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Aortic Fistulas: Pathophysiologic Features, Imaging Findings, and Diagnostic Pitfalls

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Abbreviations: AAF = aortoatrial fistula, ABF = aortobronchial fistula, ACF = aortocaval fistula, AEF = aortoenteric fistula, AEsoF = aorto-esophageal fistula, AoF = aortic fistula, APF = aortopulmonary fistula, IVC = inferior vena cava, PA = pulmonary artery, RBC = red blood cell, WBC = white blood cell

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Identify direct and indirect signs of various AoFs at CT and consider potential mimics in the differential diagnosis.
- Describe the pathophysiologic mechanisms, natural history, and current approach to management of AoFs.
- Discuss the limitations of imaging in the diagnosis of early aortic fistulization and perigraft infection and the current algorithms for management.
- See www.rsna.org/education/search/RG.

Fistulas between the aorta and surrounding organs are extremely rare but can be fatal if they are not identified and treated promptly. Most of these fistulas are associated with a history of trauma or vascular intervention. However, spontaneous aortic fistulas (AoFs) can develop in patients with weakened vasculature, which can be due to advanced atherosclerotic disease, collagen-vascular disease, vasculitides, and/or hematogenous infections. The clinical features of AoFs are often nonspecific, with patients presenting with bleeding manifestations, back or abdominal pain, fever, and shock. Confirmation with invasive endoscopy is often impractical in the acute setting. Imaging plays an important role in the management of AoFs, and multiphasic multidetector CT angiography is the initial imaging examination of choice. Obvious signs of AoF include intravenous contrast material extravasation into the fistulizing hollow organ, tract visualization, and aortic graft migration into the adjacent structure. However, nonspecific indirect signs such as loss of fat planes and ectopic foci of gas are seen more commonly. These indirect signs can be confused with other entities such as infection and postoperative changes. Management may involve complex and staged surgical procedures, depending on the patient's clinical status, site of the fistula, presence of infection, and anticipated tissue friability. As endovascular interventions become more common, radiologists will need to have a high index of suspicion for this entity in patients who have a history of aneurysms, vascular repair, or trauma and present with bleeding.

Online supplemental material and the slide presentation from the RSNA Annual Meeting are available for this article.

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Introduction

Pathologic communications between the vascular high-flow aortic lumen and the surrounding organs are extremely rare and require expeditious diagnosis and management in the face of imminent catastrophic bleeds (1,2). These aortic fistulas (AoFs) are typically a result of trauma or prior vascular interventions, but they may also occur de novo in weakened vessels, as in cases of accelerated atherosclerotic disease or blood-borne infection (1). Rare causes include vasculitides, collagen-vascular disorders, malignancy, and radiation-induced changes (2). In the absence of trauma or history of surgery, the diagnosis is often harder to make and can be inadvertently missed. There has been an increase in the incidence of AoFs in recent years, paralleling the increased burden of atherosclerotic disease and consequent increased use of endovascular graft aortic repairs (1). Affected patients typically present with intermittent herald bleeds before exsanguination, with or without other nonspecific or even misleading local symptoms (3). Given the rarity of AoFs, nonspecific clinical manifestations, and risks associated with invasive

TEACHING POINTS

- Multidetector CT angiography with multiplanar reconstruction is the initial examination of choice for diagnosing AoFs.
- When CT angiographic findings are equivocal in the differentiation of postoperative changes and infections, scintigraphy with technetium 99m (^{99m}Tc) hexamethylpropyleneamine oxime-labeled white blood cells (WBCs) or indium 111 (¹¹¹In) oxime-labeled WBCs can be used. Fluorine 18-fluorodeoxyglucose (FDG) PET/CT also can be considered for use in certain patients and provides superior resolution.
- Direct signs of AoFs are rarely visualized and include visualization of the fistula tract, extravasation of intravenous contrast medium into the fistulizing organ or oral contrast medium coursing into the paraprostatic space, and aortic graft migration into the adjacent fistulizing structure.
- Indirect signs are often the only available evidence to suggest a diagnosis of AoF. These ancillary imaging features include abnormalities of the aortic wall such as aortitis, aneurysm or pseudoaneurysm formation, aortic mural thrombus, a malpositioned graft, or discontinuous plaque. Irregularities of the interface between the aorta and the fistulizing entity, such as loss of fat planes and periaortic foci of gas, also can be seen. The fistulizing organ itself may have abnormally thickened or tethered walls. Last, the presence of air in the aortic lumen, any surrounding fluid collection, or free hemorrhage also warrants closer inspection.
- Various normal anatomic variants, postoperative appearances or complications, and pathologic processes can have imaging features that are similar to those of AoFs.

confirmatory procedures, imaging plays a pivotal role in establishing a diagnosis.

CT angiography is the mainstay for diagnosis owing to the quick acquisition, widespread availability, and superior spatiotemporal resolution (1,4). When a direct imaging sign such as contrast material extravasation from the aorta into the bowel, or, conversely, extravasation of orally administered gastrointestinal contrast material into the periaortic space; visualization of the tract; or graft migration into a communicating organ is present, a diagnosis can be established with high certainty (5). However, in practice, the diagnosis of AoF often requires piecing together sometimes subtle imaging findings such as increased perigraft soft tissue, fat stranding, bowel wall thickening, aortic mural hematoma, and ectopic air in the aorta or suspected tract (1,5,6).

Adjunctive use of scintigraphic techniques to identify infection or slow intermittent bleeds can be very helpful. An unsuspecting radiologist can easily miss or misdiagnose a potential fistula amid postoperative changes or in the presence of perigraft infection. In this pictorial review, we describe the clinical and radiologic features of the spectrum of AoFs, highlighting the distinguishing characteristics that differentiate these topographically distributed entities from confounding anatomic, postoperative, and patho-

logic mimics and focusing on imaging findings at CT angiography.

Pathogenesis of AoFs

Atherosclerotic disease leading to intimomedial fat deposition is the most common cause of loss of integrity of an otherwise intact vessel wall. With the high pulsatile intravascular pressures, this phenomenon gradually leads to aneurysmal growth. The weakened aortic wall allows abnormal radial transmission of pulsatile pressure to the surrounding soft tissues. This abnormal force causes pressure necrosis and adhesive granulation tissue between the aorta and a periaortic hollow or solid organ, resulting in a fistula (7,8). A true aneurysm occurs owing to the weakening of an otherwise intact vessel wall. This aneurysm contains all of the layers of the arterial wall, whereas a false aneurysm, or pseudoaneurysm, does not contain any wall layers and is more prone to sudden rupture instead of controlled fistula development.

Infection, postoperative inflammation, and foreign bodies (ie, sutures, particularly if they are too tight, or stents) act as catalysts for subsequent fistulization. Other less common risk factors such as penetrating atherosclerotic ulcers, vasculitides, collagen-vascular disorders, mycotic aneurysms, and radiation have a common substrate—that is, inflamed and weakened perivascular soft tissue that allows adhesion and fistulization. Rarely, AoFs can be congenital (9,10). An AoF is typically referred to as a primary fistula if it occurs in a vessel that has had no prior intervention and is typically associated with an atherosclerotic aneurysm.

If an AoF occurs in the setting of a prior aortic intervention and/or aortic graft placement, it is referred to as a secondary fistula. The pathophysiology of secondary fistulas is multifactorial and related to foreign body reaction to the graft, graft kinking, superimposed infection, endoleak coil placement, and endotension (11,12). Secondary fistulas, aortoenteric fistulas (AEFs) in particular, are more commonly seen in clinical practice, given the higher incidence of abdominal aortic aneurysms and their repair (1). Literature comparing the incidences of fistulas related to open repair versus endovascularly placed grafts is scant. One of the larger studies by Chopra et al (13) involving 50 patients suggests a higher incidence of fistulas involving open aortobifemoral bypass compared with the incidences of fistulas related to endovascular repairs and tube grafts.

Three morphologic subtypes of secondary AEF have been described in the surgical literature: (a) fistula between the bowel and the anastomotic suture line (ie, between the native aorta and the graft), (b) fistula between the bowel and

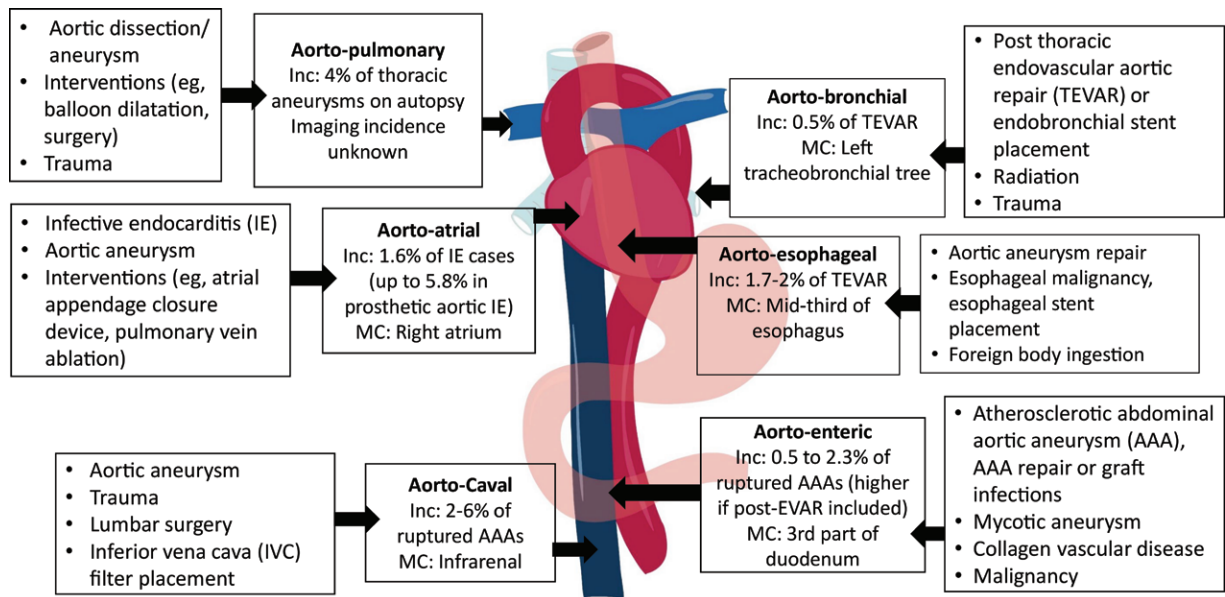


Figure 1. Diagram illustrates the incidence (*Inc*) of and risk factors for the most commonly seen aortic fistulas (AoFs). EVAR = endovascular aortic repair, MC = most common.

a suture-line pseudoaneurysm, and (c) fistula between the bowel and the graft body lining itself, which is also called a paraprosthetic fistula or graft erosion (14,15). No significant difference in clinical outcome has been reported among these subtypes (13).

Trauma is another rare etiologic factor for AoFs. Usually, penetrating trauma due to a gunshot or stab wound is the main perpetrator (see slides 29 and 30 in the online slide presentation.) Less commonly, iatrogenic injury during a vascular procedure or lumbar disk surgery also has been implicated (16). Blunt trauma is another event that can lead to vascular injury, owing to a whip-lash mechanism or the vessel being compressed between osseous structures. The latter mechanism, related to vessel compression, is seen particularly in the aortic isthmus, which can get trapped between the manubrium, first rib, and clavicular heads (17).

Spectrum of AoFs

Broadly, there are six entities that comprise the majority of clinically relevant AoFs in relation to the aorta: aortoatrial fistula (AAF), aortopulmonary fistula (APF), aortobronchial fistula (ABF), aorto-esophageal fistula (AEsoF), AEF, and aortocaval fistula (ACF). Among these, abdominal AEFs and ACFs are the most common. In addition to the great vessel itself, its larger branches can be involved in fistula formation. However, this is beyond the scope of this article. The major risk factors and incidences associated with individual AoFs, derived from the contemporary literature, are illustrated in Figure 1 (7,16–24).

Radiologic Imaging

Various radiologic modalities are available to evaluate AoFs. Each of these modalities has advantages and disadvantages.

CT Angiography

Multidetector CT angiography with multiplanar reconstruction is the initial examination of choice for diagnosing AoFs. CT angiography enables quick acquisition of images with high spatial resolution. Volume-rendered three-dimensional images provide surgeons and interventional radiologists with an anatomic overview for surgical and endovascular planning. The routine CT angiography protocol for aortic evaluation at our institution involves a noncontrast phase to assess for hemorrhage or intramural hematoma. The noncontrast phase is followed by image acquisition in the arterial phase and an optional delayed phase.

Image acquisition is performed by using automated bolus tracking with the region of interest placed in the descending aorta. Ninety to 150 mL of nonionic intravenous iodinated contrast medium (350 mg/mL) is injected at a rate of 3–5 mL/sec and followed by a 30–50-mL saline chaser to clear the venous system of contrast material.

CT angiography is performed on a 64-section multidetector CT system, with a collimation of 0.625 mm. Images are reconstructed in all three planes by using a 0.6–3.0-mm section thickness. In patients with a history of stent placement, a delayed phase image is obtained 120 seconds after contrast medium administration to assess for endoleaks. This protocol is summarized in Table 1.

A virtual noncontrast sequence can be reconstructed from the arterial phase of dual-energy CT. This examination can be particularly useful in cases in which the need is realized retrospectively. However, virtual noncontrast images are noisier than true noncontrast images, they can have significantly higher attenuation, and calcium can be partially removed from these images. However, most of these images are found to be of diagnostic quality (25,26).

At nongated imaging, cardiac motion can mimic intimal flaps or wall irregularity at the aortic root and ascending aorta and be misinterpreted as true aortic wall disease. The clinical background, presence of streaks extending outside of the lumen, absence of secondary ancillary findings that suggest acute aortic syndrome, and blurred margins at other sites provide clues to the true nature of the observation (27). Suboptimal acquisition in the setting of high clinical suspicion may require adjunctive use of advanced cardiac gating techniques. This is often the case when the indication requires interrogation of the aortic root and ascending aorta.

Oral contrast material is rarely used in emergent settings at most centers today, and, if used, it may obscure abnormal contrast material extravasation into the bowel. When an AoF is suspected, the use of conventional oral contrast material (containing barium) is not recommended, as it can obscure the extravasation of vascular contrast material into the bowel, which is an important sign of AoF (5). However, if the patient is stable, neutral oral contrast agents can be used in combination with intravascular contrast medium to enhance the ability to detect such vascular-to-bowel contrast medium extravasation.

Conventional Angiography

Although conventional angiography is rarely the initial imaging modality used in cases of AoF, it facilitates better spatial resolution than does CT angiography. Conventional angiography may be performed to delineate the arterial anatomy better, to detect bleeding rates as slow as 0.5 mL/min, and for dynamic assessment of vascular pressure gradients (28). It is particularly useful in patients who have previously undergone graft repair but is less reliable for diagnosing primary AEFs (9). Conventional angiography has the added advantage of enabling simultaneous treatment with stent placement and embolization (5).

MR Angiography

MR angiography has limited use in emergent settings but can be a viable imaging option for patients with renal insufficiency who cannot tolerate iodinated contrast medium. Use of phase-contrast

MR angiography to quantify flow through a fistula has been reported (29). The superior soft-tissue contrast has been shown to be helpful (68%–85% sensitivity, 97%–100% specificity) in detecting graft infections in cases that are equivocal with CT angiography (30). The nonionizing nature of MRI, with superior contrast resolution, is also optimal for volumetric assessment of the graft lumen at aneurysm follow-up, especially in young patients (31). However, if there are concerns regarding the imaging of areas with vascular or graft-related calcifications, metallic fixation sites, or the outer metal skeleton of the graft, adjunctive CT is essential.

Aortic endografts are modular stent grafts made of polyester (Dacron; DuPont) or expanded polytetrafluoroethylene (Teflon; Chemours) fabric supported by a metallic mesh skeleton that is usually composed of a nickel titanium alloy called nitinol. Knowledge of the endograft type used is important when considering post-endovascular repair MRI assessment. While nitinol grafts do not cause significant susceptibility artifacts that compromise MRI assessment of the perigraft soft tissues or graft lumen, ferromagnetic materials such as stainless steel and cobalt-chromium preclude MRI assessment. Of the eight endografts that are currently available, the following six have nitinol-based skeletons: Endurant (Medtronic), Excluder (Gore), Incraft (Cordis), Aorfix (Lombard Medical), and Alto and Ovation (Endologix). The AFX2 endograft (Endologix) has a cobalt-chromium skeleton, and the Zenith device (Cook Medical) has a stainless steel skeleton.

While the presence of susceptibility artifacts depends heavily on the material of the metallic construct, all endografts cause some degree of radiofrequency shielding or caging that can impair visualization of the graft lumen, regardless of the material. If needed, these radiofrequency caging artifacts can be reduced by increasing the radiofrequency power of the excitation pulse. Radiofrequency-related heating or dislodging torque forces are not of clinical concern with regard to any of the described graft materials (32–34).

Nuclear Imaging

Superimposed vascular graft infections are a major source of morbidity and mortality from septicemia and, more relevant to the subject of this review, their presence significantly increases the likelihood of AoF. When CT angiographic findings are equivocal in the differentiation of postoperative changes and infections, scintigraphy with technetium 99m (^{99m}Tc) hexamethylpropyleneamine oxime-labeled white blood cells (WBCs) or indium 111 (¹¹¹In) oxime-labeled WBCs can be used. Fluorine 18-fluorodeoxyglucose (FDG) PET/CT also can be

Table 1: CT Angiographic Protocol for Evaluation of Aorta at Authors' Institution

| Parameter | Description or Value |
|---|--|
| Acquisition phases | Noncontrast, arterial, and (optionally) delayed at 120 sec |
| Region of interest for automated bolus tracking | Descending aorta |
| Contrast medium volume | 90–150 mL |
| Collimation | 0.625 mm |
| Reconstructed section thickness | 0.6–3.0 mm |

considered for use in certain patients and provides superior resolution (30). Radiolabeled WBC studies are used because of their added specificity for infection (53%–100% sensitivity, 60%–100% specificity) (30,35). ^{111}In -labeled WBC radiotracer is preferred when evaluating fistulas of the abdominal aorta owing to the physiologic gastrointestinal or urinary excretion of $^{99\text{m}}\text{Tc}$. FDG PET/CT is a higher-resolution study and is gaining significant recognition, with a sensitivity and specificity of 95% and 80%, respectively, reported in patients with vascular graft infections in a relatively recent meta-analysis (36). FDG PET/CT is best suited for patients at least 3–4 months after surgery before antibiotics have been initiated, as postoperative inflammatory changes can cause false-positive results and use of antibiotics can cause false-negative results (5,37).

$^{99\text{m}}\text{Tc}$ -tagged red blood cell (RBC) scanning is best suited for identifying slow, intermittent, and occult-to-moderate bleeding owing to its excellent sensitivity, with bleeding as slow as 0.05–0.10 mL/min reportedly detected (5,28,38). However, this examination is not the optimal choice for patients who present with massive bleeding that is suspicious for AoF owing to the prolonged imaging time needed for this study. Rather, RBC scintigraphy studies are more suited for detecting the intermittent bleeds seen in cases involving a tenuous fistula tract or flow limitation due to interposed laminated plaque. The added sensitivity of tagged RBC scanning, as compared with CT angiography, cannot be therapeutically leveraged since patients who have positive RBC study results may not have a detectable correlate at angiography (28). In the majority of cases, the diagnosis can be made by using properly performed multiphase CT angiography with meticulous interpretation of findings.

Ultrasonography

US may have a role in the evaluation of medically unstable patients and patients with allergies to contrast medium, but visualization of the deep-seated aorta is usually poor and obscured by overlying gas and tissue (1,30). However, there have been reports of AEFs being diagnosed with

use of point-of-care US. Reported cases describe heteroechoic masslike collections abutting both the aorta and the bowel, with pouching of flow from the aorta toward the collection and/or internal air foci (39,40). Turbulent aortocaval communication and arterialized venous flow may be visualized directly (41). Contrast-enhanced US can also depict a tract when contrast medium is seen flowing from the aorta into the fistulizing structure (42). Transesophageal echocardiography has utility in the evaluation of flow through the aortic arch, aortic root, and proximal descending fistulas and can be performed intraoperatively or at the bedside of unstable patients (43). Approaches to imaging AoFs in various clinical settings are summarized in Table 2.

Principal Imaging Features

The imaging signs of AoFs can be divided into direct and indirect signs, irrespective of the fistula site. Direct signs of AoFs are rarely visualized (6,8,44) and include visualization of the fistula tract, extravasation of intravenous contrast medium into the fistulizing organ or oral contrast medium coursing into the paraprostatic space, and aortic graft migration into the adjacent fistulizing structure. Indirect signs are often the only available evidence to suggest a diagnosis of AoF. These ancillary imaging features include abnormalities of the aortic wall such as aortitis, aneurysm or pseudoaneurysm formation, aortic mural thrombus, a malpositioned graft, or discontinuous plaque. Irregularities of the interface between the aorta and the fistulizing entity, such as loss of fat planes and periaortic foci of gas, also can be seen. The fistulizing organ itself may have abnormally thickened or tethered walls. Last, the presence of air in the aortic lumen, any surrounding fluid collection, or free hemorrhage also warrants closer inspection. Specific features of each of the different types of AoFs are discussed in the following sections.

Aortoatrial Fistula

Epidemiologic and Clinical Features

AAFs are usually the result of endocarditis, which is often associated with paravalvular abscesses,

Table 2: Imaging Approaches Used in Various AoF-related Clinical Scenarios

| Clinical Scenario | Imaging Approach |
|--|--|
| Large-volume bleeding with high clinical suspicion for AoF (primary or secondary) | CTA usually suffices; US and bedside TEE may be useful; CTA may be beneficial in unstable patients—especially in the setting of intrathoracic fistulas |
| Intermittent bleeding with recent aneurysm repair (secondary) | CTA (first line), tagged RBC scanning (second line), tagged WBC scanning for infection evaluation PET/CT at 3–4 months or longer after surgery and if antibiotics have not been started |
| Intermittent bleeding with suspected spontaneous AoF (primary) or remote aneurysm repair (secondary) | CTA (first line), tagged RBC scanning (second line), infection evaluation with PET/CT and MRI to provide better resolution of infection |

Note.—AoF = aortic fistula, CTA = CT angiography, RBC = red blood cell, TEE = transesophageal echocardiography, WBC = white blood cell.

and are more to likely develop in patients with a prosthetic valve. They have been reported in 1.6% of patients with infectious endocarditis, with the prevalence increasing to 5.8% in cases of prosthetic aortic valve infection (19). AAFs can result from complications of aortic aneurysms and dissection, ruptured sinus of Valsalva aneurysms, and trauma. Interventions such as aortic root or valve surgery, atrial septal defect closure, coronary artery bypass graft placement, angiography, pulmonary vein ablation, and insertion of Watchman (Boston Scientific) devices are also risk factors. Congenital forms of AAFs are less common, manifesting as aortoatrial tunnels or coronary-cameral fistulas, and can be associated with other anomalies, most commonly ostium secundum atrial septal defect (22).

Symptoms occur owing to volume overload secondary to shunting of the blood from the aorta to the atria. Manifesting symptoms include dyspnea on exertion, dry cough, palpitations, fatigue, chest pain, and even cardiogenic shock. A new continuous murmur in patients with known aortic disease should prompt workup for an AAF (10,18). Symptoms may worsen gradually over time as the volume overload and cardiac remodeling progress; however, patients can also remain asymptomatic (45,46). Complications of AAFs include heart failure, pulmonary edema, congestive hepatopathy, atrial fibrillation, and secondary atrioventricular valvular insufficiency (10,18).

Imaging Features

In cases of acquired AAFs, the right atrium is more commonly affected than the left owing to the anatomic locations (10,18). A fistula may be visualized directly owing to contrast material leakage into the low-pressure right or left atrium from the aorta (Fig 2). Alternatively, another direct finding would be a tract arising from the true or false lumen of a dissected aorta (Fig 3,

Movie 1). Indirect features that are suggestive of an underlying AAF include adhesions and fibrosis within the soft tissues interposed between the aorta and the atrium, changes related to underlying endocarditis, and aortic root dilatation. In stable patients who can tolerate a longer examination, advanced techniques such as four-dimensional flow MRI can also be utilized for confirming and characterizing aorto-atrial fistulas, mirroring its use for other aortic fistulas (29). Transesophageal echocardiography can be used for confirmation of AAF and flow quantification, especially in the setting of unstable patients who require bedside or intraoperative assessment (43).

Aortopulmonary Fistula

Epidemiologic and Clinical Features

APFs are an uncommon intrathoracic diagnosis. They can be congenital or acquired. Their association with congenital heart disease, including repair of coarctation of the aorta, is well known. Causes of acquired APFs include rupture of a thoracic aortic aneurysm into the pulmonary artery (PA) as a complication of aortic dissection and procedures of the aortic or pulmonic valve such as the Bentall procedure, trauma, and sepsis (47). An autopsy study in 1924 (48) revealed a prevalence of 4% in cases of thoracic aneurysms, but a subsequent study in 1958 (49) found not even a single fistula among 505 cases of aortic dissection. Data on the imaging incidence of APFs are limited, and available literature suggests that they are more commonly associated with acute dissections (50). Balloon dilation of pulmonary arteries also has been reported to cause APFs in patients with a history of PA stent placement or suture lines from previous Ross or arterial switch procedures (51). APFs lead to the development of left-to-right shunt, acute pulmonary edema, and right heart failure.

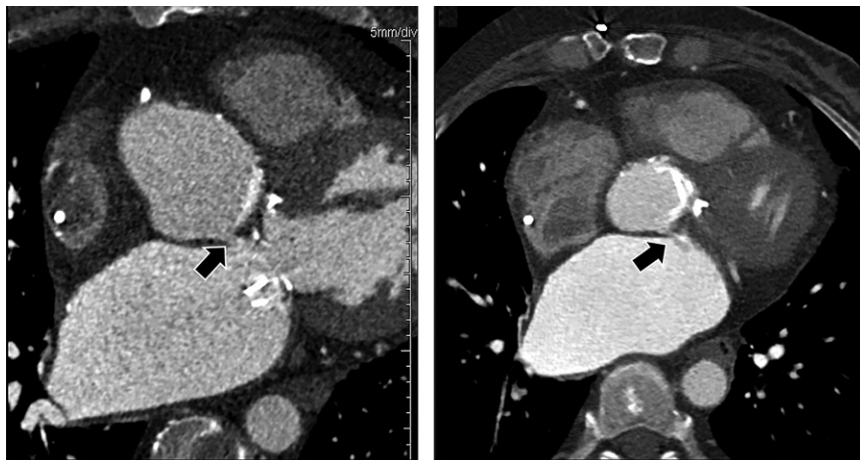


Figure 2. Aortoatrial fistula (AAF) in a 68-year-old woman with a history of aortic and mitral valve replacements who presented with recent-onset chest pain. Oblique (a) and axial (b) CT angiograms show contrast material (arrow) opacifying a communication between the aortic root and left atrium. The patient was subsequently scheduled for elective repair.



Figure 3. AAF in a 55-year-old man with previous coronary artery bypass graft placement who presented with chest pain (Movie 1). Axial (a) and coronal (b) CT angiograms show a type A aortic dissection involving the thoracic aorta (arrows) and aortic root (arrowhead in b), resulting in a fistula between the coronary sinus and right atrium seen on the sagittal image (arrow in c). During surgery, the right atrium was found to have a thrill on palpation, which was suggestive of a fistula. After dissection and identification of the extent of the tear to just above the annulus in the noncoronary sinus, the aorta was further resected down to the sinotubular junction, where a 1-cm communication between the rupture in the noncoronary sinus and the right atrium was confirmed.

Imaging Features

Continuous (systolic and diastolic) turbulent high-velocity flow between the right ventricular outflow tract–PA and aorta can be detected with transthoracic echocardiography (52) (Fig 4). High oxygen saturation in the PA and oxygen step-up between the right atrium and the PA, as demonstrated during cardiac catheterization, are confirmatory (Fig 4b, Movie 2). Cardiac CT helps in the detection of pseudoaneurysm formation and evaluation of coronary atherosclerosis in adults preparing for cardiac intervention.

Aortobronchial Fistula

Epidemiologic and Clinical Features

ABFs are rare entities. They are usually the consequence of surgical intervention involving the descending thoracic aorta or aerodigestive

tract, as a complication of radiation therapy for lung or head and neck malignancies. In adults, this usually means the repair of aneurysms or pseudoaneurysms, dissections, or traumatic rupture, and interventions such as endobronchial stent placement to relieve bronchial obstruction, endobronchial blocker placement for preferential bronchial aeration, and esophagectomy. Childhood surgeries such as repair of tetralogy of Fallot, patent ductus arteriosus, and coarctation of the aorta also can lead to the development of ABFs well into adulthood. Rarely, ABFs are due to vasculitides such as Takayasu arteritis (53).

Development of ABF has been shown to occur as early as 2 weeks and as late as 25 years after the interventions just mentioned (7,44). In a 2010 study of 1113 patients who underwent thoracic endovascular aneurysm repair (20), six of these patients later developed ABFs, with five of

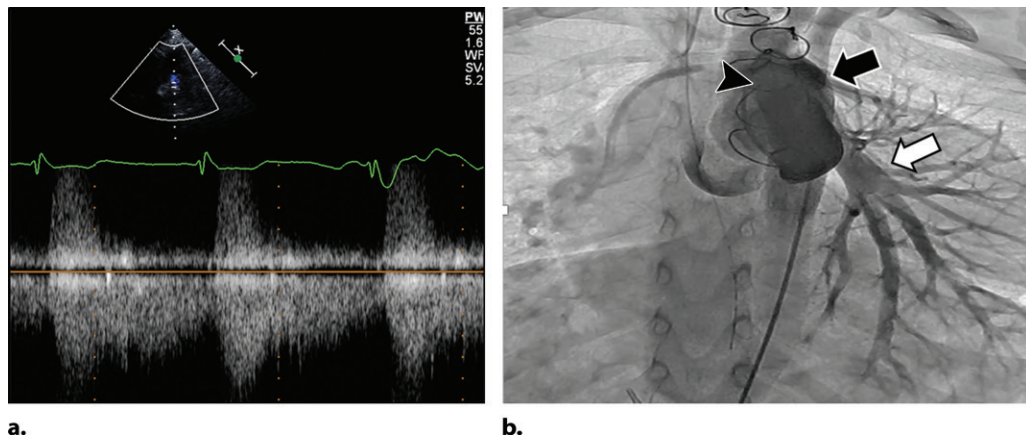


Figure 4. Aortopulmonary fistula (APF) in a 7-year-old girl with a history of double-outlet right ventricle and subpulmonary ventricular septal defect, after an arterial switch procedure involving the LeCompte maneuver and ventricular septal defect closure. The patient underwent balloon dilation of the left pulmonary artery (PA) for neopulmonary stenosis. At follow-up, a new continuous murmur was noted. **(a)** Doppler US echocardiogram shows continuous antegrade flow with systolic accentuation in the APF. **(b)** Image from cardiac catheterization shows a fistula (black arrow) between the ascending aorta (arrowhead) and left PA (white arrow) (Movie 2). (Case courtesy of Christian Pizarro, MD, Nemours Cardiac Center, Division of Pediatric Cardiothoracic Surgery, Wilmington, Del.)

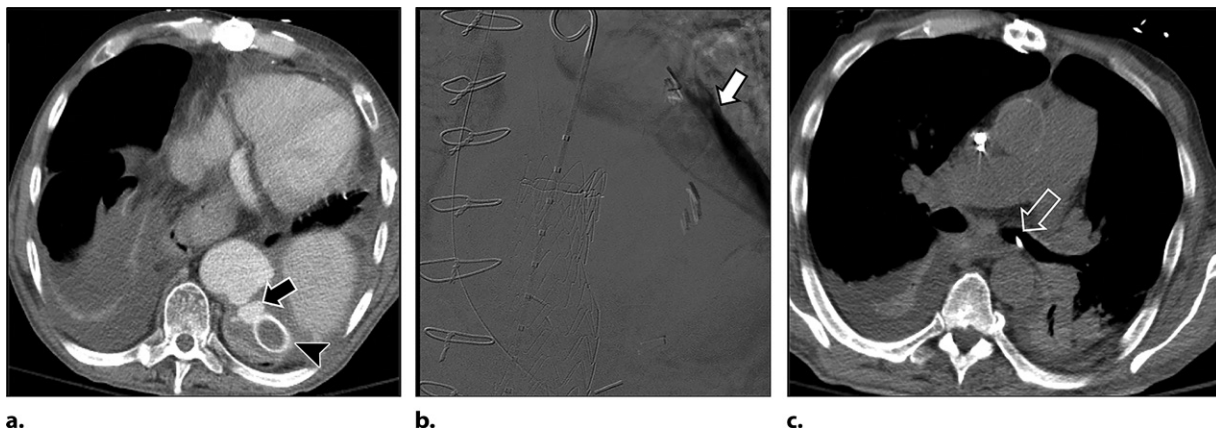


Figure 5. Aortobronchial fistula (ABF) in a 61-year-old man with hemoptysis, a history of heart transplant, and left ventricular assist device placement. **(a)** Axial CT image shows a pseudoaneurysm of the descending thoracic aorta at the anastomosis site (arrow) adjacent to the thrombosed outflow remnant graft (arrowhead). **(b)** Delayed image of the catheter angiogram demonstrates extravasation of contrast material at the time of aortic stent graft placement, representing an underlying communication (arrow). **(c)** Axial noncontrast CT image shows an endoblocker (arrow) placed in the left mainstem bronchus for treatment.

them having concomitant AEsoFs. The actual incidence of ABF is likely underestimated, as more than 30% are diagnosed only at autopsy (54).

Hemoptysis of varying degrees is the typical manifestation. Recurrent and massive bleeds should increase suspicion. Other symptoms include chest or back pain or dyspnea (7,44).

Imaging Features

ABFs with communications involving the left tracheobronchial tree are more common owing to the relative anatomic proximity (6,7,44). As with all AoFs, with ABFs, ancillary imaging features are more frequent. A common example is a focal area of ground-glass opacity (indicating pulmonary hemorrhage) in close vicinity or contiguity

with a potentially fistulizing acute aortic syndrome (eg, aneurysm, penetrating ulcer, or dissection) (10). The site of the ulcer or aneurysmal bulge should be carefully interrogated to localize the fistula (Fig 5). In a patient who has a history of surgical repair with graft placement, signs of graft infection—like thickening of the surrounding wall, perigraft fluid collection, air within the wall, loss of normal fat planes, and/or stent migration also should raise suspicion for a fistula (10).

Aortoesophageal Fistula

Epidemiologic and Clinical Features

AEsoFs most commonly occur as a result of aortic aneurysm rupture or repair, dissection,

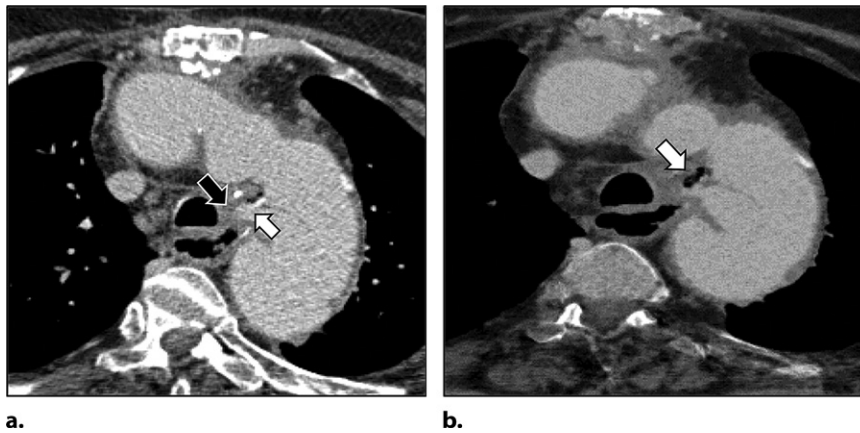


Figure 6. Aorto-esophageal fistula (AEOF) in a 70-year-old man with a prolonged tracheostomy for laryngeal cancer who presented with acute bright red hematemesis. Axial CT angiograms show loss of the outline of the medial wall of the aneurysmal aortic arch, with a nipple-like outpouching tethered to the esophagus (black arrow in a) and an adjacent focus of air (white arrow). The fistula was confirmed at surgery.

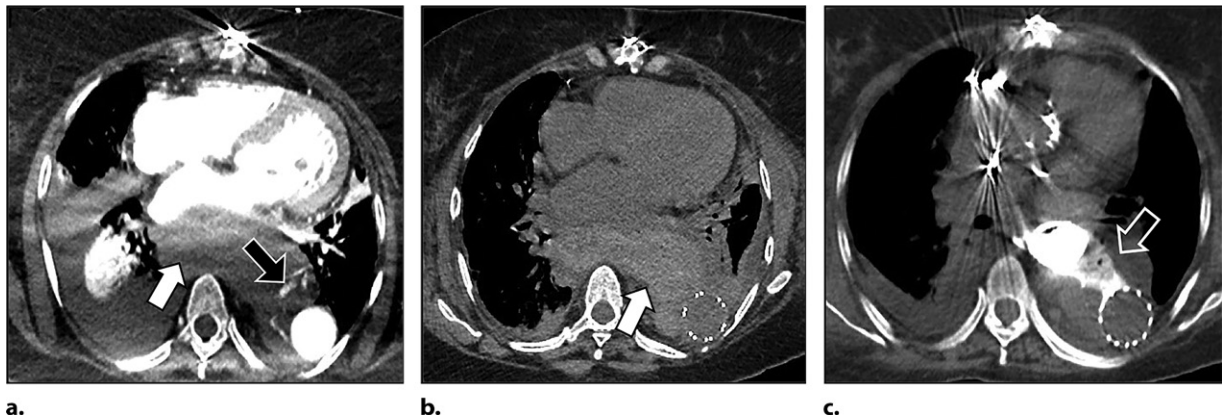


Figure 7. AEOF in a patient with an aortic dissection (not shown) complicated by rupture with extravasation of oral contrast material. (a, b) Axial contrast-enhanced (a) and noncontrast (b) CT images show the extravasation anteriorly (black arrow in a) and a mediastinal hematoma indenting the left atrium (white arrow). (c) Axial postoperative CT image after aortic stent-graft repair and esophageal stent placement shows an esophageal leak and probable AEOF outlined by extravasated oral contrast material extending to the aortic stent (arrow).

invasive esophageal malignancies, or foreign body ingestion. They have been reported to occur at frequencies ranging from 0.5% to 1.7% (21). Given increasing interventions and use of grafts, the incidence of AEOFs also is rising (21). The Chiari triad, consisting of midthoracic pain or dysphagia and sentinel hemorrhage, followed by potentially fatal arterial hematemesis leading to exsanguination after a symptom-free period, is the classically described manifestation of AEOFs (55,56). Affected patients can also have other forms of gastrointestinal bleeding, back pain, fever, and sepsis.

Imaging Features

AEOFs are seen most commonly in the middle-third portion of the esophagus, closer to the region of the change in aortic curvature. Signs of an AEOF include extravasation of intravenous contrast material into the gastrointestinal tract and stent migration (1,57,58). These may include a teatlike outpouching from the aorta toward the esophagus, as seen in Figure 6, or vice versa.

More commonly, extraluminal air and mediastinal collections, esophageal wall thickening, or aortic wall discontinuity or dilatation is seen with loss of normal fat planes (Fig 7).

Aortoenteric Fistula

Epidemiologic and Clinical Features

An AEF is an abnormal communication between the intestinal tract and the aorta. Primary AEFs occur de novo, usually in a diseased atherosclerotic vessel, and are significantly rarer, with a reported prevalence of 0.04%–0.07% (23). Secondary AEFs have a reported prevalence of 0.5%–2.3%, making them the most common type of aortic fistula, and they are usually associated with repaired atherosclerotic aneurysms (23). Foreign material-like grafts or sutures and superimposed gut flora infections act as catalysts. Fistulas at sites other than the duodenum are often related to more unusual causes such as retroperitoneal radiation, ballistic injuries, endoleak coils, ingested foreign bodies, pancreatic necrosis, or uncommon variants

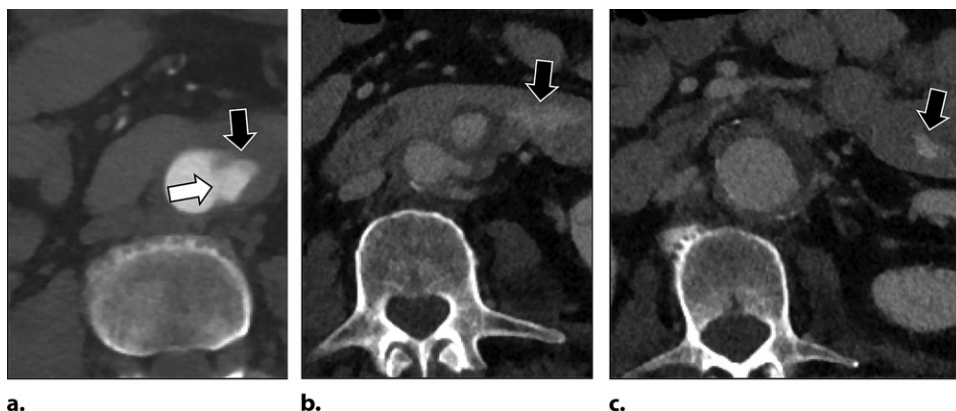


Figure 8. Aortoduodenal fistula in a 72-year-old man with a history of abdominal aortic aneurysm repair who presented with acute-onset severe midabdominal pain, gastrointestinal bleeding, and syncope. Axial CT angiograms of the abdomen and pelvis show extravasation of arterial contrast material into the duodenum and proximal jejunum (black arrow), confirming the clinical suspicion for an aortoenteric fistula (AEF) to the third portion of the duodenum (white arrow in a). The patient underwent urgent repair with explantation of an aortobiliac graft, cadaveric aortic homograft placement, and aortoduodenal fistula repair.

of diverticular disease (59,60). The classic triad for primary AEFs first described by Sir Astley Cooper has been described as an upper gastrointestinal bleed, abdominal pain, and a pulsatile abdominal mass. However, this triad is only reported in 10%–25% of patients (4,23,61). Other manifestations of AEFs include varying forms of gastrointestinal bleeding, sepsis, back or abdominal pain, fever, and shock (23,62). A “herald bleed,” which is smaller and more intermittent, often precedes exsanguination by as little as a few hours to as much as a few months (4). It is believed to occur as a result of mucosal ulceration followed by temporary tamponade by clotting and bowel loops (23). Hematemesis and melena, followed by a herald bleed and shock, have been found to occur more commonly. Hematochezia and bright red blood from the rectum have been reported to be the least common (15).

Imaging Features

AEFs most commonly involve the third part of the duodenum, followed by the fourth part, jejunum, ileum, and large bowel (63,64). The proximity of the juxtarenal and infrarenal portions of the aorta to the retroperitoneally anchored duodenum makes this site the most vulnerable.

The sensitivity and specificity of CT for AEFs (40%–90% and 33%–100%, respectively) vary widely and are consistently higher for secondary AEFs (90%) (1). Direct signs of AEF are extravasation of intravenous contrast material into the gastrointestinal tract or stent migration into the gastrointestinal tract (1,57,58) (Fig 8). These signs are extremely rare, but they are specific for a fistula (1). More common signs include air in the aneurysm sac or periaortic area and focal

bowel wall thickening (1) (Fig 9). Indirect signs include previously described aortic and periaortic abnormalities such as pseudoaneurysm, abnormal soft tissue, and loss of planes between the aorta and bowel (Fig 10).

Aortocaval Fistula

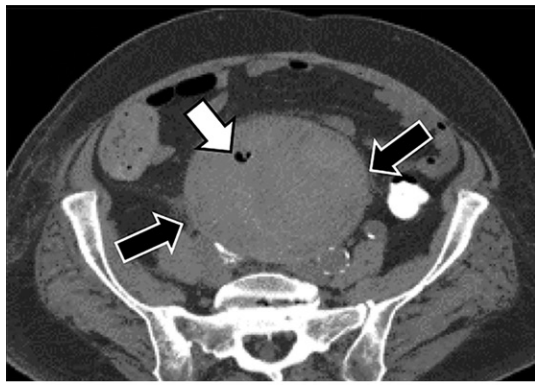
Epidemiologic and Clinical Features

ACFs are known to occur at a rate of 2%–7%, but prevalences as high as 10% also have been reported (24,65,66). They can result from ruptured aortic aneurysms, aortitis, or the use of inferior vena cava (IVC) filters. High-velocity penetrating trauma or significant spinal trauma with thoracolumbar translation also can lead to ACF formation. Less common causes include spinal surgery and IVC dissection (67,68).

ACFs have varied signs and symptoms and may manifest as acute abdominal pain or low back pain with shortness of breath and a pulsatile abdominal mass with a bruit (69). If the fistula has been gradually expanding over time, the patient may have adapted to the new hemodynamics and eventually present with signs and symptoms of high-output cardiac failure. Shock is a less frequent manifestation, as the blood remains intravascular (24,70).

Imaging Features

ACFs occur most commonly in association with the infrarenal aorta. The most obvious imaging finding of an ACF is early contrast enhancement of the IVC, with simultaneous enhancement of the aorta and IVC during the arterial phase (Fig 11). The fat planes between the two vascular structures are lost, and the IVC may show inden-



a.



b.

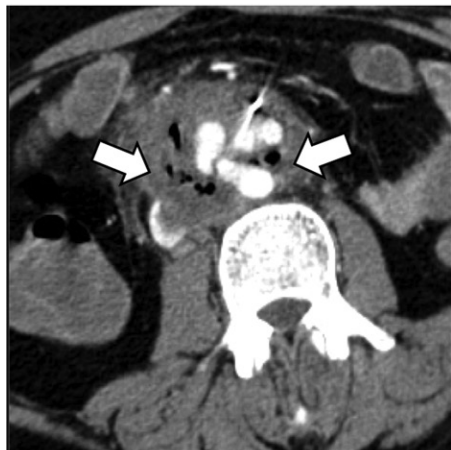


c.

Figure 9. Aortoenteric (aortoileal) fistula (AEF) in a 77-year-old man with a history of right-sided aortobifemoral bypass graft placement who presented with abdominal pain. (a, b) Axial CT images show a large hyperattenuating perigraft collection (black arrows in a), with internal foci of gas (white arrow) extending down to the right groin. (c) Axial CT image shows that there is also loss of the normal fat plane between the collection and the adjacent ileal loop (arrows), raising suspicion for a fistula. During surgery for removal of the infected aortic graft and placement of an axillofemoral bypass graft, the AEF was confirmed.



a.



b.

Figure 10. AEF in a 30-year-old man with metastatic nonseminomatous germ cell cancer who was found to have hematemesis, melena, and hematochezia with a palpable abdominal mass at clinical examination. He was also found to have a large aortocaval lymph node infiltrating into the duodenum and supplied by the superior and inferior mesenteric arteries that required coil placement in the inferior mesenteric artery. He returned 1 month later with massive hematemesis and melena. (a, b) Coronal maximum intensity projection (a) and axial (b) images from a CT angiogram show an aortic pseudoaneurysm just above the bifurcation, communicating with the small bowel (black arrow in a), with periaortic soft tissue and fluid (white arrows). The fistula was confirmed at digital subtraction angiography, when contrast material extravasation into the bowel loops was seen.

tation at the site of the fistula. A retroperitoneal hematoma may or may not be present (70). A fistula leading to the IVC from the false lumen of a ruptured dissecting aortic aneurysm may be seen (Fig 12). Owing to the vascular overload, the IVC and pelvic veins are often dilated; this should prompt thorough investigation for an ACF.

Pitfalls

Various normal anatomic variants, postoperative appearances or complications, and pathologic processes can have imaging features that are similar to those of AoFs. These mimics of AoFs are described in the following sections.

Anatomic Mimics

Normal variants of the aortic contour can simulate an aneurysm or pseudoaneurysm. In the scenario of high clinical suspicion for a fistula, these entities

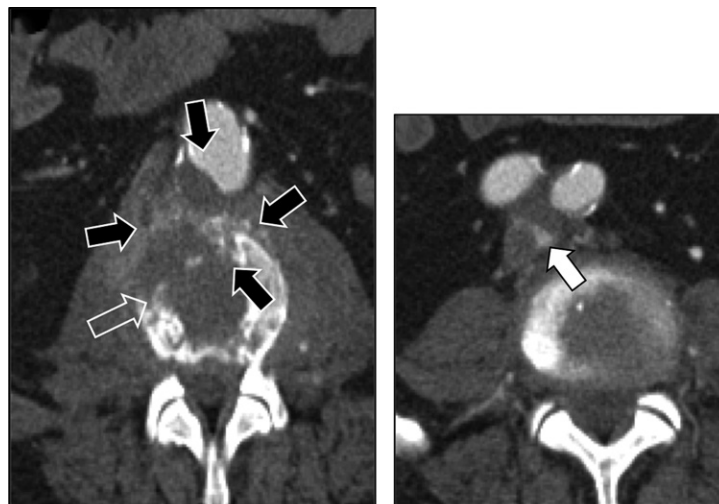


Figure 11. Aortocaval fistula (ACF) in a 60-year-old man who underwent imaging for bacteremic sepsis. Axial CT images show phlegmonous extension of infection into the aorta (black arrows in **a**) and early dense enhancement of the inferior vena cava (IVC) matching the enhancement of the aorta (arrow in **b**). These findings suggest aortocaval fistulization secondary to adjacent L3-L4 discitis-osteomyelitis (open arrow in **a**).

can mislead the interpreting radiologist. Such anatomic variants include ductus diverticulum, aortic spindle and nipple, and Kommerell diverticulum at the origin of an aberrant right subclavian artery. Thus, familiarity with the normal appearance of these benign variants is important.

A ductus diverticulum can have an appearance similar to that of a pseudoaneurysm, given that it is a focal bulge. However, its typical location at the anterior undersurface of the isthmic region and obtuse angle with the aorta are assuring that it is a benign variant. An aortic spindle is a similar bulge in the first part of the descending aorta (27,71). *Aortic nipple* refers to the prominence of the third intercostal artery origin from the aorta; it is seen as an abnormal beaking of the lateral aortic contour just distal to the isthmus. Rarely, a Kommerell diverticulum may be a confounding finding (72). Repair of a Kommerell diverticulum is associated with AEsoF formation (73).

Postoperative Mimics

Accurate diagnosis of fistulas and correct differentiation of them from perigraft infection in the immediate postoperative setting can be challenging. In addition, expected postoperative changes can have imaging features that overlap with those of fistulas, adding to the diagnostic dilemma.

The time that has elapsed since surgery can be helpful. Perigraft air is rare after a week following surgery but may persist until 4–7 weeks after surgery. Similarly, perigraft soft tissue, fluid, or hematoma can be seen in the immediate postoperative period. However, after 3 months, one should

consider the possibility of infection (5,74,75). Although blood cultures can provide conclusive evidence, they are often negative, and clinical signs and laboratory biomarkers for infection are less reliable owing to the generalized inflammatory response in the postoperative setting (5).

If any direct imaging features of the fistula are seen, establishing a diagnosis is more straightforward; however, this is a rare scenario. Focal bowel wall thickening and irregularity, ectopic air foci, and pseudoaneurysm are more likely to be related to a fistula than an infection (76,77). In addition to CT, tagged RBC scanning or conventional angiography may be required to detect subtle bleeds. Tagged WBC scans can be obtained when infection cannot be conclusively differentiated from a fistula (Fig 13) (5). A simplified approach to imaging in the postoperative period is illustrated in Figure 14 (1,5,78).

Surgical material and hemostatic packing agents such as bioabsorbable sponge or gauze can have foci of air within them, simulating perigraft air. However, in these cases, these materials tend to be uniform, do not demonstrate enhancement, do not change position across studies, and are higher in attenuation (–104 to –458 HU) than is pure air. These processes typically resolve in 7–14 days (78). Felt pledgets can mimic hyperattenuating intravenous contrast material or simulate a pseudoaneurysm. However, their typical location along the aortic wall, abutting the PA, and appearance during the noncontrast phase facilitate correct identification (78). The surgical notes also can be a valuable aid.

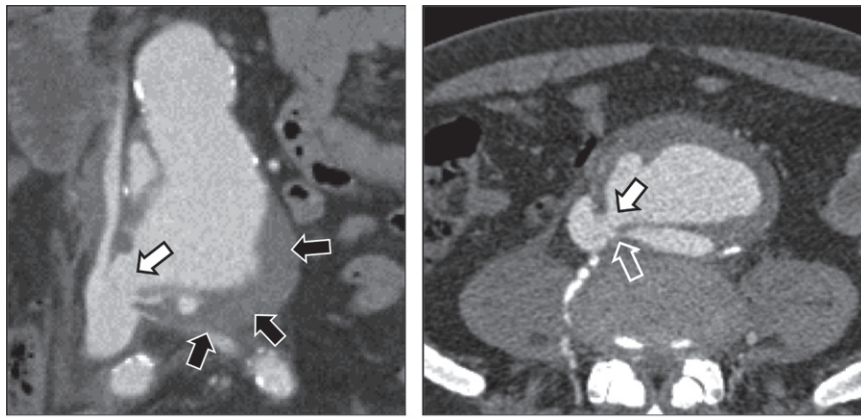


Figure 12. ACF in a 58-year-old man who became hypotensive during workup for suspected renal colic. Coronal (a) and axial (b) CT angiograms show a saccular infrarenal aortic aneurysm (black arrows in a) with a chronic dissection flap (open arrow in b) and fistulization with the IVC (white arrow). The patient underwent aortobiliac bypass surgery involving polyester graft placement and fistula repair.

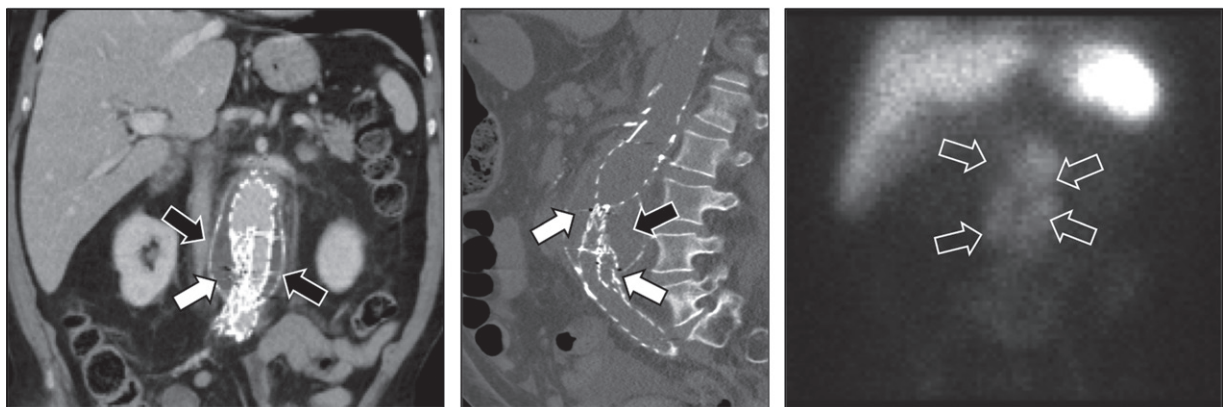


Figure 13. Infection mimicking a fistula in a 67-year-old man who presented with acute abdominal pain 4 years after endovascular aortic repair. (a, b) Coronal (a) and sagittal (b) CT images show an expanding infrarenal aortic aneurysm (black arrows) with tiny foci of air (white arrows). It was unclear whether this was an infection or an underlying fistula. (c) Tagged ¹¹¹In–white blood cell (WBC) scan shows increased uptake in the perigraft region (arrows), consistent with infection. The patient underwent excisional débridement of the aorta, with implantation of antibiotic beads and staged graft explantation.

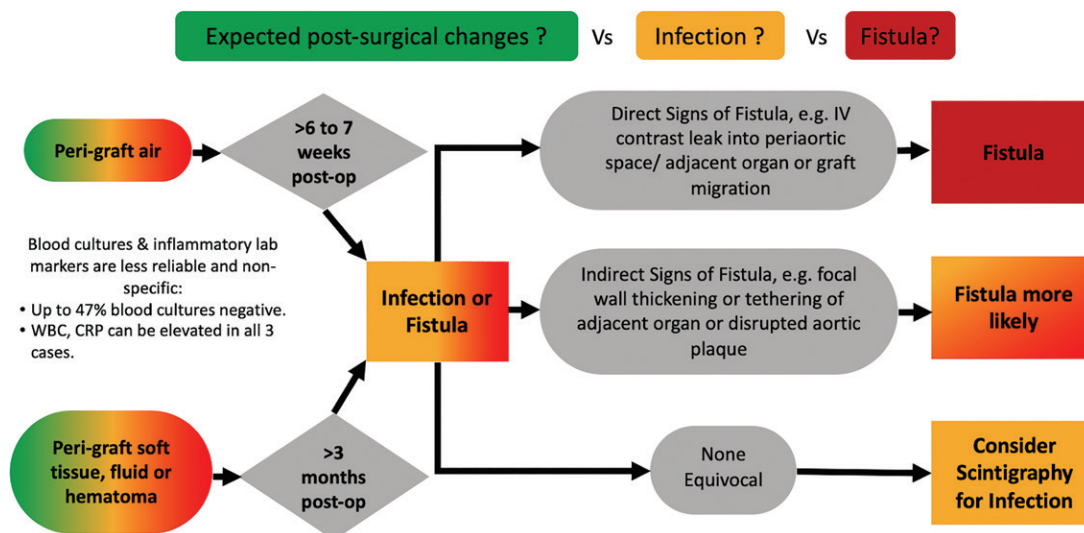


Figure 14. Diagram of a simplified approach to imaging in the postoperative period to differentiate expected changes, infection, and fistula formation. CRP = C-reactive protein, IV = intravenous.

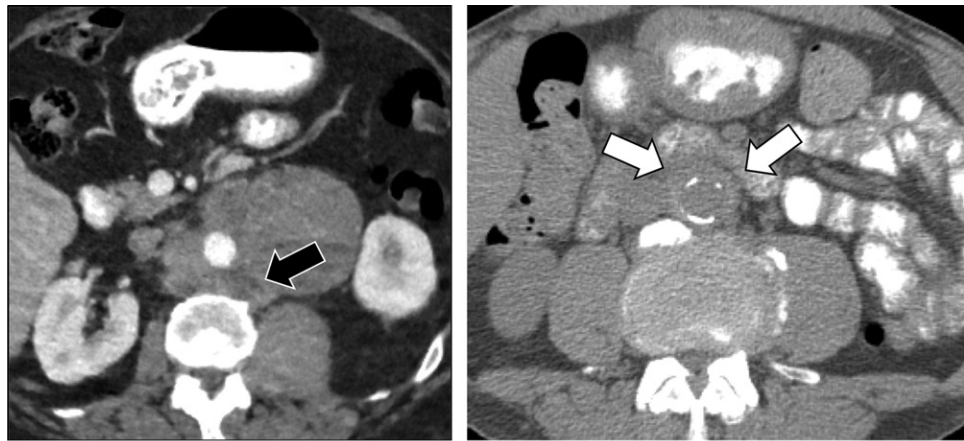


Figure 15. Axial contrast-enhanced CT images in a 56-year-old woman (**a**) and a 58-year-old man (**b**) show a soft-tissue periaortic density (arrow in **a**) and rind (arrows in **b**), which increase the distance between the aorta and spine owing to bulky retroperitoneal lymphadenopathy (**a**) and retroperitoneal fibrosis (**b**).

Pathologic Mimics

Other pathologic processes such as retroperitoneal fibrosis, lymphadenopathy, and inflammatory conditions encasing the aorta can have imaging findings similar to those of AoFs. Retroperitoneal fibrosis appears as variably enhancing soft-tissue plaques encasing the aorta and other retroperitoneal organs. It can sometimes be asymmetric, focal, and infiltrating, mimicking an inflammatory process. These plaques can demonstrate variable degrees of enhancement as well. Adenopathy can be a similar confounding entity and widen the gap between the aorta and spine (Fig 15) (1). Pathologic entities such as Erdheim-Chester disease are also known to manifest with a thick inflammatory periaortic rind (79). However, in these cases, sites of involvement other than the periaortic region and the lack of ancillary imaging features such as wall tethering and focal ectopic gas can help to differentiate these diseases from a fistula (1).

Infected (mycotic) aneurysms can contribute to the formation of fistulas. Hence, the features of these two entities often overlap. Features of an infected aneurysm include vessel-wall irregularity, fat stranding, and enhancement. The presence of air foci can be a feature of an anaerobic infection as well as a fistula. However, contiguity with an adjacent vessel, presence of a tract, or extravasation of contrast material should raise suspicion for an underlying fistula. In addition, subtle differences in features such as the shape of the mycotic aneurysm, being typically saccular, and the site, more commonly involving the suprarenal and thoracic aorta, can be helpful hints. Rarely, the adjacent vertebral bodies can also show osteomyelitis-discitis. Infected aneurysms have a higher risk of rupture and should be closely monitored (1,80).

Treatment

AoFs can have extremely grim outcomes. Accurate and prompt diagnosis and treatment are key to patient survival. AEFs are conventionally treated with open repair. It is imperative to limit aortic inflow into the operative field by clamping the supraceliac or suprarenal aorta with use of direct exposure or an endovascular balloon, which is not without associated cold ischemia time and associated morbidity (30). The traditional reference standard for open repair is staged extra-anatomic bypass surgery; excision of the infected aortic graft, leaving an oversewn aortic stump; or repair of the fistulizing organ. The bypass may be temporary or definitive—in other words, reconstruction of the native aorta may or may not be performed later, depending on the tissue friability and associated infection (21,30,81,82). Open repairs are associated with a higher rate of complications such as recurrent fistula formation, reinfection of the aortic stump, and stump blowout (30). The aortic stump or primary anastomosis usually can be protected from refistulization by interposing a generous flap of soft tissue or omentum (30). Thus, this area requires particular attention at follow-up imaging.

Given that staged repairs have only a marginal benefit, with high complication rates, there has been a gradual shift toward primary in situ reconstruction whereby the native infected or fistulizing aorta or graft is resected to healthy margins, and vascular allografts (cryopreserved or fresh) or rifampin-embedded synthetic grafts are refashioned to reconstruct the resected segment at the outset. However, this may not be feasible in cases of severe or resistant infections (eg, *Pseudomonas* or drug-resistant bacteria).

Early endovascular repair as part of definitive endovascular or staged open repair of AoFs has been shown to facilitate improved short-term morbidity and reduced mortality (14,30). However, a higher reintervention rate has been observed for patients who are treated primarily with endovascular repair. Thus, these patients require close follow-up (83). The American Heart Association (AHA) currently recommends the use of endovascular treatment as only a bridging therapy before definitive open repair for AEsoFs (30,56). Similarly, the AHA recommends endovascular repair for AEsoFs and ABFs in patients who cannot tolerate open repair or as a temporizing measure until definitive open surgery (30). Small AAFs and ABFs can be managed conservatively, and there are case reports of these fistulas being primarily treated by using endovascular approaches such as device closure, embolization coil placement, stent or balloon placement, and even cyanoacrylate procedures (7,44,46,84–87).

Conclusion

The early diagnosis of AoFs is paramount to patient survival and avoiding a grim outcome. As endovascular procedures gain more popularity, fistulas should be included in the differential diagnosis for patients with bleeding, especially those who have undergone a prior aortic intervention. Radiologists must be familiar with the various imaging features of AoFs and the associated confounding entities.

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References

- Vu QD, Menias CO, Bhalla S, Peterson C, Wang LL, Balfé DM. Aortoenteric fistulas: CT features and potential mimics. *RadioGraphics* 2009;29(1):197–209.
- Sipe A, McWilliams SR, Saling L, Raptis C, Mellnick V, Bhalla S. The red connection: a review of aortic and arterial fistulae with an emphasis on CT findings. *Emerg Radiol* 2017;24(1):73–80.
- Simon T, Feller E. Diverse presentation of secondary aortoenteric fistulae. *Case Rep Med* 2011;2011:406730. doi:10.1155/2011/406730.
- Lin TC, Tsai CL, Chang YT, Hu SY. Primary aortoduodenal fistula associated with abdominal aortic aneurysm with presentation of gastrointestinal bleeding: a case report. *BMC Cardiovasc Disord* 2018;18(1):113.
- Tagowski M, Vieweg H, Wissgott C, Andresen R. Aortoenteric fistula as a complication of open reconstruction and endovascular repair of abdominal aorta. *Radiol Res Pract* 2014;2014:383159.
- Fontana M, Tonelli R, Gozzi F, et al. An uncommon cause of hemoptysis: aortobronchial fistula. *Multidiscip Respir Med* 2018;13(1):25.
- Picichè M, De Paulis R, Fabbri A, Chiariello L. Postoperative aortic fistulas into the airways: etiology, pathogenesis, presentation, diagnosis, and management. *Ann Thorac Surg* 2003;75(6):1998–2006.
- Picichè M, De Paulis R, Chiariello L. Unusual origin and fistulization of an aortic pseudoaneurysm: “off-pump” surgical repair. *Ann Thorac Surg* 1999;68(4):1406–1407.
- Lemos DW, Raffetto JD, Moore TC, Menzoian JO. Primary aortoduodenal fistula: a case report and review of the literature. *J Vasc Surg* 2003;37(3):686–689.
- Jainandunsing JS, Linnemann R, Maessen J, et al. Aorto-atrial fistula formation and therapy. *J Thorac Dis* 2019;11(3):1016–1021.
- Bertges DJ, Villella ER, Makaroun MS. Aortoenteric fistula due to endoleak coil embolization after endovascular AAA repair. *J Endovasc Ther* 2003;10(1):130–135.
- Chenu C, Marcheix B, Barcelo C, Rousseau H. Aortoenteric fistula after endovascular abdominal aortic aneurysm repair: case report and review. *Eur J Vasc Endovasc Surg* 2009;37(4):401–406.
- Chopra A, Cieciora L, Modrall JG, Valentine RJ, Chung J. Twenty-year experience with aorto-enteric fistula repair: gastrointestinal complications predict mortality. *J Am Coll Surg* 2017;225(1):9–18.
- Kakkos SK, Bicknell CD, Tsolakis IA, Bergqvist D; Hellenic Co-operative Group on Aortic Surgery. Editor’s choice: management of secondary aorto-enteric and other abdominal frterio-enteric Fistulas—review and pooled data analysis. *Eur J Vasc Endovasc Surg* 2016;52(6):770–786.
- Pipinos II, Carr JA, Haithcock BE, Anagnostopoulos PV, Dossa CD, Reddy DJ. Secondary aortoenteric fistula. *Ann Vasc Surg* 2000;14(6):688–696.
- Spencer TA, Smyth SH, Wittich G, Hunter GC. Delayed presentation of traumatic aortocaval fistula: a report of two cases and a review of the associated compensatory hemodynamic and structural changes. *J Vasc Surg* 2006;43(4):836–840.
- Luckraz H, Kitchlu S, Youhana A. Aortoesophageal fistula as a late complication of aortic transection. *J Thorac Cardiovasc Surg* 2005;129(2):458–459.
- Fierro EA, Sikachi RR, Agrawal A, Verma I, Ojrzanowski M, Sahni S. Aorto-atrial fistulas: a contemporary review. *Cardiol Rev* 2018;26(3):137–144.
- Anguera I, Miro JM, Vilacosta I, et al. Aorto-cavitary fistulous tract formation in infective endocarditis: clinical and echocardiographic features of 76 cases and risk factors for mortality. *Eur Heart J* 2005;26(3):288–297.
- Chiesa R, Melissano G, Marone EM, Marrocco-Trischitta MM, Kahlberg A. Aorto-oesophageal and aortobronchial fistulae following thoracic endovascular aortic repair: a national survey. *Eur J Vasc Endovasc Surg* 2010;39(3):273–279.
- Kieffer E, Chiche L, Gomes D. Aortoesophageal fistula: value of in situ aortic allograft replacement. *Ann Surg* 2003;238(2):283–290.
- Gajjar T, Voleti C, Matta R, Iyer R, Dash PK, Desai N. Aorta-right atrial tunnel: clinical presentation, diagnostic criteria, and surgical options. *J Thorac Cardiovasc Surg* 2005;130(5):1287–1292.
- Xiromeritis K, Dalainas I, Stamatakos M, Filis K. Aortoenteric fistulae: present-day management. *Int Surg* 2011;96(3):266–273.
- Leigh-Smith S, Smith RC. Aorto caval fistula: the “bursting heart syndrome.” *J Accid Emerg Med* 2000;17(3):223–225.
- Lehti L, Söderberg M, Höglund P, Nyman U, Gottsäter A, Wassélius J. Reliability of virtual non-contrast computed tomography angiography: comparing it with the real deal. *Acta Radiol Open* 2018;7(7-8):2058460118790115.
- Vlahos I, Chung R, Nair A, Morgan R. Dual-energy CT: vascular applications. *AJR Am J Roentgenol* 2012;199(5 suppl):S87–S97.
- Patel NR, Dick E, Batrick N, Jenkins M, Kashef E. Pearls and pitfalls in imaging of blunt traumatic thoracic aortic injury: a pictorial review. *Br J Radiol* 2018;91(1089):20180130.
- Wortman JR, Landman W, Fulwadhwa UP, Viscomi SG, Sordickson AD. CT angiography for acute gastrointestinal bleeding: what the radiologist needs to know. *Br J Radiol* 2017;90(1075):20170076.

29. Lebenatus A, Salehi Ravesh M. Aortocaval fistula at 4D flow MRI: visualization and quantification of vascular flow. *Radiology* 2018;289(3):617.
30. Wilson WR, Bower TC, Creager MA, et al. Vascular graft infections, mycotic aneurysms, and endovascular infections: a scientific statement from the American Heart Association. *Circulation* 2016;134(20):e412–e460.
31. Zaiem F, Almasri J, Tello M, Prokop LJ, Chaikof EL, Murad MH. A systematic review of surveillance after endovascular aortic repair. *J Vasc Surg* 2018;67(1):320–331.e37.
32. van der Laan MJ, Bartels LW, Bakker CJ, Viergever MA, Blankensteijn JD. Suitability of 7 aortic stent-graft models for MRI-based surveillance. *J Endovasc Ther* 2004;11(4):366–371.
33. Cejna M, Loewe C, Schoder M, et al. MR angiography vs CT angiography in the follow-up of nitinol stent grafts in endoluminally treated aortic aneurysms. *Eur Radiol* 2002;12(10):2443–2450.
34. Bartels LW, Bakker CJ, Viergever MA. Improved lumen visualization in metallic vascular implants by reducing RF artifacts. *Magn Reson Med* 2002;47(1):171–180.
35. Love C, Palestro CJ. Radionuclide imaging of infection. *J Nucl Med Technol* 2004;32(2):47–57; quiz 58–59.
36. Reinders Folmer EI, Von Meijenfeldt GCI, Van der Laan MJ, et al. Diagnostic imaging in vascular graft infection: a systematic review and meta-analysis. *Eur J Vasc Endovasc Surg* 2018;56(5):719–729.
37. Treglia G. Diagnostic performance of ¹⁸F-FDG PET/CT in infectious and inflammatory diseases according to published meta-analyses. *Contrast Media Mol Imaging* 2019;2019:3018349.
38. Ganatra RH, Haniffa MA, Hawthorne AB, Rees JI. Aortoenteric fistula complicating an infected aortic graft: diagnosis by leukocyte scintigraphy. *Clin Nucl Med* 2001;26(9):800–801.
39. Perrone T, Pagani C, Mossolani EE. Ultrasound detection of aortoenteric fistula in a patient with sepsis. *J Ultrasound* 2017;20(2):157–159.
40. Mackenzie DC. Aortoenteric fistula identified by clinical ultrasound. *J Emerg Med* 2015;48(6):699–701.
41. Egan DJ, Saul T, Herbert-Cohen D, Lewiss RE. Bedside ultrasound diagnosis of an aortocaval fistula in the emergency department. *J Emerg Med* 2014;47(2):e55–e57.
42. Clevert DA, Stickel M, Flach P, et al. Contrast-enhanced ultrasound in detection and follow-up of an infrarenal abdominal aortic aneurysm with aorto-caval fistula and endovascular treatment. *Cardiovasc Intervent Radiol* 2007;30(3):480–484.
43. Ananthasubramaniam K. Clinical and echocardiographic features of aorto-atrial fistulas. *Cardiovasc Ultrasound* 2005;3(1):1.
44. Yuan SM. Aortobronchial fistula. *Gen Thorac Cardiovasc Surg* 2020;68(2):93–101.
45. Bouchez S, Wouters PF, Vandenplas G. Asymptomatic aorto-atrial fistula identified with intraoperative transesophageal echocardiography. *J Cardiothorac Vasc Anesth* 2012;26(6):e76–e77.
46. Erkal Z, Bayar N, Cagirci G, Arslan S. Incidentally detected aorto-right atrial fistula in asymptomatic patient with operated aortic dissection. *Int J Cardiovasc Acad* 2016;3(1-2):57–58.
47. Veerbeek AG, van der Wieken LR, Schuilenburg RM, Bloemendaal K. Acquired aorto-pulmonary fistula in acute dissection. *Eur Heart J* 1992;13(5):713–715.
48. Boyd LJ. A study of four thousand reported cases of aneurysm of the thoracic aorta. *Am J Med Sci* 1924;168:654–663.
49. Hirst AE Jr, Johns VJ Jr, Kimes W Jr. Dissecting aneurysm of the aorta: a review of 505 cases. *Medicine (Baltimore)* 1958;37(3):217–279.
50. Piciché M, De Paulis R, Chiariello L. A review of aortopulmonary fistulas in aortic dissection. *Ann Thorac Surg* 1999;68(5):1833–1836.
51. Kenny D, Holoshitz N, Turner D, Hijazi ZM. Aortopulmonary fistula after transcatheter pulmonary valve replacement. *Circ Cardiovasc Interv* 2013;6(6):e67–e68.
52. Kort S, Tunick PA, Applebaum RM, et al. Acquired aortopulmonary artery fistula: diagnosis by multiple imaging modalities. *J Am Soc Echocardiogr* 2001;14(8):842–845.
53. Knockaert DC. Cardiac involvement in systemic inflammatory diseases. *Eur Heart J* 2007;28(15):1797–1804.
54. Kokotsakis J, Misthos P, Athanasiou T, et al. Endovascular stenting for primary aortobronchial fistula in association with massive hemoptysis. *Tex Heart Inst J* 2007;34(3):369–372.
55. Heckstall RL, Hollander JE. Aortoesophageal fistula: recognition and diagnosis in the emergency department. *Ann Emerg Med* 1998;32(4):502–505.
56. Amin S, Luketich J, Wald A. Aortoesophageal fistula: case report and review of the literature. *Dig Dis Sci* 1998;43(8):1665–1671.
57. Hagspiel KD, Turba UC, Bozlar U, et al. Diagnosis of aortoenteric fistulas with CT angiography. *J Vasc Interv Radiol* 2007;18(4):497–504.
58. Hughes FM, Kavanagh D, Barry M, Owens A, MacErlaine DP, Malone DE. Aortoenteric fistula: a diagnostic dilemma. *Abdom Imaging* 2007;32(3):398–402.
59. Sevastos N, Rafailidis P, Kolokotronis K, Papadimitriou K, Papatheodoridis GV. Primary aortojunal fistula due to foreign body: a rare cause of gastrointestinal bleeding. *Eur J Gastroenterol Hepatol* 2002;14(7):797–800.
60. Sindelar WF, Mason GR. Aortocystoduodenal fistula: rare complication of pancreatic pseudocyst. *Arch Surg* 1979;114(8):953–955.
61. Karthaus EG, Post IC, Akkersdijk GJ. Spontaneous aortoenteric fistula involving the sigmoid: a case report and review of literature. *Int J Surg Case Rep* 2016;19:97–99.
62. Kim JY, Kim YW, Kim CJ, Lim HI, Kim DI, Huh S. Successful surgical treatment of aortoenteric fistula. *J Korean Med Sci* 2007;22(5):846–850.
63. Alzobydi AH, Guraya SS. Primary aortoduodenal fistula: a case report. *World J Gastroenterol* 2013;19(3):415–417.
64. Beuran M, Negoii I, Negoii RI, Hostiuc S, Paun S. Primary Aortoduodenal fistula: first you should suspect it. *Braz J Cardiovasc Surg* 2016;31(3):261–263.
65. Orion KC, Beaulieu RJ, Black JH 3rd. Aortocaval fistula: is endovascular repair the preferred solution? *Ann Vasc Surg* 2016;31:221–228.
66. Wang T, Huang B, Zhao J, Yang Y, Yuan D. Aortocaval fistula resulting from rupture of abdominal aortic dissecting aneurysm treated by delayed endovascular repair: a case report. *Medicine (Baltimore)* 2016;95(18):e3570.
67. Kotsikoris I, Papas TT, Papanas N, et al. Aortocaval fistula formation due to ruptured abdominal aortic aneurysms: a 12-year series. *Vasc Endovascular Surg* 2012;46(1):26–29.
68. Santoro G, Ramieri A, Chiarella V, Vigliotta M, Domenicucci M. Thoraco-lumbar fractures with blunt traumatic aortic injury in adult patients: correlations and management. *Eur Spine J* 2018;27(suppl 2):248–257.
69. Ravari H, Moini M, Vahedian M, Aliakbarian M. Spontaneous aortocaval fistula: a case report and literature review. *Iran Red Crescent Med J* 2011;13(4):276–279.
70. Psathas ED, Lioudaki S, Doulaptis M, Charalampoudis P, Klonaris C, Verikokos C. Clinical manifestations of aortocaval fistulas in ruptured abdominal aortic aneurysm: report of two cases. *Case Rep Surg* 2012;2012:123081.
71. Agarwal PP, Chughtai A, Matzinger FR, Kazerooni EA. Multidetector CT of thoracic aortic aneurysms. *RadioGraphics* 2009;29(2):537–552.
72. Wong HL, Liew CJY, Poh ACC. Saccular Kommerell aneurysm, a potential pitfall on MDCT imaging: a review of imaging features and potential mimics. *Eur J Radiol Open* 2017;4:89–94.
73. Tsukui H, Aomi S, Yamazaki K. Surgical strategy for Kommerell's diverticulum: total arch replacement. *J Thorac Cardiovasc Surg* 2014;148(4):1423–1427.
74. Qvarfordt PG, Reilly LM, Mark AS, et al. Computerized tomographic assessment of graft incorporation after aortic reconstruction. *Am J Surg* 1985;150(2):227–231.
75. O'Hara PJ, Borkowski GP, Hertzler NR, O'Donovan PB, Brigham SL, Beven EG. Natural history of periprosthetic air on computerized axial tomographic examination of the abdomen following abdominal aortic aneurysm repair. *J Vasc Surg* 1984;1(3):429–433.
76. Low RN, Wall SD, Jeffrey RB Jr, Sollitto RA, Reilly LM, Tierney LM Jr. Aortoenteric fistula and perigraft infection: evaluation with CT. *Radiology* 1990;175(1):157–162.

77. Mathias J, Mathias E, Jausset F, et al. Aorto-enteric fistulas: a physiopathological approach and computed tomography diagnosis. *Diagn Interv Imaging* 2012;93(11):840–851.
78. Green DB, Vargas D, Reece TB, Raptis CA, Johnson WR, Truong QA. Mimics of complications in the postsurgical aorta at CT. *Radiol Cardiothorac Imaging* 2019;1(4):e190080.
79. Antunes C, Graça B, Donato P. Thoracic, abdominal and musculoskeletal involvement in Erdheim-Chester disease: CT, MR and PET imaging findings. *Insights Imaging* 2014;5(4):473–482.
80. Rakita D, Newatia A, Hines JJ, Siegel DN, Friedman B. Spectrum of CT findings in rupture and impending rupture of abdominal aortic aneurysms. *RadioGraphics* 2007;27(2):497–507.
81. Chandrashekar G, Kumar VM, Kumar AK. Repair of aorto-esophageal fistula due to a penetrating atherosclerotic ulcer of the descending thoracic aorta and literature review. *J Cardiothorac Surg* 2007;2(1):12.
82. Xu R, Wang T, Li D, et al. Surgical approach for the treatment of aorto-esophageal fistula combined with dual aortic aneurysms: a case report. *J Cardiothorac Surg* 2013;8(1):206.
83. Maltezos K, Bisdas T, Patelis N. Endovascular repair of aorto-enteric fistulae as a definitive treatment strategy. *Vasc Dis Manage* 2020;17(3):E46–E50.
84. Sasaki A, Egashira H, Tokoro S, et al. Thoracic endovascular aortic repair of esophageal cancer-associated aorto-esophageal fistula: a case report and literature review. *Case Rep Oncol Med* 2018;2018:9851397.
85. Estévez-Loureiro R, Salgado Fernández J, Vázquez-González N, et al. Percutaneous closure of an aorto-atrial fistula after surgery for infective endocarditis. *JACC Cardiovasc Interv* 2012;5(6):e15–e17.
86. Hernández-García JM, Alonso-Briales JH, Jiménez-Navarro MF, Cabrera-Bueno F, González-Cocina E, Such-Martínez M. Transcatheter closure of aorto-left atrial fistula using an Amplatzer device [in Spanish]. *Rev Esp Cardiol* 2005;58(9):1121–1123.
87. Hiraki T, Mimura H, Kanazawa S, et al. Transcatheter embolization of an aortobronchial fistula with N-butyl cyanoacrylate. *J Vasc Interv Radiol* 2002;13(7):743–746.