Mesenteric ischemia results from lack of adequate blood flow to and oxygenation of the mesentery and intestines. The clinical course consists of rapid progression from bowel ischemia to infarction, sepsis, and usually death. The incidence of acute mesenteric ischemia is increasing because of an aging population and the prolonged survivability of patients with severe cardiovascular disease and other significant medical conditions.

Four types of mesenteric ischemia must be considered: acute mesenteric ischemia, nonobstructive mesenteric ischemia (NOMI), mesenteric vein thrombosis, and chronic mesenteric ischemia (or mesenteric angina). The most deadly of these is acute mesenteric ischemia, which results from sudden obstruction of blood flow to the intestines and has a mortality rate approaching 90%.

**EPIDEMIOLOGY**

Mesenteric ischemia accounts for 0.1% of all hospital admissions and 1% of emergency department (ED) visits for abdominal pain in geriatric patients. Cases of mesenteric vein thrombosis are more difficult to estimate accurately but have been reported at 2 per 100,000 admissions over a period of 20 years at one center.\(^1\)\(^3\)

The overall mortality associated with mesenteric ischemia is between 60% and 93% but rises precipitously once bowel wall infarction has occurred. Mortality remains greatest for acute mesenteric ischemia resulting from obstruction or embolic phenomena. Patients with an early manifestation of NOMI have mortality rates of 50% to 55%, whereas patients with mesenteric vein thrombosis have a 15% mortality at 30 days.\(^1\)\(^3\) Those affected by chronic mesenteric ischemia have a more prolonged course, are relatively protected by dual blood supply, and present the physician with more numerous chances for intervention.

**PATHOPHYSIOLOGY**

Any patient with advanced age, atherosclerosis, thromboembolic disease, atrial fibrillation, and processes leading to chronic low-flow states is at risk for the development of arterial mesenteric ischemia (Tables 34.1 and 34.2).\(^1\)\(^2\)\(^4\)\(^5\) Mesenteric venous obstruction carries its own separate risk factors, which are similar to those for venous thrombosis anywhere in the body.

Acute mesenteric ischemia is a result of the precipitous onset of hypoperfusion caused by occlusive or nonocclusive obstruction of either arterial or venous blood flow. Acute hypoperfusion occurs in 65% of cases and carries a mortality rate exceeding 60%. Occlusive arterial obstruction is most commonly caused by embolic or thrombotic obstruction of the superior mesenteric artery (SMA). NOMI is often due to vasoconstriction of the splanchnic system. Occlusive venous obstruction occurs with thrombosis or segmental strangulation. Mesenteric venous thrombosis is the main cause of mesenteric ischemia in younger patients without cardiovascular disease.

Acute arterial embolism causes a dramatic cessation of blood flow, with rapid progression from ischemia to infarction. As the bowel wall necroses, contamination with intraluminal bacteria leads to peritonitis, sepsis, and toxin-mediated hypotension.

Nonocclusive infarction, which represents 25% of all cases of acute ischemia, is most often caused by splanchnic hypoperfusion and vasoconstriction. Risk factors for nonocclusive disease include advanced age, acute myocardial infarction (AMI), acute cardiac decompensation, and heart failure.
### Table 34.1 Risk Factors for Ischemic Bowel Diseases

<table>
<thead>
<tr>
<th>RISK FACTOR</th>
<th>ARTERIAL THROMBOSIS</th>
<th>EMBOLUS</th>
<th>MESENTERIC VEIN THROMBOSIS</th>
<th>NONOBSTRUCTIVE MESENTERIC ISCHEMIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advanced age</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>+</td>
<td></td>
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<td></td>
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<tr>
<td>Aortic dissection</td>
<td>+</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Low cardiac output</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
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<tr>
<td>Congestive heart failure</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
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<tr>
<td>Shock</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Severe dehydration</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
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<tr>
<td>Cardiac arrhythmias, especially atrial fibrillation</td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Severe cardiac valvular disease</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recent myocardial infarction</td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Intraabdominal malignancy</td>
<td></td>
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<td>+</td>
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<tr>
<td>Abdominal trauma</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
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<tr>
<td>Intraabdominal infection</td>
<td></td>
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<td></td>
<td>+</td>
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<tr>
<td>Intraabdominal inflammatory conditions</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
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<tr>
<td>Parasitic infection (ascariasis)</td>
<td></td>
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<td>+</td>
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<tr>
<td>Hypercoagulable states (venous thrombosis)</td>
<td></td>
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<td></td>
<td>+</td>
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<tr>
<td>Sickle cell anemia</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Recent cardiac surgery</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Recent abdominal surgery</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Vascular aortic prosthetic grafts proximal to the superior mesenteric artery</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemodialysis</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Vasculitis</td>
<td>+</td>
<td>+</td>
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<td></td>
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<tr>
<td>Drugs that cause constriction</td>
<td></td>
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<tr>
<td>Digitalis</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
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<tr>
<td>Cocaine</td>
<td></td>
<td></td>
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<td>+</td>
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<tr>
<td>Amphetamines</td>
<td></td>
<td></td>
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<td>+</td>
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<tr>
<td>Pseudoephedrine</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
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<tr>
<td>Vasopressin</td>
<td>+</td>
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<td>+</td>
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<tr>
<td>Estrogen therapy</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Pregnancy</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Decompression sickness</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Blast lung caused by systemic air embolism</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data from references 1, 2, 4, and 5.

*A plus sign (+) indicates that the factor is a risk for the disease subtype.

†Especially after sclerotherapy.
Diuretics contribute to a decrease in splanchnic perfusion in patients with profound heart disease, whereas medications such as digoxin and alpha-blockers cause regional vasoconstriction and may add to a low-flow state. Cocaine can cause splanchnic vasoconstriction and should be suspected as a cause of mesenteric ischemia in younger patients.

Bowel perfusion is generally preserved during periods of hypotension; therefore, NOMI represents failure of the normal autoregulatory systems. Patients with chronic renal failure may have bowel ischemia after hemodialysis, probably from hypoperfusion, which promotes preferential shunting of blood from the splanchnic circulation to preserve flow to the cardiac and cerebrovascular systems.

Although acute mesenteric vein thrombosis accounts for a small proportion of cases of ischemic bowel disease (5% to 10%), the ease of diagnosis with computed tomography (CT) has allowed identification of a greater number of patients with venous thrombosis. Symptoms are even less specific than those of arterial obstruction and are manifested over a longer period before bowel infarction occurs. Thrombus secondary to hypercoagulable states develops first in the smaller vessels and later progresses into the larger veins; clots associated with cirrhosis, neoplasm, or local injury (operative, trauma) start at the site of obstruction and evolve distally.4

Thrombotic arterial ischemia occurs late in the course of severe mesenteric atherosclerotic disease and involves the three major sources of intestinal blood supply: the celiac artery, the SMA, and the inferior mesenteric artery (IMA). Symptoms are typically manifested when two of the three vessels are significantly stenosed or completely obstructed.

A review of the anatomy of arterial blood flow to the intestines is helpful in understanding the pathophysiology of mesenteric ischemia.

The celiac artery arises anteriorly from the abdominal aorta at the level of the 12th thoracic vertebra. The celiac artery branches into the common hepatic, splenic, and left gastric arteries. These vessels supply their corresponding organs with significant redundancies, so ischemia in these areas is rare.

The SMA comes off the aorta 1 cm below the celiac artery and terminates as the ileocolic artery. This latter vessel supplies the majority of the blood delivered to the small intestine, as well as some flow to the pancreas, right colon, and transverse colon.

The IMA originates from the aorta 7 cm distal to the SMA. It provides blood to the distal transverse colon, descending colon, and rectum.

There is a significant array of collateral blood vessels and flow patterns. The small intestine is especially vulnerable to ischemia, however, because the terminal arterioles enter the intestinal wall without collateral pathways.5 Splanchnic blood flow requirements vary continuously but can account for up to 35% of cardiac output.

Venous drainage of the system occurs via the superior mesenteric vein, which empties into the portal vein.

### Presenting Signs and Symptoms

#### Classic Presentation

Soon after ischemia begins, patients have complaints of severe abdominal pain that is clearly out of proportion to the findings on physical examination, such as a soft abdomen that is not very tender to palpation. The description and location of the pain vary over time. As the disease progresses, infarction develops and the symptoms may temporarily remit. Over the next several hours, bowel necrosis leads to signs of peritonitis: the abdomen becomes rigid, distended, and very painful with decreased bowel sounds. The intestinal mucosa begins to slough, and rectal bleeding occurs. At this point the stool contains occult blood in 60% of patients. The bowel may perforate, as signaled by findings of hypotension and sepsis.

Clues to diagnosis of the various ischemic bowel diseases are as follows:

- Acute abdominal pain followed by rapid and forceful evacuation of the bowels (vomiting or diarrhea) strongly suggests an embolic phenomenon in the SMA.
- Long-standing abdominal pain (weeks to months), which is then followed by acute worsening, suggests intestinal angina and SMA thrombosis.
- Patients with risk factors for NOMI may have unexplained abdominal distention or gastrointestinal bleeding; pain is totally absent in up to 25% of these patients, and unexplained distention may herald infarction.

Chronic mesenteric ischemia, or “intestinal angina,” refers to a pattern of pain typically brought on after eating that is usually episodic and recurrent, is sometimes constant, and lasts for up to 3 hours at a time. Mesenteric arterial atherosclerotic disease is generally the cause of chronic mesenteric ischemia, with a process similar to that of coronary artery disease and resultant angina pectoris.

Colonic ischemia occurs much less frequently than small bowel ischemia. Colonic ischemia often resolves spontaneously and without sequelae but can lead to significant morbidity and, in some cases, death.

#### Variations in Presentation

In patients with NOMI, the severity and location of pain vary, which complicates early diagnosis. A heightened level of suspicion is necessary for patients with significant risk factors.

Mesenteric vein thrombosis may be totally asymptomatic and might be diagnosed as an incidental finding in patients.
undergoing CT of the abdomen for reasons other than abdominal pain. Blockage of the IMA may be silent because of adequate collateral circulation.

Patients with mesenteric atherosclerosis may have symptoms of abdominal angina, classically manifested as postprandial pain. As a result, fear of eating, early satiety, weight loss, and altered bowel habits develop. This syndrome occurs in up to 50% of patients in whom thrombotic mesenteric ischemia eventually develops.\(^2\)

**DIFFERENTIAL DIAGNOSIS**

Few diagnoses portend a more serious course and risk for mortality than mesenteric ischemia does. Patients at risk for AMI are generally also at risk for aortic disease. Other items in the differential diagnosis are listed in Box 34.1.

**ANGIOGRAPHY AND COMPUTED TOMOGRAPHIC ANGIOGRAPHY**

Mesenteric angiography provides direct visualization of the vasculature and remains a valuable method of evaluation in patients with suspected bowel ischemia (Table 34.3). Angiography is both sensitive (74% to 100%) and specific (100%); the test also differentiates between occlusive and nonocclusive disease. Patients who undergo angiography in timely fashion have better survival; mortality rates of 70% to 90% are observed when bowel infarction has occurred.\(^4\)

CT angiography (CTA) is largely replacing standard angiography because it is more readily available and more quickly accomplished. A metaanalysis published in 2010 confirmed CTA as the first-line imaging modality because of its high sensitivity (93%) and specificity (96%).\(^5\) CTA is clearly more advantageous in the ED; interventional radiology (IR) involvement later offers additional treatment advantages in the perioperative or observational inpatient phases.

## DOCUMENTATION

Onset, severity, and duration of symptoms
Presence of melena or hematochezia
Presence or absence of risk factors
Vital signs: evidence of shock, sepsis
Findings of cardiac, full abdominal, genitourinary, and rectal examinations
Emergency department course: times of discussions with consultants, discussions with family, code status, availability of testing, treatments, delays

## RED FLAGS

Pain out of proportion to the findings on physical examination
Presence of one or more risk factors
Pain not responsive to narcotics
Rectal bleeding (late finding)

## DIFFERENTIAL DIAGNOSIS

Abdominal aortic aneurysm: rupture or expansion
Perforated ulcer or viscus
Ruptured ectopic pregnancy (woman of childbearing age)
Incarcerated or strangulated hernia
Septic shock
Intussusception
Volvulus
Salpingitis or tuboovarian abscess
Torsion of the ovary or testicle
Appendicitis
Pelvic mass or torsion
Pancreatitus
Diverticulitis
Ruptured ovarian cyst
Renal colic
Biliary colic
Also consider atypical manifestations of:
- Inferior wall myocardial infarction
- Pulmonary embolism
- Pneumonia
- Diabetic ketoacidosis
- Acute glaucoma

*Differential diagnoses are listed in order of urgency.

## BOX 34.1 Differential Diagnosis of Ischemic Bowel Disease*

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>FINDINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior mesenteric artery (SMA) embolus</td>
<td>Filling defects with obstruction of distal flow Secondary vasoconstriction</td>
</tr>
<tr>
<td>Nonobstructive mesenteric ischemia</td>
<td>Narrowing of the origins of SMA branches Irregularities in SMA branches Spasm of the mesenteric arcades Impaired filling of the intramural vessels</td>
</tr>
<tr>
<td>SMA thrombus</td>
<td>Occlusion of the proximal SMA Secondary distal vasoconstriction Absence of collateral flow</td>
</tr>
<tr>
<td>Superior mesenteric vein (SMV) thrombus</td>
<td>Thrombus with partial or complete occlusion Failure to visualize the SMV or portal vein Slowness or absence of filling of the mesenteric veins Failure of the arterial arcades to empty Prolonged blush in the involved segment</td>
</tr>
</tbody>
</table>

Standard angiography allows local administration of thrombolytic or vasodilatory therapy concomitant with the diagnostic procedure, thereby improving mortality. The emergency physician (EP) must weigh the diagnostic and therapeutic advantages of CTA or standard angiography against concerns for dye administration in patients susceptible to renal insufficiency (almost every patient with mesenteric ischemia), availability of an interventional radiologist, stability of the patient, and delays in surgical intervention (Fig. 34.1). In all cases of suspected mesenteric ischemia, the surgeon should be contacted for involvement before or simultaneously with the ordering of advanced diagnostic testing, with the caveat that any delay in diagnosis and operative intervention increases mortality.

With the exception of contrast-enhanced CT scans for mesenteric vein thrombosis, all other imaging tests are fraught with error and lead to diagnostic delays. Standard CT should not be considered an alternative to CTA or standard angiography for the definitive diagnosis of mesenteric ischemia. Once the diagnosis is considered seriously, all testing and consultation should be arranged in parallel rather than sequentially.

**COMPUTED TOMOGRAPHY**

Early CT findings of acute arterial ischemia are poorly sensitive and nonspecific; late findings identify disease in patients with prolonged ischemia and probable necrosis (Table 34.4). CT is the imaging test of choice only for mesenteric vein thrombosis, for which it has a diagnostic accuracy of 90%. Angiography may be avoided when the diagnosis of SMV thrombosis is confirmed by standard CT, although it may still be used for catheter placement and local papaverine infusion.

**OTHER IMAGING MODALITIES**

Magnetic resonance angiography (MRA) with gadolinium enhancement is useful for evaluation of the proximal celiac trunk and SMA. MRA less accurately identifies disease of the IMA, peripheral vessel disease, and NOMI. The findings are similar to those with CT. In the future, rapid MRA may replace angiography for the diagnosis of ischemic bowel disease.

Duplex ultrasonography can assess flow and thrombosis in the SMA or the portal vein. Ultrasonography is frequently limited by the patient’s symptoms, condition, and abdominal distention. It is accurate only for evaluation of proximal vessel disease; false-negative results are common in cases of NOMI or distal disease.

Plain radiographs have no role in the evaluation of acute ischemic bowel disease but are frequently ordered to quickly assess for the presence of processes such as a perforated viscus and free air. Plain film findings are usually normal early in the course of illness. Late findings suggest mucosal edema and hemorrhage and include bowel wall thickening, ileus, and thumbprinting, which describes the appearance of bowel wall edema, as though a thumb had been pressed into the bowel wall and caused an indentation. Pneumatosis intestinalis, the presence of gas in the bowel wall, may also be seen. Air in the portal venous system may likewise be seen (Fig. 34.2). Late findings are associated with a poor prognosis.

**LABORATORY STUDIES**

No laboratory studies are confirmatory of mesenteric ischemia. Delaying diagnostic imaging by waiting for laboratory results decreases survival. Laboratory abnormalities are nonspecific and occur late in disease (Box 34.2); their absence in no way rules out acute ischemia. An elevated serum amylase level is commonly noted with bowel infarction and sometimes leads to an incorrect diagnosis of pancreatitis. The EP should beware of the presence of elevated amylase when the serum lipase value is normal. Diffuse elevation of liver enzymes is seen with hepatic involvement and ischemia.

Levels of ischemia-modified albumin increase in AMI, as well as in several other vascular diseases; their potential role in early diagnosis remains undefined.
Early surgical consultation is a high priority in patients with suspected mesenteric ischemia. Initial ED management should include volume resuscitation, treatment of contributing cardiac abnormalities (dysrhythmias, heart failure, hypotension), and administration of broad-spectrum antibiotics (Box 34.3). Administration of antibiotics decreases the infectious complications of bowel infarction when they are given early to patients with suspected ischemia. The most difficult process for the EP is to see past the present crisis and perform a complete and thorough evaluation of the patient; atrial fibrillation with a rapid ventricular response is a common finding, but hypotension and the altered mental status attributed to it may actually be due to the resultant mesenteric ischemia and sepsis. Look for and pay attention to the other findings pointing to coexisting diagnosis.

Blood tests and radiographs might be ordered to exclude other causes during the wait for surgical consultation and angiography. Placement of a nasogastric tube reduces abdominal distention and may improve the symptoms; nothing should be given by mouth. Blood typing and cross-matching should be performed in the expectation that the patient will undergo surgery. Central venous pressure monitoring may be useful to ensure adequate volume resuscitation in critically ill patients.

Surgical exploration is mandatory when peritonitis is present to remove necrotic bowel and restore blood flow via arterial bypass or embolectomy. A second-look procedure is generally performed 12 to 24 hours after the first operation to evaluate for additional loss of bowel (Fig. 34.3).

When peritonitis and gastrointestinal bleeding are not present, thrombolytic agents or vasodilators (papaverine), or both, are useful measures that may avoid surgery. Thrombolytics given within 12 hours of the onset of symptoms may completely resolve a partially obstructing or distal thrombus.

Vasospasm may become irreversible if not addressed early; it can lead to bowel necrosis even after surgical embolectomy or thrombolysis. Papaverine is infused locally at 30 to 60 mg/hr for vasodilation via an IR-placed angiography catheter for all causes of ischemic bowel disease. Papaverine is routinely and safely administered from the preoperative period to several days after surgery. Vasodilation may be used as monotherapy in patients with minor emboli or those who have major emboli but for whom surgery poses a high risk. Reperfusion of vascular structures distal to the obstruction must be demonstrated by angiography.

Placement of intravascular stents is successful in selected cases of SMA thrombosis. Mesenteric vein thrombectomy is similarly useful in certain cases of mesenteric vein thrombosis.

Anticoagulation is routinely administered postoperatively to patients with mesenteric vein thrombosis. Immediate heparinization after surgery decreases the chance of thrombus recurrence from 25% to 13%, prevents disease progression, and reduces mortality from 50% to 13%. With the absence of peritonitis, anticoagulation may be used in lieu of surgery. If deemed successful after close observation, anticoagulation therapy is generally continued for 3 to 6 months.
Additional therapeutic possibilities currently being explored include the use of heparin-binding epithelial growth factor–like growth factor to protect the intestines and other organs from ischemic injury, percutaneous transluminal angioplasty of the SMA, tolazoline and nitroglycerin as local intraarterial infusions, and prostaglandin E\textsubscript{1} infusions in patients with NOMI.\textsuperscript{12-16}

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

Critical care admission is required for all patients with ischemic bowel disease regardless of whether they are to undergo surgical or medical management.

**PATIENT TEACHING TIPS**

Have an early discussion with the patient and family regarding the severity of the illness, need for an aggressive approach to diagnosis and treatment, and high mortality.

Determine the patient’s and the family’s desire for full resuscitation efforts given the mortality rate associated with mesenteric ischemia.

**SUGGESTED READINGS**


**REFERENCES**

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES


