Cross-sectional imaging of aortic infections

The Harvard community has made this article openly available. Please share how this access benefits you. Your story matters

| Citation       | Murphy, D. J., A. R. Keraliya, M. D. Agrawal, A Aghayev, and M. L. Steigner. 2016. “Cross-Sectional Imaging of Aortic Infections.” Insights into Imaging 7 (6) (October 19): 801–818. doi:10.1007/s13244-016-0522-5. |
|----------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Published Version | doi:10.1007/s13244-016-0522-5                                                                                                                                                               |
| Citable link   | http://nrs.harvard.edu/urn-3:HUL.InstRepos:30510287                                                                                                                                 |
| Terms of Use   | This article was downloaded from Harvard University’s DASH repository, and is made available under the terms and conditions applicable to Other Posted Material, as set forth at http://nrs.harvard.edu/urn-3:HUL.InstRepos:dash.current.terms-of-use#LAA |


Abstract

Aortic infections are uncommon clinical entities, but are associated with a high morbidity and mortality. In this review, we focus on the cross-sectional imaging appearances of aortic infections, including aortic valve endocarditis, pyogenic aortitis, mycotic aneurysm and aortic graft infections, with an emphasis on CT and MRI.
Introduction

Aortic infections are rare, but are associated with significant morbidity and mortality. The imaging appearances of aortic infections vary based on the location of infection, the culprit organism, the presence of aortic graft material and any other previous surgical intervention.

Imaging Modalities

Echocardiography, computed tomography (CT), magnetic resonance angiography (MRA) and flurodeoxyglucose positron emission tomography (18F-FDG PET/CT) are the principal imaging modalities used to image the aorta in cases of suspected infection. Transthoracic echocardiography (TTE) is often the initial imaging modality in cases of suspected aortic valve infection. It is a quick, non-invasive, with the beneficial lack of ionizing radiation. Imaging of the aortic valve by TTE can be complicated by artifacts, especially in patients with valvular calcification or with valve prostheses. Transesophageal echocardiography (TEE) is a more sensitive test to evaluate the aortic valve for suspected endocarditis, but has the disadvantage of being an invasive modality, often requiring sedation (1).

CT angiography (CTA) is a rapid, three-dimensional cross-sectional imaging technique. It is commonly the initial imaging study in cases of suspected aortic infection, offering excellent spatial and temporal resolution. The use of ECG-gating when performing CT of the aortic valve or thoracic aorta improves image quality, negating the deleterious affect of cardiac motion. The improved temporal resolution with modern scanners, as low as 66 milliseconds (ms) in third generation dual source CTs, allows for excellent anatomical assessment of the aortic valve throughout the
cardiac cycle (2). During CTA of the aorta in cases of suspected infection, acquisition of an arterial phase with an additional delayed phase (approximately 70s) may be helpful to assess for infection related complications.

MRA offers multiparametric aortic imaging with excellent soft tissue contrast and tissue characterization. Steady state free precession (SSFP) cine imaging of the aortic valve provides both anatomical and functional information on the aortic valve. A temporal resolution of less than 50ms is readily achievable, enabling accurate assessment of valve structure and function throughout the cardiac cycle. Contrast-enhanced MR angiography (CE-MRA) with gadolinium based contrast agents provides excellent anatomical detail of the aorta with superior contrast resolution. Double inversion recovery (DIR) sequences null the blood pool, providing excellent visualization of the aortic wall distinct from the aortic lumen, a unique feature of MRA. The beneficial lack of ionizing radiation makes MRA a particularly useful modality in the serial follow-up of patients.

The assessment of systemic infection/ inflammation has emerged as one of the principal applications of 18F-FDG PET/CT outside oncology. Foci of active infection are often metabolically active, and will avidly take-up glucose. 18F-FDG PET/CT’s main role is currently as a problem-solving tool in equivocal cases.

In this review, we will focus mainly on the cross-sectional imaging modalities, namely CTA, MRA and 18F-FDG PET/CT and their role in the assessment of aortic infections.
Aortic Valve Endocarditis

Infective endocarditis (IE) of the aortic valve is a serious, life-threatening condition with a mortality of 30% at one year (3). It is caused be infection of the aortic valve, either native or prosthetic. The major risk factors for native valve endocarditis include degenerative valve disease, diabetes, cancer, intravenous drug use and congenital heart disease (4). The epidemiology of infective endocarditis has changed, with nosocomial infective endocarditis now accounting for approximately 30% of cases, largely due to the increased use of long-term intravenous lines, invasive procedures, prosthetic valves and indwelling cardiac devices (5). Gram-positive cocci (Staphylococcus, Streptococcus and Enterococcus) account for 80-90% of cases of infective endocarditis, with S. aureus the most frequently isolated organism, causing up to 30% of cases (5). Staphylococcal infective endocarditis is no longer limited to the traditional at-risk groups such as renal failure patients on hemodialysis and intravenous drug users, and it can affect both native and prosthetic valves (6).

Infective endocarditis has a myriad of clinical presentations, such as pyrexia of unknown origin, stroke and systemic emboli. Definitive diagnosis requires integration of clinical, laboratory and imaging results, incorporated into the modified Duke criteria (7,8). Echocardiography is central to the imaging diagnosis, with the presence of valvular vegetations, periannular tissue destruction, abscess, aneurysms, fistulas, leaflet perforation or valvular dehiscence all signs of endomyocardial valvular infection. Transthoracic echocardiography (TTE) is a moderately sensitive and highly specific test (75% and 90% respectively) for the presence of vegetations in suspected native valve endocarditis (9). Transesophageal echocardiography (TEE) is the current imaging gold standard, with a sensitivity of more than 90%, and is useful
in cases with a high clinical suspicion and a negative TTE (9). Both TTE and TEE can be limited by factors such as patient habitus, variant anatomy, artifact from heavy valve calcification and the presence of metal prosthetic valves.

CT provides aortic valve imaging with high spatial and temporal resolution. It is an excellent imaging option in patients with suspected infective endocarditis, particularly in patients with a negative TTE who have a high clinical suspicion, and are too high risk for a TEE. A series by Feuchtner et al. compared the diagnostic performance of ECG-gated CT compared to TEE in patients with clinically suspected IE, with CT performing comparatively well, with a sensitivity of 97% and a specificity of 88% (10).

On CT, vegetations appear as irregularly shaped, low attenuation masses adherent to the valve or endocardial surface, which usually oscillate throughout the cardiac cycle (11). Vegetation size and mobility are the most important factors in determining the risk of cerebrovascular or systemic embolism. The vegetation size is defined by its maximal length, with those >10 mm carrying a very high risk of embolism (1). CT has a high sensitivity in detecting these large vegetations, with one series reporting a sensitivity of 100% for lesions larger than 10mm (12). The detection of vegetations by CT is challenging when there are pre-existing calcified degenerative lesions and when the vegetations are small, especially when < 2 mm in size (13). Large vegetations may cause perforation of the aortic valve leaflets, which is readily visible on CT. The 2015 European Society of Cardiology (ESC) guidelines on the diagnosis and treatment of IE have proposed the presence of a paravalvular lesion on CT as a major criterion in the modified Duke criteria (14).
Cardiac MRI’s (CMR) role in the initial diagnosis of suspected aortic valve endocarditis is limited. Vegetations are best depicted on SSFP cine imaging as low signal masses attached to the valve surface or endocardium, which oscillate during the cardiac cycle. The presence of off-resonance artifact, particularly at 3.0 Tesla, can hinder the use of SSFP sequences in aortic valve imaging, often requiring use of gradient echo (GRE) cine imaging (15). CMR can help quantify the severity of aortic insufficiency, if present, which can be useful in triaging patients with endocarditis to medical or surgical treatment (1).

18F-FDG PET/CT can be used to visualize vegetations, which will avidly take up the glucose tracer. Although PET/CT does not have a sensitivity comparable to echocardiography or CT in the diagnosis of native valve endocarditis, it can be a useful problem solving tool in cases with a high clinical suspicion with negative imaging, and in the detecting of distant septic emboli (16,17).

The major differential diagnoses of aortic valve vegetations are thrombi, papillary fibroelastoma, myxomatous changes and giant Lambl’s excrescences (18). Papillary fibroelastoma are usually attached to the aortic side of the valve and can potentially cause coronary ostial obstruction, whilst vegetations are often attached to the free end of the valve (19). These tumors typically enhance on CMR following gadolinium administration, which can be another distinguishing feature (20). Giant Lambl’s excrescences are formed by the coalescence of multiple filiform fronds, which form at sites of valve closure likely resulting from endothelial wear and tear (21). They are associated with thrombi, and can cause embolic events (22,23). Overall, there is
significant overlap in the imaging features between these entities and vegetations. Incorporating the imaging findings with the clinical and laboratory data is thus crucial in making an accurate diagnosis of IE.

*Local endocarditis-related complications*

Extension of infection into the peri-valvular tissue can manifest as abscesses, pseudoaneurysms and fistulae. These complications occur in 10-40% of cases of native aortic valve IE, necessitating surgical treatment (24). CT is excellent at detecting peri-valvular extension, with one series demonstrating a sensitivity of 100%, using surgery as a reference standard (10). Peri-valvular abscess appears on CT as a collection of fluid density around the aortic valve, surrounded by inflammatory tissue, which can enhance. An abscess can spread into surrounding structures, such as the interatrial septum or left ventricular myocardium. This can manifest clinically as atrioventricular (AV) nodal block, new bundle branch block or persistent sepsis. Rupture of a peri-valvular abscess into the aortic root creates a pseudoaneurysm. This manifests on CT as an abnormal contrast-containing cavity adjacent to the aortic valve, which freely communicates with the aortic lumen.

Extension of a pseudoaneurysm or abscess into the intervalvular fibrous body (IFB) is an important finding which may have implications on surgical planning, and one which can be readily assessed on CT (12,25). The IFB is a fibrous structure located between the lateral and medial fibrous trigones, connecting the anterior mitral valve leaflet, the left and non-coronary aortic valve cusps to the heart’s fibrous skeleton. Destruction of the IFB by an aortic root abscess or pseudoaneurysm complicates surgical intervention, removing the inherent local stability required to successfully
perform an aortic valve replacement. In these cases, radical debridement of all the infected tissue with reconstruction of the IFB, followed by aortic and mitral valve replacement is the only surgical option (26). Fistula formation is a rare and serious complication of aortic valve IE, occurring when a peri-valvular abscess or pseudoaneurysm ruptures into an adjacent cardiac cavity.

*Prosthetic valve endocarditis*

Aortic valve prosthetic valve endocarditis (PVE) has a poor prognosis, with a reported mortality of 20-40% (27). This is likely due to the higher propensity for infection to extend into the peri-valvular tissue compared with native valve endocarditis (1). Bioprosthetic valves (also referred to as tissue valves) are predominantly composed of soft tissue material (usually porcine xenograft), and have similar imaging appearances when infected as those described in native valve infection.

Mechanical prosthetic valve imaging by echocardiography can be limited by artifact caused by acoustic shadowing. IE is suspected on a prosthetic valve when there is a peri-valvular mass, or when valve dehiscence can be demonstrated. ECG-gated CT performs well in the identification of PVE, with one series reporting a sensitivity of 93% when compared to surgical findings (28). The major signs of PVE on CT are the presence of vegetations, thickening of the aortic root wall of >5mm, the presence of a peri-valvular abscess or pseudoaneurysm and prosthetic valve dehiscence. The latter manifests as a rocking motion of the mechanical valve throughout the cardiac cycle, which can be demonstrated on ECG-gated cardiac CT with a multi-phase acquisition throughout the cardiac cycle (11,28). Transcatheter aortic valve replacements
(TAVR) are a novel category of mechanical aortic valves, often placed in patients who are deemed to high risk to undergo open aortic valve replacement. Cases of endocarditis post TAVR have similar appearances on CT to mechanical aortic PVE (29).

A systematic review of the role of non-invasive imaging in the diagnosis of PVE supports the use of CT in addition to echocardiography in improving the diagnostic accuracy, especially in cases of life-threatening peri-valvular extension (30). One of the main limitations of CT in the assessment of PVE is the presence of streak artifact caused by the high-density valve material. Artifact caused by the valve material can also limit the role of CMR in the diagnosis of mechanical PVE, causing local spin dephasing. Given these limitations, 18F-FDG PET/CT has emerged as useful adjunct in the diagnosis of PVE. Studies by Saby et al. and Pizzi et al. demonstrated that the addition of increased tracer uptake around the prosthetic valve on 18F-FDG PET/CT in cases of suspected PVE increased the sensitivity of the modified Duke criteria from 70 to 97% and 52 to 91% respectively (31,32). The current 2015 ESC IE guidelines recommend that the presence of abnormal increased activity on a prosthetic valve by 18F-FDG PET/CT should be considered a major criterion for PVE diagnosis, once the valve has been implanted for more than 3 months (14).

**Infectious aortitis**

The aortic intima is normally very resistant to infection, with most cases of aortic wall infection occurring in patients with an underlying mural abnormality, such as atherosclerosis, cystic medical necrosis, or in the presence of an aortic prosthesis (33).
Bacteria are the most common culprit microorganisms, especially *Salmonella, Enterococcus* and *Staphylococcus* species, and are often associated with a concurrent episode of gastroenteritis or osteomyelitis (34,35). Other causative organisms include *Streptococcus pneumoniae, Listeria, Bacteroides fragilis, Clostridium, Human Immunodeficiency Virus (HIV), Mycobacterium tuberculosis* and *Treponema pallidum* (36,37). Fungal aortic infection is uncommon, usually occurring in immunosuppressed patients, such as those on chemotherapy or HIV patients.

**Pyogenic aortitis**

Bacterial aortic wall infection usually occurs when a segment of the wall is seeded by bacteria via the vasa vasorum (35). This can result from systemic septic embolism, hematogenous seeding, spread from an adjacent focus of infection or by iatrogenic direct inoculation. The clinical presentation is determined by the site and extent of infection, varying from back or abdominal pain with fever to acute severe acute aortic regurgitation. *S.pneumoniae* and *Enterococcus* are commonly implicated in thoracic aortic infection, whilst *Salmonella* is the most common cause of infectious abdominal aortitis (38). Prompt diagnosis is crucial, with a mortality of up to 44%, despite treatment (39). CT/MR aortography are the imaging modalities of choice. CT is more commonly utilized, secondary to greater availability, and the shorter acquisition time in patients who are commonly systemically unwell. Signs of pyogenic aortitis on cross sectional imaging include aortic wall thickening (often asymmetric rather than circumferential) and enhancement, periaortic fluid, high attenuation periaortic soft tissue, rapidly enlarging saccular-type aortic aneurysm/pseudoaneurysm and air in the aortic wall (36,37). Aortic mural gas is easier to appreciate on CT than MRI, whilst aortic wall enhancement is often better visualized on MRI, especially using T1-
weighted sequences pre and post gadolinium administration. The potential complications of pyogenic aortitis include septic emboli, mycotic aneurysm formation, aortic rupture and fistula formation.

*Mycotic aneurysms*

The term ‘mycotic aneurysm’ can be a source of confusion. It refers to all aortic aneurysms caused by infection, regardless of the culprit microorganism. They are rare entities, making up 0.7-2.6% of all aortic aneurysms (40). They usually result from an infectious aortitis, which weakens the wall causing a contained aortic rupture and formation of a pseudoaneurysm. They most commonly affect the infrarenal abdominal aorta, with *Salmonella* the most common causative organism (40). They typically appear as a saccular aortic aneurysm with lobulated contours, periaortic soft tissue stranding and edema/fluid (36,37,40). Asymmetric periaortic fat density or a periaortic soft tissue mass may be the only early signs of infection before aneurysm development (41). Other imaging findings include perianeurismal gas, vertebral body destruction, psoas abscess formation and renal infarction (37). Early diagnosis is crucial, as untreated mycotic aneurysms are associated with a high mortality from rupture or uncontrolled sepsis (38,42).

*Tuberculous Aortitis*

Infection of the aortic wall with *Mycobacterium tuberculosis* is rare. It can be caused by direct extension from adjacent tuberculous infected tissue, or by hematogenous seeding from a remote tuberculous focus, the latter suggesting the presence of disseminated tuberculosis (43). Tuberculous aortitis commonly affects the distal aortic arch and descending thoracic aorta, presenting with a pseudoaneurysm caused
by caseous necrosis of the aortic wall (44). On cross-sectional imaging, tubercular aortitis often appears as a focal, saccular pseudoaneurysm with multiple lobular outpouchings and an irregular, thickened aortic wall (45). Perforation of the aorta into adjacent structures such as the esophagus, pulmonary tree, peritoneal cavity and small bowel have been reported (46). Aortic dissection and focal aortic wall thickening without aneurysmal dilatation may also occur (45). The latter entity can have identical imaging appearances to a chronic periaortitis, appearing as circumferential aortic encapsulation with enhancing soft tissue, which may result in symptomatic stenosis (47). The overall prognosis of tuberculous aortitis is poor, with mortality rates as high as 60% despite appropriate anti-tubercular and surgical treatment (46).

**Syphilitic Aortitis**

Syphilis is a chronic infection caused by the sexually transmitted spirochete *Treponema pallidum*. Tertiary syphilis is characterized by cardiovascular (luetic) involvement, along with the presence of gummas and neurological involvement, typically occurring 15 to 30 years after the initial infection. Luetic syphilis is caused by an endarteritis of the vasa vasorum, and can manifest as an aortitis, aortic aneurysm, aortic regurgitation and coronary artery stenosis (37). The ascending thoracic aorta is the most commonly affected aortic site, followed by the aortic arch (48). The most common appearance on CT/MR is diffuse aortic mural thickening with associated aneurysm formation, which may be multiple and saccular (49). Syphilitic thoracic aortic aneurysms may grow to a considerable size, and can cause sternal or clavicle erosion (50). The aortic wall may have a double-ring appearance on CT, with hyperdense outer and hypodense inner layers (51). Chronic inflammation of the aorta leads to intimal fibrosis and calcification, which can give rise to a tree bark
appearance of the aortic wall, which may be visible on chest radiographs (37,52). Aneurysm rupture, severe aortic insufficiency and coronary artery ostial narrowing are the most common causes of death in patients with luetic syphilitic (53).

*Human Immunodeficiency Virus (HIV) aortitis*

HIV causes aortitis though a complex mechanism of infectious and non-infectious processes. It accelerates atherosclerotic disease, which results in both occlusive and aneurysmal aortic disease (54). There are also a broad spectrum of non-atherosclerotic vasculitides which can occur in patients with HIV, either due to opportunistic infection, or directly caused by HIV infection (55). The imaging features are non-specific, including aortic aneurysms, aneurysms involving multiple large vessels, aortic dissection and occlusive aortic disease (56).

**Aorta infection post graft repair**

Infection is a rare complication of aortic graft repair with significant mortality and morbidity. The incidence of prosthetic graft infection post open aortic repair varies from 0.6% to 5%, with a mortality rate ranging from 25% to 88% (57,58). Infection rates are lower with endovascular graft repair that with open surgical approaches, with an incidence of approximately 1% (59). The most common microorganism isolated within the first three months after an open repair is *Staphylococcus aureus*, with coagulase-negative *staphylococcus* more common in late infections (60). A similar flora is implicated in endograft infections, with *staphylococcus* and *streptococcus* most commonly isolated (59,61). There are several treatment options for infected aortic grafts, endovascular and open, including graft excision with extra-anatomic bypass and in situ reconstruction (62). Symptoms of aortic graft infection can be non-
specific, including recurrent fever and chills, back or chest pain, erythema and swelling. Early diagnosis is crucial, with treatment delays associated with significant morbidity and mortality (63).

CT is the most commonly used imaging modality to assess for aortic graft infection. Signs of infection on CT include persistent or expanding perigraft soft tissue thickening, perigraft fluid collections and gas, after the initial postoperative period (36,57,58). Persistence of perigraft soft-tissue attenuation and fluid beyond 3 months post open aortic repair is suspicious for graft infection. The presence of perigraft air beyond the initial post-operative period is suspicious for, but not pathognomonic of, an aortoenteric fistula. The combination of ectopic gas and focal bowel wall thickening and/or tethering of bowel loops adjacent to the graft site is highly suggestive for an aortoenteric fistula (64). In rare cases, extravasation of contrast from the aorta into the involved loop of bowel can be demonstrated on CT. Pseudoaneurysm formation is a serious complication of graft infection, occurring particularly in cases of endovascular graft infection (65,66).

On of the major challenges is identifying patients with low grade graft infections, with a reported sensitivity and specificity on CT of 55-64% and 86-100% respectively (67,68). Nevertheless, CT is a highly specific, relatively highly sensitive test in the diagnosis of aortic prosthetic infections, and can provide image guidance for a fine needle aspiration to provide a definitive microbiological diagnosis.

Tissue characterization is a useful feature of MRI in cases of suspected graft infection. MRI can distinguish perigraft fluid and inflammation from chronic
hematoma based on T1 and T2 signal characteristics and post-contrast enhancement. Shahidi et al. compared MRI with Indium-111 labeled white blood cell scanning in the diagnosis of aortic graft infection in 59 patients; MRI performed well, with a sensitivity of 68% and a specificity of 97% (69).

To help overcome some of the shortcomings of CT and MRI in the diagnosis of aortic graft infections, especially in low-grade infections, 18F-FDG PET/CT has been proposed as useful adjunct. Keidar et al. investigated its role in the diagnosis of vascular graft infection, reporting a sensitivity and specificity of 93% and 91% (70). False positives may occur due to chronic inflammation on the graft surface. This manifests as diffuse low to moderate FDG avidity along the graft (71). A focal or heterogeneous pattern of increased FDG uptake along the graft is suggestive of infection (72).

**Conclusion**

Cross-sectional imaging provides an important role in the diagnosis and management of the broad spectrum of aortic infections. It is crucial that reporting radiologists are vigilant to the various manifestations of aortic infections on cross-sectional imaging, as prompt diagnosis and treatment can be life saving.
References

1. Habib G, Hoen B, Tornos P et al. Guidelines on the prevention, diagnosis, and treatment of infective endocarditis (new version 2009): the Task Force on the Prevention, Diagnosis, and Treatment of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) and the International Society of Chemotherapy (ISC) for Infection and Cancer. European Heart Journal. 2009;19: 2369–2413.

2. Flohr TG, De Cecco CN, Schmidt B, Wang R, Schoepf UJ, Meinel FG. Computed tomographic assessment of coronary artery disease: state-of-the-art imaging techniques. Radiol. Clin. North Am. 2015; 2:271–285.

3. Malhotra A, Rayner J, Williams TM, Prendergast B. Infective endocarditis: therapeutic options and indications for surgery. Curr Cardiol Rep. 2014;4:464.

4. Cahill TJ, Prendergast BD. Infective endocarditis. Lancet. 2016;387:882–893.

5. Murdoch DR, Corey GR, Hoen B et al. Clinical presentation, etiology, and outcome of infective endocarditis in the 21st century: the International Collaboration on Endocarditis-Prospective Cohort Study. Am. Heart J 2009;5:463–473.

6. Durante-Mangoni E, Bradley S, Selton-Suty C et al. Current features of infective endocarditis in elderly patients: results of the International Collaboration on Endocarditis Prospective Cohort Study. Am. Heart J 2008;19:2095–2103.

7. Prendergast BD. Diagnostic criteria and problems in infective endocarditis. Heart 2004;6:611–613.

8. Li JS, Sexton DJ, Mick N et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin. Infect. Dis. 2000; 4:633–638.

9. Habib G, Badano L, Tribouilloy C et al. Recommendations for the practice of echocardiography in infective endocarditis. Eur J Echocardiogr. 2010; 2:202–219.

10. Feuchtnner GM, Stolzmann P, Dichtl W et al. Multislice computed tomography in infective endocarditis: comparison with transesophageal echocardiography and intraoperative findings. J. Am. Coll. Cardiol. 2009; 5:436–444.

11. Grob A, Thuny F, Villacampa C et al. Cardiac multidetector computed tomography in infective endocarditis: a pictorial essay. Insights Imaging. 2014;5:559–570.

12. Gahide G, Bommart S, Demaria R et al. Preoperative evaluation in aortic endocarditis: findings on cardiac CT. American Journal of Roentgenology. 2010; 3:574–578.

13. Bruun NE, Habib G, Thuny F, Sogaard P. Cardiac imaging in infectious
endocarditis. European Heart Journal. 2014; 10:624–632.

14. Habib G, Lancellotti P, Antunes MJ, et al. 2015 ESC Guidelines for the management of infective endocarditis: The Task Force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). European Heart Journal. 2015; 44:3075–3128.

15. Rajiah P, Bolen MA. Cardiovascular MR imaging at 3 T: opportunities, challenges, and solutions. Radiographics. 2014; 6:1612–1635.

16. Millar BC, Prendergast BD, Alavi A, Moore JE. 18FDG-positron emission tomography (PET) has a role to play in the diagnosis and therapy of infective endocarditis and cardiac device infection. Int. J. Cardiol. 2013; 5:1724–1736.

17. Kouijzer IJE, Vos FJ, Janssen MJR, van Dijk APJ, Oyen WJG, Bleeker-Rovers CP. The value of 18F-FDG PET/CT in diagnosing infectious endocarditis. Eur. J. Nucl. Med. Mol. Imaging. 2013; 7:1102–1107.

18. Thuny F, Grisoli D, Cautela J, Riberi A, Raoult D, Habib G. Infective endocarditis: prevention, diagnosis, and management. Can J Cardiol. 2014; 9:1046–1057.

19. Gowda RM, Khan IA, Nair CK, Mehta NJ, Vasavada BC, Sacchi TJ. Cardiac papillary fibroelastoma: a comprehensive analysis of 725 cases. Am. Heart J. 2015; 3:1298–1303.

20. Carpenter JP, Price S, Rubens MB, Sheppard MN, Moat NE, Morgan A, et al. Aortic papillary fibroelastoma as an unusual cause of angina: insights from multimodality imaging. Circ Cardiovasc Imaging. 2011;2:191–193.

21. Aziz F, Baciewicz FA. Lambl's excrescences: review and recommendations. Tex Heart Inst J. 2007; 3:366–368.

22. Buckley O, Madan R, Kwong R, Rybicki FJ, Hunsaker A. Cardiac masses, part 2: key imaging features for diagnosis and surgical planning. American Journal of Roentgenology. 2011; 5:W842–851.

23. Iaizzo PA. Atlas of Human Cardiac Anatomy (Web only). University of Minnesota 2014. Available from: http://www.vhlab.umn.edu/atlas/aorta/aortic-valve/index.shtml . Accessed 4/16/2016

24. Graupner C, Vilacosta I, SanRomán J et al. Periannular extension of infective endocarditis. J. Am. Coll. Cardiol. 2002; 7:1204–1211.

25. David TE, Regesta T, Gavra G, Armstrong S, Maganti MD. Surgical treatment of paravalvular abscess: long-term results. Eur J Cardiothorac Surg. 2007; 1:43–48.

26. Davierwala PM, Binner C, Subramanian S et al. Double valve replacement and reconstruction of the intervalvular fibrous body in patients with active infective
endocarditis. Eur J Cardiothorac Surg. 2014; 1:146–152.

27. Wang A, Athan E, Pappas PA et al. Contemporary clinical profile and outcome of prosthetic valve endocarditis. JAMA. 2007; 12:1354–1361.

28. Fagman E, Perrotta S, Bech-Hanssen O et al. ECG-gated computed tomography: a new role for patients with suspected aortic prosthetic valve endocarditis. Eur Radiol. 2012; 11:2407–2414.

29. Salgado RA, Budde RPJ, Leiner T et al. Transcatheter aortic valve replacement: postoperative CT findings of Sapien and CoreValve transcatheter heart valves. Radiographics. 2014; 6:1517–1536.

30. Habets J, Tanis W, Reitsma JB et al. Are novel non-invasive imaging techniques needed in patients with suspected prosthetic heart valve endocarditis? A systematic review and meta-analysis. Eur Radiol. 2015; 7:2125–2133.

31. Saby L, Laas O, Habib G et al. Positron emission tomography/computed tomography for diagnosis of prosthetic valve endocarditis: increased valvular 18F-fluorodeoxyglucose uptake as a novel major criterion. J. Am. Coll. Cardiol. 2013; 23:2374–2382.

32. Pizzi MN, Roque A, Fernández-Hidalgo N et al. Improving the Diagnosis of Infective Endocarditis in Prosthetic Valves and Intracardiac Devices With 18F-Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography Angiography: Initial Results at an Infective Endocarditis Referral Center. Circulation. 2015; 12: 1113–1126.

33. Foote EA, Postier RG, Greenfield RA, Bronze MS. Infectious Aortitis. Curr Treat Options Cardiovasc Med. 2005; 2: 89–97.

34. 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; 2:123–138.

35. Gornik HL, Creager MA. Aortitis. Circulation. 2008; 23: 3039–3051.

36. Katabathina VS, Restrepo CS. Infectious and noninfectious aortitis: cross-sectional imaging findings. Semin. Ultrasound CT MR. 2012; 3:207–221.

37. Restrepo CS, Ocazionez D, Suri R, Vargas D. Aortitis: imaging spectrum of the infectious and inflammatory conditions of the aorta. Radiographics. 2011; 2:435–451.

38. Lopes RJ, Almeida J, Dias PJ, Pinho P, Maciel MJ. Infectious thoracic aortitis: a literature review. Clin Cardiol. 2009; 9:488–490.

39. Fillmore AJ, Valentine RJ. Surgical mortality in patients with infected aortic aneurysms. J. Am. Coll. Surg. 2003; 3:435–441.
40. Macedo TA, Stanson AW, Oderich GS, Johnson CM, Panneton JM, Tie ML. Infected aortic aneurysms: imaging findings. Radiology. 2004; 1:250–257.

41. Azizi L, Henon A, Belkacem A, Monnier-Cholley L, Tubiana J-M, Arrivé L. Infected aortic aneurysms: CT features. Abdom Imaging. 2004; 6:716–720.

42. Cartery C, Astudillo L, Deelchand A et al. Abdominal infectious aortitis caused by Streptococcus pneumoniae: a case report and literature review. Ann Vasc Surg. 2011; 2:266.e9–16.

43. Lin M-M, Cheng H-M. Images in cardiovascular medicine: tuberculous aortitis. Intern. Med. 2012; 15:1983–1985.

44. Pierret C, Tourtier J-P, Grand B, Bodaert G, Laurian C, de Kerangal X. Multiple tuberculous aneurysms of the aorta. J. Vasc. Surg. 2011; 6:1720–1722.

45. 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; 2:522–531.

46. 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; 4:439–444.

47. Molloy CB, Filer C, Ismail A. Mycobacterium tuberculosis as a cause of chronic periaortitis. Rheumatology (Oxford). 2005; 5:696–697.

48. Roberts WC, Barbin CM, Weissenborn MR, Ko JM, Henry AC. Syphilis as a Cause of Thoracic Aortic Aneurysm. Am. J. Cardiol. 2015; 8:1298–1303.

49. Kimura F, Satoh H, Sakai F et al. Computed tomographic findings of syphilitic aortitis. Cardiovasc Intervent Radiol. 2004; 2:179–181.

50. 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; 3:499–501.

51. Liu J, Yuan Q, Golamaully R, Gong T. Syphilitic aortitis complicated by multiple aortic aneurysms: findings of multidetector CT. Int J Cardiovasc Imaging. 2011; 5:695–699.

52. Tavora F, Burke A. Review of isolated ascending aortitis: differential diagnosis, including syphilitic, Takayasu's and giant cell aortitis. Pathology. 2006; 4:302–308.

53. Roberts WC, Ko JM, Vowels TJ. Natural history of syphilitic aortitis. Am. J. Cardiol. 2009; 11:1578–1587.

54. Hemkens LG, Bucher HC. HIV infection and cardiovascular disease. European Heart Journal. 2014; 21:1373–1381.
55. Monsuez J-J, Charniot J-C, Escaut L et al. HIV-associated vascular diseases: structural and functional changes, clinical implications. Int. J. Cardiol. 2009; 3:293–306.

56. Restrepo CS, Diethelm L, Lemos JA et al. Cardiovascular complications of human immunodeficiency virus infection. Radiographics. 2006; 1:213–231.

57. Bruggink JLM, Slart RHJA, Pol JA, Reijnen MMPJ, Zeebregts CJ. Current role of imaging in diagnosing aortic graft infections. Semin Vasc Surg. 2011; 4:182–190.

58. Orton DF, LeVeen RF, Saigh JA et al. Aortic prosthetic graft infections: radiologic manifestations and implications for management. Radiographics. 2000; 4:977–993.

59. Cernohorsky P, Reijnen MMPJ, Tielliu IFJ, van Sterkenburg SMM, van den Dungen JJAM, Zeebregts CJ. The relevance of aortic endograft prosthetic infection. J. Vasc. Surg. 2011; 2:327–333.

60. Chambers ST. Diagnosis and management of staphylococcal infections of vascular grafts and stents. Intern Med J. 2005; 35 Suppl 2:S72–8.

61. Murphy EH, Szeto WY, Herdrich BJ et al. The management of endograft infections following endovascular thoracic and abdominal aneurysm repair. J. Vasc. Surg. 2013; 5:1179–1185.

62. Kilic A, Arnaoutakis DJ, Reifsnyder T et al. Management of infected vascular grafts. Vasc Med. 2016; 1: 53-60

63. Perera GB, Fujitani RM, Kubaska SM. Aortic graft infection: update on management and treatment options. Vasc Endovascular Surg. 2006; 1 :1–10.

64. Raman SP, Kamaya A, Federle M, Fishman EK. Aortoenteric fistulas: spectrum of CT findings. Abdom Imaging. 2013; 2:367–375.

65. Thakor AS, Tanner J, Ong SJ et al. Radiological Evaluation of Abdominal Endovascular Aortic Aneurysm Repair. Can Assoc Radiol J. 2015; 3:277–290.

66. Mita T, Arita T, Matsunaga N et al. Complications of endovascular repair for thoracic and abdominal aortic aneurysm: an imaging spectrum. Radiographics. 2000; 5:1263–1278.

67. Fukuchi K, Ishida Y, Higashi M et al. Detection of aortic graft infection by fluorodeoxyglucose positron emission tomography: comparison with computed tomographic findings. J. Vasc. Surg. 2005; 5:919–925.

68. Fiorani P, Speziale F, Rizzo L et al. Detection of aortic graft infection with leukocytes labeled with technetium 99m-hexametazime. J. Vasc. Surg. 1993; 1:87–96

69. Shahidi S, Eskil A, Lundof E, Klaerke A, Jensen BS. Detection of abdominal aortic graft infection: comparison of magnetic resonance imaging and indium-
labeled white blood cell scanning. Ann Vasc Surg. 2007; 5:586–592.

70. Keidar Z, Engel A, Hoffman A, Israel O, Nitecki S. Prosthetic vascular graft infection: the role of 18F-FDG PET/CT. J. Nucl. Med. 2007; 8:1230–1236.

71. Ahmed FZ, James J, Memmott MJ, Arumugam P. Radionuclide Imaging of Cardiovascular Infection. Cardiol Clin. 2016;1:149–165.

72. Saleem BR, Pol RA, Slart RHJA, Reijnen MMPJ, Zeebregts CJ. 18F-Fluorodeoxyglucose positron emission tomography/CT scanning in diagnosing vascular prosthetic graft infection. Biomed Res Int. 2014; 2014:471971.
Table 1. Aortic infection and their common causative organisms.
Figures

Figure 1

ECG-gated cardiac CT in a 22-year-old intravenous drug user with a bicuspid aortic valve, fever and a new murmur.

Coronal (A) MPR of the aortic valve demonstrates a perforation (double arrow) in the posterior leaflet of the bicuspid aortic valve. 3-chamber MPR (B) demonstrates periaortic soft tissue thickening posterior to the aortic root (arrow), extending inferiorly towards the anterior leaflet of the mitral valve (curved arrow) consistent with spread of infection into the intervalvular fibrous body. The patient underwent aortic and mitral valve replacement, and the infected valve tissue grew S. aureus.

LA = left atrium; LV = left ventricle; Ao = ascending aorta; RA = right atrium; RV = right ventricle
Fig 1B
Figure 2

65-year-old man with a previous history of bioprosthetic aortic valve replacement and ascending aorta homograft repair presents with fever and sepsis.

Axial (A) ECG-gated CT angiogram demonstrates a large aortic root abscess (dashed line) with a small pseudoaneurysm (curved arrow) arising from the right sinus of Valsalva, adjacent to the origin of the right coronary artery (*).

Coronal MPR (B) demonstrates a large vegetation on the non-coronary cusp of the bioprosthetic valve (arrow), with further delineation of the aortic root abscess (dashed line). The patient underwent a repeat aortic valve and ascending aorta homograft replacement. Tissue culture of the removed bioprosthetic valve grew coagulase-negative *Staphylococcus* and *E.faecalis*.

*Ao*=ascending aorta; *LA*=left atrium; *RA*=right atrium; *PA*= pulmonary artery; *RVOT*=right ventricular outflow tract.
Figure 3

78-year-old man who initially presented with chest pain, fever and *S. aureus* septicemia.

Axial thoracic CT non-contrast (A) demonstrates mural high attenuation in the descending thoracic aorta (*) with adjacent consolidation in the left lower lobe (arrow).

18F-FDG PET/CT (B) shows marked corresponding increased FDG uptake in the aortic wall (*). The patient was diagnosed with an infected intramural hematoma, and was treated with antibiotics.

Follow-up sagittal fat-saturated T1 MRI post contrast (C) shows development of a large saccular pseudoaneurysm (curved arrow), with persistent adjacent inflammatory tissue (*). The patient subsequently underwent surgical repair.

*Ao=Descending thoracic aorta*
Fig 3B
Figure 4
55-year-old man with a history of chronic myeloid leukemia who presented with fever and chest pain.

Axial CT angiogram of the thoracic aorta (A) demonstrates crescentic mural thickening (arrow) of the mid descending thoracic aorta.

T1 double inversion recovery (DIR) MRI pre-contrast (B) demonstrates crescentic mural high signal in the same location in the descending thoracic aorta. T1 post contrast (C) demonstrates crescentic mural enhancement in the corresponding location, consistent with an aortitis. The patient had new cryptococcal sepsis, with no other focus of infection, and a diagnosis of cryptococcal aortitis was made. The patient expired despite systemic antifungal treatment.

Ao=descending thoracic aorta
Fig 4C
Figure 5
46-year-old man with no significant medical history with abdominal pain and low-grade fever. Initial CT abdomen (A) demonstrates ill-defined fat stranding (arrow) surrounding the proximal abdominal aorta at the level of the celiac artery origin. The patient’s pain worsened, and a follow-up CT abdomen was performed 3 days later (B), which showed an interval increase in the severity of peri-aortic fat stranding (arrow). Follow-up CT abdomen one week later (C) shows development of a saccular aortic aneurysm (*) with worsening periaortic fat stranding (arrow). The patient underwent surgical excision of the mycotic aneurysm with bypass grafting, and the excised aortic wall grew S.aureus. Despite surgical intervention, the patient expired in the early post-operative period.

Ao=abdominal aorta
Figure 6

75-year-old man with a history of chronic kidney disease and type 2 diabetes mellitus presents with back pain and fever. Axial (A) and coronal (B) post contrast CT abdomen demonstrates aneurysmal dilatation of the infrarenal aorta with multiple pockets of gas within the aortic wall (arrow) and peri-aortic fat stranding (*), consistent with emphysematous aortitis. The patient underwent urgent excision of the mycotic aneurysm with placement of an axillo-bifemoral bypass graft. The resected aortic wall grew *Clostridium perfringens*, a gram-positive, gas-forming, anaerobic bacillus.

* Ao=abdominal aorta
Figure 7

15-year-old girl with cough, night sweats and back pain.

CT abdomen coronal maximum intensity projection (MIP) (A) demonstrates multifocal stenoses of the aortic hiatus, the suprarenal and infrarenal abdominal aorta (arrowheads).

Axial CT abdomen (B) of the infrarenal abdominal aorta demonstrates severe luminal narrowing (*) with circumferential periaortic soft tissue (arrow).

The patient’s sputum cultures grew *M.tuberculosis*, with the aortic appearances consistent with tuberculous aortitis.
Fig 7B
A 62-year-old woman with a history of previous open abdominal aortic aneurysm
graft repair presents with abdominal pain and emesis of brown particulate matter was
referred for CT angiogram. Post contrast CT abdomen A & B) demonstrates a fistula
(arrow) between the aorta (Ao) and duodenum (D), with layering of high-density
material in the duodenum (curved arrow). The patient was operated on emergently,
but unfortunately expired. Post-mortem culture of the abdominal aorta graft grew
gram-negative bacilli, likely *E. coli*. 
Fig 8A
65-year-old man who presented 6 months post thoracic endovascular aneurysm repair (TEVAR) with fever and hemoptysis.

Axial non-contrast CT Thorax (A) demonstrates new air within the excluded aneurysm sac (arrow), immediately posterior to the esophagus (*). There is adjacent left lower lobe consolidation with an associated pleural effusion. Axial (B) post-contrast CT thorax demonstrates a peripherally enhancing per-aortic fluid collection (curved arrow) at the inferior aspect of the stent graft consistent with an abscess. Barium swallow (C) confirms the presence of an aorto-esophageal fistula (arrows). The overall diagnosis is consistent with a thoracic stent graft infection with an aorto-esophageal fistula, likely caused by the adjacent left lower lobe pneumonia. The patient expired despite surgical intervention, with no definite organism identified.

$Ao=descending\ thoracic\ aorta;\ Eo=esophagus$
Fig 9B
Figure 10

A 70 year-old-man with chest pain underwent a CT pulmonary angiogram for suspected pulmonary embolus. Coronal (A) and sagittal (B) CT demonstrates a large, fusiform, peripherally calcified aneurysm (double arrow) of the thoracic aorta (Ao), abutting the sternum anteriorly. The patient underwent surgical repair, with pathology demonstrated classical appearances of luetic aortitis, caused by the spirochete *Treponema pallidum*.

*R*A=right atrium; *RV*= right ventricle; *LV*=left ventricle
Fig 10B