

Aortitis: Imaging Spectrum of the Infectious and Inflammatory Conditions of the Aorta¹

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CME FEATURE

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LEARNING OBJECTIVES FOR TEST 3

After reading this article and taking the test, the reader will be able to:

- List the common and less common forms of aortitis.
- Describe the pathophysiology of the different inflammatory diseases involving the aorta.
- Identify the different types of aortitis on the basis of their imaging findings.

TEACHING POINTS

See last page

Aortitis is a general term that refers to a broad category of infectious or noninfectious conditions in which there is abnormal inflammation of the aortic wall. These inflammatory conditions have different clinical and morphologic features and variable prognoses. The clinical manifestations are usually vague and nonspecific and may include pain, fever, vascular insufficiency, and elevated levels of acute phase reactants, as well as other systemic manifestations. As a result, aortitis is often overlooked during the initial work-up of patients with constitutional symptoms and systemic disorders. A multimodality imaging approach is often required for assessment of both the aortic wall and aortic lumen, as well as for surveillance of disease activity and treatment planning. Noninvasive cross-sectional imaging modalities such as magnetic resonance (MR) imaging, MR angiography, and computed tomographic angiography play a critical role in initial evaluation and further assessment of aortitis. Radiologists should be familiar with the clinical features and imaging findings of the different types of aortitis.

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Abbreviations: AIDS = acquired immunodeficiency syndrome, FDG = fluorine 18 fluorodeoxyglucose, GCA = giant cell arteritis, HIV = human immunodeficiency virus, SLE = systemic lupus erythematosus

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Introduction

Aortitis is a pathologic term for the presence of inflammatory changes of the aortic wall, regardless of the underlying cause. Aortic wall inflammation may be infectious or more commonly noninfectious. Noninfectious aortitis occurs in large-vessel vasculitides such as Takayasu arteritis and giant cell arteritis (GCA). It is also seen in other collagen vascular disorders such as rheumatoid arthritis and ankylosing spondylitis. Infectious aortitis may be secondary to tuberculosis, syphilis, or infection with *Salmonella* or other bacterial or viral pathogens.

The pattern of aortic involvement and imaging features help distinguish between noninfectious and infectious causes. Imaging is also useful for monitoring disease activity and in selected cases for guiding biopsy (eg, in temporal arteritis). Multiple imaging modalities have been used in the evaluation of inflammatory aortic diseases; regardless of the imaging technology employed, all of them finally converge in evaluation of the aortic lumen or of aortic wall changes.

Multidetector computed tomography (CT) has largely replaced conventional angiography. The invasive nature of the latter, the cumulative radiation dose from serial imaging, and the lack of information about the aortic wall have made conventional angiography fall out of favor for initial diagnosis and surveillance. CT angiography has excellent spatial resolution and is commonly the initial imaging study performed. Multidetector scanners allow multiplanar reformation and three-dimensional reconstruction, thus allowing evaluation of both arterial wall changes and abnormalities of the aortic lumen. For better imaging of the ascending aorta, electrocardiographically gated acquisition is recommended. Shortcomings include use of ionizing radiation and of iodinated contrast material in patients with impaired renal function.

Magnetic resonance (MR) imaging is recommended for serial imaging in patient follow-up and similarly allows excellent visualization of the arterial wall and vascular lumen, with multiplanar and three-dimensional reformation. Contraindications to MR imaging include the presence of pacemakers and metallic implants and inability to use gadolinium contrast material in patients with impaired renal function. Nuclear medicine

imaging with fluorine 18 fluorodeoxyglucose (FDG) positron emission tomography (PET) or gallium 67 (^{67}Ga) is helpful in assessment of inflammatory activity but limited in evaluation of morphologic changes. Ultrasonography (US) also has shortcomings, including limited anatomic coverage and inability to allow evaluation of disease activity. Therefore, US is recommended only in selected cases.

In this article, the pathophysiology, epidemiology, imaging manifestations, and differential diagnoses of the different types of aortitis and their complications are reviewed.

Classification

Classifications are often incomplete or controversial, but a simple classification of aortic inflammation into two broad categories, noninfectious and infectious aortitis, is useful for clinical purposes (Table). Noninfectious aortitis can be part of large-, medium-, or small-vessel vasculitis, since diseases in this category commonly affect other vessels and may be part of a systemic disorder (1).

Noninfectious Aortitis

The association between rheumatic diseases and aortic involvement is well known, but the prevalence of aortic involvement in the different rheumatic diseases is quite variable. Slobodin et al (2) recently reviewed the spectrum of rheumatic diseases that affect the aorta. **Rheumatic diseases with a high prevalence (>10%) of aortic involvement include Takayasu arteritis, GCA, long-standing ankylosing spondylitis, Cogan syndrome (interstitial keratitis, iritis, conjunctival or subconjunctival hemorrhage, fever, aortic insufficiency), and relapsing polychondritis.**

Rheumatic diseases in which aortic involvement is an uncommon but well-documented complication include rheumatoid arthritis, seronegative spondyloarthropathies, Behçet disease, and SLE. Rheumatic diseases with isolated case reports of aortic involvement or uncertain involvement include sarcoidosis, antineutrophil cytoplasmic antibody-associated aortitis (Wegener granulomatosis and polyarteritis nodosa), and juvenile rheumatoid arthritis. Symptoms of polymyalgia rheumatica have been reported in approximately 10% of patients with noninfectious ascending aortitis (3). Similarly, verrucous endocarditis (Libman-Sacks endocarditis) is a

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Classification of the Types of Aortitis

Noninfectious aortitis

- Large-vessel vasculitides
 - GCA
 - Takayasu arteritis
 - Rheumatoid arthritis
 - SLE
 - Ankylosing spondylitis
 - Reiter syndrome
- Medium- and small-vessel vasculitides
 - Wegener arteritis
 - Polyarteritis nodosa
 - Behçet disease
 - Relapsing polychondritis
- Idiopathic conditions
 - Idiopathic aortitis
 - Inflammatory aortic aneurysm
 - Idiopathic retroperitoneal fibrosis (periaortitis)
- Radiation-induced aortitis

Infectious aortitis

- Bacterial (eg, due to *Salmonella* or *Staphylococcus*)
- Luetic (syphilis)
- Mycobacterial (due to *Mycobacterium tuberculosis*)
- Viral (due to HIV infection or AIDS)

Source.—Reference 1.

Note.—AIDS = acquired immunodeficiency syndrome, HIV = human immunodeficiency virus, SLE = systemic lupus erythematosus.

well-known complication in cases of SLE and antiphospholipid syndrome (4).

Takayasu Arteritis

Also known as pulseless disease or Martorell syndrome, Takayasu arteritis is a necrotizing and obliterative segmental, large-vessel panarteritis of unknown cause with a predilection for young women (>80% of cases). Pathologic analysis demonstrates granulomas and inflammation of the arterial wall, with marked infiltration and proliferation of mononuclear cells in the adventitia and media; there is perivascular cuffing of the vasa vasorum in the early stage, followed by fibrosis and calcification in the late stage (5). This process leads to occlusion and narrowing of the aorta and its branches.

More common in females of Asian origin, Takayasu arteritis is thought to be secondary to an autoimmune process. It has also been associated with mycobacterial infection (tuberculosis), with occurrence after streptococcal infections, and with rheumatoid arthritis. According to the American College of Rheumatology, diagnosis is based on both clinical and imaging findings. Treatment is generally with high-dose glucocorticoids (6).

Takayasu arteritis is commonly divided into two clinical stages. In the early stage, also known as the systemic or prepulseless phase, clinical manifestations are mainly vague, systemic, and constitutional (fever, malaise, night sweats, weakness, pain), making diagnosis during this stage difficult. The late stage, also known as the occlusive or pulseless phase, is characterized by manifestations related to arterial stenosis, occlusion, or dilatation with clinical findings that depend on the vascular territory affected. During this late stage, constitutional symptoms are less prominent (7).

Aortic involvement in Takayasu arteritis is common, with the abdominal aorta affected most often, followed by the descending thoracic aorta and aortic arch (8). Aortic and branch vessel disease may manifest as stenosis or luminal narrowing, or less commonly as aneurysmal dilatation that ensues after inflammation destroys the media. Rapid expansion of aortic aneurysms and aortic rupture have been reported (8). Aneurysmal dilatation and rupture of the affected aorta are not rare, with a prevalence of 45% and 33%, respectively (8).

Historically, different imaging modalities have been used for diagnosis of Takayasu arteritis. Digital subtraction angiography was the traditional procedure of choice, with findings ranging from mild vessel stenosis to frank occlusion. Disadvantages of this technique include a higher radiation dose, greater contrast material burden, and difficulty in performance in cases of long-segment stenosis or when there is heavy arterial calcification. In addition, digital subtraction angiography is not as accurate as cross-sectional imaging in demonstrating wall architectural changes, and sometimes it is impossible to differentiate vascular obliteration caused by inflammation from stenosis resulting from chronic

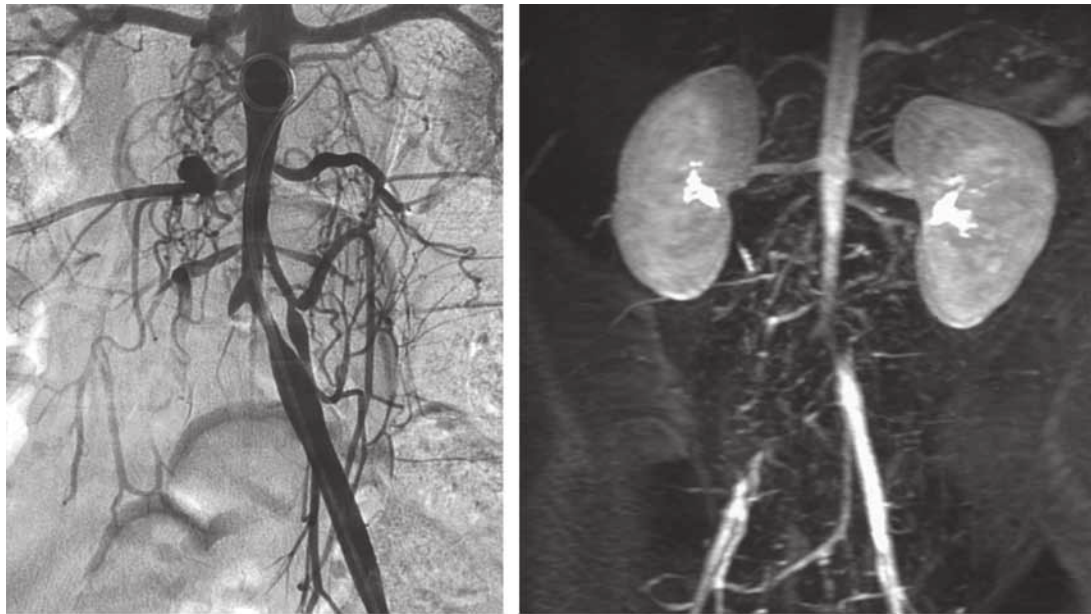


Figure 1. Takayasu arteritis in a 13-year-old girl. **(a)** Image from conventional angiography shows concentric infrarenal narrowing of the aorta and left common iliac artery and occlusion of the proximal right common iliac artery. **(b)** Image from MR angiography clearly shows the morphology of the affected vessels distal to the obstructive lesion. There is narrowing of the distal abdominal aorta and complete obstruction of the iliac arteries.

fibrosis (Fig 1). Transesophageal echocardiography and intravascular US are important tools that provide high-resolution images of subtle changes in aortic segments.

CT angiography is essential in the work-up of these patients because it allows early diagnosis. Findings include concentric thickening of the vessel wall, thrombosis, stenosis, and occlusion. Other associated findings include vessel ectasia, aneurysms, and ulcers (Fig 2). Aortic wall thickening, which has been described as a “double ring” appearance at contrast-enhanced CT, is the typical finding in the early stage, with a poorly enhanced internal ring (the swollen intima) and an enhancing outer ring (the inflamed media and adventitia) (9).

Arterial wall calcification can develop in chronic cases, typically after 5 or more years of inflammatory involvement. Aortic wall calcification is typically linear and usually spares the ascending aorta. Radiation and contrast material administration remain an issue with CT; however, the doses are lower than those of conventional angiography. CT angiography has high sensitivity and specificity (95% and 100%, respectively) for demon-

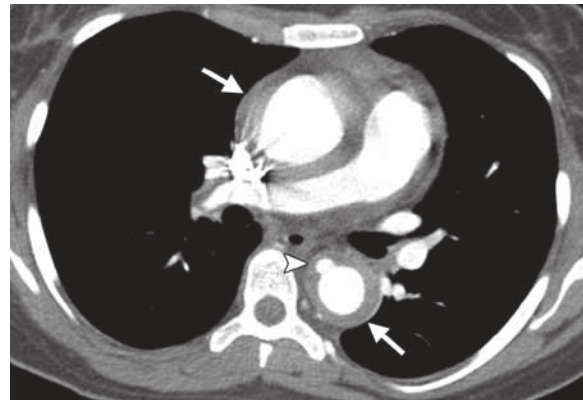


Figure 2. Takayasu arteritis in a 22-year-old woman. Contrast material-enhanced CT image shows extensive thickening of the aortic wall that involves the entire thoracic aorta (arrows). There is an area of ulceration in the anteromedial wall of the descending aorta (arrowhead).

strating the abnormalities of the affected vessels and is better than conventional angiography in demonstrating wall thickening, calcification, and mural thrombi (10).

MR imaging has become of considerable significance because it demonstrates early wall thickening even before luminal narrowing occurs

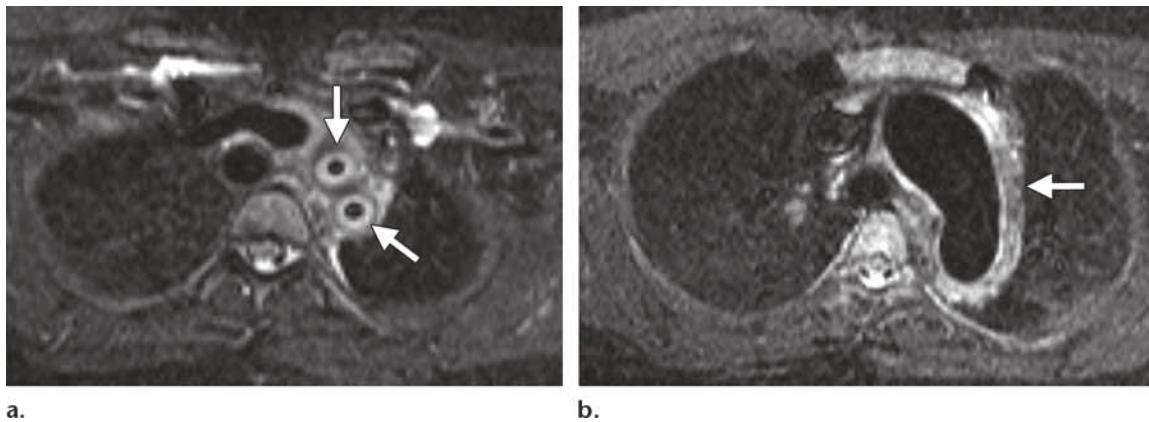


Figure 3. Takayasu arteritis in a 20-year-old woman. Short inversion time inversion-recovery MR images show abnormal arterial wall thickening and edema involving the arch vessels (arrows in **a**) and aortic wall (arrow in **b**), findings similar to the double ring appearance seen at CT. The wall thickening and edema are better seen in the left carotid and left subclavian arteries.

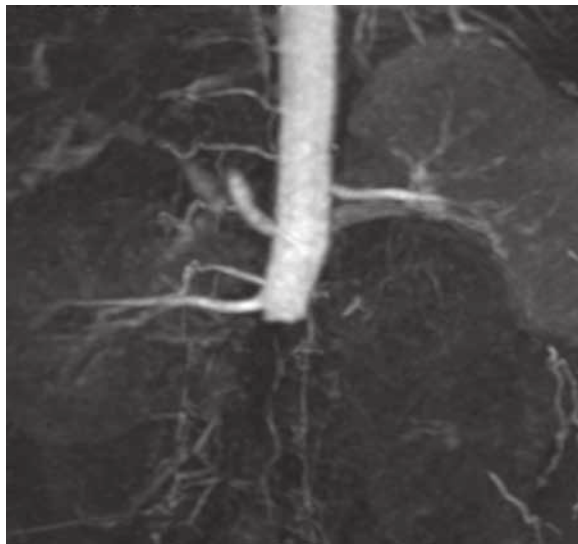


Figure 4. Aortic interruption in a 27-year-old man with Takayasu arteritis. Image from MR angiography shows complete occlusion of the mid abdominal aorta, occlusion of the left renal artery, and multiple retroperitoneal collateral vessels.

(6,11). Gadolinium-enhanced fat-suppressed T1-weighted images show a thickened, enhancing arterial wall. High signal intensity on T2-weighted images represents mural edema (Fig 3). Similarly to CT angiography, MR angiography may show stenoses at multiple levels, mural thrombi, thickening of aortic valve cusps, and pericardial effusions (5–7,12) (Fig 4). MR imaging is superior to conventional angiography because it allows early detection of subtle changes in the aortic wall as well as disease activity. It has been proposed that for early diagnosis of Takayasu arteritis, there

should be crescentic or ringlike aortic thickening of more than 3 mm at contrast-enhanced MR imaging or CT (11).

Giant Cell Arteritis

GCA is a chronic vasculitis that affects large and medium-sized vessels and usually involves the superficial cranial arteries. It is being increasingly recognized as a systemic vascular disease not limited to the cranial arteries. Aortic involvement occurs in 15% of GCA patients; this is the most common form of aortitis in North America, accounting for more than 75% of cases (13).

The prevalence rises with increasing age: GCA is rarely diagnosed in patients less than 50 years old. The prevalence is also higher among whites than in other racial groups. The pathogenesis is secondary to a systemic granulomatous vasculitis.

GCA is closely related to polymyalgia rheumatica. The acute stage is characterized by disruption of the internal elastic lamina and an inflammatory cellular infiltrate with multinucleated giant cells and lymphocytes. In the chronic stage, there is progressive fibrosis of the vessel wall.

Vascular inflammation may be widespread, most commonly involving the external carotid branches, especially the superior temporal artery, and also involving the vertebral arteries, coronary arteries, and mesenteric arteries as well as the aorta and its branches (14). Widespread involvement has been associated with significantly reduced 5-year survival (15). Aortic involvement usually manifests as annuloaortic ectasia or as an ascending aortic aneurysm that can extend

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into the aortic arch (16–18). Aortic involvement can also manifest as acute dissection, aortic valve insufficiency, or abdominal aortic aneurysm (Fig 5). Thoracic aortic aneurysms are usually a late complication of the disease (19,20).

Diagnostic modalities useful in diagnosis of extracranial GCA include chest radiography, CT angiography, and MR angiography. CT and in particular MR angiography are able to demonstrate vessel wall edema, which reflects activity of the disease, as well as smooth tapering proximal and distal to the lesion (21,22) (Figs 6, 7). FDG PET has been shown to be sensitive for extracranial vasculitis but not for intracranial vasculitis on account of its poor spatial resolution (23). FDG PET reveals abnormal uptake in the aortic arch or large thoracic arteries in more than one-half of affected patients (sensitivity = 56%, specificity = 98%, positive predictive value = 93%, negative predictive value = 80%) (24). CT angiography is also useful and reveals luminal changes such as stenosis, occlusion, dilatation, aneurysm formation, calcification, and mural thrombi (25).

Other Rheumatic Diseases Associated with Aortitis

Ankylosing Spondylitis.—Ankylosing spondylitis was the first rheumatic disease found to be associated with aortitis. Aortic root disease and aortic valve disease are common in patients with ankylosing spondylitis (80% of cases), with aortic wall thickening present in about 60% of affected patients (26). Aortic valve thickening and nodularity, associated with valvular insufficiency, are also common manifestations of ankylosing spondylitis and are associated with significant morbidity (heart failure, stroke) and death. The frequency of aortic insufficiency and regurgitation parallels that of the duration of disease.

Relapsing Polychondritis.—Relapsing polychondritis is a debilitating multisystem inflammatory and autoimmune disorder, which is characterized by recurrent episodes of cartilage inflammation and degeneration that may affect other connective tissue such as the elastic element of cardiac valves and the aorta. It involves proteoglycan-rich structures such as the aorta and may manifest as aortic root dilatation and aortitis. The prevalence of cardiac involvement is 15%–45% and includes aortic dilatation, with secondary regurgitation, mitral regurgitation, and aortitis. Histopathologic studies of the aorta reveal cystic degeneration of collagen, destruction of elastic fiber,

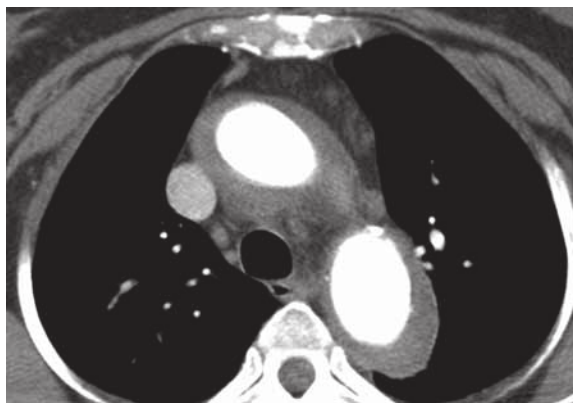


Figure 5. GCA in a 72-year-old woman. Contrast-enhanced CT image shows diffuse abnormal thickening of the thoracic aorta. The thickening was due to an extensive intramural hematoma.

lymphocytic infiltration, and decreased content of acid mucopolysaccharides (27).

During the active aortitis phase, there is an increase in the vasa vasorum with endothelial swelling, with the vasa vasorum extending through the entire aortic wall thickness (28). Aortic wall calcification and ossification with nodular wall formation have also been described (29). Aortic wall involvement results in aneurysm formation in the thoracic and abdominal aorta (5% of cases) and obliterans vasculitis in medium-sized and large arteries (30,31).

Rheumatoid Arthritis.—Rheumatoid arthritis may affect the heart, aortic valve, and great vessels. In an autopsy study of 188 patients with rheumatoid arthritis, a 5.3% prevalence of aortitis and 1.6% prevalence of aneurysm formation were reported (32). The aortic valve and annulus may also be affected with granulomatous or non-granulomatous inflammation, leaflet thickening, and secondary regurgitation (33).

Aortitis in rheumatoid arthritis is rare and may well be associated with rheumatoid vasculitis in other vessels. If there is involvement of the coronary ostia, this may lead to myocardial ischemia (2). Multiple aortic aneurysms as well as spontaneous rupture in patients with rheumatoid arthritis receiving long-term steroid therapy have also been reported (34,35).

Systemic Lupus Erythematosus.—SLE is known to affect the cardiovascular system mostly as serositis of the pericardium. Aortitis is uncommon in patients with SLE, but it has been described and is associated with aortic dissection, aneurysm, and thrombus. Recently, a case of recurrent aortitis was diagnosed with FDG PET, which showed

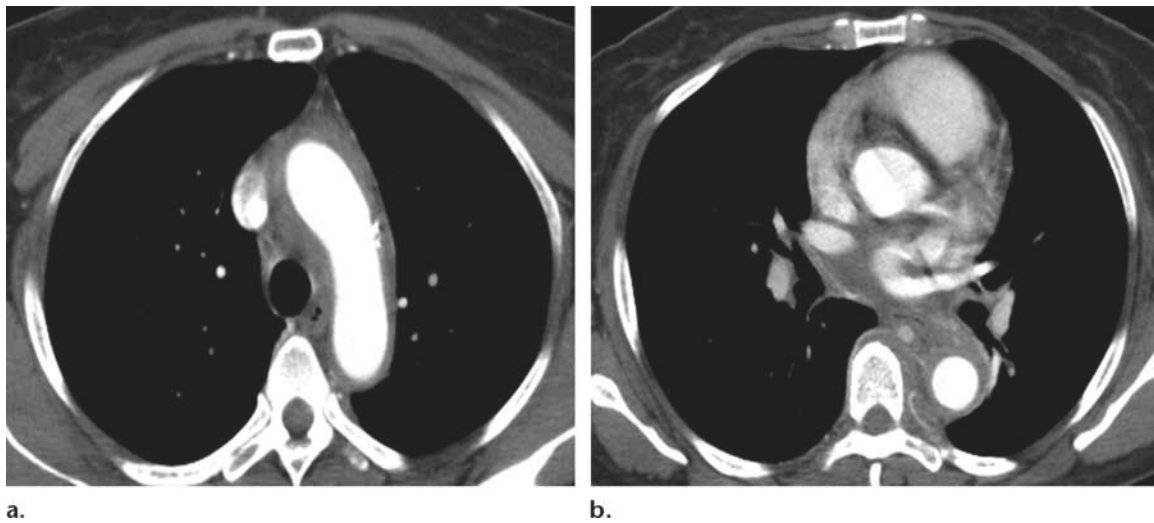


Figure 6. GCA in a 65-year-old woman. Contrast-enhanced CT images (**a** obtained at a higher level than **b**) show diffuse wall thickening of the thoracic aorta.

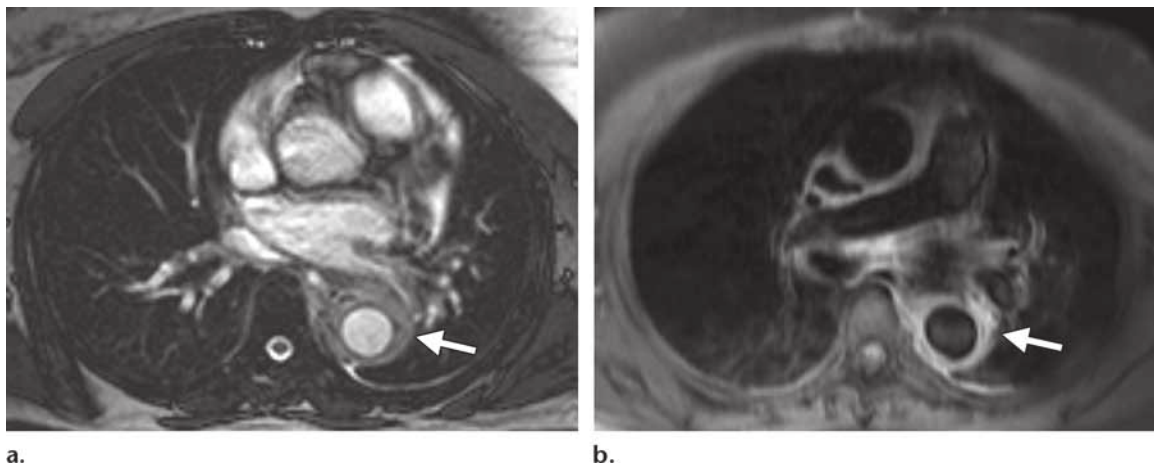


Figure 7. GCA in a 67-year-old woman. **(a)** Gradient-echo MR image, obtained at the level of the aortic root, shows abnormal thickening of the middle one-third of the descending aorta (arrow). **(b)** Fat-suppressed T2-weighted MR image shows increased signal intensity in the aortic wall (arrow), a finding consistent with edema.

a high metabolic rate in the ascending aorta; the diagnosis was confirmed with MR imaging, which showed wall thickening and high signal intensity on T2-weighted images (36).

Lupus-related aortic aneurysms tend to manifest at a younger age and are characterized by destruction of the medial elastic lamina. Prolonged steroid therapy and vasculitis-related aortic wall damage have been proposed as contributing factors for the development of aortic aneurysms in these patients (37).

Cogan Syndrome.—Cogan syndrome is a rare autoimmune disease with systemic manifestations, including ocular, inner ear, and vascular inflammation. Manifestations include eye redness, photophobia, or eye pain from interstitial keratitis;

audiovestibular manifestations similar to those in Ménière syndrome; nerve deafness; and various cardiovascular manifestations including aortitis complicated by aortic insufficiency and necrotizing vasculitis, which may induce coronary, iliac, or renal artery stenosis (38).

Young white adults are more commonly affected. Aortitis and valvulitis with aortic insufficiency may be seen in almost 10% of patients and warrant early imaging if there is suspicion of aortic involvement. Histologic analysis of the aortic wall reveals inflammation with prominent lymphocytic infiltration, destruction of medial elastic tissue, fibrosis, and neovascularization, which finally result in aneurysm formation (39).

Figure 8. Aortic aneurysm secondary to Behçet disease in a 48-year-old man. Contrast-enhanced CT images (**a** obtained at a higher level than **b**) show an irregular aneurysm in the abdominal aorta. Extensive soft tissue effaces the anatomic planes between the aorta and the psoas muscles, with vertebral body remodeling. (Case courtesy of Naim Ceylan, MD, Ege University School of Medicine, Bornova/Izmir, Turkey.)

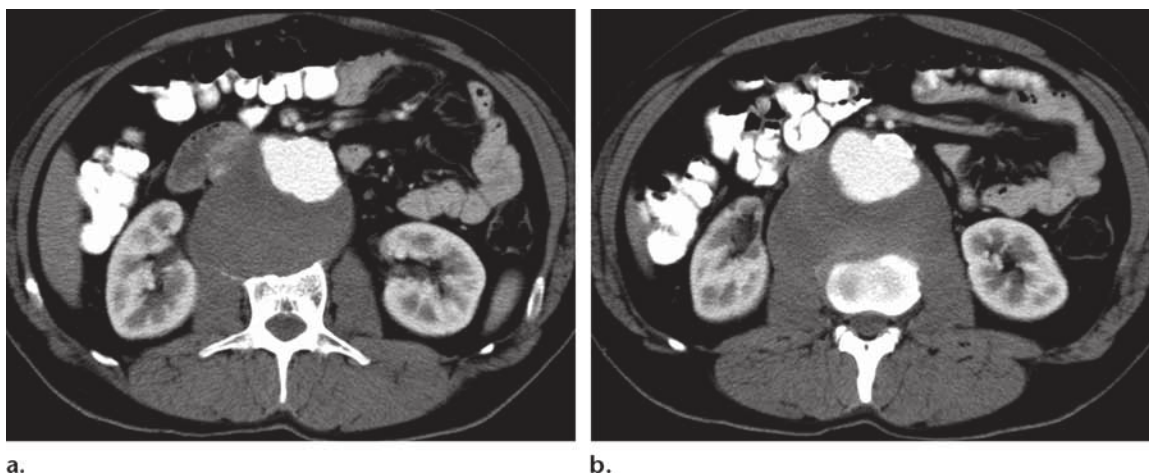
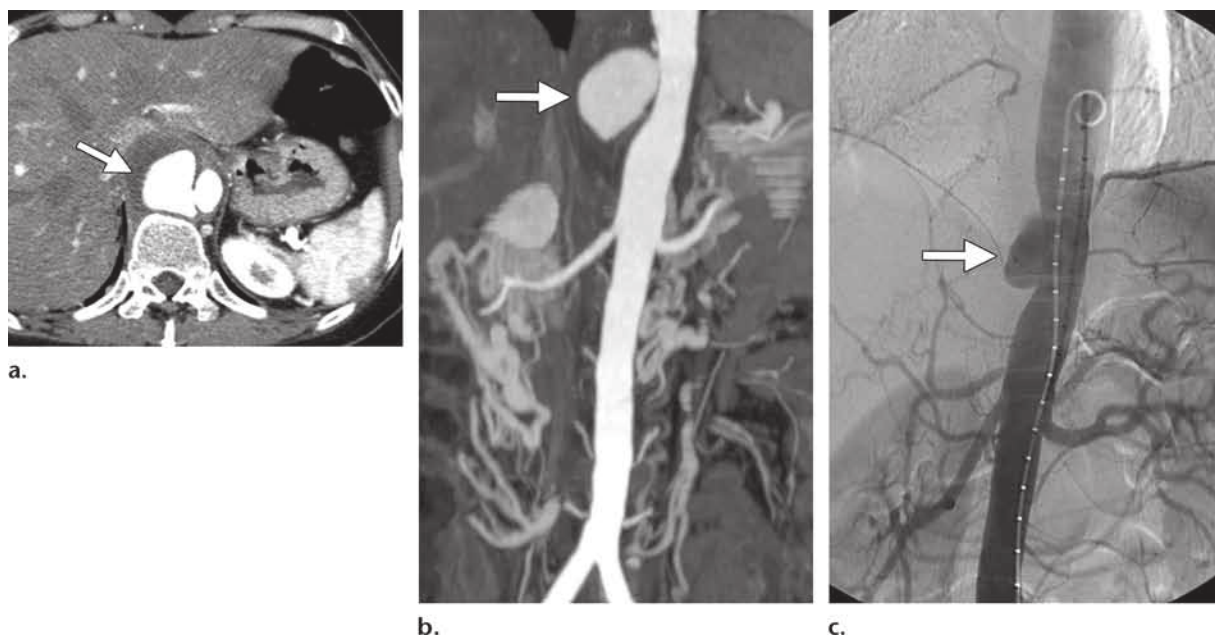


Figure 9. Aortic pseudoaneurysm secondary to Behçet disease in a 51-year-old woman. (**a, b**) Axial (**a**) and coronal (**b**) contrast-enhanced CT images show a saccular pseudoaneurysm (arrow) that arises from the lateral wall of the aorta, resulting in extrinsic compression of the aortic lumen. (**c**) Image from conventional angiography shows the pseudoaneurysm (arrow) and the luminal narrowing. (Case courtesy of Jae Hyung Park, MD, Seoul National University College of Medicine, Seoul, Korea.)



Behçet Disease.—Behçet disease is a rare, chronic and relapsing, inflammatory systemic disorder of unknown cause that is characterized by mucocutaneous ulcers; inflammatory vascular involvement is described in 5%–40% of affected patients (40). Wall-enhancing saccular pseudoaneurysms may develop in the abdominal and thoracic aorta (Figs 8, 9). In one-fifth of affected patients, multiple pseudoaneurysms develop in large vessels

such as the aorta and the iliac, femoral, popliteal, and subclavian arteries (41).

Idiopathic Aortitis

An idiopathic form of aortitis that is typically asymptomatic and is diagnosed only by the pathologist after surgery for aortic aneurysms has been described. Of 1,204 aortic surgical and pathologic specimens studied over a 20-year period, 4.3% were clinically and pathologically classified as representing idiopathic aortitis. Two-thirds of patients with this form of idiopathic aortitis were women;

Figure 10. Idiopathic aortitis in a 57-year-old man. Contrast-enhanced CT images (**a** obtained at a higher level than **b**) show abnormal concentric wall thickening that involves only the ascending aorta (arrow), with a normal-caliber lumen.

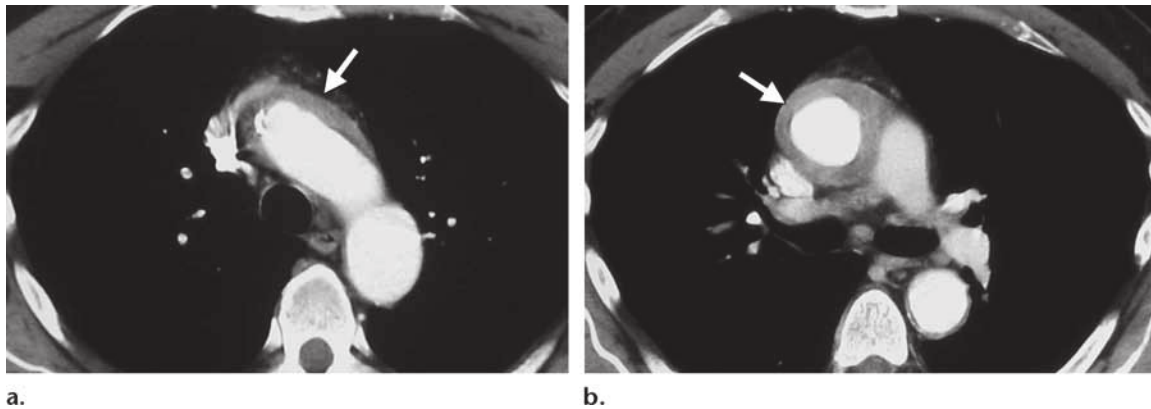
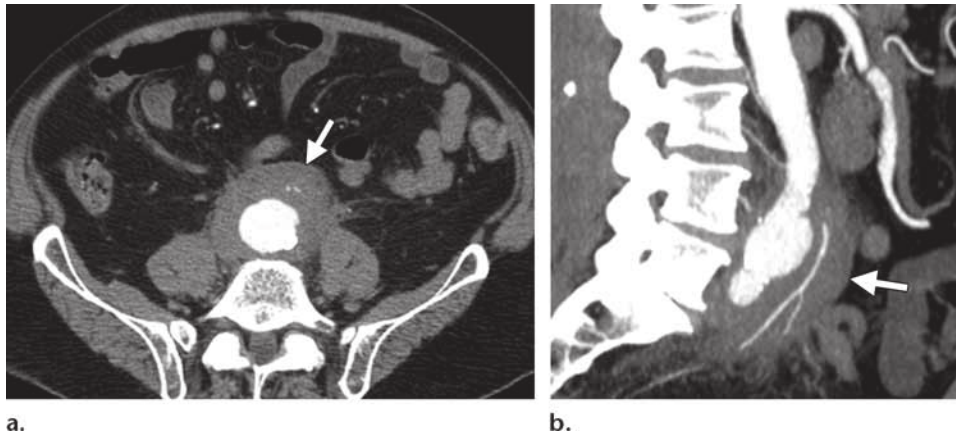


Figure 11. Inflammatory aortic aneurysm in a 65-year-old man. Axial (**a**) and sagittal (**b**) contrast-enhanced CT images show a significant amount of soft tissue (arrow) surrounding a fusiform dilatation of the distal aorta. The inflammatory soft tissue mainly involves the anterior and lateral aortic walls, sparing the posterior wall.



in 96% of patients in whom aortitis was associated with an aortic aneurysm, aortitis was present only in the thoracic aorta (42) (Fig 10).

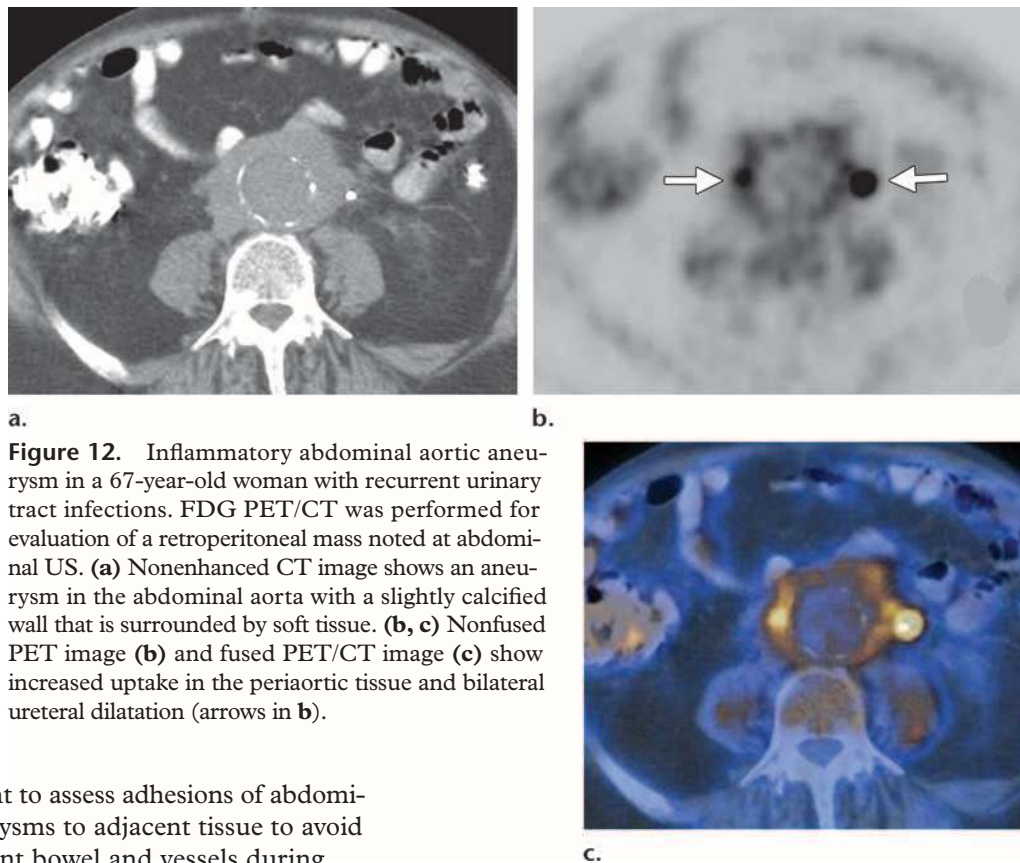
In addition, among the surgical specimens of 383 thoracic aortic aneurysms, 12% had idiopathic inflammatory features in the aortic wall. During a mean follow-up of 41 months, new aneurysms developed in one-fourth of the patients who did not receive glucocorticoid therapy (42).

Idiopathic Inflammatory Aortic Aneurysm

Idiopathic inflammatory aneurysms differ from atherosclerotic aneurysms because of the presence of dense perianeurysmal fibrosis and a thickened aortic wall. The prevalence has been reported as 5%–25% of all abdominal aortic aneurysms. Inflammatory aneurysms of the ascending aorta and aortic arch are much less frequent, with only a handful of cases reported in the literature, many of them with concomitant inflammatory aneurysms in the abdominal aorta (43).

Aneurysms with periaortic fibrosis are a distinct entity because of the higher mortality (23%) during surgical repair. Complications related to retroperitoneal extension of the inflammatory process are seen in one-third of affected patients and include secondary ureteral involvement with resultant hydronephrosis, aortic–sigmoid colon fistula with bleeding, and secondary bacterial infection (eg, with *Salmonella*).

CT shows a hypoattenuating mass with periaortic wall thickening that spares the posterior wall. After intravenous administration of contrast material, rapid luminal opacification is followed by delayed enhancement of the soft-tissue component. A characteristic feature of inflammatory abdominal aortic aneurysms is that the thickening of the aortic wall typically affects the anterior wall (Fig 11). Contrast-enhanced CT has been reported to have 83% sensitivity and almost 100% specificity for this diagnosis (44).



a.
Figure 12. Inflammatory abdominal aortic aneurysm in a 67-year-old woman with recurrent urinary tract infections. FDG PET/CT was performed for evaluation of a retroperitoneal mass noted at abdominal US. **(a)** Nonenhanced CT image shows an aneurysm in the abdominal aorta with a slightly calcified wall that is surrounded by soft tissue. **(b, c)** Nonfused PET image **(b)** and fused PET/CT image **(c)** show increased uptake in the periaortic tissue and bilateral ureteral dilatation (arrows in **b**).

It is important to assess adhesions of abdominal aortic aneurysms to adjacent tissue to avoid injury to adjacent bowel and vessels during surgery and for planning a transperitoneal versus retroperitoneal approach for repair. FDG PET and MR imaging play a significant role in preoperative assessment and in determining the extent (suprarenal vs infrarenal) of the aneurysm. FDG PET also helps in the identification and grading of adhesions and the extent of inflammation (Fig 12). MR imaging demonstrates periaortic inflammation, adventitial fibrosis, and turbulence in intraluminal flow (45).

US shows a hypoechoic mass that represents the inflammatory process with surrounding echogenic tissue and thickening of the aortic wall.

Chronic Periaortitis

Retroperitoneal fibrosis, also known as sclerosing retroperitoneal granuloma, chronic periaortitis, and Ormond disease, is characterized by mass-like fibrosis mainly in the retroperitoneum that extends to adjacent viscera, including the inferior vena cava and ureters. Under microscopic evaluation, this tissue is composed mainly of fibroblasts along with inflammatory cells (lymphocytes and macrophages) and vascular endothelial cells. The initial fibrosis tends to initiate near the aorta, about the aortic bifurcation extending through

the retroperitoneum (Fig 13). Cases in which periaortitis was confined to the thoracic aorta have been reported (46).

Retroperitoneal fibrosis is commonly idiopathic but has been associated with certain drugs, malignant disease, and systemic disorders. An autoimmune cause is suspected, and an exaggerated inflammatory response to a component of the atheroma seen in advanced atherosclerotic disease has been postulated (47,48).

Typical imaging findings include a retroperitoneal and paraspinal mass of soft-tissue attenuation that is isoattenuating to adjacent muscles at CT, with a variable degree of extension to adjacent organs. As a rule, the mass does not displace the aorta and inferior vena cava anteriorly from the spine. Increased uptake at ^{67}Ga scanning and FDG PET is particularly common during the active inflammatory stage (49).

Radiation-induced Aortitis

Radiation-induced vascular injury may manifest early after radiation therapy but most commonly develops more than 10 years after exposure to a usually high dose of therapeutic radiation. Affected elastic arteries may develop thrombosis, pseudoaneurysm, rupture, stenosis, and ac-

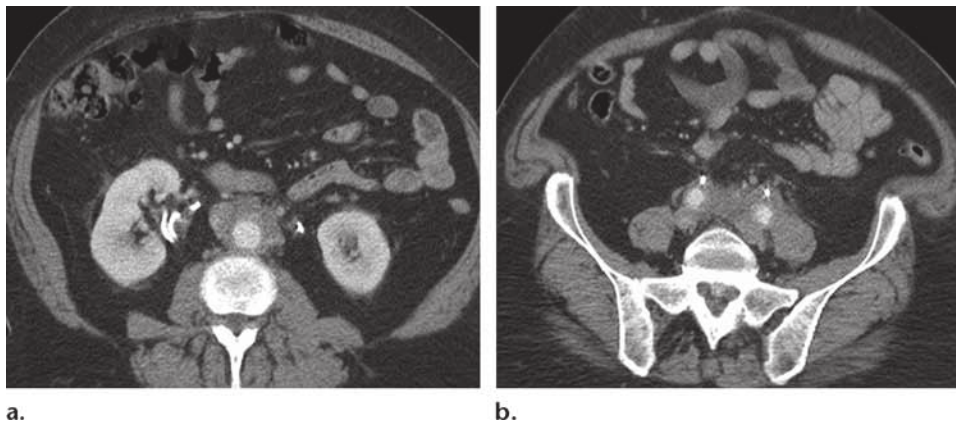


Figure 13. Retroperitoneal fibrosis in a 61-year-old woman with hydronephrosis. Axial CT images show low-attenuation soft tissue surrounding the aorta (**a**) and iliac vessels (**b**). Bilateral ureteral stents have been placed for obstructive uropathy.

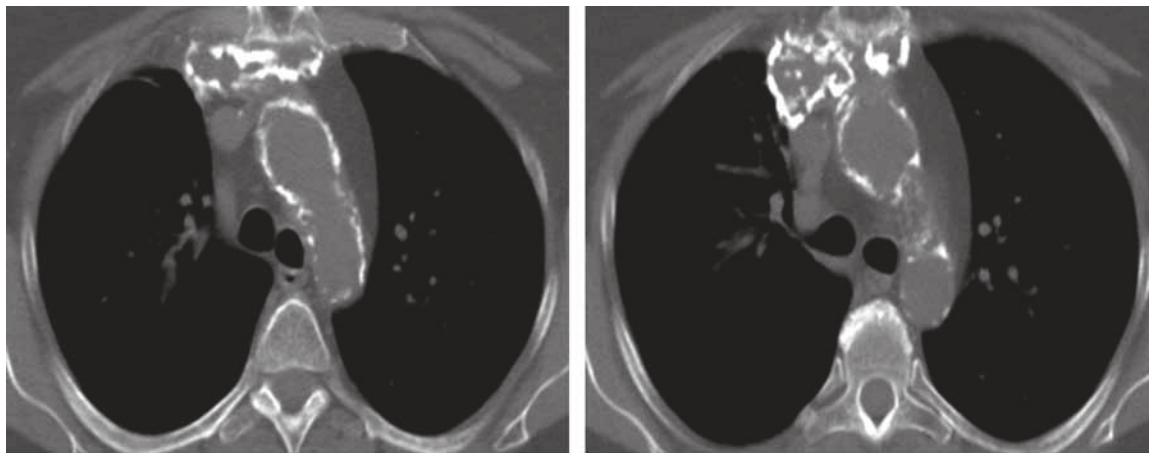


Figure 14. Radiation-induced aortitis in a 50-year-old woman with a remote history of radiation therapy for mediastinal lymphoma. Nonenhanced CT images (displayed from superior [**a**] to inferior [**b**]) show a densely calcified residual mass in the anterior mediastinum, as well as significant calcification of the aortic wall within the radiation field. The aortic wall distal to the radiation field is free of calcification (**c**).



celerated wall calcification. As a rule, radiation-induced arteritis is confined to the irradiated field (50,51). Calcification of the ascending aorta and proximal coronary arteries in young adults may occur as a late complication of mediastinal radiation therapy for Hodgkin disease (52) (Fig 14).

Infectious Aortitis

Infectious aortitis is an infectious and inflammatory process of the aortic wall induced by microorganisms. In the preantibiotic era, it was most likely a complication of bacterial endocarditis secondary to *Streptococcus pyogenes*, *Streptococcus pneumoniae*, and *Staphylococcus*. **The aorta is normally very resistant to infection; however, an abnormal aortic wall, like that associated with atherosclerotic disease, preexisting aneurysm, cystic medial necrosis, diabetes, vascular malformation, medical devices, or surgery, makes it more susceptible to infection (53,54).**

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Nowadays, the most common pathogens, which account for almost 40% of infections, include *Staphylococcus aureus* and *Salmonella* species. Other pathogens involved include *Treponema pallidum*, *M tuberculosis*, and other bacteria such as *Listeria*, *Bacteroides fragilis*, *Clostridium septicum*, and *Campylobacter jejuni* (55,56). Mechanisms of infection include hematogenous spread, contiguous seeding from adjacent infection, and traumatic or iatrogenic inoculation. Infected aortic or mycotic aneurysms are part of the spectrum of infectious aortitis (57). Men are affected more often than women, with most cases seen in adults after the 5th decade of life.

Contrast-enhanced CT is usually the imaging modality of choice. Imaging manifestations of infectious aortitis include aortic wall thickening, periaortic fluid or soft-tissue accumulation, rapidly progressing saccular aneurysm or pseudoaneurysm, and occasionally air in the aortic wall (58) (Fig 15).

Syphilitic Aortitis

Syphilis is a sexually transmitted chronic systemic infection caused by the spirochete *T pallidum*. It is characterized by episodes of activity with interspersed episodes of latency. The clinical findings depend on the duration of infection and are divided into four stages: primary, secondary, early or late latent, and tertiary. Tertiary syphilis is defined as neurosyphilis, gummas, and cardiovascular involvement. Cardiovascular manifestations are usually evident 5–30 years after the primary infection and are secondary to endarteritis obliterans of the vasa vasorum.

Syphilitic (luetic) heart disease has been divided into syphilitic aortitis, syphilitic aortic aneurysm, syphilitic aortic valvulitis with aortic regurgitation, and syphilitic coronary ostial stenosis. Chronic aortic inflammation results in fibrosis and wrinkling of the intima (“tree barking”), which ultimately leads to aneurysm formation. Calcification of the ascending aorta is typical but uncommon. Luetic aortitis involves the ascending thoracic aorta in 60% of cases and the aortic arch in 30% (59) (Fig 16). Early sternal erosion associated with luetic aneurysms of the ascending aorta has been noted; the erosion mainly affects the right side of the manubrium, as well as the medial end of the right clavicle (60).



Figure 15. Infectious aortitis due to *C septicum* in a 74-year-old man with adenocarcinoma of the ascending colon. Nonenhanced CT image shows circumferential mural gas in and around the abdominal aorta (arrow).

Diagnosis is based on sensitive nontreponemal serologic tests (rapid plasma reagin test, Venereal Disease Research Laboratory test) and specific treponemal serologic tests (fluorescent treponemal antibody–absorption test, microhemagglutination–*T pallidum* test). The preferred antibiotic therapy is penicillin.

Tuberculous Aortitis

The prevalence of tuberculous aortitis is expected to increase with the rise in *M tuberculosis* infection due to the rise in co-infection with HIV and multiple drug-resistant tuberculosis. Tuberculous aortitis usually involves the distal aortic arch and descending aorta. It is generally due to direct extension from contiguous mediastinal lymph nodes, empyema, or pericarditis or to hematogenous or lymphatic spread of distant infection. It occurs in less than 1% of patients with latent tuberculosis, with mortality rates as high as 60% (61).

Recently, an association between vertebral tuberculosis and tubercular thoracoabdominal pseudoaneurysm has been reported (62). Pathologic samples demonstrate caseous granulomas, multinucleated giant cells, and epithelioid cells. The risk of perforation increases when a cold abscess is present (63). In a study of 39 cases of tuberculous mycotic aneurysms by Long et al (64), 75% of the aneurysms appeared to originate from aortic wall erosion by a contiguous focus (Fig 17); most of the aneurysms (90%) were saccular and false, and disseminated tuberculosis was present in almost one-half of cases.

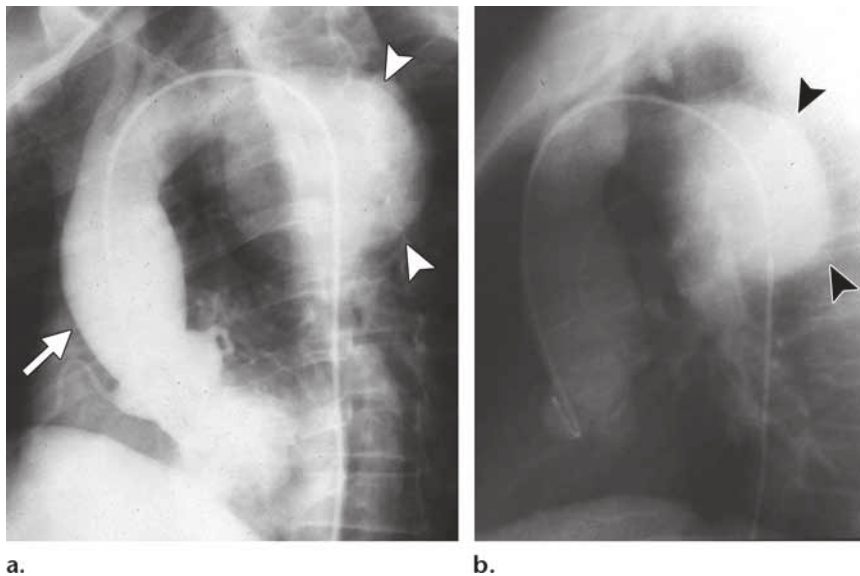


Figure 16. Syphilitic aortic aneurysm in a 59-year-old man with a long-standing history of syphilis. Images from conventional angiography show a fusiform aneurysm in the ascending aorta (arrow in **a**) and a saccular aneurysm in the proximal descending aorta (arrowheads).

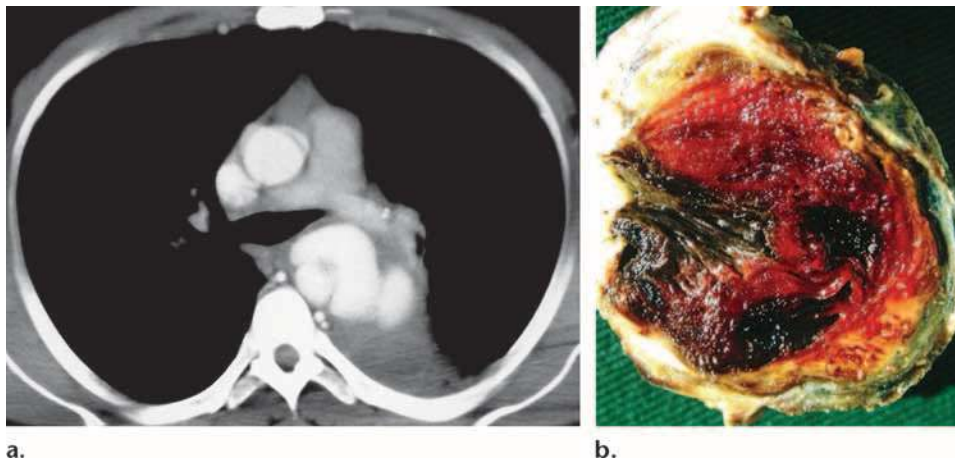


Figure 17. Tuberculous aortitis in a young man with atypical chest pain. **(a)** Contrast-enhanced CT image shows a pseudoaneurysm in the descending thoracic aorta, with hypoattenuating aortic wall thickening and surrounding inflammatory reaction. **(b)** Photograph of the surgical specimen shows necrosis and hemorrhage in the aortic wall.

Pyogenic Aortitis

Aortitis due to *Salmonella* infection generally affects the native aorta. It is secondary to bacteremia with endovascular seeding. It should be suspected in elderly or immunocompromised patients who develop high-grade bacteremia with prolonged fever and pain in the back, chest, or abdomen after an acute episode of gastroenteritis. Although complications are seen in less than

1% of cases, early detection is crucial to avoid such complications as endomyocardial abscess, mycotic aneurysm, aneurysm rupture, dissection, and even aortoenteric fistula (Fig 18). Treatment options include a prolonged course of antibiotics as well as surgical removal of infected tissue with restoration of distal flow (54).

Pneumococcal aortitis is seen mainly in the elderly. The endarteritis is believed to be secondary to bacteremia in 59% of cases. The most common sites of involvement in order of frequency are the abdominal aorta, descending thoracic aorta, and ascending aorta (65).

C septicum has a strong association with colonic neoplasms that spread hematogenously (66).

Infected (Mycotic) Aortic Aneurysm

The adjective *mycotic* in reference to infectious aneurysms has been a source of confusion. Used by Osler in 1885 when describing a mushroom-shaped aneurysm associated with endocarditis and not in regard to a fungal pathogenesis, the term *mycotic aortic aneurysm* now encompasses all aortic aneurysms that are secondary to an infectious cause. Predisposing factors include atherosclerosis, arterial grafts, intravascular catheters, joint prostheses, neoplasia, alcoholism, corticosteroid therapy, chemotherapy, diabetes mellitus, and other conditions that cause immunosuppression (67). Infected aortic aneurysm is infrequent, with a documented prevalence of 0.06%–2.6% among all aneurysms. If the aneurysm is left untreated, severe hemorrhage or sepsis may lead to early death (68).

Infected aortic aneurysm is a consequence of infectious aortitis of a vulnerable vessel that ultimately disrupts and weakens the vessel wall, creating a false lumen or pseudoaneurysm (69–72). The most common location is the infrarenal aorta, followed by the descending thoracic aorta, thoracoabdominal aorta, juxtarenal aorta, and ascending aorta (73). *Salmonella* has been found to be the most common organism implicated (57).

Mycotic aneurysms of the aortic root as well as aneurysms of the sinus of Valsalva have been associated with infectious endocarditis, unicuspid or bicuspid aortic valve, and infected prosthetic aortic valve (74).

At CT, the morphology of these aneurysms is mostly saccular (>90% of cases) rather than fusiform, with a diameter of 1–11 cm. Other CT findings include perianeurysmal gas, stranding, and fluid; vertebral body destruction with psoas abscess; and kidney infarct. Early in the course of disease, a periaortic soft-tissue mass with or without rim enhancement (depending on the degree of necrosis) may be the only finding before development of the aneurysm. This periaortic mass may be confused with neoplasia, infectious lymphadenopathy, or hematoma; the presence of a hypoattenuating concentric rim in the aortic

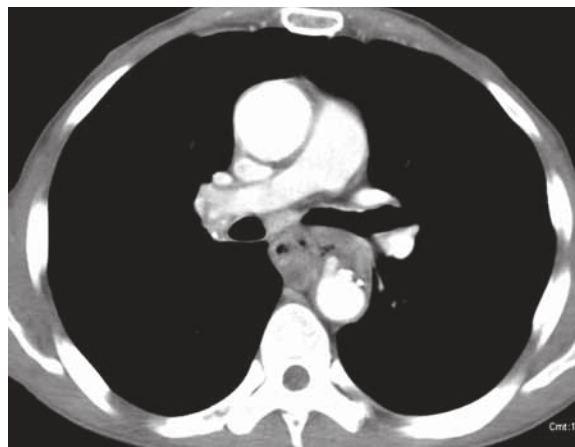


Figure 18. Mycotic pseudoaneurysm and penetrating ulcer in a 51-year-old man. Contrast-enhanced CT image shows deformity of the aortic lumen with areas of ulceration, periaortic soft tissue, and small gas bubbles. There is no clear plane between the inflammatory aortic lesion and the adjacent esophagus.

wall helps differentiate between these lesions (69,73) (Fig 19).

Delineation of the extent of the aneurysm is essential to decide between simple prolonged antibiotic therapy versus surgical management, such as resection of the diseased segment, débridement of periaortic tissue, or abscess drainage. Untreated mycotic aneurysms of the aorta have a poor outcome, with high mortality from rupture (50% of cases) or uncontrolled septic complications (68,75).

Aortitis Due to HIV Infection or AIDS

Several forms of infectious and noninfectious vasculitis have been reported in association with HIV infection, with complications including aortic and large-vessel aneurysmal dilatation, multiple aneurysms in the same patient, and occlusive vascular disease (Fig 20). The pathophysiology is complex and multifactorial, including vasculitis of the vasa vasorum with chronic inflammation and accelerated atherosclerotic disease (76).

Conclusions

Aortitis is a nonspecific term referring to inflammatory changes that affect the aortic wall. The term encompasses a wide array of infectious and noninfectious inflammatory conditions with different clinical and morphologic features and variable prognoses. Clinically, aortitis is characterized by nonspecific symptoms, leading to a complex diagnostic process; as a result, the condition is often overlooked during the initial work-up of patients with constitutional symptoms and systemic disorders.

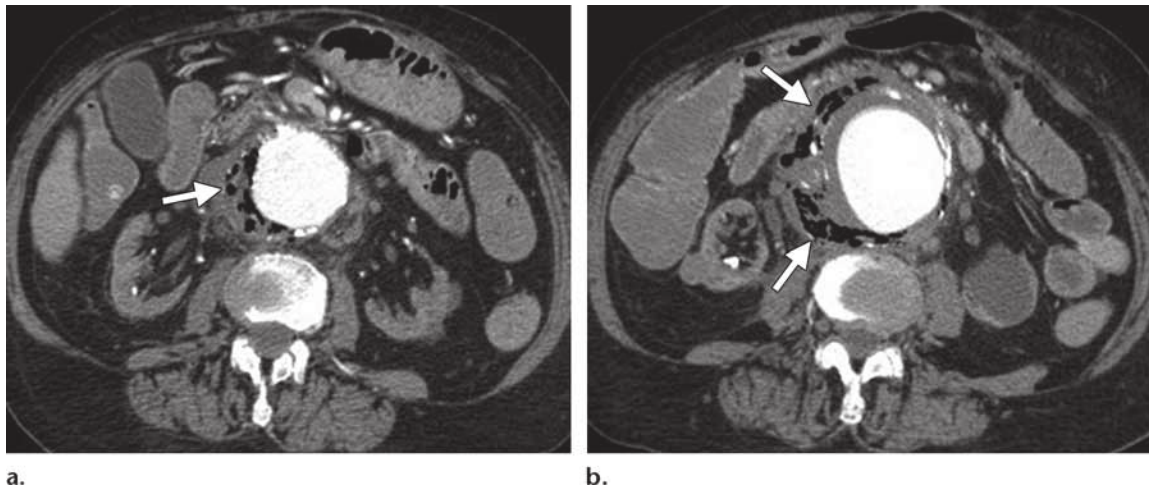


Figure 19. Infected abdominal aneurysm in a 77-year-old man. Contrast-enhanced CT images (**a** obtained at a higher level than **b**) show irregular collections of gas (arrows) in the wall of a large aneurysm in the distal abdominal aorta.

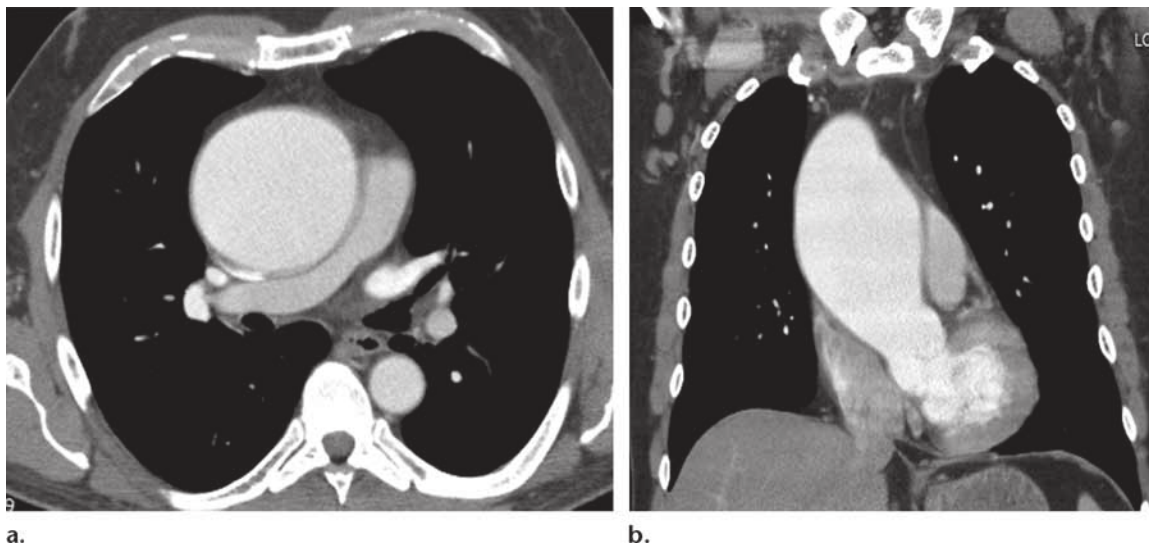


Figure 20. Aortic aneurysm in a 40-year-old man with AIDS. Axial (**a**) and coronal (**b**) contrast-enhanced CT images show a large fusiform aneurysm of the ascending aorta.

A multimodality imaging approach is often required for assessment of both the aortic wall and aortic lumen, as well as for surveillance of disease activity and treatment planning. Cross-sectional imaging with multidetector CT and MR imaging has largely replaced conventional angiography for initial diagnosis and follow-up. Radiologists should be familiar with the clinical features and imaging findings associated with the different types of aortitis.

References

- Gornik HL, Creager MA. Aortitis. *Circulation* 2008; 117(23):3039–3051.
- Slobodin G, Naschitz JE, Zuckerman E, et al. Aortic involvement in rheumatic diseases. *Clin Exp Rheumatol* 2006;24(2 suppl 41):S41–S47.
- Liang KP, Chowdhary VR, Michet CJ, et al. Non-infectious ascending aortitis: a case series of 64 patients. *J Rheumatol* 2009;36(10):2290–2297.
- Lee JL, Naguwa SM, Cheema GS, Gershwin ME. Revisiting Libman-Sacks endocarditis: a historical review and update. *Clin Rev Allergy Immunol* 2009; 36(2-3):126–130.
- Gotway MB, Araoz PA, Macedo TA, et al. Imaging findings in Takayasu's arteritis. *AJR Am J Roentgenol* 2005;184(6):1945–1950.
- Nastri MV, Baptista LP, Baroni RH, et al. Gadolinium-enhanced three-dimensional MR angiography of Takayasu arteritis. *RadioGraphics* 2004;24(3): 773–786.
- Matsunaga N, Hayashi K, Sakamoto I, Ogawa Y, Matsumoto T. Takayasu arteritis: protean radiologic manifestations and diagnosis. *RadioGraphics* 1997; 17(3):579–594.

8. Sueyoshi E, Sakamoto I, Hayashi K. Aortic aneurysms in patients with Takayasu's arteritis: CT evaluation. *AJR Am J Roentgenol* 2000;175(6):1727-1733.
9. Hayashi H, Katayama N, Takagi R. CT analysis of vascular wall during the active phase of Takayasu's aortitis [abstr]. *Eur Radiol* 1991;1(suppl):S239.
10. Yamada I, Nakagawa T, Himeno Y, Numano F, Shibuya H. Takayasu arteritis: evaluation of the thoracic aorta with CT angiography. *Radiology* 1998;209(1):103-109.
11. Choe YH, Han BK, Koh EM, Kim DK, Do YS, Lee WR. Takayasu's arteritis: assessment of disease activity with contrast-enhanced MR imaging. *AJR Am J Roentgenol* 2000;175(2):505-511.
12. Desai MY, Stone JH, Foo TK, Hellmann DB, Lima JA, Bluemke DA. Delayed contrast-enhanced MRI of the aortic wall in Takayasu's arteritis: initial experience. *AJR Am J Roentgenol* 2005;184(5):1427-1431.
13. Pacini D, Leone O, Turci S, et al. Incidence, etiology, histologic findings, and course of thoracic inflammatory aortopathies. *Ann Thorac Surg* 2008;86(5):1518-1523.
14. Bley TA, Ghanem NA, Foell D, et al. Computed tomography coronary angiography with 370-millisecond gantry rotation time: evaluation of the best image reconstruction interval. *J Comput Assist Tomogr* 2005;29(1):1-5.
15. Crow RW, Katz BJ, Warner JE, et al. Giant cell arteritis and mortality. *J Gerontol A Biol Sci Med Sci* 2009;64(3):365-369.
16. Bley TA, Uhl M, Venhoff N, Thoden J, Langer M, Markl M. 3-T MRI reveals cranial and thoracic inflammatory changes in giant cell arteritis. *Clin Rheumatol* 2007;26(3):448-450.
17. Zehr KJ, Mathur A, Orszulak TA, Mullany CJ, Schaff HV. Surgical treatment of ascending aortic aneurysms in patients with giant cell aortitis. *Ann Thorac Surg* 2005;79(5):1512-1517.
18. Lie JT, Failoni DD, Davis DC Jr. Temporal arteritis with giant cell aortitis, coronary arteritis, and myocardial infarction. *Arch Pathol Lab Med* 1986;110(9):857-860.
19. Gelsomino S, Romagnoli S, Gori F, et al. Annuloaortic ectasia and giant cell arteritis. *Ann Thorac Surg* 2005;80(1):101-105.
20. Haddad FG, El-Nemnoun R, Haddad F, Maalouly G, El-Rassi I. Giant cell arteritis of the aorta: catastrophic complications without a preexisting aneurysm. *Eur J Intern Med* 2008;19(7):e59-e60.
21. Bongartz T, Matteson EL. Large-vessel involvement in giant cell arteritis. *Curr Opin Rheumatol* 2006;18(1):10-17.
22. Tso E, Flamm SD, White RD, Schwartzman PR, Mascha E, Hoffman GS. Takayasu arteritis: utility and limitations of magnetic resonance imaging in diagnosis and treatment. *Arthritis Rheum* 2002;46(6):1634-1642.
23. Walter MA. [(18)F]fluorodeoxyglucose PET in large vessel vasculitis. *Radiol Clin North Am* 2007;45(4):735-744.
24. Blockmans D, Stroobants S, Maes A, Mortelmans L. Positron emission tomography in giant cell arteritis and polymyalgia rheumatica: evidence for inflammation of the aortic arch. *Am J Med* 2000;108(3):246-249.
25. Bau JL, Ly JQ, Borstad GC, Lusk JD, Seay TM, Beall DP. Giant cell arteritis. *AJR Am J Roentgenol* 2003;181(3):742.
26. Roldan CA, Chavez J, Wiest PW, Qualls CR, Crawford MH. Aortic root disease and valve disease associated with ankylosing spondylitis. *J Am Coll Cardiol* 1998;32(5):1397-1404.
27. Dib C, Moustafa SE, Mookadam M, Zehr KJ, Michet CJ Jr, Mookadam F. Surgical treatment of the cardiac manifestations of relapsing polychondritis: overview of 33 patients identified through literature review and the Mayo Clinic records. *Mayo Clin Proc* 2006;81(6):772-776.
28. Selim AG, Fulford LG, Mohiaddin RH, Sheppard MN. Active aortitis in relapsing polychondritis. *J Clin Pathol* 2001;54(11):890-892.
29. Wilson GE, Hasleton PS, Manns JJ, Marks JS. Relapsing polychondritis: bone marrow and circular fibrous nodules in the aorta. *Ann Rheum Dis* 1990;49(10):795-797.
30. Giordano M, Valentini G, Sodano A. Relapsing polychondritis with aortic arch aneurysm and aortic arch syndrome. *Rheumatol Int* 1984;4(4):191-193.
31. Manna R, Annese V, Ghirlanda G, et al. Relapsing polychondritis with severe aortic insufficiency. *Clin Rheumatol* 1985;4(4):474-480.
32. Gravalles EM, Corson JM, Coblyn JS, Pinkus GS, Weinblatt ME. Rheumatoid aortitis: a rarely recognized but clinically significant entity. *Medicine (Baltimore)* 1989;68(2):95-106.
33. Levine AJ, Dimitri WR, Bonser RS. Aortic regurgitation in rheumatoid arthritis necessitating aortic valve replacement. *Eur J Cardiothorac Surg* 1999;15(2):213-214.
34. Hoshina K, Koyama H, Miyata T, et al. Aortic wall cell proliferation via basic fibroblast growth factor gene transfer limits progression of experimental abdominal aortic aneurysm. *J Vasc Surg* 2004;40(3):512-518.
35. Smith DC, Hirst AE. Spontaneous aortic rupture associated with chronic steroid therapy for rheumatoid arthritis in two cases. *AJR Am J Roentgenol* 1979;132(2):271-273.
36. Breynaert C, Cornelis T, Stroobants S, Bogaert J, Vanhoof J, Blockmans D. Systemic lupus erythematosus complicated with aortitis. *Lupus* 2008;17(1):72-74.
37. Ohara N, Miyata T, Kurata A, Oshiro H, Sato O, Shigematsu H. Ten years' experience of aortic aneurysm associated with systemic lupus erythematosus. *Eur J Vasc Endovasc Surg* 2000;19(3):288-293.
38. Grasland A, Pouchot J, Hachulla E, et al. Typical and atypical Cogan's syndrome: 32 cases and review of the literature. *Rheumatology (Oxford)* 2004;43(8):1007-1015.
39. Tseng JF, Cambria RP, Aretz HT, Brewster DC. Thoracoabdominal aortic aneurysm in Cogan's syndrome. *J Vasc Surg* 1999;30(3):565-568.
40. Chae EJ, Do KH, Seo JB, et al. Radiologic and clinical findings of Behçet disease: comprehensive review of multisystemic involvement. *RadioGraphics* 2008;28(5):e31.
41. Ko GY, Byun JY, Choi BG, Cho SH. The vascular manifestations of Behçet's disease: angiographic and CT findings. *Br J Radiol* 2000;73(876):1270-1274.

42. Rojo-Leyva F, Ratliff NB, Cosgrove DM 3rd, Hoffman GS. Study of 52 patients with idiopathic aortitis from a cohort of 1,204 surgical cases. *Arthritis Rheum* 2000;43(4):901–907.
43. Girardi LN, Coselli JS. Inflammatory aneurysm of the ascending aorta and aortic arch. *Ann Thorac Surg* 1997;64(1):251–253.
44. Iino M, Kuribayashi S, Imakita S, et al. Sensitivity and specificity of CT in the diagnosis of inflammatory abdominal aortic aneurysms. *J Comput Assist Tomogr* 2002;26(6):1006–1012.
45. Cullenward MJ, Scanlan KA, Pozniak MA, Acher CA. Inflammatory aortic aneurysm (periaortic fibrosis): radiologic imaging. *Radiology* 1986;159(1):75–82.
46. Lee YK, Seo JB, Kim SS, Lim TH. Thoracic periaortic fibrosis mimicking malignant tumor: CT and 18F-FDG PET findings. *AJR Am J Roentgenol* 2007;188(2):345–347.
47. Meier P, Vogt B, Blanc E. Rethinking the triggering inflammatory processes of chronic periaortitis. *Nephron Exp Nephrol* 2007;105(1):e17–e23.
48. Vaglio A, Salvarani C, Buzio C. Retroperitoneal fibrosis. *Lancet* 2006;367(9506):241–251.
49. Cronin CG, Lohan DG, Blake MA, Roche C, McCarthy P, Murphy JM. Retroperitoneal fibrosis: a review of clinical features and imaging findings. *AJR Am J Roentgenol* 2008;191(2):423–431.
50. Mesurole B, Qanadli SD, Merad M, et al. Unusual radiologic findings in the thorax after radiation therapy. *RadioGraphics* 2000;20(1):67–81.
51. Modrall JG, Sadjadi J. Early and late presentations of radiation arteritis. *Semin Vasc Surg* 2003;16(3):209–214.
52. Coblenz C, Martin L, Tuttle R. Calcified ascending aorta after radiation therapy. *AJR Am J Roentgenol* 1986;147(3):477–478.
53. Foote EA, Postier RG, Greenfield RA, Bronze MS. Infectious aortitis. *Curr Treat Options Cardiovasc Med* 2005;7(2):89–97.
54. Fernández Guerrero ML, Aguado JM, Arribas A, Lumbreras C, de Gorgolas M. The spectrum of cardiovascular infections due to *Salmonella enterica*: a review of clinical features and factors determining outcome. *Medicine (Baltimore)* 2004;83(2):123–138.
55. Gomes MN, Choyke PL, Wallace RB. Infected aortic aneurysms: a changing entity. *Ann Surg* 1992;215(5):435–442.
56. Bucknell SJ, Le T, Amerena J, Hill DG, McDonald M. Aortic dissection associated with *Campylobacter* aortitis. *Heart Lung Circ* 2000;9(2):88–91; discussion 91.
57. Mendelowitz DS, Ramstedt R, Yao JS, Bergan JJ. Abdominal aortic salmonellosis. *Surgery* 1979;85(5):514–519.
58. Narang AT, Rathlev NK. Non-aneurysmal infectious aortitis: a case report. *J Emerg Med* 2007;32(4):359–363.
59. Kennedy JL, Barnard JJ, Prahlow JA. Syphilitic coronary artery ostial stenosis resulting in acute myocardial infarction and death. *Cardiology* 2006;105(1):25–29.
60. Bodhey NK, Gupta AK, Neelakandhan KS, Unnikrishnan M. Early sternal erosion and luetic aneurysms of thoracic aorta: report of 6 cases and analysis of cause-effect relationship. *Eur J Cardiothorac Surg* 2005;28(3):499–501.
61. Allins AD, Wagner WH, Cossman DV, Gold RN, Hiatt JR. Tuberculous infection of the descending thoracic and abdominal aorta: case report and literature review. *Ann Vasc Surg* 1999;13(4):439–444.
62. Jain AK, Chauhan RS, Dhammi IK, Maheshwari AV, Ray R. Tubercular pseudoaneurysm of aorta: a rare association with vertebral tuberculosis. *Spine J* 2007;7(2):249–253.
63. Choi JB, Yang HW, Oh SK, Yun KJ. Rupture of ascending aorta secondary to tuberculous aortitis. *Ann Thorac Surg* 2003;75(6):1965–1967.
64. Long R, Guzman R, Greenberg H, Safneck J, Hershfield E. Tuberculous mycotic aneurysm of the aorta: review of published medical and surgical experience. *Chest* 1999;115(2):522–531.
65. Bronze MS, Shirwany A, Corbett C, Schaberg DR. Infectious aortitis: an uncommon manifestation of infection with *Streptococcus pneumoniae*. *Am J Med* 1999;107(6):627–630.
66. Mohamed HK, Elliott BM, Brothers TE, Robison JG. Suprarenal *Clostridium septicum* aortitis with rupture and simultaneous colon cancer. *Ann Vasc Surg* 2006;20(6):825–829.
67. Parellada JA, Palmer J, Monill JM, Zidan A, Giménez AM, Moreno A. Mycotic aneurysm of the abdominal aorta: CT finding in three patients. *Abdom Imaging* 1997;22(3):321–324.
68. Gomes MN, Choyke PL. Infected aortic aneurysms: CT diagnosis. *J Cardiovasc Surg (Torino)* 1992;33(6):684–689.
69. Rozenblit A, Bennett J, Suggs W. Evolution of the infected abdominal aortic aneurysm: CT observation of early aortitis. *Abdom Imaging* 1996;21(6):512–514.
70. Chan FY, Crawford ES, Coselli JS, Safi HJ, Williams TW Jr. In situ prosthetic graft replacement for mycotic aneurysm of the aorta. *Ann Thorac Surg* 1989;47(2):193–203.
71. Yang CY, Liu KL, Lee CW, Tsang YM, Chen SJ. Mycotic aortic aneurysm presenting initially as an aortic intramural air pocket. *AJR Am J Roentgenol* 2005;185(2):463–465.
72. Sueyoshi E, Sakamoto I, Kawahara Y, Matsuoka Y, Hayashi K. Infected abdominal aortic aneurysm: early CT findings. *Abdom Imaging* 1998;23(6):645–648.
73. Macedo TA, Stanson AW, Oderich GS, Johnson CM, Panneton JM, Tie ML. Infected aortic aneurysms: imaging findings. *Radiology* 2004;231(1):250–257.
74. Feigl D, Feigl A, Edwards JE. Mycotic aneurysms of the aortic root: a pathologic study of 20 cases. *Chest* 1986;90(4):553–557.
75. Malouf JF, Chandrasekaran K, Orszulak TA. Mycotic aneurysms of the thoracic aorta: a diagnostic challenge. *Am J Med* 2003;115(6):489–496.
76. Restrepo CS, Diethelm L, Lemos JA, et al. Cardiovascular complications of human immunodeficiency virus infection. *RadioGraphics* 2006;26(1):213–231.

Aortitis: Imaging Spectrum of the Infectious and Inflammatory Conditions of the Aorta

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Page 436

Rheumatic diseases with a high prevalence (>10%) of aortic involvement include Takayasu arteritis, GCA, long-standing ankylosing spondylitis, Cogan syndrome (interstitial keratitis, iritis, conjunctival or subconjunctival hemorrhage, fever, aortic insufficiency), and relapsing polychondritis.

Page 439

GCA is a chronic vasculitis that affects large and medium-sized vessels and usually involves the superficial cranial arteries.

Page 439

GCA is closely related to polymyalgia rheumatica.

Page 445

The aorta is normally very resistant to infection; however, an abnormal aortic wall, like that associated with atherosclerotic disease, preexisting aneurysm, cystic medial necrosis, diabetes, vascular malformation, medical devices, or surgery, makes it more susceptible to infection (53,54)

Page 448

Mycotic aneurysms of the aortic root as well as aneurysms of the sinus of Valsalva have been associated with infectious endocarditis, unicuspid or bicuspid aortic valve, and infected prosthetic aortic valve (74).