Mesenteric arterial occlusive and aneurysmal disease
James C. Stanley, MD
University Hospital, 1500 East Medical Center Drive, Ann Arbor, MI 48109-0329, USA

Mesenteric ischemia

Severe reductions in intestinal blood flow result in cellular hypoxia, accumulation of oxygen free radicals, elevation of portal venous pressure, adverse effects of digestive enzymes on gut tissues, and intraluminal bacterial proliferation. In instances of severe ischemia, the cellular coverage of the villus in its entirety can be destroyed before reperfusion. Reactive oxygen metabolites cause further injury with reperfusion of ischemic intestinal tissues. The fact that ischemic injury often results in invasion of the microcirculation by intestinal bacteria (as evidenced by the presence of portal venous gas; Fig. 1) means that intestinal ischemia is much more damaging than ischemia that affects other tissues such as skeletal or cardiac muscle.

The small intestine can withstand modest reductions in blood flow without ischemic injury as long as there is not more than a 50% decrease in oxygen consumption. Certain drugs that influence mesenteric blood flow (such as glucagon) frequently have major effects on intestinal motility. Agents that decrease intestinal motility might lessen the oxygen needs of the gut. At the same time they relax the intestine’s muscularis mucosa, with subsequent decreases in the resistance to blood flow from submucosal vessels to mucosal and villous vessels.

Three categories of mesenteric ischemia deserve note, including (1) chronic arteriosclerotic occlusive disease, (2) acute embolic occlusive disease, and (3) low-flow nonocclusive ischemia (Table 1).

Chronic arteriosclerotic occlusive disease

Chronic mesenteric ischemia secondary to arteriosclerosis in its classic form is well recognized and its treatment has been widely reported [1–16]. Two of the three major splanchnic vessels (celiac, superior mesenteric, and inferior mesenteric arteries) must usually be severely diseased to produce clinical symptoms (Fig. 2). Isolated superior mesenteric artery atherosclerosis located beyond its proximal inferior pancreaticoduodenal and middle colic branches prevents collateral blood flow from the celiac and inferior mesenteric arterial circulations, respectively, and can cause chronic mesenteric ischemia. Splanchnic arterial occlusive disease invariably occurs as aortic spillover atherosclerosis involving the ostia of these vessels and coexistence of significant disease in the mesenteric branches is often overlooked. This is a potentially serious oversight, especially in diabetics, in whom diffuse disease extends into the smaller vessels, akin to similar arteriosclerotic disease in coronary, renal, and lower extremity vessels.

A predilection for women to develop splanchnic arteriosclerosis is clearly dissimilar to the male gender predominance of arteriosclerosis elsewhere. The basis for this gender difference is unknown, but these individuals might have pre-existing developmental intimal cushions or hypoplastic vessels that carry a propensity to accelerated arteriosclerosis. There appears to be a bimodal distribution of female patients having chronic mesenteric ischemia, including (1) women in their fifth and sixth decades of life who have a history of heavy cigarette consumption, often in the neighborhood of 50 or more pack years, and (2) more elderly women in their seventh or eighth decades of life who have generalized arteriosclerotic cardiovascular disease with underlying lipid disorders and hypertension.

Clinical sequelae of chronic mesenteric ischemia caused by arteriosclerosis are defined by two events. First is the rapidity with which the occlusive disease evolves, and second is the adequacy of the evolving collateral circulation. Individuals
can exhibit severe splanchnic arterial occlusive disease without clinical symptoms because of the extensive collateral network providing blood flow to their intestines. Unfortunately, the incidence of splanchnic arteriosclerosis in the general population and the frequency with which it causes acute or chronic symptoms have never been established. Most clinical descriptions of chronic mesenteric ischemia involve patients undergoing surgical therapy, and as such these reports represent a select group of individuals who might not be representative of the disease in the general population.

Patients carrying a diagnosis of chronic mesenteric ischemia have a mean age of 60 years with abdominal discomfort that has averaged 18 months in duration. In most patients mid-abdominal and epigastric discomfort begins 10 to 15 minutes following consumption of large meals initially, and somewhat later when ingesting smaller amounts of food. This pain is often referred to as abdominal angina, intestinal angina, or mesenteric angina. The duration of abdominal discomfort ranges from 1 to 4 hours, and it is thought to represent the time required for transit of food through the small bowel. This discomfort may be ill-defined initially, but it usually becomes more severe with time. In the advanced stages of chronic mesenteric ischemia, patients develop the familiar “small meal” syndrome because of a fear of eating. This form of sitophobia results in progressive weight loss, usually averaging 10 to 15 kg by the time that a diagnosis of chronic mesenteric ischemia is entertained.

In certain patients, collateral circulation might be insufficient to sustain bowel viability and intestinal infarction can occur. The literature is replete with case histories of chronic intestinal ischemia progressing to infarction. The majority of patients with chronic mesenteric ischemia are more likely to have modest symptoms that are difficult to differentiate from other intra-abdominal disease processes for long time periods before they develop manifestations of intestinal ischemia with infarction. Occasional patients with splanchnic vascular disease present with gastric ischemia[17]. They experience dyspepsia and exhibit evidence of mul-

Fig. 1. Portal venous air bubbles (arrows) associated with invasion of profoundly ischemic bowel wall by gas-forming bacteria. (Reprinted with permission from Wakefield TW, Stanley JC. The intestine. In: Zelenock GB, editor. Clinical ischemic syndromes: mechanisms and consequences of tissue injury. St. Louis, MO: CV Mosby; 1990.)

multiple antral ulcers that appear to be resistant to conservative therapy. The presence of such findings when associated with weight loss and a fear of eating large quantities of food should suggest the possibility of underlying mesenteric ischemia. Diagnostic criteria pathognomonic of ischemic intestinal dysfunction do not exist. In reality, significant weight loss, postprandial abdominal pain, and the anatomic presence of splanchnic arterial occlusive lesions are the best indicators of chronic mesenteric ischemia, but these findings are far from being diagnostic of the disease. Nevertheless, deep abdominal ultrasonography has proven to be an increasingly useful noninvasive means of evaluating patients suspected of having arteriosclerotic stenoses of the proximal celiac and superior mesenteric arteries [5,18,19]. Peak systolic flow velocities of greater than 275 and 200 cm/second in these two vessels, respectively, are compatible with a 70% stenosis. Gadolinium-enhanced magnetic resonance angiography with a breath-hold technique provides a second safe method of revealing proximal celiac and superior mesenteric arterial stenoses [20–22].

Conventional contrast arteriography with or without digital enhancement provides the most information in defining the anatomy of splanchnic circulation. Lateral aortography is essential in establishing the presence of atherosclerotic narrowings involving the origins of the three major splanchnic arteries. Among patients reported to have chronic mesenteric ischemia undergoing operative intervention, 85% have severe stenoses or actual occlusion of their celiac and superior mesenteric arteries. In 50% of these patients the inferior mesenteric artery also exhibits a critical stenosis, but the severity of stenotic disease affecting this artery is usually less than that affecting the celiac and superior mesenteric arteries. The simple relation of stenotic disease to the existence of clinical symptoms must be viewed with caution because 6% to 10% of the general population have critical lesions affecting their major splanchnic vessels at death, and similar lesions are found in 14% to 24% of individuals undergoing aortography, usually for vascular disease. Few of these patients have symptoms suggestive of mesenteric ischemia. Thus, documentation of occlusive lesions

### Table 1: Mesenteric Ischemic Disease

<table>
<thead>
<tr>
<th>Category</th>
<th>Acute Embolic Occlusion</th>
<th>Low-Flow States</th>
<th>Chronic Arteriosclerotic Occlusive Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usual Pathology</td>
<td>Embolic occlusion of SMA beyond collaterals from CA and IMA, from dislodged atrial thrombus (due to atrial fibrillation)</td>
<td>Cardiac failure</td>
<td>Aortic spillover arteriosclerosis causing critical stenoses of at least two of the three major splanchnic arteries. Usually involves orificial stenoses of CA and SMA.</td>
</tr>
<tr>
<td>Gender Ratio (M:F)</td>
<td>2:1</td>
<td>1:5:1</td>
<td>1:2</td>
</tr>
<tr>
<td>Clinical Manifestation</td>
<td>Marked disparity between acute excruciating midabdominal pain and a paucity of early physical findings</td>
<td>3 to 4 day history of vague abdominal pain, distention</td>
<td>Gradual, but progressive development of postprandial pain, lasting 3–4 hours. Associated “small meal syndrome” and weight loss.</td>
</tr>
<tr>
<td>Diagnostic Studies</td>
<td>Emergent arteriography, operative diagnosis</td>
<td>Arteriography, occasional barium enema in subacute cases revealing colon “thumb-prints”</td>
<td>Lateral aortography.</td>
</tr>
<tr>
<td>Treatment</td>
<td>Emergent SMA embolectomy, chronic anticoagulation</td>
<td>Improve cardiac function, vasodilators (glucagon) and fluids</td>
<td>Bypass or endarterectomy of CA and SMA.</td>
</tr>
<tr>
<td>Mortality</td>
<td>Operative mortality 60% without intestinal infarction, 95% with intestinal infarction</td>
<td>Low, but 60% with intestinal infarction</td>
<td>Operative mortality less than 10%.</td>
</tr>
</tbody>
</table>
is useful in supporting a diagnosis of chronic mesenteric ischemia, but such lesions are not confirmatory of the clinical disease.

Treatment of chronic intestinal ischemia caused by arteriosclerotic disease necessitates revascularization of the intestines. Endarterectomy of the celiac and superior mesenteric arteries or bypass procedures involving these vessels are the most successful means of restoring normal blood flow to the intestines. The contemporary results of surgical interventions for chronic mesenteric ischemia are excellent. In general, the overall operative mortality among cumulated experiences approaches 7.5% with salutory long-term benefits in 87% of survivors. Endarterectomy appears to have the lowest recurrence of clinical symptoms, followed by antegrade bypass reconstructions, then retrograde reconstructions. Recurrent symptoms of chronic mesenteric ischemia occur disproportionately in female patients and patients who are heavy smokers. Re-operations provide salutory outcomes in nearly 75% of individuals with recurrent ischemia [14]. Splanchnic revascularizations benefit the majority of patients with chronic mesenteric ischemia.

Experience with percutaneous balloon angioplasty is limited, and the procedure carries the risk of acute embolic infarction of the intestines. Nevertheless, in select patients, especially those at high risk for open surgical procedures, catheter-based therapy might be most appropriate [1,7,8,10,11,13]. In patients who are capable of withstanding surgical reconstruction, percutaneous angioplasty with stenting does not provide benefits comparable to those of the open procedure [7].

Clinicians should not forget about the possibility of mesenteric venous thrombosis as an etiology for abdominal pain and weight loss mimicking typical mesenteric arterial ischemic syndromes. If evaluation for mesenteric arterial disease is negative in a patient with appropriate symptoms, a CT scan with contrast or a venous phase of a mesenteric arteriogram should be obtained to be certain that the diagnosis of mesenteric venous thrombosis is not overlooked.

**Acute embolic occlusive disease**

The most catastrophic form of intestinal ischemia is macroembolism involving the superior mesenteric artery [23–25]. Such emboli have a cardiac origin in nearly 95% of patients, with most originating from atrial thrombi associated with arteriosclerotic heart disease and atrial fibrillation. Ventricular thrombi following myocardial infarction are a less common cause. Men are affected twice as often as women. Half of these patients had experienced earlier embolism of extraintestinal vessels, most often involving the femoral or popliteal arteries. Superior mesenteric artery emboli usually lodge beyond the origin of the inferior pancreaticoduodenal and middle colic arteries (Fig. 3), which effectively isolates the distal superior mesenteric arterial circulation and causes critical ischemia of the distal jejunum, entire ileum, and right colon.
Patients experiencing acute superior mesenteric artery embolic occlusions usually present with unremitting, intense mid-abdominal pain with nausea and vomiting that might be accompanied by explosive diarrhea. Early physical examination might be normal, without evidence of abdominal tenderness, rigidity, or mass lesions. The diagnosis should be suspected in the face of such a disparity in the severity of symptoms and the absence of physical findings.

Leukocytosis, hemoconcentration, and systemic acidosis often accompany embolic occlusion of the superior mesenteric artery. Bowel infarction can be accompanied by elevated levels of serum amylase, inorganic phosphorous, creatinine phosphokinase, and alkaline phosphatase, but none of these are reliable biochemical markers of intestinal ischemia.

Early radiographic features of superior mesenteric artery embolism are usually minimal. Tissue invasion by gas-forming organisms with evidence of mesenteric or portal venous gas on abdominal films implies a grave prognosis. Computed tomography can document intravascular gas and intestinal wall thickening accompanying gut infarction.

Patients suspected of acute superior mesenteric artery embolism should undergo emergent operative embolectomy. Arteriographic studies should be obtained when the diagnosis is in question. Such studies usually reveal a characteristic meniscus sign at the site of acute embolic occlusion. Revascularization of ischemic intestine is best performed by superior mesenteric artery embolectomy. Intestines appearing marginally viable at initial operation should remain, with the intent
of undertaking a second-look operation 24 to 36 hours later. Reperfusion of the intestine after embolectomy might cause sloughing of ischemic mucosa and massive gastrointestinal hemorrhage, a serious problem in patients who require anticoagulants to lessen further peripheral embolization and a reason that lytic therapy is not an appropriate intervention.

Vasodilators have been advocated as a logical means of improving flow to noninfarcted, marginally viable intestine associated with acute mesenteric embolic occlusion, but no controlled studies exist to document the efficacy of such therapy. Papaverine requires intra-arterial administration and might be associated the tachyphylaxis if administered over prolonged periods of time. Glucagon is not associated with autoregulatory escape, and when administered intravenously it has the same effect as superior mesenteric artery catheter infusion. Aggressive diagnosis and prompt surgical therapy have lessened the mortality of superior mesenteric artery embolism from 95% with infarction to 60% without intestinal infarction.

Microembolization from aortic atherosclerotic material, diseased heart valves, prosthetic valves, or infective endocarditis might also cause acute intestinal ischemia. These minute emboli usually produce segmental intestinal infarctions. Clinical manifestations of microemboli are those of an acute abdomen, and they frequently lead to operations without suspicion of the underlying disease. Limited bowel resections are appropriate in these settings.

Other causes of acute mesenteric ischemia deserve mention. In particular, acute superior mesenteric artery thrombosis can present in a manner that is similar to that of embolic occlusion. In most instances of thrombosis, underlying arteriosclerosis affects the proximal superior mesenteric artery, and prodromata of intestinal angina might precede the thrombotic event. In these cases the entire mid-gut, including the proximal jejunum, becomes ischemic. Acute mesenteric thrombosis necessitates an urgent endarterectomy or bypass procedure. Unfortunately, thrombolytic therapy and balloon angioplasty have little value in this setting because of subsequent hemorrhage from the reperfused intestine.

Low-flow nonocclusive ischemia

Intestinal ischemia might also follow nonmechanical reductions in mesenteric blood flow. Patients in cardiac failure are at particular risk. Men are affected one and a half times more often than women. Digitalis in this setting causes an increase in mesenteric vascular resistance, as will many vasopressors used for the management of acute cardiac failure. Elderly patients who become hypovolemic and hypotensive from various illnesses (ranging from simple dehydration to sepsis) are also at high risk for nonocclusive mesenteric ischemia.

The portion of the intestine that is most vulnerable to low-flow ischemia is the colon’s splenic flexure because of its watershed blood supply. The entire intestine can be affected by low flow, however. The frequency of nonocclusive intestinal ischemia remains ill defined because many patients have self-limited, unreported sequelae such as mild ischemic colitis.

Nonocclusive mesenteric ischemia is often manifested by a 3- to 4-day history of vague lower or mid-abdominal discomfort with distention that is accompanied later by nausea and vomiting. The severity will become intense with transmural bowel infarction and peritoneal irritation, the latter of which is usually accompanied by a leukocytosis. Biochemical evidence of intestinal infarction, as in the case of embolic disease, is an unreliable diagnostic finding. Plain radiographic studies might reveal bowel wall edema, and barium enema studies often reveal a “thumb-printing” pattern in the colon, which is indicative of submucosal hemorrhage and edema. Arteriographic studies in low-flow intestinal ischemia usually exhibit delayed arterial emptying and delayed venous filling with a segmental or diffuse appearance of vasoconstriction. The latter is reversible with intra-arterial administration of papaverine or other vasodilators.

Treatment of low-flow, nonocclusive mesenteric ischemia requires restoration of a normal hemodynamic state by appropriate administration of blood, colloids, and electrolyte fluids while treating the patient’s compromised cardiac status. Cardiac function might deteriorate further as a direct effect of intestinal ischemia and the release of myocardial depressant substances into circulation. Intestinal ischemia can be lessened by the administration of various vasoactive drugs. Glucagon, which has inotropic and chronotropic actions, has the theoretic potential to improve cardiac function while producing splanchnic vasodilation and reducing intestinal motor activity. Papaverine, administered directly into the superior mesenteric artery by a percutaneously inserted catheter, can also be used in these patients. The efficacy of these therapies has not been documented in controlled studies. Nevertheless, patients
with low-flow, nonocclusive intestinal ischemia appear to benefit when treated aggressively by these means. Operations should be undertaken only when transmural infarction is suspected and resection of the bowel appears to be necessary. The mortality of low-flow, nonocclusive intestinal ischemia if intestinal infarction has occurred is 60%.

**Splanchnic artery aneurysms**

Splanchnic artery aneurysms are an unusual but important group of diseases [24]. Nearly 22% of these aneurysms present as emergencies, and 8.5% are fatal. The most commonly involved vessels (in decreasing order of frequency) include the splenic, hepatic, superior mesenteric, celiac, gastroduodenal, jejunal-ileal-colic, pancreaticoduodenal-pancreatic, and gastroartery (Table 2).

**Splenic artery aneurysms**

Aneurysms of the splenic artery account for more than 60% of all splanchnic artery aneurysms [26,27]. Their incidental recognition in 0.78% of abdominal arteriographic studies probably reflects the frequency in the general population. Splenic artery aneurysms are usually saccular, occur at bifurcations, and are multiple in 20% of patients (Fig. 4).

Women are affected four times more often than men. Three distinct conditions are associated with these aneurysms: (1) arterial fibrodysplasia, (2) portal hypertension with splenomegaly, and (3) multiple pregnancies, with 40% of women with these lesions having completed six or more pregnancies. These aneurysms often exhibit arteriosclerotic changes, but such is considered a secondary—not an etiologic—event. Trauma and inflammatory disease such as chronic pancreatitis with pseudocyst erosion of the splenic artery are less common but important causes of some aneurysms. Microaneurysms of the splenic artery, which are usually associated with connective tissue disorders, are of less clinical importance.

Most splenic artery aneurysms are asymptomatic. These aneurysms can be suspected with signet-ring calcifications in the left upper quadrant on plain abdominal radiographs. Diagnosis is usually the result of arteriography that is performed for some other disease. Ultrasonography, CT, or MRI can prove useful in the initial recognition of splenic artery aneurysms, but arteriography is usually necessary to confirm their presence.

Life-threatening rupture affects less than 2% of bland splenic artery aneurysms in nonpregnant women. Symptoms of a “double rupture” occur in 25% of such cases, with bleeding into the retrogastric area occurring first and frank intraperitoneal hemorrhage and shock developing later when the lesser space tamponade is lost. The contention that rupture is less likely to occur in calcified aneurysms, normotensive patients, or patients older than 60 years is not supported by clinical experience. Rupture has affected nearly 95% of aneurysms recognized during pregnancy. Maternal mortality is 70% and fetal mortality is 75% in this setting.

Operative intervention is easily justified for aneurysms in pregnant patients, women of childbearing age who might become pregnant, and for symptomatic aneurysms. Aneurysm size greater than 2.5 cm is a relative indication for surgical therapy.

Treatment consists of aneurysmectomy or ligation and exclusion of the aneurysm. Splenectomy might be necessary if the aneurysm is within the splenic parenchyma. The mortality rate for ruptured aneurysms in nonpregnant patients is 25%. Thus, on the basis of a 2% incidence of rupture with an accompanying 25% mortality rate, operative mortality must be less than 0.5% to justify elective surgical therapy. Catheter-directed occlusion of these aneurysms with thrombogenic material is an appropriate therapy for high-risk patients.

**Hepatic artery aneurysms**

Hepatic artery aneurysms account for 20% of all splanchnic artery aneurysms. Men are affected twice as often as women. These aneurysms are usually recognized during the sixth decade of life. Causes include atherosclerosis (32%), acquired medial degeneration (24%), trauma (22%), and infection associated with illicit drug use (10%). Traumatic pseudoaneurysms are more frequently recognized in contemporary times with the greater use of CT to evaluate the injured patient. Polyarteritis nodosa, cystic medial necrosis, and other arteriopathies are uncommon causes of these aneurysms. Most aneurysms of the hepatic artery are solitary, being fusiform when less than 2 cm in diameter and saccular when more than 2 cm in diameter. Hepatic artery aneurysms are extrahepatic in 80% of cases and intrahepatic 20% of the time.

Most hepatic artery aneurysms are asymptomatic. Symptoms, if present, characteristically include right upper quadrant and epigastric pain,
<table>
<thead>
<tr>
<th>Aneurysm Location</th>
<th>Frequency Within Splanchnic Circulation</th>
<th>Male: Female Ratio</th>
<th>Contributing Factors</th>
<th>Frequency of Reported Rupture</th>
<th>Site of Rupture</th>
<th>Mortality with Rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Splenic artery</td>
<td>60%</td>
<td>1:4</td>
<td>Medial degeneration; arterial fibrodysplasia; multiple pregnancies; portal hypertension; trauma; chronic pancreatitis with arterial erosion by pseudocysts</td>
<td>2% bland aneurysms, 95% with pregnancy</td>
<td>Intraperitoneal within lesser sac; intragastric with pancreatitis-related aneurysms</td>
<td>25% bland and unassociated with pregnancy; during pregnancy 70% maternal, 75% fetal</td>
</tr>
<tr>
<td>Hepatic artery</td>
<td>20%</td>
<td>2:1</td>
<td>Medial degeneration; blunt and penetrating liver trauma; infection related to intravenous substance abuse</td>
<td>20%</td>
<td>Intraperitoneal and biliary tract with equal frequency</td>
<td>35%</td>
</tr>
<tr>
<td>Superior mesenteric artery</td>
<td>5.5%</td>
<td>1:1</td>
<td>Medial degeneration; infection related to bacterial endocarditis, often associated with nonhemolytic streptococci and more recently with bacteremia from intravenous substance abuse</td>
<td>Uncommon, thrombosis more common</td>
<td>Intraperitoneal and retroperitoneal</td>
<td>50%</td>
</tr>
<tr>
<td>Celiac artery</td>
<td>4%</td>
<td>1:1</td>
<td>Medial degeneration</td>
<td>13% Intraperitoneal</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td>Gastric and gastroepiploic arteries</td>
<td>4%</td>
<td>3:1</td>
<td>Periarterial inflammation; medial degeneration</td>
<td>90% 70% intestinal tract; 30% intraperitoneal</td>
<td>70%</td>
<td></td>
</tr>
<tr>
<td>Pancreaticoduodenal, pancreatic, and Gastroduodenal arteries</td>
<td>3.5%</td>
<td>4:1</td>
<td>Pancreatitis-related arterial necrosis and arterial erosion by pseudocysts in 60% of gastroduodenal and 30% of pancreaticoduodenal artery aneurysms; medial degeneration</td>
<td>75% inflammatory 85% intestinal tract; 15% intraperitoneal</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td>Jejunal, ileal, and colic arteries</td>
<td>3%</td>
<td>1:1</td>
<td>Medial degeneration; connective tissue diseases</td>
<td>30% Intraperitoneal and intestinal tract</td>
<td>20%</td>
<td></td>
</tr>
</tbody>
</table>
which (when severe) are similar to the pain of pancreatitis. Large aneurysms uncommonly obstruct the biliary tract. Hepatic artery aneurysms are discovered most often during arteriographic evaluations of other gastrointestinal or abdominal diseases.

Rupture of a hepatic artery aneurysm causes bleeding into the peritoneal cavity and biliary tract with equal frequency. The latter results in hemobilia with acute gastrointestinal bleeding and fever from cholangitis. Melena and chronic anemia are uncommon with this complication. Hepatic artery aneurysms rupture in less than 20% of cases. The mortality rate with rupture approaches 35%.

Aneurysms of the common hepatic artery are treated by aneurysmectomy or exclusion, often without arterial reconstruction. If the aneurysm is in the proper hepatic artery, blood flow to the liver should be restored after aneurysmectomy, preferably with an autologous vein graft. Simple ligation or percutaneous transcatheter obliteration of a hepatic artery aneurysm might also be appropriate in selected patients. Partial hepatectomy is rarely required, but it might be necessary to successfully treat hemobilia caused by a ruptured intraparenchymal aneurysm.

**Superior mesenteric artery aneurysms**

Aneurysms of the superior mesenteric artery account for 5.5% of all splanchnic artery aneurysms. Men and women are affected equally. Infection accounts for nearly 50% of cases, occurring most often as a result of nonhemolytic streptococci originating from left-sided bacterial endocarditis. Infection associated with illicit intravenous drug abuse is a second cause of increasing frequency. Medial degeneration, atherosclerosis, and trauma are less common causes. Aneurysms in young patients are usually mycotic, whereas aneurysms in patients older than 50 years are most often noninfectious in origin.

Superior mesenteric artery aneurysms are usually recognized when vascular calcifications are identified on radiographs of the abdomen or arteriograms are performed for unrelated diseases. Epigastric pain is common. Symptoms of intestinal angina can accompany nonmycotic lesions,
in which aneurysmal dissection or thrombus has compromised intestinal blood flow. The reported frequency of rupture is low, but thromboembolic complications of these aneurysms are relatively high, and most lesions should be treated operatively. Mortality is 50% with rupture. Aneurysmectomy with formal arterial reconstruction is preferred in most cases, but simple ligation of vessels entering and exiting the aneurysm has been successful in about 30% of reported cases. The operative mortality rate in treating superior mesenteric artery aneurysms is less than 15%.

Celiac artery aneurysms

Celiac artery aneurysms represent 4% of splanchnic artery aneurysms. Most of these aneurysms exhibit medial degeneration. There is no gender predilection. Celiac artery aneurysms are usually asymptomatic or they cause vague abdominal discomfort. The rupture rate approaches 13% with a mortality of 50%. Treatment usually consists of aneurysmectomy and arterial reconstruction. Celiac artery ligation can be performed in selected cases if circulation to the liver is adequately maintained through collateral vessels. More than 90% of operations for celiac artery aneurysms are successful.

Gastric and gastroepiploic artery aneurysms

Aneurysms of the gastric and gastroepiploic arteries account for 4% of all splanchnic artery aneurysms. Gastric artery aneurysms are ten times more common than gastroepiploic artery aneurysms. Men are affected three times more often than women. These aneurysms are usually acquired, most frequently as a result of medial degeneration or a consequence of periarterial inflammation. They often present as vascular emergencies without preceding symptoms. Rupture occurred in more than 90% of reported cases with 70% causing serious gastrointestinal bleeding. The rest caused life-threatening intraperitoneal hemorrhage. Nearly 70% of patients with rupture die from this complication.

Fig. 5. Gastroduodenal artery aneurysm in a patient with alcoholic-related pancreatitis and pseudocyst erosion of the artery. (A) CT scan. (B) Arteriography. (Reprinted with permission from Eckhauser FE, Stanley JC, Zelenock GB, Freier DT, Lindenauer SM. Gastroduodenal and pancreaticoduodenal artery aneurysms: a complication of pancreatitis causing spontaneous gastrointestinal hemorrhage. Surgery 1980;88:335–44.)
Ligation of aneurysmal vessels with or without aneurysm excision is appropriate for extragastric lesions. Intramural lesions should be excised with the involved portion of the stomach.

**Jehunal, ileal, and colic artery aneurysms**

Aneurysms affecting the intestinal branches of the superior and inferior mesenteric arteries account for 3% of all splanchnic artery aneurysms. Men and women are affected equally. These aneurysms are recognized most often in the seventh decade of life. Ninety percent of these aneurysms are solitary, except for microvascular aneurysms associated with connective tissue disorders. Congenital or acquired medial defects cause most of these lesions. Certain aneurysms present the sequela of an endarteritis from septic cardiac emboli.

Most mesenteric artery branch aneurysms are incidental findings during exploratory surgery for intestinal or intraperitoneal bleeding. Seventy percent of these aneurysms cause abdominal pain or bleeding, and some might present as a tender abdominal mass. Arteriography is necessary to establish a preoperative diagnosis.

Although the risk of rupture is low, the reported mortality with rupture is 20%. Treatment of these small aneurysms usually consists of ligation or aneurysmectomy for extraintestinal aneurysms and bowel resection for intramural aneurysms.

**Pancreaticoduodenal, pancreatic, and gastroduodenal artery aneurysms**

Pancreatic and pancreaticoduodenal artery aneurysms account for 2% of all splanchnic artery aneurysms. Gastroduodenal artery aneurysms represent an additional 1.5% of these lesions. These aneurysms are among the most hazardous of all splanchnic artery aneurysms. Men are affected four times more often than women. The most common cause is periarterial inflammation, which usually occurs as a consequence of pancreatitis with vascular necrosis or vessel erosion by an adjacent pseudocyst (Fig. 5). Most patients with these aneurysms have epigastric pain and discomfort. This might reflect the fact that about 60% of gastroduodenal and 30% of pancreaticoduodenal artery aneurysms are complications of pancreatitis. Asymptomatic aneurysms of these vessels are unusual.

Aneurysmal rupture of gastroduodenal and pancreaticoduodenal aneurysms occurs in 75% of inflammatory lesions and 50% of noninflammatory lesions. Bleeding into the biliary or pancreatic ductal system might occur. Endoscopy, ultrasonography, CT, and MRI often contribute to the diagnosis, but arteriography is essential to confirm the presence of these lesions. Mortality with rupture approaches 50%.

Operative intervention with aneurysmectomy is justified in all reasonable-risk patients. Aneurysms embedded within the pancreas are best treated by suture ligation of entering and exiting vessels from within the aneurysm. Eventual extirpation of the diseased pancreas might be necessary. Transcatheter embolization can temporarily control acute bleeding from some of these aneurysms, but it is associated with a high rate of rebleeding and abscess formation.

**References**


