



Case Report: Postoperative Recurrent Stercoral Ulcer - Management and Prevention

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Received: April 11, 2025

Published: April 30, 2025

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Abstract

Stercoral ulcer perforation is a rare but life-threatening complication of stercoral colitis, with the mortality rate as high as 35%. Early diagnosis and prompt surgical intervention is the key to a favourable prognosis. To the best of our knowledge, there have been only 2 reported cases of recurrent stercoral ulcer perforation proximal to an end colostomy created for management of the initial stercoral ulcer perforation. High grade of suspicion for its recurrence in the postoperative period along with excision of the entire affected segment, clearance of the proximal loaded colon during and appropriate management of chronic constipation is the accepted modality of treatment for this condition.

Keywords: Postoperative; Stercoral Ulcer; Stercoral Colitis

Introduction

Stercoral colitis and its sequelae was first described by Berry [1]. in 1894. Stercoral ulcer perforation is the “perforation of the colon caused by ischemic pressure necrosis due to impacted hard stool or fecaloma”. Around 200 cases have been reported, however the actual incidence and prevalence of stercoral colitis and its complications are not known. The estimated post mortem incidence of stercoral ulcer ranges from 0.04% to 2.3% [2]. According to Maurer, et al. [3] stercoral perforation of the colon was found in 0.5% of all surgical colorectal procedures, 1.2% of all emergency colorectal procedures, and 3.2% of all colonic perforations. When stercoral colitis is associated with colonic ulcer perforation, a 35% mortality rate has been reported [4].

To the best of our knowledge till date, there have been 14 reported cases of stercoral perforation of the colon proximal to an end colostomy [5,6]. Among these, Serpell, et al. [7]. described two cases of recurrent stercoral perforation of the colon as an early postoperative complication in a patient undergoing end colostomy for stercoral perforation. We hereby present a case of recurrent stercoral perforation in 7th postoperative day in a 75 year old female who underwent Hartmann's procedure with end descending colostomy for stercoral ulcer perforation with diverticulitis.

Case Report

75 year old female who is known case of hypertension and rheumatic heart disease (status post BMV in 2019) with atrial fi-

brillation. She had recent history of cardio-embolic stroke - right MCA infarct 1.5 months ago and was re admitted with recurrent bilateral cerebellar infarct around 1 month back - currently without any neurological defects and on blood thinners. She was admitted under gastroenterology department with complaints of recurrent vomiting, chronic constipation, decreased appetite and generalised weakness. She underwent an OGDscopy which was suggestive of hiatus hernia with lax LES and gastric antral ulcer. The following day, the patient developed generalised pain in abdomen with mild distension, with non passage of flatus since 1 day. Surgical reference was given and CECT abdomen was done showing pneumoperitoneum with extra luminal fecal matter and free fluid in peritoneum suggestive of bowel perforation. The large bowel was distended with fecal matter and revealed multiple small air and fluid filled diverticuli especially in the sigmoid colon with no inflammatory changes. The patient was taken up for exploratory laparotomy where a large (~2.5cm) sigmoid perforation was noted on the antimesenteric border with fecaloma protruding through the perforation. Multiple pellets of hard stools noted in the abdominal cavity with loaded colon. Hartmann's procedure was done with an end descending colostomy leaving a 25cm sigmoid stump. Thorough wash was given to the abdominal cavity using warm saline and 2 drains inserted in the right subhepatic space and pelvis. Patient was shifted to ICU post surgery for further medical management.

On post op day 2, stoma appeared a bit dusky which further darkened the following day. The patient was tolerating liquids orally and stoma was not functional. Per stomal endoscopy was done on POD 3 which showed ulceration with blackish discoloration of 2cm of colonic mucosa at the stomal opening. Beyond that, the colonic mucosa showed a healthy pink colour with few stercoral ulcers (discoloured thinned out mucosa without perforation) and small quantity of hard impacted stools. In the following days, manual per stomal enema and evacuation of hard stool pellets were done to reduce the risk of fecal impaction. The patient improved clinically, nasogastric tube was removed and tolerated soft blended diet orally. On POD 5 and 6, the patient had increased serosanguinous discharge from midline wound leading to a suspicion of a burst abdomen. However the patient was vitally stable and both drains in situ were serosanguinous.

On POD 7, the patient developed low grade fever spikes, tachycardia and showed a raised leukocyte count. The midline wound discharge turned feculent with a strong odour. The patient was taken up for re-exploration the next morning (POD8). Intraoperatively, fecal matter noted concentrated in the left parastomal region. Another large stercoral perforation of around 3 cm in size was noted in the antimesenteric border of the descending colon around 20 cm proximal to the stomal opening. The entire diseased segment upto mid transverse colon was excised. The hepatic flexure and transverse colon was mobilised to create an end transverse colostomy at previous stoma site. Entire small bowel and remaining large bowel was traced, showing no stool loading or perforation. An abdominal drain was placed in the left paracolic region and the location of previous two abdominal drains confirmed. Thorough wash given and full thickness tension closure of midline wound was done. The patient recovered post operatively, tolerating oral diet. The stoma was functional by POD3 and the drains were clear. The patient was discharged on 11th day after the second surgery with left paracolic drain in situ.

On follow up after a week, the left paracolic drain was removed. The patient improved clinically with a healthy functional stoma and a healing midline wound with VAC system.

Discussion

Stercoral perforation has high mortality rates [8]. Early diagnosis and intervention is key in the management of bowel perforation. The greatest risk factor is chronic constipation seen in 81% of all patients [9]. Old age, chronic constipation, bed ridden patients with multiple comorbidities leading to abnormal bowel motility, and increased intraluminal colonic pressure are contributing factors to both stercoral colitis and diverticular diseases [3,10].

The clinical presentation of stercoral perforation and diverticulitis with or without perforation can be similar. In contrast to diverticular perforations, stercoral perforations present with the proximal colon loaded with multiple fecalomas (63%), the inflammatory and necrotic process involves a longer segment of colon beyond the area of perforation and perforations can be multiple (21-28%) [10].

In selected cases of perforated diverticulitis, medical management or CT-guided percutaneous drainage is appropriate. However all stercoral perforations mandate immediate surgical intervention [7]. Common locations for stercoral colitis are anterior rectum, anti-mesenteric border of the recto sigmoid junction, and the apex of the sigmoid colon which are described as the “watershed” area of the colon i.e., Sudeck’s point between the inferior mesenteric artery and superior rectal artery. Therefore, these areas are at risk of ischemia, particularly related to hypoperfusion [11].

CT abdomen has an important role in identifying this life-threatening complication of stercoral ulcers. Some of the findings are: colonic dilatation >6 cm, colonic wall thickening >3 mm, pericolic fat stranding, mucosal discontinuity, presence of free air, free fluid, and pericolic abscess [12]. Maurer, *et al.* [3], presented the diagnostic criteria of stercoral perforation, which includes the following: (1) round or ovoid perforation, > 1 cm in diameter; (2) fecalomas present within the colon, protruding through the perforation site or lying within the abdominal cavity; and (3) pressure necrosis or ulcer and chronic inflammatory reaction around the perforation site seen microscopically. Huttunen, *et al.* [13] reported that in a perforated stercoraceous ulcer, the perforation was a round or an ovoid hole with necrotic and inflammatory edges; however, in the idiopathic form, the perforation was a tear with a normal appearance of the colonic wall without being involved in the diverticulum.

The principles of treatment includes preoperative resuscitation, broad spectrum antibiotics stepped up based on peritoneal fluid cultures, elimination of all faecal soiling, resection of the entire involved bowel with exteriorization - to prevent recurrent ulcer perforations, as seen in this case report. Guyton, *et al.* [14] reviewed surgically managed cases which showed that resection with end colostomy and Hartmann’s procedure as the operation of choice with the lowest operative mortality (23%) when compared to those patients treated by either loop colostomy or exteriorization (71%) or proximal colostomy with plication of the perforation (44%). Serpell, *et al.* [7] who reported the first two cases of recurrent stercoral ulcer perforation in early postoperative period, emphasizes the necessity to resect the entire diseased segment rather than exteriorization of the perforation alone. Durrans, *et al.* [15] described two prerequisites that are necessary for limited resec-

tion 1. stool-filled proximal colon needs to be cleared 2. following reanastomosis, recurrent constipation needs to be addressed. According to Koruth, *et al.* [16] intra-operative orthograde colonic lavage is used to clear the proximal loaded colon which could help protect colonic anastomoses. Huang, *et al.* [17] were the first to perform an intraoperative colonoscopy around 10 minutes after clearing the colon of impacted stools to ensure the adequacy of the colonic resection and rule out the presence of additional stercoral ulcerations that could lead to delayed colonic perforation.

In the above described case, grossly dilated loaded proximal colon was not cleared. It is suspected to have caused a perforation in pre-existing proximal stercoral ulcer even when an end colostomy was established.

Conclusion

Although stercoral ulcer perforations are rare, it should be a differential diagnosis in elderly, bedridden patients with chronic constipation who present with sudden diffuse abdominal pain. Appropriate management of constipation with high fibre diet, laxatives and enemas is important to prevent stercoral perforation during follow-up. Early diagnosis and prompt surgical intervention is necessary to decrease mortality rates. Segmental resection of the entire affected segment with end colostomy is preferred over primary repair/exteriorisation. Resection along with emptying of the proximal loaded bowel and prevention of constipation are the most important steps in management and prevention of recurrent perforations.

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