Large-bowel obstruction: CT findings, pitfalls, tips and tricks
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To cite this version:
Cécile Verheyden, Céline Orliac, Ingrid Millet, Patrice Taourel. Large-bowel obstruction: CT findings, pitfalls, tips and tricks. European Journal of Radiology, Elsevier, 2020, 130, pp.109155. 10.1016/j.ejrad.2020.109155 . hal-03369488

HAL Id: hal-03369488
https://hal.umontpellier.fr/hal-03369488
Submitted on 22 Aug 2022

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Paper title:
Large-bowel obstruction: CT findings, pitfalls, tips and tricks.

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Abstract
Large bowel obstruction (LBO) is associated with high morbidity and mortality due to
delayed diagnosis and/or treatment. MDCT has become the standard of care to identify the
site, severity, and etiology of obstruction. The goal of this review is fourfold. The first
objective is to give clues to differentiate LBO from colonic pseudo-obstruction. The second
objective is to describe CT features in the most common cause of LBO which is colonic
cancer by illustrating classical and atypical features of colonic cancer responsible for LBO
and by giving the features which must be reported when differentiating malignant from
benign: presence of local lymph nodes, other colic localizations, length of involved segment,
presence of diverticula, or other. The third objective is to illustrate the various causes of LBO
which can mimic a colon cancer by leading to a thickening of the colonic wall: diverticulitis,
ischemic colitis, endometriosis, inflammatory disease and to give tips which permit to evoke
another diagnosis than a colon cancer in patient with a LBO and a thickening of the colic
wall. The fourth objective is to describe the common signs of cecal and sigmoid volvulus and
to give tips for a diagnosis sometimes difficult particularly for cecal volvulus: one of two
transition points according to the type of volvulus and the presence of a whirl sign with a
torsion of the mesenteric vessels.

Keyword
Large-bowel obstruction.
Acute Colonic Pseudo-Obstruction.
Common and uncommon causes of LBO.
Large-bowel obstruction (LBO) is an abdominal pathology defined by a mechanical interruption of the flow of colic contents. LBO accounts for 2% to 4% of all surgical admissions (1). It is four to five times less frequent than small-bowel obstruction (SBO), but represents at least 25% of all intestinal obstructions (2-3).

However, it is an abdominal emergency with high morbidity and mortality rates, not to be left untreated. Actually, LBO is associated with an immediate risk of perforation and subsequent peritonitis. Recent studies highlight high morbidity and mortality rates of 42% to 46% and 13% to 19%, respectively, following surgery (4), which is often required (75%) (2). These complications are often due to delayed diagnosis or treatment.

The radiologist plays a major role in accurate diagnosis and in helping to choose the best treatment plan.

Abdominal radiography is usually the first imaging study performed in patients suspected of having LBO. LBO is demonstrated by dilatation of the colon, up to the level of the obstruction, with distal collapse. The reported sensitivity of abdominal radiography for the detection of LBO is 84%, whereas the reported specificity is 72%; as a result, it may be difficult to distinguish between obstruction and colonic pseudo-obstruction in a patient with a distended colon (5).

Computed tomography is the imaging method of choice, as stated by the consensus conference of the World Society of Emergency Surgery focused on the obstruction and perforation in colon and rectal cancer (6). Indeed, computed tomography can establish the diagnosis of LBO, it helps to distinguish the cause of LBO, and it permits to reveal its complications.

**Positive diagnosis**

The diagnosis of LBO is based on the presence of a dilated large bowel proximal to a transition point, and a decompressed bowel distal to the obstruction. The visualization of a transition point is considered a reliable finding for the diagnosis of LBO (1, 7).

Two main pitfalls are to be avoided when confronted by suspicion of LBO: mistake a functional obstruction for a mechanical one, and a small-bowel obstruction for a large-bowel one.
How to differentiate LBO from ACBO

The major mimic of LBO is acute colonic pseudo-obstruction (ACPO) and is the major differential diagnosis challenge. ACPO, also known as Ogilvie syndrome, is defined as an acute dilatation of the colon due to altered autonomic innervation of the colon. Its causes are numerous and suggest the diagnosis of ACPO when they are identified: all surgeries, cardiopulmonary and neurological diseases, metabolic troubles, medications and all systemic infections. Unlike in an adynamic ileus with small-bowel and colic dilatation, the important distension of the cecum in ACPO may lead to cecal ischemia and subsequent perforation, despite the absence of mechanical obstacle.

Even if ACBO is defined as a colic dilatation without mechanical obstacle, presentation at CT can be tricky, as in some cases, the dilatation only concerns some parts of the colon. The most frequent presentation is a dilatation of the colon lumen until the splenic flexure, without any thickening of the colic wall. In this case ACBO is very likely (Fig 1). Actually, spasm at the splenic flexure in a normal colon may mimic a fixed narrowing (8) and transitional region in ACBO tends to be at or near the splenic flexure (9). In these cases, relative cecal size may be useful in determining if a large bowel obstruction is present: if the colon is diffusely distended and the cecal diameter is clearly less than that of other colonic segments, LBO is unlikely (10). By contrast, in LBO, the cecum is usually the most dilated part, since, according to Laplace law, the largest diameter bowel requires the least pressure to distend and thus perforate.

In some cases, particularly in inflammatory process of the right upper and lower quadrants such as appendicitis or cholecystitis, and of the epigastrium such as pancreatitis, the dilatation of the colon may be limited to the ascending colon and to the hepatic flexure, responsible for the colon cut-off sign.

It is important, when looking at an isolated dilatation of the right colon to carefully search for short annular desmoplastic colonic lesions, which can be easily missed on CT scans (8), 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 than in left-colonic tumors (11).

How to differentiate proximal LBO from SBO
The other potential difficulty is to differentiate LBO from SBO in patients with dilatation of the small bowel and of the proximal large bowel. In patients with LBO, the competence of the ileocecal valve influences the pattern of the colon and of the small bowel. 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 (12). An incompetent ileocecal valve will decompress the LBO into the small bowel. The resultant small-bowel distension may mimic a distal SBO. This can be differentiated from SBO by tracing the dilated distal small bowel to the IC valve, in the setting of colonic dilation. Careful attention is needed in patients with feature of distal SBO to look for the presence of a right-sided colic tumor with an incompetent ileocecal valve (Fig 2).

**Diagnosis of cause**

**The common causes**

The major causes of LBO are primary colon carcinoma (60-80%), volvulus (11-15%) and fecal impaction (12). They account for about 90% of cases of LBO.

**Colon carcinoma : diagnosis**

Colon carcinoma is the most common cause of LBO (> 60% of cases), and mortality is high (10%–30%) in patients requiring emergency surgery (13, 14, 15). The two most frequent locations of obstruction due to colonic malignancy are the sigmoid colon (Fig 3) and the splenic flexure.

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. Obstructing colon cancers often produce a shouldering appearance and may be large enough to have central necrosis or rarely air within the mass, which may resemble an abscess (16). It is especially important to look for colic malignancy in the setting of unexplained abscess in the pelvis in elderly female patients.

How to differentiate colon cancer and colic diverticulitis

Colonic malignancy may mimic diverticulitis if there is pericolonic spread with infiltration of the pericolonic fat (Fig 4). Studies have assessed the value of CT findings to differentiate sigmoid cancer versus chronic diverticular disease (17, 18), the most recent being focused on CT colonography (19). Radiographic features used for differential diagnosis are the pattern of
wall thickening, the presence of a pericolic infiltration and the characteristics of eventual associated lymph nodes.

Table adapted from (19).

<table>
<thead>
<tr>
<th>Features</th>
<th>Colon cancer</th>
<th>Diverticulitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of the affected segment</td>
<td>&lt; 10 cm</td>
<td>&gt;10 cm</td>
</tr>
<tr>
<td>Signs of inflammation</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Stricture margins</td>
<td>Shouldering</td>
<td>Smooth</td>
</tr>
<tr>
<td>Diverticula within the involved segment</td>
<td>Not necessarily</td>
<td>Yes</td>
</tr>
<tr>
<td>Enhancement</td>
<td>Strong</td>
<td>Target pattern</td>
</tr>
<tr>
<td>Lymph nodes</td>
<td>&gt;1 cm (in short axis)</td>
<td>Absent or &lt;1 cm (in short axis)</td>
</tr>
</tbody>
</table>

However there are some pitfalls
- The presence of diverticula within unaffected segments has less diagnostic value, particularly because of the epidemiologic association between diverticulitis and colon carcinoma
- In chronic forms of diverticulitis, colonic infiltration may show a mass-like pattern and differentiating it from colorectal carcinoma can be difficult. It involves the necessity of follow-up colonoscopy for definitive pathologic differentiation.
- Infiltration of the pericolic fat in colon carcinoma may mimic diverticulitis, pericolic infiltration may be due to either tumor infiltration or to inflammation caused by microperforation (18)
- Normal-sized nodes may have microscopic tumor involvement (12)

It must be noted that most published studies do not include patients in the setting of bowel obstruction. In clinical practice the presence of colonic obstruction is a significant factor in favour of colon cancer. Actually the incidence of LBO in patients with colon cancer is high, reaching 15 to 29% in single-institution studies (20,21). Although this rate is likely overestimated, with a rate of 8% in a large population of patients with stage IV colon cancer (22), the frequency of colon cancer makes LBO due to colon cancer a common setting. By contrast, in a trial assessing CT imaging predictive factors for progression from uncomplicated to complicated acute diverticulitis, the rate of patients developing a large-bowel obstruction requiring surgical intervention was only 1% (23)
The other main differential diagnosis in LBO with thickening of the colon wall and narrowing of the colonic lumen is colonic ischemia in a chronic phase, with short segment ischemic strictures. There is fibrotic reaction in the injured colonic wall, leading to continuous mild and irregular circumferential wall thickening with narrowing of the lumen. Pericolic fluid is not usually found in the setting of chronic stricture related to ischemia, as recurrent episodes of ischemia or other unassociated causes of abdominal pathology (e.g. cirrhosis or renal disease) may result in pericolic fluid.

**Colon carcinoma: staging**

CT staging in colon carcinoma is based on the American Joint committee on Cancer (AJCC) TNM system and includes the assessment of tumoral extension (T), of lymph node extension (N) and of metastasis extension (M). Some subtle findings for the evaluation of tumoral extension must be known (24).

How to better assess tumoral extension

- Look for advanced T3 (T3c, T3d) sub-stage disease: infiltration more than 5 mm beyond the bowel wall,
- Look for T4 disease: invasion of adjacent organ, tumor perforation, peritoneal infiltration
- Look for extramural vein invasion: nodular spread into small vessels, definite enhancing tumour spread along a large vein

These CT findings have important prognostic value, as shown in a recent trial which demonstrates that advanced T3 sub-stage disease, T4 disease and extramural vein invasion identified by CT are strongly associated with worse disease-free survival in colon cancer.

In patients with LBO due to cancer, special attention must be given to review the entire colon for synchronous lesions, which occur in 2-7% of patients (25), especially as the tumoral stenosis may be impassable with endoscopy, making impossible to diagnose a second tumor by colonoscopy. When there is a thickening of the colic wall proximally to the colon cancer, the differential diagnosis between a second carcinoma and ischemic colitis proximally to the cancer is raised. In ischemic colitis, colic wall thickening is symmetric, with typically a conservation of the different layers. Overall, ischemic segments are contiguously proximal to the tumoral segment.

An accurate staging of the colon cancer, its extent, its location, the presence of added complications such as perforation are of importance in the choice of therapeutic options in
obstructive colon cancer, which include Hartmann’s procedure, resection with or without primary anastomosis, decompression with proximal colostomy, tube decompression or endoscopic colic stenting (Fig 10).

**Volvulus**

Acute colonic volvulus accounts for approximately 11%–15% of LBO. 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. 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. Colonic volvulus may involve the sigmoid colon, the cecum and the transverse colon. 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%). According to demographics, sigmoid volvulus is more common in older patients, cecal in younger

Sigmoid volvulus
CT is the best imaging modality for the diagnosis of sigmoid volvulus. Classic signs of sigmoid volvulus on CT scan are well known (26). On CT scanogram, the most sensitive signs were the absence of rectal gas (90%) and the U-shaped appearance (an inverted ahuastral dilated sigmoid in the shape of an inverted “U” extending into the right upper quadrant) (86%), while the most sensitive findings on cross-sectional imaging were a single transition point in the sigmoid (95%) and disproportionate enlargement of the sigmoid (86%). A “beak” is usually found at the twisting point of the sigmoid colon, and describes the smooth, tapering transition point of the obstruction. The whirl sign is defined as the appearance of spiraled loops of collapsed bowel with engorged vessels radiating from the twisted bowel, and should be searched for at the point of obstruction (27) (Fig 5). The location of this sign is highly accurate in discriminating cecal volvulus from sigmoid volvulus, according to Macari et al (28)

Two types of sigmoid volvulus are defined:
- Mesenterico-axial form: The most classic pattern of sigmoid volvulus where there is a bird beak aspect of both the afferent and efferent segments, thus constituting a closed loop obstruction. However this classical form might be missing (26).
- Organo-axial form: the sigmoid colon rotates along its long axis, showing only one beak.
Cecal volvulus

Cecal volvulus results from an abnormal mobility of the cecum because of improper fusion of the cecal mesentery with the posterior parietal pneumoperitoneum. CT appearance depends on the pathophysiological mechanism of the volvulus (Fig 6). Three types of cecal volvulus are defined:

- The axial torsion type: the cecum rotates along its axis and remains located in the right lower quadrant and twists in the axial plan
- The loop type: the distended cecum both twists and inverts. The distended cecum is located in the left upper quadrant
- The bascule type: the distended cecum folds anteriorly without any torsion. The distended cecum is located in the central abdomen and the whirl sign is absent.

Because the diagnosis of cecal volvulus may be tricky and often underestimated even with CT, it is useful to follow strict rules to perform the diagnosis by CT:

1. To identify the cecum and not to confound it with the stomach, the transverse colon or the sigmoid colon by scrolling the colon from the rectum to the distal ileum. Sometimes the identification of the displaced appendix may be an ancillary finding to identify the cecum
2. To look for a beak finding at the level of the volvulus. This beak finding permits to differentiate a cecal volvulus from an acute colonic pseudo-obstruction with predominant cecal distension
3. To look for a whirl finding which means a volvulus with a real twist. The whirl finding is generally located in the right lower quadrant regardless the location of the cecum
4. To identify the ileocecal valve in order not to perform a false diagnosis of small bowel obstruction

Unlike sigmoid volvulus, nonoperative decompression is not usually possible. Emergent surgery with detorsion is recommended. Resection and anastomosis is the favored option for both gangrenous and viable bowel. Cecopexy and cecostomy constitute alternatives in unstable high-risk patients (29).

Fecal impaction

Fecal impaction (FI) is a common cause of LBO. It is the result of chronic or severe constipation and is most commonly found in the elderly and neuropsychiatric patients. About
40% of patients with fecal impaction had a history of prior impactions (30). CT shows the presence of large fecal matter in the colon and rectum with or without signs of colonic perforation (Fig 7). In addition to diagnosing the cause of LBO, CT allows to diagnose stercoral colitis which constitutes the main complication of LBO due to fecal impaction. Common sites of ulceration are anterior rectum, anti-mesenteric border of the recto sigmoid junction, and the apex of the sigmoid colon. CT may show mural thickening greater than 3 mm, increased mucosal density which reflects intramural hemorrhage and discontinuous colonic mural enhancement due to perfusion defects (31).

Treatment of fecal impaction includes digital manipulation, enema instillation, or disimpaction under anesthesia. Laparotomy is needed in cases of failure of these procedures or in stercoral peritonitis.

Other causes
The other causes of LBO account for about 10% of LBO. There are intrinsic causes, including intussusception and foreign body, mural causes including inflammatory bowel disease, acute diverticulitis, post anastomosis stenosis, radiation stricture, NSAID colopathy, ischemic colitis and extrinsic causes including peritoneal carcinomatosis, endometriosis and hernias (4).

Table 1 summarizes CT key findings, pitfalls, tips and tricks for diagnosis of these various entities.

Diagnosis of complications
The main complications of large bowel obstruction are perforation and ischemia, which may be associated.

In LBO due to cancer, perforation may have two mechanisms: perforation of the tumor itself (which is the most common cause) or perforation arising from the distended cecum above the tumor. The amount of pneumoperitoneum and the stranding of the peritumoral fat are key findings to distinguish these two causes of perforation. In perforation arising from the tumor, free pneumoepiritoneum is rare, more often small bubbles with fluid and mesenteric stranding are detected in the pericolic fat adjacent to the tumor. In the case of diastatic perforation of the cecum, pneumoperitoneum is abundant and there is no particular peritumoral fat infiltration. The presence of pneumatosis within the cecal wall may constitute an argument for cecal ischemia, however pneumatosis in dilated cecum proximal to bowel cancer may also be the consequence of the dilatation without any finding of ischemia (32).
The differentiation between perforation at the tumor site and diastatic perforation may have impact on the surgical procedure (6): perforation at the tumor site may need formal resection with or without anastomosis and with or without stoma, diastatic perforation needs simultaneous tumor resection and management of proximal perforation. Depending on the colic wall condition, a subtotal colectomy may be required.

In LBO due to sigmoid volvulus, intraluminal reduction via colonoscopy or contrast enema can often be successfully performed. Surgical intervention is required in patients who show signs and symptoms of bowel ischemia or when intraluminal reduction is not successful. CT signs have been assessed as ineffective for the prediction of the presence of pathologically proven ischemia until there was frank bowel necrosis (26). A newly defined CT imaging factor of ischemia has recently been described, the “dark torsion knot sign” shown as a sudden loss of mucosal enhancement in the volvulus torsion knot (33). This CT factor and a clinical factor (sepsis) were the only factors able to predict complicated volvulus sigmoid necessitating emergent surgery instead of colonoscopic detorsion as a primary treatment of choice.

To conclude, a rapid and accurate diagnosis of LBO, of its cause and of eventual added complication is crucial in the management of suspicion of LBO. CT is the reference exam and has strong impact on the management of the obstruction for which the treatment options are numerous and depend mainly on the cause, on the general condition of the patient, and on the location of the obstruction.
References


**Figures**

Fig 1:
85-year-old man with abdominal distension caused by acute colonic pseudo-obstruction (ACPO)
Axial (a), coronal (b) and sagittal (c) contrast-enhanced abdominal CT images show diffusely dilated colon, especially for the sigmoid colon (asterisk). Absence of transition point. No argument for an other diagnosis : no mural thickening, no whirl-sign, no pericolonic anomaly.

Fig 2:
Images in a 81-year-old man with LBO from an ascending colon cancer.
(a) Axial contrast-enhanced abdominal CT image shows a short wall thickening (arrow) with luminal narrowing, without any abnormality in the pericolonic fat. Note the dilatation of distal small bowel caused by an incompetent ileocecal valve (asterisk).
(b) and (c) Coronal (b) and sagittal (c) contrast-enhanced abdominal CT images show a short wall thickening with shouldering margins (arrows) and an cecal distension with pneumatosis (arrowheads), consequence of the dilatation without any finding of ischemia during the emergent surgery.

Fig 3:
Images in a 90-year-old woman with LBO from a descending colon cancer.
A, b and c: axial (a), coronal (b) and sagittal (c) contrast-enhanced abdominal CT images show a short mural thickening, with shouldering margins, and no sign of inflammation. Note the dilatation of distal small bowel caused by an incompetent ileocecal valve (asterisk).

Fig 4:
85-year-old woman with abdominal pain and abdominal distension.
(a), (b) and (c) sagittal (a) and axial (b, c) contrast-enhanced abdominal CT images show a mural thickening (arrow), infiltration of the pericolonic fat and diverticula within the involved segment (arrowhead).
Acute diverticulitis was diagnosed and medical therapy was successfully done.
(d) and (e) New episode of abdominal pain and abdominal distension, 6 month later. Axial (d) and sagittal (e) contrast-enhanced abdominal CT images show asymmetric mural thickening (arrow). Colon carcinoma was diagnosed during emergent surgery. The initial diagnosis was likely inappropriately called diverticulitis. Actually, the segment of wall thickening is short and the diverticulum is not convincing.

Fig 5:
Images in a 79-year-old man 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 (arrow). There is no gas in the rectum (arrowhead).
(b) Axial contrast-enhanced CT image of the abdomen and pelvis shows dilated sigmoid colon proximal to the volvulus (asterisk) with a distal “whirl” of the mesentery at the point of volvulus (arrow).

Fig 6:
40-year-old man with abdominal distension: diagnostic of LBO caused by cecal volvulus. Contrast-enhanced abdominal CT axial (a), coronal (b, c) and sagittal (d) images.
(a) Axial contrast-enhanced abdominal CT image shows an cecal distension (asterisk) and a cecal twist, rotates into the left upper quadrant (arrow).
(b) Coronal contrast-enhanced abdominal CT 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).
(c) Coronal contrast-enhanced abdominal CT image shows the cecal malposition (arrow), the whirl sign (arrowhead), and a dilatation of distal small bowel caused by an incompetent ileocecal valve (asterisk).
(d) Sagittal contrast-enhanced abdominal CT image shows the site of the twist forming an appearance that resembles a bird’s beak (arrow).

Fig 7:
69 year-old man with constipation and abdominal pain caused by fecaloma. Axial (a) and coronal (b) contrast-enhanced abdominal CT show significant dilatation and fecal impaction of rectosigmoid colon (asterisk), and pneumatosis (arrow) caused by dilatation. Note the ascendant colon dilatation (arrowhead).

Fig 8:
Images in a 86-year-old woman with abdominal pain and abdominal distension : diagnostic of LBO caused by a colocolonic intussusception. (a-b) Axial (a) and coronal (b) contrast-enhanced abdominal CT images show a cecal and right colonic intussusception (arrows), like a “target” in cross-section (a), like a sausage-shaped mass in longitudinal plan(b). (c) and (d) Axial (c) and coronal (d) reformatted CT images of the abdomen and pelvis show at the lead point for the obstruction an oval and well-marginated mass with fat density : lipoma of the cecal (arrows).

Fig 9:
Images in a 29-year-old woman with LBO caused by Crohn colitis involving the descending colon. (a) Coronal contrast-enhanced CT image demonstrates a fibrostenotic disease with luminal narrowing, prestenotic dilatation and wall thickening. The thickened segment is long, has an hyperenhancement of the mucosa (arrow), is associated with adjacent perforation and abscess (arrowhead). (b) Sagittal contrast-enhanced CT image shows hyperemic vascular engorgement (Comb sign) (arrowhead).

Fig 10:
52-year-old man with abdominal pain and obstipation : diagnostic of LBO from a descending colon cancer complicated by cecal perforation. (a) Axial contrast-enhanced abdominal CT image shows short wall thickening (arrow) with luminal narrowing, with discreet infiltration of the pericolonic fat. Note the cecal distension (asterisk). (b) Coronal contrast-enhanced abdominal CT image shows dilate and stool-filled cecum (arrow), proximal to the descending colon tumor.
(c) Coronal contrast-enhanced abdominal CT image shows a free pneumoperitoneum (arrow).
| **Table 1 : Uncommon causes of large bowel obstruction : CT findings, pitfalls, tips and tricks** |
|-------------------------------------------------|-----------------|-----------------|-----------------|
| **Luminal causes** | **Imaging : CT findings** | **Pitfalls** | **Tips and tricks** |
| Intussusception (1-2% LBO) (Fig 8) | **Colo-colonic intussusception** :  
Intussuscipiens : distended colon with a thickened wall  
Intraluminal intussusceptum telescoping within the intussuscipiens  
Curvilinear area of fat : invaginated mesenteric fat of the intussusceptum  
Invaginated vessels accompanying the intussusceptum. | Distinguish between lead point intussusception and non-lead point intussusception  
Look for a cause +++ : Not always easily determined  
Not always seen | Colo-colonic intussusception like a “target” in cross-section  
like a sausage-shaped mass in longitudinal plane  
Causes  
Malign tumor : Carcinoma  
Irregular wall thickening  
Greater caliber of large bowel lumen  
Lipoma : oval and well-margined mass with fat density  
Adenomatous polyp : regular marginated mass with fat density  
 Appendiceal mucocele : well-encapsulated cystic mass protruding in the cecum lumen |
| Foreign body | Many causes with various CT findings : gallstones, enteroliths, intentionally inserted foreign body, medications, illegal drugs  
By insertion +++ , ingestion, migration, surgery | May be inconspicuous because of its composition  
Not mainly the cause of obstruction | Complications : mostly the cause of obstruction  
Indent perforation :  
Metallic and sharp objects  
Ileocecal region and appendix  
Infection : inflammation pitfalls  
Fistula and abscesses |
| **Mural causes** | **Imaging : CT findings** | **Pitfalls** | **Tips and tricks** |
| Acute diverticulitis | Colonic diverticular disease  
Bowel wall :  
Segmental and symetric wall thickening  
Hyperemia  
Pericolonic inflammation  
Fat stranding  
Fluid in the mesentry  
Vascular engorgement  
Complications :  
Abscesses  
Perforation  
Fistula | Mimicking of colonic tumor +++  
Colonoscopic evaluation with biopsy is often required to distinguish between diverticulitis and a colonic malignancy | Look for signs of chronic diverticulitis :  
More diffuse colonic thickening (length > 10 cm)  
Signs of inflammation  
Smooth margins  
Diverticula in the involved segment  
Target pattern enhancement |
| Inflammatory bowel disease  
Crohn disease (Fig 9)  
Ulcerative colitis | Fibrostenotic disease :  
Luminal narrowing  
Prestenotic dilatation  
Wall thickening  
Signs of active inflammation :  
Mucosal hyperemia  
Hyperemic vascular engorgement (Comb sign)  
Fluid in the mesentery  
Abscesses  
Fistulae | Exclude malignancy +++  
Risk 2 to 3 times higher  
Difficult on imaging  
Colonoscopic evaluation with biopsy is often required  
Ulcerative colitis :  
Less common  
Suspicion of an underlying malignancy +++  
Crohn disease : distinguish active inflammation and chronic fibrotic stenosis | More diffuse colonic thickening (length > 10 cm)  
In active inflammation :  
Mural stratification  
Mucosal hyperenhancement  
In chronic involvement :  
Marked wall thickening  
Homogeneous attenuation of the wall  
Shortening and luminal narrowing  
Mimicking of colonic tumor |
<p>| Radiation | Sigmoid colon and rectum | Surgical and treatment history |</p>
<table>
<thead>
<tr>
<th>Early phase</th>
<th>Various lengths</th>
<th>Various degrees of narrowing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular wall thickening</td>
<td></td>
<td></td>
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<tr>
<td>Edema and sloughing, with associated</td>
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<td></td>
</tr>
<tr>
<td>Chronic phase</td>
<td>Fibrotic strictures</td>
<td></td>
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<tr>
<td>Ischemic colitis</td>
<td>Chronic phase</td>
<td>Fibrotic strictures</td>
</tr>
<tr>
<td>Ischemic segment</td>
<td>Smoothly thickened</td>
<td>Homogeneously enhanced</td>
</tr>
<tr>
<td>Postsurgical or Anastomotic stricture</td>
<td>Abrupt transition or focal narrowing of large-bowel caliber at the site of anastomosis</td>
<td></td>
</tr>
<tr>
<td>Decompression of large-bowel loops distal to the anastomosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical colopathy NSAID</td>
<td>Typical localisations: cecum, ascending colon, rectum</td>
<td></td>
</tr>
<tr>
<td>Distinguish inflammatory disease and NSAID colopathy may be difficult</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Extrinsic causes

<table>
<thead>
<tr>
<th>Hernias</th>
<th>Most affected extragenital location: sigmoid and rectum</th>
<th>May simulate colon carcinoma or serosal metastatic disease</th>
<th>Distinguish endometriosis and carcinoma: Age and clinical history</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Variables findings on CT Serosal soft-tissue mass Penetrating thickened colon wall Severe rectosigmoid stricture</td>
<td></td>
<td>Involved the serosa and muscularis layer Sparing the mucosa</td>
</tr>
<tr>
<td></td>
<td>Hernias discovered: Internal: Foramen of Winslow hernia+++ Externel: Inguinal +++, every hernias Hernia sac contains large bowel Obstruction: dilated proximal colon and decompressed distal colon</td>
<td>Overwhelming majority of hernias are incidentally discovered on imaging and do not cause obstruction</td>
<td>Look for other localisations of endometriosis</td>
</tr>
<tr>
<td></td>
<td>Foramen of Winslow hernia Right colon herniate through the normal communication between the greater and lesser peritoneal cavities Association with herniation of small bowel</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neoplasia</th>
<th>Site of extracolonic tumor Metastatic disease Presence of a soft-tissue mass</th>
<th>May simulate colon carcinoma</th>
<th>Enhanced serosal soft-tissue mass with colonic luminal compression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peritoneal metastasis Serosal metastases Prostatic malignancies Gynecologic malignancies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intra-abdominal pathology</td>
<td>Pancréatitits</td>
<td>Intra abdominal abscesses</td>
<td>Lymphadenopathy</td>
</tr>
<tr>
<td>---------------------------</td>
<td>---------------</td>
<td>--------------------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Adhésions</td>
<td>Colonic obstruction without an obvious cause</td>
<td></td>
<td>Surgical history</td>
</tr>
</tbody>
</table>
Table 1: Causes of Acute Colonic Pseudo-Obstruction

<table>
<thead>
<tr>
<th>Various causes</th>
<th>Risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgical</td>
<td>Orthopedic surgery, organ transplantation, spine surgery, cardiac surgery</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td>Mechanical ventilation, pneumonia, myocardial infarction, congestive heart failure, chronic obstructive lung disease</td>
</tr>
<tr>
<td>Neurological</td>
<td>Spinal cord injury, stroke, dementia, multiple sclerosis, parkinson disease</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Electrolyte imbalance, diabetes, renal failure, hepatic failure</td>
</tr>
<tr>
<td>Medications</td>
<td>Opiates, anticholinergics, antiparkinson agents, chemotherapy, antipsychotics</td>
</tr>
<tr>
<td>Gynaecological</td>
<td>Normal pregnancy, vaginal delivery, caesarean section, pelvic surgery</td>
</tr>
<tr>
<td>Infectious</td>
<td>All systems infections</td>
</tr>
<tr>
<td>Others</td>
<td>Severe sepsis, burns,</td>
</tr>
</tbody>
</table>