Acute mesenteric ischemia is a life-threatening vascular emergency that requires early diagnosis and intervention to adequately restore mesenteric blood flow and to prevent bowel necrosis and patient death. The underlying cause is varied, and the prognosis depends on the precise pathologic findings. Despite the progress in understanding the pathogenesis of mesenteric ischemia and the development of modern treatment modalities, acute mesenteric ischemia remains a diagnostic challenge for clinicians, and the delay in diagnosis contributes to the continued high mortality rate. Early diagnosis and prompt effective treatment are essential to improve the clinical outcome.

Acute mesenteric ischemia (AMI) is a potentially fatal vascular emergency with overall mortality of 60% to 80%,1-3 and its reported incidence is increasing.3 Acute mesenteric ischemia comprises a group of pathophysiologic processes that have a common end point—bowel necrosis. The survival rate has not improved substantially during the past 70 years, and the major reason is the continued difficulty in recognizing the condition before bowel infarction occurs.1,8,7

Clinical presentation is nonspecific in most cases and can be characterized by an initial discrepancy between severe abdominal pain and minimal clinical findings. Physical examination does not reliably differentiate between ischemic and infarcted bowel. Complications such as ileus, peritonitis, pancreatitis, and gastrointestinal bleeding may also mask the initial signs and symptoms of AMI. The risk factors for AMI, and the clinical course, differ according to the underlying pathologic condition.6-9 As bowel ischemia rapidly progresses to irreversible bowel necrosis, severe metabolic derangements ensue, leading to a series of events that culminate in multiple organ dysfunction and death. The timely use of diagnostic and therapeutic methods to quickly restore blood flow is the key to reducing the high mortality rate associated with AMI.2,5,7-10

PATHOPHYSIOLOGIC PROCESSES

The splanchnic circulation receives approximately 25% of the resting and 35% of the postprandial cardiac output.11,12 Seventy percent of the mesenteric blood flow is directed to the mucosal and submucosal layers of the bowel, with the remainder supplying the musculairis and serosal layers. The physiologic characteristics of splanchnic blood flow are complex and incompletely understood. Multiple major elements interact to provide the intestinal tract with an appropriate share of the blood supply, including the intrinsic (metabolic and myogenic) and the extrinsic (neural and humoral) regulatory systems.12,13

Pressure-flow autoregulation, reactive hyperemia, and hypoxic vasodilation are considered intrinsic controls and are responsible for instantaneous fluctuations in splanchnic blood flow. In the metabolic theory, oxygen delivery rather than blood flow causes adaptive changes in splanchnic circulation. An imbalance between tissue oxygen supply and demand will raise the concentration of local metabolites (eg, hydrogen, potassium, car-
blood flow include vasopressin, pounds that decrease splanchnic tors. Other pharmacologic com-
stimulation of adrenergic recep-
tense vasoconstriction through the levels of epinephrine produce in-
culation. Norepinephrine and high
inapplicable of affecting the splanchnic cir-
ular. 

The extrinsic neural compo-
ent of splanchnic circulatory regu-
lation comprises the α-activated vasoconstrictor fibers. Intense activ-
ation of vasoconstrictor fibers through α-adrenergic stimulation re-
results in vasoconstriction of small ves-
sels and a decrease in mesenteric blood flow. After periods of pro-
lnged α-adrenergic vasocon-
striction, blood flow increases, presumably through β-adrenergic stimula-
tion, which acts as a protective response. After cessation of α-adrenergic stimulation, brief hyperemia makes the response tri-
phasic. Although various types of neural stimulation (eg, vagal, cholinergic, histaminergic, and sympa-
thetic) can affect the gut, the adrener-
ergic limb of the autonomic nervous system is the predominant and pos-
sibly the sole neural influence on splanchic circulation.

Numerous endogenous and ex-
genous humoral factors are cap-
able of affecting the splanchic cir-
ulation. Norepinephrine and high levels of epinephrine produce in-
tense vasoconstriction through the stimulation of adrenergic recep-
tors. Other pharmacologic com-
ponents that decrease splanchic blood flow include vasopressin, phenylephrine, and digoxin.14 Low-
dose dopamine causes splanchic vasodilation, whereas higher doses lead to vasoconstriction by stimu-
lating α-adrenergic receptors. Pap-
averine, adenosine, dobutamine, fenoldopam mesylate, and sodium nitroprusside are exogenous agents that increase mesenteric blood flow. In addition, various naturally occur-
ing agents can serve as splanchic vasodilators, including acetylcholine, histamine, nitric oxide, leuko-
atrienes, thromboxane analogues, glucagon, and an assortment of gas-
trointestinal hormones. The effects of prostaglandins are variable.

Table 1. Physiologic and Pharmacologic Factors Regulating Mesenteric Blood Flow (Extrinsic Regulatory System)

<table>
<thead>
<tr>
<th>Decrease Blood Flow</th>
<th>Increase Blood Flow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine (high dose)</td>
<td>Epinephrine (low dose)</td>
</tr>
<tr>
<td>Norepinephrine (moderate to high dose)</td>
<td>Norepinephrine (low dose)</td>
</tr>
<tr>
<td>Dopamine (high dose)</td>
<td>Dopamine (low dose)</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>Dobutamine</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Sodium nitroprusside</td>
</tr>
<tr>
<td>Angiotensin II</td>
<td>Papaverine</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Nitric oxide</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>α-Adrenergic receptors</th>
<th>β-Adrenergic receptors</th>
</tr>
</thead>
</table>

In summary, the splanchic cir-
culation is regulated by a complex array of physiologic and pharmaco-
logic factors (Table 1).

**REPERFUSION INJURY**

Tissue damage due to alterations in mesenteric blood flow is often the result of cellular injury associated with reperfusion.15,16 Brief periods of mesenteric ischemia lead to an increase in microvascular permeabil-
ity, whereas prolonged ischemia leads to disruption of the intestinal mucosal barrier, primarily through the actions of reactive oxygen me-
tabolites and polymorphonuclear neutrophils.

The role of oxygen free radicals in reperfusion injury is demon-
imated by the reduction of tissue damage in the presence of anti-
oxidants, xanthine oxidase inhibitors, and free-radical scavenging sub-
stances. Polymorphonuclear leuko-
cytes contain enzymes that reduce molecular oxygen to superoxide an-
ions and produce hypochloric acid, providing an additional source of re-
active oxygen metabolites. Epithelial cells may produce xanthine oxida-
tase–derived oxidants and initiate the production of proinflammatory agents that attract polymorpho-
uclear leukocytes.17 In addition, phospholipase A2 is activated during reperfusion, increasing the for-
mation of cytotoxic lysophospholip-
ids within the ischemic tissue and up-regulating the production of pros-
taglandins and leukotrienes.18 Fur-
ther understanding of the role of reperfusion injury may present op-
portunities for protective pharmaco-
logic therapies with agents such as captorpril and carvedilol.19,20 Carvedi-
ilol, a new β-adrenoreceptor block-
ing agent and a free-radical scavenger, has been demonstrated to have an antishock and endothelial-
protective effect in a rat splanchic ischemia reperfusion model.20

The degree of reduction in blood flow that the bowel can tol-
erate without activating these reper-
fusion mechanisms is remarkable. Only one fifth of the mesenteric cap-
illaries are open at any given time, and normal oxygen consumption can be maintained with only 20% of maximal blood flow. When splanch-
nic blood flow is restored, oxygen extraction increases, providing rela-
tively constant oxygen consump-
tion over a wide range of blood flow rates.12 However, when blood flow decreases below a threshold level, oxygen consumption is reduced and oxygen debt ensues.

**CAUSE**

Acute mesenteric ischemia can be categorized into 4 specific types based on its cause (Table 2).

**Arterial Embolism**

Arterial emboli are the most fre-
quent cause of AMI and are responsible for approximately 40% to 50% of cases.2,3 Most mesenteric emboli originate from a cardiac source. Myocardial ischemia or infarction, atrial tachyarrhythmias, endocardi-
tis, cardiomyopathies, ventricular aneurysms, and valvular disorders are risk factors for the development of mural thrombus, which can...
subsequently embolize to mesenteric arteries.21 Rarely, a mesenteric artery embolus can occur during or after angiography of the coronary or cerebral circulation. Most visceral arterial emboli preferentially lodge in the superior mesenteric artery (SMA) because it emerges from the aorta at an oblique angle. Whereas 15% of arterial emboli occur at the origin of the SMA, 50% lodge distally to the origin of the middle colic artery, which is the first major branch of the SMA.5,21 Nearly one third of all patients with an SMA embolus have a history of an antecedent embolic event.

The onset of symptoms is usually dramatic as a result of the poorly developed collateral circulation, and it is characterized by the abrupt onset of severe abdominal pain associated with diarrhea, which may become bloody. Frequently, the diagnosis of SMA embolism can be made intraoperatively based on the distribution of ischemic bowel. Because most SMA emboli lodge distally to the origin of the middle colic artery, allowing the inferior pancreaticoduodenal branches to be perfused, the proximal jejunum is spared, whereas the rest of the small bowel is ischemic or infarcted.

**Table 2. Clinical Features of Acute Mesenteric Ischemia**

<table>
<thead>
<tr>
<th>Cause</th>
<th>Incidence, %</th>
<th>Presentation</th>
<th>Risk Factors</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial embolism</td>
<td>40-50</td>
<td>Acute catastrophe</td>
<td>Arrhythmia, myocardial infarction, rheumatic valve disease, endocarditis,</td>
<td>Embolectomy, papaverine, excise</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>cardiomyopathies, ventricular aneurysms, history of embolic events,</td>
<td>infarction</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>recent angiography</td>
<td></td>
</tr>
<tr>
<td>Arterial thrombosis</td>
<td>25</td>
<td>Insidious onset with</td>
<td>Atherosclerosis, prolonged hypotension, estrogen, hypercoagulability</td>
<td>Papaverine, thrombectomy, excise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>progression to constant</td>
<td></td>
<td>infarction, revascularization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonocclusive</td>
<td>20</td>
<td>Acute or subacute</td>
<td>Hypovolemia, hypotension, low cardiac output status, α-adrenergic agonists,</td>
<td>Treat cause first, papaverine,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>digoxin, β-receptor blocking agents</td>
<td>excise dead bowel</td>
</tr>
<tr>
<td>Venous thrombosis</td>
<td>10</td>
<td>Subacute</td>
<td>Right-sided heart failure, previous deep vein thrombosis, hepatosplenomegaly,</td>
<td>Thrombectomy, excise dead bowel,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>primary clotting disorder, malignancy, hepatitis, pancreatitis, recent</td>
<td>heparinize, long-term</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>abdominal surgery or infection, estrogen, polycythemia, sickle cell</td>
<td>complication</td>
</tr>
</tbody>
</table>

**Nonocclusive Mesenteric Ischemia**

Approximately 20% of patients with mesenteric ischemia have nonocclusive disease.8,23 The pathogenesis of nonocclusive mesenteric ischemia (NOMI) is poorly understood but often involves a low cardiac output state associated with diffuse mesenteric vasoconstriction. Splanchnic vasoconstriction in response to hypovolemia, decreased cardiac output, hypotension, or vasopressors best explain the difference between this entity and other forms of AMI. The resultant low-flow state causes intestinal hypoxia and necrosis. Endogenous and exogenous vasoconstrictors, disseminated intravascular coagulation, and reperfusion injury may also contribute. Vasoactive drugs, particularly digoxin, have been implicated in the pathogenesis of NOMI. Digitalis preparations induce contraction of splanchnic venous and arterial vascular smooth muscle in vitro and in vivo.19 Watershed areas of circulation are more vulnerable in NOMI.

Conditions predisposing to NOMI include age older than 50 years, myocardial infarction, congestive heart failure, aortic insufficiency, cardiopulmonary bypass, renal or hepatic disease, and major abdominal or cardiovascular surgery. However, patients may not have any clear risk factors.10,22 Because this condition frequently affects critically ill patients who have considerable comorbidities, the onset may be insidious, and the mortality rates are high. Between 1960 and 1980, because of the frequent use of vasopressors in cardiac patients, mortality was nearly 100%. With increasing use of afterload-reducing agents and vasodilators, the mortality rate associated with NOMI has declined.

An unusual form of nonocclusive ischemia has been described in patients who have undergone the stress of a surgical procedure or trauma and are receiving enteral nutrition in intensive care units.23 The reported incidence of AMI in these patients is 0.3% to 8.5%. The proposed mechanism is an imbalance between demand (created by the enteral feedings) and supply (decreased by systemic hyperperfusion and mesenteric vasoconstriction). Most pa-
tients manifest signs of sepsis, with abdominal distention as a late clinical sign. Survival is poor (56%).

Mesenteric Venous Thrombosis

Mesenteric venous thrombosis (MVT) is the least common cause of mesenteric ischemia, representing up to 10% of all patients with mesenteric ischemia and 18% of those with AMI. In the past, most cases were thought to be secondary to other intra-abdominal pathologic conditions (such as malignancy, intra-abdominal sepsis, or pancreatitis) or were classified as idiopathic. With improved diagnostic techniques, more cases have been shown to be related to primary clotting disorders, with only 10% of cases now being classified as idiopathic.2,25-26

Mesenteric venous thrombosis is usually segmental, with edema and hemorrhage of the bowel wall and focal sloughing of the mucosa. Thrombi usually originate in the venous arcades and propagate to involve the arcuate channels. Hemorrhagic infarctions occur when the intramural vessels are occluded. The thrombus is usually palpable in the superior mesenteric vein.27 Involvement of the inferior mesenteric vein and large bowel is uncommon. The transition from normal to ischemic intestine is more gradual with venous embolism than with arterial embolism or thrombosis.

Mortality depends on the type of MVT (acute vs chronic) and the extent of venous involvement. Patients with acute disease with involvement of the superior mesenteric or portal vein have a 30-day mortality approaching 30%. Long-term survival is 30% to 40% in patients with acute MVT compared with at least 80% in those with the chronic form.26

PRESENTATION

Many of the signs and symptoms associated with AMI are common to other intra-abdominal pathologic conditions, such as pancreatitis, acute diverticulitis, small-bowel obstruction, and acute cholecystitis. In addition, the clinical presentation often depends on the underlying pathologic abnormalities. In general, patients with SMA embolism or thrombosis have an acute onset of symptoms and a rapid deterioration in their clinical condition, whereas those with NOMI or MVT have a more gradual onset and a more protracted clinical course.

With SMA embolism, the onset of symptoms is usually dramatic because of lack of collateral circulation, and it manifests as severe and unrelenting abdominal pain, nausea, vomiting, and urgent bowel evacuation. Classically, the severity of abdominal pain is out of proportion to the physical findings. Dehydration and excessive fluid loss from third-spacing of fluid lead to mental confusion, tachycardia, tachypnea, and circulatory collapse. Laboratory findings include metabolic acidosis with elevated anion gap and lactate levels, leukocytosis, and hemoconcentration.

Patients with SMA thrombosis frequently report a prodromal symptom complex of postprandial pain, nausea, and weight loss associated with chronic intestinal insufficiency.3,24 Patients with a subacute onset tend to seek medical care much later than those with arterial emboli.2,27 However, when ischemia from mesenteric thrombosis becomes acute, patients present similarly to those who have acute SMA embolism.

Nonocclusive mesenteric ischemia occurs most frequently in elderly, critically ill patients and in those with severe mesenteric atherosclerosis in the setting of an acute hemodynamic insult. Such patients are often intubated and sedated and, therefore, are unable to alert the clinician to their symptoms. In these circumstances, the intestinal ischemia may not become clinically evident until hours or days after the initial hemodynamic insult. This is particularly important in cases of severe hypotension treated with α-adrenergic agonists. The hemodynamic insult and its treatment predispose the patient to NOMI. These patients frequently experience unexplained worsening in their clinical condition or a failure to thrive or to follow their anticipated recovery course.

Except in the most fulminant cases, patients with MVT typically present late (ie, 1-2 weeks after onset), complaining of diffuse, non-specific abdominal pain associated with anorexia and diarrhea. If the pain is localized, it is most often in the lower quadrants. Compared with arterial thrombosis, MVT generates fewer prodromal symptoms with eating or postprandial pain. Fever, abdominal distention, and Hemocult-positive stool samples are the most common findings. Bloody ascites and large fluid losses with third-spacing may occur, leading to dehydration and hypotension, causing further propagation of the venous thrombosis and worsening of the mesenteric ischemia.

The final common pathway of all the specific causes of mesenteric ischemia is bowel infarction. When infarction occurs, the patient has peritoneal signs, hemodynamic instability, and signs of sepsis with multiorgan failure.

DIAGNOSIS

Because AMI may proceed to fatal intestinal infarction rapidly, prompt diagnosis and treatment are paramount. A high index of suspicion in the setting of a compatible history and physical examination serves as the cornerstone to early diagnosis of mesenteric ischemia.5,7,22 Once it is suspected, the clinician should act promptly to confirm the diagnosis and initiate appropriate treatment. Acute mesenteric ischemia should particularly be considered in the differential diagnosis when a patient is older than 60 years; has a history of atrial fibrillation, recent myocardial infarction, congestive heart failure, arterial emboli, or postprandial abdominal pain and weight loss; and is initially seen for abdominal pain that is out of proportion to that suggested by physical examination. Survival is approximately 50% when diagnosis occurs within 24 hours after onset of symptoms, but it drops sharply to 30% or less when diagnosis is delayed.21

The most common laboratory abnormalities are hemoconcentration, leukocytosis, and metabolic acidosis, with high anion gap and lactate concentrations. High levels of serum amylase, aspartate aminotransferase, lactate dehydrogenase,
Barium enema has no place in the diagnosis of AMI. The introduction of barium and air may increase intraluminal pressure, causing reduced perfusion to the bowel wall, translocation of bacteria, and, potentially, perforation. In addition, the presence of barium may compromise subsequent diagnostic tests, such as computed tomography (CT) and angiography. Rarely, the barium enema can be useful in the diagnosis of colonic ischemia, but it has essentially been replaced by flexible sigmoidoscopy or colonoscopy.

Recent interest in CT for the diagnosis of AMI has resulted in several studies describing thickened bowel walls, intramural hematoma, dilated fluid-filled bowel loops, engorgement of mesenteric vessels, pneumatosis, mesenteric or portal venous gas, infarction of other viscera, and arterial or venous thrombus. Abdominal CT has poor sensitivity and specificity in mesenteric ischemia, its role in the diagnosis of AMI seems limited. Magnetic resonance imaging has been used to identify infarcted bowel in animals, but clinical studies in humans have yet to be performed. Doppler ultrasonography has been used to detect a significant stenosis (>50%) in the mesenteric vessels in patients with chronic mesenteric arterial occlusive disease, but its role in AMI seems limited. Magnetic resonance imaging has shown promise in detecting altered flows in the superior mesenteric vessels in chronic ischemia, but its reliability has not been documented in controlled trials. The relatively long time needed by most medical centers for scheduling and performing magnetic resonance imaging has made its use impractical in this rapidly progressive disorder.

Peritoneoscopy may also be a useful tool for investigating AMI due to venous thrombosis. Serosanguineous fluid in the abdominal cavity of an older patient with abdominal pain, hemocoagulation, and leukocytosis is strongly suggestive of MVT.

**TREATMENT**

Once the diagnosis of AMI is made, treatment should be initiated without delay. This should include active resuscitation and treatment of the underlying condition, with efforts directed toward reducing the associated vasospasm, preventing propagation of the intravascular clotting process, and minimizing the
reperfusion injury. An algorithm summarizing treatment procedures is shown in the Figure.

Intravenous fluid resuscitation with crystalloids and blood products should be started promptly to correct the volume deficit and metabolic derangement. Placement of a Swan-Ganz catheter may be required for judicious fluid resuscitation and hemodynamic monitoring, especially in critically ill patients. Ideally, fluid resuscitation should begin before angiography, and crystalloids may be administered in amounts as high as 100 mL/kg. Supranormalization of hemodynamic values has been attempted, with equivocal results, and it remains to be proven whether such an approach offers an advantage to patients with AMI.

Broad-spectrum antibiotics should be given as early as possible. If there are no contraindications to anticoagulation, therapeutic intravenous heparin sodium should be administered to maintain the activated partial thromboplastin time at twice the normal value. After the patient’s hemodynamic condition has been optimized and anticoagulation therapy has been initiated, efforts should aim at reducing the mesenteric vasospasm. If the diagnosis of AMI is made without the use of mesenteric arteriography, intravenous glucagon infused initially at 1 µg/kg per minute and titrated up to 10 µg/kg per minute as tolerated may help reduce the associated vasospasm. When angiography is used to establish the diagnosis, the angiographic catheter should be left in the SMA for infusions of papaverine or other vasodilators. Papaverine, a phosphodiesterase inhibitor, increases mesenteric blood flow to marginally perfused tissues and may considerably improve bowel salvage. The usual dose is 30 to 60 mg/h. Papaverine use is recommended in cases of arterial embolic or nonocclusive disease because in both conditions the arterial vasospasm persists even after successful treatment of the precipitating event.

The presence of peritoneal signs generally indicates bowel infarction rather than ischemia alone and mandates emergency laparotomy. Even in the absence of bowel necrosis, surgical procedures are generally required, except in NOMI, in which the management is primarily medical. Therapeutic improvement during preoperative resuscitation may offer a false sense of security, but bowel infarction, sepsis, and multiple organ failure usually follow unless laparotomy, revascularization, and excision of infarcted bowel segments are performed. Visceral revascularization (embolectomy, thrombectomy, endarterectomy, or bypass) should precede bowel resection in almost all patients with occlusive AMI.

For acute mesenteric embolism, a standard embolectomy via a transverse arteriotomy in the proximal SMA should be performed. After embolectomy, the arteriotomy is closed primarily with interrupted nonabsorbable sutures (polypropylene). If the cause of an acute mesenteric embolism is in doubt or if SMA thrombosis is suspected, then a longitudinal arteriotomy is preferred. With this ap-
proach, if a bypass is necessary, the longitudinal arteriotomy can be used as the site for the distal bypass graft anastomosis. If flow is adequately re-established without a bypass, then the longitudinal arteriotomy can be closed by patch angioplasty to ensure that the luminal diameter is not compromised.

For AMI from arterial thrombosis due to atherosclerotic disease, a vascular bypass graft is usually necessary. The graft can originate from the infrarenal or supraceliac aorta. To avoid potential intra-abdominal contamination from perforated, infarcted bowel, revascularization should be performed using an autologous vein.

As in embolic disease, revascularization should be performed first, with subsequent resection of clearly nonviable bowel. This allows preservation of potentially viable gut and reduces the possibility of creating a "short-gut syndrome." If the adequacy of perfusion to the bowel is in question, the ends of the bowel may be brought out as stomas. Although nonspecific, the presence of arterial pulsations and the return of bowel peristalsis and normal bowel color suggest intestinal viability.

Visual examination of the exterior of the bowel is unreliable, especially in cases of NOMI, in which the serosa may appear viable despite the presence of infarcted mucosa. The use of intravenous fluorescein and inspection under a Wood lamp has been shown to be more sensitive and specific, but this method is not widely accepted.46,50 Uniform uptake of fluorescein generally suggests bowel viability. Patchy uptake suggests questionable bowel viability; and these segments are better left in situ, with a plan for a "second-look" operation. Doppler ultrasonography is an alternative and may be used intraoperatively, but studies have not demonstrated any advantage over clinical judgment in assessment of bowel viability.31

Effective treatment of NOMI largely depends on the underlying cause. Initial therapy should aim at removing the offending stimulus and correcting the underlying medical condition. Logically, any infarcted bowel must be resected. Vasodilators, anticoagulation, and mesenteric regional blockade have been used in cases in which infarction has not yet occurred. Occasionally, direct transcatheter infusion of papaverine into the SMA restores normal blood flow within minutes. However, patients with NOMI have fewer treatment options than those with other forms of AMI because the offending stimulus often originates from the necessary treatment of another disorder. Unless the original provocation or insult is reversed, mortality in NOMI is similar to that in other forms of AMI.

Treatment of MVT is somewhat controversial and depends on the extent of intestinal ischemia. Patients without evidence of bowel infarction often recover spontaneously without operative intervention, and many are treated with anticoagulation alone. The presence of peritoneal signs necessitates emergency laparotomy. Infarcted bowel should be widely resected. Postoperatively, recurrence and progression of thrombosis are common. Heparinization has been shown to reduce the recurrence of thrombosis from 26% to 14% and mortality from 59% to 22%.52,53 For acute main superior mesenteric vein or portal vein thrombosis, a thrombectomy may be beneficial. Cases of successful treatment with intravascular thrombolytic agents have also been reported.52-54 However, thrombolysis is contraindicated when bowel infarction is suspected. After initial treatment of the acute event, the possibility of thrombophilia should be investigated. If a prothrombotic condition is detected, long-term warfarin therapy may be necessary.

Even when the primary operation is successful, the intraoperative assessment of bowel viability is often inaccurate, and few reliable signs are available to detect persistent ischemia or developing infarction in the postoperative period. For this reason, a second-look laparotomy after 24 to 48 hours is usually recommended.3,55 The rationale for this second look is based in part on the frequent occurrence of vasospasm after revascularization. Second-look laparoscopy has been advocated as a substitute for second-look laparotomy, but the reliability of this approach remains unproved.56,57

Postoperatively, patients treated for AMI are invariably critically ill. Metabolic acidosis and hyperkalemia should be aggressively corrected. Persistent acidosis, especially in the absence of renal failure, should raise concerns about ongoing uncorrected bowel ischemia or infarction. Adequate volume resuscitation is essential to avoid persistent mesenteric hypoperfusion. The mesenteric capillary leak syndrome after mesenteric revascularization is well recognized. Frequently, patients with this condition require 10 to 20 L of crystalloid resuscitation during the first 24 to 48 hours after surgery.

After successful revascularization, efforts should be directed toward limiting any reperfusion injury that may cause progressive mesenteric ischemia or infarction. If the patient's hemodynamic condition allows, infusion of vasodilators should be considered (intravenous glucagon or intra-arterial papaverine). The use of allopurinol, angiotensin-converting enzyme inhibitors, and other free oxygen scavengers may help reduce the reperfusion syndrome.

Sepsis and multiple organ dysfunction syndromes occur in many patients with AMI.59 The presentation and management of such complications are similar to those of complications from other causes; however, the use of vasopressors may worsen ischemia in marginally viable bowel and exacerbate the condition. Vasopressor options include dopamine (3-8 µg/kg per minute) and epinephrine (0.05-0.10 µg/kg per minute); pure α-adrenergic agents should be avoided, if possible.

PROGNOSIS

Perioperative mortality in patients undergoing revascularization for AMI ranges from 44% to 90%.59 Published data on long-term results after successful revascularization are few, and, in general, prognosis is not as favorable as that for patients with chronic mesenteric ischemia. Recurrence is not uncommon, and it carries a poor prognosis. A small proportion of patients survive massive bowel resection and develop short-
Acute mesenteric ischemia is a challenging clinical problem with diverse causes, which often results in delayed diagnosis and treatment. A strong clinical suspicion and an aggressive approach should be adopted in dealing with this condition because the outcome crucially depends on rapid diagnosis and treatment. With better understanding of the pathogenesis of AMI and the availability of a range of diagnostic and interventional techniques and adjuvant pharmacotherapies, an improved outcome can be achieved.

CONCLUSIONS

Acute mesenteric ischemia is a challenging clinical problem with diverse causes, which often results in delayed diagnosis and treatment. A strong clinical suspicion and an aggressive approach should be adopted in dealing with this condition because the outcome crucially depends on rapid diagnosis and treatment. With better understanding of the pathogenesis of AMI and the availability of a range of diagnostic and interventional techniques and adjuvant pharmacotherapies, an improved outcome can be achieved.

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