

A Systematic Review of the Clinical Presentation, Diagnosis, and Treatment of Small Bowel Obstruction

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Abstract

Purpose of Review This study aimed to systematically review small bowel obstruction (SBO), focusing on recent changes in diagnosis/therapy.

Recent Findings SBO incidence is about 350,000/annum in the USA. Etiologies include adhesions (65%), hernias (10%), neoplasms (5%), Crohn's disease (5%), and other (15%). Bowel dilatation occurs proximal to obstruction primarily from swallowed air and secondarily from intraluminal fluid accumulation. Dilatation increases mural tension, decreases mucosal perfusion, causes bacterial proliferation, and decreases mural tensile strength that increases bowel perforation risks. Classical clinical tetrad is abdominal pain, nausea and emesis, abdominal distention, and constipation-to-obstipation. Physical exam may reveal restlessness, acute illness, and signs of dehydration and sepsis, including tachycardia, pyrexia, dry mucous membranes, hypotension/orthostasis, abdominal distention, and hypoactive bowel sounds. Severe direct tenderness, involuntary guarding, abdominal rigidity, and rebound tenderness suggest advanced SBO, as do marked leukocytosis, neutrophilia, bandemia, and lactic acidosis. Differential diagnosis includes postoperative ileus, narcotic bowel, colonic pseudo-obstruction, mesenteric ischemia, and large bowel

obstruction. Medical resuscitation includes intravenous hydration, correcting electrolyte abnormalities, intravenous antibiotics, nil per os, and nasogastric suction. Abdominal CT with oral and intravenous gastrografin contrast is highly sensitive and specific in detecting/characterizing SBO. SBO usually resolves with medical therapy but requires surgery, preferentially by laparoscopy, for unremitting total obstruction, bowel perforation, severe ischemia, or clinical deterioration with medical therapy. Overall mortality is 10% but increases to 30% with bowel necrosis/perforation.

Summary Key point in SBO is early diagnosis, emphasizing abdominal CT; aggressive medical therapy including rehydration, antibiotics, and nil per os; and surgery for failed medical therapy.

Keywords Small bowel obstruction (SBO) · Postoperative adhesions · Mechanical obstruction · Abdominal surgery · Laparoscopy

Introduction

Review of small bowel obstruction (SBO) is important and timely. First, SBO is a relatively important cause of hospital admissions, patient morbidity, and mortality. SBO imposes a substantial economic burden on the American health care system accounting for about 300,000–350,000 hospital admissions annually, at a direct cost of >\$3 billion per annum [1, 2]. It comprises about 15% of all acute surgical gastrointestinal admissions [3, 4] and about 15% of all emergency admissions for abdominal pain [5]. SBO causes about 30,000 deaths per annum and commonly results in decreased quality of life, mostly from chronic postoperative pain or obstructive symptoms [6]. Second, this subject not only pertains to gastroenterologists and gastrointestinal surgeons but increasingly

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pertains to gastrointestinal radiologists and intensivists involved in the diagnosis and management of SBO. Third, the algorithm for management of SBO has recently changed with recognition of the paramount diagnostic role of abdominal computed tomography (CT) and the increasing role of laparoscopic surgery rather than open surgery, with 29% of cases of SBO from adhesions (aSBO) currently treated by laparoscopy [7]. Proper management of SBO is important to avoid unnecessary surgery for SBO that should be managed medically to decrease patient morbidity and to avoid delays in necessary surgery to decrease mortality. This article reviews the pathophysiology, clinical presentation, radiologic findings, therapy, and prognosis of SBO, with particular emphasis on recent developments.

Methods

A systematic computerized search was conducted using PubMed for publications in peer-reviewed journals with the following subject headings or keywords: ["small bowel" and "obstruction"] OR ["management" and "bowel obstruction"] OR ["treatment" and "bowel obstruction"] OR "adhesions" OR "laparoscopic adhesiolysis" OR "small intestinal obstruction" OR "SBO." Both authors reviewed all the identified articles. Articles were incorporated into this review by consensus. Recently published studies, from 2010 to 2016, were preferentially selected. Articles were also prioritized according to the following: prospective original studies > retrospective original studies > clinical series > case reports. Review articles were also selected, with high priority assigned to recent systematic or comprehensive reviews.

Epidemiology

Despite advances in laparoscopy, abdominal adhesions remain the most common etiology of SBO, comprising 60–70% of the cases [8, 9]. Adhesion-related complications account for 20% of readmissions within 1 year and 30% of readmissions within 10 years after abdominal surgery [8, 10]. Intra-abdominal adhesions are fibrous bands, consequent to postoperative inflammation [11]. Adhesions can begin forming within hours after abdominal surgery and can cause SBO from weeks to many years after abdominal surgery [12].

Adhesion formation is related to wound healing, which is significantly affected by surgical technique and tissue inflammation from tissue exposure to intestinal contents, foreign material, and desiccation [13, 14]. Laparoscopic surgery reduced the incidence of adhesions after gastrointestinal surgery by 25%, reduced the adhesion severity score by 1.7 points, and reduced the need for surgery for aSBO as compared to open surgery [14–16] because laparoscopy involves smaller incisions, decreases the risk of contamination, and causes less

tissue trauma, intraoperative blood loss, and tissue desiccation [17].

While all abdominal surgeries can produce adhesions, surgical site is an important determinant of risk. Surgery on the lower gastrointestinal tract or other lower abdominal sites increases the risk of aSBO, as compared to surgery at other abdominal locations [18, 19]. In a meta-analysis incorporating 196 publications, the incidence of aSBO after all types of abdominal surgery was 2.4% [13]. The rate of aSBO ranges from 0.05% for cesarean section, to 1% for appendectomy, and to 10% for colorectal surgery [18, 20, 21].

Based on a recent European study, total costs for one admission for aSBO treated surgically are \$16,972 ± \$2615 but for aSBO treated medically are \$2370 ± \$275 ($p < 0.001$ for cost difference for operative vs. medical therapies) [22].

About 10–15% of SBO are due to abdominal hernias [9, 23]. External hernias, such as femoral, inguinal, and incisional hernias, much more commonly cause SBO than internal hernias [24]. About 5–7% of SBOs are due to Crohn's disease [25]. Neoplasms account for 5–10% of SBOs [26]. Neoplasms usually cause SBO by extrinsic compression, especially from colon or ovarian cancer [27, 28]. The etiologies of SBO are listed in Table 1.

Pathophysiology

Normal small intestinal functions include absorption of nutrients, electrolytes, and water from ingested food. In SBO, the small bowel dilates proximal to an obstruction primarily from accumulation of swallowed air and secondarily from accumulation of intestinal fluids, as first demonstrated by Wangenstein [29]. Intestinal stasis results in further intestinal gas from bacterial proliferation and fermentation of ingested food. These derangements cause mural edema, loss of intestinal absorptive functions, and fluid sequestration in the lumen [30]. Transudative loss of fluid from the intestinal lumen into the peritoneal cavity may occur. Emesis from proximal SBO additionally causes systemic loss of fluids resulting in hypovolemia and electrolyte abnormalities. If bowel dilation is severe or persistent, intestinal mural perfusion may decrease from increased intramural pressure and hypovolemia, resulting in progressive bowel ischemia and necrosis [31]. Mucosal ischemia promotes intramural bacterial invasion.

Progressive bowel dilatation weakens the bowel wall tensile strength from mural thinning, while increasing intramural tension according to Laplace's Law. These effects resemble those of blowing up a party balloon: the balloon becomes more firm from increased intra-balloon pressure as its wall becomes thinner as indicated by the balloon becoming more transparent. These effects together with decreased wall integrity from ischemia promote bowel perforation, the risk of which increases with duration of failed medical therapy and clinical deterioration. Bowel mucosa is highly vulnerable to

Table 1 Etiologies of small bowel obstruction (SBO)

Extrinsic causes

Adhesions

External hernias: femoral, inguinal, midventral, periumbilical, and incisional

Internal hernias: obturator, paraduodenal, transmesenteric, and transomental (acquired secondary to prior surgery)

Primary abdominopelvic cancers: most commonly extrinsic compression by colon cancer or ovarian cancer

Metastasis: most commonly from extrinsic compression from metastases

Volvulus

Chronic mesenteric ischemia with stricture

Intrinsic causes

Inflammatory causes: diverticulitis, appendicitis, Crohn's disease

Malignant causes: primary small bowel cancers, metastatic cancer to small bowel

Radiation enteritis

Gallstone ileus

Endometriosis

IgG4-related sclerosing mesenteritis

Rare causes: Meckel's diverticulum, hamartoma, bezoars, ingested foreign bodies, intussusception, massive hookworm infestation

Iatrogenic—migration of endoscopically placed stents, deflated intragastric balloons and gastrostomy tubes, impaction of video capsules

Post-surgical: Roux-en-Y gastric bypass (RYGB)

Congenital—anomalous congenital bands manifesting at later age

Medications: strictures induced by NSAIDs, strictures induced by enteric coated potassium chloride tablets

Internal hernias: obturator hernias, paraduodenal, transmesenteric, and transomental

[21, 24]

ischemia because it is perfused by end arteries and has high metabolic activity. A collateral circulation provided by marginal vessels helps bypass ischemia from an occluded local vessel but does not defend against generalized small bowel ischemia, as occurs with SBO. Perforation usually occurs at a bowel segment that is particularly dilated and particularly compromised by ischemia. Perforation is a surgical emergency. It rapidly causes peritonitis and overwhelming sepsis.

Clinical Presentation

The clinical presentation varies somewhat depending upon severity, location, duration, and etiology of the obstruction. More severe SBO manifests more classic and specific clinical findings. The classic clinical tetrad is colicky abdominal pain, nausea and emesis, abdominal distention, and progressive constipation-to-obstipation. The nausea and emesis may be acute or subacute in onset and may be bilious, non-bilious, or feculent, depending on the location and severity of the obstruction. Feculent vomiting strongly suggests high-grade SBO. The abdominal pain can also be variably characterized as crampy, constant, or intermittent. The abdominal pain becomes more intense and unremitting if bowel ischemia or perforation supervenes. Pyrexia is often an ominous sign heralding mucosal ischemia and sepsis. Attention is directed in the medical history on prior abdominal surgeries, indications for the prior abdominal surgeries, prior SBO, its treatment, prior abdominal radiation, and narcotic use [3, 32].

Patient confinement to strict bed rest can contribute to bowel dilatation by promoting hypoperistalsis, as occurs after certain orthopedic surgeries. A history of Crohn's disease suggests that the SBO is from bowel stricture from Crohn's disease.

Physical examination may reveal a restless, acutely ill, patient with signs of dehydration and sepsis, including tachycardia, pyrexia, dry mucous membranes, poor skin turgor, and hypotension or orthostasis. Abdominal examination may reveal moderate abdominal distention with proximal SBO or relatively severe abdominal distention with distal SBO. Bowel sounds may initially be hyperactive due to muscular propulsive reflexes designed to overcome the obstruction and are often initially high-pitched (tinkling) but become hypoactive-to-absent with advanced SBO because of intestinal muscular fatigue. Inspection rarely reveals visible peristalsis, and auscultation rarely demonstrates borborygmi (audible rushes) with early obstruction. Air-filled bowel loops produce abdominal tympany, while liquid-filled loops produce abdominal dullness. Direct tenderness on abdominal palpation frequently occurs; the location of the tenderness is variable and does not correlate well with the location of bowel obstruction. Surgical scars and external hernias should be noted, and incarceration of external hernias should be excluded by physical examination. Malignant obstruction is suggested by an abdominal mass, hepatomegaly, and lymphadenopathy, including abnormal periumbilical, inguinal, or right supraclavicular (Virchow's) nodes. Rectal examination may demonstrate fecal impaction or a rectal mass that causes rectal obstruction. Signs

that the SBO is complicated by transmural ischemia or bowel perforation (peritonitis) include rebound tenderness, voluntary or involuntary guarding, and abdominal rigidity.

Laboratory abnormalities are generally non-specific. These abnormalities include hemoconcentration and electrolyte derangements from vomiting and transudation of fluid. The blood urea nitrogen (BUN) and creatinine levels may increase from prerenal azotemia consequent to third spacing of fluids. Marked leukocytosis, neutrophilia, a “left” shift to immature leukocyte forms especially band forms, and otherwise unexplained metabolic acidosis often indicate sepsis. In particular, lactic acidosis may herald impending intestinal ischemia. Intestinal fatty acid binding protein, which is released by necrotic enterocytes, may constitute a useful marker of bowel ischemia [33]. Bowel perforation may produce marked hyperamylasemia.

A beta-human chorionic gonadotropin (HCG) test should be performed to determine pregnancy status in women of child-bearing age. A coagulation profile, including international normalized ratio, platelet count, and partial thromboplastin time, should be determined because of the potential need for urgent surgery [24]. Patients with significant ascites should undergo diagnostic paracentesis to exclude peritonitis. With peritonitis from bowel perforation, the ascitic fluid is typically turbid and has a neutrophil count $>250/\text{mm}^3$, total protein $>1 \text{ g/dl}$, and glucose level $<50 \text{ mg/dl}$ [24]. The ascitic fluid should also be sent for bacterial culture and sensitivity.

Differential Diagnosis

The differential diagnosis of SBO includes the following four other diseases that produce prominent, acute, bowel dilatation.

1. *Postoperative (adynamic) ileus* is an acute functional “obstruction” due to intestinal hypoperistalsis from subtle bowel injury during abdominal surgery that manifests soon after the surgery. The bowel progressively dilates because of inability to pass flatus and stools due to the hypoperistalsis. Postoperative pain, administered analgesia, and relative immobility can all contribute to the ileus. Narcotics should be reduced to the lowest possible dose to promote bowel motility and prevent a narcotic bowel.
2. *Narcotic bowel* is caused by excessive administration of narcotics which tend to cause constipation by decreasing the amplitude of intestinal muscular contractions, interfering with the normal coordination of muscular contractions necessary for peristalsis, promoting hard stool by increasing water absorption from bowel lumen, and increasing the resting tone of the anal sphincter. Narcotics often contribute to postoperative ileus and colonic pseudo-obstruction. The diagnosis is usually suggested by the history of narcotic use and its high dosage. The primary treatment is aggressively reducing the dosage and frequency of narcotic administration.
3. In *acute colonic pseudo-obstruction* (Ogilvie’s syndrome), intestinal transit is functionally delayed without mechanical obstruction because of attenuated or uncoordinated colonic muscular contractions. This produces severe bowel dilatation in the colon, but some small bowel dilatation may occur. No transition point exists in colonic pseudo-obstruction. The patient nearly always has risk factors for colonic pseudo-obstruction including administration of antikinetic drugs such as calcium channel antagonists, anticholinergic drugs, phenothiazines, or anti-Parkinsonian medications; severe electrolyte disturbances; neurologic disorders such as Parkinsonism or diabetic neuropathy; thyroid disorders; and major acute medical illnesses such as myocardial infarction or recent surgery such as orthopedic surgery [34]. Patients with SBO are generally more acutely ill than patients with colonic pseudo-obstruction for the same degree of abdominal distention. SBO is differentiated from colonic pseudo-obstruction by predominantly small bowel dilatation and an absence of rectal air [24].

Radiologic studies in these three disorders generally reveal no transition point between dilated and collapsed bowel. These three disorders generally have a relatively benign outcome in patients without major comorbidities and less often require surgery than SBO.

4. In *acute mesenteric ischemia* or *ischemic colitis*, the primary event is bowel ischemia and bowel dilatation is secondary to the ischemia. Contrariwise, in SBO, the bowel ischemia is secondary to the bowel dilatation. Most patients with mesenteric ischemia present with prominent symptoms, especially abdominal pain, which is classically out of proportion to the paucity of clinical signs. They may have self-limited rectal bleeding. They usually have risk factors for bowel ischemia, including risk factors for embolism of atrial fibrillation, other cardiac arrhythmias, left atrial thrombus, and endocarditis; risk factors for thrombus formation of hypertension, hyperlipidemia, diabetes, and cigarette smoking; hypercoagulable states such as protein C or protein S deficiency, and anticardiolipin syndrome; and the risk factor of hypotension for non-occlusive mesenteric ischemia (NOMI) [35, 36].

Mechanical obstruction is classified as SBO versus large bowel obstruction according to the obstructed organ. Air-filled small bowel loops have a central abdominal location as opposed to the “picture frame” arrangement of colonic loops, have a narrower caliber even when dilated, and have valvulae conniventes that extend across the entire luminal diameter as opposed to the intrahaustral colonic folds that

incompletely extend across the luminal diameter. In SBO, only the small bowel is dilated; while in large bowel obstruction, both the small bowel and large bowel are dilated.

Initial Medical Resuscitation

Patients should be resuscitated and medically stabilized, including attention to major comorbidities. This is critical for medical management and for management before contemplated surgery. Patients should be maintained at nil per os (NPO) because food in the gut places greater metabolic stress on potentially already ischemic gut mucosa, promotes bacterial proliferation, and provides a substrate for contaminated intraperitoneal leakage if bowel perforation supervenes. Two, wide-bore, intravenous (IV) lines should be secured, and IV crystalloid fluids, consisting of either normal saline or lactated Ringer's solution, should be administered to rapidly restore euvolemia. Antiemetics should be administered intravenously to reduce nausea and emesis and prevent aspiration. Electrolyte disorders should be corrected; disorders in potassium, calcium, magnesium, or bicarbonate levels may contribute to hypoperistalsis. Blood cultures should be obtained if sepsis is suspected. Broad-spectrum bactericidal antibiotics are required to effectively treat gram-negative and anaerobic bacteria if sepsis is suspected. The most commonly recommended regimen for suspected intra-abdominal infection is cephalosporins or fluoroquinolones together with metronidazole [37]. Antibiotics should be administered intravenously due to uncertain enteral absorption in the face of potentially compromised bowel. Septic patients should have a Foley catheter inserted to continuously monitor urinary output. Narcotics should be avoided because they retard gastrointestinal peristalsis and can mask critical clinical findings of abdominal pain, rebound tenderness, and abdominal rigidity, which comprise early signs of bowel necrosis and perforation.

SBO is primarily a surgical condition that is best managed on the surgical service. A study of 107,603 admissions for aSBO reported management on the medical service was an independent risk factor for longer hospitalization, greater inpatient costs, and a higher rate of 30-day readmission following non-operative management. Similarly, management on the medical service of patients eventually requiring surgery was associated with delayed surgery, longer hospitalizations, greater inpatient costs, higher 30-day mortality, and higher rates of 30-day readmission [38]. Thus, regardless of operative or non-operative management, patients benefit from admission to the surgical service, with medical support to manage medical comorbidities.

Acutely ill patients with suspected SBO should be managed by a team of specialists, including a surgeon to monitor the abdominal findings and treat the surgical complications; a gastroenterologist to assist in the diagnosis and administration of medical and colonoscopic therapy for volvulus,

intussusception, or colonic pseudo-obstruction; an intensivist to manage metabolic and hemodynamic abnormalities in medically metastable or unstable patients; and a gastrointestinal radiologist for specialized imaging [24]. Patients with clinically evident deterioration or impending peritonitis during medical observation should undergo emergency surgery, rather than radiological studies that would unnecessarily delay surgery [32].

Once SBO is diagnosed, the dilated stomach and proximal small bowel is decompressed by placing a nasogastric tube or long nasoenteral tube either blindly or fluoroscopically to primarily remove air to prevent clinical deterioration and to secondarily remove fluid to decompress dilated bowel and prevent emesis [29]. Decompression using a 300-cm-long nasoenteral tube can effectively relieve obstructive symptoms, help avoid emergency surgery, and resolve obstruction [39, 40]. Before Wangenstein's classic studies in the early 1930s, bowel obstruction was nearly uniformly fatal because of inattention to bowel dilatation [29].

Abdominal examinations should be performed serially to detect early signs of bowel ischemia, necrosis, and perforation including pyrexia, tachycardia, increasing leukocytosis, hypotension, increasing abdominal pain, and increasing abdominal distention or contrariwise to detect signs of SBO resolution, such as decreasing NG tube output, decreasing abdominal pain, passage of flatus, increasing bowel movements, and decreasing abdominal distention [3].

Abdominal Radiographs

During medical resuscitation and stabilization, abdominal radiographs should be performed as initial radiologic screening for suspected SBO. Plain films are fairly accurate, with 60–93% sensitivity in the diagnosis of SBO, especially when reviewed by experienced radiologists [41]. SBO is more accurately diagnosed if radiographs are obtained in both dependent (supine or prone) and non-dependent (upright or decubitus) positions [41]. However, radiographs cannot reliably determine the site or etiology of obstruction or detect early bowel ischemia [41, 42]. Abdominal radiographs in a patient with SBO typically demonstrate dilatation of small bowel out of proportion to that of the colon. Small bowel dilatation is defined as ≥ 3 cm in diameter [11, 41]. Signs of SBO on upright or left lateral decubitus radiographs include multiple air-fluid levels, air-fluid levels ≥ 2.5 -cm long, differential air-fluid levels (>5 mm difference in heights of individual air-fluid levels) in loops of small bowel, and small amounts of remaining gas trapped within the uppermost part of folds between valvulae conniventes in fluid-filled loops of bowel, in an arrangement that resembles a string of beads and is called the "string of beads" sign [41]. These signs are relatively sensitive and specific for SBO [11, 41]. For the

diagnosis of aSBO, abdominal radiographs have a sensitivity of 79–83%, specificity of 67–83%, and accuracy of 64–82% [43]. Abdominal radiographs with the patient (partially) upright may detect pneumoperitoneum (intraperitoneal “free” air), a reliable radiographic sign of bowel perforation.

Abdominal CT

Abdominal CT plays a paramount role in diagnosing SBO and in detecting radiographic signs of evolving bowel ischemia not detected by plain abdominal radiographs. CT should be performed on *all* patients with suspected SBO, with rare exceptions [32]. It has a sensitivity of 94%, specificity of 96%, and accuracy of 95% in detecting SBO [44]. CT can help determine the etiology of SBO, identify the transition point, distinguish between complete versus partial obstruction, and distinguish between high-grade versus low-grade obstruction. It is more accurate for high-grade or complete obstruction than for low-grade, partial obstruction [45]. Additionally, CT can reliably demonstrate signs of ischemia, necrosis, or perforation including mural thickening, mural enhancement, mesenteric edema, and pneumatosis intestinalis (intramural air) [46, 47]. CT has 63–100% sensitivity and 61–96% specificity in identifying ischemia [48].

Radiological findings suggestive of SBO include dilated gas-filled, proximal small bowel loops; collapsed, gasless, distal small bowel loops; and an abrupt transition point between these bowel segments. Major CT criteria for SBO include small bowel dilatation ≥ 3 cm in diameter without significant colonic dilatation (i.e., <6 cm colonic diameter) and a transition point from dilated to collapsed small bowel. Minor radiologic criteria for SBO include air-fluid levels and a decompressed colon [11•]. CT usually fails to identify bowel adhesions, but their presence is suspected by an abrupt transition from dilated to collapsed bowel loops without an otherwise identified cause at the transition point [11•]. Accuracy of localization of the transition point ranges from 63 to 93%. Multidetector row CT (MDCT) with three-dimensional reconstruction is a relatively new technique that shows promise in improving the diagnosis of the site and etiology of SBO as compared to conventional CT [49].

Gastrografin is a water-soluble, radiopaque solution containing a mixture of 168 g/100 ml of diatrizoate meglumine and 10 g/100 ml of diatrizoate sodium. It is administered orally for abdominal CT if the patient is not vomiting or otherwise by NG tube either at admission or after failed conservative treatment for 48 h for suspected SBO [32, 50•]. Correct NG tube placement should be documented before administering gastrografin via the NG tube to prevent contrast administration into the lungs, either directly or by aspiration, which can be fatal [3, 51]. Gastrografin has both diagnostic and therapeutic effects. Its osmolality is six times that of extracellular

fluid. Oral administration therefore increases the pressure gradient across an obstruction, promotes shifting of fluid into bowel lumen, decreases mural edema, and enhances bowel motility by promoting SBO resolution. Administration of oral gastrografin is safe and reduces the need for surgery, length of stay, and time to resolution of SBO but does not significantly reduce patient morbidity, overall complication rate, or mortality [52]. Gastrografin did not influence the rate of SBO recurrence [50•, 53]. Patient age >65 years, multiple previous laparotomies, and previous abdominal surgery for aSBO are risk factors for unsuccessful management with gastrografin [54].

For abdominal CT, oral contrast is usually ingested between 1 and 4 h before CT to permit opacification of small bowel and passage of contrast into the colon. Passage of oral contrast distally into decompressed bowel excludes complete or high-grade obstruction [11•]. The presence of this contrast in the colon <24 h after administration predicts non-surgical resolution of the SBO, with 92% sensitivity and 93% specificity [50•, 55]. Contrariwise, failure of gastrografin to reach the cecum by 24 h indicates suspected complete bowel obstruction that will likely require surgery. Oral contrast should not be administered in patients with suspected GI perforation because of risks of intraperitoneal leakage. Oral contrast should not be administered for CT scans in patients with suspected advanced SBO with sepsis to avoid delays in performing the test and making the diagnosis. In patients with potential but unlikely bowel perforation, water-soluble oral contrast should be used instead of barium contrast to prevent peritoneal spillage and barium-induced chemical peritonitis [56]. Positive contrast may obscure mural enhancement, which limits evaluation of ischemia, acute inflammation, or an underlying enhancing lesion. Iodinated contrast may be contraindicated in patients with renal insufficiency because this contrast may precipitate renal failure. In these circumstances, CT enterography with neutral oral contrast may be substituted. CT should be performed with IV contrast, in the absence of contraindications, such as contrast allergy or renal insufficiency, to assess mesenteric vessel patency by mural enhancement [42]. Identifying mesenteric branches by contrast-enhanced CT helps detect vascular engorgement or swirling that can occur in certain types of obstruction, such as volvulus [42]. The usual clinical symptoms and signs of mural ischemia, such as pyrexia, rigors, intense abdominal pain, leukocytosis, tachycardia, and metabolic acidosis, and the clinical experience of surgeons are neither sensitive nor specific for the diagnosis of strangulation or requirement for surgery. Performing both unenhanced and contrast-enhanced CT improves test sensitivity, diagnostic confidence, and interobserver agreement in the diagnosis of ischemia. Decreased or absent mural enhancement after administration of IV contrast is a useful, highly specific, indicator of intestinal ischemia (95–100%), despite variable sensitivity of 40–60% [46, 57, 58]. A recent meta-analysis showed that decreased mural

enhancement was more predictive of ischemia than any other single CT sign [59, 60]. This sign was 100% specific and 56% sensitive for bowel ischemia [57–60]. Other CT signs of ischemia include bowel wall thickening and mucosal thumbprinting from intramural edema, inflammation, or hemorrhage; pneumatosis intestinalis from intramural gas produced by bacteria; and streaky mesentery from adjacent inflammatory infiltration as listed in Table 2 [11•, 24]. CT is up to 83% sensitive and 92% specific in detecting intestinal ischemia [48]. CT findings are helpful to diagnose SBO and its complications, but none of these findings are consistently positive for bowel ischemia and they have to be considered with the entire clinical presentation.

“Free” intraperitoneal air (pneumoperitoneum) usually indicates bowel perforation [42]. Coronal and/or sagittal multiplanar reformations help identify the transition point, assess for volvulus, and detect closed-loop obstruction. In patients with known Crohn’s disease, CT enterography is preferred to assess for small bowel mural enhancement and to delineate the contribution of active inflammation to suspected SBO, which may alter the patient management [42].

Common CT Signs

A *transition point*, where bowel loops abruptly change from dilated to collapsed, indicates the site of obstruction [61] (Fig. 1a, b). A transition point in patients with non-strangulated SBO may be a significant predictor of operative management [62], but this finding is controversial [63]. *Complete obstruction* shows severely discrepant luminal diameters between proximal and distal small bowel adjacent to the transition point, with collapse of the distal small bowel and colon, and no passage of air or fluid beyond the transition point. *Incomplete high-grade obstruction* shows moderately discrepant luminal diameters between the proximal and distal small bowel adjacent to the transition point with minimal passage of air or fluid into the distal small bowel and ascending colon. High-grade obstruction is predictive of failure of non-

operative management [64] and a need for early surgery [63]. *Low-grade obstruction* shows mildly discrepant luminal diameters between proximal and distal small bowel adjacent to the transition point, with the passage of air or fluid into the distal small bowel and ascending colon [65].

In the *whirl sign*, both mesenteric vessels and bowel loops are twisted together and the bowel loops in the whirl exhibit luminal narrowing with a “beak” appearance [65]. The *whirl sign* occurs when afferent and efferent bowel loops rotate around a fixed point of obstruction, resulting in tightly wound (twisted) mesentery along the axis of rotation [66]. These twisted loops of bowel and branching mesenteric vessels create swirling strands of soft tissue attenuation within a background of mesenteric fat attenuation that resembles a hurricane on a weather map [66]. The whirl sign is highly suggestive of intestinal volvulus [11•, 66]. A patient with the whirl sign on CT is 25 times more likely than a patient without this sign to require surgery for SBO [67].

Small bowel feces sign (SBFS) refers to the presence of mottled, feculent material, resembling colonic contents, in dilated small bowel immediately proximal to the transition point [42]. Although potentially an incidental finding, it suggests high-grade or chronic obstruction [68].

When associated with small bowel dilation, SBFS may help locate the transition point. Jacobs et al. [69] reported that all patients with SBFS and a small bowel diameter >3 cm had SBO and that the SBFS was immediately proximal to the transition point in 75% of cases. SBFS may also predict non-operative treatment of SBO [70, 71]. *The mesenteric fluid sign* results from venous congestion and transudation of fluid across serosa from mesenteric venous outflow obstruction; the absence of the mesenteric fluid sign decreases the probability of strangulation sixfold [59]. The presence of high-density intra-abdominal fluid (>10 Hounsfield units) on CT may predict the need for surgical intervention [72].

In *closed-loop SBO (CL-SBO)*, a bowel segment is occluded at two points, an arrangement that prevents bowel decompression. In CL-SBO, the closed loops are fluid-filled and gasless because swallowed air cannot enter the closed loop [73]. Patients often have more than one transition point [42]. Common causes of CL-SBO include intestinal volvulus in which a loop of bowel is mechanically compressed at both ends of a twist formed around a single band and an incarcerated hernia in which a loop of bowel is compressed at both ends within a hernial sac [24]. Patients with Roux-en-Y gastric bypass are at increased risk of developing CL-SBO from surgically created rents in the mesentery [11•]. CT findings of CL-SBO include radial distribution of the incarcerated bowel arranged like the rims of a bicycle wheel with mesenteric vessels converging towards a central point of torsion arranged like the spokes in a bicycle wheel, a “coffee bean,” “C,” or “V” loop, and the whirl sign [11•, 24]. While CL-SBO without ischemia can sometimes be managed conservatively, it usually

Table 2 CT findings of intestinal ischemia

1. Mural thickening (>3 mm)
2. Mesenteric edema
3. Fluid in mesentery and/or peritoneal cavity
4. Increased (or decreased) bowel wall enhancement
5. Mesenteric vessel occlusion
6. Mesenteric vein engorgement
7. Whirl sign
8. Closed-loop obstruction or volvulus
9. Pneumatosis intestinalis
10. Mesenteric venous gas
11. Portal venous gas

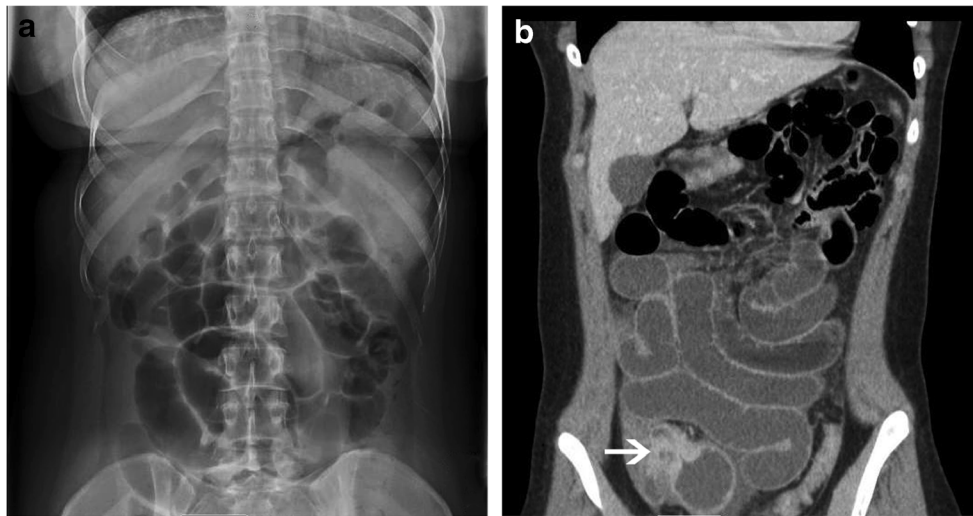


Fig. 1 **a** Supine abdominal plain radiograph in a patient with a flare of chronic ileocolonic Crohn's disease demonstrates numerous distended, air-filled, loops of small bowel, measuring up to 3.4 cm in diameter, without dilated loops of large bowel. These radiographic features are suspicious for small bowel obstruction. **b** Axial image of abdominopelvic computerized tomography (CT) with oral and intravenous contrast in the same patient as

Fig. 1a with a flare of chronic ileocolonic Crohn's disease demonstrates prominent small bowel dilatation, with bowel loops measuring up to 3.4 cm in diameter, proximal to severe luminal narrowing of the terminal ileum (*arrow*). The terminal ileum demonstrates active disease with severe circumferential wall thickening from mural edema and mucosal hyperemia from inflammation

constitutes a surgical emergency because it usually causes progressive vascular occlusion, mural ischemia, and has a high mortality, up to 35%, after delayed diagnosis for 36 h [60, 74].

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is more time-consuming, more expensive, and more variable in image quality than CT [75]. It is primarily available at tertiary hospitals. Patients who have difficulty in holding their breath for MRI are better suited to undergo CT examination. Moreover, CT is preferred in the emergency setting because it detects perforation more accurately and rapidly than MRI and can exclude clinically unsuspected extra-intestinal pathology that may cause an acute abdomen [75]. MRI of the small bowel is indicated for patients with Crohn's disease, for patients with concerns about radiation such as children, patients with contraindications to CT such as pregnant patients, and patients with low-grade SBO [76].

Abdominal Ultrasound

Abdominal ultrasound (USD) is a non-invasive test devoid of radiation exposure. It provides an alternative for pregnant patients and children in whom radiation entails greater risk. USD has a sensitivity and specificity of 83 and 100%, respectively, for diagnosis of SBO but has a low ability to identify the etiology of obstruction because of persistent air within the

bowel lumen in early obstruction [77]. Underlying etiologies often identified by ultrasound include external hernias, intussusception, tumors, ascariasis, superior mesenteric artery syndrome, bezoars, foreign bodies, and Crohn's disease. USD findings that suggest the need for urgent surgery include intraperitoneal free fluid, mural thickness >4 mm, and decreased or absent peristalsis in mechanically obstructed bowel. The sonographic signs of akinetic bowel loops, hyperechoic thickening of attached mesentery, and free peritoneal fluid are typical of strangulation [78].

Non-operative Management

SBO usually resolves spontaneously with conservative management but sometimes is refractory to medical therapy or is complicated by strangulation or bowel perforation, depending on the etiology. Non-operative management may be successful in up to 90% of patients without peritonitis [43]. Emergency laparotomy increases mortality, especially in patients ≥ 80 years old [79]. In a study of 1853 patients from 35 hospitals, the reported 30-day mortality was 15% for all patients versus 25% for patients aged ≥ 80 years old [80].

When to consider surgery versus medical therapy is a diagnostic dilemma for surgeons in relatively stable patients. Delaying surgery for strangulated SBO substantially increases the risk of bowel resection, increases the length of the hospital stay, and substantially increases the mortality (up to 40%) [81, 82]. Even if conservative treatment is successful, the underlying cause of the obstruction still exists [50]. Contrariwise,

patients with uncomplicated SBO who unnecessarily undergo surgery are exposed to inherent surgical risks, longer hospitalizations, and subsequent adhesion-related complications, including recurrent SBO. Radiologic imaging aids this decision process because traditional clinical signs of vascular compromise are often unreliable predictors. CT findings indicative of poor evolution of non-operative management include mesenteric edema, free intra-abdominal fluid, absence of stool in large intestine, or signs of intestinal devascularization [23]. Younger patients (<47 years old) with no previous surgery or known adhesive disease more likely require surgery [81]. Ischemia occurs in 10% of patients with SBO [83]. Mural ischemia is associated with 30% mortality versus 3% mortality in SBO without ischemia. In a retrospective analysis, patients with ≤ 24 h wait before surgery had only a 12% rate of bowel resection, while patients with ≥ 24 h wait before surgery had a 29% rate of bowel resection [81].

Non-operative management should not extend beyond 3–5 days for non-resolving SBO, even in the absence of clinical deterioration [32, 50, 84]. Teixeira et al. [85] reported that surgery delayed >72 h increases mortality threefold, and increases systemic infectious complications by twofold, compared to surgery performed <24 h after presentation. Schraufnagel et al. [86] reported higher rates of complications, bowel resection, longer hospital stay, and increased mortality in patients operated for aSBO after ≥ 4 days. Based on the Delphi consensus study [87], criteria for immediate surgery include strangulated hernia, >10-cm cecal diameter, signs of vascular obstruction, and refractory metabolic acidosis. Surgery should be considered if there are signs of intra-abdominal complications, >18,000 leukocytes/mm³, lactic acidosis, or doubling of creatinine level compared to that on admission.

Uncontrolled Crohn's disease can result in SBO from inflammatory or fibrotic strictures (Fig. 1a, b). Radiological signs of active inflammation in Crohn's disease include mural thickening, stratified mural hyperenhancement, adjacent mesenteric fat stranding, and engorgement of the supplying mesenteric vessels [42, 88]. Inflammatory strictures are potentially reversible and commonly respond to medical therapy, including IV corticosteroids and immunosuppressive agents, and rarely require surgery, while fibrotic strictures from Crohn's disease are unlikely to respond to anti-inflammatory medications. Also, patients with Crohn's disease who had prior abdominal surgery are vulnerable to aSBO; physicians should recognize that this may constitute a life-threatening indication for emergency surgery instead of conservative medical management of the Crohn's disease. Hyperbaric oxygen therapy may have a role in aSBO, but this treatment is controversial [89, 90].

Surgery

After selecting surgery, the surgeon must decide whether to perform open versus laparoscopic surgery. Laparotomy was

the traditional surgical standard for SBO. Bastug et al. [91] first reported laparoscopic resection of a single band causing SBO. Since then, laparoscopic exploration and adhesiolysis are increasingly reported [92]. Laparoscopy is becoming the preferred choice at centers with extensive laparoscopic experience. The frequency of laparoscopic adhesiolysis, as compared to open adhesiolysis, increased by 1.6% per annum from 17.2% in 2006 to 28.7% in 2013 [7]. However, some surgeons are still reluctant to use laparoscopy for SBO because of reduced working space and risk of iatrogenic injuries from bowel distention [93].

Laparoscopy should not be performed in hemodynamically unstable patients with an acute abdomen but is recommended in patients with non-resolving SBO based on a gastrografin study or in stable patients with concern for underlying ischemia [3]. Laparoscopy is associated with favorable short-term and long-term outcomes. It is safe and effective, especially in patients with isolated adhesive bands, simple enteral angulation, foreign body, or tumor, while dense and matted adhesions often require open surgery [94]. In a large, prospective database, the conversion rate from laparoscopy to open surgery was 32% [93], but this rate can vary from 0 to 50% depending on clinical circumstances and surgical skills [95]. Conversion to laparotomy may be required for SBO due to internal hernia, inguinal hernia, intussusception, and neoplasms [50].

Laparoscopic adhesiolysis is safe in patients with aSBO if conservative, non-operative, measures fail. Laparoscopy significantly reduces length of hospitalization, postoperative complications, and postoperative mortality when compared to open surgery [7, 96–98]. Additionally, the risks of respiratory, cardiac, and neurological complications, or of deep vein thrombosis were significantly reduced after laparoscopic adhesiolysis [99, 100]. Li et al. [101] reported no statistically significant differences in the rates of intraoperative bowel injuries, wound infections, or overall mortality between open versus laparoscopic adhesiolysis. Prolonged ileus was reduced after laparoscopy as compared to open surgery. The laparoscopic approach is safer than laparotomy but only in selected patients and with experienced laparoscopic surgeons. In a series of 9500 cases of laparoscopic adhesiolysis, iatrogenic enterotomies occurred in 4.7% of cases, of which 1.3% were missed at laparoscopy [93]. Predictive factors for successful laparoscopic adhesiolysis are patients with ≤ 2 previous laparotomies, non-median previous laparotomy incisions, adhesions secondary to appendectomy as the previous surgery, single band adhesion as cause of SBO, laparoscopic management ≤ 24 h from onset of symptoms, no signs of peritonitis on physical examination, and greater surgical experience [95].

Laparoscopy has its own limitations and complications. It is technically challenging, given the bowel distension with SBO and the risk of iatrogenic injuries like inadvertent, unappreciated enterotomy [3]. Key technical measures include

avoiding grasping distended bowel loops and grasping only mesentery or distal collapsed bowel. The small bowel must be thoroughly explored starting from the cecum and running from distal to proximal small bowel until the transition point is found and site of obstruction is identified [43].

Palliation

Surgery plays a limited role in malignant SBO. Management is focused on symptomatic treatment and palliation. Systemic chemotherapy and concurrent total parenteral nutrition may not be the best approach because these therapies result in high morbidity and mortality [102]. Decompressive percutaneous endoscopic gastrostomy (PEG), decompressive percutaneous endoscopic jejunostomy (PEJ), or endoluminal stenting is effective in relieving obstructive symptoms and improving quality of life in selected patients with advanced malignancy [103, 104].

Prognosis Unless promptly and appropriately treated, SBO is associated with considerable morbidity and mortality. The mortality of uncomplicated SBO is only 3% but rises to 30% if the SBO is complicated by necrosis or perforation [105].

Prevention Intestinal obstruction has a 30-day readmission rate of 16% and a 5-year rate of recurrent symptoms of SBO of 57.4% [106]. The strategy of preventing readmissions for SBO has focused on adhesions because it is the most common etiology of SBO and is potentially preventable by anti-adhesive agents or treatment of asymptomatic adhesions. However, complete adhesiolysis does not decrease the risk of subsequent aSBO and is therefore not recommended at surgery [93]. Only pathological adhesions should be lysed [50•]. Prolonged surgery and complete adhesiolysis are also associated with an increased risk of iatrogenic enterotomy [107].

Incidence of abdominal adhesions is reduced by performing laparoscopy over laparotomy when appropriate, emphasizing “proper” surgical techniques including meticulous hemostasis, avoiding excessive tissue destruction and desiccation, early operation when bowel ischemia occurs, and introducing anti-adhesive barriers [108•]. Four anti-adhesive agents are currently approved for clinical use in the USA, including hyaluronate carboxymethylcellulose (Seprafilm), oxidized regenerated cellulose, polyethylene glycol, and icodextrin. Seprafilm adhesion barrier has reduced the incidence of aSBO [109]. Icodextrin has been shown to reduce adhesions in patients undergoing gynecologic surgeries. However, surgical complications, especially anastomotic leakage, remain a concern after icodextrin use. A randomized prospective trial of 300 patients, with pre-planned safety analysis, did not demonstrate significant differences in the rate of surgical complications related to the use of icodextrin versus controls after 30 days [110].

Conclusions

This work systematically reviews SBO, with a particular focus on recent changes in the diagnostic evaluation and surgical therapy. About 350,000 patients are admitted for SBO per annum in the USA. The bowel obstruction typically produces a symptomatic tetrad of colicky abdominal pain, nausea and emesis, abdominal distention, and progressive constipation-to-obstipation. Physical examination may reveal an acutely ill patient with signs of dehydration and sepsis. The abdominal examination may reveal severe direct tenderness, voluntary or involuntary guarding, a rigid abdomen, and rebound tenderness with advanced intestinal ischemia or perforation. The patient should be managed on the surgical service, with consultation by a team of specialists. After medical resuscitation, abdominal CT is performed with use of IV and oral gastrografin contrast to diagnose SBO, determine whether the SBO is complete or incomplete, detect the SBO etiology, identify the transition point, and detect signs of bowel ischemia. Surgery is required for unremitting or total bowel obstruction, advanced bowel ischemia, bowel perforation, and deterioration of clinical findings while under conservative therapy. Laparoscopy is increasingly preferred over open surgery as the surgical technique.

Compliance with Ethical Standards

Conflict of Interest Mitchell Cappell reports personal fees as a consultant to the United States Food & Drug Administration (FDA) Advisory Committee for Gastrointestinal Drugs but affirms that this paper does not discuss any proprietary, confidential, and pharmaceutical data submitted to the FDA and reports personal fees as a member of the speaker's bureau for AstraZeneca and Daiichi Sankyo, co-marketers of Movantik. This work does not discuss any drug manufactured or marketed by AstraZeneca or Daiichi Sankyo. Srinivas Reddy declares no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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