Aneurysms of the Peripheral Arteries

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Definitions

From the Greek roots aneurysm are arteries that are broad (eurys) across (ana). In the strictest sense, exactly what aneurysmal must be defined, for example, an artery, vein, or ventricle. Just how broad across an artery must be to be considered an aneurysm has been the subject of many hallway discussions and interdisciplinary jokes. Johnston et al.2 suggested that a 50% or greater increase in size should be considered an aneurysm (Table 77.1). In the population from which the data were obtained, Johnston et al. noted that age, gender, and body habitus all affected the arterial size. He suggested an adjacent nonaneurysmal segment of artery be used to help clarify the departure from normal for the individual.

Widening of less than 50% of normal diameter is referred to as ectasia. While ectasia is not an aneurysm, it is still not normal and must be considered pathologic. Ectatic arteries may progress to aneurysm.

Arteriomegaly is the diffuse enlargement of multiple arterial segments, often contingously, of greater than 50% the expected diameter.3,4 This is seen most commonly in the aorto-iliofemoral distribution and has three patterns (Table 77.2). Patients with arteriomegaly tend to be younger than patients with aneurysms. Just over a third of subjects had a first-degree relative with an aneurysm, all of which were aortic. Providing evidence that supports arteriomegaly as a separate entity for aneurysmal disease, Sandgren and colleagues4 studied 183 consecutive patients with abdominal aortic aneurysms imaged by ultrasound to determine if the lower extremity arteries were of increased diameter versus age-matched control subjects. In this cohort eight common femoral aneurysms (4.4%) and four popliteal aneurysms (2.2%) were found. Excluding the aneurysmal vessels, the lower extremity vessels were no different in the controls.

A true aneurysm involves all three layers of the vessel wall. True aneurysms have two basic shapes. Fusiform aneurysms are symmetric widening of an artery. Saccular aneurysms bulge asymmetrically to one side. In practice, there is a continuum between fusiform and saccular. A saccular aneurysm is generally regarded as less stable than a fusiform aneurysm.5

A false aneurysm or pseudoaneurysm does not involve all vessel wall layers. Injury to a vessel results in a rupture that is contained by surrounding connective tissue. The pseudoaneurysm communicates with the artery via a small neck allowing back and forth flow of blood. Pseudoaneurysms are seen most commonly after trauma such as cannulation or when a surgical anastomosis degenerates and fails. Treatment of pseudoaneurysms has changed in recent years from ligation to ultrasound guided compression and most recently to percutaneous thrombin injection. The feasibility of this depends on several factors including the location, the width of the neck (a large neck may result in spillover of thrombin into the artery), and the ability to compress or isolate the neck by direct pressure or balloon catheter.4,6

A dissecting aneurysm occurs as a result of a nonruptured, limited dissection that expands over time. This is
thought to be due to hemodynamic stress on the structurally weakened arterial wall and chronic inflammation. Dissecting aneurysm most commonly occur in the aorta, but may be seen in other vessels including the carotid arteries, the splanchnic arteries, and the lower extremity arteries (Table 77.3).

Prevalence

The incidence and prevalence of aneurysms is difficult to assess. The aorta is the vessel most commonly found to be aneurysmal. A study found that 4.8% of men age 65 to 69 had an aneurysm and 10.8% of men age 80 to 89 had an aortic aneurysm.\textsuperscript{10} It is well accepted that the greatest risk for an aneurysm is having an aneurysm in a different location. In a selected population of 1500 patients with an aortic aneurysm, 3.5% had at least one other aneurysm, most often in the femoral or popliteal region (3%), followed by the mesenteric vessels (0.5%). In those with a femoral aneurysm, just over 90% had an aortic aneurysm, and in those with a popliteal aneurysm over 60% had an abdominal aortic aneurysm.\textsuperscript{11} A later study of 313 patients followed over 10 years demonstrated that 14% of the men (35 of 251 men in the population) and, surprisingly, none of the women had or developed popliteal aneurysms.\textsuperscript{12}

Aneurysm Etiology

Several factors are thought to be responsible, or in part responsible, for the weakening of the arterial wall and resultant aneurysm (Table 77.4). These include degenerative processes, inflammatory processes, giant cell arteritis, Behçet’s disease, connective tissue diseases, congenital causes, trauma, and

### TABLE 77.1. Usual size of arteries in centimeters and accepted size for aneurysm (female/male distribution, when available)

<table>
<thead>
<tr>
<th>Vessels</th>
<th>Normal size (cm)</th>
<th>Aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesenteric</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Celiac</td>
<td>0.53</td>
<td>1.0 cm</td>
</tr>
<tr>
<td>Superior mesenteric</td>
<td>0.63</td>
<td>1.1 cm</td>
</tr>
<tr>
<td>Carotids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Common</td>
<td>0.77/0.75</td>
<td>1.5 cm/1.5 cm</td>
</tr>
<tr>
<td>Internal</td>
<td>0.49/0.55</td>
<td>1.0 cm/1.1 cm</td>
</tr>
<tr>
<td>Bulb</td>
<td>2 × adjacent ICA or</td>
<td>1.5 × CCA diameter</td>
</tr>
<tr>
<td>Common Iliac</td>
<td>1.00/1.20</td>
<td>2.0 cm/2.5 cm</td>
</tr>
<tr>
<td>Internal Iliac</td>
<td>0.49/0.55</td>
<td>1.0 cm/1.1 cm</td>
</tr>
<tr>
<td>Common femoral</td>
<td>0.80/0.95</td>
<td>1.5 cm/2.0 cm</td>
</tr>
<tr>
<td>Popliteal</td>
<td>/0.90</td>
<td>1.5 cm [2.0 cm often used]</td>
</tr>
</tbody>
</table>

CCA, common carotid artery; ICA, internal carotid artery.

### TABLE 77.2. Arteriomegaly patterns

<table>
<thead>
<tr>
<th>Type</th>
<th>Aneurysms of the aorta to femoral segments</th>
<th>Ectasia distally</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Aneurysms of the femoral to popliteal segments</td>
<td>Ectasia proximally</td>
</tr>
<tr>
<td>II</td>
<td>Aneurysms of the aorta and popliteal segments</td>
<td>Ectasia in the intervening nonaneurysmal segments [iliofemoral]</td>
</tr>
<tr>
<td>III</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### TABLE 77.3. Location of aneurysms requiring surgical treatment as percentage of total

<table>
<thead>
<tr>
<th>Location</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortoiliac</td>
<td>68</td>
</tr>
<tr>
<td>Supraarenal</td>
<td>10</td>
</tr>
<tr>
<td>Femoral</td>
<td>1</td>
</tr>
<tr>
<td>Other</td>
<td>6</td>
</tr>
<tr>
<td>Popliteal</td>
<td>4</td>
</tr>
<tr>
<td>Splanchnic</td>
<td>3</td>
</tr>
<tr>
<td>Carotid</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

### TABLE 77.4. Syndromes associated with arterial aneurysms

<table>
<thead>
<tr>
<th>Cause</th>
<th>Etiology</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerosis</td>
<td>Degenerative</td>
<td>Any</td>
</tr>
<tr>
<td>Fibromuscular dysplasia</td>
<td>Degenerative</td>
<td>Any—renal, carotid, intracranial, iliac, mesenteric</td>
</tr>
<tr>
<td>Cystic medial necrosis</td>
<td>Degenerative</td>
<td>Any</td>
</tr>
<tr>
<td>Coarctation</td>
<td>Post-stenotic</td>
<td>Aorta</td>
</tr>
<tr>
<td>Thoracic outlet syndrome</td>
<td>Post-stenotic</td>
<td>Subclavian</td>
</tr>
<tr>
<td>Ehlers-Danlos syndrome</td>
<td>Connective tissue</td>
<td>Mesenteric, renal</td>
</tr>
<tr>
<td>Marfan syndrome</td>
<td>Connective tissue</td>
<td>Ascending aorta, pulmonary</td>
</tr>
<tr>
<td>Behçet’s disease</td>
<td>Inflammatory</td>
<td>Pulmonary</td>
</tr>
<tr>
<td>Kawasaki’s disease</td>
<td>Inflammatory</td>
<td>Coronary</td>
</tr>
<tr>
<td>Polyarteritis nodosa (parenchymal)</td>
<td>Inflammatory</td>
<td>Mesenteric (distal) and renal</td>
</tr>
<tr>
<td>Takayasu’s arteritis</td>
<td>Inflammatory</td>
<td>Great vessels and renal [poststenotic]</td>
</tr>
<tr>
<td>Giant cell arteritis</td>
<td>Inflammatory</td>
<td>Great vessel, poststenotic</td>
</tr>
<tr>
<td>Infectious</td>
<td>Bacterial, Fungal, Syphilitic, HIV</td>
<td></td>
</tr>
<tr>
<td>Posttraumatic</td>
<td>Deceleration, Instrumentation</td>
<td>Great vessels, Femoral, radial, axillary</td>
</tr>
<tr>
<td>Repetitive trauma</td>
<td>Ulnar</td>
<td></td>
</tr>
<tr>
<td>Tuberous sclerosis</td>
<td>Congenital</td>
<td>Mesenteric, aortic</td>
</tr>
<tr>
<td>Pregnancy associated</td>
<td>Idiopathic</td>
<td>Splenic, renal, mesenteric</td>
</tr>
</tbody>
</table>
Fibromuscular Dysplasia

Fibromuscular dysplasia (FMD) has been reported in nearly all arteries. The renal arteries are the most commonly affected [Fig. 77.1 A,B], followed by the carotids, the mesenteric arteries and iliac arteries. Stenosis is the most common consequence of FMD resulting in hypertension, claudication, pulsatile tinnitus, or an incidentally discovered bruit. Women are affected eight times more often than men. There are five types of FMD described, with the fibromuscular type being the most common. This appears as a "string of beads" on angiography with ectasia or microaneurysmal changes in the post-stenotic segments. Wall weakening caused by the FMD may result in macroaneurysm formation and rupture in any vessel, the renal, iliac, and hepatic arteries are most commonly reported. Dissection has been reported in the renal and carotid arteries. Carotid artery dissection should be considered in the setting of acute carotidodynia (lateral neck pain) with or without neurologic symptoms such as headache or Horner’s syndrome, especially in women with known FMD.

Treatment is most often required for the stenotic complications. When an aneurysm is large, repair should be considered. Renal artery aneurysms and rarely other splanchnic aneurysms of greater than 2 cm diameter should be considered for repair. Ex-vivo repair may be required for distal or branch vessel aneurysms of the renal artery.

Ehlers-Danlos Syndrome

Ehlers-Danlos syndrome (EDS) is caused by abnormal synthesis or storage of collagen. There are six different phenotypes. For all types of EDS combined, the syndrome occurs at a rate of 1 in 5000. Type IV, which is estimated to account for only 4% of cases, is caused by point mutation(s) in gene COL3A1, resulting in abnormal synthesis and secretion of procollagen III. Type IV affects the arteries and is the most fatal. The prevalence is less than 1 in 100,000 and it is transmitted in an autosomal dominant inheritance pattern. The clinical features include easy bruising; thin translucent skin with visible veins; facial appearance of a narrow nose, thin lips, and hollow cheeks; and rupture of the intestine, uterus, and arteries. The descending aorta may be affected, but medium-sized vessels such as the mesenteric and renal arteries are most frequently involved. Unlike the more commonly occurring types I, II, and III, joint hypermobility is typically limited to the fingers, and stretchy skin is not a prominent feature.

The typical presentation of EDS type IV is a young patient in the third or fourth decade with abdominal pain due to rupture of a splanchnic vessel. Imaging incidentally may reveal multiple aneurysms (Fig. 77.2). Cannulation of vessels frequently results in dissection or hemorrhagic complications including retroperitoneal hematoma and death. Noninvasive imaging with ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) is recommended.

In a review of 31 patients requiring surgery for symptomatic or very large arterial aneurysms, the initial surgical outcomes were good in terms of low mortality, but were fraught with frequent postoperative bleeding, and one out
of five patients required reexploration. Late outcomes showed that 40% of patients had graft-related complications and 11 of the 12 deaths over the follow-up period were vascular related. Endovascular placement of covered stents have been reported with expectedly uninspiring outcomes.

Spontaneous rupture of splanchnic and peripheral vessels, including the carotid, is frequently reported. Survival is poor, with over half dying prior to or during the perioperative period. Prevention of arterial damage by avoidance of activities that might entail the risk of sharp or blunt trauma is advised. Collagen analysis of the patients’ cultured fibroblasts or genetic analysis is advised whenever the diagnosis is considered.

Behçet’s Syndrome

Behçet’s syndrome is a medium-size and small vessel systemic vasculitis of unclear etiology. It was first described in Turkey by Dr. Hulusi Behçet as a syndrome of relapsing oral ulcers and iritis with hypopyon. It is uncommon, affecting less than 1 in 100,000 in Western countries. The frequency is much higher in the Mediterranean, Middle Eastern, and Far Eastern countries [most prevalent in Turkey, at 1 in 1000]. Vascular manifestations include superficial phlebitis and
arterial aneurysm of both the systemic and pulmonary vessels. There are no specific laboratory criteria for Behçet’s syndrome. Diagnosis is made in the setting of recurrent (three or more in 12 months) oral ulcers, with two of four minor criteria met: [1] recurrent genital ulcers, [2] eye lesions—anterior or posterior uveitis or retinal vasculitis, [3] skin lesions—erythema nodosum, pseudofolliculitis, or pustulopapular lesions, or [4] pathergy—sterile pustules at sites of needle sticks.

Vascular involvement most frequently presents as thrombosis of either arteries or veins. In one major review, 14% had venous complications including deep, superficial, mesenteric, and pulmonary venous thrombosis. An increase in thrombotic tendency is present but the mechanism is not defined. Arterial involvement, typically aneurysms, although stenotic lesions may also be seen, was present in only 1.6%. These general numbers were supported in a more recent review by Tohme et al. Popliteal, coronary, carotid, and aortic aneurysms have all been reported. The incidence of aneurysms is likely to increase as imaging techniques improve and become more readily available. Saba et al. found 3.9% of patients with Behçet’s have had arterial aneurysms, with pulmonary aneurysms being the most common. In fact, the most common pulmonary manifestation is hemothysis owing to a pulmonary artery aneurysm.

Historically, treatment of aneurysms in Behçet’s has been surgical repair. While outcomes are good initially, there is a high rate of late pseudoaneurysm formation and rupture. Endovascular treatment has been reported in a number of cases with mixed results.

**Neurofibromatosis**

Aneurysms may be associated with neurofibromatosis. This autosomal dominant disorder presents as café-au-lait spots and cutaneous neurofibromas. Vascular involvement is present in less than 4% of affected individuals. Most often seen is smooth tapered stenosis that may involve large, medium-size, or small vessels. Hypertension is common when the renal vessels are affected. Claudication and or hypertension are commonly seen when aortic stenosis is present. Aneurysmal change has been reported, most often in the renal arteries, as post-stenotic dilatation (Fig. 77.3). Peripheral artery aneurysms and rupture of splanchnic vessel aneurysms have been described. Peripartum rupture of mesenteric aneurysms has been well described and should be considered in pregnant women with abdominal pain and known neurofibromatosis.

**Kawasaki’s Disease**

Originally described in Japan, Kawasaki’s disease has now been described worldwide with an incidence of 10 per 100,000. Children 5 years of age or younger are most commonly affected. Coronary and systemic aneurysms are found in 20% to 30% of patients and rupture has been reported. Thrombosis is a risk in large aneurysms, although the degree of risk and optimal treatment strategies are not yet defined. In adults aortic, iliac, mesenteric, and renal artery aneurysms may be found.

**Infected Aneurysms**

Wilson et al. categorized infected aneurysm into four types: mycotic aneurysm, microbial arteritis, infection of existing aneurysm, and posttraumatic infected pseudoaneurysms. The incidence of infected aneurysms is increasing due to invasive procedures, immunocompromised hosts, and intravenous drug use. Infected aneurysms develop from septic emboli that lodge in the lumen or in the vasovasorum of arteries, causing infection of the vessel wall. Destruction of the media by the inflammatory response then leads to aneurysm formation.

The clinical presentation of infected aneurysm includes localized pain, fever, leukocytosis, positive blood cultures, palpable mass, and rupture. Blood cultures are frequently negative due to recent antibiotic use. Infected aortic aneurysms may cause vertebral body erosion. The angiographic appearance of an infected aneurysm may be sacular in an otherwise normal-appearing vessel (in contrast to a saccular aneurysm associated with atherosclerosis), multilobulated or eccentric with a relatively narrow neck. White blood cell scans are useful when the diagnosis is in doubt or the extent of the infected segment is unclear. Repair of an infected aneurysm is a surgical resection with local debridement, and arterial reconstruction most commonly by extra-anatomic bypass, although primary reconstruction has been reported. Extended antibiotic treatment is usually required. The different subtypes are briefly discussed below.

**Mycotic Aneurysms**

The term was coined by Osler for a patient with endocarditis that embolized and seeding the arterial wall causing an infected aneurysm. The term mycotic aneurysm is still
used interchangeably with infected aneurysm to this day. The overall incidence of mycotic aneurysms has decreased in the era of antibiotics. Infected aneurysms may involve any segment of the arterial tree. Arteries commonly affected include the aorta, intracranial arteries, the superior mesenteric artery, and the femoral arteries. Organisms most often found include *Streptococcus viridans*, *Staphylococcus aureus*, *Streptococcus faecalis*, *Staphylococcus epidermidis*, and *Candida albicans*.

**Microbial Arteritis**

Microbial arteritis with secondary aneurysm formation has become more common than mycotic aneurysms. Disruption of the arterial wall, most often from atherosclerosis, is the most important factor that allows blood-borne bacteria to penetrate and colonize the arterial wall. Common organisms include *Escherichia coli*, *Salmonella*, and *Staphylococcus*. HIV infection appears to increase the risk of microbial arteritis as well.

**Infection of an Existing Aneurysm**

The abdominal aorta is the most common site for secondary infection of an aneurysm, but other sites have been reported. Gram-positive bacteria are most frequently involved.

**Posttraumatic Infected Pseudoaneurysm**

Posttraumatic infected pseudoaneurysms are common in intravenous drug users who inject at the femoral or carotid arteries. Invasive procedures and closure devices can also cause iatrogenic posttraumatic infected pseudoaneurysms.

**Pregnancy**

Pregnancy is associated with both aneurysm formation and rupture. Several factors may account for this including hemodynamic changes and collagen or elastic tissue changes. The rupture risk is elevated in labor and in the peripartum period. Because of this well-described relationship, all symptomatic aneurysms during pregnancy and large aneurysms meeting criteria for repair in women of child-bearing years are recommended for repair.

**Aneurysms of Specific Vessels**

Aneurysms of specific locations are covered in this section. Where available the incidence and natural history are presented. See Table 77.5 for a summary.

**Splanchnic Artery Aneurysms**

Aneurysms of the splanchnic arteries (arteries supplying the thorax and abdominal viscera) are rare. Data on incidence and outcomes are limited to case studies and institutional reviews. As imaging techniques have improved with CT and MR angiography, the result has been a recognition of these previously unrecognized aneurysms. Despite the improvement in imaging modalities, the distribution of splanchnic artery aneurysms has not changed. Etiology is varied and is discussed with the specific sites.

### TABLE 77.5. Location, etiologies, incidence and natural history of systemic aneurysms

<table>
<thead>
<tr>
<th>Site</th>
<th>Etiology</th>
<th>Incidence</th>
<th>Natural history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid</td>
<td>Arterial dysplasia</td>
<td>Very rare</td>
<td>Thrombosis, occlusion</td>
</tr>
<tr>
<td></td>
<td>Atherosclerotic</td>
<td></td>
<td>Embolism</td>
</tr>
<tr>
<td></td>
<td>Inflammatory</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Trauma (blunt or penetrating)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Spontaneous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subclavian/axillary</td>
<td>Post-stenotic</td>
<td>Rare</td>
<td>Thrombosis, embolism</td>
</tr>
<tr>
<td></td>
<td>Thoracic outlet</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Trauma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mesenteric</td>
<td>Multiple pregnancies</td>
<td>Rare</td>
<td>Rupture (more in pregnancy)</td>
</tr>
<tr>
<td>Splenic</td>
<td>Fibrodyplasia</td>
<td></td>
<td>Dissection</td>
</tr>
<tr>
<td>Hepatic</td>
<td>Portal hypertension, pancreatitis</td>
<td></td>
<td>Pain</td>
</tr>
<tr>
<td>Celiac</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior mesenteric</td>
<td>Atherosclerosis</td>
<td>Uncommon</td>
<td>Rupture, embolism</td>
</tr>
<tr>
<td></td>
<td>FMD, Ehlers-Danlos</td>
<td></td>
<td>HTN, pain</td>
</tr>
<tr>
<td></td>
<td>Inflammatory</td>
<td></td>
<td>Local compression</td>
</tr>
<tr>
<td>Renal</td>
<td>Trauma, iatrogenic</td>
<td>3 per 100,000</td>
<td>Rupture</td>
</tr>
<tr>
<td>Iliac</td>
<td>Atherosclerotic</td>
<td>4 per 100,000</td>
<td>Not known</td>
</tr>
<tr>
<td></td>
<td>FMD, infection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Femoral</td>
<td>Atherosclerotic</td>
<td>7.8 per 100,000</td>
<td>Thrombosis, occlusion</td>
</tr>
<tr>
<td></td>
<td>Iatrogenic</td>
<td></td>
<td>Embolism</td>
</tr>
<tr>
<td>Popliteal</td>
<td>Atherosclerotic</td>
<td></td>
<td>Rupture (rare)</td>
</tr>
<tr>
<td></td>
<td>FMD, Trauma</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

FMD, fibromuscular dysplasia; HTN, hypertension.
Celiac Artery Aneurysms

Aneurysms of the celiac artery are uncommon, accounting for only 4% of all splanchnic artery aneurysms. Post-stenotic dilatation is common, usually the result of an arcuate ligament compression, but this rarely progresses to aneurysm. The most common etiology of celiac aneurysm is atherosclerosis, although other etiologies occur [Fig. 77.4].

Symptoms indicate expansion and may mimic pancreatitis. A pulsatile epigastric mass may be present. Rupture has been found to be as high as 13% in one study. Treatment by surgical resection and primary anastomosis or aortoiliac reconstruction is successful in the vast majority of cases. A hostile (surgically difficult field due to ventral mesh, adhesions etc.) abdomen is a contraindication to elective surgical reconstruction. Aneurysms with narrow necks is successful and may be the only option in the setting of the severely ill or those with a hostile abdomen.

Splenic Artery Aneurysms

Splenic artery aneurysms account for 60% of splanchnic artery aneurysms and are fairly common. Several associated conditions are recognized, including portal hypertension, chronic pancreatitis with pseudocyst formation, trauma, and infection, but the most strongly associated conditions are fibromuscular dysplasia and repeated pregnancy. Whether the hormonal changes affecting collagen tissues or the local hemodynamic changes during pregnancy or both account for the association with multiple pregnancies is not yet determined. Whatever the reason may be, the female-to-male ratio for splenic artery aneurysms is 4:1.

The presentation is usually incidental as a finding on abdominal X-ray, CT, MRI, or ultrasound. When symptomatic, left upper quadrant fullness or tenderness may be present. Symptoms that worsen acutely may be due to expansion, and if proven should be treated. Rupture is rare, estimated as less than 2% of previously known asymptomatic aneurysms, but may result in death. There is no evidence to support the notion that calcification of a splenic artery aneurysm is protective.

Treatment is indicated for all asymptomatic patients. Elective repair is indicated for good-risk patients, particularly women of childbearing age who may become pregnant, when aneurysm size is greater than 2 cm in diameter. The highest rate of rupture for splenic aneurysms is in pregnant women, where rupture risk is estimated at up to 9.6%. Mortality of both the mother and fetus is 70% or greater after rupture. Open surgical repair, coil embolization, and endovascular or open ligation have been used for repair. Splenic artery ligation for proximal splenic artery aneurysms will leave the spleen adequately perfused via collaterals in most cases. Previously, aneurysms in the splenic hilum and pancreatic locations had required distal ligation and often splenectomy. Alternative approaches, including endovascular coil embolization and direct thrombin injection, have proven effective in selected cases.

Superior Mesenteric Artery Aneurysms

The superior mesenteric artery (SMA) is the third most commonly affected splanchnic artery aneurysm, accounting for 6% of cases. Infection is more common in this location than in others, especially at the proximal portion. Dissection with subsequent aneurysm formation is rare in general, but is seen more often at the SMA than at other sites.

The presentation, when not incidental on imaging, may mimic intestinal ischemia due to the central location of the artery. A pulsatile epigastric mass, tender and mobile, may be present. Rupture rate is as high as 40%. Extensive thrombus may effectively eliminate the collateral blood supply from both the celiac and inferior mesenteric vessels. If the origin of the SMA is compromised by dissection, the resulting ischemia is severe.

Treatment is indicated in most cases. Surgical resection and reconstruction can be performed safely in the majority of patients. Endovascular embolization of saccular aneurysms with narrow necks is successful and may be the only option in the setting of the severely ill or those with a hostile abdomen.

Hepatic Artery Aneurysms

Hepatic artery aneurysms account for 20% of splanchnic aneurysms. Infection (commonly in intravenous drug users), trauma, and atherosclerosis are common causes. Men are affected more often than women, at a ratio of 2:1. Polyarteritis nodosa and cystic medial necrosis are less common causes. Hepatic artery aneurysms as reported tend to be large (greater than 3 cm) and saccular. Smaller aneurysms

**TABLE 77.6. Splanchnic artery aneurysms**

<table>
<thead>
<tr>
<th>Location</th>
<th>Relative percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Celiac trunk</td>
<td>4</td>
</tr>
<tr>
<td>Splenic</td>
<td>60</td>
</tr>
<tr>
<td>Hepatic</td>
<td>20</td>
</tr>
<tr>
<td>Gast/epiploic</td>
<td>4</td>
</tr>
<tr>
<td>Gastroduodenal</td>
<td>1.5</td>
</tr>
<tr>
<td>Superior mesenteric trunk</td>
<td>5.5</td>
</tr>
<tr>
<td>Pancreatic</td>
<td>2</td>
</tr>
<tr>
<td>Jejunal/Ileal/Colic</td>
<td>3</td>
</tr>
<tr>
<td>Inferior mesenteric</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

**FIGURE 77.4. Patient with fibromuscular dysplasia of the celiac artery with beading and post-stenotic aneurysms.**
Renal Artery Aneurysms

The incidence of renal artery aneurysms is not well established. Estimates range from slightly more than 1 in 10,000 on autopsy series to up to 1% on an angiography series. This latter number likely overestimates the incidence since the patients undergoing angiography were preselected for renal arteriography due to hypertension. Women are affected more often than men, primarily due to FMD. When FMD is excluded, the male-to-female ratio is 1:1. Ten percent of renal artery aneurysms are bilateral.

True aneurysms are most often saccular (75%) or extraparenchymal (90%), and the incidence peaks in the fifth and sixth decades. Location is most often at the bifurcation of the main renal artery. Atherosclerosis is commonly present but not seen in all cases, and is likely not causal since poststenotic aneurysms tend to be fusiform whether due to atherosclerosis or FMD. Fibromuscular dysplasia is frequently associated with small, multiple renal artery aneurysms. Large aneurysms have also been seen with FMD, most often as a post-stenotic complication. When available for histopathology the overwhelming majority of renal artery FMDs are of the medial type (99%), with the medial subtype in 55%, the subadventitial subtype in 28%, and the combined subtype in 16%.

The majority of aneurysms are found incidentally or as part of a hypertension evaluation. The vast majority of aneurysms associated with hypertension are fusiform, with the arterial stenosis causing both the hypertension and the aneurysm. But in some cases the hypertension may be due to a distal embolism from thrombus within the aneurysm. Hypertension due to branch vessel compression by the aneurysm and, rarely, hydronephrosis due to compression of a ureter or the renal calyx have both been described. Natural history studies suggest that rupture is rare, with no deaths due to renal artery rupture noted in over 36,000 autopsies in southern Sweden. The presence or absence of calcification and rupture risk is in dispute. At this time there is no convincing evidence that calcification is protective. False aneurysms of the renal artery are most often due to trauma, blunt, penetrating, or iatrogenic trauma may cause the disruption of the vessel wall.

Intraparenchymal Renal Aneurysms

Intraparenchymal renal aneurysms are very rare, accounting for only 10% of all renal artery aneurysms. Most often these aneurysms are small and tend to be multiple. Many disease entities have been associated with these aneurysms. Trauma may be a cause, but connective tissue disease (EDS, for example) and polyarteritis are more common. Those due to PAN tend to be located very distally, in the cortex, and often regress with medical treatment.

Ideal treatment for renal artery aneurysms is not yet defined. Symptomatic aneurysms (especially in pregnancy), acute false aneurysms, and those with acute dissection should be repaired early. Elective repair has been proposed for women who may become pregnant—or are pregnant and can be treated safely—for aneurysms associated with FMD due to an already weakened vessel wall, and aneurysms of greater than 3.0 cm in size that can be treated without nephrectomy. Surgical repair has proven effective by a number of techniques. Aneurectomy and closure or ligation and bypass have proven to be effective and durable. Stent graft covering has been reported, but long-term and large series reports are not yet available.

Intraparenchymal aneurysms pose a challenge for repair. Observation for aneurysms less than 2 cm is reasonable. Treatment is recommended when they are larger or are causing hypertension, distal embolization, local expansion,
urologic symptoms, or are symptomatic in pregnancy. Catheter-directed embolization has also proven successful. Nephrectomy with bench repair and reimplantation is established in a number of centers.

Renal aneurysm due to arteriovenous fistulas or malformation is very rare. When present, the affected vessels may be striking in appearance (Fig. 77.6). Both congenital and acquired etiology occur. Significant shunting can develop, resulting in pulmonary hypertension and right heart failure. The majority of patients do not have symptoms or signs justifying treatment. When symptoms or signs are present, intervention must be carefully considered and planned to preserve renal function. Surgical, endovascular, or combined approaches may be utilized.

Other Splanchnic Artery Aneurysms
Aneurysms of other splanchnic arteries such as the inferior mesenteric may occur but are very uncommon.

Coronary Artery Aneurysms
Aneurysms of the coronary arteries are most commonly seen as a result of Kawasaki’s disease. Post-stenotic aneurysm (Fig. 77.7), EDS, and Behçet’s syndrome are just a few of the causes of these uncommon aneurysms. When coronary aneurysms are present, treatment with anticoagulation should be considered. This is especially true if intraaneurysmal thrombus is present or if distal embolization is evident.

Pulmonary Artery Aneurysms
Pulmonary artery aneurysm are most commonly seen as a late complication of vasculitis such as Behçet’s disease or Takayasu’s arteritis as a post-stenotic change. Rarely, diffuse pulmonary aneurysm is present in the setting of chronic pulmonary hypertension.

Subclavian and Axillary Artery Aneurysms
Subclavian artery aneurysms have multiple causes including trauma, fibromuscular dysplasia, syphilis, cystic medial necrosis, and tuberculous lymphadenitis. Aneurysms of the subclavian artery are more common in men older than 60 years. The most common cause of distal subclavian aneurysm is thoracic outlet obstruction [see below]. False aneurysm is seen secondary to instrumentation. Subclavian aneurysms present in a number of ways. Pain and ischemic symptoms from occlusion or distal thrombosis are fairly common. Neurologic dysfunction from brachial plexus compression, hoarseness due to recurrent laryngeal nerve compression, tracheal compressions, transient ischemic attacks [TIAs], or hemoptysis have all been described.

Physical signs of a subclavian aneurysm include a bruit, diminished pulse, blue finger syndrome, vocal cord paralysis, or Horner’s syndrome. Diagnosis can be established with duplex ultrasound, CT angiography or MR angiography.

Subclavian-Axillary Artery Aneurysms
Aneurysms of the distal subclavian artery and proximal axillary artery are frequently associated with cervical ribs and thoracic outlet syndrome. Although cervical ribs are present in 0.6% of the population, they are asymptomatic and bilateral in 50% to 80% of patients. A cervical rib or other bony abnormalities such as a prior clavicle fracture with poor union or excessive callus can result in repetitive trauma leading to stenosis. Eventually a post-stenotic
dilation and aneurysm formation occurs. Alternatively, the artery may course between the heads of the scalene anterior and scalene medius scalene muscles. Chronic compression of the subclavian artery leads to arterial wall changes, intimal injury, thickening, mural thrombus, and peripheral embolization.

The earliest arterial manifestation of thoracic outlet syndrome (TOS) is from an aneurysm-associated embolic event to the hand of the affected upper extremity [Fig. 77.8] (Raynaud’s syndrome may be present earlier, but this is not strictly an arterial syndrome). Arterial involvement of TOS is less common than venous or nerve compression, but has the potential for digit- and limb-threatening complications.

In patients with embolic events, aneurysm should be repaired regardless of the aneurysm size. Most patients require a short interposition vein or prosthetic graft in addition to cervical and/or first rib resection. Asymptomatic patients with a large aneurysm containing a large amount of thrombus should be considered for repair. Those with or without post-stenotic dilation in the setting of a cervical rib should be considered for cervical rib resection if they are an acceptable surgical risk. Endovascular angioplasty may be indicated for acute limb salvage, but angioplasty with or without stenting is typically not advised as definitive treatment unless the bony and muscular structural abnormalities are addressed.

Axillary artery aneurysms are mostly caused by blunt trauma (e.g., crutch-induced, shoulder dislocation) or penetrating trauma. There may be concomitant injury to the brachial plexus. An arteriogram should be performed in all cases of penetrating trauma to the shoulder and with blunt trauma if the patient has an abnormal pulse examination. Computed tomography angiography or MR angiography should be performed in patients with blunt trauma to the shoulder. Surgical resection with interposition vein graft is often performed. It is important to protect the brachial plexus and its branches during surgical treatment.

Ulnar Artery Aneurysms

Hypotenar hammer syndrome was first reported by Guattani in 1771, but it was Conn and associates who coined the term hypotenar hammer syndrome. Aneurysms of the ulnar artery develop in people who use the palms of their hands as a substitute for a hammer—pounding and banging stubborn materials into position. It has been described in occupations and sports as disparate as farming, soccer, and badminton. In practice, people who use their hands in physical activity are at risk. The ulnar artery and nerve enter the hand by Guyon’s canal, where the artery lies directly over the hook of the hamate bone. Trauma to the artery in this location can cause intimal injury, leading to thrombosis, and medial injury, leading to true or false aneurysm formation. Symptoms include pain, cold sensation, paresthesias, and mottling or cyanosis of the digits. Raynaud’s phenomenon, if present, is unilateral and spares the thumb. On examination there may be ischemic changes of the fingers, hypotenar callus, an abnormal Allen test, and a sensory deficit. Ulnar aneurysms may form a thrombus, resulting in complete occlusion of the ulnar artery or distal emboli in the ulnar distribution (digits 3, 4, and 5 most commonly involved).

Noninvasive studies are helpful to confirm the diagnosis. Plethysmography, digital pressures, duplex ultrasound, or CT or MR angiography can confirm the diagnosis [Fig. 77.9].
Aneurysms of the Lower Extremity

Iliac Artery Aneurysms

An iliac artery aneurysm, as an extension of an aortic aneurysm, is common. Isolated iliac artery aneurysms are rare. Of reported aortoiliac artery aneurysms, fewer than 1% were isolated to the iliac segment only. Prevalence is low, reported at only 0.03% in autopsy series. The vast majority of aneurysms involve the common iliac artery (70–90%), with a minority involving the internal iliac artery (10–30%). External iliac aneurysms are very rare. Males outnumber females by a ratio of 5:1 or more. Fifty percent of iliac aneurysms are bilateral.154

In surgical series the average size of iliac aneurysms on repair is 4 to 5 cm, and for those that had ruptured, the size has been estimated at 6 cm. Rupture rates have been reported to be 10% to 70% after 5 years, but no clear correlation between rupture risk and size has been demonstrated. There is a high mortality from rupture, greater than 25%, with surgical mortality low, at less than 5% for good-risk patients. In a study of 189 veterans with 323 iliac artery aneurysms, only six patients had symptoms, and two ruptured. All of these were greater than 4 cm in diameter. Expansion rates were slow for aneurysms smaller than 3 cm. Observation by ultrasound or other means for aneurysms of less than 3 cm is recommended at least yearly. Elective repair is indicated when the iliac size reaches 3 to 4 cm in good-risk patients. But if the risk is poor, observation for expansion ought to occur more frequently, and any symptoms should be investigated immediately. Repair may be interposition graft or aortoiliac reconstruction in cases of bilateral aneurysm. Endovascular repair of isolated iliac aneurysms using covered stents is an option. Occlusion of the internal iliac by coil embolization is required to avoid a type II endoleak.

Femoral Artery Pseudoaneurysms

Traumatic pseudoaneurysm of the femoral artery is common following instrumentation. A pulsatile mass and systolic and diastolic components to a bruit (to and fro) are the common physical findings of a pseudoaneurysm. This may be a subacute incident, independent of an immediate post-interventional hematoma or other complication. Treatment depends on the symptoms, size, and anatomy of the aneurysm. If the aneurysm is small and stable, observation and reassessment is appropriate. For large or expanding aneurysms, compression of the neck, or, increasingly common, injections of thrombin under ultrasound guidance are appropriate. For large pseudoaneurysms with a wide neck not amenable to thrombin injection, artery repair and ligation of the pseudoaneuhrism neck with evacuation of the hematoma is appropriate.

Popliteal Artery Pseudoaneurysms

The popliteal artery is considered aneurysmal when size is greater than 1.5 cm, but clinically 2 cm is often used as a guide. Pseudoaneurysms are frequently bilateral [Fig. 77.10]. The incidence of popliteal aneurysms in the population is not clear. In a prospective screening trial of asymptomatic men age 65 to 80 years, the incidence of a popliteal aneurysm (defined as greater than 1.5 cm) was only 1% (11 out of 1074 patients).

There is a well-accepted relationship between abdominal aortic and popliteal aneurysms. The incidence of popliteal aneurysms is higher in patients with aortic aneurysms, with studies reporting between 10% and 14% having concurrent aneurysms. Conversely, in patients with popliteal aneurysm the incidence of abdominal aortic aneurysms (AAAs) has been reported as 33% to 43%. In general, any patient with a popliteal aneurysm should be screened for an AAA. Nearly all popliteal aneurysms are associated with atherosclerosis. Case reports of trauma [often causing pseudoaneurysm], fibromuscular dysplasia, and other diverse etiologies can be found.
The clinical presentation is variable but includes distal embolization (55%) manifesting as claudication, blue toe syndrome, absent pedal pulses, critical limb ischemia, local compression (6.5%), and rupture, which is very rare (1.4%). Pain due to local compression or peroneal nerve paresis may occur. Venous congestion including acute venous thrombosis has been reported. Physical examination is most often normal, but when a large pulsatile mass is present at the popliteal fossa an aneurysm should be excluded. Diagnosis can be established by duplex ultrasonography, which provides important information about the diameter of the artery, the presence or absence of thrombus, and the patency of the distal vessels. Computed tomography and MR and conventional angiography are all useful modalities.

The rate of expansion appears to be slow. Due to associated complications, elective repair of aneurysms greater than 2 cm is recommended for good-risk patients. Smaller aneurysms with thrombus present and associated claudication or absent pedal pulses may warrant intervention or anti-coagulation and close observation with duplex ultrasound scanning. Surgical treatment may include interposition graft, bypass graft, or stent grafts.

Extracranial Carotid Artery Aneurysms

Extracranial carotid artery aneurysms are rare. True and false aneurysms may occur. Etiologies include trauma, fibro-dysplasia, carotid surgery, carotid dissection, or infection. True aneurysms are the most common type, more common in men, and nearly all are degenerative, that is, associated with atherosclerosis. The average age at diagnosis is 60 years. The carotid bulb is the most frequently involved segment.

Bilateral aneurysms are present in 12% of patients. The bulb is anatomically larger than the proximal and distal vessel normally. It is considered aneurysmal if maximal diameter is greater than twice the distal internal carotid artery (ICA) diameter. Extracranial carotid aneurysm may present as stroke, TIA, or cranial nerve palsies from compression. A non-tender cervical mass or pulsatile mass may be present. Duplex ultrasound is helpful to define the size and extent of involvement. Redundant vessel length with tortuosity is a common finding when imaged.

Surgical treatment is recommended for aneurysms greater than 2 cm in size, with resection and reconstruction with interposition saphenous vein graft. Complications associated with aneurysmectomy in patients with true aneurysms relate to stroke resulting from dislodgment of atherosclerotic debris, with distal embolization and injury of the cranial nerves. Transection of carotid sinus nerve and loss of its bar receptors function could result in neurogenic hypertension. False aneurysms of the carotid artery caused by trauma are rare and commonly occur after penetrating neck injury or blunt trauma.

Local repair may be possible if injury is limited or more extensive repair is needed in case of extensive tissue disruption. False aneurysms have been reported after carotid endarterectomy, and up to 20% may exhibit infection, often treated with excision and reconstruction with vein graft. Adequacy of intracranial collateral circulation in these patients may be assessed by preoperative positron emission tomography studies.

References


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