Acute Colonic Pseudo-obstruction and Volvulus: Pathophysiology, Evaluation, and Treatment

Joshua Underhill, MD, MPH[1] Emily Munding, MD[1] Dana Hayden, MD, MPH, FACS, FASCRS[1]

[1] Department of General Surgery, Rush University Medical Center, Chicago, Illinois

Clin Colon Rectal Surg 2021;34:242–250.

Address for correspondence Dana Hayden, MD, MPH, FACS, FASCRS, Department of General Surgery, Rush University Medical Center, 1725 W. Harrison, Suite 1138, Chicago, IL 60612 (e-mail: Dana_m_hayden@rush.edu).

Abstract

Acute colonic pseudo-obstruction (ACPO) and volvulus are two disease processes that affect the colon causing abdominal distension and may necessitate operation intervention. ACPO may be associated with multiple comorbidities, infectious diseases, and cardiac dysfunction. It may be treated with conservative management including endoscopic decompression or neostigmine. If the distension is not addressed, high mortality may result if peritonitis develops. Volvulus most commonly occurs in the sigmoid colon or cecum. If left-sided, endoscopic decompression may resolve the obstruction if detorsion is successful, although sigmoid colectomy should be performed during the admission. If cecal volvulus is identified, right hemicolectomy should be performed.

Keywords
- Ogilvie syndrome
- pseudo-obstruction
- volvulus
- large bowel obstruction

Acute colonic pseudo-obstruction (ACPO) is a rare but serious disease process involving severe colonic distension without mechanical obstruction. It most commonly occurs in critically ill patients with multiple other comorbid conditions. The disease mechanism is poorly understood but thought to be due to relative overactivity of the sympathetic nervous system on the colon. When suspected, ACPO is best evaluated with abdominal plain films followed by either water-soluble contrast enema or computed tomography (CT) of the abdomen and pelvis to confirm the diagnosis. A majority of patients can be successfully treated with intravenous neostigmine, with initial success rates ranging from 84 to 94%. All patients should also receive treatment for any other underlying medical conditions that may be contributory. Patients that fail medical therapy may benefit from colonoscopic decompression or surgical decompression with a cecostomy. Any patient with peritonitis or other signs of bowel ischemia requires a laparotomy with either right hemicolectomy or subtotal colectomy. Laparotomy is also recommended for patients with a colonic diameter of 12 cm or greater due to a high risk of perforation. In patients who require laparotomy, mortality rates may range from 40 to 60%, most likely related to the severity of the disease process and underlying medical conditions seen in these patients.

Introduction

ACPO was first detailed in the medical literature by Sir William Heneage Ogilvie in 1948 in the *British Medical Journal*, resulting in the disease now being referred to as Ogilvie syndrome. In this chapter, he discussed two cases that clinically appeared to be large bowel obstructions (LBO); however, barium enemas showed no point of obstruction. In both cases, exploratory laparotomy demonstrated subdiaphragmatic malignancies near the crura without visible colonic pathology, aside from distension. These findings led Sir Ogilvie to hypothesize that the tumors were interrupting sympathetic activity to the colon. The resulting overactive parasympathetic stimulation would lead to an atonic colon.[1] While Ogilvie originally only described pseudo-obstruction associated with subdiaphragmatic tumors, in more recent years, the term has been used to describe all cases of acute colonic dilatation without mechanical obstruction, regardless of cause.[2]

Epidemiology

ACPO is a rare disease process, with a calculated incidence of ~100 cases per 100,000 inpatient admissions in the United States.[3] It occurs most commonly in the sixth decade of life,

Issue Theme Bowel Obstructions; Guest Editor: Michael Benjamin Hopkins, MD, FACS, FASCRS

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DOI https://doi.org/10.1055/s-0041-1727195.
ISSN 1531-0043.

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with a slight predilection toward males with 60% of cases reported in men.[4] Most cases are associated with recent illness or surgery; only 5% of cases considered to be idiopathic.[4] The most common underlying conditions associated with ACPO include nonoperative trauma, infection, and cardiac dysfunction.[4] A more comprehensive list of medical conditions associated with ACPO is detailed in ►**Table 1**.[1,4–7] ACPO carries a high mortality rate: ~15% in all cases, and increasing to 36 to 44% when there is associated intestinal ischemia or perforation.[4,8] More recent research indicates mortality has decreased to as low as 6.4% in 2011, but this may be due to more frequent diagnosis of ACPO.[3]

Pathophysiology

The exact etiology of ACPO remains poorly understood, but it appears to be related to autonomic imbalance. Sir Ogilvie originally proposed that the disease is caused by relative overactivity of parasympathetic innervation after sympathetic interruption.[1] However, more recent literature indicates the opposite: that parasympathetic interruption leads to relative sympathetic overactivity, causing pseudo-obstruction.[7,9] While not proven in animal models, it is plausible for several reasons. ACPO largely occurs in critically ill patients who have greater sympathetic drive.[10] Parasympathetic fibers from S2 to S4 contribute to emptying the left colon and rectum, making it reasonable to assume that loss of this function could lead to ACPO.[11] Furthermore, acute colonic dilation could stimulate mechanoreceptors further increasing sympathetic activity and halting intestinal motility, the "colo-colonic reflex."[12] Lastly, ACPO is often successfully treated with neostigmine, which increases parasympathetic activity in the colon. This suggests the lack of parasympathetic activity to be a cause.[13] The greatest concern in ACPO is that worsening dilation of the colon will result in ischemia and perforation, which can rapidly progress to peritonitis and death.[4]

Evaluation

Clinical Signs and Symptoms

As suggested by the term "pseudo-obstruction," patients with ACPO present with signs and symptoms very similar to a LBO. Patients can experience cramping lower abdominal pain, nausea, vomiting, and fever.[4] While many patients experience constipation, others will continue to pass flatus and may have diarrhea.[4,10] One of the most striking symptoms is progressive abdominal distension with a tympanic abdomen on physical exam.[10,14] Although abdominal tenderness was classically considered to be a sign of intestinal ischemia and impending perforation, it is common in ACPO patients with viable bowel as well. However, patients with ischemia and perforation are more likely to be febrile and peritonitic.[4] As the presentation of ACPO can be quite variable, it is important to always consider it in the differential diagnosis for critically ill patients with abdominal pain and distension, along with mechanical obstruction and *Clostridium difficile* infection.[15]

Table 1 Underlying medical conditions in patients diagnosed with ACPO[1,4–7]

Trauma
Hip fracture and surgery
Pelvic fracture and surgery
Burns
Infection
Pneumonia
Herpes zoster
Pelvic abscess
Sepsis
Obstetrics/gynecology
Cesarean section
Normal pregnancy
Cardiovascular
Myocardial infarction
Congestive heart failure
Stroke
Cardiothoracic surgery
Neurologic
Parkinson's disease
Alzheimer's disease
Multiple sclerosis
Neurosurgery
Malignancy
Leukemia
Retroperitoneal tumors
Subdiaphragmatic tumors
Disseminated pelvic malignancy
Gastrointestinal
Cholecystitis
Pancreatitis
Liver failure
Abdominal and pelvic surgery
Metabolic
Electrolyte imbalance
Pulmonary
Acute respiratory failure
Mechanical ventilation
Chronic obstructive pulmonary disease
Renal
Acute renal failure
Nephrolithiasis
Urologic surgery
Transplant surgery
Heart
Lung
Liver
Kidney
Pharmacologic
Antidepressants
Phenothiazines
Opiates
Antiparkinsonian agents
Miscellaneous
Alcohol abuse
Amyloidosis
Idiopathic

Abbreviation: ACPO, acute colonic pseudo-obstruction.
ACPO is noted to most commonly occur in critically ill patients, with many co-occurring conditions linked to the development of ACPO, listed in this table.

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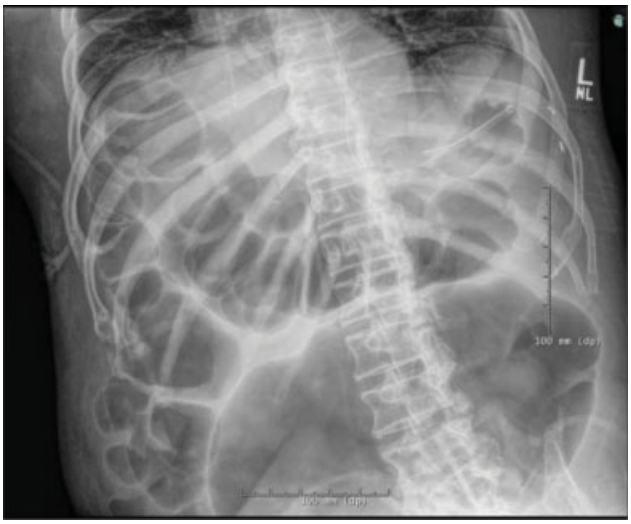

Fig. 1 Abdominal radiograph of a patient with acute colonic pseudo-obstruction.

Laboratory and Radiologic Evaluation

When ACPO is considered, it is important to obtain plain abdominal radiographs to evaluate for colonic distension, which would suggest either mechanical obstruction or pseudo-obstruction (►**Fig. 1**). Upright chest X-ray and plain films of the abdomen performed in left lateral decubitus can also demonstrate pneumoperitoneum in cases of perforation.[16,17] This imaging should be followed by either a water-soluble contrast enema or CT of the abdomen and pelvis. Contrast enema has excellent sensitivity (96%) and specificity (98%) when differentiating mechanical obstruction from pseudo-obstruction.[17] CT with intravenous contrast has slightly lower sensitivity (91%) and specificity (91%) but has the benefit of more accurately measuring the degree of colonic distension and assessing the condition of the bowel mucosa.[18,19] Both are acceptable imaging modalities for the diagnosis of ACPO.

During the initial evaluation, laboratory testing for electrolyte abnormalities, blood cell count, and *C. difficile* infection should be obtained. ACPO may be associated with electrolyte abnormalities, particularly hypokalemia.[2] While it is unclear if electrolyte imbalances are a cause or effect of ACPO, correction is warranted as patients with electrolyte abnormalities are less likely to respond favorably to neostigmine therapy.[20] White blood cell count is also important to determine; one study demonstrated that 100% of patients with ischemia or perforation had leukocytosis, compared with only 26% of patients found to have viable bowel.[4] Lastly, *C. difficile* infection can present very similarly to colonic pseudo-obstruction.[15] Therefore, patients should be evaluated for *C. difficile* toxin and treated accordingly if the results are positive.

Treatment

Once a diagnosis of ACPO is confirmed, conservative medical management is the first-line of treatment. However, if a patient develops signs of impending perforation or peritonitis, then laparotomy should be performed to investigate viability of bowel and repair or resect as needed. Since the cecum is the most likely site of perforation, cecal diameter on radiograph is also an important indicator of severity of disease and need for operative intervention.[10] There is no clear consensus regarding the cutoff in cecal diameter that requires operative intervention.[13] In early studies, cecal perforation was likely to occur at diameters greater than 9 cm.[21] However, more recent studies show that perforation was less likely to occur at diameters less than 12 cm. Actually, perforation occurred in 23% of cases with cecal diameters of 14 cm or more.[4] Considering the poor consensus, clinicians should maintain high suspicion for perforation at diameters between 9 and 12 cm. It is also prudent to obtain serial abdominal films every 12 to 24 hours to evaluate for worsening cecal distension and pneumoperitoneum.[6,10] Above 12 cm, laparotomy is a reasonable option.

Medical Management

First-line conservative medical management includes supportive care similar to that for bowel obstruction and postoperative ileus. Patients should be maintained *nil per os*. Nasogastric decompression should be performed. Opiates should be minimized as they slow intestinal transit and can worsen the condition. Osmotic laxatives can increase colonic gas production and therefore should be avoided as well. Careful placement of a rectal tube has limited success in colonic decompression.[6,9,10,13] Patients should be closely monitored with serial abdominal exams and abdominal films to evaluate progression of disease. Also, all underlying conditions associated with ACPO, including electrolyte abnormalities and poor mobility, should be addressed, if feasible, as this may lead to resolution of colonic distension.[10]

Pharmacologic decompression of the colon can be attempted with intravenous neostigmine. Neostigmine is an acetylcholinesterase inhibitor that acts a parasympathomimetic agent in the gastrointestinal system. In multiple studies, a 2.0 mg dose of intravenous neostigmine has shown great effectiveness in quick colonic decompression, with initial success rates ranging from 84 to 94%.[20,22,23] The most common adverse effect associated with neostigmine is abdominal pain, but patients can also frequently experience excessive salivation, nausea and vomiting, and symptomatic bradycardia. In cases of bradycardia, 0.5 to 1.0 mg atropine generally reverses the cardiac effects of neostigmine.[20,22,23] Some patients experience recurrence of ACPO after decompression with neostigmine. If this occurs, a second dose of neostigmine can be attempted or endoscopic decompression may be warranted.[20,22,24]

Endoscopic Management

Colonoscopic decompression of ACPO demonstrated success in multiple studies before neostigmine was widely used, although it has been largely replaced by neostigmine.[25–27] Endoscopy carries a higher risk profile than neostigmine, but it may be warranted in patients who fail neostigmine therapy or in patients with contraindications to neostigmine. It also

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has the added benefit of definitively diagnosing ACPO by ruling out any obstructing lesion. It is a difficult procedure in the context of ACPO, as the colon is unable to be prepped and is full of viscous stool. Given that patients with ACPO are already at high risk for cecal perforation, insufflation from colonoscopy adds to that risk.[13] In fact, colonoscopy in the setting of ACPO is noted to have a 2% perforation rate and 1% mortality rate.[28,29] If any colonic ischemia is noted during colonoscopy, the procedure should be aborted to perform laparotomy.[13]

Surgical Management

Surgical intervention is indicated for ACPO refractory to nonoperative interventions or if peritonitis is present. Historically, supportive therapy was attempted for 48 to 72 hours before surgical treatment.[13] However, more recent literature suggests conservative and pharmacologic therapy can be continued up to 5 days as long as the patient's exam and imaging do not worsen or suggest ischemia.[6] ACPO lasting for 6 days or longer is associated with a higher risk of perforation.[8] Surgery is indicated at any time the patient clinically deteriorates.

In patients who fail nonoperative management but who do not show signs of peritoneal inflammation, tube cecostomy is a good option. It provides immediate colonic decompression. It is most commonly performed through a limited laparotomy in the right lower quadrant allowing direct access to the cecum.[30] While it is a definitive therapy for ACPO, cecostomy has risk of local complications including pericatheter leak, surgical site infection, and tube displacement.[31,32] Other methods of decompressive cecostomy have also been described such as laparoscopic cecostomy, CT-guided percutaneous cecostomy, and percutaneous endoscopic cecostomy (PEC). Laparoscopic cecostomy is beneficial as it allows for surveillance of the entire colon for signs of ischemia or perforation requiring further intervention.[33] PEC is an option for poor surgical candidates since it requires minimal sedation and can be performed during attempted colonoscopic decompression with direct visualization of the tube being placed into the cecum.[34]

In any patient with signs of peritoneal inflammation suggesting ischemia or perforation, exploratory laparotomy is indicated.[4,6,8,13,30] Depending on intraoperative findings, there are multiple options during laparotomy. If all bowel appears viable, then a tube cecostomy or formal cecostomy are good options for immediate decompression. In cases of bowel ischemia, frank perforation, or over-distension sheer injury (evidenced by spontaneous serosal tears), formal resection with a right hemicolectomy, or subtotal colectomy is required (►**Fig. 2**). If laparotomy is performed, mortality can be as high as 40 to 60%.[4,35] This is likely a reflection of the severity of disease as well as the underlying critical comorbid conditions in these patients. Laparotomy therefore is a last chance option for refractory ACPO in critically ill patients.

Colonic Volvulus

Colonic volvulus is a torsion of the large bowel around its mesentery that creates a closed loop obstruction.

It is the third most common cause of LBO and requires rapid identification and treatment due to its associated high morbidity and mortality. Although it can occur at any or multiple points in the colon, it most commonly occurs in the sigmoid and cecum. The diagnosis is made by clinical history, physical examination, and classic imaging on plain abdominal films and CT.

Current management entails endoscopic and/or surgical intervention depending on the type of volvulus. If sigmoid volvulus is diagnosed, treatment includes decompression and detorsion along with resection with the goal of minimizing morbidity and also risk of recurrence.

Epidemiology and Pathophysiology

Incidence of colonic volvulus is not well established. In a recent large US population-based study, it was found to be responsible for ~2% of bowel obstructions (including both small and large bowel).[36] Sigmoid volvulus is more common than cecal volvulus, accounting for 65 to 80% of all colonic volvulus.[37,38] However, the same population-based study also reported that the incidence of cecal volvulus is increasing by ~5% per year, while the incidence of sigmoid volvulus remains the same.[36] Other types of colonic volvulus such as transverse, synchronous sigmoid and cecal volvulus and splenic flexure volvulus have been described, but remain rare.

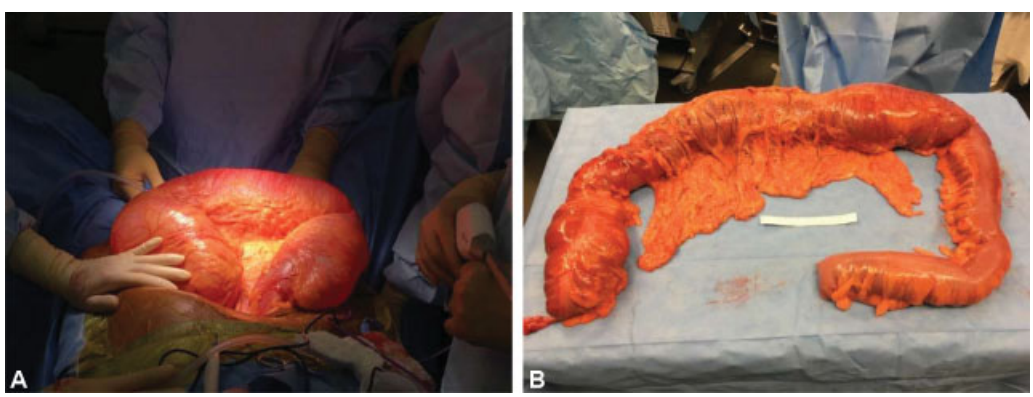

Fig. 2 (A and B) Intraoperative images from a colectomy for a patient with acute colonic pseudo-obstruction.

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A volvulus is defined as a twist (between 180–-360 degrees) around a mesenteric point that results in obstruction. It is theorized that for a volvulus to occur, it requires (1) a long and redundant colon with poor peritoneal fixation and (2) a thinning of the mesentery with a lead-point within the abdomen to serve as the site of rotation. A lead-point can be an adhesion or mass, either intra- or extraluminal. Based on this pathophysiology, various factors contribute to the risk of volvulus. Sigmoid volvulus is classically seen in elderly, institutionalized patients with multiple comorbidities. Volvulus usually occurs in these patients due to colonic dysmotility, which can be secondary to chronic constipation or laxative use.[39,40] Dysmotility leads to an elongation and dilation of the colon, thus creating redundancy and making the bowel prone to torsion.[41] This may also be observed in Hirschsprung's disease, where sigmoid volvulus can be a presenting symptom in children.[42]

There are three types of cecal volvulus that have been described, all of which require a mobile cecum and ascending colon. It is commonly theorized that a congenital or acquired (i.e., from surgery, pregnancy) absence of the embryogenic fixation of the cecum to posterior parietal peritoneum is required for cecal volvulus to occur.[43,44] This anatomic anomaly combined with multiple other factors such as age and colonic dysmotility may lead to the torsion event.[45,46] Whereas the average age of sigmoid volvulus is 70 years old, cecal volvulus is more frequent in younger women, with the mean age in the mid-50s.[36,44] A recent population-based study demonstrated that there are two peaks for age on onset, the previously described mid-50s as well as the early-70s.[47] Another well-known but rare cause of LBO is cecal bascule, where the cecum folds up directly on itself anteriorly without any twisting.[48]

Overall, the incidence of colonic volvulus in the United States is lower than the worldwide incidence. Colonic volvulus, specifically sigmoid volvulus, has been described as 45 to 80% of the cause of LBOs in Africa, the Middle East, South America, and Southern Asia.[49–55] This finding is commonly attributed to higher fiber intake in these populations, which is hypothesized to stretch out the colon, increasing the risk of torsion.[56] However, recent studies indicate that the previously observed increase in incidence may no longer hold true. A study conducted by Hussein et al[57] (2008) reported that in the Middle East, which is commonly referred to as the "volvulus belt," colonic volvulus comprises only 10% of all LBOs. The etiology of this trend is likely multifactorial, due to population migration and westernization of diet.[57,58]

Evaluation

Presentation

A patient suffering from volvulus may present with symptoms of abdominal distention, intermittent episodes of colicky diffuse pain, nausea, vomiting, and obstipation. Sigmoid volvulus presents with symptoms of a LBO and typically has an insidious onset with vomiting occurring before the onset of pain. Only 17% of patients present acutely within 48 hours of symptom onset.[47] Cecal volvulus presents with symptoms of a small bowel obstruction and can vary in presentation, with duration of symptoms ranging from hours to days.[59]

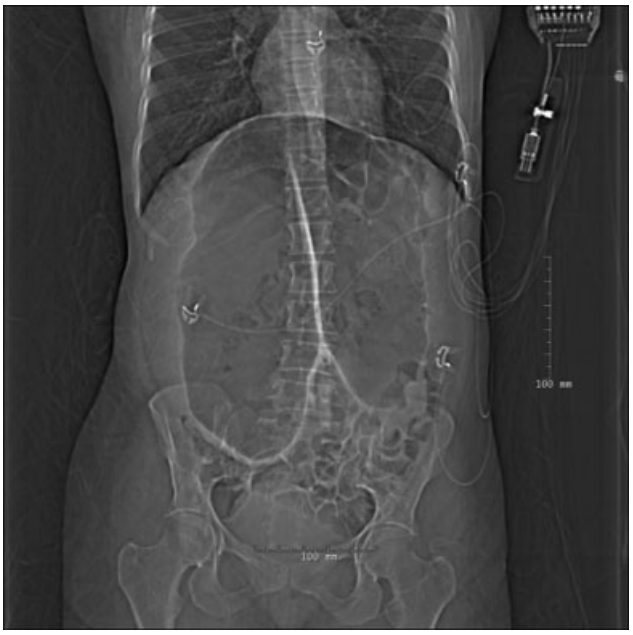

Fig. 3 Coffee bean sign.

In rare late presentations, patients may have signs of sepsis or end-organ damage including hypotension, altered mental status, acute kidney injury, or decreased urine output. Chronic volvulus has also been described, especially in patients with chronic constipation. Atypical cases of sigmoid volvulus have been described in adolescents. These patients present with multiple self-resolving episodes of abdominal pain due to torsion and spontaneous detorsion of the colon.[60]

Imaging

Modality of evaluation depends on the clinical status of the patient and location of the volvulus. Common initial workup includes an abdominal X-ray to identify pneumoperitoneum or obstruction. Classic X-ray findings for volvulus include a bent inner tube for sigmoid volvulus, a distended colon projecting into the left upper quadrant for cecal volvulus, and a kidney or coffee bean sign on the right side of the abdomen for cecal bascule[61] (►**Fig. 3**). It is important to note that the cecum may be displaced anywhere in the abdomen.[62] Patients with any evidence of perforation on imaging should be taken immediately to the operating room.

Abdominal X-ray can be limited if the closed loop is fluid-filled, oriented anteroposteriorly, or obscured by loops of air-distended bowel.[63] If a patient is clinically stable, CT of the abdomen and pelvis may be obtained. In sigmoid volvulus, the sigmoid colon will appear dilated and a whirl pattern of the mesentery is formed. A bird's beak may be seen both proximally and distally along with absence of air in the rectum.[63] In cecal volvulus, the cecum typically appears in the upper mid and left abdomen and can be traced back to the level of the volvulus that shows the classic swirling of the bowel and mesentery known as the "whirl sign."[64]

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There are three types of cecal volvulus commonly described.[65(30)] Type I is an axial cecal volvulus that develops from clockwise axial torsion along the long axis with the volvulized cecum remaining in the right lower quadrant.[48,65,66] Type II is a loop cecal volvulus that includes the terminal ileum and involves a counterclockwise twisting of both the cecum and terminal ileum, with the cecum ending up in an abnormal location (most commonly the left upper quadrant).[48,65,66] The third type of cecal volvulus, cecal bascule, involves the cecum folding upwards on itself and is often seen as a dilated loop in the mid-abdomen.[48]

Medical Treatment

Principles of Treatment

Given that volvulus is a closed loop obstruction, the bowel must be urgently detorsed to avoid bowel compromise and potential perforation. As with any acute abdominal process, the patient should be properly resuscitated prior to proceeding with a decompressive or operative procedure. Because of the high risk of torsion recurrence without resection, volvulus is rarely treated with decompression alone. The most commonly accepted treatment algorithm for sigmoid volvulus includes decompression with contrast enemas or flexible sigmoidoscopy followed by prompt, not necessarily urgent resection (usually during the same hospital admission). In contrast, if cecal volvulus is diagnosed, there is little role for decompression due to risk of perforation. The patient should be taken immediately to the operating room; however, variations in practice do exist.

Contrast Enemas

Historically, contrast enemas (either water soluble or barium) are both diagnostic and therapeutic for sigmoid volvulus. Imaging will demonstrate a beak-shaped area at the level of the distal aspect of the twist, with no contrast passing proximal to that point. Contrast enemas are diagnostic in ~88% of cases.[44] The enema may also reduce the volvulus. Comparatively, for cecal volvulus, once diagnosed, there is little role for contrast enema due to risk of perforation. The patient should be taken urgently to the operating room. However, if an enema study is performed, findings for cecal volvulus demonstrate distal colon decompression with beak-like tapering at the level of the contrast and lack of contrast in the dilated proximal colon and terminal ileum.[61] Overall, endoscopic decompression (which is mainstay of treatment for sigmoid volvulus) is now more commonly utilized than contrast enemas as they provide the opportunity to evaluate the viability of the bowel. Furthermore, decompression alone is not sufficient treatment for volvulus due to documented high rates of recurrence.

Endoscopic Decompression

Decompression with endoscopy can be done using rigid proctoscopy, flexible sigmoidoscopy, or colonoscopy. Certain studies support the use of colonoscopy over sigmoidoscopy due to the lower success rate of identifying the torsion with a sigmoidoscope (24%).[67] Endoscopic decompression of the colon entails use of a scope to advance slowly through the volvulized part of the colon, using minimal insufflation to detorse the colon, restoring blood supply. The point of torsion may appear as a spiral or sphincter. Once the torsion has been passed, there is a release of fluid and gas with dilated bowel visualized proximally. All gas and fluid are suctioned to visualize the bowel lumen and assess viability. Rectal tubes are commonly left in place, positioned proximal to the point of torsion, allowing for decreased distention of the colon and possibly recurrence. Various studies have demonstrated successful rates of endoscopic decompression, some report up to 90% of cases.[68,69]

Surgical Treatment

Sigmoid Volvulus

While decompression successfully detorses the bowel, decompression alone does not sufficiently treat sigmoid volvulus due to the high risk of recurrence. Studies demonstrate recurrence in up to 50 to 70% of patients, with an associated 6% mortality after decompression without subsequent resection.[36,70,71] The benefits of decompression include transitioning an operation from emergent to urgent, allowing for resuscitation and administration of mechanical bowel preparation. Currently, there is not a standardized time to wait between decompression and resection, but the typical time has been reported as 1 to 3 days. As in standard practice, patients with gangrenous bowel identified on endoscopy, hemodynamic instability, or signs of perforation should undergo emergent exploration.

Intraoperative surgical management depends on multiple factors including patient-specific factors (age, comorbidities, immunosuppression) and medical acuity (hemodynamic stability, presence of necrotic bowel). The mainstay of surgical treatment is resection of the diseased bowel, typically with a Hartmann's procedure or resection with primary anastomosis.[71] Most procedures are conducted through laparotomy; however, a few studies have demonstrated that laparoscopy may be safe and feasible.[72]

Overall outcomes of sigmoid volvulus depend on the viability of the bowel at time of presentation; less than 10% mortality is seen in patients who do not have gangrenous bowel upon initial presentation. Studies have demonstrated that the extent of colectomy correlates with morbidity and mortality.[70] Furthermore, recurrence of volvulus from lack of prior surgical resection carries the greatest mortality risk, over 20%.[69]

Nonresectional methods such as a sigmoidopexy and mesosigmoidopexy exist; however these methods are associated with higher recurrence and then subsequent increased morbidity and mortality.

Cecal Volvulus

In contrast to sigmoid volvulus, nonoperative reduction in cecal volvulus is only successful in 5 to 30% of cases. Attempts at nonoperative reduction can increase the risk of perforation.[43,73] These patients are usually taken urgently to the operating room after proper resuscitation. There are

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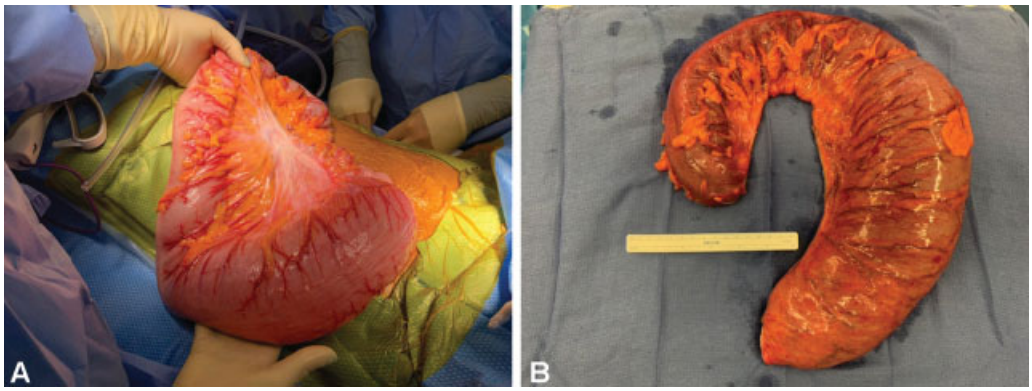

Fig. 4. Intraoperative images from a partial colectomy for a cecal volvulus.

different surgical options for cecal volvulus: resective versus fixative. Operative decision-making depends on a variety of factors, including viability of the bowel and clinical status of the patient. If there is obvious perforation or necrosis of the bowel, resection with diversion or primary anastomosis should be performed. If bowel is viable, surgeons may opt to detorse with a fixation procedure such as a cecopexy or cecostomy tube.

Cecopexy entails mobilizing peritoneum off of the right lateral abdominal wall and retroperitoneal surface followed by securing the serosa of the anterior right colon to the created peritoneal flap. This maneuver can be difficult due to the thinness of the distended colonic wall. Cecopexy is performed less frequently (22% of cecal volvulus operations) as it is associated with higher recurrence (20–30%) compared with resection.[59,74] Studies examining mortality have been equivocal; some report higher mortality rates of up to 22% and others indicate similar rates when compared with resection. One retrospective analysis demonstrated a mortality of 14.2% with operative detorsion compared with a mortality rate of 7.2% with cecal resection.[44]

The least invasive option is a cecostomy tube, which is rarely used (only 2% of cases) due to the high rate of mortality (up to 66.6%) and complications. It involves placing a tube within the cecum, connected to the anterior abdominal wall, for both decompression and fixation.[44]

Surgical resection remains the most common treatment for cecal volvulus.[36] Options include resection of cecum and ascending colon (►**Fig. 4**) followed by either primary anastomosis alone, end ileostomy without anastomosis, or primary anastomosis with a proximal diverting loop ileostomy. Similar to the management of sigmoid volvulus, surgical decision-making for cecal volvulus depends on standard surgical principles such as the clinical condition of the patient and anatomical factors including condition of the bowel (viability and length). Resection has an extremely low recurrence (<10%) and a low mortality (5–10%).[75]

However, complication rates can be as high as 15%.[36] Resection-specific complications include leak, abscess, and fistula formation. Even with the best surgical outcomes, volvulus remains a highly morbid condition with long hospitalizations; average length of stay has been reported to be 11 and 15 days for cecal and sigmoid volvulus, respectively.[36]

Conclusions

Colonic volvulus is important to recognize early due to its high morbidity and mortality. Fortunately, early recognition is feasible given the classic presenting symptoms and imaging. Patients are more ill and tend to be elderly. Treatment entails decompression and surgical resection versus fixation, depending on the type of volvulus. Decision-making for both the conservative and surgical treatments depend on various patient factors. Understanding the pathophysiology of colonic volvulus and the best treatment strategy for each volvulus type are the keys to minimizing complications and recurrence.

Conflict of Interest
None declared.

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