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Case Report

Case Reports in Clinical Radiology



Cecal gangrene – Unusual complication of manual bowel decompression

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Received: 20 December 2022 Accepted: 31 December 2022 EPub Ahead of Print: 13 March 2023 Published: 26 July 2023

DOI

10.25259/CRCR_52_2022

Quick Response Code:



ABSTRACT

We present a case of a 31-year-old female who presented with complaints of obstipation. Imaging features were suggestive of small bowel obstruction with transition point at distal ileum. On emergency laparotomy, the patient was found to have fecal impaction, for which manual bowel decompression (milking) was done. The patient developed hypotension intraoperatively requiring ICU admission for post-operative management. Post-operative CT demonstrated features of hypotension with gangrenous changes in the cecal wall. Re-exploration surgery demonstrated features of cecal gangrene consistent with imaging findings.

Keywords: Milking, Hypotension, Cecal gangrene

INTRODUCTION

Acute intestinal obstruction is a surgical emergency frequently encountered in clinical practice.^[1] It can lead to damage to mucosal epithelial cells, increased mucosal permeability and inflammatory changes.^[2,3] Manual bowel decompression, commonly called "milking" is a commonly used method of intestinal decompression.^[4] Possible complications of milking include paralytic ileus, post-operative peritoneal adhesions, sepsis and peritoneal contamination.^[4-6]

CASE REPORT

Clinical details and imaging findings

A 31-year-old female with no previous comorbidities presented to casualty with complaints of colicky abdominal pain and obstipation for 8 days. On examination, the patient was vitally stable; however, she had abdominal distension and tenderness. Erect and supine abdominal radiographs [Figure 1a] were obtained which showed dilated small bowel loops with multiple air-fluid levels on erect radiograph. Contrast-enhanced abdominal CT [Figure 1b-d] showed features of small bowel obstruction with transition point in the distal ileum with no definite mass or thickening seen at the transition point and normal enhancement of the involved bowel loops.

The patient was taken for emergency laparotomy and was found to have fecal impaction, for which manual bowel decompression (milking) was done. The patient developed hypotension intraoperatively and was immediately stabilized; however, she required ICU admission for postoperative management. She had progressive abdominal distension with increased requirement

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Figure 1: (a) Erect radiograph of abdomen showing multiple air fluid levels, (b-d) contrast-enhanced abdominal CT showing features of small bowel obstruction with transition point in the distal ileum (d, white arrow). Cecum is collapsed $(c,^*)$ with normal ileocecal junction (c, black arrowhead).



Figure 2: Contrast-enhanced abdominal CT reduced enhancement of liver (b) and hyperenhancing adrenals (b, black arrows) in arterial phase, collapsed IVC (c, white arrow) and mucosal hyperenhancement of the bowel with diffuse edematous thickening. Cecum and transverse colon are distended with impacted fecal matter (*). The wall of the cecum appears thinned out with imperceptible walls near the anterior inferior end of cecum (a, e, dashed arrows). Hyperdense fluid is seen in the peritoneal cavity.

of vasopressors in the post-operative period and contrastenhanced CT was done to evaluate the same.

Post-operative CT scan [Figure 2] showed reduced enhancement of liver, hyperenhancing adrenals, collapsed IVC, and mucosal hyperenhancement of the bowel with diffuse edematous thickening. Cecum and transverse colon were distended with impacted fecal matter. The wall of the cecum appeared thinned out with imperceptible walls near the anterior inferior end of cecum. Hyperdense fluid was seen in the peritoneal cavity. No evidence of vascular occlusion was seen. These findings were suggestive of CT hypotension complex with the early gangrenous changes in the cecum and features of peritonitis.

Re-exploration surgery was done which confirmed gangrenous changes in the dilated cecum with no vascular occlusions. Partial right hemicolectomy was done for



Figure 3: Gross specimen of partial right hemicolectomy showing gangrenous changes in the cecum.

the same and tissue specimen [Figure 3] was sent for microbiological and histopathological evaluation. Aerobic culture from the tissue sample demonstrated growth of *Enterococcus faecium*.

The patient was shifted back to intensive care unit; however, patient's status did not improve post-surgery and she succumbed to the consequences of multi-organ dysfunction on 2^{nd} day after re-exploration.

DISCUSSION

Acute intestinal obstruction is a surgical emergency frequently encountered in clinical practice.^[1] Impaction of the intraluminal content proximal to the obstruction site causes distension of the bowel loops and bacterial overgrowth contributed to stasis of the contents. Persistent distension may cause reduced blood circulation to involved bowel loops, leading to damage to mucosal epithelial cells, increased mucosal permeability, and inflammatory changes. In addition, increase in gut bacterial load contributed bacterial overgrowth may promote translocation of bacteria.^[2,3]

The treatment is aimed at either relieving or bypassing the cause of mechanical obstruction and decompressing the intra-abdominal pressure. In addition to enterotomy followed by aspiration and aspiration with intraluminal tube insertion, manual bowel decompression, commonly called "milking," is a commonly used method of intestinal decompression.^[4]

The technique involves caressing the intestinal contents cranially into the stomach or caudally into the cecum and colon, followed by nasogastric decompression or enema as required.^[4] Possible complications of milking including inhibition of gastrointestinal motility leading to paralytic ileus, post-operative peritoneal adhesions, risk of bacterial

translocation leading to sepsis, and peritoneal contamination have been hypothesized;^[4-6] however, not many extensive studies have been performed to validate the same.

CT hypotension complex is a widely described constellation of CT findings associated with hypovolemic shock related to trauma as well as non-traumatic conditions including sepsis, neurogenic shock, cardiogenic shock, and diabetic ketoacidosis. Imaging findings comprise diffusely thickened and edematous bowel wall with intense mucosal enhancement, collapsed IVC and aorta, abnormal pancreatic enhancement, peripancreatic fluid, hypoenhancement of the liver and spleen, and hypoenhancing renal parenchyma. These findings can be reversed with adequate fluid resuscitation and may not always lead to ischemic bowel injury. However, if treated inadequately, it has been associated with a mortality rate as high as 70%.^[7]

Cecal infarction is a rarely encountered condition which may develop as a consequence to various underlying causes including vascular occlusion and non-occlusive conditions like in particular shock and cardiac failure. Most commonly, non-occlusive hypoperfusion of the intestine involves the "watershed areas," including the splenic flexure (Griffith's point) and the rectosigmoid junction (Sudeck's point); however, cecum can also be frequently involved.^[8]

Non-occlusive hypoperfusion typically does not cause isolated cecal involvement but if present, it can be attributable to other underlying causes that may, in turn, make the cecum more vulnerable to ischemia. The cecum, having the largest caliber among all the bowel loops, is at higher tension (according to Laplace's law) which may make it susceptible to reduction in microcirculation. In addition, high transit time of colonic contents is commonly seen in the cecum, thus, increasing the possibility of bacterial translocation and superadded infection on top of the prior ischemic damage.^[8]

Since no occlusions of the cecal vessels could be identified in our patient, it may be speculated that the present case is an example of non-occlusive ischemia. However, since there was isolated gangrene of the cecum while sparing the other two watershed areas, it would be wise to assume that other factors came into play. In an ongoing ischemic damage contributed by hypotension, cecum, being overloaded, and overdistended with fecal matter contributed by the effect of manual bowel decompression could have made it susceptible to reduction of its microcirculation and resultant ischemia. These factors could have been complicated by bacterial overgrowth in the milked fecal matter in the cecum contributed by previous mechanical obstruction increasing the possibility of bacterial translocation and superadded infection on top of the prior ischemic damage.

CONCLUSION

Cecal gangrene should be kept in the differential diagnosis as a complication post manual decompression if cecum is over distended on follow-up imaging.

TEACHING POINTS

- CT hypotension complex can be associated with nontraumatic conditions such as septic shock, bacterial endocarditis, and diabetic ketoacidosis.
- Isolated cecal infarction is an uncommon entity which may develop secondary to several causes including occlusive and non-occlusive underperfusion.

MCQs

- 1. Intestinal "watershed areas" include all of the following except
 - a) Splenic flexure
 - b) Mid-transverse colon
 - c) Rectosigmoid junction
 - d) None of the above

Answer Key: b

- 2. Possible complications of manual bowel decompression include
 - a) Paralytic ileus
 - b) Post-operative peritoneal adhesions
 - c) Risk of bacterial translocation
 - d) All of the above

Answer Key: d

- 3. Isolated cecal infarction can be attributed to all except
 - a) Large bowel obstruction
 - b) Hypotension
 - c) Bacterial translocation
 - d) Overhydration

Answer Key: d

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Sonker S, Sureka B, Soni SC, Kothari N. Cecal gangrene – Unusual complication of manual bowel decompression. Case Rep Clin Radiol 2023:1:123-6.