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Motility Disorders of the Small Bowel

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Pearls and Pitfalls

- A mechanical cause of intestinal obstruction should be excluded before the diagnosis of a functional intestinal obstruction is made.
- A thorough history and clinical suspicion are both important in recognizing and diagnosing small intestinal motility disorders.
- Whenever possible, enteral feeding is preferable to parenteral feeding.
- Postoperative ileus is normal, but prolonged ileus is not and needs to be investigated.
- The development of obstipation after apparent resolution of ileus, with passage of flatus or stool, is a sign of mechanical obstruction.
- Patients with intestinal “pseudo-obstruction” can have true, and even complete, non-mechanical bowel obstruction.
- Intestinal pseudo-obstruction may involve either part or all of the gastrointestinal tract.
- If the diagnosis of chronic intestinal pseudo-obstruction is not considered, unnecessary laparotomy is often performed.
- Patients with suspected primary intestinal pseudo-obstruction require very specialized evaluations to diagnose precisely the type of myopathy or neuropathy present.
Introduction

Classification of Small Bowel Motility Disorders

The two types of small bowel motility disorder included here are paralytic or adynamic ileus and the chronic small intestinal hypomotility or pseudo-obstruction syndromes. Intestinal pseudo-obstruction syndromes can be either primary or secondary motility disorders, whereas ileus occurs secondary to other pathology. Both pseudo-obstruction and ileus are characterized by abnormally delayed transport of gastrointestinal contents. Postoperative ileus is encountered very frequently by surgeons, unlike the rare chronic intestinal pseudo-obstruction syndromes. Despite promising recent research findings, the difficulty of conducting clinical studies of small intestinal motility means that the complex pathophysiology of both these conditions remains unclear.

Although it may be very difficult to distinguish paralytic ileus or intestinal pseudo-obstruction from mechanical small bowel obstruction due to adhesions, tumor, or other causes, there are important differences in the typical features of non-mechanical and mechanical bowel obstructions. These might alert a prudent surgeon and possibly avoid a non-therapeutic celiotomy in patients with pseudo-obstruction.

Paralytic Ileus

Ileus is defined as the temporary loss of gastrointestinal motor function. Ileus thus is an acute, reversible condition that results in a non-mechanical intestinal obstruction. Apart from its almost invariable development after intra-abdominal operations, paralytic ileus can occur after other major operations and after trauma, intra-abdominal or generalized sepsis, myocardial infarction, pneumonia, and electrolyte derangement. A list of causes is shown in Table 2.1. During resolution of ileus, contractile activity usually returns to the small bowel first, followed by the stomach, and only then the large bowel.
The esophagus is not affected. Clinical features of ileus are usually less and may be absent after laparoscopic surgery compared with open abdominal operations.

The pathogenesis of ileus is not well understood. It is not simply a state of hypomotility, as evidenced by the fact that intestinal electrical and mechanical activity usually return rapidly after laparotomy, long before the clinical features of ileus disappear. Loss of both the contractile activity itself and the normal organization of contractile activity are important. Recent studies suggest that important etiologic factors are likely to include recruitment of inflammatory cells to the handled bowel, mast cell degranulation, and activation of inhibitory neural pathways. Also important may be the motilin-related peptide ghrelin, which has been identified as a strong promotility agent in postoperative ileus. Ileal resection and anastomosis results in the early loss, and later partial return, of electrical slow waves and phasic contractions in

<table>
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<th>Table 2.1. Causes of ileus.</th>
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<tr>
<td>Abdominal surgery, especially laparotomy</td>
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<td>Major non-abdominal operations</td>
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<td>Intra-abdominal infection</td>
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<td>Extra-abdominal infection, e.g. systemic sepsis, pneumonia</td>
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<td>Peritonitis, e.g. anastomotic leak, bile peritonitis, perforated viscus</td>
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<td>Retroperitoneal processes, e.g. pancreatitis, retroperitoneal hemorrhage, ureterolithiasis</td>
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<td>Metabolic and electrolyte disturbances, e.g. hypokalemia, hyponatremia, hypomagnesemia, hypophosphatemia</td>
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<td>Drugs, e.g. opiates, anticholinergic agents, autonomic blockers, psychotropic agents, general anesthesia</td>
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<td>Renal failure</td>
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<td>Spinal or orthopedic injury</td>
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<td>Diabetic coma</td>
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<td>Hypoparathyroidism</td>
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muscle near the resection area. This loss of electrical rhythmicity is associated with disruption to the network of interstitial cells of Cajal (ICCs), the “pacemaker” cells of the gut.

Clinical Presentation and Diagnosis

The diagnosis of ileus should be considered when signs of bowel obstruction develop in patients with known causes of ileus (see Table 2.1).

The clinical features of ileus are abdominal distension, obstipation, and either vomiting or a large quantity of nasogastric aspirate. Pain is usually absent, but, if present, it is non-colicky. In the postoperative setting, the pain is usually no more severe than would be expected with typical postoperative incisional pain and with the associated abdominal distension. Although the abdomen may be generally tender, it is usually not localized except at the wound. While the abdomen is typically silent on auscultation without the groans and rushes of a mechanical obstruction, tinkles are common, and a succussion splash may result from the large volume of fluid contained within the distended stomach or bowel. Plain erect and supine films of the abdomen show both small and large bowel dilatation with scattered air-fluid levels.

Distinguishing paralytic ileus from mechanical obstruction in the postoperative period may be difficult but is essential. Ileus should be managed nonoperatively, whereas mechanical obstruction may require operation, including urgent surgery in some patients. Careful examination and investigation of patients with continuing signs of obstruction after operation are important in order to detect complications responsible for an ongoing ileus (such as an anastomotic leak) and to detect mechanical obstruction. Mechanical obstruction in the early postoperative period may be caused by fibrinous adhesions or by an internal hernia. Patients with persistent clinical features of bowel obstruction must be evaluated to exclude a mechanical obstruction. Passing of flatus or stool, followed by a return to absolute constipation, may be a sign that mechanical obstruction has occurred. Similarly, severe
or colicky pain, localized tenderness, radiographic findings of one or more loops of dilated small intestine with a deflated colon, or the absence of gas in the colon is each a sign of mechanical obstruction and not of ileus. Investigation by computed tomography (CT) with luminal contrast or a small bowel follow-through contrast examination may be needed to exclude mechanical obstruction.

Management

The management of ileus in the postoperative period is controversial. The standard treatment has been supportive, with nasogastric decompression, no oral intake, and intravenous fluids until the passage of flatus signifies resolution of ileus. In the absence of strong data showing that nasogastric aspiration reduces the duration of postoperative ileus, there is an increasing trend toward avoidance of nasogastric tubes postoperatively. Similarly, many surgeons now introduce a water or clear liquid diet before the passage of flatus. The management of patients with prolonged postoperative ileus or nonoperative ileus is not controversial. In these patients, the stomach should be decompressed with a nasogastric tube to relieve vomiting and reduce the risk of aspiration. Serum electrolytes should be checked regularly, medications that may induce ileus should be discontinued, and complications such as pneumonia or intra-abdominal sepsis should be excluded and treated aggressively when found. Ileus will resolve spontaneously if the underlying cause(s) is identified and treated successfully. Nutritional support, including TPN, is not usually needed but may be helpful in patients who have both a prolonged ileus and antecedent malnutrition.

There is no convincing evidence that any pharmacologic treatments are beneficial. Attempts to shorten the duration of ileus using prokinetic agents such as acetylcholine, cisapride, motilin, the motilin receptor agonist erythromycin, and other drugs have been disappointing. Cisapride has been reported to be beneficial occasionally, but the use of this drug is associated with a risk of life-threatening cardiac events and
is no longer available. Recent studies suggest that effective pharmacologic therapies may be imminent. Promising agents include drugs that block peripheral opioid activity, leukocyte migration, or mast cell degranulation. As well as being a potent gastrokinetic agent, ghrelin also seems to accelerate postoperative small intestinal transit. The identification of the role of an inhibitory neural pathway involving sensory neurons of the lumbar dorsal horn of the spinal cord in the acute phase of postoperative ileus suggests other therapeutic targets. The initial suggestion that chewing gum might be a simple effective treatment for ileus has lost support after a randomized placebo-controlled trial found that chewing gum did not reduce the duration of postoperative ileus.

**Intestinal Pseudo-Obstruction**

Intestinal pseudo-obstruction is a term applied to a group of rare, incurable conditions that have in common permanent or recurrent hypomotility of either a part or the whole of the gastrointestinal tract. Although in sporadic cases the gut as a whole is abnormal, the most severely affected organ is usually the small intestine. Despite the name “pseudo-obstruction,” patients with these conditions after previous abdominal surgery can also have true intestinal obstruction, sometimes requiring total parenteral nutrition (TPN). An increased awareness of these disorders by physicians has resulted in an increase in their recognized prevalence. With the exception of acute colonic pseudo-obstruction (Ogilvie’s syndrome), intestinal pseudo-obstruction syndromes are chronic progressive diseases, with subacute and recurrent episodes in most patients that usually progress to chronic, non-resolving pseudo-obstruction.

Physiologically, both the fasted and fed motility patterns are abnormal. Migrating motor complexes (MMCs) are absent or abnormally infrequent during fasting, resulting in stasis, distension, and bacterial overgrowth. Similarly, postprandial motility is either markedly depressed, absent, or poorly coordinated.
The intestinal pseudo-obstruction syndromes are classified as either primary (idiopathic) or as secondary to a known disease. These syndromes are sometimes sub-classified according to whether they have primarily a neuropathic or a myopathic etiology. Familial visceral myopathies and, less frequently, familial neuropathies have both been described, with a clear autosomal inheritance in many myopathic cases. Sporadic idiopathic forms have been termed chronic idiopathic intestinal pseudo-obstruction (CIIP) for the neuropathic patients and nonfamilial hollow visceral myopathy for the myopathic patients. The natural history of CIIP was reported recently in a study of 59 patients followed for a mean 4.6 years. The diagnosis was made a median 8 years after symptom onset and only after most patients had undergone a therapeutic laparotomy for presumed mechanical obstruction. Long-term outcome was poor, and home TPN was needed for almost one third of patients.

A fibrotic myopathy is found in patients in whom the pseudo-obstruction is secondary to connective tissue diseases, with scleroderma being the most common of these disorders. Individuals with myotonic dystrophy, progressive muscular dystrophy, and other muscle diseases have gut involvement but may have few symptoms referable to the alimentary tract. The more common neuropathic causes are diabetes mellitus, hypothyroidism, amyloidosis, and medication use (antidepressants, antipsychotics, anti-Parkinsonian drugs, narcotics, and some antihypertensive and chemotherapeutic agents). Abuse of laxatives, especially those containing anthraquinone, can cause a chronic pseudo-obstruction that effects predominantly the colon. Pseudo-obstruction may also be secondary to a paraneoplastic syndrome.

Clinical Presentation and Diagnosis

Patients with intestinal pseudo-obstruction have a more gradual onset of progressively worsening intestinal obstruction or recurrent symptoms of subacute obstruction. Some
patients come to attention only after complete cessation of bowel activity. Common symptoms include abdominal bloating, distension, and discomfort. Patients with colonic involvement may have severe constipation, although diarrhea may also occur as a result of bacterial overgrowth. Involvement of the foregut can cause nausea, vomiting, heartburn, dysphagia, and regurgitation. Children are seen frequently with failure to thrive and weight loss.

Except for those with a known causative disease such as scleroderma, the diagnosis of intestinal pseudo-obstruction is often not suspected or entertained early in its course. As a result, patients with these syndromes undergo exploratory laparotomy frequently to treat a presumed mechanical obstruction, and it is not uncommon for multiple laparotomies to have been performed without the correct diagnosis ever being reached. A careful history, including a detailed family and medication history, should prompt consideration of the diagnosis so that mechanical obstruction can be excluded by radiologic and endoscopic evaluation rather than by operation. The astute clinician may suspect intestinal pseudo-obstruction in the patient without a previous history of abdominal surgery, and thus no adhesions, in whom the clinical presentation is not characteristic of mechanical small bowel obstruction (i.e. slow onset, absence of crampy pain, history of less severe episodes). A slow-growing neoplasm causing a progressive mechanical obstruction may present with similar clinical features and needs to be excluded as does the diagnosis of sprue, which can mimic intestinal obstruction. Radiologic imaging will often show small bowel dilatation, although the diameter can be normal in early or mild cases. Foregut motility studies and gastrointestinal transit studies can be particularly helpful. If a full-thickness biopsy of the small bowel is needed to establish the diagnosis, a laparoscopic approach, in the appropriate setting, is preferred to reduce the risk of subsequent adhesions. The details of the tests used to establish the specific myopathic or neuropathic diagnosis in unclear cases are described in the review by Coulié and Camilleri.
Management

The treatment of known intestinal pseudo-obstruction syndromes is nonoperative. The goals of treatment are to provide nutritional support and improve intestinal motility. Attempts to provide nutritional needs with high calorie, high protein soft or liquid diets are indicated, along with vitamin and mineral supplementation. Antibiotic treatment is used for those with steatorrhea and diarrhea resulting from bacterial overgrowth. Promotility agents, including cisapride (if available), erythromycin, and metoclopramide, may be beneficial in some patients.

In those patients with the most severe myopathic disease and diffuse involvement of the gut, home TPN may be necessary. In patients with less severe disease, enteral feeding may be possible and is almost always preferable to parenteral feeding. Oral feeding can be supplemented or replaced by enteral feeding via a feeding jejunostomy. Surgical resection or bypass is much less effective for these syndromes than it is for isolated colonic hypomotility, but the rare patient with truly localized disease can benefit from resection. Studies of the regeneration of small intestinal motility in an animal model suggest that it may be preferable to construct an end-to-end rather than an end-to-side anastomosis.

Relief of distension and bloating by construction of a venting enterostomy has been reported to reduce the number of hospitalizations, nasogastric intubations, and laparotomies. This operation may even allow some patients to return to enteral feeding. In end-stage disease, small bowel transplantation may be lifesaving.

Selected Readings


Surgery of the Small Bowel
Handbooks in General Surgery
(Eds.) K.I. Bland; M.G. Sarr; M.W. Büchler; A. Csendes; O.J. Garden; J. Wong
2011, XVI, 218 p. 40 illus., 10 in color., Softcover