Large-Bowel Obstruction in the Adult: Classic Radiographic and CT Findings, Etiology, and Mimics[1]

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Large-bowel obstruction is an abdominal emergency with high morbidity and mortality rates if left untreated. Although abdominal radiography is usually the initial imaging study performed in patients suspected of having large-bowel obstruction, it may not be sufficient to distinguish obstruction from other causes of colonic dilatation. Computed tomography is the imaging method of choice as it can establish the diagnosis and cause of large-bowel obstruction. A contrast agent enema may be used to confirm or exclude large-bowel obstruction. In this review, the imaging findings in multiple causes of large-bowel obstruction are illustrated and compared with acute colonic pseudo-obstruction.

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Acute complete large-bowel obstruction (LBO) is an abdominal emergency, with high morbidity and mortality rates if left untreated (1,2). While LBO may develop over a protracted period of time, the clinical presentation is often acute and includes abdominal pain, constipation or obstipation, and abdominal distension (3). The marked distension of colon proximal to the level of obstruction leads to mucosal edema, bowel ischemia, and, if not treated, bowel infarction and perforation. While the same principles of initial management of small-bowel obstruction (SBO) (attention to strangulation, hydration, and nasogastric suction) are used in LBOs, emergency surgery or colonoscopy is usually required to relieve the obstruction (4).

LBO is four to five times less frequent than SBO and the causes of LBO and SBO differ substantially (5) (Table 1). Colonic malignancy remains the most common cause of LBO (> 60%) (4,6). Additional causes of LBO include entities such as diverticulitis, colonic volvulus, and adhesions. Colonic obstruction is most often seen in elderly individuals, as the aforementioned causes of obstruction are more common in advanced age groups. Of note, the etiology of LBO worldwide varies substantially as does the patient population affected; in Africa and India, volvulus is the primary cause of LBO (50%), and patients in these areas are usually young and healthy (7).

Essentials

- Large-bowel obstruction (LBO) differs substantially from small-bowel obstruction (SBO), and LBO is an abdominal emergency.
- Abdominal radiography can differentiate LBO from SBO.
- CT has become the standard imaging procedure for patients with both SBO and LBO.
- CT is highly accurate and well tolerated and allows acquisition of images in all patients; CT also helps distinguish the cause of LBO and its complications.

The purpose of this review is to familiarize radiologists and radiology residents with basic knowledge of the imaging findings diagnostic of LBO and to review the complications that require emergent surgical and endoscopic intervention. This review will focus on the most widely used imaging methods for the evaluation of LBO: radiography, contrast agent enema, and multidetector computed tomography (CT). The final portion of this review describes the major mimic of LBO, acute colonic pseudo-obstruction (ACPO).

Clinical Findings and Pathophysiology

An LBO occurs when there is occlusion of the lumen of the colon anywhere along its course and dilatation of the large bowel proximal to the site of obstruction. Both the clinical findings and the pathophysiology of LBO differ substantially from SBO. Patients with LBO are usually elderly and the signs and symptoms of LBO are often insidious in contrast to the abrupt onset of symptoms seen in most SBOs; these symptoms include abdominal pain, constipation or obstipation, and abdominal distension (3,5). The major sites of obstruction include the cecum, hepatic and splenic flexures, and recto-sigmoid colon. LBO occurs more frequently within the left colon (5).

The etiology of the LBO may be suggested by the specific symptoms and presentation of the patient. LBO caused by obstruction in the left colon manifests earlier than that caused by obstruction in the right colon because the lumen of the sigmoid and descending colon is smaller and the stool is more inspissated in the distal colon (3). Obstruction from sigmoid diverticulitis may manifest with symptoms of left lower quadrant pain, fever, and a palpable mass. Colonic volvulus, especially in the setting of chronically distended colon, may include symptoms of chronic abdominal distension and abdominal pain. At the time of the acute volvulus, these patients rapidly develop acute pain and distension. Bowel sounds are usually hypoactive in patients with LBO; this is caused by the cessation of peristalsis (8). In the setting of vascular compromise and ischemia, patients often demonstrate substantial abdominal tenderness.

The competence of the ileocecal valve influences the response of the colon. If the ileocecal valve is competent, which occurs in about 75% of patients, an LBO will result in a closed -loop obstruction, which cannot decompress into the small bowel (4). According to the La Place law, the intraluminal pressure needed to stretch the wall of a hollow tube is inversely proportional to the radius of the tube. Because the cecum is the largest diameter of the colon, it requires the least amount of pressure to distend (9,10). Cecal distension will lead to increased wall tension and without intervention, will progress to ischemia and necrosis. The exact size of the cecum at risk for perforation ranges in the literature from 9 to 12 cm (5). In intermittent or chronic obstruction, however, the cecal wall may become hypertrophied and the colon may greatly exceed 10 cm in diameter without perforation (11). It is important to note that the exact size of the cecum is less important than the duration and rapidity of cecal distension (12–13). An incompetent ileocecal valve will decompress the LBO into the small bowel. The resultant small-bowel distension may mimic a distal SBO.

Abdominal Radiography Technique

Abdominal radiography is usually the first imaging study performed in patients suspected of having LBO (4,5,14). The examination should include supine and nondependent (either upright or left lateral decubitus) radiographs to aid in the diagnosis of LBO and exclude an SBO and to detect pneumoperitoneum.

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Abbreviations:
ACPO = acute pseudo-obstruction
LBO = large-bowel obstruction
SBO = small-bowel obstruction

Conflicts of interest are listed at the end of this article.

Table 1

Causes of LBO

Cause	Specific Signs
Common (>95%)	Neoplasm (primary colon carcinoma) (60%–80%)
	Volvulus (11%–15%)
	Sigmoid
	Cecum
	Transverse colon
	Diverticulitis (4%–10%)
Uncommon (<5%)	Intussusception
	Hernia
	Inflammatory bowel disease
	Extrinsic compression from abscess or other masses
	Fecal impaction
	Intraluminal foreign body

Source.—References 16 and 86.

Figure 1

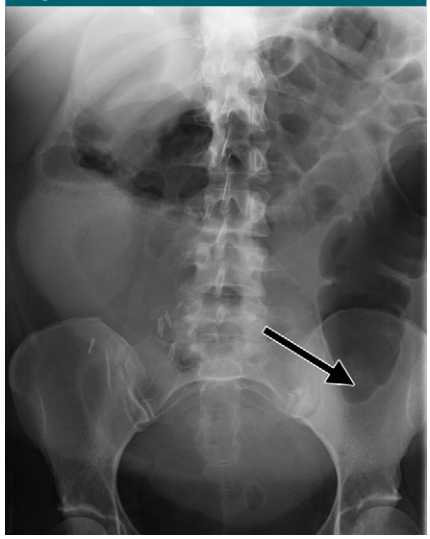


Figure 1: Anteroposterior supine abdominal radiograph in a 67-year-old man with LBO shows dilated ascending, transverse, and descending colon. A transition point is identified in the region of splenic flexure from an obstructing colon carcinoma (arrow).

Figure 2

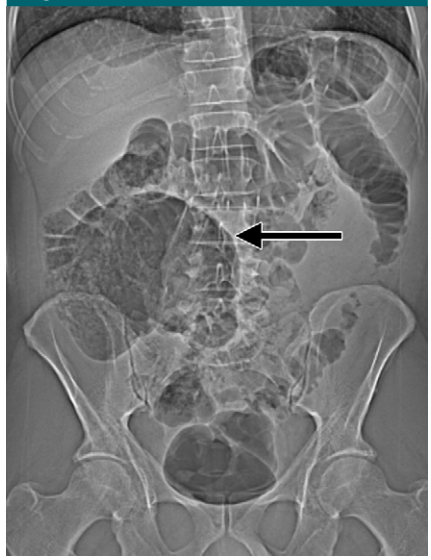


Figure 2: CT scout radiograph in a 51-year-old woman with chronic abdominal pain and cecal ileus shows a distended and medially displaced cecum (arrow). Gas is present throughout the entire colon. CT showed no colonic obstruction.

Merits of Abdominal Radiography

While the reported sensitivity of abdominal radiography for the detection of LBO is similar to that for the detection of SBO (84% vs 82%, respectively), the reported specificity is considerably different (72% vs 83%, respectively) and as a result, it may be difficult to distinguish between obstruction and colonic pseudo-obstruction in a patient with a distended colon (15,16). Normal colonic caliber ranges from 3 to 8 cm, with the largest diameter in the cecum; the remainder of the colon is dilated when it is greater than 6 cm and the cecum is not larger than 9 cm in diameter. In the setting of LBO, the colon is dilated proximal to the site of obstruction with a paucity or absence of gas distal to the obstruction (Fig 1). Air-fluid levels are often seen in the dilated colon on the upright or decubitus radiographs (5). The presence of air-fluid levels suggest that the cause of obstruction is more acute since the colonic fluid has not been present long enough to be absorbed.

Abdominal radiography that includes an upright or decubitus radiograph can also be used to identify complications of LBO, such as pneumatosis, portal venous gas, and pneumoperitoneum. It is important to note that although intramural gas is recognized as a sign of necrosis and developing perforation, the presence of pneumatosis in the setting of LBO does not always indicate transmural infarction but should be considered a worrisome finding for threatened necrosis (12,17). Pseudopneumatosis intestinalis, the appearance of gas trapped within feces or against the mucosal surface, may mimic pneumatosis and is commonly seen in the cecum and the ascending colon (18), CT is helpful in distinguishing between these two diagnoses.

Challenges of Abdominal Radiography in Patients with LBO

One of the challenges facing radiologists and clinicians is determining the cause of a diffusely dilated colon (≥ 6 cm) on abdominal radiographs. Pseudoobstruction, dilatation of the colon without mechanical obstruction, can occur as a result of adynamic ileus, ACPO (also known as Ogilvie syndrome), or toxic megacolon. Adynamic ileus can be characterized by diffuse small- and large-bowel dilatation without a transition point. Common causes of adynamic ileus include recent gastrointestinal surgery, recent opiate use, critical illness, neurologic disorders, and metabolic disturbances (19). ACPO is described as an acute dilatation of the colon due to altered autonomic innervation of the colon. Unlike in an adynamic ileus, perforation may occur with ACPO. Both entities are characterized by colonic dilatation with preserved haustration, smooth inner wall contour, and normal colonic wall thickness. Adynamic ileus is routinely characterized by small-bowel dilatation as well (19). Colonic distension due to these entities usually occurs with minimal fluid; the presence of air-fluid levels should raise the suspicion of an obstruction (19,20). Toxic megacolon, a complication of a variety of infectious, ischemic, and inflammatory diseases of the colon, is characterized by its hallmark feature of marked bowel wall thickening, loss of haustration, and segmental parietal wall thinning (11,21).

The presence of cecal distension may be seen in LBO, colonic ileus, ACPO, and toxic megacolon. Johnson et al (22) described the phenomenon known as cecal ileus as the clinical condition that occurs when patients with a mobile ce-

cum develop an adynamic ileus with displacement of the cecum anteromedially (Fig 2). Relative cecal size may be useful in determining if a large bowel is present; as Wittenberg (9) notes, if the colon is diffusely distended and the cecal diameter is clearly less than that of the other colonic segments LBO is unlikely. It may be difficult to distinguish between a low colonic obstruction and a colonic ileus as the absence of distal rectal gas is seen in both entities. The converse is also true as small amounts of distal rectal gas may be present in the setting of LBO as well as ileus. The presence of rectal gas should not exclude the diagnosis of LBO, but frank distention of the rectum implies a colonic ileus. Acquisition of a prone or right lateral decubitus radiograph may also be helpful in ruling out LBO: Gas will be restricted in a bowel obstruction but will move to the distal colon and decompress in the setting of pseudo-obstruction (Fig 3). Despite these dissimilarities in presentation, the differentiation between LBO and pseudo-obstruction remains difficult and CT should be used to better distinguish between the two diagnoses.

Multidetector CT

CT is the imaging modality of choice for the diagnosis of the cause of LBO. Multidetector CT is a well-tolerated, rapid imaging examination that allows acquisition of images in one breath hold in the frail without the need for the use of rectal contrast agent or air insufflation. Thin sections and multiplanar reformatting provide accurate delineation of large-bowel morphology. CT can be used to diagnose intraluminal, mural, and extramural causes of LBO. In patients with LBO secondary to malignancy, CT offers the additional benefit of detecting local and regional metastases. CT is also an excellent imaging modality for the detection of inflammation and bowel ischemia. The detection of LBO with CT has been reported to have a sensitivity and specificity of 96% and 93%, respectively (3,23–25). The diagnosis of LBO is based on dilated large bowel proximal to a transition point and decompressed bowel distal to the obstruction. The presence of a transition point is considered a reliable finding for the diagnosis of LBO (3,24).

CT Technique

A reasonable scanning protocol for a routine abdominal and pelvic CT with a 64-detector scanner would include the following acquisition parameters: helical mode, 120 kVp; beam pitch, 0.8–1.375; automated tube current modulation with minimum tube current, 100–150 mAs; reconstruction section thickness, 5 mm. If possible, the administration of intravenous contrast agent is recommended as it adds to the identification of the presence of a mass, as well as signs of inflammation and bowel wall ischemia. Iodinated intravenous contrast agent can be given with a weight-based protocol or in a routine volume (eg, 150 mL) and a rate of 3 mL/sec with a delay of 70 seconds, which is sufficient for portal venous imaging in most patients. Oral contrast agent administration is controversial in the setting of acute abdominal pain, and its use is quite variable (26–29).

Coronal and multiplanar reformations aid in the identification of the course of the distended bowel and the exact location of obstruction. If confusion about the diagnosis of LBO persists, water-soluble rectal contrast agent can be administered to better document obstruction.

Pitfalls of CT Imaging of LBO

Spasm at the splenic flexure in a normal colon may mimic a fixed narrowing (25). It is important to note that the transitional region in pseudo-obstruction tends to be at or near the splenic flexure (30). Dilatation of the ascending and transverse colon with distal collapse can be seen in both ACPO and chronic colonic pseudo-obstruction. Additionally, the "colon cut-off" sign, an isolated gaseous distension of the ascending colon and hepatic flexure in the setting of pancreatitis, can also mimic an LBO (31). Finally, as Beattie et al (25) note, there is also potential for missing short annular desmoplastic colonic lesions on CT scans, particularly if there is partial luminal obstruction with limited distension of the proximal colon to delineate the lesion. This pitfall is more common in right-sided colonic tumors (19).

Figure 3

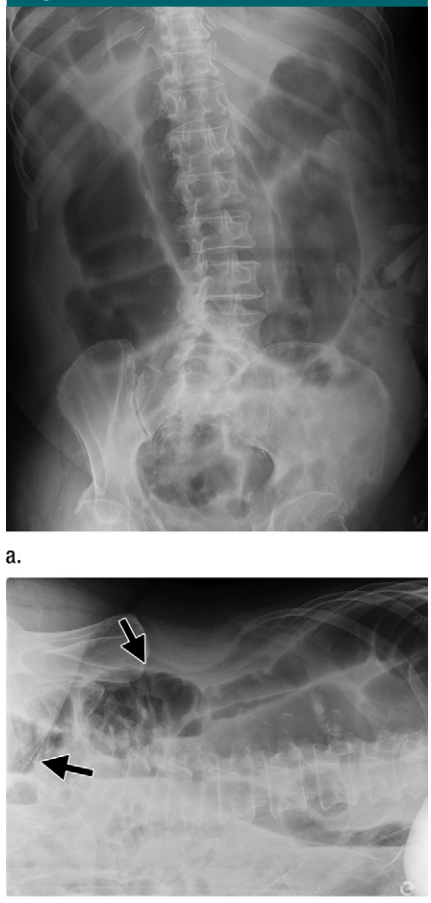
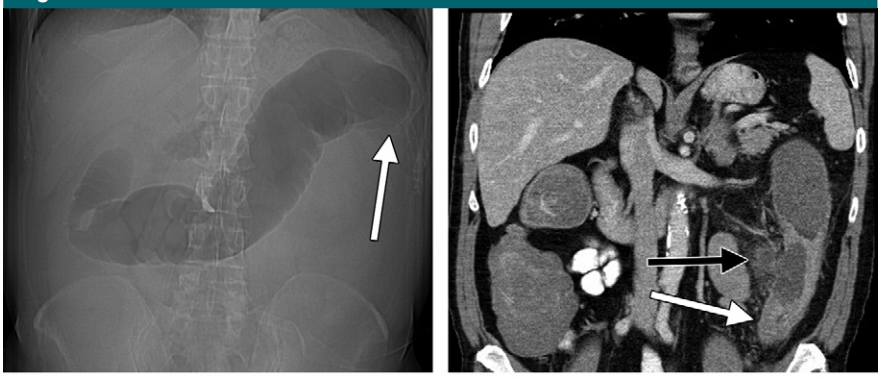


Figure 3: Images in a 71-year-old man in a persistent vegetative state after a pontine hemorrhage. **(a)** Anteroposterior supine abdominal radiograph shows marked dilatation of the entire colon. **(b)** Right lateral decubitus radiograph shows gas filling the entire colon down into the sigmoid colon and rectum (arrows).

The Contrast Enema

Although CT has become the preferred imaging study for evaluation of LBO, there are some indications for performing a contrast enema. The major advantage of the contrast en-

Figure 4



a. b.

Figure 4: Images in a 76-year-old man with LBO from a descending colon cancer. **(a)** CT scout image shows air-filled dilated colon terminating in the left upper quadrant (arrow). **(b)** Midline coronal reformatted CT image of the abdomen and pelvis after administration of intravenous contrast material shows obstructing left colonic adenocarcinoma (white arrow) with adjacent perforation and abscess (black arrow).

ema is that it usually allows easy distinction between a LBO and colon pseudo-obstruction (4,5,32). It may also be used to confirm a colonic volvulus (5). The goal of the examination is to fill the colon adequately enough to detect the obstruction or demonstrate dilated colon without a transition point. Water-soluble iodinated contrast material should be used as it is easily absorbed in the peritoneum should there be a perforation (32). Additionally, if the enema is performed first, water-soluble contrast material does not cause an artifact on CT scans. The study should be performed under low pressure without inflation of the balloon.

To completely evaluate the colon, the patient must be able to rotate on the fluoroscopy table. This is particularly true for the sigmoid colon, which can be very redundant in the elderly patient. For these reasons, the examination may be an insufficient diagnostic tool in the large, elderly, immobile, or uncooperative patient.

LBO: Major Causes

The following section provides an overview of the clinical and radiographic features of the various causes of LBO and a discussion of how to differentiate LBO from ACPO and chronic colonic pseudo-obstruction. The entities are presented in order of frequency.

Colon Carcinoma

Colon carcinoma is the most common cause of LBO (> 60% of cases), and mortality is high (10%–30%) in patients requiring emergency surgery (3,17,33–35). The two most frequent locations of obstruction due to colonic malignancy are the sigmoid colon and the splenic flexure (33). The most common site of perforation in LBO is not at the site of the tumor but at the cecum, with a reported incidence of perforation of 3%–8% (36). The clinical manifestation of LBO from a colon malignancy depends on a number of factors, including location of the tumor and competency of the ileocecal valve. Right-sided tumors with an incompetent ileocecal valve can mimic SBO. Left-sided malignancies cause diffuse distension of the colon up to the level of obstruction.

CT findings include asymmetric and short-segment colonic wall thickening or an enhancing soft-tissue mass centered in the colon that narrows the colonic lumen with or without findings of ischemia and perforation (Fig 4). Obstructing colon cancers often produce a shouldering appearance and may be large enough to have central necrosis or rarely air within the mass, the latter appearance may resemble an abscess (37). Recognition of proximal colonic dilatation aids in identification of the transition point at the site of tumor. Colonic malignancy may mimic diverticulitis if there is pericolonic spread with infiltration of the pericolonic fat. The identification of pericolonic lymph nodes larger than 1 cm in short axis should raise the suspicion of malignancy (38). It should be noted that not all enlarged pericolic lymph nodes contain tumor, and normal-sized nodes may have microscopic tumor involvement (37). Nodal metastases can be located in expected regional drainage routes. Care should be made to review the entire colon for synchronous lesions, which occur in 2%–7% of patients (39).

Volvulus

Acute colonic volvulus accounts for approximately 10%–15% of LBO (3). Volvulus is defined as a twisting of the intestine upon itself that causes obstruction. If the twist is greater than 360°, the volvulus is unlikely to resolve without intervention. The symptoms of obstruction, severe abdominal pain and distension, are due to the narrowing produced at the site of torsion. Vascular compromise at the site of volvulus leads to ischemia, necrosis, and perforation. Sigmoid volvulus is three to four times more common than cecal volvulus (60%–75% vs 25%–33%, respectively), and volvulus of the transverse colon and splenic flexure is very rare (< 1%) (4,5). A major predisposing factor leading to a colonic volvulus is a mobile redundant colon on a mesentery and a fixed point about which the colon can twist. Sigmoid volvulus commonly occurs in the elderly, who have an elongated and chronically dilated sigmoid colon. The more proximal colon volvuli occur due to a congenital defect in the cecum or transverse colon mesentery, which makes these segments of the colon more mobile and prone to twisting (40). Patients with a large-bowel volvulus causing obstruction present with acute abdominal pain and abdominal distension.

Figure 5

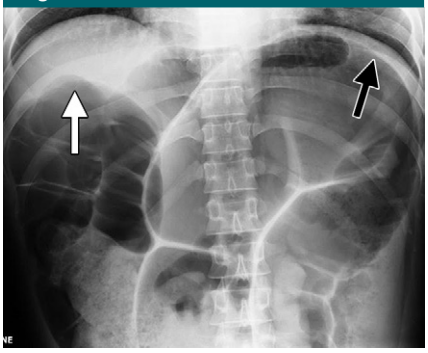
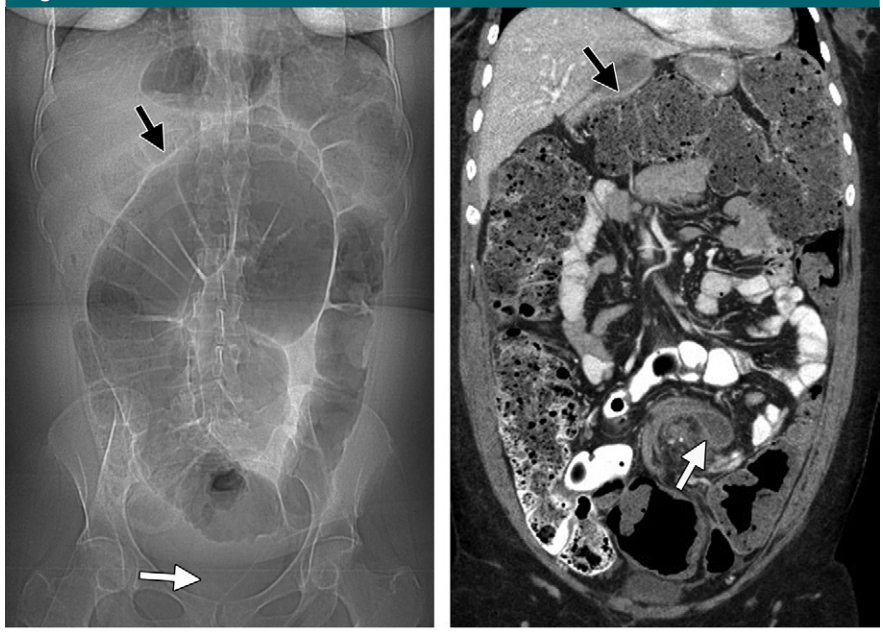


Figure 5: Anteroposterior supine abdominal radiograph in a 58-year-old man with sigmoid volvulus and "northern exposure" sign shows markedly dilated sigmoid colon (black arrow) extending above the transverse colon (white arrow).

Figure 6



a. **b.**

Figure 6: Images in a 72-year-old woman with LBO caused by sigmoid volvulus. **(a)** CT scout image shows dilated, air-filled colon terminating in markedly dilated sigmoid colon folded upon itself with its apex (the "coffee bean sign") in the midline upper abdomen (black arrow). The sigmoid also conforms to an "upside down U" configuration. There is no gas in the rectum (white arrow). **(b)** Midline coronal reformatted CT image of the abdomen and pelvis shows dilated, stool-filled colon proximal to the volvulus (black arrow) with a distal "whirl" of the mesentery at the point of volvulus (white arrow).

Sigmoid Volvulus

Sigmoid volvulus is the abnormal twisting of the sigmoid colon along the mesenteric axis, which leads to a closed-loop obstruction. The diagnosis of sigmoid volvulus is evident on abdominal radiographs in 57%–90% of cases (40–42). There are several classic signs describing the findings of colonic volvulus; these include the coffee bean and bird beak signs. The coffee bean sign describes the appearance of the volvulus, with apposition of the medial walls of the dilated loop of bowel forming the cleft of the bean and the lateral walls forming the outer walls of the bean; it can be seen in both sigmoid and cecal volvulus (43–45). The bird beak sign, seen in all colonic volvuli, describes the smooth, tapering transition point of the obstruction. The inverted U sign, an inverted ahaustral dilated sigmoid in the shape of an inverted "U" extending into the right upper quadrant, is specific to sigmoid volvulus and is seen in 25%–78% of patients (43,46) The northern exposure sign, also specific to sigmoid volvulus, describes the repositioning of the dilated sigmoid colon out of the pelvis to extend above the transverse colon (Fig 5); among a series of 30 cases of sigmoid volvulus, Javors et al (41) found this sign in 26 (87%) of the cases. It is by far the most specific sign described related to sigmoid volvulus. Because sigmoid volvulus can be a closed-loop obstruction, there may be a substantial amount of gas in the more proximal colon and the small bowel. Absence of rectal gas is a common finding in sigmoid volvulus.

CT is extremely helpful in the diagnosis of sigmoid volvulus. Levsky et al (46) reviewed the classic signs of sigmoid volvulus on CT scans and found that the most sensitive signs on the CT scanogram were the absence of rectal gas (90%) and the U sign (86%), while the most sensitive findings on cross-sectional images were a single transition point in the sigmoid (95%) and disproportionate enlargement of the sigmoid (86%). The coffee bean, kidney bean, and bent inner tube signs, all descriptors of the appearance of air-filled closed loop of colon, can all be seen in the setting of sigmoid volvulus (Fig 6). A "beak" can be found at the point of twisting of the sigmoid colon and if necessary, may be confirmed with colonic contrast material. The whirl sign, the appearance of spiraled loops of collapsed bowel with enhancing engorged vessels radiating from the twisted bowel, is often evident at the point of obstruction (Fig 6b) (47–50). Macari et al (50) found that the location of the whirl was highly accurate in discriminating cecal from sigmoid volvulus. CT is also used to exclude findings of ischemia and necrosis of the effected sigmoid.

The water-soluble enema is a helpful diagnostic tool in the confirmation of sigmoid volvulus. The examination is performed under low pressure, without insufflation of the balloon (5). The classic beak sign is usually encountered at the site of torsion, and contrast material may not pass proximal to the transition point (Fig 7). In some cases, however, the sigmoid volvulus does not produce a complete obstruction and contrast material may pass proximal to the beak, indicating a partial LBO. In these cases, the right

Figure 7

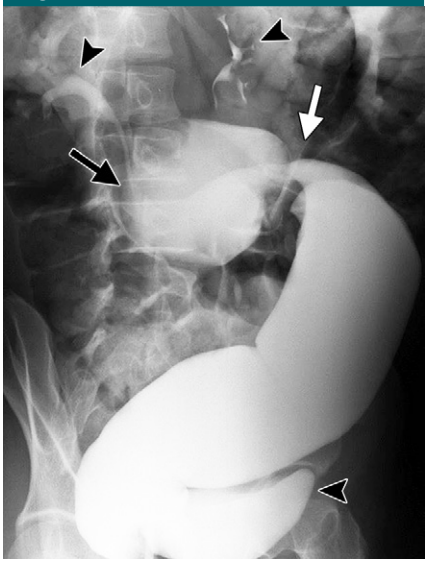
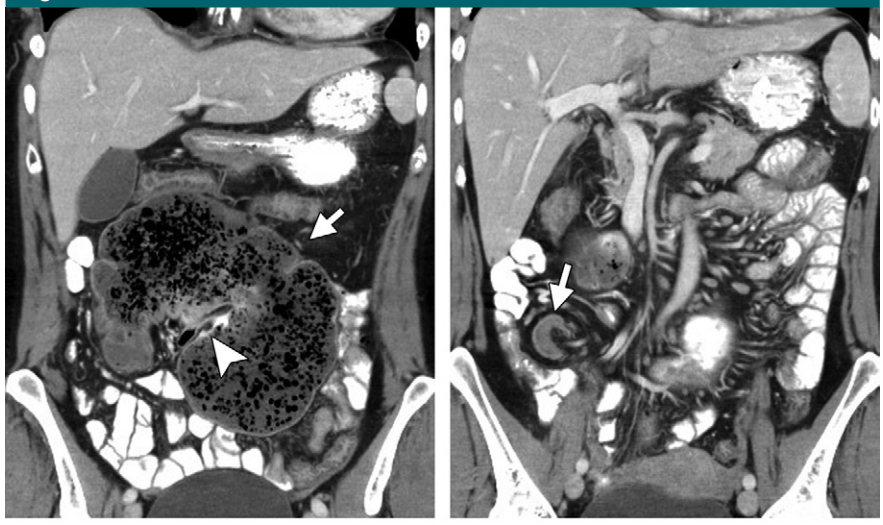


Figure 7: Anteroposterior supine abdominal radiograph after administration of water-soluble enema in a 64-year-old man with sigmoid volvulus shows a "beak" sign at the site of torsion (white arrow). Some contrast material is noted to pass above the level of obstruction (black arrow). Residual CT contrast material is seen in the renal collecting systems and bladder (arrowheads).

Figure 8



a. **b.**

Figure 8: Coronal reformatted CT images of the abdomen and pelvis in an 81-year-old woman with LBO caused by cecal volvulus. **(a)** Image shows displaced cecum in the mid abdomen, with its apex located in the left upper quadrant (arrow). The ileocecal valve is displaced toward the left upper quadrant as well (arrowhead). **(b)** Image after administration of intravenous contrast material demonstrates the "whirl" sign (arrow), confirming the cecal volvulus originating in the right lower quadrant (arrow).

colon and cecum are usually less dilated than the more distal colon.

Cecal Volvulus

Cecal volvulus is characterized by twisting of the cecum causing a proximal LBO. This phenomenon occurs when the right colon is not fused to the posterior abdominal wall (5,51). Pregnancy and recent colonoscopy, factors that result in dilatation of the right colon, predispose patients to cecal volvulus (52). In half of patients with cecal volvulus, the cecum twists in the axial plane, rotating along its long axis, appearing in the right lower quadrant. The other half of patients has a "loop" type of cecal volvulus, with the cecum twisting and inverting, resulting in the apex of the cecal twist in the left upper quadrant. The terminal ileum usually twists with the cecum. Identification of the displaced, gas-filled appendix confirms the diagnosis (34).

The diagnosis of cecal volvulus can be made in 75% of cases from the abdominal radiograph alone (5). The cecum rotates out of the right lower quadrant into the left upper quadrant and occasionally into the left lower abdomen or mid line. There is often substantial cecal distension (> 9 cm), with little distal colonic gas. An incompetent ileocecal valve causes dilatation of distal small bowel. The key to diagnosis with abdominal radiography is the recognition of displacement of the cecum out of the right lower quadrant. If a contrast enema is performed, a classic beak sign will be demonstrated in the displaced ascending colon (5,52). It is important to recognize findings of ischemia in the cecum, which include pneumatosis in the cecal wall, pneumoperitoneum, and/or portal venous gas.

CT findings of cecal volvulus include marked distension of the cecum in an abnormal location, usually in the mid or left upper abdomen. The ileocecal valve is also displaced into the left upper quadrant. Coronal reformations confirm the abnormal location of the cecum (Fig 8). The two limbs of the looped obstructed bowel taper and meet at the site of the twist, forming an appearance that resembles a bird's beak. The whirl sign can be found at the site of the twist. The tightness of the twist is proportional to the degree of rotation. Given the proximal location of this LBO, small-bowel dilatation may also be an associated finding (34). CT findings of ischemia associated with cecal volvulus include wall thickening, mural hypoenhancement, and pneumatosis. Mesenteric stranding and peritoneal fluid aid in the diagnosis of bowel wall ischemia.

A variant of cecal volvulus, the cecal bascule, occurs when the cecum folds anteriorly on itself without twisting (52,53). It appears as a dilated loop in the midabdomen. Johnson et al (22) challenged this concept and felt most of these cases were due to focal ileus in an anteriorly displaced cecum. It is important to note that a distended cecum, 9 cm or greater, is at risk for perforation.

Transverse Colon Volvulus

The transverse colon volvulus is very uncommon, accounting for between 1%–4% of all colonic volvulus (5,54,55). It occurs in patients with a redundant transverse colon on a long mesentery; failure of fixation of the mesentery may

lead to mobility of the ascending colon and hepatic flexure, leaving these patients predisposed to transverse colon volvulus. Because the diagnosis may not be established early, and the twisting may occur at the root of the mesentery, the mortality rate in these patients has been reported to be 33% (46). A contrast enema can confirm the diagnosis by demonstrating the classic beak at the point of obstruction in the transverse colon. Findings at CT include LBO proximal to the twist in the mesentery. The right colon and cecum are midline or displaced to the left.

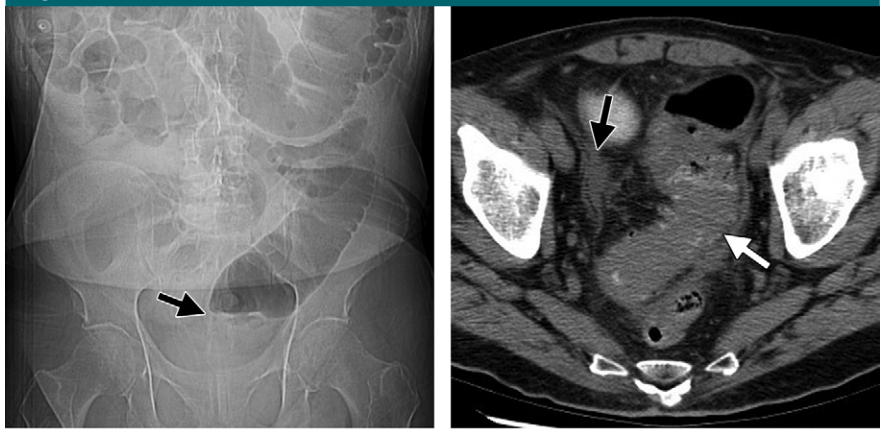
By far the least common site for reported colonic volvulus is the splenic flexure (56). Causes include postoperative adhesions, abnormal peritoneal attachments, and chronic constipation. A CT or contrast enema is usually needed to establish the diagnosis. Findings will include marked distension of the distal transverse colon, with a whirl sign in the region of the splenic flexure (57,58).

Diverticulitis

Although less common (10% of all cases of LBO), patients with acute diverticulitis can present with LBO due to bowel wall edema and pericolonic inflammation (3). High-grade obstruction is less common in the setting of diverticulitis; more commonly, obstruction occurs in the setting of multiple episodes of diverticulitis, which causes narrowing and stricture formation (5). Chronic diverticulitis can produce both LBO and a chronically dilated colon. While the most common location for obstructing diverticulitis is the sigmoid, LBO caused by diverticulitis may occur at any location in the colon and is not uncommon in the right colon in Asian countries (59).

Patients with sigmoid diverticulitis usually present with left lower quadrant pain, fever, a palpable left lower quadrant mass, and constipation. If there is accompanying LBO, they will also have abdominal distension. These symptoms may mimic a colon carcinoma–producing LBO. Large LBO due to right colon or cecal diverticulitis may mimic a distal SBO, with dilatation of the small bowel upstream of the inflammation.

Figure 9



a. b.

Figure 9: Images in a 47-year-old man with LBO caused by diverticulitis. **(a)** CT scout image shows air-filled dilated colon terminating in the left pelvis (arrow) **(b)** Transverse CT image of the pelvis after the administration of intravenous contrast material shows dilated, stool-filled large bowel extending into the pelvis where the sigmoid colon is thick walled and inflamed (white arrow). There is fluid in the root of the mesentery (black arrow).

Figure 10

a. b.

Figure 10: Images in a 64-year-old man with LBO caused by a colocolonic intussusception. **(a)** CT scout image shows air-filled dilated colon terminating abruptly in the left upper quadrant (arrow). **(b)** Coronal reformatted CT image of the abdomen and pelvis shows a transverse colonic intussusception (arrow). The lead point for the obstruction was a tubulo-villous adenoma.

Diverticulitis on CT scans is characterized by segmental, symmetric bowel wall thickening with hyperemia, which is typically in a longer segment (≥10 cm) than malignant lesions (Fig 9) (38,60). Pericolonic inflammation and fat stranding are hallmarks of diverticulitis. If the inflammation is extreme, intramural and extramural abscesses, as well as perforation with pneumoperitoneum, may be seen. Fluid in the root of the mesentery and vascular engorgement favor the diagnosis of diverticulitis (61). In contrast, a short (< 10 cm) segment of colonic wall thickening and the presence of lymph nodes raise the suggestion of a colonic malignancy (38,60). In some cases, it is impossible to distinguish between diverticulitis and a colonic malignancy without colonoscopy with biopsy. Although both the American Society of

Figure 11

Figure 11: Transverse CT image of the pelvis in an 85-year-old woman with LBO caused by distal fecal impaction. Image obtained after administration of intravenous contrast material and displayed by using lung windows shows a dilated colon and large mass of impacted stool in the rectum (arrow). Lung windows aid in the delineation of air-containing structures.

Figure 12

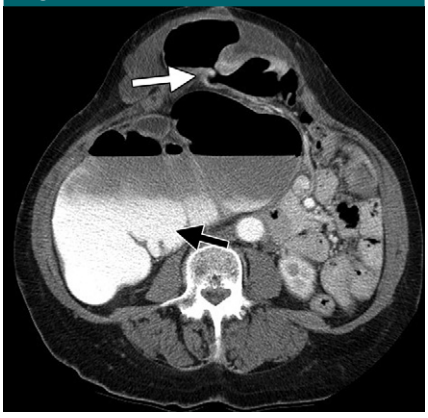


Figure 12: Anterior transverse CT image of the abdomen and pelvis in a 67-year-old man with LBO caused by a colon-containing ventral hernia. Image obtained after administration of oral and intravenous contrast material shows dilated, fluid-filled cecum (black arrow) and a portion of colon obstructed in a ventral hernia (white arrow).

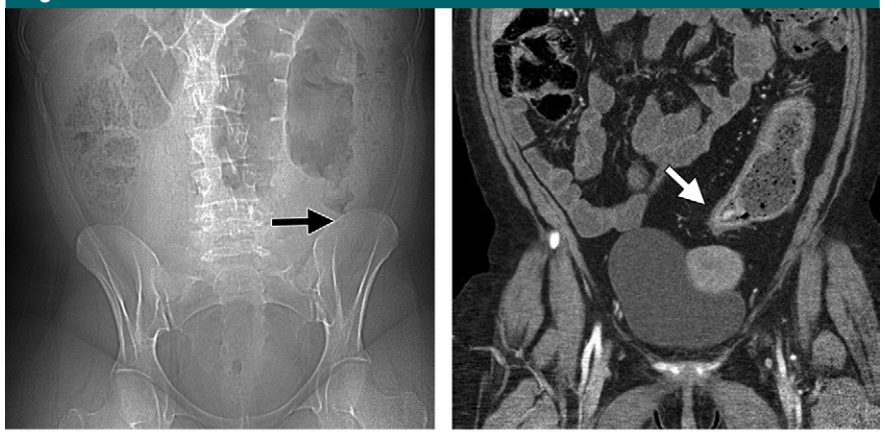
Colon and Rectal Surgeons and the American College of Gastroenterology routinely recommend that patients undergo colonoscopy to exclude colon cancer after an episode of acute diverticulitis, there are limited data to support this recommendation (62).

Adult Intussusception

Intussusception accounts for only a small number (< 1%–2%) of adult LBO cases. Demonstration of a lead point is found in more than 80% of adults (5). The most common cause of a colocolonic intussusception is a primary colon carcinoma (63). In addition, there are a number of benign lesions that can serve as lead points in colonic intussusception, the most common being adenomatous polyps and lipomas (64). Many other lesions have been reported to cause intussusception, including gastrointestinal stromal tumors, as well as a variety of appendiceal lesions, including the inverted appendiceal stump, endometriosis involving the appendix, and benign masses such as a mucocele (65,66). Other reported causes of LBO due to intussusception include eosinophilic colitis, pseudomembraneous colitis, and epiploic appendagitis (63,67–69).

Abdominal radiographs may show only evidence of bowel obstruction, and if the lesion is in the right colon, the findings may mimic a SBO. A contrast enema can identify the obstructing colonic mass and the classic "coil spring" appearance as the contrast material is trapped between the intussusceptum and intussuscipiens (70). However, with signs and symptoms of a LBO, most patients will undergo an abdominal CT.

Figure 13



a. b.

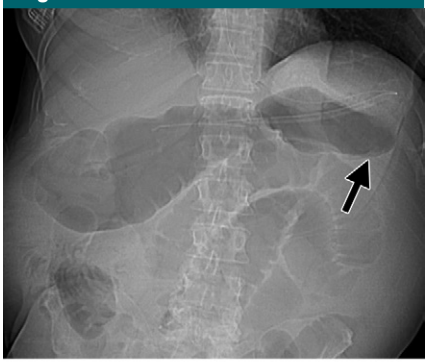
Figure 13: Images in a 59-year-old man with LBO caused by Crohn colitis involving the distal descending colon. **(a)** CT scout image demonstrates substantial colonic distension with stool. Arrow marks the site of obstruction. **(b)** Midline coronal reformatted CT image shows wall thickening and hyperenhancement of the mucosa of the descending colon with a distal stricture from Crohn colitis (arrow).

The CT findings of an ileocolic or colocolic intussusception include distended colon (the intussuscipiens) with a thickened wall, an intraluminal intussusceptum telescoping within the intussuscipiens, and a curvilinear area of fat representing the invaginated mesenteric fat of the intussusceptum (Fig 10). Invaginated vessels may also be seen accompanying the intussusceptum. The bowel has the appearance of a "target" in cross-section or sausage-shaped mass if in the longitudinal plane (63).

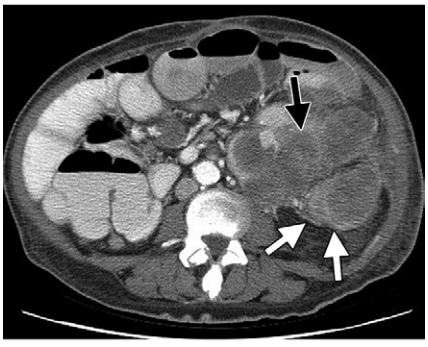
Intraluminal Contents Causing LBO

The most likely sites of colonic obstruction from intraluminal contents are the rectum (70%) and sigmoid colon (20%) (4,5). There are many reported causes of intraluminal contents resulting in colonic obstruction, including gallstones, enteroliths, intentionally inserted foreign body, medications, and illegal drugs. The most common cause is fecal impaction, a clinical entity occurring primarily in the elderly, chronically debilitated patients, and in those taking certain medications (3,71). Abdominal radiographs will demonstrate colonic obstruction, with a large amount of stool distal to the obstruction (Fig 11). CT findings include a large amount of stool located distal to the dilated colon.

Figure 14



a.



b.

Figure 14: Images in a 55-year-old man with LBO caused by a metastasis from adenocarcinoma of the lung. **(a)** CT scout image shows dilated, air-filled colon and small bowel terminating in the left upper abdomen (arrow). **(b)** Transverse CT image of the abdomen and pelvis after intravenous injection of contrast material shows large necrotic metastasis from lung adenocarcinoma (black arrow) in the left abdomen compressing and deviating the descending colon posteriorly (white arrows). Both upstream small bowel and large bowel are dilated.

Table 2

Causes and Associations in ACPO

Cause	Association
Surgical	
Inflammatory	Abscess, appendicitis, cholecystitis, pancreatitis
Trauma	Fractures and orthopedic procedures, burns
Urologic	Renal tumor ablation, calculi
Obstetric	Normal pregnancy and delivery, cesarean section, hysterectomy, complications of pregnancy
Organ transplantation	…
Other	Emergency laparotomy, craniotomy, thoracotomy
Medical conditions	
Cardiopulmonary	Mechanical ventilation, pneumonia, myocardial infarction, congestive heart failure, chronic obstructive lung disease
Metabolic	Hypokalemia, hyponatremia, hypocalcemia, hypothyroidism, diabetes
Neurologic	Dementia, multiple sclerosis, Parkinson disease
Infectious	All system infections
Oncologic	All malignancies and their treatments
Miscellaneous	Organ failure, alcoholism
Medications	Narcotics, anticholinergics, antiparkinsonian, laxative abuse

Hernias

Although a considerably less common result of a hernia than a SBO, LBO can occur secondary to inguinal, femoral, umbilical, Spigelian, incisional, lumbar, and diaphragmatic hernias (Fig 12) (5). The most common internal hernia to produce an LBO is the foramen of Winslow hernia, the condition in which small bowel and, in one-third of cases, the right colon herniate through the normal communication between the greater and lesser peritoneal cavities, between the free edge of the lesser omentum and the hepatoduodenal ligament (72). Abdominal radiographs will demonstrate findings of LBO. Most of these patients will undergo CT for definitive diagnosis, where colon will be found in a hernia with dilated proximal colon and decompressed distal colon.

Inflammatory Bowel Disease

Between 20% and 50% of patients with Crohn disease will have colonic involvement, and stricture formation of the large bowel occurs in 5%–17% of patients (73,74). It is important to exclude malignancy in these individuals as the risk of colon cancer is two to three times higher in patients with Crohn disease compared with age-matched standard populations (75). Radiographic findings will demonstrate an LBO. Contrast enemas are rarely performed in these patients, and most will undergo CT for diagnosis.

CT findings of colonic Crohn disease include wall thickening, luminal narrowing with prestenotic dilatation, and dilatation of the vasa recta supplying the affected bowel loop (Fig 13). Mural stratification with hyperenhancement of the mucosal wall suggests active inflammation, while homogeneous attenuation of the wall of the colon suggests a more chronic fibrotic stenosis. Other CT findings of colonic Crohn disease include abscesses and fistulae. Stricture formation and obstruction is less common in ulcerative colitis and should raise suspicion of an underlying malignancy.

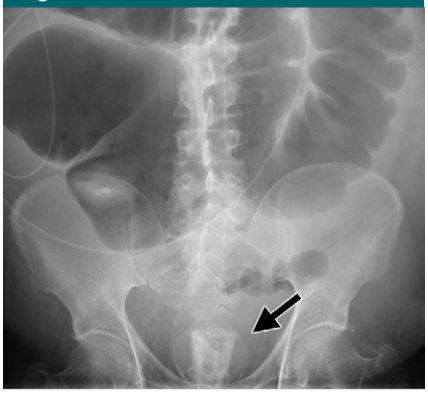
Adhesions

Adhesions are a very rare cause of LBO. Adhesive bands causing LBO have been reported in the right, transverse, and sigmoid colon (5,76,77). Abdominal radiographs show a colonic obstruction, and contrast barium enema will demonstrate a short area of circumferential narrowing with intact mucosa. Similar to findings seen in SBO, CT will demonstrate a colonic obstruction without an obvious cause.

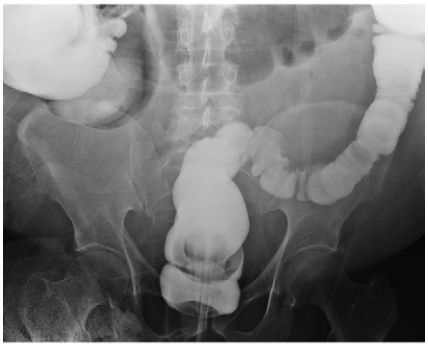
External Compression

The large bowel can rarely become obstructed from external compression. This type of LBO is most commonly caused by adjacent masses. Sources of external compression are extensive and include endometriosis, lymphadenopathy, pancreatitis, intra-abdominal abscesses, mesenteric or colonic surface involvement of peritoneal carcinomatosis, and direct invasion from gynecologic or prostatic malignancies

Figure 15



a.



b.

Figure 15: Anteroposterior supine abdominal radiographs obtained after cardiac surgery in a 55-year-old man with abdominal distension. **(a)** Radiograph shows marked distension of the entire colon despite rectal tube (arrow) in place. **(b)** Radiograph after administration of water-soluble enema demonstrates patent colon without evidence of obstruction. The pseudo-obstruction resolved with colonic decompression tube placement.

(3). Abdominal radiographs will show LBO and occasionally a suggestion of the presence of a mass. A contrast enema will show extrinsic compression producing the LBO. The CT findings include large-bowel dilatation from a soft-tissue mass (Fig 14).

ACPO or Ogilvie Syndrome: An Important Mimic of LBO

ACPO (Ogilvie syndrome) was first described by Ogilvie in 1948 as a pseudo-obstruction secondary to interruption of sympathetic innervation of the colon (78). A number of etiologies have been implicated in causing colonic pseudo-obstruction (Table 2). Although the exact pathophysiology is still unclear, the treatment with neostigmine is based on parasympathetic stimulation. This medication has been reported to show rapid resolution in more than 80% of ACPO patients (79). Treatment with lower endoscopic decompression is also very beneficial (80).

ACPO is most common in male patients over 60 years of age, and most are already hospitalized with a severe illness (81). The symptoms of ACPO mimic those of LBO and include abdominal distension, pain, nausea, and vomiting. While they usually develop over 3 to 7 days, symptoms may occur more quickly. Abdominal tenderness, a common sign in the setting of LBO, is not a prominent feature of ACPO and its presence, especially in the presence of other signs of an acute abdomen, should prompt an immediate work-up to exclude perforation.

Abdominal radiographs in patients with ACPO often demonstrate marked colonic distension predominantly involving the cecum, ascending colon, and transverse colon. Gas may also extend to the sigmoid colon and rectum (Fig 15). Because the cecum is routinely distended in ACPO, cecal ischemia and perforation are a major concern. The risk of spontaneous cecal perforation in ACPO is 3%–15%, with a mortality of 50% (82). While there is no clear relationship between cecal diameter and perforation, duration of cecal distension does correlate with risk of perforation. Prolonged cecal dilatation beyond 2 to 3 days should prompt strong consideration for decompression with colonoscopy or surgery (22,80). The presence of pneumatosis in the cecum and/or ascending colon indicates ischemia of the bowel, and if not treated, the bowel will perforate. Free intraperitoneal air in ACPO suggests a colonic perforation and should prompt immediate surgery (59–61).

Distinguishing between LBO and pseudo-obstruction is a major diagnostic challenge. In patients with diffuse colonic distention in the setting of ACPO, repositioning the patient after an initial supine radiograph of the abdomen and obtaining additional images in a right lateral decubitus and/or prone position after a few minutes usually results in air filling the distal colon. This allows distinction between LBO and pseudo-obstruction (83). Furthermore, patients with a chronic colonic pseudo-obstruction can usually be established with prior abdominal radiographs and a history of chronically dilated large bowel (84).

Ultimately, if indistinguishable at abdominal radiography, the diagnosis may be made with a contrast enema (Fig 15) (22,80). If the differentiation of LBO and ACPO remains problematic, CT may play a role in the diagnosis of ACPO. CT will allow characterization of the entire large bowel and help identify the presence or absence of a transition point (85).

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References

1. Lopez-Kostner F, Hool GR, Lavery IC. Management and causes of acute large-bowel obstruction. Surg Clin North Am 1997;77(6):1265–1290.
2. Sawai RS. Management of colonic obstruction: a review. Clin Colon Rectal Surg 2012;25(4):200–203.
3. Taourel P, Kessler N, Lesnik A, Pujol J, Morcos L, Bruel JM. Helical CT of large bowel obstruction. Abdom Imaging 2003;28(2):267–275.
4. Welch J. Bowel obstruction: differential diagnosis and clinical management. Philadelphia, Pa: Saunders, 1989; 59–95.
5. Gore RM, Levine MS. Textbook of gastrointestinal radiology. 3rd ed. Philadelphia, Pa: Saunders/Elsevier, 2008.
6. Biondo S, Parés D, Frago R, et al. Large bowel obstruction: predictive factors for postoperative mortality. Dis Colon Rectum 2004;47(11):1889–1897.
7. Sule AZ, Ajibade A. Adult large bowel obstruction: a review of clinical experience. Ann Afr Med 2011;10(1):45–50.
8. Balfour DC Jr, Bollman JL, Grindlay JH. Intestinal lymph flow following simple intesti-

nal obstruction in the rat. Surgery 1951; 29(4):500–501.

9. Wittenberg J. The diagnosis of colonic obstruction on plain abdominal radiographs: start with the cecum, leave the rectum to last. AJR Am J Roentgenol 1993;161(2):443–444.
10. Saegesser F, Chapuis G, Rausis C, Tabrizian M, Sandblom P. Intestinal distension and colonic ischemia: occlusive complications and perforations of colo-rectal cancers—a clinical application of Laplace's law [in French]. Chirurgie 1974;100(7):502–516.
11. Krajewski K, Siewert B, Eisenberg RL. Colonic dilation. AJR Am J Roentgenol 2009; 193(5):W363–W372.
12. Kottler RE, Lee GK. The threatened caecum in acute large-bowel obstruction. Br J Radiol 1984;57(683):989–990.
13. Davis L, Lowman RM. An evaluation of cecal size in impending perforation of the cecum. Surg Gynecol Obstet 1956;103(6):711–718.
14. Nadrowski L. Clinical presentation and preoperative management of bowel obstruction. Edinburgh, Scotland: Churchill Livingstone, 1987; 66–77.
15. Thompson WM, Kilani RK, Smith BB, et al. Accuracy of abdominal radiography in acute small-bowel obstruction: does reviewer experience matter? AJR Am J Roentgenol 2007;188(3):W233–W238.
16. Cappell MS, Batke M. Mechanical obstruction of the small bowel and colon. Med Clin North Am 2008;92(3):575–597, viii.
17. Taourel P, Garibaldi F, Arrigoni J, Le Guen V, Lesnik A, Bruel JM. Cecal pneumatosis in patients with obstructive colon cancer: correlation of CT findings with bowel viability. AJR Am J Roentgenol 2004;183(6): 1667–1671.
18. Wang JH, Furlan A, Kaya D, Goshima S, Tublin M, Bae KT. Pneumatosis intestinalis versus pseudo-pneumatosis: review of CT findings and differentiation. Insights Imaging 2011;2(1):85–92.
19. Batke M, Cappell MS. Adynamic ileus and acute colonic pseudo-obstruction. Med Clin North Am 2008;92(3):649–670, ix.
20. Bryk D, Soong KY. Colonic ileus and its differential roentgen diagnosis. Am J Roentgenol Radium Ther Nucl Med 1967;101(2): 329–337.
21. Moulin V, Dellon P, Laurent O, Aubry S, Lubrano J, Delabrousse E. Toxic megacolon in patients with severe acute colitis: computed tomographic features. Clin Imaging 2011;35(6):431–436.
22. Johnson CD, Rice RP, Kelvin FM, Foster WL, Williford ME. The radiologic evaluation of gross cecal distension: emphasis on cecal ileus. AJR Am J Roentgenol 1985;145(6): 1211–1217.
23. Frager D, Rovno HD, Baer JW, Bashist B, Friedman M. Prospective evaluation of colonic obstruction with computed tomography. Abdom Imaging 1998;23(2):141–146.
24. Godfrey EM, Addley HC, Shaw AS. The use of computed tomography in the detection and characterisation of large bowel obstruction. N Z Med J 2009;122(1305):57–73.
25. Beattie GC, Peters RT, Guy S, Mendelson RM. Computed tomography in the assessment of suspected large bowel obstruction. ANZ J Surg 2007;77(3):160–165.
26. Huynh LN, Coughlin BF, Wolfe J, Blank F, Lee SY, Smithline HA. Patient encounter time intervals in the evaluation of emergency department patients requiring abdominopelvic CT: oral contrast versus no contrast. Emerg Radiol 2004;10(6):310–313.
27. Stoker J, van Randen A, Laméris W, Boermeester MA. Imaging patients with acute abdominal pain. Radiology 2009;253(1): 31–46.
28. Hill BC, Johnson SC, Owens EK, Gerber JL, Senagore AJ. CT scan for suspected acute abdominal process: impact of combinations of IV, oral, and rectal contrast. World J Surg 2010;34(4):699–703.
29. Winter T. A plea for oral contrast administration in CT for emergency department patients. AJR Am J Roentgenol 2010;195(1): W90, W.
30. Bachulis BL, Smith PE. Pseudoobstruction of the colon. Am J Surg 1978;136(1):66–72.
31. Schwartz S, Nadelhaft J. Simulation of colonic obstruction at the splenic flexure by pancreatitis: roentgen features. Am J Roentgenol Radium Ther Nucl Med 1957;78(4): 607–616.
32. Chapman AH, McNamara M, Porter G. The acute contrast enema in suspected large bowel obstruction: value and technique. Clin Radiol 1992;46(4):273–278.
33. Garcia-Valdecasas JC, Llovera JM, deLacy AM, et al. Obstructing colorectal carcinomas: prospective study. Dis Colon Rectum 1991; 34(9):759–762.
34. Gandrup P, Lund L, Balslev I. Surgical treatment of acute malignant large bowel obstruction. Eur J Surg 1992;158(8):427–430.
35. Irvin GL 3rd, Horsley JS 3rd, Caruana JA Jr. The morbidity and mortality of emergent operations for colorectal disease. Ann Surg 1984;199(5):598–603.
36. Desai MG, Rodko EA. Perforation of the colon in malignant tumors. J Can Assoc Radiol 1973;24(4):344–349.
37. Horton KM, Abrams RA, Fishman EK. Spiral CT of colon cancer: imaging features and role in management. RadioGraphics 2000;20(2):419–430.
38. Chintapalli KN, Chopra S, Ghiatas AA, Esola CC, Fields SF, Dodd GD 3rd. Diverticulitis versus colon cancer: differentiation with helical CT findings. Radiology 1999;210(2): 429–435.
39. Rex DK, Kahi CJ, Levin B, et al. Guidelines for colonoscopy surveillance after cancer resection: a consensus update by the American Cancer Society and US Multi-Society Task Force on Colorectal Cancer. CA Cancer J Clin 2006;56(3):160–167; quiz 185–186.
40. Ballantyne GH, Brandner MD, Beart RW Jr, Ilstrup DM. Volvulus of the colon: incidence and mortality. Ann Surg 1985;202(1): 83–92.
41. Javors BR, Baker SR, Miller JA. The northern exposure sign: a newly described finding in sigmoid volvulus. AJR Am J Roentgenol 1999;173(3):571–574.
42. Burrell HC, Baker DM, Wardrop P, Evans AJ. Significant plain film findings in sigmoid volvulus. Clin Radiol 1994;49(5):317–319.
43. Feldman D. The coffee bean sign. Radiology 2000;216(1):178–179.
44. Salati U, McNeill G, Torreggiani WC. The coffee bean sign in sigmoid volvulus. Radiology 2011;258(2):651–652.
45. Rosenblat JM, Rozenblit AM, Wolf EL, DuBrow RA, Den EI, Levsky JM. Findings of cecal volvulus at CT. Radiology 2010;256(1): 169–175.
46. Levsky JM, Den EI, DuBrow RA, Wolf EL, Rozenblit AM. CT findings of sigmoid volvulus. AJR Am J Roentgenol 2010;194(1): 136–143.
47. Hirao K, Kikawada M, Hanyu H, Iwamoto T. Sigmoid volvulus showing "a whirl sign" on CT. Intern Med 2006;45(5):331–332.
48. Frank AJ, Goffner LB, Fruauff AA, Losada RA. Cecal volvulus: the CT whirl sign. Abdom Imaging 1993;18(3):288–289.
49. Shaff MI, Himmelfarb E, Sacks GA, Burks DD, Kulkarni MV. The whirl sign: a CT finding in volvulus of the large bowel. J Comput Assist Tomogr 1985;9(2):410.
50. Macari M, Spieler B, Babb J, Pachter HL. Can the location of the CT whirl sign assist in differentiating sigmoid from caecal volvulus? Clin Radiol 2011;66(2):112–117.

51. Delabrousse E, Sarlième P, Sailley N, Aubry S, Kastler BA. Cecal volvulus: CT findings and correlation with pathophysiology. Emerg Radiol 2007;14(6):411–415.

52. Peterson CM, Anderson JS, Hara AK, Carenza JW, Menias CO. Volvulus of the gastrointestinal tract: appearances at multimodality imaging. RadioGraphics 2009;29(5):1281–1293.

53. Moore CJ, Corl FM, Fishman EK. CT of cecal volvulus: unraveling the image. AJR Am J Roentgenol 2001;177(1):95–98.

54. Gumbs MA, Kashan F, Shumofsky E, Yerubandi SR. Volvulus of the transverse colon: reports of cases and review of the literature. Dis Colon Rectum 1983;26(12):825–828.

55. Newton NA, Reines HD. Transverse colon volvulus: case reports and review. AJR Am J Roentgenol 1977;128(1):69–72.

56. Ballantyne GH. Volvulus of the splenic flexure: report of a case and review of the literature. Dis Colon Rectum 1981;24(8):630–632.

57. Machado NO, Chopra PJ, Subramanian SK. Splenic flexure volvulus presenting with gangrene. Saudi Med J 2009;30(5):708–711.

58. Mindelzun RE, Stone JM. Volvulus of the splenic flexure: radiographic features. Radiology 1991;181(1):221–223.

59. Law WL, Lo CY, Chu KW. Emergency surgery for colonic diverticulitis: differences between right-sided and left-sided lesions. Int J Colorectal Dis 2001;16(5):280–284.

60. Lefèvre F, Béot S, Chapuis F, et al. Computed tomography study of the sigmoid colon: discriminating diagnostic criteria and interobserver correlations [in French]. J Radiol 1999;80(5):447–456.

61. Horton KM, Corl FM, Fishman EK. CT evaluation of the colon: inflammatory disease. RadioGraphics 2000;20(2):399–418.

62. Sai VF, Velayos F, Neuhaus J, Westphalen AC. Colonoscopy after CT diagnosis of diverticulitis to exclude colon cancer: a systematic literature review. Radiology 2012;263(2):383–390.

63. Gayer G, Zissin R, Apter S, Papa M, Hertz M. Pictorial review: adult intussusception—a CT diagnosis. Br J Radiol 2002;75(890):185–190.

64. Kim YH, Blake MA, Harisinghani MG, et al. Adult intestinal intussusception: CT appearances and identification of a causative lead point. RadioGraphics 2006;26(3):733–744.

65. Duncan JE, DeNobile JW, Sweeney WB. Colonoscopic diagnosis of appendiceal intussusception: case report and review of the literature. JSLS 2005;9(4):488–490.

66. Laalim SA, Toughai I, Benjelloun B, Majdoub KH, Mazaz K. Appendiceal intussusception to the cecum caused by mucocele of the appendix: laparoscopic approach. Int J Surg Case Rep 2012;3(9):445–447.

67. Yamada H, Morita T, Fujita M, Miyasaka Y, Senmaru N, Oshikiri T. Adult intussusception due to enteric neoplasms. Dig Dis Sci 2007;52(3):764–766.

68. Box JC, Tucker J, Watne AL, Lucas G. Eosinophilic colitis presenting as a left-sided colocolonic intussusception with secondary large bowel obstruction: an uncommon entity with a rare presentation. Am Surg 1997;63(8):741–743.

69. Ikuhara M, Tsang TK, Tosiou A, White EM, Buto SK, Meyer KC. Intussusception in an adult with pseudomembranous colitis. J Clin Gastroenterol 1995;21(4):336–338.

70. Levine MS, Trenkner SW, Herlinger H, Mishkin JD, Reynolds JC. Coiled-spring sign of appendiceal intussusception. Radiology 1985;155(1):41–44.

71. Stewart RB, Moore MT, Marks RG, Hale WE. Correlates of constipation in an ambulatory elderly population. Am J Gastroenterol 1992;87(7):859–864.

72. Martin LC, Merkle EM, Thompson WM. Review of internal hernias: radiographic and clinical findings. AJR Am J Roentgenol 2006;186(3):703–717.

73. Berg DF, Bahadursingh AM, Kaminski DL, Longo WE. Acute surgical emergencies in inflammatory bowel disease. Am J Surg 2002;184(1):45–51.

74. Swaminath A, Lichtiger S. Dilation of colonic strictures by intralesional injection of infliximab in patients with Crohn's colitis. Inflamm Bowel Dis 2008;14(2):213–216.

75. Laukoetter MG, Mennigen R, Hannig CM, et al. Intestinal cancer risk in Crohn's disease: a meta-analysis. J Gastrointest Surg 2011;15(4):576–583.

76. Brodey PA, Schuldt DR, Magnuson A, Esterkyn S. Complete colonic obstruction secondary to adhesions. AJR Am J Roentgenol 1979;133(5):917–918.

77. Holt RW, Wagner RC. Adhesional obstruction of the colon. Dis Colon Rectum 1984;27(5):314–315.

78. Ogilvie H. Large-intestine colic due to sympathetic deprivation; a new clinical syndrome. BMJ 1948;2(4579):671–673.

79. Ponec RJ, Saunders MD, Kimmey MB. Neostigmine for the treatment of acute colonic pseudo-obstruction. N Engl J Med 1999;341(3):137–141.

80. Jain A, Vargas HD. Advances and challenges in the management of acute colonic pseudo-obstruction (ogilvie syndrome). Clin Colon Rectal Surg 2012;25(1):37–45.

81. Vanek VW, Al-Salti M. Acute pseudo-obstruction of the colon (Ogilvie's syndrome): an analysis of 400 cases. Dis Colon Rectum 1986;29(3):203–210.

82. Rex DK. Colonoscopy and acute colonic pseudo-obstruction. Gastrointest Endosc Clin N Am 1997;7(3):499–508.

83. Low VH. Colonic pseudo-obstruction: value of prone lateral view of the rectum. Abdom Imaging 1995;20(6):531–533.

84. De Giorgio R, Cogliandro RF, Barbara G, Corinaldesi R, Stanghellini V. Chronic intestinal pseudo-obstruction: clinical features, diagnosis, and therapy. Gastroenterol Clin North Am 2011;40(4):787–807.

85. Choi JS, Lim JS, Kim H, et al. Colonic pseudoobstruction: CT findings. AJR Am J Roentgenol 2008;190(6):1521–1526.

86. Drożdż W, Budzyński P. Change in mechanical bowel obstruction demographic and etiological patterns during the past century: observations from one health care institution. Arch Surg 2012;147(2):175–180.