37 Diseases of the Aorta

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INTRODUCTION

The largest artery in the body, the aorta receives blood pumps from the left ventricle and distributes it distally to the branch arteries. While it is one continuous vessel, its segments have been distinguished anatomically. The aorta begins in the anterior mediastinum above the aortic valve as the ascending aorta, the most proximal portion of which is also called the aortic root. This is followed in the superior mediastinum by the aortic arch, which gives rise to the brachiocephalic arteries. The descending thoracic aorta then courses in the posterior mediastinum to the level of the diaphragm, after which it becomes the abdominal aorta that then bifurcates distally into the common iliac arteries.

AORTIC ANEURYSMS

Aortic aneurysms, defined as pathologic dilatation of the aorta, are one of the most commonly encountered aortic diseases. Aneurysms may involve any part of the aorta, but occur much more commonly in the abdominal than in the thoracic aorta. Abdominal aortic aneurysms have a prevalence of at least 3% in a population greater than 50 yr old *(1)*—although the exact prevalence varies with the age and risk of the population studied—and are five to 10 times more common in men than in women. The infrarenal aorta is the segment most often involved. Among thoracic aortic aneurysms, aneurysms of the ascending aorta are most common. When aneurysms involve the descending thoracic aortic aorta, they often extend distally and involve the abdominal aorta as well, producing a *thoracoabdominal* aortic aneurysm.

Etiology

Atherosclerosis has long been recognized as a major underlying cause of abdominal aortic aneurysms. While the mechanism by which atherosclerosis promotes the growth of aneurysms is uncertain, it appears that the atherosclerotic thickening of the aortic intima reduces diffusion of oxygen and nutrients from the aortic lumen to the media, in turn causing degeneration of the elastic elements of the media and a weakening of the aortic wall *(2)*. More recent research suggests that inflammation within the aortic wall may lead to degradation of the extracellular matrix, and thus also contribute to the development of abdominal aortic aneurysms *(3)*. Once the aorta begins to dilate, tension on the wall increases, thereby promoting further expansion of the aneurysm. There also appears to be a genetic predisposition to the development of abdominal aortic aneurysms, as 13 to 32% of first-degree relatives of those with abdominal aneurysms may be affected, compared with the 2 to 5% risk in the general population.

Atherosclerosis is also a common cause of aneurysms of the descending thoracic aorta. However, the most important etiology of ascending thoracic aortic aneurysms is a process known as *cystic medial necrosis* or *degeneration*, which appears histologically as smooth muscle cell necrosis and degeneration of elastic layers within the media. Cystic medial necrosis is found in almost all patients with Marfan's syndrome, placing this group at very high risk for aortic aneurysm formation at a

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relatively young age. Among patients without overt evidence of connective tissue disease, ascending thoracic aortic aneurysms occur commonly among those with an underlying bicuspid aortic valve, and also among those with a family history of similar aneurysms (i.e., familial thoracic aortic aneurysm syndrome). In addition, a history of long-standing hypertension is a common risk factor. Syphilis was once a common cause of thoracic aortic aneurysms, but is now a rarity. Less common causes of thoracic aortic aneurysms include great-vessel arteritis (aortitis), aortic trauma, and aortic dissection. Often thoracic aortic aneurysms are idiopathic.

Clinical Manifestations

The large majority of patients with abdominal and thoracic aortic aneurysms are asymptomatic and the aneurysms are discovered incidentally on a routine physical exam or imaging study. When patients with abdominal aortic aneurysms do experience symptoms, the most frequent complaint is of pain in the hypogastrium or lower back. The pain typically has a steady gnawing quality and may last for hours or days. New or worsening pain may herald aneurysm expansion or impending rupture. Rupture of an abdominal aneurysm is often accompanied by the triad of pain, hypotension, and the presence of a pulsatile abdominal mass. Those with thoracic aortic aneurysms may experience chest or back pain from aneurysm expansion or compression of adjacent structures. Aneurysms of the ascending aorta often will produce aortic insufficiency (due to dilatation of the aortic root), so patients may present with congestive heart failure or a diastolic murmur.

Diagnosis

Abdominal aortic aneurysms may be palpable on physical examination, although even large aneurysms are sometimes obscured by body habitus *(4)*. Typically abdominal aortic aneurysms are hard to size accurately by physical examination alone, as adjacent structures often make an aneurysm feel larger than it actually is. Thoracic aortic aneurysms, on the other hand, cannot be palpated at all on physical examination.

The definitive diagnosis of an aortic aneurysm is made by radiologic examination. Abdominal aortic aneurysms can be detected and sized by either abdominal ultrasonography or computed tomography (CT). Ultrasound is extremely sensitive and is the most practical method to use in screening for abdominal aortic aneurysms. While mass screening of the population has not yet become widely accepted, screening with ultrasound is generally recommended for patients considered to be at risk for aortic aneurysms *(5)*. CT is even more accurate and can size aneurysms to within a diameter of ± 2 mm, and is therefore the preferred modality for following aneurysm growth over time (Fig. 1).

Thoracic aortic aneurysms are frequently recognized on chest radiographs, often producing widening of the mediastinal silhouette, enlargement of the aortic knob, or displacement of the trachea from midline. CT is an excellent modality for detecting and sizing thoracic aneurysms and for following growth over time. Transthoracic echocardiography, which generally visualizes the aortic root and ascending aorta well, is useful for screening patients with Marfan's syndrome because they are at particular risk for aneurysms in this location.

Prognosis

Most aneurysms expand over time, and the rate of growth tends to increase with increasing aneurysm size. The major risk associated with an aortic aneurysm in any location is that of rupture. The risk of rupture rises with increasing aneurysm size because—in accordance with Laplace's law (which states that wall tension is proportional to the product of pressure and radius)—as the diameter of the aorta increases its wall tension rises. Abdominal aortic aneurysms of less than 4.0 cm in size have only a 0.3% annual risk of rupture, those 4.0 to 4.9 cm have a 1.5% annual risk of rupture, and those 5.0 to 5.9 cm have a 6.5% annual risk of rupture *(6)*. For aneurysms 6.0 cm or greater the risk of rupture rises sharply, although an exact risk cannot be estimated. The overall mortality from rupture of an abdominal aortic aneurysm is 80%, with a mortality of 50% even for those who

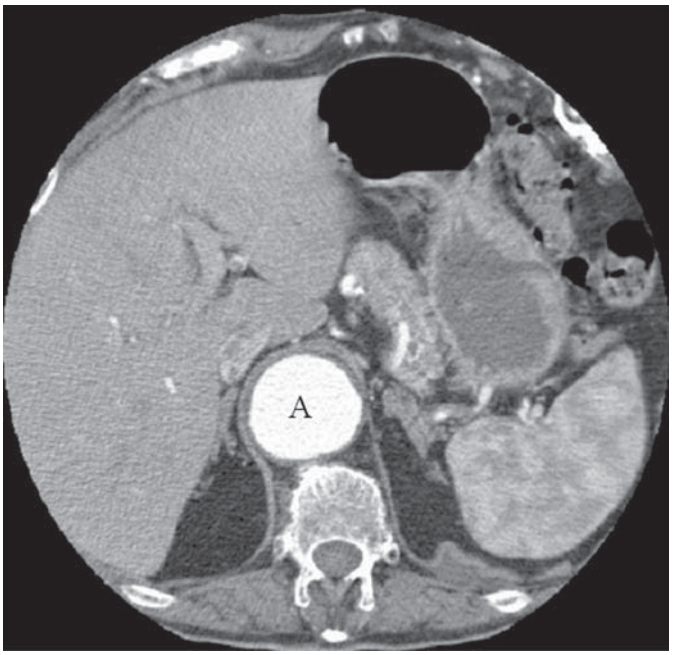

Fig. 1. A contrast-enhanced CT scan of the abdomen showing a 5.1 × 5.6 cm suprarenal abdominal aortic aneurysm (A).

reach the hospital. Thoracic aneurysms of less than 5.0 cm in size typically expand slowly and rarely rupture, but the rate of growth and risk of rupture increase significantly when the aneurysms are 6.0 cm or larger. Rupture of thoracic aneurysms carries an early mortality of 76% at 24 h *(7)*.

Treatment

Patients whose aneurysms are not at significant risk of rupture should be managed medically. The goal of medical therapy is to reduce the rate of aneurysm expansion and risk of future rupture. The use of β-blockers is the mainstay of this approach, but additional antihypertensive agents are often required. Both thoracic and abdominal aortic aneurysms should be followed closely with serial imaging studies (such as CT) to detect progressive enlargement over time that may indicate the need for surgical repair.

Size is the major indicator for repair of aortic aneurysms. Abdominal aortic aneurysms larger than 5.0 to 5.5 cm should be repaired in good operative candidates, and aneurysms greater than 4.0 cm is size should be monitored every 6 to 12 mo. Patients with ascending thoracic aortic aneurysms of greater than 5.5 cm in size should undergo surgical repair, while those with Marfan's syndrome should have repair when the aneurysm is ≥5.0 cm in size. Aneurysms of the descending thoracic aorta should be repaired when they are ≥6.0 cm in size.

Surgical repair consists of resection of the aneurysmal portion of the aorta and insertion of a synthetic prosthetic tube graft. When aneurysms involve aortic segments with branch arteries, such branches may need to be reimplanted into the graft. Similarly, when a dilated aortic root must be replaced in the repair of an ascending thoracic aortic aneurysm, the coronary arteries must be reimplanted.

A less-invasive alternative approach for repair of many abdominal and some descending thoracic aortic aneurysms is the placement of an expandable endovascular stent-graft inside the aneurysm via a percutaneous catheter-based approach. The device consists of a collapsible prosthetic tube graft that is inserted remotely (e.g., via the femoral artery), advanced transluminally across

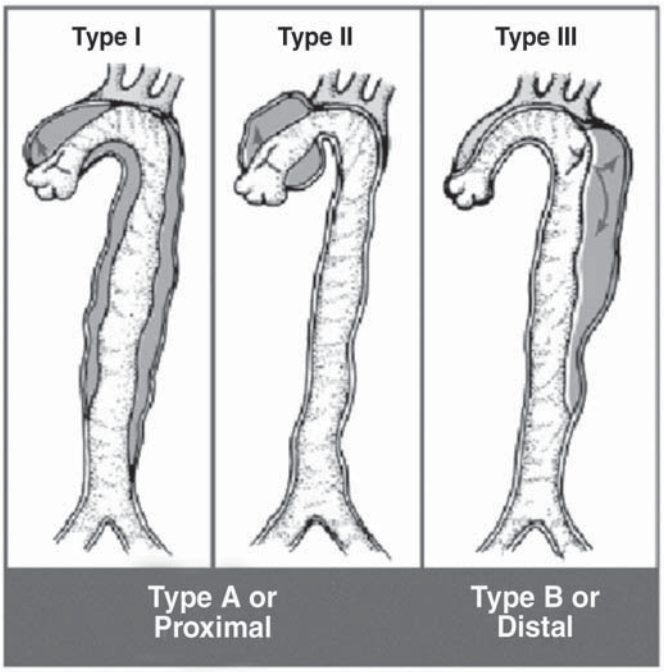

Fig. 2. Classification systems for aortic dissection. (From ref. *18*. Copyright 1998; with permission from Elsevier.)

the aneurysm under fluoroscopic guidance, and then secured at both its proximal and distal ends with an expandable stent attachment system. Once deployed the stent-graft serves to bridge the region of the aneurysm, thereby excluding it from the circulation while allowing aortic blood flow to continue distally through the prosthetic stent-graft lumen. However, only 30 to 60% of patients with abdominal aortic aneurysms—and fewer with descending thoracic aortic aneurysms—have aneurysm anatomy suitable for endovascular repair. The success rate of stent-graft implantation has been high, but in some instances patients are left with *endoleaks,* which means there is some residual blood flow into the aneurysm sac because of failure to completely exclude the aneurysm from the aortic circulation. Moreover, the long-term outcomes of endovascular repair versus conventional surgical repair are not yet known. Therefore, at present the use of stent-grafts for endovascular repair of abdominal aortic aneurysms has generally been limited to a subset of patients, typically older patients or those at high operative risk.

AORTIC DISSECTION

While far less common than aortic aneurysms, aortic dissection is a life-threatening condition with an early mortality as high as 1 to 2% per hour. However, with prompt early diagnosis and treatment, survival can be dramatically improved. The process of aortic dissection begins with a tear in the aortic intima that exposes a diseased medial layer to the systemic pressure of blood within the aortic lumen. The systolic force of aortic blood flow may cleave the media longitudinally into two layers, producing a blood-filled false lumen within the aortic wall that propagates distally (or sometimes retrograde) for a variable distance. The result is the presence of both a true and a false lumen separated by an intimal flap.

Aortic dissections are classified according to location, based on one of several systems as depicted in Fig. 2. Two thirds of aortic dissections are type A and the remainder are type B. The classification schemes are intended to distinguish those dissections that involve the ascending aorta from those that do not. Involvement of the ascending aorta carries a high risk of early aortic rupture and

death from cardiac tamponade, while those not involving the ascending aorta carry a much lower risk. Therefore prognosis and management differ according to the extent of aortic involvement.

Etiology

Disease of the aortic media, with degeneration of the medial collagen and elastin, is the most common predisposing factor for aortic dissection. Patients with Marfan's syndrome have classic cystic medial degeneration and are at particularly high risk of aortic dissection at a relatively young age. The peak incidence of aortic dissection in patients without Marfan's syndrome is in the sixth and seventh decades of life, with men affected twice as often as women *(8)*. A history of hypertension is present in the large majority of cases. A bicuspid aortic valve is a less common risk factor. Iatrogenic trauma from catheterization procedures or cardiac surgery may also cause aortic dissection.

Clinical Manifestations

The most common presenting symptom of aortic dissection is severe pain, occurring in 80% of cases *(8)*. The pain is typically retrosternal or interscapular, but it may also appear in the neck or throat, in the lower back, in the abdomen, or in the lower extremities, depending on the location of the aortic dissection. In fact, the pain may migrate as the dissection propagates distally. The pain is often of abrupt onset and at its most severe at the start. It is most often described as "sharp" or "stabbing," or alternatively as "tearing" or "ripping," in quality *(8)*. On the other hand, the description of the pain is sometimes relatively nonspecific. Less typical presentations include congestive heart failure (due to acute aortic insufficiency), syncope, stroke, or mesenteric ischemia.

Hypertension on presentation is a common finding, especially among most of those with type B aortic dissection. Hypotension may also occur, particularly among those with type A dissections, and suggests the presence of rupture into the pericardium (causing cardiac tamponade) or the presence of severe aortic insufficiency. It is essential to recognize the presence of *pseudohypotension*, which represents a falsely low measure of blood pressure due to involvement of the affected extremity's subclavian artery by the dissection. Pulse deficits are a common finding on physical examination when there is involvement of any of the subclavian, carotid, or femoral arteries. Acute aortic insufficiency may occur in up to one-half of those with type A dissection. While the presence of congestive heart failure or a widened pulse pressure should raise one's suspicion of acute aortic insufficiency, the diastolic murmur is often difficult to appreciate.

Involvement of branch arteries by the aortic dissection may produce a variety of vascular complications. Compromise of the ostium of a coronary artery—the right is most often involved—may cause myocardial ischemia or acute infarction. Involvement of the brachiocephalic or left common carotid artery may produce a stroke or coma. When a dissection extends into the abdominal aorta it may compromise flow to one or both renal arteries, producing acute renal failure with an exacerbation of hypertension. Another consequence may be mesenteric ischemia presenting as abdominal pain. Finally, an extensive dissection may compromise one of the common iliac arteries, causing femoral pulse deficits or lower-extremity ischemia.

The findings on chest roentgenography are typically nonspecific and rarely diagnostic. An enlarged mediastinal silhouette is present in 62% of cases *(8)*, and is often the factor that first prompts suspicion of aortic dissection among patients with chest pain. A small left pleural effusion (an exudate produced by the inflamed aortic wall) is commonly seen when there is involvement of the descending thoracic aorta. It should be emphasized that under no circumstances does a normal chest roentgenogram exclude the diagnosis of aortic dissection, as the chest roentgenogram is indeed normal in 12% of cases *(8)*.

Diagnosis

When the possibility of aortic dissection is being considered it is essential that one promptly confirms or excludes the diagnosis with an appropriate imaging study. Computed tomography,

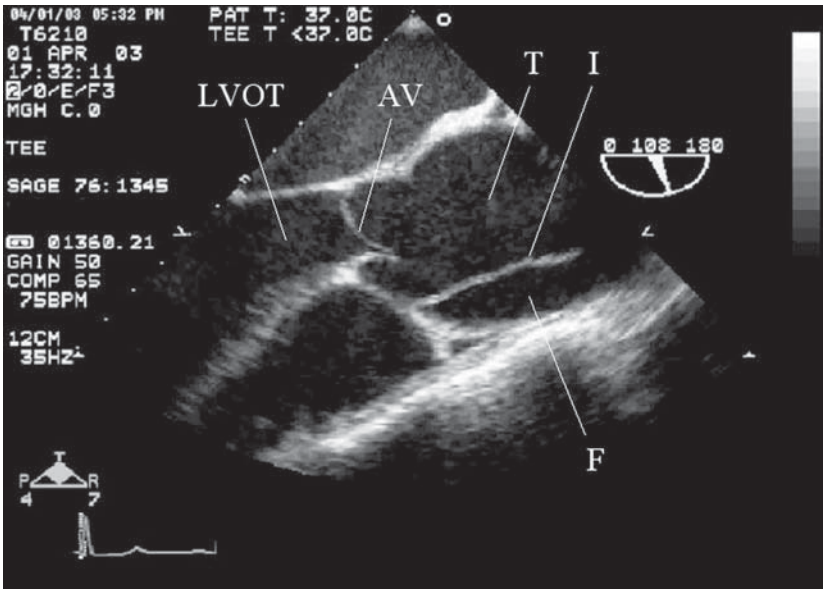

Fig. 3. A transesophageal echocardiogram of the ascending aorta in long-axis in a patient with a type A aortic dissection. The left ventricular outflow tract (LVOT) and aortic valve (AV) are on the left and the ascending aorta extends to the right. Within the aorta is an intimal flap (I) that originates at the level of the sinotubular junction. The true (T) and the false (F) lumens are separated by the intimal flap.

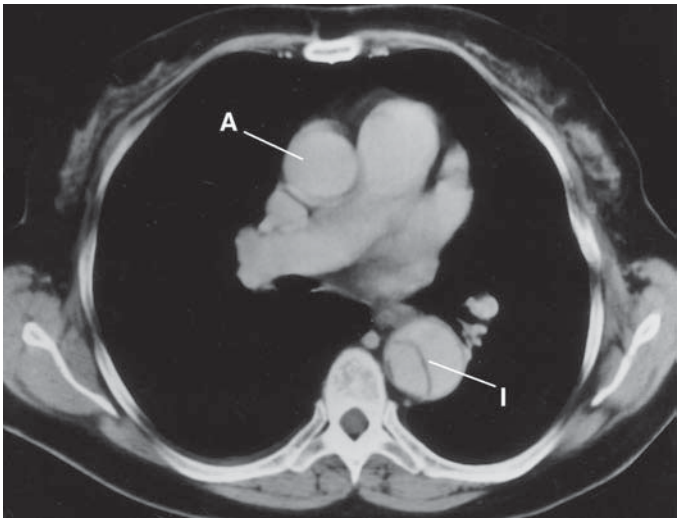

Fig. 4. A contrast-enhanced CT scan of the chest showing an intimal flap (I) separating the two lumens of the descending thoracic aorta in a type B aortic dissection. Note that there is no evidence of a dissection flap in the ascending aorta (A).

magnetic resonance imaging (MRI), transesophageal echocardiography (TEE), and aortography can accurately diagnose the presence of aortic dissection. In a tertiary care center when suspicion of aortic dissection is high, a transesophageal echocardiogram (Fig. 3) is often the study of choice as this examination provides sufficient detail to enable the surgeon to take the patient directly to the operating room for aortic repair if necessary *(1)*. When one's clinical suspicion is lower and the goal is to "rule out" aortic dissection, contrast-enhanced CT scanning (Fig. 4) is generally

preferred since it is entirely noninvasive. In community hospitals where TEE is not readily available contrast-enhanced CT scanning should be performed in all cases; if positive the patient can then be transferred promptly to a tertiary center for definitive treatment. When clinically significant branch artery involvement is suspected, aortography may be necessary to adequately define the arterial anatomy *(1)*. However, a good CT angiogram may be sufficient to provide the same anatomical detail.

TREATMENT

The goal of medical therapy is to halt any further progression of the aortic dissection and to reduce the risk of rupture. Whenever there is a suspicion of aortic dissection medical therapy should be instituted immediately while imaging studies are ordered, rather than waiting for the diagnosis to be confirmed. The primary goal of therapy is to reduce the systolic force of blood ejected from the heart into the aortic lumen by reducing *dP/dt*. The secondary goal is to reduce systolic blood pressure to 100 to 120 mmHg, or to the lowest level that maintains cerebral, cardiac, and renal perfusion. β-blockers are the first-line therapy to achieve these goals, and intravenous agents such as propranolol, metoprolol, or esmolol (ultra-short-acting) should be administered. Intravenous labetalol, which acts as both an α- and a β-blocker, may be particularly useful in aortic dissection for reducing both *dP/dt* and hypertension. Finally, after β-blockers have been initiated intravenous nitroprusside may be added to control hypertension more precisely on a minute-by-minute basis.

When a patient first presents with aortic dissection one must always document which arm has the higher blood pressure and then use only that arm for subsequent hemodynamic monitoring. Moreover, when patients present with significant hypotension, *pseudohypotension* should be carefully excluded. When true hypotension occurs due to hemopericardium and cardiac tamponade, patients should be treated with volume expansion and taken to surgery without delay, as early mortality in this setting is extremely high. Pericardiocentesis should be performed only as a last resort in this setting as it may precipitate hemodynamic collapse and death *(9)*.

After the diagnosis of aortic dissection has been confirmed, one must choose between medical and surgical therapy. Whenever an acute dissection involves the ascending aorta, surgical repair is indicated in order to minimize the risk of life-threatening complications such as rupture, cardiac tamponade, or severe aortic insufficiency. Conversely, if the dissection is confined to the descending aorta patients have been found to fare as well with medical therapy as with surgical repair *(10)*. However, when a type B dissection is associated with a serious complication, such as end-organ ischemia, surgery is indicated. Over the past decade there have been advances in endovascular techniques that can be used, in some cases, for nonsurgical management of acute vascular complications of aortic dissection.

Prognosis

Whether treated medically or surgically, patients with acute aortic dissection who survive the initial hospitalization generally do well thereafter. However, potential late complications include aneurysm formation (and possible rupture), recurrent dissection, and aortic insufficiency. Medications to reduce *dP/dt* and control hypertension can dramatically reduce the incidence of such late complications and should therefore be continued indefinitely *(11)*. β-blockers are the drug of choice in this setting, but typically additional medications will be needed to achieve the goal of a systolic blood pressure below 130 mmHg. Patients are at highest risk of complications during the first 2 yr after aortic dissection. Progressive aneurysm expansion typically occurs without symptoms, so patients must be followed closely with serial aortic imaging. This can be done using CT, MRI, or TEE, although most prefer CT. All patients should have a baseline imaging study prior to hospital discharge, with follow-up examinations performed at 6-mo intervals initially and then annually thereafter, provided that the anatomy is stable.

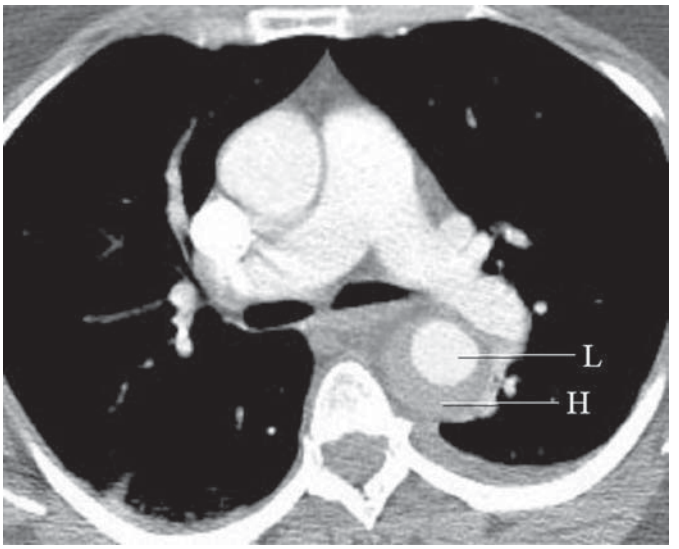

Fig. 5. Intramural hematoma of the aorta. A contrast-enhanced CT scan of the chest demonstrates crescentic thickening of the aortic wall consistent with an intramural hematoma (H). Note that there is no intimal flap within the lumen (L), nor does any contrast enter the hematoma. A small left pleural effusion is also present.

Intramural Hematoma of the Aorta

Intramural hematoma of the aorta is best defined as an atypical form of classic aortic dissection. Its etiology is not entirely certain, but it likely occurs when there is rupture of the vasa vasorum within the aortic media, resulting in a contained hemorrhage within the aortic wall. This hematoma may then propagate longitudinally along a variable length of the aorta, but since the intimal layer remains intact the hematoma does not communicate with the aortic lumen. While intramural hematoma of the aorta is clinically indistinguishable from aortic dissection, on cross-sectional imaging it appears as a crescentic thickening around the aortic wall (Fig. 5) rather than as true and false lumens separated by an intimal flap. It is important to note that the presence of an intramural hematoma may go undetected on aortography. The prognosis and management of intramural hematoma is essentially the same as that of classic aortic dissection *(12)*.

TAKAYASU'S ARTERITIS

Takayasu's arteritis is a chronic inflammatory disease of unknown etiology that involves the aorta and its branches. It typically affects young women, with a mean age of onset of 29 and women affected eight times as often as men *(13)*. It occurs more often in Asia and Africa than in Europe or North America. It typically has two stages. The first is an early stage in which there is active inflammation involving the aorta and its branches. This then progresses at a variable rate to a later sclerotic stage in which there is intimal hyperplasia, medial degeneration, and obliterative changes of the aorta and affected arteries. The majority of the resulting arterial lesions are stenotic, but aneurysms may occur as well. The aortic arch and brachiocephalic vessels are most often affected, but the abdominal aorta is also commonly involved. The pulmonary artery is occasionally involved. The disease may be diffuse or patchy, with affected areas separated by lengths of normal aorta.

Clinical Manifestations

Most patients present initially with symptoms of a systemic inflammatory process, such as fever, night sweats, arthralgia, and weight loss. However, there is often a delay of months to years between the onset of symptoms and the time the diagnosis is made. Indeed, at the time of diagnosis 90% of

patients have already entered the sclerotic phase and suffer symptoms of vascular insufficiency, typically with pain in the upper (or less often lower) extremities *(14)*. There will often be absent pulses and diminished blood pressures in the upper extremities, and there may be bruits over affected arteries. Significant hypertension due to renal artery involvement occurs in more than half of patients, but its presence may be difficult to recognize due to the diminished pulses. Aortic insufficiency may result from proximal aortic involvement. Congestive heart failure may result from either the hypertension or aortic insufficiency. Involvement of the coronary artery ostia may cause angina or myocardial infarction, and carotid artery involvement may cause cerebral ischemia or stroke. Abdominal angina may result from mesenteric artery compromise. The overall 15-yr survival for those diagnosed with Takayasu's arteritis is 83%, with the majority of deaths due to stroke, myocardial infarction, or congestive heart failure *(15)*. The survival rate for those with major complications of the disease is as low as 66%, while it may be as high as 96% for those without a major complication.

Diagnosis

During the acute phase, laboratory abnormalities include an elevated erythrocyte sedimentation rate, mild leukocytosis, anemia, and elevated immunoglobulin levels. The diagnosis is most accurately made, however, by the angiographic findings of stenosis of the aorta and stenosis or occlusion of its branch vessels, often with poststenotic dilation or associated aneurysms. Specific clinical criteria have been proposed for making a definitive diagnosis of Takayasu's arteritis *(16)*.

Treatment

The primary therapy for those in the acute inflammatory stage of Takayasu's arteritis is corticosteroids, which may be effective in improving the constitutional symptoms, lowering the erythrocyte sedimentation rate, and slowing disease progression *(17)*. When steroid therapy is ineffective cyclophosphamide or methotrexate may be added. Nevertheless, it remains unknown whether medical therapy actually reduces the risk of major complications or prolongs life. Surgery may be necessary to bypass or reconstruct segments of the aorta or branch arteries. Most commonly surgery is performed to bypass the coronary, carotid, or renal arteries, or to treat aortic insufficiency. More recently, as an alternative to surgery, balloon angioplasty has been used to successfully dilate stenotic lesions of either the aorta or renal arteries.

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