SMALL BOWEL OBSTRUCTION

Perspective

The signs and symptoms of small bowel obstruction (SBO), as well as the potentially life-threatening nature of the condition, have been recognized since ancient times. The Egyptian Ebers Papyrus, which is thought to date to as early as 3400 BC, contains an ominous passage detailing what some modern researchers have suggested may describe a small intestinal volvulus. It states, “if thou examinet one who suffers...with colicky pains, and whose belly is stiff through it, and has pain in his cardia...nor is there any way it can come out, then it shall rot in his belly...it grows into a twist in the bowel.”

Over the centuries numerous therapeutic treatments have been used to treat SBO, including the administration of metallic mercury, opium, enemas, inflation of the bowels with air, electrical stimulation, percutaneous puncture of the bowel, horseback riding, leeches, and even “suspending the patient in an upside-down position over an attendant’s shoulder,” as suggested by Sir Astley Cooper in 1804 to assist in the reduction of a hernia, and by Jonathan Hutchinson in 1878 to treat intussusception. By the close of the 19th century, proximal intestinal decompression via gastric suctioning was found to quickly reduce the symptoms of intestinal obstruction. Although the management of SBO has remained challenging, over the last century advancements in imaging as well as operative techniques have greatly improved the prognosis for patients with this condition and have decreased the mortality rate from nearly 60% in 1900 to less than 8% today.

Intestinal obstruction is a relatively common problem encountered in the emergency department (ED), accounting for an estimated 15% of all emergency admissions for abdominal pain. This translates to over 300,000 patients per year in the United States, at an estimated yearly cost of over 1.3 billion dollars for the treatment of SBOs caused by adhesions alone. Despite the high frequency with which SBO is encountered, the ideal management remains controversial.

There are several different types of SBO with a vast array of causes ranging from the common to the truly unusual. The term mechanical obstruction implies the presence of a physical barrier to the movement of the intestinal contents. Obstructions of this type can be further subclassified according to the cause of the obstruction relative to the intestinal wall (Box 92-1). Lesions external to the intestinal tract can cause obstruction via compression from outside the gut. This is most commonly a result of postoperative adhesions, but hernias and intraperitoneal neoplasms are other causes. Lesions intrinsic to the intestinal wall itself can cause mechanical obstruction; such lesions include primary intestinal neoplasms, localized infection (i.e., intestinal wall TB), and trauma-related conditions (i.e., a hematoma of the intestinal wall). Lesions within the intestinal lumen itself can lead to obstruction. Bezoars, ingested foreign bodies, and gallstone ileus are all examples of the cause of this type of obstruction.

Another important distinction of SBO is whether the obstruction is a simple or closed-loop obstruction. A simple obstruction occurs at a single point. On the other hand, in a closed-loop type obstruction the intestine is obstructed at two locations, which creates a segment of bowel with both proximal and distal blood flow compromised. This can occur when a twist develops in the mesentery or, in the case of an internal hernia, when a loop of bowel becomes entrapped in a defect in the mesentery (Fig. 92-1). If not promptly recognized and relieved, a closed loop obstruction can quickly lead to intestinal infarction and necrosis, which, in most studies, has been shown to double the mortality rate.

In contrast to a mechanical obstruction, a neurogenic or functional obstruction occurs as a result of disruption of the normal coordinated peristaltic activity of the gastrointestinal tract in the absence of a physical blockage within the intestinal lumen. This is also commonly referred to as an adynamic ileus. The causes of adynamic ileus are listed in Box 92-2. Most often it occurs in patients who have undergone abdominal surgery and is transient in nature. In fact, some degree of functional obstruction is considered normal after surgery and is thought to result from multiple factors, including an inflammatory response to intestinal manipulation, the effects of analgesics, and the release of hormones and several neurotransmitters. In addition to surgery, a number of metabolic abnormalities and medical conditions can lead to the development of a functional SBO, including infection, medications, and metabolic abnormalities.

Finally, the term pseudo-obstruction refers to a poorly understood and complex syndrome in which the signs and symptoms of a mechanical obstruction, including the appearance of dilated bowel on radiography, are present in the absence of a mechanical lesion. This is thought to involve disruption of the intestinal “pacemaker activity” controlled by a specialized group of cells found in the gastrointestinal tract called the interstitial cells of Cajal (ICC). These cells regulate the contractility of the intestinal smooth muscle and are under the influence of the enteric nervous and autonomic systems. Because of this, pathology at any one of these sites can lead to pseudo-obstruction. Pseudo-obstruction can arise from many causes, including degenerative neuropathies, autoimmune and paraneoplastic disease, and...
progressive development of symptoms. In the presence of a mechanical obstruction, the bowel proximal to the blockage first becomes mildly dilated by the accumulation of partially digested food and normal intestinal secretions. These secretions are referred to as succus entericus and are secreted by cells lining the intestinal wall in response to mechanical stimulation. Increased intestinal dilation causes an increase in peristalsis throughout the intestines, which can trigger frequent and loose bowel movements early in the progression of the obstruction as well as episodes of nausea and vomiting. As the process continues, the bowel wall becomes edematous and the normal absorptive function of the intestinal wall decreases, leading to further accumulation of contents in the intestinal lumen proximal to the obstruction. Owing to the loss of normal intestinal motility, bacterial overgrowth begins to occur in the proximal small bowel. It is this overgrowth in a location of the intestines that is normally relatively sterile that explains the feculent nature of the emesis frequently observed in patients with SBO. As the obstruction continues, transudative fluid loss occurs into the peritoneal cavity, leading to worsening hypovolemia and dehydration. In addition, if the obstruction is proximal in location, continued bouts of emesis can lead to electrolyte abnormalities, metabolic alkalosis, and severe hypovolemia and shock.

In the presence of a closed-loop obstruction, the increase in the intraluminal pressure occurs much more rapidly because the intestinal contents cannot flow retrograde. Intestinal venous congestion and then arterial obstruction can also progress quickly to intestinal ischemia and infarction. This is referred to as a strangulation obstruction. If it is not promptly relieved, necrosis and intestinal perforation can occur. The resulting leakage of the intestinal contents into the peritoneum can lead to peritonitis and sepsis.

In the developed world the most common cause of SBO is postoperative adhesions, which account for approximately 60% of cases. These adhesions develop as a result of a process involving the interaction among numerous types of cells, cytokines, and coagulation factors caused by damage to peritoneal surfaces. It has been estimated that 93 to 100% of patients who undergo transperitoneal surgery will develop postoperative adhesions. Otherwise, up to 25% will develop SBO, with those undergoing intestinal or pelvic surgeries at greater risk. Over the last several years, numerous physical bioabsorbable barriers and pharmacologic agents have been evaluated as potentially useful in decreasing the formation of postoperative adhesions.

The second most common cause of SBO is tumors, which are responsible for roughly 20% of cases. This includes both malignancies, such as adenocarcinomas, carcinoid tumors, lymphomas, and sarcomas, and benign conditions, including adenomas, leiomyomas, and lipomas. In addition to these primary gastrointestinal tumors, gynecologic cancers, especially ovarian cancer, are a very common cause of SBO. There are also numerous case reports of metastatic disease leading to SBO, including metastatic breast, skin, and testicular cancers.

Hernias are the third most common cause of SBO, found in approximately 10% of cases. Similar to their relative frequency in general, ventral and inguinal hernias are most often encountered, but femoral hernia, parastomal hernia, lateral ventral hernia (also called spigelian hernia), and internal hernia may also lead to SBO.

Although rare in the general population, internal hernias (Fig. 92-1) are a recognized complication of bariatric surgery, especially when a Roux-en-Y type procedure has been performed. In this group, internal hernias have been described in up to 5% of patients and usually develop at the mesocolic window.

Another rare type of hernia is the obturator hernia. This hernia develops into the obturator foramen and is especially common in elderly women who have recently lost a significant amount of weight. The female pelvis is wider and the obturator canal is more narrow, creating a very common cause of SBO. 17 There are also numerous reports of metastatic disease leading to SBO, including metastatic breast, skin, and testicular cancers.

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oblique in women than in men. This, in combination with a loss of preperitoneal fat in older, often emaciated patients, predisposes to the development of an obturator hernia. Because an external mass is absent, the diagnosis can be especially challenging, which explains why it carries the highest mortality of any abdominal hernia, at nearly 70% when incarcerated.20

Gallstone ileus is another rare but important cause of mechanical SBO (Fig. 92-2). It is responsible for 1 to 4% of all cases of mechanical obstruction and is most frequently seen in elderly patients with significant underlying medical problems.21 The pathogenesis involves the entry of a gallstone into the intestinal tract through a biliary-enteric fistula. This results from the localized inflammation of cholecystitis, and in the majority of cases entry occurs via a cholecystoduodenal fistula, although cholecystocolonic and cholecystogastric fistulae can also be involved. After entering the intestinal lumen, the gallstone migrates distally. As a stone moves through the intestinal lumen, it often increases in size as bowel content sedimentation attaches. Eventually, the gallstone becomes lodged, most often in the ileum, which is the narrowest segment of the small bowel, and the patient then develops symptoms of obstruction.

Small bowel volvulus occurs infrequently in this country but is a potentially catastrophic cause of SBO. This condition results from the abnormal twisting of a loop of small bowel around the axis of its own mesentery. This accounts for only 3.5 to 6.2% of SBO in the Western world. However, it is much more common in Africa, India, and the Middle East, where it is responsible for up to 20% of cases of SBO.22 Primary small bowel volvulus occurs in an otherwise normal abdominal cavity; secondary small bowel volvulus occurs when a congenital or acquired abnormality leads to the development of the volvulus, as in the case of intestinal malrotation or as a result of postoperative adhesions.23 Although primary small bowel volvulus is poorly understood, there are several proposed factors that may predispose to its development. Diet is thought to play a role in some cases. One study found a tenfold increase in the incidence of small bowel volvulus among Muslims in Afghanistan during the festival of Ramadan.24 During this period observers abstain from food or drink from dawn to sunset and then consume large quantities of food and liquid. The researchers proposed that eating a large amount of food bulk after prolonged fasting causes the proximal jejunum to descend into the pelvis, displacing empty small bowel loops upward and initiating malrotation. Alterations in gut motility and increased small bowel length have also been suggested as possible predisposing factors. Secondary causes of small bowel volvulus include

intestinal malrotation caused by the arrest of normal rotation of the embryonic gut or as a result of postoperative adhesions. In the case of malrotation, more than 50% of affected children present for evaluation before 1 month of age with small bowel volvulus.25 Because a small bowel volvulus is a classic closed-loop obstruction, prompt recognition and surgical treatment are imperative, as the risk of strangulation is high.

Intussusception describes the invagination or “telescoping” of a part of the small intestine into itself. This results in the development of venous and lymphatic congestion with consequent intestinal edema, which can lead to intestinal ischemia and perforation. Intussusception occurs in patients of all ages but is most frequently seen in children younger than 2 years. It is the most common cause of intestinal obstruction in infants 6 to 36 months of age.26 In children the cause is most often idiopathic, but several studies have shown an association with adenovirus infection. It has been postulated that enteric adenovirus infection may trigger stimulation of the lymphatic tissue in the intestinal tract, which may create a “lead point” for the intestine to be “dragged” into itself by the normal peristaltic activity of the intestines.27 In contrast to the idiopathic nature of intussusception in children, a mechanical cause is found in more than 90% of adult cases. Tumors, either benign or malignant, are discovered as the initiating cause in more than 65% of adult cases. Several reports have also described adult intussusception in association with acquired immunodeficiency syndrome (AIDS).28 These patients are at an increased risk of lymphoma or unusual infections, such as atypical mycobacterial infections, as the cause of the development of intussusception.

Clinical Features

History

Patients with SBO commonly report crampy abdominal pain, abdominal distention, nausea, vomiting, constipation, or the inability to pass flatus. The pain is often described as periumbilical in location and typically has a crescendo-decrescendo pattern. The recurrent waves of discomfort can last from seconds to minutes. In more proximal obstruction, symptoms of nausea and vomiting can be much more severe, and the onset of symptoms is often more abrupt. Distal obstructions typically cause symptoms over a slower period of 1 to 2 days and are frequently accompanied by greater abdominal distention. It is important to remember that the colon requires up to 24 hours to empty after the formation of SBO, and the associated small bowel distention stimulates peristalsis. Because of this, flatus and the passage of stool may continue even in the presence of a complete obstruction. A thorough past surgical history should be obtained, and any history of malignancy or inflammatory bowel disease should be determined.

Physical Examination

As with all patients in the ED, the physical examination should start with a careful evaluation of the patient’s hemodynamic status, degree of distress, and general condition. Thus patients requiring resuscitation can be quickly identified, and the appropriate interventions, including aggressive intravenous fluids, can be initiated early. Inspection of the patient should include a careful search for abdominal distention and hernias and should include a genital examination. Although bowel sounds in SBO are frequently described as high pitched and “tinkling” in nature, studies have shown that they are also frequently decreased or absent in SBO. One study showed that physicians listening to recordings of bowel sounds were able to correctly identify SBO in only 42% of affected patients.29
The presence of peritoneal signs usually indicates late obstruction with complications, including strangulation. However, it should be noted that abdominal palpation in the setting of significant bowel dilation can give the false impression of peritonitis, because quick compression-decompression of dilated bowel may elicit a significant pain response. For this reason it may be helpful to determine the presence of pain with cough or gentle shaking of the patient’s pelvis to better investigate for true peritonitis.

**Complications**

There are numerous serious complications associated with SBO. Persistent vomiting can lead to significant hypovolemia, metabolic alkalosis, and shock. If strangulation occurs, necrosis of the bowel can lead to perforation and leakage of contaminated bowel contents into the peritoneal space. This can cause peritonitis, intra-abdominal abscess formation, and sepsis. As one would expect, complications are more common in older patients and in those with comorbidities. There are also several potential complications related to surgical intervention for SBO, including wound infection and short bowel syndrome. Unfortunately, in addition to these adverse events, the rate of recurrence of SBO is quite high. For patients treated nonoperatively the rate is 40%, and in those treated operatively, 27%. For patients with SBO secondary to adhesions, the relative risk of recurrence increases with the number of prior episodes of obstruction. For those with four or more episodes of adhesional SBO, the recurrence rate is more than 80%.

**Diagnostic Strategies**

Although laboratory tests are not helpful in diagnosing the presence of SBO, they can be very useful in assessing the degree of dehydration and metabolic disruption resulting from the obstruction. Several studies have evaluated the use of various biomarkers to identify strangulation complicating SBO. These have included lactate and creatinine phosphokinase (CPK), and some studies have evaluated the use of intestinal fatty acid binding protein (1-FABP), which is released by necrotic enterocytes, to attempt to identify strangulation. Unfortunately, these may be normal until very late in the process of intestinal strangulation and have proven to be overall unreliable.

Several imaging modalities are applicable in the diagnosis and evaluation of SBO. Plain radiographic findings are estimated to be diagnostic in approximately 50 to 60% of cases of SBO, equivocal in 20 to 30%, and normal, nonspecific, or misleading in 10 to 20%. The cause of the obstruction can rarely be elicited from plain radiographs. In addition, there is occasionally a discrepancy between terms used by emergency physicians and radiologists to describe abdominal plain film results. For example, many emergency physicians use the term “nonspecific bowel gas pattern” to mean normal. However, one survey showed that 65% of radiologists use that same term to describe normal bowel gas findings, 22% use it to mean that they cannot tell if the findings are normal or abnormal, and 13% use it to indicate an abnormal study, but one in which they cannot determine if the findings represent mechanical obstruction or adynamic ileus.

Abdominal plain film radiographs should include both supine and upright or decubitus studies. An upright chest radiograph may also be obtained to evaluate for subdiaphragmatic free air resulting from a bowel perforation. Characteristic plain radiographic findings of SBO include distended loops of bowel, normally greater than 3 cm in diameter, seen centrally in the radiograph (Fig. 92-3). In addition, unlike the haustra of the large intestine, which do not cross the full diameter of the bowel, the valvulae conniventes (or plicae circulares) of the small bowel cross the entire lumen of the small intestine. Noting these structures crossing the lumen of distended bowel on plain radiographs helps to differentiate SBO from large bowel obstruction. In general, the greater the number of distended loops, the more distal the obstruction. No gas should be seen in the large bowel, unless the films are obtained early in the course of the obstruction, or in the presence of a partial SBO.

![Figure 92-3](image_url)

**Figure 92-3.** A, Supine plain film radiograph showing dilated loops of small bowel in a patient with small bowel obstruction. B, Upright abdominal plain radiograph revealing multiple air-fluid levels and small bowel dilation, consistent with a diagnosis of small bowel obstruction.
If the obstructed small bowel contains more fluid than air, the classic radiographic findings may be absent. In this setting, a row of air bubbles seen on upright or decubitus plain films may be present. These represent small amounts of air trapped between the valvulae conniventes. The meniscal effect of the surrounding fluid gives the air an ovoid or rounded appearance, which is referred to as the “string of pearls” or “string of beds” sign and is very suggestive of SBO. An adynamic ileus, on the other hand, tends to show extensive air-filled loops throughout the entire gastrointestinal system, and no small bowel dilation.

Computed tomography (CT) has become an increasingly popular imaging modality in the evaluation of SBO and has largely replaced the small bowel series, in which barium or water soluble contrast that is swallowed or introduced into the duodenum via a nasogastric tube is followed fluoroscopically. CT has been shown to detect SBO with a high degree of sensitivity and specificity. In addition, unlike plain films and the small bowel follow-through studies, CT provides more information about the cause of obstruction, such as a tumor.

Finally, any discussion of the diagnostic strategies in SBO would be incomplete without addressing the challenges of identifying strangulation complicating SBO. Despite several decades of investigations into the problem, numerous studies have failed to elicit clinical indicators that reliably predict this life-threatening condition. One study that evaluated 72 preoperative clinical, laboratory, and radiologic findings at admission showed that only the CT finding of reduced intestinal wall enhancement, elevated white blood cell (WBC) count, and guarding on physical examination were independent predictors of bowel strangulation. Of these, the CT finding was by far the most specific at 96%, with a likelihood ratio of 9.3 for the presence of strangulation. The likelihood ratios for leukocytosis and guarding for the presence of strangulated bowel were much less impressive at 1.7 and 2.8, respectively.

**Differential Considerations**

The diagnosis of SBO should be considered in any patient with abdominal pain and vomiting, especially if he or she has a history of prior abdominal surgery. It may be difficult to differentiate SBO from nonobstructive intestinal motility disorders such as adynamic ileus or intestinal pseudo-obstruction by history and physical examination alone.

The other conditions to consider in the differential diagnosis range widely from benign to potentially life-threatening. These include constipation, cholelithiasis or nephrolithiasis, ectopic pregnancy, pancreatitis, peptic ulcer disease, atypical myocardial infarction, leaking abdominal aortic aneurysm (AAA), and mesenteric ischemia. These pathologies have typical signs, symptoms, and diagnostic findings that can help differentiate them from one another and from SBO, but this may be challenging, especially early in the course of the particular disorder.

**Management and Disposition**

Hemodynamically unstable patients should be aggressively resuscitated with crystalloid solution via a large-bore catheter. Although use of nasogastric decompression is considered dogma by many emergency physicians and surgeons, its effect in decreasing the duration of SBO has scant support in the medical literature. In the era of modern antiemetics, if the patient’s symptoms of nausea and vomiting can be controlled with medication, it may be reasonable to delay nasogastric tube insertion. However, if symptoms persist or if the patient has an altered level of consciousness that places him or her at risk for aspiration, a nasogastric tube should be promptly inserted and attached to wall suction. Studies have shown no benefit to the use of long intestinal tubes over a traditional nasogastric tube. Placement of a nasogastric tube is a noxious procedure, and attempts should be made to anesthetize the patient’s nasopharynx with topical anesthetic before insertion.

All patients with SBO merit admission to the hospital. One recent study found that patients with SBO admitted to a surgical service for inpatient management had a shorter length of stay, lower hospital charges, and lower mortality than those admitted to the medical service. This was attributed largely to the fact that those patients in whom conservative management was failing and who needed surgical intervention were identified more quickly when being managed primarily by the surgical team. However, in the community setting, where hospitals with internal medicine and family practice training have assumed a large role in the management of surgical patients, it may not be logistically possible to admit patients with SBO directly to a surgical service.

Bacterial translocation is thought to result from simple SBO; in one series, 59% of patients who underwent operative intervention for simple SBO had bacteria (most commonly *Escherichia coli*) cultured from mesenteric lymph nodes, compared with 4% of patients who underwent operations for other reasons. However, this has not been shown to be clinically relevant, and there is no convincing evidence to recommend the empirical use of antibiotics in the nonoperative management of a simple SBO. In patients in whom surgical exploration is planned or perforation is suspected, antibiotics are recommended. The antibiotic used should provide coverage against the gram-negative and anaerobic organisms that colonize the intestinal tract (e.g., second-generation cephalosporins).

SBO in the presence of known malignancy is very common, occurring in up to 30% of patients with colon cancer and 50% of patients with ovarian cancer at some time in the course of their disease. Surgery should be the primary treatment for selected patients with malignant bowel obstruction (MBO). In patients who do not qualify for surgical intervention because of intra-abdominal carcinomatosis, massive ascites, or poor overall health status, treatment with self-expanding metal stents and the use of octreotide to rapidly reduce gastrointestinal secretions may provide significant palliative relief. A collaborative approach with the patient’s oncologist and consulting surgeon can provide the ideal individualized treatment for the patient in this situation.

Finally, although laparoscopic surgery was once considered inappropriate for the management of SBO, several studies have shown that laparoscopy is a safe and effective surgical method in the treatment of SBO in adults and children, particularly those with obstructions caused by adhesions. The risk of recurrence seems to be similar to that of an open surgical approach. As surgeons continue to gain experience with laparoscopy, it will likely become more commonly applied to the surgical management of SBO.
ACUTE MESENTERIC ISCHEMIA

Perspective

In his 1926 treatise on mesenteric vascular occlusion, A.J. Cokkinis wrote, “occlusion of the mesenteric vessels is apt to be regarded as one of those conditions of which...the diagnosis is impossible, the prognosis is hopeless and the treatment almost useless.” Despite significant advances in the understanding of the pathophysiology of this condition since that time, the mortality rate has remained as high as 60 to 80%, and the diagnosis and treatment of this vascular catastrophe have remained difficult.

Acute mesenteric ischemia (AMI) involves the acute insufficiency of the blood supply to the small bowel and may also involve the right colon. The left colon has a much higher degree of collateral blood flow and is less prone to AMI. When AMI occurs, rapid intestinal injury results. This condition should be clearly differentiated from chronic mesenteric ischemia (CMI), which is often referred to as “intestinal angina” and often manifests as recurrent episodes of abdominal pain resulting from insufficient intestinal blood flow during periods of increased postprandial metabolic demand. CMI does not usually require emergent therapy; however, it is possible for AMI to develop in these patients as well.

Overall AMI is a rare clinical problem, accounting for 0.1% of hospital admissions. Within the diagnosis of AMI there are four specific clinical categories that make up the overwhelming majority of causes, each having distinct epidemiologic risk factors. These categories include mesenteric arterial embolus, mesenteric arterial thrombosis, nonocclusive mesenteric ischemia, and mesenteric venous thrombosis.

Principles of Disease

The mesenteric vessels arise from the primitive ventral segmental arteries. Although there is a significant amount of person-to-person variability, as embryologic development proceeds, these vessels typically regress with the exception of the 10th, 13th, and 21st segmental arteries. These become the celiac trunk, superior mesenteric artery (SMA), and inferior mesenteric artery (IMA), respectively. The celiac trunk arises from the anterior aspect of the abdominal aorta and branches into the common hepatic, splenic, and left gastric arteries. These vessels supply the distal esophagus to the duodenum at the entrance of the bile duct. The SMA normally arises 1 cm below the celiac trunk and runs toward the cecum, terminating as the ileocolic artery. The SMA supplies the distal half of the duodenum to the proximal two thirds of the transverse colon. The IMA originates approximately 6 to 7 cm below the SMA and gives rise to the left colic artery, the sigmoid arteries, and the hemorrhoidal arteries. Anatomically, this vessel provides blood flow to the distal third of the transverse colon to the rectum.

The gut receives 20% of cardiac output at rest, and up to 35% after eating. Of this, up to 70% supplies the mucosa. This is because of the high metabolic demands required for the absorptive function of this intestinal layer.

Intestinal blood flow is regulated by a complex combination of intrinsic and extrinsic mechanisms to match intestinal demands with blood supply. Intrinsic factors provide the moment-to-moment control of the intestinal circulation; they function independently from neural control. This intrinsic modulation has been proposed to involve the release of local metabolites produced as a result of mucosal ischemia. These metabolites are then thought to diffuse to the local arterioles, triggering relaxation in the smooth muscle and increased blood flow, thereby allowing for efficient adjustments to the intestinal blood supply. Smooth muscle relaxation can also be brought about directly by a decrease in the perfusion pressure in the arterioles themselves. These two mechanisms are referred to as the metabolic and myogenic pathways. Intestinal blood flow is also controlled extrinsically through neural and hormonal mechanisms. Increased sympathetic tone to the paired celiac ganglia located adjacent to the celiac trunk results in mesenteric and arteriolar vasoconstriction. Hormonal influences include the direct action of angiotensin II released as a result of decreased extracellular volume, as well as vasopressin to cause mesenteric vasoconstriction.

Although these mechanisms allow for the mesenteric circulation to adapt to wide variations in the metabolic needs of the gut and systemic perfusion, in the setting of acute compromise the bowel is very quickly injured. Because of the high metabolic demands of the intestinal mucosa, within 15 minutes of absolute ischemia, structural damage to the intestinal villi can be observed histologically (Fig. 92-4). If not corrected, within 3 hours mucosal sloughing occurs. By 6 hours, transmural necrosis is complete. Complicating the situation even further, reestablishment of blood flow at this point results in the systemic release of several proinflammatory cytokines and toxic oxygen radicals caused by reperfusion, which can lead to multiorgan failure and rapid death.

Mesenteric Arterial Embolism

Arterial emboli are the most common cause of AMI and are responsible for approximately 50% of cases. The median age of patients with mesenteric arterial emboli is 70 years, and two thirds are women. Emboli are most often cardiac in origin and arise from left atrial or ventricular mural thrombi or valvular lesions. Risk factors for the development of such thrombi include myocardial ischemia or infarction, cardiomyopathies, ventricular aneurysms, endocarditis, and atrial dysrhythmias, specifically atrial fibrillation. Compared with the estimated annual risk of stroke of 2.3%, the annual risk of AMI caused by thromboembolism secondary to atrial fibrillation is 0.14%. The SMA is most frequently affected because of the large caliber of the vessel and its narrow takeoff angle from the aorta. The embolus typically lodges 3 to 10 cm distal to the origin of the SMA (Fig. 92-5). The jejenum is most often involved, as it is distant from the collateral flow provided from the celiac and inferior mesenteric arteries.

Mesenteric Arterial Thrombosis

Mesenteric arterial thrombosis results from the progression of atherosclerotic disease of the mesenteric vasculature. Risk factors for development include advanced age, hypertension, diabetes, and tobacco use. Affected patients frequently have a history...
suggestive of CMI of several months’ or years’ duration. Unlike embolic occlusions, thrombosis usually occurs in the proximal SMA at the origin of the vessel.56

Nonocclusive Mesenteric Ischemia

Nonocclusive mesenteric ischemia occurs as a result of mesenteric vasospasm in the absence of a physical obstruction. This vasospasm is triggered by mesenteric hypoperfusion or excessive sympathetic nervous system activity. Mesenteric hypoperfusion can result from a wide variety of conditions, including sepsis, severe dehydration, pancreatitis, or hemorrhagic shock. Excessive sympathetic activity can result from congestive heart failure, or the use of medications and drugs such as vasopressors, cocaine, or digoxin.57 Once initiated, this vasospasm often persists even after correction of the underlying condition, and repeated episodes of ischemia and reperfusion occur. Studies suggest that this recurrent pattern of ischemia and reperfusion may result in more severe histologic injury than a single episode of prolonged ischemia.58

Mesenteric Venous Thrombosis

Mesenteric venous thrombosis is the least common cause of AMI, accounting for only 5 to 15% of all mesenteric ischemic events. It most frequently involves the superior mesenteric vein and its branches. In the vast majority of cases (over 75%), an underlying inherited thrombotic disorder or an inherited or acquired hypercoagulable state can be identified. The most common cause is factor V Leiden mutation, which is thought to account for 20 to 40% of cases. Other inherited prothrombotic states implicated in the development of mesenteric venous thrombosis include deficiency in antithrombin III, protein C, or protein S. Hematologic conditions predisposing to this condition include polycythemia vera and essential thrombocytopenia. Oral-contraceptive use accounts for 9 to 18% of the episodes of mesenteric venous thrombosis in young women. Local intra-abdominal inflammation secondary to pancreatitis, malignancy, or inflammatory bowel disorders also increase the risk of mesenteric venous thrombosis.

Finally, the venous stasis caused by portal hypertension is a recognized risk factor.59

Box 92-3 summarizes causes of mesenteric venous thrombosis.

Unusual Causes of Mesenteric Ischemia

In addition to the conditions already described, there are numerous case reports of unusual causes of mesenteric ischemia. These include SMA dissection leading to occlusion, tumor emboli, retroperitoneal fibrosis, and various types of vasculitis, including Buerger’s disease, polyarteritis nodosa, and Takayasu’s arteritis. Because these conditions involve a rare cause of an already rare condition, they frequently go unrecognized until patients develop major adverse outcomes.60

Clinical Features

History

The history at presentation is largely dependent on the nature of the underlying cause. The traditional historical triad of AMI is the sudden onset of poorly localized abdominal pain and gastric emptying (vomiting or diarrhea) in a patient with cardiac disease. This is especially true in cases of SMA embolism or thrombosis, in which symptoms and clinical deterioration can rapidly occur. In cases of mesenteric venous thrombosis the symptoms are slower in onset and often have been present for several days by the time the patient seeks medical attention. Approximately one third of patients with acute embolic mesenteric ischemia and one half of patients with acute mesenteric venous thrombosis have a personal history of an embolic event such as a pulmonary embolism, deep vein thrombosis, or ischemic stroke.61 Patients with nonocclusive mesenteric ischemia are often already critically ill and in the hospital, making it difficult or impossible for them to provide historical details to the treating physician.

Physical Examination

The pain is classically described as being “out of proportion” to the physical examination findings. The patient may be writhing in
pain but have a soft abdomen without guarding, especially early in the course of the event when only the visceral structures are ischemic. As the parietal peritoneum becomes ischemic, the abdominal physical findings progress. If the ischemia progresses to infarction, peritonitis may be present. Heme-positive stools may also be noted. Hypotension, tachycardia, and tachypnea are all signs of severe ischemia and suggest a poor prognosis.

Complications

Even with prompt recognition and aggressive treatment of AMI, a complicated course is expected. Secondary reperfusion injury is common, and bowel initially identified as viable may progress to necrosis. Other complications include wound infections, sepsis, and pneumonia. Given the population in which AMI tends to occur, the physiologic stress of this disease process also places patients at high risk for myocardial infarction, renal failure, and pulmonary embolism while in the hospital.

Diagnostic Strategies

Because of the rare and potentially devastating nature of AMI, it is imperative that a high index of suspicion for this condition be maintained. The emergency physician should be especially vigilant in patients older than 50 years with the complaint of the sudden onset of abdominal pain. Despite significant advances in the understanding of the pathology of AMI, its rapid diagnosis remains challenging. Owing to the rarity of this condition, there are few clinical data comparing treatments, and most suggested treatment guidelines have been created from expert opinion.

Initial laboratory results are often nonspecific and may include leukocytosis, an elevated hematocrit secondary to hemococoncentration, and metabolic acidosis. Several serum biomarkers have been investigated as early indicators of AMI. These include lactate, D-dimer, interleukin (IL)-6, and serum ischemia-modified albumin levels. One small study showed that an elevated plasma lactate level was 100% sensitive for AMI but only 42% specific. To date, no biomarkers have been found that are sufficiently sensitive and specific to diagnose or eliminate mesenteric ischemia. In addition, several of the biomarkers currently being investigated require up to several hours for results to become available, which significantly limits their usefulness in this time-sensitive emergency.

Mesenteric angiography remains the “gold standard” in the radiographic evaluation for mesenteric ischemia, and offers the benefit that diagnosis and initial treatment can occur concurrently. However, in reality, angiographic services may not be widely available to the treating emergency physician. Plain radiographs in mesenteric ischemia are most often nonspecific. Later in the disease course, plain radiograph findings may show so-called thumbprinting, in which multiple, round, smooth soft tissue densities project into the intestinal lumen because of mucosal and submucosal edema and hemorrhage. More specific but very late plain radiographic findings indicating infarction include pneumatosis intestinalis and portal venous gas.

CT has largely replaced conventional angiography as the initial imaging study of choice in the evaluation of mesenteric ischemia (Fig. 92-6). Several studies have shown that with the emergence of multidetector scanners the sensitivity and specificity for mesenteric ischemia are quite high, at approximately 96% and 94%, respectively. Although the current management guidelines for AMI created by the American Gastroenterological Association suggest frequent and rapid traditional angiographic evaluation in cases of suspected AMI, these recommendations predate numerous studies on the accuracy of CT angiography in the diagnosis of AMI. Future revisions of these guidelines will likely be amended to include more use of CT angiography in the initial evaluation of these patients.

Duplex sonography has also been evaluated as an imaging tool in the diagnosis of AMI. It has been found to be very specific (92-100%) for the detection of occlusions, but its sensitivity is decreased (70-89%) by the limited evaluation beyond the proximal main vessel that is possible with ultrasound. It is unable to provide much information about complications of AMI, including bowel infarction.

Differential Considerations

Other potentially devastating conditions to consider in the differential diagnosis of acute-onset severe abdominal pain include leaking AAA, perforated viscus, bowel obstruction, biliary disease, and atypical myocardial infarction.

Management and Disposition

Once AMI has been diagnosed, the goals of treatment are to restore mesenteric blood flow as rapidly as possible, manage underlying conditions, treat persistent mesenteric vasospasm if present, and mitigate the risk of further clot propagation. Initial interventions should focus on aggressive fluid resuscitation and hemodynamic stabilization. Because these patients are often elders with significant cardiac comorbidities, invasive monitoring may be indicated. If vasopressors are required, dobutamine, low-dose dopamine, or milrinone is advised, as these agents have been shown in human and animal studies to have less of a vasoconstrictive effect on the mesenteric vasculature than other agents. Broad-spectrum antibiotics should be administered without delay, and a general or vascular surgeon should be immediately consulted. Further management is dependent on the cause of ischemia, and controversies exist as to the optimal management of these critically ill patients.

Regardless of the cause, if signs of intestinal infarction or perforation with peritonitis are present, prompt emergent laparotomy is warranted. Some have suggested that preoperative conventional angiography may be beneficial to attempt rapid revascularization, and recent data suggest that initial endovascular revascularization may significantly improve patient outcomes and
dramatically alter the future treatment of AMI. Numerous reports exist detailing the use of thrombolytic agents, angioplasty, embolectomy, or vascular stenting to restore mesenteric blood flow. In addition, the phosphodiesterase inhibitor papaverine may be continuously infused directly into the compromised vessel. This agent results in elevated levels of cyclic adenosine monophosphate (cAMP), which results in profound smooth muscle relaxation. Because this medication undergoes over 90% first-pass hepatic metabolism, few systemic effects are noted when the agent is infused directly into the mesenteric circulation. However, catheter migration into the aorta can cause rapid hemodynamic collapse if not quickly recognized. A “second-look” laparotomy may be indicated, as bowel that initially appears viable may become gangrenous later.

Primary treatment of nonocclusive mesenteric ischemia involves aggressive interventions to reverse the underlying cause and consideration for papaverine infusion via angiographic catheter as well as intravenous heparin to prevent thrombosis in the vasospastic vessel. Papaverine infusion is often maintained for 24 hours, at which time repeated angiography may be performed to evaluate for the resolution of vasospasm. If peritoneal signs develop, laparotomy is indicated. If the underlying medical condition persists, the mortality of nonocclusive mesenteric ischemia remains high.

The treatment of mesenteric venous thrombosis is unique in that, in the absence of peritoneal findings, initial treatment with heparin infusion alone may be adequate. However, if peritoneal findings are present or develop later in the patient’s hospital course, bowel necrosis is likely and prompt laparotomy. If the patient recovers, long-term anticoagulation with warfarin usually is provided to prevent recurrence. An appropriate workup for hypercoagulable conditions should be undertaken.

Acknowledgement

The contributors would like to thank Drs. Susan P. Torrey and Philip Henneman for their work in earlier editions.

KEY CONCEPTS

- Acute mesenteric ischemia is a rare vascular catastrophe with a very high mortality. It should be considered in patients older than 50 years with a history of cardiac disease who have acute abdominal pain, which may initially appear as severe pain that seems “out of proportion” to physical examination findings.
- Within the diagnosis of AMI are four distinct clinical entities with specific associated risk factors, clinical presentations, and treatments: mesenteric arterial embolism, mesenteric arterial thrombosis, nonocclusive mesenteric ischemia, and mesenteric venous thrombosis.
- Traditionally, conventional angiography has been the imaging test of choice. However, recent advancements in multidetector CT angiographic scanning have made this modality a very sensitive and specific tool for the diagnosis of AMI.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.