Large Bowel Obstruction
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Large Bowel Obstruction

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Large Bowel Obstruction

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INTRODUCTION

Large bowel obstruction (LBO) is a clinical entity that results from a mechanical hindrance to the intestinal passage distal to the ileocecal valve. The extent of a mechanical obstruction can be described as either partial (allowing for some passage of gas or stool) or complete (no passage of stool or gas); the onset of the developing LBO is characterized as acute or chronic.1

There are numerous etiologies of LBO (Table 1) as well as wide variation in etiology with age and geography.2 For example, cancer is the most common cause of LBO in the United States, whereas colonic volvulus is more common in Africa, South America, Russia, and Eastern Europe (geographical distribution termed as “volvulus belt”).3 The neonatal and pediatric population presents with LBO in the context of intussusception or congenital malformations such as Hirschsprung’s disease and imperforate anus.

Depending on the timing and dynamic course of the LBO, the clinical presentation can evolve either acutely or slowly and progressively. Common early complaints are varying levels of pain, obstipation, and increasing abdominal distention; later in the course, nausea, vomiting, and systemic signs (eg, electrolyte imbalances, hemodynamic changes, sepsis, multiorgan dysfunction) may develop.1,4 The colonic obstruction leads to an intraluminal accumulation of stool and intestinal gas proximal to the site of obstruction. Initially, increased peristaltic waves attempt to overcome the obstacle (hypermotility phase). With continuing distention, however, the intraluminal and intra-abdominal pressure may ultimately exhaust the colonic muscle contractility (ileus phase) and compromise the lymphatic, venous, and arterial blood flow. As the events progress, the large bowel suffers a worsening vascular compromise that may lead to bowel ischemia up to full-thickness gangrene and perforation of the colon.

A number of key components are important for successful management of patients with LBO:1 early recognition of the clinical symptoms, a good understanding of the impact of different underlying causes, timely initiation of diagnostic and management steps, and swift, strategic planning of subsequent interventions. This article reviews the management of LBO using 3 illustrative patient presentations. Definitions of terms pertinent to this discussion are presented in Table 2.

CASE 1: MALIGNANT OBSTRUCTION

CASE PRESENTATION

A 56-year-old woman presents with a 6- to 8-week history of abdominal cramping, constipation, and poor appetite. Apart from an unintentional 7-lb weight loss and a history of asthma, the past medical and surgical history are negative. The patient denies any rectal bleeding. On clinical examination, she has a distended abdomen with mild diffuse tenderness to palpation, but no guarding, rebound, or percussion tenderness (ie, no local or diffuse peritoneal signs). There is no evidence for a hernia, and there are no palpable masses. The digital rectal exam reveals an empty rectal vault, but no mass or gross blood are found in reach of the finger. Laboratory test results are as follows: hemoglobin, 10.4 mg/dL; blood urea nitrogen (BUN), 25 mg/dL; creatinine, 2.0 mg/dL; sodium, 138 mEq/L; potassium, 3.6 mEq/L; amylase, 23 U/L; and lipase, 35 U/L.

- What is the differential diagnosis in this patient?

This patient’s clinical presentation is consistent with a relatively slowly evolving LBO. Excluding hernias, the most common cause of LBO in adults in the United States is carcinoma (60%–80%), followed by a diverticular stricture (10%–20%) and a colonic volvulus (5%).2 Other causes are possible, but short of a documented negative colonic evaluation in the very recent past, the index of suspicion for a malignant obstruction in a previously healthy individual should be high based on the progressive painless worsening and the associated anemia. Simple poor-habit constipation or colonic inertia (slow transit constipation) are theoretically possible but unlikely given both a weight loss and the presence of anemia in this patient. The presentation is not characteristic for a volvulus, which commonly would be more acute and associated with significant pain. A benign
The clinical symptoms of a LBO depend on the underlying etiology and the site of the obstruction. The more proximal the location of a colonic obstruction, the less likely that the obstruction will result in a change in bowel habits or a significant distention prior to becoming a complete obstruction (only about 2%–5% of colon cancers).5 Gradual or sudden onset of crampy abdominal pain is common. Severe pain—potentially out of proportion to the exam—suggests an acute event, typically either a perforation or some ischemic component (eg, from a volvulus or a closed loop obstruction).

**History and Physical Examination**

A problem-oriented history should focus on understanding the exact timeframe for the development of the obstructive symptoms and whether the obstruction is complete or incomplete, as a complete obstruction may not tolerate any delay.1 The patient should be asked whether there have been previous similar episodes, a change in bowel habits or stool consistency (more likely with lesions distal to the splenic flexure), or visible bleeding per rectum (more likely from a distal source); documented anemia or suggestive symptoms (fatigue, weakness) are equally important. It is crucial to know of any previous intestinal surgeries or colonic evaluations (particularly if they occurred in the recent past). The general and cardiopulmonary performance status and respective comorbidities should be elucidated.

The clinical exam should actively assess the patient’s overall condition and vital signs and determine whether signs of toxicity or sepsis are present before focusing on the abdominal and mandatory rectal exam. A rigid proctoscopy is considered an "extension of the digital exam";
Large Bowel Obstruction

Laboratory Tests

Laboratory tests are not suited to establish or rule out a particular diagnosis. They can at best support the overall assessment and management of the patient when preparing for possible interventions. In that sense, a set of basic laboratory values is indicated: complete blood count (CBC, with white blood cell count, hemoglobin, and platelets), electrolytes, renal function tests (creatinine, BUN), and coagulation parameters may be useful in assessing and optimizing the patient’s clinical state prior to further intervention. Neither fecal occult blood test or tumor markers (eg, carcinoembryonic antigen) are indicated as neither a positive nor a negative result would change the management of the LBO.

Diagnostic Imaging

Radiological imaging is essential in the workup of a patient suspected of having a LBO, but images alone are not sufficient for a definitive diagnosis. An upright chest radiograph and an abdominal series can rule out or confirm free intraperitoneal air and document the distribution and extent of the colonic distention (Figure 1). Specific diagnostic features (eg, “bent inner tube,” or “coffee bean,” sign) may occasionally give a clear hint to the diagnosis of a volvulus.

A limited colonic enema with a water-soluble contrast agent is a useful first step to determine the location and level of an obstruction and to suggest the possible nature of the obstruction (eg, “apple core” lesion in neoplasms, “bird’s beak” in sigmoid volvulus). Furthermore, it may provide information that allows for stenting of the obstruction. This study, however, should be performed with caution to avoid trapping of the contrast agent proximal to the obstruction, where the hyperosmolar fluid may add to the osmotic fluid accumulation, leading to further distention and possible rupture of the colon.

If the patient does not require urgent surgical intervention, computed tomography (CT) scanning can add to the preoperative evaluation by providing information on the colonic diameter, bowel wall integrity (pneumatosis, portal venous gas), transition zone, lymph node status, and presence of distant metastasis. Use of contrast agent should be avoided if it would aggravate the clinical situation (eg, oral contrast when abdomen is very distended; intravenous contrast in prerenal kidney dysfunction), and rectal contrast may be of value in such settings. In the case patient, intravenous contrast should be avoided unless the prerenal failure is corrected.

Instrumentation

A flexible endoscopy with minimal insufflation should be performed with the goal of visualizing the obstructing lesion and obtaining biopsy specimens. Given the substantial incidence of synchronous neoplasms, a full colonic evaluation would be desirable; however, this is not realistic in an LBO unless the obstruction is incomplete or overcome to allow for an orthograde bowel cleansing.

CASE 1 CONTINUED

The patient is worked up with a chest radiograph and abdominal radiograph series, which do not show evidence of free air. The colon is distended from the cecum to the descending colon, with a cecal diameter of 10 cm. A few distended small bowel loops are seen as well. A small amount of air is seen in the rectum. A flexible sigmoidoscopy is performed, and an obstructing tumor is found 40 cm from the anal verge, leaving no doubt that a malignant process is the cause of this patient’s LBO. The lesion cannot be passed.
Biopsy specimens are taken. The patient continues to be highly symptomatic, with abdominal cramping and occasional passage of gas and stool.

• What are the next steps in the management of this patient?

**MANAGEMENT**

There are 3 major concerns when dealing with an obstructing colon cancer: the potential for a complication of the obstructed and distended bowel segment (eg, gangrene and perforation with fecal peritonitis and sepsis); the actual obstruction and underlying disease responsible for it; and the inability to reliably assess the colon proximal to the obstruction. From a practical and patient standpoint, however, the biggest question is whether the situation can be solved in a single stage or requires more than one intervention and possibly a stoma. Clinical signs and the degree of obstruction, perforation, and progression of symptoms determine the urgency and type of surgical intervention in the setting of LBO, as well as the outcome.

In this case, the bowel is not completely obstructed and the patient does not have peritonitis, but she is highly symptomatic. The cecum has reached a substantial diameter, even if it is still below the alarming threshold of 12 cm. The risk of a perforation does not strictly correlate with the absolute diameter, but it undoubtedly becomes significant when a cecum greater than 12 cm or a transverse colon of greater than 6 cm is found. Only expeditious decompression can avert a disaster.

**General Management**

Preoperative optimization is beneficial, if time allows. Intravenous fluid resuscitation should focus on correcting the patient’s electrolyte and fluid status and normalizing the prerenal azotemia. A nasogastric tube is inserted to decompress the upper gastrointestinal tract and avoid vomiting with aspiration. Cardiopulmonary risk factors (eg, the patient’s asthma) should be addressed and optimized. Because an obstruction is a sign of an advanced colon cancer, further evaluation with CT (see discussion above) may assist in the surgeon’s decision-making. Patients and their family should be informed of the substantial morbidity and mortality historically associated with LBO and of the high probability that a stoma will be required, of which up to 30% may never be reversed. If a patient has a clinically relevant obstruction, a chemotherapy approach instead of an intervention is not typically recommended as the risk for decompression in a most vulnerable period is too high.

**Decompression**

The best option to decompress a left-sided colon obstruction and turn an urgent/emergency situation into a (semi)-elective one is to endoscopically place a self-expandable metal stent. Depending on the endoscopist’s experience, the rate of successful decompression is around 80% to 90%. The course of action following stent placement depends on the patient’s overall condition and the tumor burden. In a minority of patients with metastatic disease or contraindications to surgery, the stent will be considered the definitive palliative procedure, but more commonly stenting is only a “bridge to surgery”; after stabilization and appropriate workup, including full colon cleansing and evaluation, the standard resection with primary anastomosis is planned. A successful stent placement reduces the risk of an anastomotic leak, the need for an ostomy, and the overall mortality rate all to less than 5%. The 2 most common complications of stent placement are migration and reobstruction. Stent migration can lead to intractable tenesmus, urgency, and incontinence. Similar symptoms can be seen if the stent is primarily placed too distally in the rectum. In rare circumstances, when a left-sided obstruction cannot be stented and a decompression plus resection would not be tolerated by a fragile patient, creation of a stoma proximal to the site of obstruction may achieve the decompression and allow for stabilization of the patient. If required, such a colostomy can be performed under local anesthesia.

**Decompression Plus Resection**

Most right-sided colon obstructions are not stented or diverted but rather are directly approached with a resection of the obstruction and the obstructed/dilated colon. For left-sided obstructions that are not amenable to stenting, there are a number of surgical options.

**Hartmann-type resection.** This option involves resection of the obstruction and bringing out the proximal bowel as an end colostomy while leaving a blind distal end. This discontinuous option eliminates the obstructing cancer and preserves colon length; it allows the distended colon to decompress through the colostomy and avoids an anastomosis and potential anastomotic leak. However, it requires a second, sometimes difficult, operation to reverse the stoma. Evaluation of the proximal colon before the stoma takedown is mandatory.

**Resection with primary anastomosis, with or without proximal diversion.** This restorative option, which also eliminates the obstructing cancer but immediately reconstitutes the intestinal continuity, should be reserved for stable patients whose bowels have an excellent tissue quality. There is a risk of an anastomotic leak. A proximal loop colostomy or ileostomy may reduce the septic
sequelae from a leak. This option also requires a second operation, which is often much easier to perform than a Hartmann reversal. Evaluation of the proximal colon before the stoma takedown is mandatory.

(Sub-)total colectomy with ileosigmoidostomy or ileorectostomy. This restorative option eliminates the obstructing cancer as well as the distended large bowel that potentially harbors synchronous lesions in it. This extension of the right-sided management to tumors on the left is an elegant compromise with acceptable bowel function if the anastomosis is proximal to the rectum.

Resection with on-table lavage and primary anastomosis. This restorative option removes the obstructing cancer and eliminates the stool load in the distended colon before reconstituting continuity with a colo-colonic anastomosis, emulating a bowel cleansing similar to an elective resection. The proximal colon could be visualized intraoperatively after the clean-out or postoperatively after 4 to 6 weeks. As there is the risk of an anastomotic leak, this surgical option should be reserved for stable patients whose bowels have a good tissue quality.

The decision regarding which of these specific options to use depends on the patient’s overall condition and the quality of the bowel at the time of surgery. The primary goal is to create a condition that affords the best chance for a smooth recovery while preserving as much quality of life as possible.

**CASE 1 CONCLUSION**

On the same day she presented, the patient undergoes a colonoscopic stent placement, which successfully decompreses the colon. The previously obtained biopsy specimens confirm the presence of adenocarcinoma. Further workup reveals no evidence of metastatic disease. A barium-air double-contrast enema is performed 1 week after the stent placement, which reveals no evidence of synchronous colon lesions. Eight days after presentation, the patient undergoes an elective laparoscopic sigmoid resection with primary anastomosis. The final tumor stage is a pT3N1 cM0 (stage III) with 3 out of 21 lymph nodes positive for cancer. Adjuvant chemotherapy is initiated.

**CASE 2: SIGMOID VOLVULUS**

**CASE PRESENTATION**

A 63-year-old man presents to the emergency department with sudden onset of crampy abdominal pain and increasing abdominal distention. He has not had any bowel movements or flatus since symptoms started 4 hours prior. His bowel function prior to this episode was normal. A colonoscopy performed 2 years ago was normal except for internal hemorrhoids. The patient’s past surgical and medical history are significant for a left-sided inguinal hernia repair, arterial hypertension, and a 20-year history of chronic back pain for which he has been taking oxycodeone and nonsteroidal anti-inflammatory drugs (NSAIDs). In addition to his earlier complaints, the patient is now throwing up and is slightly disoriented.

On clinical examination, the abdomen is massively distended. There is an unremarkable scar in the left groin and a small reducible inguinal hernia on the right side. The abdomen shows local tenderness to palpation with guarding and rebound tenderness (local peritoneal signs) in the left lower quadrant. The rectal exam shows no masses and no gross blood.

Laboratory testing results are as follows: white blood cell count, 18,000/µL; hemoglobin, 16 mg/dL; BUN, 30 mg/dL; creatinine, 2.5 mg/dL; and HCO3-, 8 mg/dL. Electrolytes and liver function tests are within normal limits. An abdominal radiograph series shows a massively dilated loop of colon but no free air.

- **What diagnosis is suggested by this patient’s clinical presentation?**

This patient’s clinical presentation is consistent with an acute complete LBO. While the normal colonoscopy 2 years prior does not completely rule out a malignancy, it would be rather unusual to have a locally advanced lesion that presents primarily with a complication. Hence, the most likely cause for this obstruction is a sigmoid volvulus. Other differential diagnoses should be kept in mind (eg, a megacolon with complications).

Volvulus can be described as an axial rotation of the colon around its narrow-based mesentery and vascular pedicle. The twist results in a closed loop obstruction and strangulation, which, if left untreated, will lead to gangrene and perforation. Colonic volvulus accounts for 3% to 10% of all LBOs and is the third most common cause of LBO in the United States (after malignancy and chronic diverticulitis). The sigmoid colon is the most common site (60%–75% of the time), followed by the cecum and the transverse colon. Various clinical states are thought to contribute to the pathophysiology of this disease. These include congenital anatomic hypermobility from incomplete retroperitoneal attachments and/or incomplete embryological rotation, floppy mesentery with a narrow base, and a fiber rich-diet (which is thought to explain the much higher incidence in the geographical “volvulus belt” in Africa, South America, Russia, and Eastern Europe). Multiple other conditions
have been associated with volvulus, such as pregnancy, constipation, Chagas disease, inflammatory bowel disease, Parkinson’s disease, and diabetes. The incidence of volvulus is higher in elderly men and institutionalized patients. There is a high mortality rate (50%–80%) in cases involving intestinal ischemia, and therefore early diagnosis and treatment are crucial.

- What is an appropriate diagnostic plan for this patient?

**DIAGNOSIS**

**Clinical Presentation**

The clinical signs and symptoms of sigmoid volvulus can be summarized in a clinical triad: acute crampy abdominal pain, abdominal distention, and cessation of bowel activity. Late signs related to bowel compromise are fever, chills, nausea, vomiting, hypotension, shock, and peritoneal signs. In the case patient, several parameters suggest a serious condition: the patient’s concerning abdominal exam, lethargy, leukocytosis, prerenal renal failure, and metabolic acidosis are all late findings that suggest advanced strangulation with ischemia or perforation.

**History and Physical Examination**

Despite the acuteness of this patient’s symptoms, there are occasionally patients who report a long history of intermittent obstructive symptoms and distentions that would suggest episodes of subvolvulus. The time and progression after onset of symptoms are valuable parameters for patient assessment. Physical signs consistent with strangulation, ischemia, necrosis, and perforation can range from pain out of proportion to the exam to peritonitis (localized or diffused) and dark bloody discharge on rectal exam as a result of mucosal sloughing.

**Laboratory Tests**

Similar to any clinical presentation of LBO, laboratory tests must be tailored to the specific circumstances based on history and physical exam, the patient’s overall condition, and the planned management. With rapid clinical deterioration, blood draws should not delay the intended intervention. However, a CBC and a basic metabolic panel provide a good picture of the patient’s baseline function and can guide the resuscitative measures. Arterial blood gas analysis may be ordered, depending on the clinical picture (eg, if needed to guide corrective measures in acidotic patients and those requiring resuscitation). The value of measuring serum lactic acid is limited in bowel ischemia related to bowel strangulation because there is blood flow neither in nor out of bowel wall. Therefore, the accumulation of lactic acid within the tissue may not be paralleled by a rise in the systemic lactate levels despite the ongoing cellular destruction.

**Diagnostic Imaging**

Plain radiograph can detect a sigmoid volvulus in 70% to 90% of cases, with an air-fluid level seen in the characteristically dilated loop of colon and a relative lack of air in the rectum. The classic radiographic finding for a sigmoid volvulus is the “bent inner tube,” or “coffee bean,” sign with the convexity of the loop lying in the right upper quadrant. Analyzing a 2-dimensional image of a 3-dimensional event can sometimes be difficult; therefore, a water-soluble contrast enema may be used to rule out a pseudoobstruction and demonstrate the sigmoid volvulus with the “bird’s beak” appearance (sharp narrowing of the contrast column at the site of the volvulus). The contrast enema has a potential to achieve a detorsion of the sigmoid volvulus, but the success rate is only about 5%. Only if the diagnosis is still in doubt, a CT scan may be useful. The characteristic finding is a whirl in the mesentery.

**Instrumentation**

A rigid or flexible sigmoidoscopy at the bedside is generally the first measure, less in diagnostic than in therapeutic intent. A detorsion maneuver should be attempted unless there is a high index of suspicion that the bowel is already necrotic. Through a combination of gentle insufflation with a small rotating movement of the instrument, a sigmoid volvulus can be successfully decompressed 85% to 90% of the time, typically with an explosive gush of liquid stool and gas. The rate of a recurrent volvulus is generally high, affecting more than 50% of patients who are not taken to surgery. Immediate recurrent volvulus can be minimized by insertion of a soft rectal tube (25–32 F) for 48 to 72 hours. During that time, the patient should be monitored clinically while steps are initiated to optimize the patient’s general condition and to perform a full colon evaluation prior to an elective sigmoid resection.

**CASE CONTINUED**

The patient is aggressively resuscitated and remains hemodynamically stable. His clinical exam remains unchanged. A rigid sigmoidoscopy is performed at the bedside, but the volvulus cannot be untwisted. The patient is brought to the operating room and the area is again visualized with a flexible sigmoidoscopy. At 25 cm there is mucosal ulceration and dark blood.
• What is the next step to resolve this patient’s sigmoid volvulus?

There is considerable concern for ischemia or perforation, and therefore any intervention should be performed in the operating room with access to anesthesia and an exploratory laparotomy. The visualization supports the impression from the clinical exam that the bowel wall has already been compromised. Mucosal ulcerations and dark blood at the site of volvulus are strong indicators of ischemic changes. The depth of the injury cannot be assessed endoscopically. Despite reasonable short-term success with colonoscopic decompression in a majority of cases, it would be unsafe to try to push the instrument through this weakened segment. A surgical exploration should be performed.

CASE CONTINUED

An exploratory laparotomy is performed, which confirms the presence of a sigmoid volvulus. The involved bowel segment is massively dilated and shows obvious gangrene but no frank perforation.

• What concerns does nonviable bowel present?

Gangrenous bowel is encountered in 5% to 10% of patients with colonic volvulus.19,20 Only a combination of a timely presentation, timely recognition of the symptoms, and swift action can prevent this complication. Ideally, detorsion of the volvulized segment with decompression of the bowel would allow for recovery of the large bowel proximal to the volvulus. Unless there are prohibitive contraindications for any kind of surgery, an elective resection should be planned, as the recurrence rates are very high.19,21,22 By the time of an elective resection, the colon segment to be resected will be better delineated, and a primary anastomosis is routine. Once the bowel becomes necrotic, however, the affected segment remains problematic even after detorsion, releasing inflammatory toxins and bacteria that result in overwhelming systemic stress and a septic response. Furthermore, the bowel proximal to the volvulus may also be distended and structurally compromised, possibly making a reconnection less than optimal. Mortality rates of up to 50% have been reported for sigmoid volvulus with nonviable bowel.19–21

• What are the surgical options for sigmoid volvulus?

PROCEDURES

The selection of the operative strategy should again be based on the findings of the index segment, the quality of the proximal colon, and the patient’s overall condition (see also Case 1). A Hartmann resection is a safe choice at the price of requiring a second surgery to reverse the stoma. Alternatively, and particularly if the remaining colon is also very dilated and damaged, one may consider a subtotal colectomy; depending on the quality of the small bowel, either an ileostomy or a primary anastomosis is performed.

Even if an acute volvulus is successfully reduced, an elective resection is recommended.19,22 A resection with primary anastomosis remains the most reliable option, but alternatives include a mesosigmoidoplasty, which entails a widening of the mesenteric base without bowel resection; tube sigmoidopexy has been reported, but the results have not been corroborated.21

OTHER SITES OF COLONIC VOLVULUS

Colonic volvulus affects the cecum in 20% to 35% of cases. It occurs in a slightly younger population than sigmoid volvulus and is more common in females.19,20 Cecal volvulus can occur by 2 mechanisms: ileocolic axial rotation or a horizontal and upward folding of the floppy cecal pole, referred to as “cecal bascule.” This latter mechanism may be triggered by colonic hypermobility, pregnancy, previous surgery, congenital malrotation, and obstructing lesions of the left colon. Plain radiographs are diagnostic for a cecal volvulus in only 20% of cases, and the majority are diagnosed on exploration. Endoscopic decompression is seldom successful. There is a higher incidence of ischemic changes in cecal volvulus (20%–30%) compared to sigmoid volvulus (5%–10%).20 The procedure of choice is a segmental resection with primary anastomosis, although the combination of cecopexy with a tube cecostomy may have similarly low recurrence rates.21,22 When detorsion, cecostomy, and cecopexy are performed alone, the recurrence rates are considerably higher (12%–14%). A cecostomy may carry a historical mortality rate of up to 32%.

The transverse colon and splenic flexure area represent the third most common site for a colonic volvulus, but volvulus in these areas is still rare. It is usually diagnosed on exploration only and is therefore most commonly addressed with a segmental resection or an extended hemicolecction.

CASE RESOLUTION

The patient undergoes a technically successful Hartmann procedure (resection of the necrotic sigmoid with proximal end colostomy and blind rectal stump). However, he postoperatively requires admission to the intensive care unit after intraoperatively suffering a subendocardial myocardial infarct. His ostomy starts functioning on postoperative day 4. Four months after discharge, he has an elective colonoscopy followed by an uneventful ostomy takedown 2 months later.
CASE 3: PSEUDO OBSTRUCTION

CASE PRESENTATION

An 89-year-old nursing home resident is found down in her room during morning rounds. She is transferred to the emergency department, where she is found to have a left-sided femur neck fracture. A hemiarthroplasty with implantation of a bipolar hip endoprosthesis is carried out in spinal anesthesia. On postoperative day 2, general surgery is called to evaluate the patient for increasing abdominal distention. The patient has a history of dementia, depression, and hypertension. Her past surgical history is only significant for a hysterectomy with bilateral salpingo-oophorectomy 20 years prior. She currently is on a beta-blocker and morphine patient-controlled analgesia (PCA). The patient has had a few loose bowel movements in the past 24 hours. She has never had a colonoscopy. On clinical examination, the patient is hemodynamically stable. She has a body mass index of 21. The abdominal exam shows an unremarkable scar without hernia, significant distention, and hypoactive bowel sounds, but no tenderness to palpation or guarding. On digital rectal exam, there is small amount of stool in the rectal vault; a mass cannot be palpated, and there is no blood on the stool. An abdominal radiograph shows a significantly distended colon with a cecal diameter of 11 cm.

- What is the differential diagnosis for abdominal distention?

There is a wide range of possible differential diagnoses for abdominal distention. These include, among others, a tumor (colorectal cancer, carcinomatosis, pseudomyxoma), a decompensated benign stricture (from ischemia, diverticulitis), toxic or pre-toxic megacolon (eg, from *Clostridium difficile* colitis), a hematoma, ascites, portal vein thrombosis, as well as functional disorders (fecal impaction, pseudoobstruction). Because colonic distention is a key finding, a LBO with a mechanical obstacle has to be postulated until proven otherwise. This patient’s circumstances and clinical presentation very much suggest a functional cause of the colon distention; that is, colonic pseudoobstruction, or Ogilvie’s syndrome.26 The clinical presentation and colonic distention with pseudoobstruction are not fundamentally different from a true LBO, but they develop in the absence of a mechanical obstruction. This acquired condition commonly develops in hospitalized or nursing home patients and is triggered by other events. Numerous factors have been associated with the condition (Table 3), including medications (eg, opiates, psychotropics, anticholinergics, Table 4), surgery (eg, cardiothoracic or spine), trauma, immobility, sepsis and malignancy.26,27 Pseudoobstruction is thought to result from an imbalance in the autonomic nervous system, with excess sympathetic stimulation and a paucity of parasympathetic input, producing an inhibitory effect on colonic motility.26 The dilatation is most prominent in the right and transverse colon.

- How is the diagnosis of colonic pseudoobstruction established?

DIAGNOSIS

Colonic pseudoobstruction is a diagnosis of exclusion. A mechanical obstruction (true LBO) or a toxic megacolon must be ruled out.

Clinical Presentation

Clinical symptoms are mainly increasing abdominal distention, most commonly associated with constipation and occasionally with diarrhea. Pain is usually absent, but the patient may not feel comfortable and may even develop some respiratory distress as the increasing abdominal volume limits respiratory excursions.
Abdominal tenderness on exam, fevers, or chills should prompt a search for ischemia or perforation.

**History and Physical Examination**

Pseudoobstruction can be suspected based on circumstantial evidence. It usually develops while patients are hospitalized for another clinical problem. The key to diagnosis is the underlying medical condition and medication that contribute to pseudoobstruction (anticholinergics, opiates). Pseudoobstruction most commonly is not associated with an intrinsic colonic pathology, but occasionally it can be a first manifestation of *C. difficile* or ischemic colitis. Serial clinical and radiological abdominal exams are the key components of monitoring the disease progression and success of therapeutic interventions.

**Laboratory Tests**

Metabolic and electrolyte imbalances can contribute to the colonic dysmotility and pseudoobstruction. A CBC and basic metabolic panel should be obtained and electrolytes corrected appropriately. Since this patient’s symptoms developed in the postoperative period after a hip surgery with respective antibiotic prophylaxis, the stool should be checked for *C. difficile* toxins A and B.

**Imaging and Instrumentation**

Plain abdominal and chest radiographs are used to initially document the colonic distention and rule out free peritoneal air (Figure 2). Subsequent diagnostic management should aim at ruling out a true LBO or structural abnormalities other than the colonic distention. A watersoluble contrast enema is indicated as a first step. However, it may be contraindicated if signs of toxicity (fevers, tachycardia, elevated white blood cell count) are present. The contrast should rapidly and freely flow from the rectum to the dilated bowel segment (Figure 2). Alternatively, an endoscopic evaluation (preferably with disconnected air insufflation) may be considered. Apart from ruling out an obstructive transition point, the endoscopy allows for assessment of the mucosal integrity and for colonic decompression at the same time.

**CASE CONTINUED**

The patient is found to have hypokalemia, which is corrected over the next 24 hours. In
addition, the PCA is stopped and pain control is transitioned over to NSAIDs. A water-soluble contrast enema exam does not show any mechanical obstruction. While the initial radiographs showed a diffuse colonic distention with a cecal diameter of 11 cm, a follow-up radiograph after 24 hours demonstrates mild progression with a cecal dilatation to 14 cm. The patient’s clinical condition and physical exam remain unchanged. Testing for C. difficile toxin is negative.

- **What are the 2 main methods for decompressing a colonic pseudoobstruction?**

**DECOMPRESSION METHODS**

Worsening colonic distention carries a risk of secondary bowel injury and perforation if left untreated. Because these complications dramatically increase morbidity and mortality, proactive management is mandatory. Supportive measures in treating colonic pseudoobstruction involve identifying and correcting contributing factors, if possible. Unfortunately, many cofactors cannot be eliminated, such as the need for postoperative pain control and immobility, retroperitoneal or mediastinal swelling, and hematoma after spinal or cardiothoracic surgery. Enemas and prokinetic medications may be considered as supportive measures. Unless success is objectively documented by serial exams and radiographs, a pharmacological or interventional decompression is necessary.

**Colonic Stimulation**

Neostigmine is the most effective drug used for treating colonic pseudoobstruction. It is an acetylcholinesterase inhibitor that potentiates the action of acetylcholine on parasympathetic receptors. In the colon, it results in increased smooth muscle contraction and supports colonic motility. It is effective in decompressing the colon 90% of the time. Side effects include abdominal cramping, nausea, diaphoresis, excessive salivation, and bradycardia. All these adverse reactions are clearly more frequent if the medication is given over just a few minutes. Because of the risk of bradycardia, neostigmine must be administered in a monitored setting (ICU) with atropine available as an antidote. However, Ogilvie’s syndrome typically evolves over a few days, and there is no need to resolve it in a few minutes. Compared with rapid administration, neostigmine given at a dose of 2.0 to 2.5 mg (in 100 mL of normal saline) as an intravenous drip over 2 to 4 hours is much better tolerated, equally effective, and has virtually none of these side effects. It may be necessary to repeat the cycle 1 to 2 times per day for several days before the colon successfully maintains its contractility without stimulation.

**Colonoscopic Decompression**

As the dilatation is greatest in the proximal colon, placing a rectal tube is rarely effective. Only a full colonoscopic decompression has a realistic chance of success. Efficacy rates of up to 80% have been reported if the colonoscope is advanced past the hepatic flexure. To minimize the recurrence rate, a decompression tube may be placed over a guide wire, but repeat colonoscopic decompressions may be necessary. The risk of perforation with the colonoscope is fairly low (estimated range, 2%–3%). The risk is probably related more to the underlying weakening of the colonic wall than to the procedure itself. Avoiding overinsufflation by intentionally disconnecting the air insufflation may help reduce the risk.

**CASE 3 CONTINUED**

Because the patient’s most recent radiograph showed a cecal diameter of 14 cm, a colonoscopic decompression is initially performed. The colon mucosa appears healthy and the decompression is seemingly successful, but the radiograph still demonstrates a cecal diameter of 12 cm. The patient is transferred to a monitored bed. Neostigmine is administered intravenously over 4 hours, and the patient passes large amounts of gas. The abdominal distention improves clinically and on abdominal radiographs. However, after 24 hours, the distention recurs. Therefore, 2 cycles of neostigmine infusions are ordered every day with success.

- **What are the options if both decompression methods are unsuccessful?**

Nonsurgical treatment options fail in a minority of patients. In order to avert a colonic perforation, these patients should be recommended a surgical approach. Several surgical options could be considered, and the selection again depends on the overall clinical picture and the bowel quality. A tube cecostomy for colonic decompression and venting may be placed either open, laparoscopically, or percutaneously. Creation of a loop colostomy may also serve as an effective decompressive measure, but this requires a later operation for reversal. Resective strategies are indicated if there is evidence of altered bowel wall. Injury to the bowel wall can be chronic or acute (ischemia, tears, perforation). The extent of resection has to be individualized and ranges from a subtotal colectomy with primary anastomosis to a discontinuous resection with ileostomy or colostomy.
Large Bowel Obstruction

**SUMMARY POINTS**

- Large bowel obstruction is a potentially life-threatening condition that results from a wide variety of malignant and benign etiologies.
- Clinical management requires a combination of careful history and physical exam, targeted diagnostic studies, and immediate resuscitation.
- Following a swift initial assessment, the surgeon should be able to conclude whether an immediate surgery or procedure is warranted, or whether conservative management might be an option.
- Surgical decision making around LBO is complex and should include a plan on how to deal with (1) the diseased segment causing the obstruction, (2) the dilated colon proximal to the site of obstruction, (3) possible complications (perforation, ischemia), and (4) the possibility of synchronous pathology in the nonaccessible dilated colon.
- The patient’s overall condition and bowel quality are important cofactors to determine whether a single-stage procedure or a two-stage or multistage approach is most appropriate.
- Outcome parameters are overall survival, disease-specific morbidity and mortality, and quality of life aspects (eg, presence of an ostomy).

**REFERENCES**

TYGACIL® (tigecycline) Brief Summary

See package insert for full Prescribing Information. For further product information and current package insert, please visit www.wyeth.com or call our medical communications department toll-free at 1-800-934-5556.

INDICATIONS AND USAGE
TYGACIL is indicated for the treatment of adults with complicated skin and skin structure infections caused by Escherichia coli, Enterococcus faecalis (vancomycin-susceptible and -resistant species), Staphylococcus aureus (methicillin-resistant and -susceptible), Streptococcus agalactiae, Streptococcus anginosus group (includes: S. anginosus, S. intermedius, and S. constellatus), Staphylococcus pyogenes, Enterobacter cloacae, Klebsiella pneumoniae, and Bacteroides fragilis.

TYGACIL is indicated for the treatment of adults with complicated intra-abdominal infections caused by Citrobacter freundii, Enterococcus faecalis (vancomycin-susceptible and -resistant species), Staphylococcus aureus (methicillin-resistant and -susceptible), Streptococcus agalactiae, Streptococcus anginosus group (includes: S. anginosus, S. intermedius, and S. constellatus), Bacteroides fragilis, Bacteroides thetaiotaomicron, Bacteroides uniformis, Bacteroides vulgatus, Clostridium perfringens, and Peptostreptococcus micros.

TYGACIL is indicated for the treatment of adults with community-acquired pneumonia infections caused by Streptococcus pneumoniae (pneumocillin-susceptible isolates), including cases with concurrent bacteremia, Haemophilus influenzae (beta-lactamase negative isolates), and Legionella pneumophila.

CONTRAINdicATIONS
TYGACIL is contraindicated for use in patients who have known hypersensitivity to tigecycline.

WARNINGS AND PRECAUTIONS
Anaphylaxis/Anaphylactoid Reactions
Anaphylactic/anaphylactoid reactions have been reported with nearly all antibacterial agents, including tigecycline. Hypersensitivity reactions can be severe. TREATMENT: Discontinue TYGACIL and institute appropriate therapy. Ensure that patients are monitored after treatment is discontinued because severe, sometimes fatal, reactions can occur following the administration of antibiotic agents in patients with history of sensitivity to these agents (see WARNINGS AND PRECAUTIONS).

Epidemic E. coli bacteremia is also known. When treating E. coli bacteremia, the patient should be apprised of the potential hazard to the fetus. Results of animal studies indicate that tigecycline crosses the placenta and is found in fetal tissues. Decreased fetal weight was noted in rabbits (with associated delays in ossification) and fetal loss in rabbits have been observed with tigecycline (see USE IN SPECIFIC POPULATIONS).

Preclinical studies were performed with TYGACIL. TYGACIL should not be used during tooth development unless otherwise indicated. For use during tooth development (last half of pregnancy, infancy, and childhood to the age of 12), oral use is not likely to be effective or is contraindicated.

Adverse Reactions
In clinical trials, adverse reactions were reported in 59% of patients treated with TYGACIL. The most common adverse reactions were nausea and vomiting which generally occurred during the first 1 – 2 days of therapy. The majority of cases of nausea and vomiting associated with TYGACIL and comparators were either mild or moderate in severity. In patients treated with TYGACIL, nausea incidence was 20% (17% mild, 8% moderate, 1% severe) and vomiting incidence was 18% (11% mild, 6% moderate, 1% severe). In patients treated for complicated skin and skin structure infections (cSSSI), nausea incidence was 35% for TYGACIL and 5% for comparators; vomiting incidence was 20% for TYGACIL and 5% for comparators. In patients treated for complicated intra-abdominal infections (cIAI), nausea incidence was 25% for TYGACIL and 21% for comparators; vomiting incidence was 18% for TYGACIL and 9% for comparators. In patients treated for community-acquired bacterial pneumonia (CABP), nausea incidence was 24% for TYGACIL and 8% for levofloxacin; vomiting incidence was 10% for TYGACIL and 6% for levofoxacin.

Nausea and vomiting were more common in patients treated with TYGACIL than in comparator treatment groups. In the CABP clinical study, nausea incidence was 24% for TYGACIL and 8% for levofloxacin; vomiting incidence was 10% for TYGACIL and 6% for levofoxacin.

Discontinuation from tigecycline was most frequently associated with nausea (1%) and vomiting (1%). For comparators, discontinuation was most frequently associated with nausea (1%). The relationship of these events to treatment cannot be established.

In comparative clinical studies, infection-related serious adverse events were more frequently reported for patients treated with TYGACIL (7%) versus comparators (6%). Serious adverse events of sepsis/septic shock were more frequently reported for patients treated with TYGACIL (2%) versus comparators (1%). Due to baseline differences between treatment groups in this study, the relationship of this outcome to treatment cannot be established.

The following adverse reactions have been identified during postapproval use of TYGACIL. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure. Anaphylaxis/anaphylactoid reactions, acute pancreatitis, hemorrhagic, hypocalcemia, hypoglycemia, hypokalemia, toxicity.

Drug Interactions
Probenecid time or other suitable antibiotic test should be monitored if tigecycline is administered with warfarin [see CLINICAL PHARMACOLOGY (12.9) in Full Prescribing Information].

Concurrent use of antibacterial drugs with oral contraceptives may render oral contraceptives less effective.

USE IN SPECIFIC POPULATIONS
Pregnancy
TYGACIL is not indicated for use in women of childbearing age. There are no adequate and well-controlled studies of TYGACIL in pregnant women. TYGACIL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers
TYGACIL is not indicated for use in women of childbearing age. Nursing mothers should be advised to discontinue breast feeding if he or she are being treated with TYGACIL. TYGACIL is structurally similar to tetracycline-class antibiotics and should be used with caution in patients with known hypersensitivity to tetracycline-class antibiotics.

The use of TYGACIL during tooth development (last half of pregnancy, infancy, and childhood to the age of 12) is not likely to be effective or is contraindicated.

TYGACIL is not indicated for use in women of childbearing age. There are no adequate and well-controlled studies of TYGACIL in pregnant women. TYGACIL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

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TYGACIL is indicated for the treatment of adults with:

- Complicated skin and skin structure infections caused by *Escherichia coli*, *Enterococcus faecalis* (vancomycin-susceptible isolates), *Staphylococcus aureus* (methicillin-susceptible and -resistant isolates), *Streptococcus agalactiae*, *Streptococcus anginosus* grp. (includes *S. anginosus*, *S. intermedius*, and *S. constellatus*), *Streptococcus pyogenes*, *Enterobacter cloacae*, *Klebsiella pneumoniae*, and *Bacteroides fragilis*

- Complicated intra-abdominal infections caused by *Citrobacter freundii*, *Enterobacter cloacae*, *Escherichia coli*, *Klebsiella oxytoca*, *Klebsiella pneumoniae*, *Enterococcus faecalis* (vancomycin-susceptible isolates), *Staphylococcus aureus* (methicillin-susceptible and -resistant isolates), *Streptococcus anginosus* grp. (includes *S. anginosus*, *S. intermedius*, and *S. constellatus*), *Bacteroides fragilis*, *Bacteroides thetaiotaomicron*, *Bacteroides uniformis*, *Bacteroides vulgatus*, *Clostridium perfringens*, and *Peptostreptococcus micros*

- Community-acquired bacterial pneumonia caused by *Streptococcus pneumoniae* (penicillin-susceptible isolates), including cases with concurrent bacteremia, *Haemophilus influenzae* (beta-lactamase negative isolates), and *Legionella pneumophila*

Important Safety Information

- TYGACIL is contraindicated in patients with known hypersensitivity to tigecycline
- Anaphylaxis/anaphylactoid reactions have been reported with nearly all antibacterial agents, including tigecycline, and may be life-threatening. TYGACIL should be administered with caution in patients with known hypersensitivity to tetracycline-class antibiotics
- Isolated cases of significant hepatic dysfunction and hepatic failure have been reported in patients being treated with tigecycline. Some of these patients were receiving multiple concomitant medications. Patients who develop abnormal liver function tests during tigecycline therapy should be monitored for evidence of worsening hepatic function. Adverse events may occur after the drug has been discontinued
- The safety and efficacy of TYGACIL in patients with hospital-acquired pneumonia have not been established
- An increase in all-cause mortality has been observed across phase 3 and 4 clinical studies in TYGACIL-treated patients versus comparator-treated patients. The cause of this increase has not been established. This increase in all-cause mortality should be considered when selecting among treatment options

- TYGACIL may cause fetal harm when administered to a pregnant woman
- The use of TYGACIL during tooth development may cause permanent discoloration of the teeth. TYGACIL should not be used during tooth development unless other drugs are not likely to be effective or are contraindicated
- Acute pancreatitis, including fatal cases, has occurred in association with tigecycline treatment. Consideration should be given to the cessation of the treatment with tigecycline in cases suspected of having developed pancreatitis
- *Clostridium difficile*-associated diarrhea (CDAD) has been reported with use of nearly all antibacterial agents, including TYGACIL, and may range in severity from mild diarrhea to fatal colitis
- Monotherapy should be used with caution in patients with clinically apparent intestinal perforation
- TYGACIL is structurally similar to tetracycline-class antibiotics and may have similar adverse effects. Such effects may include: photosensitivity, pseudotumor cerebri, and anti-anabolic action (which has led to increased BUN, azotemia, acidosis, and hyperphosphatemia). As with tetracyclines, pancreatitis has been reported with the use of TYGACIL
- To reduce the development of drug-resistant bacteria and maintain the effectiveness of TYGACIL and other antibacterial drugs, TYGACIL should be used only to treat infections proven or strongly suspected to be caused by susceptible bacteria. As with other antibacterial drugs, use of TYGACIL may result in overgrowth of non-susceptible organisms, including fungi
- The most common adverse reactions (incidence >5%) are nausea, vomiting, diarrhea, abdominal pain, headache, and increased SGPT
- Prothrombin time or other suitable anticoagulant test should be monitored if TYGACIL is administered with warfarin
- Concurrent use of antibacterial drugs with oral contraceptives may render oral contraceptives less effective
- The safety and effectiveness of TYGACIL in patients below age 18 and lactating women have not been established

Please see brief summary of Prescribing Information on adjacent page.


Expanded broad-spectrum coverage is on your side