Each shift, the emergency physician must consider the uncommon diagnoses grouped together as abdominal vascular emergencies in the differential diagnosis of the patient with nausea, vomiting, diarrhea, or with abdominal, back, or flank pain. Identification of vascular emergencies is made even more difficult by clinical findings often being nonspecific or equivocal.

Vascular abdominal emergencies are not common but, when present, are often catastrophic. Most of the conditions are time sensitive, putting perfusion of critical organs (eg, the bowel) at risk, leading to the potential for ischemia, infarction, and translocation of enteric microbes, bacteremia, and sepsis. Aneurysmal dilation of the aorta with rupture leads to rapid hypovolemic shock and death if not diagnosed.

A high index of suspicion is critical to the successful diagnosis of abdominal vascular emergencies. Because most emergencies ultimately require surgical intervention, diagnostic testing should be performed in parallel with resuscitation, consultation, and involvement of the vascular or general surgeon.

VASCULAR ABDOMINAL ANATOMY

The aorta gives rise to several paired and unpaired vessels within the abdomen. The adrenal, renal, and gonadal arteries are paired, and provide blood flow to their respective organs. The unpaired branches (celiac artery, superior mesenteric artery [SMA], and inferior mesenteric artery [IMA]) deliver blood to most of the digestive tract. The celiac trunk branches off the aorta at approximately 90 degrees, making it less susceptible to embolic phenomena compared with the SMA and IMA. The 3 branches of the celiac trunk (the splenic, left gastric, and common hepatic arteries) supply the foregut structures from the distal esophagus to the second part of the duodenum, the spleen, the liver, and parts of the pancreas (Fig. 1).1

**KEYWORDS**

- Abdominal aorta
- Mesenteric ischemia
- Abdominal aortic aneurysm
- Embolus
- Arterial thrombus
- Venous thrombus

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The SMA typically arises about 1 cm below the celiac trunk, at the approximate level of the first lumbar vertebra.\(^2\) The SMA gives rise to several branches that supply the midgut structures extending from the second part of the duodenum to the distal third of the transverse colon. The SMA leaves the aorta at an angle of less than 30 degrees, making it susceptible to thromboembolism (Fig. 2).\(^1\)

Just before the bifurcation of the aorta at the level of the fourth lumbar vertebra, the IMA branches off the aorta and supplies all of the structures of the hindgut, which extend from the transverse colon to the rectum. It terminates in the superior rectal artery (see Fig. 2).\(^2\) Although each branch is separate, there is extensive collateral blood flow

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*Fig. 1. The celiac artery and its 3 major branches: the splenic, left gastric, and hepatic arteries. (From Walker JS, Dire DJ. Vascular abdominal emergencies. Emerg Med Clin North Am 1996;14(3):573; with permission.)*

*Fig. 2. The superior and inferior mesenteric arteries and their anastomotic connections. (From Walker JS,Dire DJ. Vascular abdominal emergencies. Emerg Med Clin North Am 1996;14(3):574; with permission.)*
between the celiac artery and SMA via the pancreaticoduodenal artery, and between the SMA and the IMA by way of the marginal artery of Drummond and arc of Riolan. The venous system of the gastrointestinal tract differs from the arterial system in that, rather than draining into the inferior vena cava (IVC), it passes via the portal vein into the liver. Blood passes through the hepatic lobules into the hepatic veins, which pass into the IVC. In normal health, portal blood is sequestered from the systemic venous circulation. Pathologic conditions (eg, cirrhosis, obstruction, thrombosis) that obstruct portal flow cause anastomoses to form between systemic and portal vessels in tissues that are at the watershed junction between the 2 systems. These locations are the lower esophagus, the umbilicus, and the rectum. When anastomoses become very large they are referred to as varices.

ABDOMINAL VASCULAR THROMBOSES

Aortic Thrombosis

Acute occlusive aortic thrombosis is a rare condition that is lethal if not diagnosed. Nonocclusive aortic thrombosis is more common and occurs in the setting of aneurysmal disease, dissection, or severe atherosclerotic disease. Other conditions associated with aortic thrombotic disease include diabetes, cardiomyopathy, blunt and penetrating abdominal trauma, spinal surgery, polycythemia, nephrotic syndrome, exogenous estrogens, the classic hypercoagulable conditions (protein C and S deficiency, factor V Leiden deficiency, antithrombin III deficiency), malignancy, use of certain chemotherapeutic agents, antiphospholipid antibody syndrome, and previous aortic grafting. Because of the large diameter of the aorta, emboli (typically from the left ventricle) rarely lead to aortic occlusion. Patients with an acute thrombotic event typically present with symptoms of lower extremity ischemia: bilateral lower extremity pulselessness, pallor, pain, paresthesias, and possible paralysis. If the occlusion involves the artery of Adamkowicz, patients may also develop spinal cord infarction. Signs of the anterior spinal artery syndrome include paralysis and loss of sensation to both light touch and pinprick, but preservation of vibratory sensation and proprioception. In cases of chronic occlusion caused by atherosclerotic disease, there is time for the formation of collateral circulation to the distal structures. Leriche syndrome results from chronic obstruction of the distal aorta leading to chronic ischemia of the pelvis and lower extremities. Classically described in men, the triad of symptoms includes claudication, abnormal or absent lower extremity pulses, and erectile dysfunction. Acute ischemia from aortic occlusive disease warrants emergent consultation with a vascular surgeon. Patients will likely need emergent laparotomy for thrombectomy or embolectomy, often with aortic bypass. Overall mortality in a retrospective case series of 48 patients by Babu and colleagues was 52%. Dossa and colleagues reported a 40-year experience of 46 patients with an in-hospital mortality of 35%. The urgency of surgical consultation and intervention in patients with subacute or chronic occlusive disease is determined by the severity and progression of symptoms. Most patients will be admitted for observation, often with anticoagulant or antiplatelet agents.

Renal Artery Thrombosis

Renal artery thrombosis is also a rare condition, most commonly seen in individuals aged 30 to 50 years. Similar to the other thromboses, the most common cause
involves the development of a clot in situ on established atherosclerotic lesions. Thrombosis is also a known complication after renal transplantation because of both the surgical anastomoses, and immunosuppressive drugs being prothrombotic. Intra-aortic catheter or balloon pump placement, intravenous (IV) cocaine use, and renal angiography are also risk factors. The transplanted renal artery graft may also develop thrombosis secondary to surgical technique including torsion of the artery, kinking of the anastomosis, or dissection into the wall. Large case series have described rates of thrombosis after transplant between 0.5% and 3.5%. Patients with acute renal artery occlusion typically present with flank pain, which may be associated with hypertension. Accompanying symptoms may include nausea, vomiting, and upper abdominal pain on the affected side. Hematuria only occurs in between 30% and 50% of patients. An untreated occlusion of the renal artery leads to renal infarction. In the setting of renal infarct, the serum lactate dehydrogenase (LDH) is typically increased. Symptoms may be difficult to distinguish from renal colic. When renal colic is suspected, unenhanced computed tomography (CT) is the initial study. However, with severe symptoms and a CT without renal stones, the study can be repeated with intravenous contrast if renal artery thrombosis is a consideration. In the renal transplant recipient, duplex ultrasound is the preferred initial test.

Renal artery occlusion can also occur in the setting of trauma. Blunt trauma to the abdomen can lead to compression of the artery, as well as dissection and thrombosis. Patients typically require surgical revascularization, and the timeliness of this intervention is linked with improved outcomes. Nontraumatic thrombosis is treated with surgical revascularization or sometimes thrombolysis. Long-term sequelae of both traumatic and nontraumatic thromboses include renal artery stenosis, renal insufficiency or failure (more likely with bilateral disease), and hypertension.

Renal Vein Thrombosis

Renal vein thrombosis may be either an acute or chronic process. Diagnosis depends on a high level of clinical suspicion, because the clinical findings mimic those of renal colic, renal artery occlusion, and pyelonephritis. With acute thrombosis, patients experience flank pain often associated with nausea and vomiting. Hematuria and proteinuria may also be noted. In the setting of chronic thrombosis, the diagnosis may not be made until the development of complications such as impaired renal function or pulmonary embolism. The left renal vein is affected more often than the right, but up to two-thirds of patients have bilateral thrombosis. In contrast to occlusive arterial disease, renal vein thrombosis is also a disease of children and neonates. In the setting of severe volume depletion, dehydration, or sustained hypotension, blood flow is shunted from the renal vein, leading to sluggish flow that may eventually lead to the formation of a clot. In children, a palpable mass in the flank may be present because of the enlargement of the kidney on the affected side. The classic triad of symptoms in children includes a palpable mass, gross hematuria, and thrombocytopenia, although most patients do not have all three. Thrombosis of the renal vein may occur as a result of the hypercoagulable, post-transplant, and postoperative states mentioned in previously. In addition, blunt trauma and infection play roles in its development. The most common disease associated with the development of renal vein thrombosis is nephrotic syndrome. Patients have direct loss of protein S and antithrombin III in their urine. With excessive proteinuria, the liver is stimulated to produce new proteins, many of which are prothrombotic.
In patients receiving transplants, color Doppler sonography should be used to evaluate the flow in the graft. In all other patients, CT scan is the diagnostic study of choice. Intravenous contrast should be administered to visualize the vascular structures. In addition to visualizing the thrombus, the kidney on the affected side is typically engorged because of impaired venous drainage. Delayed images show a persistent enhancement from the contrast on CT scan because of the limited venous outflow.

Treatment of renal vein thrombosis is generally medical with systemic anticoagulation. Patients with, or at risk for, severe disease (eg, extensive clot progressing to the IVC, renal failure, bilateral disease, renal transplant) may be candidates for systemic or catheter-directed thrombolytic therapy.27

**Portal Vein Thrombosis**

The estimated lifetime risk of developing portal vein thrombosis in the general population is 1%.31 Up to 15% of cirrhotics develop this condition. It is rarely seen in patients without known liver disease or other risk factors that include adjacent inflammatory conditions (eg, pancreatitis, cholecystitis, diverticulitis, inflammatory bowel disease, appendicitis), malignancies (local or systemic), or hypercoagulable conditions (especially sepsis).32,33 Mortality caused by portal vein thrombosis itself is low; however, these patients frequently have other significant comorbidities that combine to give them poor outcomes with this condition.

The mechanism by which cirrhosis leads to portal vein thrombosis is not clear. It is believed that decreased portal blood flow, periportal inflammation and fibrosis, and impaired production of anticoagulation factors lead to thrombosis.34 Fifty percent of portal vein thrombosis in children and neonates is associated with an intra-abdominal infection, including umbilical infections in the very young.34

With clot in the portal vein, the liver loses approximately two-thirds of its blood supply. In the acute phase, several compensatory mechanisms occur, including dilation of the hepatic artery to increase blood supply, the development of variceal collaterals between the portal and systemic venous systems, and collateral cavernoma formation. The cavernomas are a matted plexus of collateral vessels that form at the porta hepatis, often leading to secondary biliary effects including cholecystitis, biliary obstruction, and jaundice. Although collateral formation ultimately restores some degree of splanchnic circulation, hepatocytes often continue to be underperfused, leading to ongoing ischemia and cell death.32,35

Acute portal vein thrombosis may present with abdominal pain (which may be localized in the right upper quadrant, but is frequently diffuse), nausea, and fever. Signs of intestinal ischemia (discussed later), which is a secondary effect of acute portal vein thrombosis, may also be present.32,34 With chronic thrombosis, patients may remain clinically silent until the secondary effects of the thrombosis occur. These effects include worsening hepatic function, worsening portal hypertension, and hematemesis from esophageal varices.32,34,36

Ultrasound with Doppler is the diagnostic modality of choice for portal vein thrombosis. The absence of flow within the lumen and, in some cases, the presence of a cavernoma identifies the disease. If suspicion exists for extension of the clot into the mesenteric venous system, CT with intravenous contrast is indicated to evaluate the vasculature and intestines.

Patients with portal vein thrombosis without complications of bleeding or intestinal ischemia are managed with systemic anticoagulation.31,32,37 Empiric thrombolytic therapy in cases of acute thrombosis may be indicated in severely ill patients in consultation with a gastroenterologist, although there may be significant complication
rates. Thrombolytics are also considered when standard anticoagulation does not lead to recanalization. A transjugular intrahepatic portosystemic shunt (TIPS) procedure may be considered in patients having liver transplants or as an alternative to thrombolytic therapy.

**Mesenteric Artery and Venous Thrombosis**

Mesenteric arterial and venous thromboses are discussed later.

**ISCHEMIC BOWEL**

Ischemic bowel can be caused by 1 of 4 mechanisms: arterial thrombosis, arterial embolism, venous thrombosis, or nonocclusive mesenteric ischemia (NOMI). Survival rates vary depending on the mechanism. Overall mortality is as high as 60% to 80%, especially with delays in diagnosis or presentation of greater than 24 hours. Park and colleagues reported worse survival rates in older patients, those not candidates for bowel resection, and those whose cause is NOMI. The overall survival rates for patients with mesenteric ischemia have improved according to a review that analyzed 45 studies and 3692 patients according to cause in almost 4 decades (1966–2002). Mesenteric ischemia is categorized as occlusive or nonocclusive in origin. Occlusive mesenteric ischemia either involves the SMA or the superior mesenteric vein (SMV). Arterial occlusion may be embolic or thrombotic in origin.

Mesenteric ischemia and ischemic colitis are different clinical entities. The former refers to occlusion of the SMA and SMV, whereas the latter refers to ischemia in the distribution of the IMA. Patients with mesenteric ischemia primarily present with abdominal pain. Patients with ischemic colitis present with lower gastrointestinal bleeding and are less likely to report abdominal pain as the primary complaint. Ischemic colitis has been reported in marathon runners with similar clinical presentations. Patients with ischemic colitis tend to be older (77 vs 61 years in a retrospective review of 100 patients presenting to the emergency department [ED]) and show lower overall mortality. Angiography is not indicated in cases of ischemic colitis.

Between 40% and 50% of patients with mesenteric ischemia have an arterial embolus as the cause. Emboli typically lodge in the SMA because of the narrow angle it subtends as it branches off the abdominal aorta. Less commonly, emboli lodge in the IMA and, rarely, in the celiac artery. In most cases, a patient with underlying cardiac abnormalities presents with acute onset of pain. Predisposing cardiac conditions include arrhythmia (most commonly atrial fibrillation), myocardial infarction, cardiomyopathy, recent angiography, valvular disorder (eg, rheumatic valve disease), or ventricular aneurysm. Following surgical embolectomy, the mortality from analyzed results from 4 decades is 54%.

**Thrombotic Occlusion of the Mesenteric Artery**

Atherosclerosis is the major cause of arterial thrombosis leading to ischemia of the SMA and is the cause of mesenteric ischemia in 25% of patients. The onset of pain is usually more insidious and may be initially intermittent and eventually becoming constant. Other causative factors are hypercoagulability, estrogen therapy, and prolonged hypotension. Patients with chronic ischemia may present with intestinal angina (typically epigastric pain precipitated by eating) and describe food fear and ensuing weight loss. Chronic mesenteric ischemia is usually caused by atherosclerosis and is more common in women and smokers. It is also associated with radiation
arteritis, autoimmune arteritides, and fibromuscular dysplasia.\textsuperscript{52} Mortality is high for this disorder and, following surgical treatment, is reportedly 77% (Fig. 3).\textsuperscript{44}

**Thrombotic Occlusion of the Mesenteric Vein**

Mesenteric venous thrombosis accounts for 10% to 15% of mesenteric ischemia cases. Risk factors for mesenteric venous thrombosis include oral contraceptives or estrogen therapy, malignancy, hypercoagulability, portal hypertension, portal vein thrombosis or other deep vein thrombosis, sickle cell disease, clotting disorders, hepatosplenomegaly, hepatitis, pancreatitis, coagulopathic states, sepsis, cigarette smoking, prior abdominal surgery, and alcohol use.\textsuperscript{42,50,52–56} Twenty percent of mesenteric venous thrombosis cases are found to be idiopathic. Patients may present in a subacute fashion with abdominal pain and diarrhea. Clinical findings are likely to be more severe, with frank peritonitis and bleeding in patients with extensive transmural ischemia. With chronic mesenteric vein thrombosis, the collateral circulation usually allows for adequate venous drainage, limiting symptoms and consequent secondary effects of portal hypertension and varix formation. Mesenteric venous thrombosis is usually diagnosed by CT with intravenous contrast. This modality has the advantage compared with color Doppler ultrasound of also allowing for assessment of the bowel and other intra-abdominal conditions. Most mesenteric venous ischemia is treated nonoperatively with anticoagulation. As with portal vein thrombosis, a consideration of the harm-to-benefit profile of thrombolytics can be undertaken in consultation with a gastroenterologist as clinical indications dictate. The pooled reported mortality following surgical treatment of venous thrombosis is 32%.\textsuperscript{44}

**NOMI**

NOMI is defined as ischemia in the absence of identifiable occlusive lesions in the splanchnic arteries or veins. It tends to be precipitated by low-flow states such as hypotension, hypovolemia, heart failure, or sepsis, in which a vicious cycle develops with hypoperfusion leading to vasoconstriction, which leads to ischemia, followed

![Fig. 3](image-url). Selective conventional angiography shows an abrupt cutoff of the SMA secondary to embolus (arrow). (From Martinez JP, Hogan GJ. Mesenteric ischemia. Emerg Med Clin North Am 2004;22(4):912; with permission.)
by elaboration of inflammatory mediators that further exacerbate hypotension and hypoperfusion. Mechanical processes, such as obstructive tissue bands, intussusception, volvulus, and incarcerated hernias, are nonocclusive causes of mesenteric infarction but are nonvascular in nature and therefore not considered in this article.57 Twenty percent of cases of mesenteric ischemia have nonocclusive causes. Many patients with NOMI have underlying illness, which contributes to the high mortality. Medications associated with NOMI include cocaine, digitalis (splanchnic vasoconstriction), ergot alkaloids and vasopressors, \( \alpha \) constricting agents, and \( \beta \)-blockers.39,42,50,52

**Clinical Findings**

Abdominal pain is the chief complaint in most patients with mesenteric ischemia, followed by associated nausea, vomiting, and diarrhea.40,41,51 In one retrospective study of 83 patients, hypertension and diabetes mellitus were the most common risk factors. The features of the abdominal pain tend to vary depending on the mechanism of occlusion. Pain caused by embolic disease is often acute in onset and the patient may have associated nausea, vomiting, or diarrhea (both may be either bloody or nonbloody). Bleeding per rectum is reported in 14% to 16% of cases.43,51 On examination, the patient may have remarkably mild abdominal tenderness compared with the severity of symptoms. With progression of ischemia, patients may be hypotensive with peritoneal signs on abdominal examination. Thrombotic ischemia is likely to have a more insidious onset that may progress to being constant in nature. Patients with NOMI have more varied presentation with underlying illness, abdominal distension, hypertension, and nausea.41,57

**Laboratory Evaluation**

There is no single specific test for the diagnosis of mesenteric ischemia.39 Most commonly, an anion gap acidosis, leukocytosis, and hemoconcentration are found with mesenteric ischemia. Increased lactate is independently associated with higher mortality.40 Studies evaluating the association between mesenteric ischemia and \( \beta \)-dimer note a high sensitivity but low specificity. There is no correlation between \( \beta \)-dimer value and severity of mesenteric ischemia.58

**Diagnostic Imaging**

Deciding on the most appropriate diagnostic imaging test once the possibility of mesenteric ischemia has been recognized is challenging. Plain abdominal radiographs, CT, ultrasound, magnetic resonance imaging (MRI), multislice CT angiography, and angiography are all radiographic modalities for consideration.

Plain radiographs may show pathognomonic findings of mesenteric ischemia (eg, thumb-printing or bowel loop thickening) in up to 40% of cases.39 Often, these are useful as a first test to evaluate for obstruction or free air. Commonly, plain films show nonspecific findings. Abdominal radiographs have low sensitivity for the detection of air in the splanchnic vascular system and underestimate its extent if it is seen.59 If portal gas is identified, mesenteric ischemia is the most likely diagnosis and emergent surgical intervention is indicated.

Doppler ultrasound has limited usefulness in the acute and emergent evaluation of mesenteric ischemia because of the time required and operator-dependent nature of this modality. Air-filled bowel makes ultrasound difficult to interpret, although more proximal occlusions in the celiac and SMA may be visualized. MRI is a time-consuming and costly measure whose accuracy has not been confirmed but may in the future prove to be sensitive.39
In the early phase of mesenteric ischemia, CT with IV contrast may show vascular opacification in the vessel lumen as with SMA thrombosis (Fig. 4). Dilated loops of bowel, air-fluid levels, and changes in bowel wall enhancement, or a combination of these, are nonspecific findings but may indicate more advanced stages of mesenteric ischemia. In later phases of ischemia, CT shows thickened bowel and air in the bowel wall or pneumotosis intestinalis (Fig. 5).39 Mesenteric venous gas seen on CT alone is not pathognomonic and does not mark the extent of mesenteric ischemia. It can occur in more benign inflammatory or infectious abdominal processes such as ulcer disease, pancreatitis, diabetes mellitus, and caustic ingestions.59–61 On CT, it is typically appreciated ventrally first, appearing in the left lobe peripherally and in the subcapsular region of the liver.59,62 Therefore, mesenteric vascular gas, although sensitive, is not specific for mesenteric ischemia and occurs more commonly in mesenteric venous thrombosis (Fig. 6).61,63

Angiography is still the gold standard in the diagnosis of mesenteric ischemia, but multidetector CT angiography may emerge as a preferred imaging modality because of its ability to assess the bowel wall (Fig. 7). When angiography is performed, a catheter is left in the SMA for administration of vasodilators or papaverine, which is particularly useful in embolic mesenteric ischemia or NOMI.39,42

Treatment

Treatment is tailored to the cause of the mesenteric ischemia. All patients require hemodynamic monitoring, resuscitation, intravenous fluid, broad-spectrum antibiotic administration, and pain management.39,46 Emergent surgical consultation is warranted in acute ischemia, and the patient should not be given anything to eat or drink by mouth. Without contraindications, heparin therapy should be initiated, and, if

Fig. 4. Embolic occlusion of SMA. (A) Unenhanced CT scan; the SMA is normal (arrowhead). (B) Contrast-enhanced CT scan at the same level; the SMA has a regular enhancement. (C, D) Contrast-enhanced CT at a lower level; unenhanced aspect of the SMA caused by endoluminal embolus (arrowhead). (From Angelelli G, Scardapanie A, Memeo M, et al. Acute bowel ischemia: CT findings. Eur J Radiol 2004;50(1):39; with permission.)
Fig. 5. Late-stage bowel infarction. (A) Axial scan, (B) coronal multiplanar reformation (MPR) image, and (C) sagittal MPR image. Pneumatosis; gas within bowel walls (arrowheads). (From Angelelli G, Scardapane A, Memeo M, et al. Acute bowel ischemia: CT findings. Eur J Radiol 2004;50(1):44; with permission.)

Fig. 6. Patients with bowel infarction. Gas in hepatic portal branches is peripherally located (arrowheads). (From Angelelli G, Scardapane A, Memeo M, et al. Acute bowel ischemia: CT findings. Eur J Radiol 2004;50(1):45; with permission.)
vasopressors are indicated, pure α agonists should be avoided to minimize vaso-
spasm and exacerbation of ischemia. Vasopressors and digitalis should be avoided
when possible.42

A patient with peritoneal signs usually requires emergent laparotomy.42,50 A patient
with mesenteric ischemia caused by embolic phenomena would require surgical inter-
vention for embolectomy, excision of infarcted bowel, and consideration of intra-
arterial papaverine. The patient with an arterial thrombus is a candidate for similar
interventions: thrombectomy and revascularization. Similarly, if the patient has
mesenteric venous thrombosis, thrombectomy and excision of gangrenous bowel
may be indicated, and heparin therapy should be initiated. Patients with NOMI should
have the underlying cause treated as well as resection of the affected segment of
bowel. With angiographic evidence of occlusion, intra-arterial papaverine may be
injected directly via the catheter.50

Risk Management Strategy

Factors affecting mortality from mesenteric ischemia include older age, delay in
presentation, signs of peritonitis, and delay in surgical intervention. One retrospective
study of 60 patients who underwent surgery for mesenteric ischemia noted that age
greater than 70 years conferred a 3.8-fold increased risk of mortality compared with
those less than this age.40 Another series reported an overall survival rate of 40% at
30 days after surgery, with an 81% survival rate for patients less than 71 years of

Fig. 7. Sagittal CT angiography of the aorta shows normal origins of the celiac axis (arrow-
head) and SMA (arrow). (From Martinez JP, Hogan GJ. Mesenteric ischemia. Emerg Med Clin
North Am 2004;22(4):919; with permission.)
age, compared with a 30% survival rate in those aged 71 to 84 years. Surgical inter-
vention within 6 hours also resulted in significantly improved survival.64

ANEURYSM

A vascular aneurysm is defined by the focal dilatation of an artery to at least 1.5 times
its normal diameter. A true aneurysm involves all 3 layers: the intima, media, and
adventitia of the vessel wall; otherwise the dilated segment is referred to as a pseudaneurysm.65 There is a broad spectrum of disease severity associated
with arterial aneurysms in the abdominal cavity. Some present as catastrophic
abdominal emergencies, whereas others are asymptomatic and incidental for the life-
time of the patient. With the increased use of abdominal imaging modalities, earlier
diagnosis of these lesions is more common. Emergency physicians should understand
their natural history as well as their management if they rupture.

Abdominal Aortic Aneurysm

The aorta is the most common location of an aneurysm in the abdominal cavity. Most
abdominal aortic aneurysms (AAAs) occur in the infrarenal region. Most aneurysms are
fusiform rather than saccular, meaning that the entire circumference of the vessel is
involved.65 The mean diameter of the infrarenal aorta is 1.66 to 2.16 cm in older
women and 1.99 to 2.39 cm in older men. By convention, an anteroposterior diameter
of greater than 3.0 cm is classified as an aneurysm.65,66

The prevalence of AAA in men between 65 and 79 years of age is 5% to 10% but
less common in women at 2%.67,68 The most important complication of AAA is
rupture, which occurs almost exclusively in aneurysms larger than 4 cm.69 According
to one recent population-based study, the prevalence of aneurysms of this magnitude
in men is 1.1% for age 55 to 64 years, 4.1% for age 65 to 74 years, and 8.6% for age 75
to 84 years. Larger aneurysms are present in only 0.4% of women older than 55
years.68 AAA is very rare before the age of 50 years. Ruptured AAA is estimated to
cause 15,000 deaths in the United States each year.70

A large prospective cohort of more than 120,000 veterans undergoing screening for
AAA has elucidated the risk factors for this disease (Table 1). For clinically significant
aneurysms (>3.9 cm), the most important risk factors are smoking, family history of
AAA, increasing age, and history of atherosclerotic diseases. Factors that were not
associated with the development of AAA included chronic obstructive pulmonary
disease (COPD) and hypertension. This latter finding is surprising in view of the clear
association between AAA and atherosclerosis. In addition, this study also showed that

<table>
<thead>
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<th>Factors</th>
<th>Odds Ratio (95% Confidence Interval)</th>
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<tr>
<td>Smoking</td>
<td>5.07 (4.13–6.21)</td>
</tr>
<tr>
<td>Family history of AAA</td>
<td>1.94 (1.63–2.32)</td>
</tr>
<tr>
<td>Age, per 7 y</td>
<td>1.71 (1.61–1.82)</td>
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<tr>
<td>History of atherosclerotic disease</td>
<td>1.66 (1.49–1.84)</td>
</tr>
<tr>
<td>Female sex</td>
<td>0.18 (0.07–0.48)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>0.52 (0.45–0.61)</td>
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<tr>
<td>Black race</td>
<td>0.53 (0.40–0.69)</td>
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female sex, black race, and diabetes were traits associated with a decreased risk of AAA.  

The pathophysiology of AAA development and subsequent rupture is incompletely understood at this time. The aortic wall consists of 3 layers: the intima, media, and adventitia. Within the media and adventitia, elastin and collagen are the most important components for maintaining the mechanical integrity of the vessel wall. Elastin is highly concentrated in the medial layer, whereas collagen is concentrated in the adventitia. Damage to and loss of elastin fibers and an associated reduction in the density of smooth muscle cells seem to be integral to the early development of AAA, whereas collagen degradation in the adventitia might be the most important factor in rupture. There seems to be an inflammatory component to this process that ultimately results in the destruction of elastin and collagen. Matrix metalloproteinases have been implicated as one of the key enzymes responsible for the breakdown of these structural proteins.  

In most patients, the natural history of AAA is one of expansion. Size is the most important predictor of AAA rupture. Accordingly, determining the rate of growth and the size at which aneurysms are at significant risk for rupture are important for predicting who will benefit from elective repair. These principles are also valuable to the emergency physician managing patients with AAA. First, understanding the association between AAA size and rupture can aid in estimating the likelihood that a patient’s symptoms are related to an aortic rupture or some other abdominal process. Second, it can guide the type and urgency of follow-up needed in asymptomatic patients in whom AAA is detected in the ED.  

The average growth rate of AAAs is from 0.2 to 0.6 cm/y, with larger aneurysms growing at a faster rate than smaller ones. It is currently impossible to predict which aneurysms will undergo significant growth. Although it seems that there is a substantial increase in rupture risk for aneurysms greater than 5.0 to 6.0 cm, estimating the precise risk associated with a specific aneurysm diameter is challenging. The Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery have pooled results from various studies and predicted annual rupture risks based on AAA diameter: size less than 4 cm, 0%; size 4 to 5 cm, 0.5% to 5%; size 5 to 6 cm, 3% to 15%; size 6 to 7 cm, 10% to 20%; size 7 to 8 cm, 20% to 40%; larger than 8 cm, 30% to 50%. Because the mortality associated with surgical repair of AAA is considerable (2.1%–5.5%), elective repair is generally not recommended until aneurysms have reached 5.5 cm (or increase at a rate >1 cm/y), at which point the annual risk of rupture outweighs the risks associated with surgery. Other factors, including female sex, current smoking, and hypertension, have been associated with an increased risk for rupture and should be factored on a case-by-case basis into decisions regarding elective operative repair versus ongoing ultrasound surveillance for AAA growth. Two large trials have addressed the question of whether elective repair versus surveillance for small aneurysms (4.0–5.5 cm) decreases mortality, and it seems that early repair does not improve survival. As previously mentioned, the most widely accepted and feared complication of AAA is rupture. It is estimated that 50% of patients with a ruptured AAA die before reaching the hospital. Of those who reach the hospital alive, the overall mortality is 80%. This figure comes from one of the few studies reporting on all patients with ruptured AAA arriving to the hospital rather than just those undergoing operative repair. In this same cohort, 43% did not undergo operative repair, all of whom died. Fifty-seven percent received surgery, with a subsequent mortality of 64%. A recent meta-analysis including more than 21,000 patients with ruptured AAA taken for operative repair suggests a lower mortality of close to 50%. 

Vascular Abdominal Emergencies
Ruptured or leaking AAA should be suspected in any older patient presenting to the ED with the clinical findings, in isolation or combination, of abdominal pain, back pain, and shock. Based on the cohort examined, these symptoms and signs are variably present. One of the larger case series suggests that abdominal pain or back pain is present in 95% of patients with a ruptured AAA. Based on another large series, most patients presenting with a ruptured AAA are hypotensive in the prehospital setting. Of patients who arrive at the hospital alive with a ruptured AAA, about two-thirds have ruptured into the retroperitoneal space, a quarter intraperitoneal, with the remaining rupturing into the IVC or duodenum. Before leaking or rupture, aortic aneurysms rarely produce prodromal symptoms; therefore, most patients presenting to the ED with this vascular emergency are unaware that they have this disease.

The early management of patients with suspected unstable AAA, either leaking or ruptured, should focus on rapid diagnosis and early definitive treatment. The presence of known risk factors for the disease (such as male gender, advanced age, and a history of smoking) in conjunction with acute abdominal or back pain should heighten suspicion for an unstable AAA. Abdominal palpation for a pulsatile mass has a widely ranging sensitivity of 44% to 97%.

At this time, bedside ultrasound is the most useful tool in the diagnostic evaluation of AAA. For emergency physicians, ultrasound is a highly accurate test, with a sensitivity and specificity approaching 100% in detecting AAA. Ultrasound has the added advantage of being able to detect free fluid in the abdomen, associated with a free intraperitoneal rupture. However, sonography does not reliably distinguish between ruptured and asymptomatic aneurysms because hemorrhage (in patients who have not already exsanguinated into the peritoneum) most frequently occurs into the retroperitoneal tissues, where ultrasound assessment is limited. Regarding the mechanics of the test, the low-frequency abdominal probe should be used and measurements taken in a plane perpendicular to the axis of the vessel. CT might be a useful imaging modality for asymptomatic patients with a newly diagnosed AAA to fully define the anatomic details of the aneurysm; however, it is generally not considered as the initial test of choice for patients with suspected ruptured AAA if ultrasound is rapidly available. CT may be appropriate if the ultrasound is technically limited or cannot distinguish between a symptomatic or incidental AAA.

For those with a suspected ruptured AAA, resuscitation, diagnostic measures, and consultation with appropriate specialties, ideally vascular surgery, should occur simultaneously. With large-bore intravenous lines in place, judicious fluid and blood replacement should begin for patients in shock, while recognizing that the underlying disorder is an arterial rupture and hemorrhage that can only be corrected by prompt surgical repair. Although prognosis for a ruptured AAA is poor, there do not seem to be early prognostic factors that reliably predict who can and cannot survive a ruptured AAA; therefore, most patients should be aggressively resuscitated.

When aneurysms are detected in asymptomatic ED patients and rupture has been excluded, referral for outpatient follow-up is adequate in most cases. For patients with an aneurysm greater than 5.4 cm, referral should be made to vascular surgery. For those between 4.0 and 5.4 cm, surveillance with ultrasound is recommended every 6 to 12 months, and, for those less than 4.0 cm, every 2 to 3 years is probably sufficient.

Iliac Artery Aneurysm

Iliac artery aneurysms are defined by a focal dilatation in the vessel greater than 1.5 cm. In most case series, symptoms and rupture do not occur until aneurysm
size is much larger, usually greater than 4 cm.\textsuperscript{83} The risk factors and pathophysiology associated with aneurysms at this site are similar to those for AAA. About 10\% to 20\% of patients with AAA also have an iliac artery aneurysm.\textsuperscript{84,85} Isolated iliac artery aneurysms are rare but, when present, the common iliac artery is the most commonly affected site.\textsuperscript{85} There are a few key factors regarding this disease of which the emergency physician should be aware. Compared with AAA, they are more likely to present with urologic symptoms consistent with renal colic.\textsuperscript{84,85} In addition, they are more difficult to visualize with sonography, with one cohort study suggesting that up to 50\% of isolated iliac artery aneurysm are missed with this imaging modality.\textsuperscript{84} When patients with a ruptured iliac artery aneurysm are taken for operative repair, the mortality is about 60\%, higher than for AAA, potentially explained by the increased technical difficulties associated with an operation at this site.\textsuperscript{84} Unlike AAA, there are no large-scale studies to inform the management of this disease in asymptomatic individuals; however, elective surgery is generally considered when aneurysm size is greater than 3.5 cm, and strongly recommended when greater than 5 cm.\textsuperscript{85}

**Visceral Artery Aneurysms**

Visceral artery aneurysms, which include renal and splanchnic lesions, receive less attention than AAA because they are rare and usually asymptomatic.\textsuperscript{66} The prevalence of these aneurysms is unknown. Based on one of the larger case series, 95\% of the visceral artery aneurysms are detected during routine investigation into unrelated abdominal symptoms.\textsuperscript{86} Of those detected, splenic and hepatic artery aneurysms are by far the most common, comprising 80\% of visceral artery aneurysm.\textsuperscript{87} The natural history is not well understood, and the clinical presentation poorly defined. In about one-third of patients, multiple aneurysms are present.\textsuperscript{88} Most patients are diagnosed or present with symptoms in the sixth decade of life.\textsuperscript{88} Aneurysms that rupture are typically greater than 2 cm, so this is often considered the threshold for repair in patients with asymptomatic disease.\textsuperscript{86} Because of the low prevalence of these aneurysms, their investigation is most likely to be prompted by high-risk clinical findings in patients whose abdominal symptoms have not been otherwise explained.

**Splenic Artery Aneurysm**

Splenic artery aneurysm accounts for 60\% of visceral artery aneurysms.\textsuperscript{89} Most are true aneurysms. Common risk factors include arteriosclerosis, portal hypertension, pancreatitis, and trauma.\textsuperscript{88} There is an increased prevalence of the disease in women, particularly those who are multiparous. With the increased use of high-resolution ultrasonography in pregnancy, more of these aneurysms are being detected in otherwise healthy pregnant women.\textsuperscript{89}

Most patients are asymptomatic until the time of rupture, which is a rare occurrence. Pregnancy and aneurysm size greater than 2 cm increase the risk of rupture. Patients in whom the aneurysm has ruptured most commonly present with abdominal pain. Shock can be delayed if the initial aneurysm rupture is contained within the lesser peritoneal sac, leading to a double-rupture phenomenon. The treatment of rupture is both resuscitative and operative, historically including a splenectomy.\textsuperscript{88} Endovascular techniques, specifically transarterial embolization, have also been successfully used.\textsuperscript{90} There is no consensus on the management of asymptomatic aneurysms.

**Hepatic Artery Aneurysm**

Aneurysms of the hepatic artery are the second most common visceral artery aneurysm, comprising 20\%. More than half (77\%) occur in the common hepatic artery, and most of the rest in the extrahepatic segment of the proper hepatic artery. In
contrast with splenic aneurysms, these aneurysms are more common in men.\textsuperscript{87} About 50% of these lesions are pseudoaneurysms, most likely related to complications of interventional biliary procedures.\textsuperscript{88} Vascular diseases, in particular fibromuscular dysplasia and polyarteritis nodosa, are associated with both aneurysm formation and rupture. Although abdominal pain and shock suggest rupture, even unruptured hepatic artery aneurysms can be symptomatic secondary to compression on the biliary tree. About 50% of aneurysms rupture into the biliary tract and can present with the classic triad of biliary colic, hematemesis (caused by hemobilia), and jaundice.\textsuperscript{88} The mortality of 40% associated with rupture seems to be consistent across case series data.\textsuperscript{91} As with splenic artery aneurysm rupture, the treatment has historically been surgical repair; however, transarterial embolization is an increasingly used technique.\textsuperscript{91}

\textbf{Renal Artery Aneurysm}

Like other visceral artery aneurysms, renal artery aneurysms are most commonly incidental findings during abdominal imaging or investigations into renovascular hypertension. Similar to splenic aneurysms, there is an association with multiparous women. Arterial fibrodysplasia is the most common vascular disorder associated with these aneurysms.\textsuperscript{92} Based on case series data, about 20% are bilateral. Rupture seems to be extremely rare.\textsuperscript{92,93}

\textbf{SUMMARY}

The emergency physician should consider the possibility of vascular disorders in patients presenting with abdominal complaints. These clinical entities tend to be dangerous and time urgent. In maintaining an appropriate level of suspicion and assessing a patient’s risk, the factors associated with the various diseases need to be considered. If a vascular emergency is deemed likely, the appropriate diagnostic studies need to be initiated promptly and consultants engaged early. Clinician-performed bedside ultrasonography is an invaluable tool, particularly if the concern is for ruptured or leaking AAA. CT angiography is becoming the initial imaging test of choice for abdominal vascular diseases caused by occlusive thrombosis. With rapid diagnosis and appropriate resuscitation, many of these vascular emergencies are correctable.

\textbf{REFERENCES}