Intestinal Obstruction

Dr.Mohammed Tareq
Lecturer in College of Medicine
University of Al-Anbar

SMALL BOWEL OBSTRUCTION

Epidemiology

Mechanical small bowel obstruction is the most frequently encountered surgical disorder of the small intestine.Although a wide range of etiologies for this condition exists, the obstructing lesion can be conceptualized according to its anatomic relationship to the intestinal wall as:

1. *Intraluminal* (e.g., foreign bodies, gallstones, or meconium)

2. *Intramural* (e.g., tumors, Crohn's disease–associated inflammatory
strictures)

3. *Extrinsic* (e.g., adhesions, hernias, or carcinomatosis)

Intra-abdominal adhesions related to prior abdominal surgery
account for up to 75% of cases of small bowel obstruction. Less prevalent etiologies for small bowel obstruction
include hernias, malignant bowel obstruction, and Crohn's disease. The frequency with which obstruction related to these conditions is encountered varies according to the patient population and practice setting. Cancer-related small bowel obstructions are commonly due to extrinsic compression or invasion by advanced malignancies arising in organs other than the small bowel;
congenital abnormalities capable of causing small bowel

obstruction usually become evident during childhood, they sometimes elude detection and are diagnosed for the first time in adult patients presenting with abdominal symptoms.

For example, intestinal malrotation and mid-gut volvulus should not be forgotten when considering the differential diagnosis of adult patients with acute or chronic symptoms of small bowel obstruction, especially those without a history of prior abdominal surgery. A rare etiology of obstruction is the superior mesenteric artery syndrome, characterized by compression of the third portion of the duodenum by the superior mesenteric artery as it crosses over this portion of the duodenum.

This condition should be considered in young asthenic individuals who have chronic symptoms suggestive of proximal small bowel obstruction.

Pathophysiology

With onset of obstruction, gas and fluid accumulate within the intestinal lumen proximal to the site of obstruction. The intestinal activity increases in an effort to overcome the obstruction, accounting for the colicky pain and the diarrhea that some experience even in the presence of complete bowel obstruction. Most of the gas that accumulates originates from swallowed air, although some is produced within the intestine.

The fluid consists of swallowed liquids and gastrointestinal secretions (obstruction stimulates

intestinal epithelial water secretion). With ongoing gas and fluid accumulation,
the bowel distends and intraluminal and intramural pressures rise. The intestinal motility is eventually reduced with fewer contractions. With obstruction, the luminal flora of the small bowel, which is usually sterile, changes, and a variety of organisms have been cultured from the contents. Translocation of these bacteria to regional lymph nodes has been demonstrated, although the significance of this process is not well understood.

If the intramural pressure becomes high enough, intestinal microvascular perfusion is impaired, leading to intestinal ischemia and, ultimately, necrosis. This condition is termed *strangulated bowel obstruction*.

With *partial small bowel obstruction*, only a portion of the intestinal lumen is occluded, allowing passage of some gas and fluid. The progression of pathophysiologic events described earlier tends to occur more slowly than with *complete small bowel obstruction*, and development of strangulation is less likely.

A particularly dangerous form of bowel obstruction
is *closed-loop obstruction* in which a segment of intestine is obstructed both proximally and distally (e.g., with volvulus). In such cases, the accumulating gas and fluid cannot escape either proximally or distally from the obstructed segment, leading to a rapid rise in luminal pressure and a rapid progression to strangulation.

Clinical Presentation

The symptoms of small bowel obstruction are colicky abdominal pain, nausea, vomiting, and obstipation. Vomiting is a more prominent symptom with proximal obstructions than distal.

The character of vomitus is important because with bacterial overgrowth, the vomitus is more feculent, suggesting a more established obstruction. Continued passage of flatus and/or stool beyond 6 to 12 hours after onset of symptoms is characteristic of partial rather than complete obstruction.

The signs of smallbowel obstruction include abdominal distention, which is most pronounced if the site of obstruction is in the distal ileum and may be absent if the site of obstruction is in the proximal small intestine. Bowel sounds may be hyperactive initially, but in late stages of bowel obstruction, minimal bowel sounds may be heard. Laboratory findings reflect intravascular volume depletion and consist of hemoconcentration and electrolyte abnormalities.

Mild leukocytosis is common.

Features of strangulated obstruction include abdominal pain often disproportionate to the degree of abdominal findings, suggestive of intestinal ischemia. Patients often have tachycardia, localized abdominal tenderness, fever, marked leukocytosis, and acidosis. Any of these findings should alert the clinician to the possibility of strangulation and need for early surgical intervention.

Diagnosis

The diagnostic evaluation should focus on the following goals:
(a) distinguish mechanical obstruction from ileus,
 (b) determine the etiology of the obstruction,
(c) discriminate partial from complete obstruction,
(d) discriminate simple from strangulating obstruction.
Important elements to obtain on history include prior abdominal operations (suggesting the presence of adhesions) and the presence of abdominal disorders (e.g., intra-abdominal cancer or inflammatory bowel disease) that may provide insights into the etiology of obstruction. Upon examination, a meticulous search for hernias (particularly in the inguinal and femoral regions) should be conducted.
The diagnosis of small bowel obstruction is usually confirmed with radiographic examination. The *abdominal series* consists of :
(a) a radiograph of the abdomen with the patient
in a supine position,
(b) a radiograph of the abdomen with the patient in an upright position, and
 (c) a radiograph of the chest with the patient in an upright position.
 The finding most specific for small bowel obstruction is the triad of dilated small bowel loops (>3 cm in diameter), air-fluid levels seen on upright films, and a paucity of air in the colon. The sensitivity of abdominal radiographs in the detection of small bowel obstruction ranges
70% to 80%. Specificity is low, because ileus and colonic obstruction can be associated with findings that mimic those observed with small bowel obstruction. False-

negative findings on radiographs can result when the site of obstruction is located in the proximal small bowel and when the bowel lumen is filled with fluid but no gas, thereby preventing visualization of airfluid levels or bowel distention. The latter situation is associated with closed-loop obstruction. Despite these limitations, abdominal radiographs remain an important study in patients with suspected small bowel obstruction because of their widespread availability and low cost .

Computed tomography (CT) scanning is 80% to 90% sensitive and 70% to 90% specific in the detection of small bowel obstruction. The findings of small bowel obstruction include a discrete transition zone with dilation of bowel proximally, decompression of bowel distally, intraluminal contrast that does not pass beyond the transition zone, and a colon containing little gas or fluid. CT scanning may also provide evidence for the presence of closed-loop obstruction and strangulation. Closed-loop obstruction is suggested by the presence of a U-shaped or C-shaped dilated bowel loop associated with a radial distribution of mesenteric vessels converging toward a torsion point.

Strangulation is suggested by thickening of the bowel wall, pneumatosis intestinalis (air in the bowel wall), portal venous gas, mesenteric haziness, and poor uptake of intravenous contrast into the wall of the affected bowel . CT scanning also offers a global evaluation of the abdomen and may therefore reveal the etiology of obstruction. This feature is important in the acute setting when intestinal obstruction represents only one of many diagnoses in patients with acute abdominal conditions.

Small bowel obstruction: common etiologies

Adhesions
Neoplasms
    Primary small bowel neoplasms
    Secondary small bowel cancer (e.g., melanoma-derived metastasis)
    Local invasion by intra-abdominal malignancy (e.g., desmoid tumors)
    Carcinomatosis
Hernias
    External (e.g., inguinal and femoral)
    Internal (e.g., following Roux-en-Y gastric bypass surgery)
Crohn's disease
Volvulus
Intussusception
Radiation-induced stricture
Postischemic stricture
Foreign body
Gallstone ileus
Diverticulitis
Meckel's diverticulum
Hematoma
Congenital abnormalities (e.g., webs, duplications, and malrotation)

Figure 28-12. Small bowel obstruction. Plain radiographs (A) supine, which show dilated loops of small bowel in the right upper quadrant and (B) erect, which confirm the presence of air-fluid level in the loops of small bowel as well as the stomach, consistent with small bowel obstruction.

The CT scan is usually performed after administration of oral water-soluble contrast or diluted barium. The water-soluble contrast has been shown to have prognostic and therapeutic value too. Several studies and a subsequent meta-analysis have shown that appearance of the contrast in the colon within 24 hours is predictive of nonsurgical resolution of bowel obstruction.

A limitation of CT scanning is its low sensitivity (<50%) in the detection of low-grade or partial small bowel obstruction. A subtle transition zone may be difficult to identify in the axial images obtained during CT scanning. In such cases, contrast examinations of the small bowel, either *small bowel series*
(small bowel follow-through) or *enteroclysis*, can be helpful.(Enteroclysis is the use of a contrast agent normally administered through a nasogastric tube. Enteroclysis is valuable in detecting the presence of obstruction and in differentiating partial from complete blockages. This study is useful when plain radiographic findings are normal in the presence of clinical signs of small-bowel obstruction (SBO) or when plain radiographic findings are nonspecific. Enteroclysis is often less desirable when compared to computed tomography (CT) scanning due to the risk of perforation or aspiration with the administration of contrast medium.)

For standard small bowel series, contrast is swallowed or instilled into the stomach through a nasogastric tube. Abdominal radiographs are then taken serially as the contrast travels distally in the intestine. Although barium can be used, watersoluble contrast agents, such as Gastrografin, should be used if the possibility of intestinal perforation exists.

These examinations are more labor-intensive and less rapidly performed than CT scanning but may offer greater sensitivity in the detection of luminal and mural etiologies of obstruction, such as primary intestinal tumors. For enteroclysis, 200 to 250 mL of barium followed by 1 to 2 L of a solution of methylcellulose in water are instilled into the proximal jejunum via a long nasoenteric catheter. The double-contrast technique used in enteroclysis

permits a better assessment of mucosal surface and detection of relatively small lesions, even through overlapping small bowel loops. Enteroclysis is rarely performed in the acute setting but offers greater sensitivity than small bowel series in the detection of lesions that may be causing partial small bowel obstruction.

Recently, CT enteroclysis has been used and reported to be superior to plain x-ray small bowel contrast studies.

Therapy:

Small bowel obstruction is usually associated with a marked depletion of intravascular volume due to decreased oral intake, vomiting, and sequestration of fluid in bowel lumen and wall.

Therefore, fluid resuscitation is integral to treatment. Isotonic fluid should be given intravenously, and an indwelling bladder catheter may be placed to monitor urine output. Central venous or pulmonary artery catheter monitoring may be necessary to assist with fluid management in patients with underlying cardiac

disease and severe dehydration. Broad-spectrum antibiotics are given by some because of concerns that bacterial translocation may occur in the setting of small bowel obstruction;

however, there are no data to support this approach.

The stomach should be continuously evacuated of air and fluid using a nasogastric (NG) tube. Effective gastric decompression decreases nausea, distention, and the risk of vomiting and aspiration. Longer nasoenteric tubes, with tips placed into the jejunum or ileum, were favored in the past but are rarely used today, as they are associated with higher complication rates than NG tubes, with no proven greater efficacy in several studies.

The standard therapy for *complete* small bowel obstruction has generally been expeditious surgery, with the dictum that "the sun should never rise and set on a complete bowel obstruction."

Recently, however, some have advocated nonoperative approaches in management of these patients, provided closedloop obstruction is ruled out and there is no evidence of intestinal ischemia. Such patients need to be observed closely and undergo serial exams. The rationale for those favoring early surgical intervention is to minimize the risk for bowel strangulation, which is associated with an increased risk for morbidity and mortality.

Clinical signs and currently available laboratory tests and imaging studies do not reliably permit the distinction between patients with simple obstruction and those with strangulated obstruction prior to the onset of irreversible ischemia.

Therefore, the goal is to operate before the onset of irreversible ischemia. Others note, however, that a period of observation and nasogastric decompression, provided no tachycardia, tenderness, or an increase in white cell count is noted, are appropriate.

However, conservative therapy, in the form of NG decompression and fluid resuscitation, is commonly recommended in the initial recommendation for:

1. Partial small bowel obstruction
2. Obstruction occurring in the early postoperative period
3. Intestinal obstruction due to Crohn's disease
4. Carcinomatosis

In *partial obstruction*, progression to strangulation is unlikely to occur, and an attempt at nonoperative resolution is warranted. Nonoperative management has been documented to be successful in 65% to 81% of patients with partial small bowel obstruction. Of those successfully treated nonoperatively, only 5% to 15% have been reported to have symptoms that were not substantially improved within 48 hours after initiation of therapy. Therefore, most patients with partial small bowel obstruction whose symptoms do not improve within 48 hours after initiation of nonoperative therapy should undergo surgery.

In a recent study, using the National Inpatient Sample, this principle was further highlighted. The authors concluded that a 2-day limit of watchful waiting before surgery is not associated with an increase in mortality or postoperative morbidity, although inpatient costs were higher.

Patients undergoing nonoperative therapy should be closely monitored for signs suggestive of peritonitis, the development of which would mandate urgent surgery. As stated before, the administration of hypertonic water-soluble contrast agents, such as Gastrografin used in upper gastrointestinal (GI) and small bowel follow-through examinations, causes a shift of fluid into the intestinal lumen, thereby increasing the pressure gradient across the site of obstruction. This effect may accelerate resolution

of partial small bowel obstruction; however, there is less evidence that administration of water-soluble contrast agents increases the probability that an episode of bowel obstruction will be successfully managed nonoperatively. Obstruction presenting in the *early postoperative period* has been reported to occur in 0.7% patients undergoing laparotomy. Patients undergoing pelvic surgery, especially colorectal procedures, have the greatest risk for developing early postoperative small bowel obstruction. The presence of obstruction should be considered if symptoms of intestinal obstruction occur after the initial return of bowel function or if bowel function fails to return within the expected 3 to 5 days after abdominal surgery. Plain radiographs may demonstrate dilated loops of small intestine with air-fluid levels but are interpreted as normal or nonspecific in up to a third of patients with early postoperative obstruction. CT scanning or a small bowel series is often required to make the diagnosis. Obstruction that occurs in the early postoperative period is usually partial and only rarely is associated with strangulation. Therefore, a period of extended nonoperative therapy (2–3 weeks) consisting of bowel rest, hydration, and total parental nutrition (TPN) administration is usually warranted. However, if complete obstruction is demonstrated or if signs suggestive of peritonitis are detected, expeditious reoperation should be undertaken without delay.

Crohn's disease as a cause of small bowel obstruction .Twenty-five to thirty-three percent of patients with a history of cancer who present with small bowel obstruction have adhesions as the etiology of their obstruction and therefore should not be denied appropriate

therapy. Even in cases in which the obstruction is related to recurrent malignancy, palliative resection
or bypass can be performed. Patients with obvious carcinomatosis pose a difficult challenge, given their limited prognosis. Management must be tailored to an individual patient's prognosis and desires, and relief of the obstruction may be best achieved by a bypass procedure, avoiding a potentially difficult bowel resection.
The operative procedure performed for small bowel
obstruction varies according to the etiology of the obstruction.
For example, adhesions are lysed, tumors are resected,
and hernias are reduced and repaired. Regardless of the etiology, the affected intestine should be examined, and nonviable bowel resected. Criteria suggesting viability are normal color, peristalsis, and marginal arterial pulsations. Usually, visual inspection alone is adequate in judging viability. In borderline cases, a Doppler probe may be used to check for pulsatile flow to the bowel, and arterial perfusion can be verified by visualizing intravenously administered fluorescein dye in the bowel wall under ultraviolet illumination. However, neither technique
has been found to be superior to clinical judgment. In general, if the patient is hemodynamically stable, short lengths of bowel of questionable viability should be resected and primary anastomosis of the remaining intestine performed. However, if the
viability of a large proportion of the intestine is in question, a concerted effort to preserve intestinal tissue should be made. In such situations, the bowel of uncertain viability should be left intact and the patient re-explored in 24 to 48 hours in a "secondlook" operation.

At that time, definitive resection of nonviable bowel is completed. Successful laparoscopic surgery for bowel obstruction is being reported with greater frequency. Those who undergo successful laparoscopic procedure have a quicker recovery, less complications, and lower costs. Since distended loops of bowel can interfere with adequate visualization, early cases of proximal small bowel obstruction that are likely due to a single adhesive band are best suited for this approach. Presence of bowel distention and multiple adhesions can cause these procedures to be difficult and potentially hazardous. Conversion rate to open surgery is between 17% and 33%.
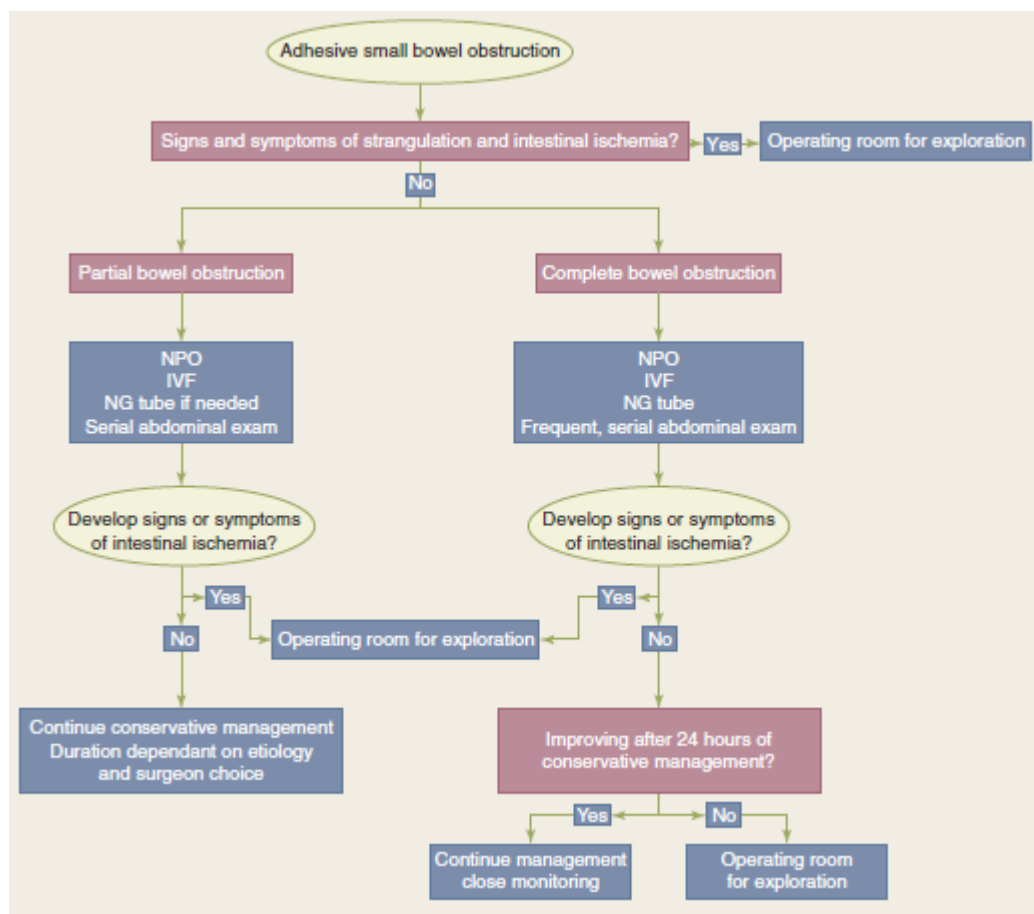
Outcomes

Prognosis is related to the etiology of obstruction. The majority of patients who are treated conservatively for adhesive small bowel obstruction do not require future readmissions; less than 20% of such patients will have a readmission over the subsequent 5 years with another episode of bowel obstruction.

The perioperative mortality rate associated with surgery for nonstrangulating small bowel obstruction is less than 5%, with most deaths occurring in elderly patients with significant comorbidities. Mortality rates associated with surgery for strangulating obstruction range from 8% to 25%. Considering the frequency of small bowel obstruction and the varied degree of clinical severity and presentation, there is often no consistency as to whether the patient is admitted to the medical or surgical service, and further variability in whether and when surgery is consulted. Recent studies have shown that a standard hospital-wide policy can help improve care of patients with bowel obstruction, reducing their time to surgery and shorteningtheir length of hospital stay.24

Prevention

With adhesive small bowel obstruction representing a large therapeutic burden, prevention of postoperative adhesions has become an area of great interest. Good surgical technique, careful handling of tissue, and minimal use and exposure of peritoneum to foreign bodies form the cornerstone of adhesion prevention. These measures alone are often inadequate. In patients undergoing colorectal or

pelvic surgery, hospital readmission rates of greater than 30% over the subsequent 10 years have been reported for adhesive small bowel obstruction.

Use of laparoscopic surgery, when possible, has been strongly promoted. A recent study using the Swedish Inpatient Register has shown that, compared to laparoscopy, open surgery is associated with a four-fold increase in risk of small bowel obstruction within 5 years of the index procedure even after accounting for other risk factors such as age, comorbidity, and previous abdominal surgery.

In those undergoing open surgery, several strategies for adhesion prevention have been tried; however, the only therapy that has shown some success has been the use of hyaluronan based agents, such as Seprafilm. The use of this barrier has been clearly shown to reduce the incidence of postoperative bowel adhesions; however, its effect in actually reducing the incidence of small bowel obstruction remains less well defined.

 The use of these products is often left to the discretion of the surgeon and the clinical context. Wrapping of an intestinal anastomosis with the material may be associated with increased leak rates and is generally discouraged.

ILEUS AND OTHER DISORDERS OF INTESTINAL MOTILITY

Ileus and intestinal pseudo-obstruction designate clinical syndromes caused by impaired intestinal motility and are characterized by symptoms and signs of intestinal obstruction in the absence of a lesion-causing mechanical obstruction.

Ileus is a major cause of morbidity in hospitalized patients. Postoperative ileus is the most frequently implicated cause of delayed discharge following abdominal operations.

Ileus is a temporary motility disorder that is reversed with time as the inciting factor is corrected. In contrast, chronic intestinal pseudo-obstruction comprises a spectrum of specific disorders associated with irreversible intestinal dysmotility.

Pathophysiology

The most frequently encountered factors are abdominal operations, infection and inflammation, electrolyte abnormalities, and drugs.

Following most abdominal operations or injuries, the motility of the GI tract is transiently impaired. Among the proposed mechanisms responsible for this dysmotility are surgical stress-induced sympathetic reflexes, inflammatory response mediator release, and anesthetic/analgesic side effects, each of which can inhibit intestinal motility. The return of normal motility generally follows a characteristic temporal sequence, with small-intestinal motility returning to normal within the first 24 hours after laparotomy and gastric and colonic motility returning to normal by 48 hours and 3 to 5 days,

respectively. Because small bowel motility is returned before colonic and gastric motility, listening for bowel sounds is not a reliable indicator that ileus has fully resolved. Functional evidence of coordinated GI motility in the form of passing flatus or a bowel movement is a more useful indicator. Resolution of ileus may be delayed in the presence of other factors capable of inciting ileus such as the presence of intra-abdominal abscesses or electrolyte abnormalities.

Chronic intestinal pseudo-obstruction can be caused by a large number of specific abnormalities affecting intestinal smooth muscle, the myenteric plexus, or the extraintestinal nervous system. Visceral myopathies constitute a group of diseases characterized by degeneration and fibrosis of the intestinal muscularis propria. Visceral neuropathies encompass a variety of degenerative disorders of the myenteric and submucosal plexuses. Both sporadic and familial forms of visceral myopathies and neuropathies exist. Systemic disorders involving the smooth muscle, such as progressive systemic sclerosis and progressive muscular dystrophy, and neurologic diseases, such as Parkinson's disease, can also be complicated by chronic intestinal pseudo-obstruction. In addition, viral infections, such as those associated with cytomegalovirus andEpstein-Barr virus, can cause intestinal pseudo-obstruction.

Ileus: common etiologies

Abdominal surgery
Infection
    Sepsis
    Intra-abdominal abscess
    Peritonitis
    Pneumonia
Electrolyte abnormalities
    Hypokalemia
    Hypomagnesemia
    Hypermagnesemia
    Hyponatremia
Medications
    Anticholinergics
    Opiates
    Phenothiazines
    Calcium channel blockers
    Tricyclic antidepressants
Hypothyroidism
Ureteral colic
Retroperitoneal hemorrhage
Spinal cord injury
Myocardial infarction
Mesenteric ischemia

Chronic intestinal pseudo-obstruction: etiologies

Primary Causes
- Familial types
  - Familial visceral myopathies (types I, II, and III)
  - Familial visceral neuropathies (types I and II)
  - Childhood visceral myopathies (types I and II)
- Sporadic types
  - Visceral myopathies
  - Visceral neuropathies

Secondary Causes
- Smooth muscle disorders
  - Collagen vascular diseases (e.g., scleroderma)
  - Muscular dystrophies (e.g., myotonic dystrophy)
  - Amyloidosis
- Neurologic disorders
  - Chagas disease, Parkinson's disease, spinal cord injury
- Endocrine disorders
  - Diabetes, hypothyroidism, hypoparathyroidism
- Miscellaneous disorders
  - Radiation enteritis
- Pharmacologic causes
  - E.g., phenothiazines and tricyclic antidepressants
- Viral infections

Clinical Presentation

The clinical presentation of ileus resembles that of small bowelobstruction. Inability to tolerate liquids and solids by mouth, nausea, and lack of flatus or bowel movements are the most common symptoms. Vomiting and abdominal distension may occur.

Bowel sounds are characteristically diminished or absent, in contrast to the hyperactive bowel sounds that usually accompany mechanical small bowel obstruction. The clinical manifestations of chronic intestinal pseudo-obstruction include variable degrees of nausea and vomiting and abdominal pain and distention.

Diagnosis

Routine postoperative ileus should be expected and requires no diagnostic evaluation. If ileus persists beyond 3 to 5 days postoperatively or occurs in the absence of abdominal surgery, diagnostic evaluation to detect specific underlying factors capable of inciting ileus and to rule out the presence of mechanical obstruction is warranted.

Patient medication lists should be reviewed for the presence of drugs, especially opiates, known to be associated with impaired intestinal motility. Measurement of serum electrolytes may demonstrate electrolyte abnormalities commonly associated with ileus. Abdominal radiographs are often obtained, but

the distinction between ileus and mechanical obstruction may be difficult based on this test alone. In the postoperative setting, CT scanning is the test of choice because it can demonstrate the presence of an intra-abdominal abscess or other evidence of peritoneal sepsis that may be causing ileus and can exclude the presence

of complete mechanical obstruction. Distinction of postoperative ileus from early postoperative obstruction can be difficult but is helpful in developing the appropriate management plan.

The diagnosis of chronic pseudo-obstruction is suggested by clinical features and confirmed by radiographic and manometric studies. Diagnostic laparotomy or laparoscopy with full-thickness biopsy of the small intestine may be required to establish the specific underlying cause.

Therapy

The management of ileus consists of limiting oral intake and correcting the underlying inciting factor. If vomiting or abdominal distention is prominent, the stomach should be decompressed using a nasogastric tube. Fluid and electrolytes should be administered intravenously until ileus resolves. If the duration of ileus is prolonged, TPN may be required.

Given the frequency of postoperative ileus and its financial impact, a large number of investigations have been conducted to define strategies to reduce its duration. Although often recommended, the use of early ambulation and routine nasogastric intubation has not been demonstrated to be associated with earlier resolution of postoperative ileus. There is some evidence that early postoperative feeding protocols and are generally well tolerated, reduce postoperative ileus, and can result in a shorter hospital stay. The administration of nonsteroidal anti-inflammatory drugs such as ketorolac and concomitant reductions in opioid dosing have been shown to reduce the duration of ileus in most studies. Similarly, the use of perioperative thoracic epidural anesthesia/analgesia with regimens containing local anesthetics combined with limitation or elimination of systemically administered opioids has been shown to reduce duration of postoperative ileus, although they have not reduced the overall length of hospital stay. Interestingly, recent data have suggested that limiting intra- and postoperative fluid administration can also result in reduction of postoperative ileus and shortened hospital stay.

Most other pharmacologic agents, including prokinetic agents, are associated with efficacy-toxicity profiles that are too unfavorable to warrant routine use. Recently, administration
of alvimopan, a novel peripherally active mu-opioid receptor antagonist with limited oral absorption, has been shown to reduce duration of postoperative ileus, hospital stay.
focuses on palliation of symptoms as well as fluid, electrolyte, and nutritional management. Surgery should be avoided if at all possible. No standard therapies are curative or delay the natural history of any of the specific disorders causing intestinal pseudo-obstruction. Prokinetic agents, such as metoclopramide and erythromycin, are associated with poor efficacy. Cisapride has been associated with palliation of symptoms;
however, because of cardiac toxicity and reported deaths, this agent is restricted to compassionate use in the United States.
Patients with refractory disease may require strict limitation of oral intake and long-term TPN administration. Despite these measures, some patients will continue to have severe abdominal pain or such copious intestinal secretions that vomiting and fluid and electrolyte losses remain substantial.
These patients may require a decompressive gastrostomy or an extended small bowel resection to remove abnormal intestine.

Measures to reduce postoperative ileus

Intraoperative measures
- Minimize handling of the bowel
- Laparoscopic approach, if possible
- Avoid excessive intraoperative fluid administration

Postoperative measures
- Early enteral feeding
- Epidural anesthesia, if indicated
- Avoid excessive intravenous fluid administration
- Correct electrolyte abnormalities
- Consider mu-opioid antagonists

Chronic intestinal pseudo-obstruction: etiologies

Primary Causes
- Familial types
  - Familial visceral myopathies (types I, II, and III)
  - Familial visceral neuropathies (types I and II)
  - Childhood visceral myopathies (types I and II)
- Sporadic types
  - Visceral myopathies
  - Visceral neuropathies

Secondary Causes
- Smooth muscle disorders
  - Collagen vascular diseases (e.g., scleroderma)
  - Muscular dystrophies (e.g., myotonic dystrophy)
  - Amyloidosis
- Neurologic disorders
  - Chagas disease, Parkinson's disease, spinal cord injury
- Endocrine disorders
  - Diabetes, hypothyroidism, hypoparathyroidism
- Miscellaneous disorders
  - Radiation enteritis
- Pharmacologic causes
  - E.g., phenothiazines and tricyclic antidepressants
- Viral infections