DISEASE SUMMARY

Definition

An aneurysm is a permanent, localized dilation or abnormal outpouching of the wall of a blood vessel with an increase in diameter of >50%. More than 90% of abdominal aortic aneurysms are inferior to the renal arteries, and many (i.e., two thirds) extend into the common iliac arteries, which provide blood to the lower extremities. An aneurysm of the abdominal aorta (AAA) is defined by an aortic diameter that exceeds 3 cm (normal infrarenal aorta = 2 cm). Both the location and appearance of the AAA are illustrated in Disease Summary Figure 2.1. The term aneurysm is derived from the Greek word aneurysma, meaning “widening.”

Prevalence

Abdominal aortic aneurysms can affect anyone, but they most commonly occur after 60 years of age. In the United States, these abnormalities are present in 5–7% of the population older than age 60, and males are five times more likely than females to be affected. Peak incidence occurs at age 70. Abdominal aortic aneurysms are significantly more common in Caucasians than in African-Americans, Asians, or persons of Hispanic heritage. The frequency has tripled during the last 30 years and, as the population ages, is expected to increase further. The increasing incidence has been noted in other Western countries as well and appears to be more than a reflection of the increasing age of the population and improved diagnostic methods.

Significance

Abdominal aortic aneurysms typically increase in size by approximately 10% of their diameter each year. Most AAAs are asymptomatic. However, once they progress to a diameter of 5 cm, they can rupture and cause profuse bleeding and hypovolemic shock (a medical emergency) or exert pressure on adjacent viscera. Due to the element of stagnant blood flow (i.e., stasis) within the dilated pouch, AAAs may also give rise to thrombi (i.e., intravascular blood clots). Thrombi may fragment, circulate as thromboemboli, and cause sudden obstruction of smaller vessels (e.g., cause a stroke when emboli obstruct cerebral vessels). Approximately 40,000 patients undergo surgical treatment for AAAs in the United States each year, and more than
15,000 deaths are attributed to this condition annually. Abdominal aortic aneurysms are currently the 9th leading cause of death in men and the 13th most common cause of death in women in the United States. The mortality rate from a ruptured aorta is approximately 90%.

**Causes and Risk Factors**

The risk factor that is most strongly associated with the development of AAAs is atherosclerosis. Other risk factors include increasing age, male gender, hypertension, a medical history of aneurysms in the lower extremities (e.g., popliteal or femoral arteries), and a positive family history of aneurysms. Approximately one in four cases is observed in persons with a first-degree relative who has had an AAA.

Abdominal aortic aneurysms are manifestations of a degenerative process of the abdominal aorta that is often attributed to atherosclerosis—a cardiovascular disease characterized by inflammation and the accumulation of lipids, fibrous connective tissue, and calcium salts in the walls of large muscular and elastic arteries. At least 80% of aortic aneurysms arise from atherosclerosis, which weakens the aortic wall in localized areas. Blood pressure within the aorta subsequently causes dilation at the site of weakness. Other causes of AAA include:

- inherited diseases (such as Ehlers-Danlos syndrome) of defective connective tissue (e.g., elastin) components responsible for the strength of the aorta
- physical trauma to the aorta
- aortitis, in which an inflammatory process in the wall of the aorta results in localized regions of weakening
- mycotic (i.e., fungal) infections that may be associated with immunodeficiency, intravenous drug use, or open-heart surgery. Microorganisms circulating in the blood occasionally become lodged in the wall of the aorta.

**Pathophysiology**

Due to constant stress on the vessel wall from arterial blood pressure and the absence of vasa vasorum (i.e., small arteries that perfuse the outer and middle layers of larger blood vessels), the aorta is particularly susceptible to the development of aneurysms. The wall of the aorta
contains smooth muscle and two major structural proteins (elastin and collagen) arranged in concentric layers to withstand arterial blood pressure. Abdominal aortic aneurysms arise as a result of a failure in the function of elastin and collagen. The specific inciting factors are unknown, but a genetic predisposition clearly exists. Elastin is the principal load-bearing element in the aorta, and both elastin fragmentation and degeneration are observed in the aortic wall at the site of an aneurysm. The smooth muscle layer (i.e., tunica media) of the aorta appears to degrade from proteolysis during the evolution of an AAA. Reports have documented increased expression and activity of metalloproteinases (a group of zinc-dependent enzymes responsible for tissue remodeling) in persons with AAA. These enzymes have the capacity to degrade virtually all components of the extracellular matrix of the aortic wall. A decreased level of tissue inhibitor of metalloproteinases may also play a critical role in the development of the aortic aneurysm.

Surgical specimens reveal inflammation (white cells are a source of metalloproteinases), thinning of the tunica media, and marked loss of elastin in weakened regions of the aorta. Blood pressure within the aorta then promotes bulging of the vessel at the site of a weakness.

**Diagnosis: Clinical Manifestations and Laboratory Tests**

Most AAAs are asymptomatic and are detected during a routine physical examination or with ultrasound or a CT (i.e., computed tomography) scan. Some patients describe a "pulsing sensation" in the abdomen. Peripheral pulses are often normal, but coexisting renal or lower extremity arterial occlusive disease is present in 1 of 4 patients. Occasionally, an AAA may cause clinical manifestations from local compression, such as early satiety (i.e., feeling "filled up" after having eaten very little), nausea, vomiting, urinary symptoms, or venous thrombosis when veins are compressed. Back pain can be caused by erosion of an aneurysm into adjacent vertebrae. Other clinical findings include groin pain, embolic phenomenon to the toes, elevated erythrocyte sedimentation rate, and fever. Patients with ruptured aortic aneurysms present with severe back, abdominal, or flank pain, temporary loss of consciousness, and hypotension (i.e., systolic blood pressure <90 mm).

*Abdominal ultrasonography is the screening test of choice,* is 98% accurate in measuring aneurysm size, and is valuable for monitoring aneurysm growth in patients with small aneurysms. Contrast-enhanced CT scanning not only precisely sizes the aneurysm but also defines its relationship with the renal arteries. Magnetic resonance imaging (MRI) is as sensitive as the CT scan and is useful if renal insufficiency (i.e., elevated blood urea nitrogen and serum creatinine concentrations) precludes the use of contrast-enhanced CT.

Angiography is especially beneficial for detecting an extension of the aneurysm above the renal arteries and in defining a thrombus within the aneurysm. The aorta is studied by injecting contrast media through a catheter that has been positioned into the aorta and then taking a rapid series of x-rays to capture blood flow.

A complete blood count (CBC) is often performed to determine if the aneurysm is leaking and the patient is losing blood internally.

**Appropriate Therapy**

*The primary goal of treatment is to perform surgery before serious complications develop.* For patients with a small AAA, attempts are made to reduce the rate of expansion and the risk for rupture. Smoking cessation and an aggressive approach to control hypertension are critical. Beta-blocker therapy should be instituted to reduce blood pressure and stress on the wall of the aorta. Beta blockers can be safely administered unless contraindications to their use exist, such as chronic obstructive pulmonary disease (COPD), drug allergies, bradycardia, or severe congestive heart failure. The aneurysm should be evaluated every 6 months with ultrasound. Surgical intervention should be offered if the aneurysm expands or becomes symptomatic irrespective of aneurysm size.

When AAAs are 4–5 cm in diameter, repair may be beneficial if patients are young, have a low operative risk, and have a relatively long life expectancy. Patients with an AAA of 5–6 cm in diameter may benefit from repair if they have major contributing factors for rupture—hypertension, smoking, or COPD. For patients at higher operative risk, the threshold for repair may be 6–7 cm in diameter, depending on their condition.
Surgical excision and synthetic graft replacement is the treatment of choice for most aneurysms of the abdominal aorta greater than 5 cm. An incision is made from below the breastbone to just below the navel. The graft is stitched in place in the aorta, the walls of the aneurysm are wrapped around the graft, and the incision is closed. The procedure takes 3–6 hours and the hospital stay is usually 5–8 days. Recommendation for repair must be balanced with the risk for rupture. The patient’s current quality of life has to be carefully considered when determining if an operative intervention is justified. Acute myocardial infarction (i.e., heart attack), arrhythmia (i.e., irregular heart rhythm), and stroke are the most common complications of this type of surgery.

Endovascular stent grafting has evolved over the last decade as an alternative treatment for AAA. Through a small incision made in the groin, a long, thin guide wire is threaded into the aorta to the aneurysm. A tube (catheter) containing the stent graft (which resembles a meshed collapsible straw) is guided over the wire and positioned into the aneurysm. The stent graft is activated by heat and expands, forming a stable channel for blood flow. Contraindications for operative intervention include severe COPD, severe cardiac disease, active infection, and other medical problems that preclude surgical intervention. Patients with these conditions may benefit best from endovascular stenting of the aneurysm. The procedure can be performed with epidural anesthesia, often in <2 hours and with minimal blood loss. These advantages have made repair of AAA feasible in high-risk patients previously considered inoperable. Additional advantages of endovascular repair include reduced incisional pain and fewer cardiopulmonary complications. Most patients are discharged from the hospital on the second post-operative day. Long-term durability of endovascular grafts needs to be established, however, before comparisons can be made with open repair for use in the good operative risk patient with asymptomatic AAA.

The mortality rate during surgery to insert a graft is approximately 2–5%.

Serious Complications and Prognosis

The diameter of the aneurysm highly correlates with the risk for rupture. The probability for rupture within 1 year is 2% for 4.0–5.9 cm aneurysms; 7% for 6.0–6.9 cm aneurysms; and 25% for 7+ cm aneurysms. Up to 90% of patients die from hemorrhagic shock before they reach the hospital or in the immediate perioperative period.

Mortality following elective open or endovascular repair is 1–5%. In general, a patient with an AAA >5 cm in diameter has a three-fold greater chance of dying from rupture of the aneurysm than from surgical resection. The 5-year survival rate after surgical repair is 60–80%. Five to ten percent of patients will develop another aortic aneurysm either adjacent to the graft or higher up in the thoracic aorta.

Suggested Readings


