggressive bowel cleansing and manual disimpaction,¹¹ which may reduce intraluminal pressure, thus decreasing the risk of subsequent bowel wall ulceration and ischemic colitis. Diagnostic delays can lead to ischemic colitis, bowel perforation, and subsequent septic shock. These patients have a mortality rate as high as 60%.² Lower morbidity and mortality have been noted in patients who were diagnosed early and managed appropriately.

Thus, stercoral colitis should be considered a potential diagnosis in elderly patients presenting with acute abdominal pain and constipation. Early diagnosis is challenging, and physicians should maintain a high level of suspicion followed by prompt radiological imaging to aid diagnosis. Delayed diagnosis can lead to bowel wall ulceration, ischemia, perforation, peritonitis, septic shock, and death. Rapid fecal disimpaction for elderly patients with constipation will help avoid the associated complications and achieve improved outcomes.

CONFLICTS OF INTEREST

Nothing to disclose.

AUTHOR CONTRIBUTIONS

Gan conceived the idea and contributed to the writing of the manuscript. Liew contributed to the writing of the manuscript. Sohil contributed to the reviewing and editing of the manuscript.

ORCID

Szemein Gan ២ https://orcid.org/0000-0001-6767-0680 Sohil Pothiawala 🝺 https://orcid.org/0000-0002-4789-4326

REFERENCES

- Gallegos-Orozco JF, Foxx-Orenstein AE, Sterler SM, Stoa JM. Chronic constipation in the elderly. Am J Gastroenterol. 2011;107:18-25.
- Naseer M, Gandhi J, Chams N, Kulairi Z. Stercoral colitis complicated with ischemic colitis: a double-edge sword. BMC Gastroentrol. 2017;17:129.
- Maurer CA, Renzulli P, Mazzucchelli L, Egger B, Seiler CA, Büchler MW. Use of accurate diagnostic criteria may increase incidence of stercoral perforation of the colon. *Dis Colon Rectum*. 2000;43:991-998.
- 4. Morano C. Stercoral Colitis. In: Sharman T, ed. *StatPearls*. Treasure Island, FL: StatPearls Publishing; 2020.
- Chew KKY, Law C, Lee D. Stercoral colitis: a rare cause of bowel ischaemia. ANS J Surg. 2020;https://doi.org/10.1111/ans.16293
- Katz S, Bank S, Greenberg RE, Lendvai S, Lesser M, Napolitano B. Hyperamylasemia in inflammatory bowel disease. J Clin Gastroenterol. 1988;10:627-630.
- Wilson C, Imrie CW. Amylase and gut infarction. Br J Surg. 1986;73:219-221.
- Canders CP, Shing R, Rouhani A. Stercoral colitis in two young psychiatric patients presenting with abdominal pain. J Emerg Med. 2015;49:e99-e103.
- Wu CH, Wang LJ, Wong YC, et al. Necrotic stercoral colitis: Importance of computed tomography findings. World J Gastroenterol. 2011;17:379-384.
- Wu CH, Huang CC, Wang LJ, et al. Value of CT in the discrimination of fatal from non-fatal stercoral colitis. *Korean J Radiol*. 2012;13:283.
- 11. Mohamed A, Nguyen M, Tsang R, LoSavio A, Khan O. Early diagnosis and treatment of stercoral colitis with an aggressive bowel regimen. *Am J Gastroenterol.* 2014;109:S399.

How to cite this article: Gan S, Liew YK, Pothiawala S. A case of colonic obstruction combined with ischemic colitis. *Aging Med.* 2021;4:58–60. <u>https://doi.org/10.1002/agm2.12145</u>