A CASE REPORT

Endoscopic Diagnosis and Treatment of Stercoral Colitis

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Stercoral colitis is an uncommon inflammatory condition resulting from hard, impacted stool causing pressure necrosis and ulceration of the colonic mucosa. This condition is usually only recognized post hoc in its most severe clinical manifestation when it has already led to perforation and peritonitis requiring surgical exploration. Here we present three cases diagnosed endoscopically, two of which were successfully treated thus avoiding surgery.

Case 1

A 67 year-old man presented to the Emergency Department (ED) with two weeks of intermittent constipation, severe abdominal pain, and vomiting brown, feculent-appearing material. He had a history of diabetes and congestive heart failure. He had passed flatus that morning but had not had a bowel movement for two days. The patient appeared acutely ill and was mildly hypotensive. His abdomen was distended with hypoactive bowel sounds, tender to palpation but no signs of peritonitis. His WBC was 19,600. A computed axial tomography (CT scan) was performed and showed “abnormal thickening of the wall of the sigmoid colon with pericolonic soft tissue stranding”; in addition there was proximal colonic dilation up to 7.8 cm diameter, without significant small bowel dilatation. He underwent a flexible sigmoidoscopy that showed hard stool impacted in the sigmoid colon with mucosal ulceration at the impaction site. The stool was disrupted with grasping forceps and then pushed proximally. Liquid stool began to pass, and a rectal tube was placed. There was good output from rectal tube for 12 hours but the abdominal exam worsened. On the second hospital day the abdomen was diffusely tender with guarding but no rebound tenderness. The WBC increased to 28,200; abdominal X-ray was negative for pneumoperitoneum. The patient was taken for exploratory laparotomy which revealed full thickness necrosis of the left colon with areas of micraperforation but no gross contamination. This was resected and a Hartman’s procedure was performed. The pathology results confirmed multifocal transmural necrosis (Figure 1). The patient developed multi-organ dysfunction and subsequently died.

Figure 1. Gross pathological specimen showing transmural necrosis with mucosal erosions.

Case 2

A 63 year-old woman presented with a one-day history of severe, intermittent, crampy left-lower-quadrant abdominal pain. She had not had a bowel movement for seven days although she was passing flatus. Her abdomen

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Figure 2. Endoscopic view showing discrete stercoral ulceration.

was distended; there was direct and rebound tenderness but no guarding. The rectal vault was empty. Her WBC was 15, 800 with 89% neutrophils. CT scan showed multiple stool balls proximal to an abrupt narrowing in the mid-sigmoid colon associated with mild colonic wall thickening and some infiltration of the pericolonic fat. The differential diagnosis by the radiologist was colitis or malignant stricture. A gastrograffin enema demonstrated complete obstruction of the sigmoid and a large amount of proximal stool. A colonoscopy was requested for placement of a self-expanding metallic stent to provide colonic decompression.

The patient underwent unprepped colonoscopy using carbon dioxide for insufflation. At the sigmoid-descending junction there was inflamed mucosa and mild narrowing of the lumen; a large hard fecalith was impacted proximal to this with multiple superficial mucosal ulcers (Figure 2). There was no stricture. The fecalith was mechanically broken into small pieces with a rat-toothed forceps; the endoscope was passed proximal to the obstructing stool and lavage was accomplished with 800 cc of polyethylene glycol along with 300 cc of mineral oil flushed through the colonoscope using a Velocity endoscopic pump (US Endoscopy, Mentor OH). The patient had multiple bowel movements overnight. The next day her abdomen was not distended, and much less tender and her WBC declined to 12,700. She was discharged two days later without complaints.

Case 3

A 72 year-old woman presented with sudden crampy abdominal pain associated with nausea but no vomiting. She had passed flatus and a bowel movement one day prior to admission. She had no prior history of obstructive symptoms. The patient had undergone a barium esophagram to evaluate dysphagia three weeks prior to admission. Her abdomen was softly distended with tympanic high-pitched bowel sounds. No rebound or guarding was elicited. There was no stool in the rectal vault. The WBC was 14,100, other labs were unremarkable. Abdominal X-ray demonstrated a large bowel obstruction with a 3 cm ovoid, densely radiopaque object in the pelvis, consistent with retained barium (Figure 3). A CT scan showed colonic obstruction by a 4 x 3.8 cm barium-impregnated fecaloma lodged in the sigmoid colon along with colonic wall thickening consistent with colitis. A colonoscopy was requested to decompress the colonic obstruction.

The colonoscopy was performed with CO2 for insufflation. A large yellowish fecalith was impacted completely obstructing the lumen at the angulation of sigmoid-descending colon junction. The mucosa was erythematous and inflated but there was no stricture or ulceration (Figure 4). The barium fecalith was mechanically broken into pieces by a tedious process with a rat-toothed forceps and a screw-tipped Soehendra stent extractor (Cook Medical, Bloomington, IN). Despite mechanical degradation, the fecalith was too large be delivered by balloon or basket extraction. The endoscope was passed proximal to the obstructing fecalith, the proximal colon was decompressed, and 300 ml of mineral oil was instilled proximally. That night the patient had multiple spontaneous bowel movements. The next morning her abdominal pain had resolved, the WBC decreased to 10,000 and no barium-fecalith was seen on abdominal X-ray.
DISCUSSION

Stercoral colitis most likely occurs more commonly than it is diagnosed. The adjective “stercoral” is derived from the Latin stercoraceus that means “consisting of feces”. Stercoral ulceration was first described by a pathologist at autopsy as the cause of a colonic perforation in 1894. The vast majority of cases continue to be reported in the surgical literature as patients who present with colonic perforation and a high mortality rate, as our first patient did. Patients with perforation, however, probably only represent the “tip of the iceberg” of the incidence stercoral colitis. This series of three patients illustrates the spectrum of the illness from crampy abdominal pain to colonic obstruction, radiologic evidence of colitis, endoscopic evidence of ulceration, and progressing to necrosis and perforation. It also demonstrates how the correct diagnosis and aggressive endoscopic management can successfully treat these patients so they do not progress to perforation requiring surgical treatment.

Serpell and Nicholls reported that 77% of stercoral ulceration occurs in the sigmoid or rectosigmoid colon, as it did in our three patients. They hypothesized that it occurs in the distal colon because the stool is dehydrated and hard, there is relative narrowing and angulation along with the higher intraluminal pressures. The proposed disease sequence is as follows: a large fecalith impacts and obstructs in a narrow, angulated portion of the sigmoid. The hard stool presses continually on the mucosa leading to colitis and pressure necrosis. The obstruction increases the intraluminal pressure and that reduces mucosal blood flow, further accelerating the mucosal necrosis. This sets up a cycle that, unless the obstruction is removed, leads to colonic perforation.

Heffernan et al. highlighted that stercoral colitis could be identified on CT scan; they reported a series of four elderly, debilitated patients who presented with stercoral perforation induced peritonitis all of whom died. These represent the most severe patients. The CT findings included focal colonic thickening, stranding of the pericolonic fat, and extra-luminal bubbles of gas in patients who have progressed to perforation. It is key that focal colonic wall thickening and pericolonic stranding were associated with a fecaloma or impaction—since this changes the diagnosis from “non-specific colitis” to “stercoral colitis.” Heffernan et al. suggest using CT scans for early diagnosis and then aggressive bowel cleaning and disimpaction.

Endoscopic treatment offers several advantages over traditional “bowel cleansing and disimpaction.” Oral laxatives are contraindicated in patients with colonic obstruction and may serve to accelerate the stercoral ulceration. Manual disimpaction is limited to the rectum and cannot reach impactions more proximally as was the case in all three of our patients. Endoscopy permits direct access, visualizes the cause of the obstruction- an impacted fecalith, stricture or cancer and further provides visualization of the degree of mucosal ulceration and the likelihood of perforation. Finally, endoscopy provides direct therapy to remove the primary cause, the fecalith, and cure the condition, preventing the mucosal damage from progressing to perforation.

The high mortality from the surgical literature would suggest that conservative management may not be appropriate for these patients. Our series of patients underscores the need to consider the diagnosis of stercoral colitis, in the correct clinical setting, and when identified consider aggressive endoscopic treatment before it progresses to perforation.

References


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