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CARDIOVASCULAR IMAGING

REVIEW ARTICLE

Non-pulmonary postoperative complications of cardiothoracic surgery

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ABSTRACT

Cardiothoracic surgery, including coronary artery bypass grafting, valve replacement, and transplantation, has considerably advanced, improving patient survival and outcomes. However, non-pulmonary postoperative complications remain a major concern, contributing to morbidity and mortality. These complications encompass cardiovascular events, vascular injuries, infections, and device-related issues that can severely impact recovery. Early diagnosis and timely intervention are crucial to mitigating risks and improving patient outcomes. Advanced imaging modalities such as computed tomography, magnetic resonance imaging, and echocardiography play a pivotal role in identifying and characterizing complications before clinical deterioration occurs. This review highlights the spectrum of acute non-pulmonary complications following cardiothoracic surgery, emphasizing the diagnostic value of imaging in guiding clinical decision-making. By improving the awareness of imaging findings associated with postoperative complications, radiologists and clinicians can facilitate early detection, enabling prompt surgical or medical interventions. A multidisciplinary approach that integrates imaging surveillance with clinical assessment is essential for optimizing patient care and reducing long-term morbidity.

KEYWORDS

Abscess, aorta, bypass, cardiac, cardiac catheterization, complications, computed tomography, dissection, endocarditis, hemorrhage, infection, mediastinum, surgery

Cardiothoracic surgery–such as coronary artery bypass grafting (CABG), valve replacement, and transplantation–has greatly evolved in both technique and perioperative care.[1,2] Despite being lifesaving, these complex procedures carry risks, especially when comorbidities are present.[2] Non-pulmonary postoperative complications–including vascular, infectious, and structural problems–can considerably affect recovery, morbidity, and mortality. Early detection and prompt management are critical. Imaging techniques such as chest radiography, computed tomography (CT), and magnetic resonance imaging (MRI) are vital for identifying complications, often before clinical symptoms arise.[3-6] Radiologists play a key role by enabling timely interventions such as surgical revisions or targeted therapies (Table 1). This review examines acute non-pulmonary complications following cardiothoracic surgery, highlights the diagnostic value of imaging, and explores strategies for improving outcomes through interdisciplinary collaboration.

2. Non-pulmonary complications

2.1. Cardiovascular complications

Cardiovascular complications considerably contribute to morbidity and mortality after cardiothoracic surgery. Key concerns include myocardial infarction (MI), cardiac tamponade, graft stenosis or occlusion, and pseudoaneurysms. Early detection with advanced imaging and prompt intervention are critical for optimal patient outcomes.

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a. Hemopericardium and cardiac tamponade

Cardiac tamponade is a life-threatening complication of cardiovascular surgery, commonly caused by surgical trauma, vascular injury, or anticoagulation.[3,5] Blood or fluid accumulates in the pericardial space, raising intrapericardial pressure and compressing the heart, thereby compromising cardiac output. Prompt recognition is essential to avert circulatory collapse, and imaging findings are crucial in detecting and assessing the severity of tamponade.[7,8] On chest radiography, a markedly enlarged, globular (water bottle) cardiac silhouette suggests considerable pericardial effusion.[8,9] Echocardiography is vital for identifying pericardial fluid and signs of cardiac chamber compression, such as right ventricular or right atrial collapse.[7] CT can delineate hyperattenuating or complex fluid collections indicative of hemopericardium, locate potential bleeding sources, and reveal secondary signs of tamponade, including inferior vena cava distension (Figure 1).[5] MRI further characterizes fluid composition and evaluates myocardial function.[10] Urgent pericardiocentesis is the primary intervention to evacuate the effusion and relieve cardiac compression. If substantial hemorrhage persists or the source of bleeding is unclear, surgical exploration may be needed.[7]

Main points

- Non-pulmonary complications after cardiothoracic surgery, including cardiovascular, vascular, infectious, and device-related issues, substantially impact patient recovery and outcomes. Early diagnosis and intervention are crucial to reducing morbidity and mortality.
- Cardiovascular issues such as myocardial infarction, cardiac tamponade, and thrombus formation, as well as vascular problems such as hemothorax and aortic dissection, require prompt detection through imaging techniques such as echocardiography, computed tomography (CT), and magnetic resonance imaging (MRI) to prevent severe outcomes.
- Postoperative infections, including mediastinitis, surgical site infections, and endocarditis, pose considerable risks. Sternotomy-related complications, such as sternal dehiscence and osteomyelitis, necessitate early recognition using CT and MRI for effective management.
- Prosthetic valves, pacemakers, and left ventricular assist devices (LVADs) can develop dysfunction, infections, or mechanical failures. Imaging plays a crucial role in detecting issues such as valve thrombosis, pacemaker lead displacement, and LVAD-associated thrombi.

b. Myocardial infarction

MI is an uncommon but serious complication of cardiothoracic surgery. Postoperative MI can complicate not only CABG but also other cardiac operations–including valve replacement/repair, resection of intracardiac masses, and complex congenital or aortic procedures–due to ischemia–reperfusion injury, graft or native-vessel occlusion, or embolization.[5] Perioperative MI occurs in about 2%–10% of patients undergoing CABG, underscoring the importance of early and accurate detection.[11] Recent diagnostic algorithms highlight postoperative troponin elevation as a crucial prognostic marker, even without overt ischemic symptoms.[12] Echocardiography is the first-line imaging modality, offering rapid assessment of cardiac function and wall motion. Coronary CT angiography (CTA) provides rapid, non-invasive evaluation of native coronary arteries, assesses bypass graft and stent patency, and demonstrates low-attenuation subendocardial perfusion defects or hypo-enhancement indicative of acute or evolving MI (Figure 2 and Supplementary Figure 1). Cardiac MRI quantifies infarct size, transmurality, and location; detects microvascular obstruction and hemorrhage; visualizes edema and acute injury; differentiates viable from non-viable myocardium for revascularization decisions; and flags early complications such as thrombus, papillary-muscle infarction, septal defect, and adverse remodeling when performed in hemodynamically stable patients.[13,14] Treatment focuses on prompt revascularization with percutaneous coronary intervention or reoperative CABG, supplemented by antiplatelet therapy, anticoagulants, beta-blockers, and other medical support.[12] A multidisciplinary approach that integrates advanced imaging with timely intervention is vital for optimal patient outcomes.

c. Cardiac thrombus

Cardiac thrombus is a serious postoperative complication, occurring in 1%–6% of cardiothoracic surgeries, particularly in valve replacements or procedures involving prosthetic devices.[15] Thrombi often form due to intraoperative endothelial injury, blood stasis, or hypercoagulability and carry a high risk of embolic events such as stroke or pulmonary embolism.[5,15] Echocardiography is the primary imaging method for detecting these thrombi, with transesophageal echocardiography (TEE) considered the gold standard, because it can reveal hyperechoic masses attached to cardiac structures or prosthetic valves.[16] Cardiac CT effectively identifies filling defects within heart chambers, especially in the left atrial appendage (Figure 3 and Supplementary Figure 2), whereas cardiac MRI uses late gadolinium enhancement (LGE) with extended inversion times (TI approximately 600 ms at 1.5T) to improve thrombus detection and assess its composition and surrounding flow dynamics.[14,16] Management typically involves anticoagulation therapy to prevent thrombus growth and embolization, although surgical removal may be necessary if there is considerable hemodynamic risk or embolic potential.[16] Timely imaging and diagnosis are essential for guiding intervention and reducing life-threatening complications.

d. Coronary artery bypass grafting complications

CABG complications include graft occlusion, which affects 10%–15% of patients within the first month (Figure 2). Early occlusion often arises from endothelial injury, platelet dysfunction, or hypercoagulability, whereas late occlusion is largely driven by atherosclerosis. Venous grafts are especially prone to failure, with more than half occluding within 15 years.[17] Recent studies show dual antiplatelet therapy can lower early occlusion risk, emphasizing the importance of optimal postoperative medical management.[12,17] Graft stenosis, often localized to anastomotic sites, compromises perfusion. However, refined surgical techniques and intraoperative imaging have reduced its incidence, prolonging graft durability.[18] Less frequently, pseudoaneurysms (occurring early from suture line disruption or infection) and true aneurysms (developing later from atherosclerosis) can arise (Figure 4). Both carry risks of rupture, thrombosis, or fistula formation and require urgent intervention.[19] Advances in surveillance imaging, including CTA and Doppler ultrasound, allow early detection and guide timely surgical or endovascular repair.

2.2. Vascular and hemorrhagic complications

Vascular and hemorrhagic complications are critical events that can have severe consequences in the postoperative period following cardiothoracic surgery. Prompt identification and management are essential to avoid catastrophic outcomes.

Table 1. Non-pulmonary postoperative complications following cardiothoracic surgery and imaging findings

Complication category	Complication	Imaging findings
Cardiovascular	Hemopericardium and cardiac tamponade	Radiograph: Globular (water bottle) silhouette Echo: Pericardial effusion with chamber collapse CT: Hyperattenuating pericardial fluid, active bleeding MRI: Hemorrhagic vs. serous characterization
Cardiovascular	Myocardial infarction	Echo: Wall motion abnormalities CT: Graft occlusion, myocardial hypoenhancement MRI: Infarct size, edema, hemorrhage, MVO
Cardiovascular	Cardiac thrombus	TEE: Hyperechoic mass on valve/chamber CT: In-chamber filling defect MRI: Thrombus vs. tumor with LGE
Cardiovascular	CABG complications	CT: Graft stenosis, pseudoaneurysm Doppler US: Turbulent or absent graft flow
Vascular and hemorrhagic	Hemothorax	Radiograph: Opacification, effusion US: Hyperechoic pleural fluid CT: Hyperdense pleural fluid, active extravasation
Vascular and hemorrhagic	Mediastinal hemorrhage	Radiograph: Mediastinal widening CT: High-density mediastinal fluid, active extravasation (multiphase imaging)
Vascular and hemorrhagic	Aortic dissection	CT: Intimal flap, double lumen MRI: Intimal flap, thrombus, late-stage complications
Vascular and hemorrhagic	Aortic graft complications	CT: Pseudoaneurysm, endoleak, thrombosis Doppler US: Graft stenosis
Infectious	Mediastinitis	Radiograph: Mediastinal widening, gas CT: Rim-enhancing abscess, wire displacement, osteomyelitis
Infectious	Surgical site infections	US: Fluid collection CT: Abscess, deep tissue gas
Infectious	Endocarditis and bloodstream infections	TEE: Vegetations, abscesses CT: Septic emboli, vegetations, pseudoaneurysm PET-CT: Prosthetic valve infection, emboli
Sternotomy	Sternal dehiscence	Radiograph: Wire displacement, sternal gap CT: Malalignment, hardware failure
Sternotomy	Sternal wound infection	CT: Bone destruction, abscess MRI: Marrow edema, early osteomyelitis
Sternotomy	Hardware failure	Radiograph: Wire fracture/migration CT: Displacement, associated infection
Device-related	Valve prosthesis dysfunction	TEE: Leak, dehiscence CT: Thrombosis, pannus, calcification MRI: Leak quantification, inflammation
Device-related	Pacemaker/ICD malfunction	Radiograph: Lead migration Echo: Pericardial effusion CT: Lead/device malposition, hemorrhage
Device-related	LVAD related complications	Echo: Thrombus, flow obstruction CT: Device thrombosis, debris, kinking, infection
Postoperative syndromes	Post-pericardiotomy syndrome	Radiograph: Enlarged silhouette, effusion Echo: Pericardial effusion MRI: Pericardial edema, effusion, inflammation
Postoperative syndromes	Post-sternotomy syndrome	CT: Chronic Bone changes MRI: Edema, infection, dehiscence
Neurological	Phrenic nerve injury	Radiograph: Elevated hemidiaphragm Fluoroscopy: Paradoxical motion MRI: Diaphragm atrophy
Neurological	Recurrent laryngeal nerve injury	Laryngoscopy: Vocal cord paralysis CT/MRI: Nerve compression, edema
Neurological	Brachial plexus injury	MRI: Plexus thickening, T2 hyperintensity, avulsion, pseudomeningocele

CT, computed tomography; MRI, magnetic resonance imaging; US, ultrasonography; TEE, transesophageal echocardiography; PET, positron emission tomography; ICD, implantable cardioverter defibrillator; LVAD, left ventricular assist devicess; MVO, microvascular obstruction.

a. Hemothorax (non-pulmonary origin)

Hemothorax is a serious postoperative complication of cardiothoracic surgery, often resulting from iatrogenic vessel injury, vascular graft failure, or bleeding from cardiac structures, with anticoagulation further heightening the hemorrhage risk.[1-3] Patients commonly present with acute hemoglobin decline, respiratory distress, hypotension, and diminished breath sounds, requiring prompt intervention to avoid hypovolemic shock or respiratory failure. Although most cases occur early in the postoperative period, delayed hemothorax can develop weeks later, emphasizing the importance of extended monitoring in high-risk patients.[3,4] Imaging is pivotal for diagnosis. A chest radiograph may show rapid, diffuse opacification of the hemithorax, whereas ultrasound offers a valuable bedside tool, revealing fluid collections of varying echogenicity. On non-contrast thoracic CT, hemothorax is characterized by hyperattenuating pleural collections–typically 30–60 Hounsfield units (HU)–that layer dependently and may demonstrate internal septations or fluid–fluid levels (Figure 5). When CTA is performed, active extravasation appears as a focal contrast blush within the pleural space, and injured thoracic aortic branches or intercostal vessels can often be localized to the site of vascular leak. In delayed cases, it can also characterize hematoma progression and identify complications such as infection or loculated collections.[3-6] Management typically involves thoracostomy drainage, surgical exploration if ongoing bleeding is suspected, and supportive measures such as blood transfusion and correction of coagulopathies.[20]

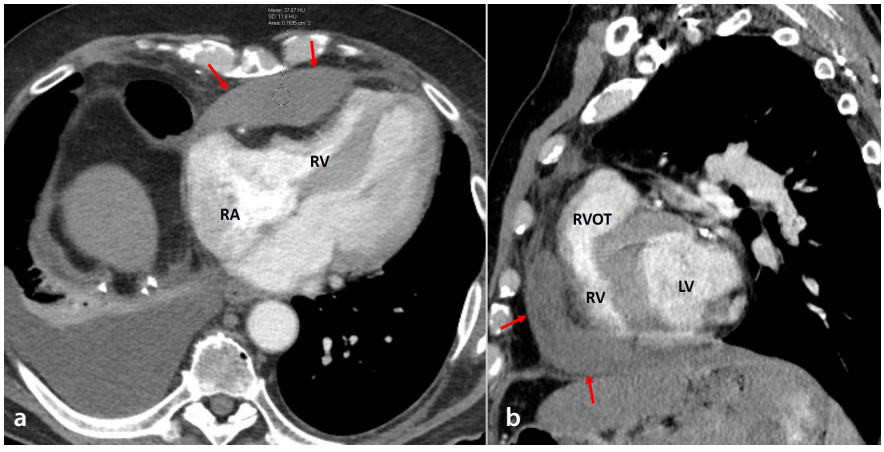

Figure 1. A 62-year-old male status post-robotic coronary artery bypass surgery. **(a)** Axial and **(b)** sagittal CT images demonstrate a loculated right pericardial hematoma (arrows; attenuation of 37 HU) causing considerable compression of the right ventricle, concerning for tamponade. LV, left ventricle; RVOT, right ventricular outflow tract; RA, right atrium; CT, computed tomography; HU, Hounsfield units.

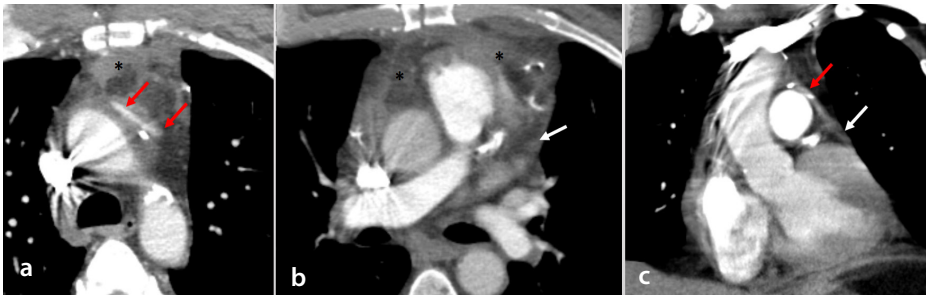

Figure 2. A 66-year-old male status post-coronary artery bypass surgery. **(a, b)** Axial and **(c)** coronal CT images demonstrate an anterior (prevascular) mediastinal postoperative changes with areas of fat stranding (*) and thrombotic occlusion (white arrows) of the aorto-obtuse marginal coronary artery bypass grafting (red arrows). CT, computed tomography.

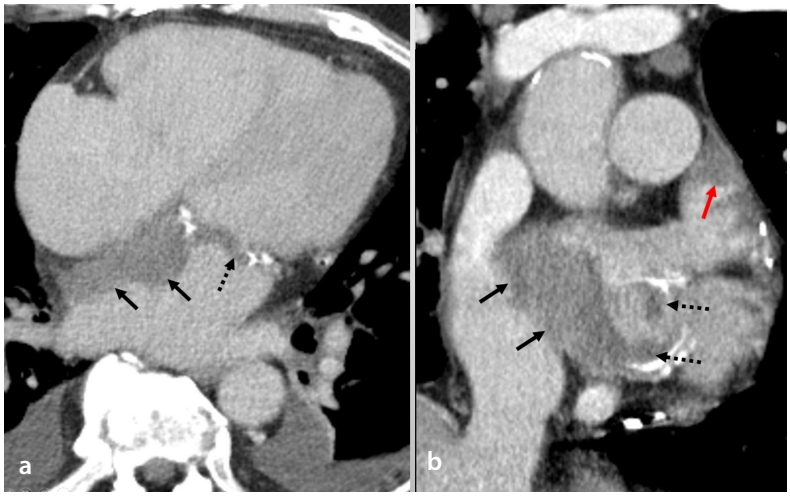

Figure 3. A 74-year-old female status post-mitral valve replacement. Axial and coronal CT images demonstrate an intermediate attenuated lesion most consistent with thrombus along the interatrial septum (solid arrows) and a prosthetic mitral valve, consistent with thrombus (dashed arrows) within the left atrium, as well as a left atrial appendage thrombus (red arrow). Follow-up echocardiography demonstrated complete resolution of the thrombus. CT, computed tomography.

b. Mediastinal hemorrhage

Mediastinal hemorrhage is a life-threatening complication of cardiac surgery, occurring in approximately 3.8% of cases requiring re-sternotomy for bleeding.[1,21] It typically results from intraoperative vascular injury involving the pulmonary arteries, internal thoracic arteries, or azygos vein, as well as postoperative coagulopathy.[3-5,21] Clinically, patients may present with hemodynamic instability, chest pain, and radiographic mediastinal widening, all of which necessitate urgent intervention to prevent progressive blood accumulation that can lead to cardiac tamponade, respiratory compromise, or fatal compression of mediastinal structures. Delayed diagnosis is associated with increased mortality, emphasizing the critical need for prompt imaging and treatment. Chest radiography may reveal mediastinal widening but lacks specificity. CT provides a more definitive evaluation, demonstrating high-density mediastinal fluid and identifying potential bleeding sources. Multiphase CT with non-contrast, arterial, and venous phase imaging is particularly useful for localizing active bleeding, assessing compression of adjacent structures, and guiding therapeutic decisions (Figure 6).[3-7] Management requires

immediate surgical exploration to control hemorrhage, with interventional radiology techniques such as vessel embolization serving as a less invasive alternative in select cases. Hemodynamic stabilization through volume resuscitation and blood product administration is essential to support circulation and prevent further deterioration.[21]

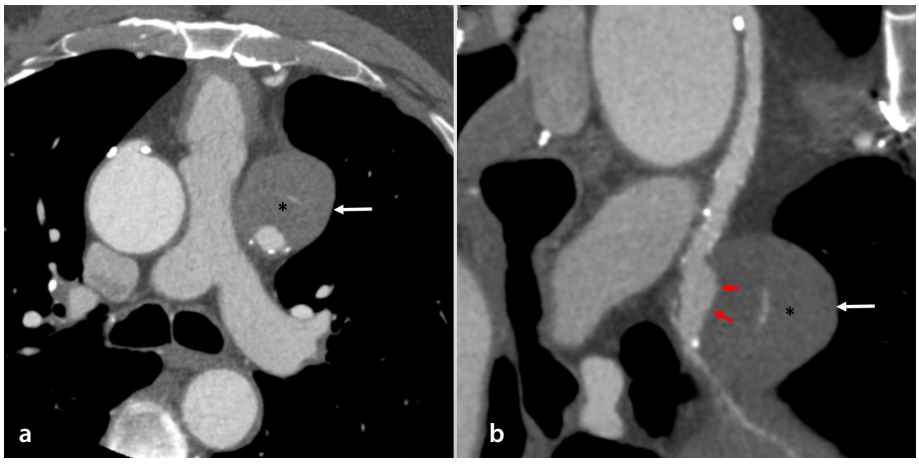

Figure 4. A 48-year-old female status post-robotic coronary artery bypass surgery. **(a)** Axial and **(b)** multiplanar reformat coronary CT angiography images demonstrate a partially thrombosed (*) pseudoaneurysm of the aorto-left circumflex coronary artery bypass grafting (white arrows). Wall irregularities of the bypass graft are also noted (red arrows). CT, computed tomography.

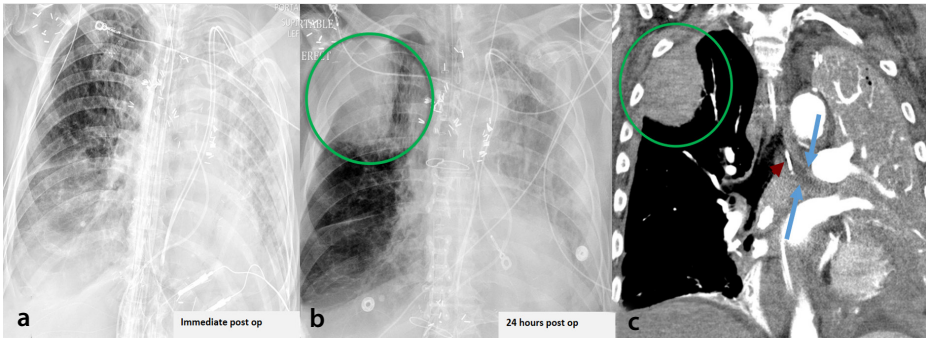

Figure 5. A 66-year-old male status post-heart transplant presenting with hemoptysis. **(a)** The immediate postoperative chest radiograph shows expected post-surgical findings. **(b)** Chest radiograph obtained 24 hours after surgery demonstrates a new broad-based density in the right upper hemithorax (circle). **(c)** Chest CT angiography reveals the opacity corresponding to a loculated hemothorax. A bronchial occluder extends into the left mainstem bronchus (arrowhead), which is filled with hemorrhagic debris (arrows). The left lung is entirely consolidated. CT, computed tomography.

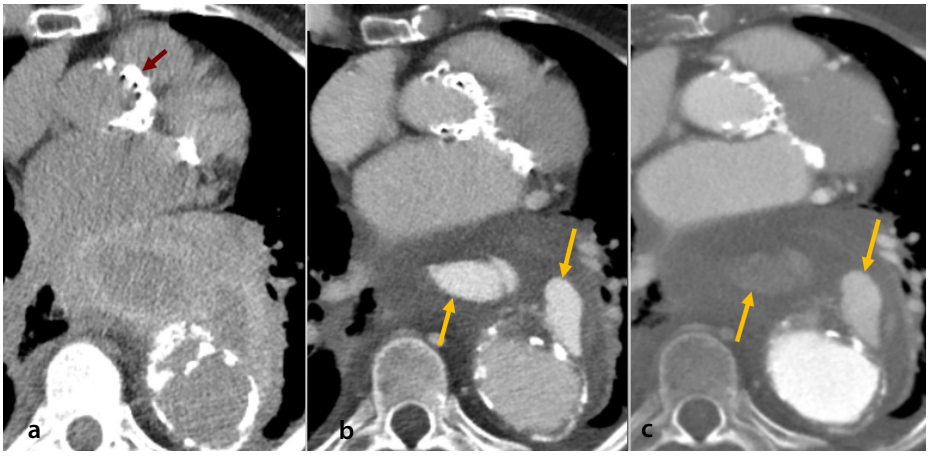

Figure 6. A 79-year-old female status post-transcatheter aortic valve replacement (red arrow). **(a)** Non-contrast, **(b)** arterial, and **(c)** venous phase chest CT images demonstrate arterial contrast extravasation within a periaortic collection, with contrast pooling on the venous phase (yellow arrows), consistent with aortic rupture. The collection contains areas of hyperdensity on the non-contrast image, suggestive of acute blood products/thrombus. CT, computed tomography.

c. Aortic dissection and pseudoaneurysms

Aortic dissection and pseudoaneurysms are rare but severe complications following cardiac or aortic surgery, each carrying high morbidity and mortality. Aortic dissection, occurring in 0.06%–0.23% of procedures, typically arises from intraoperative aortic wall trauma during cannulation, cross-clamping, or graft anastomosis (Figure 7 and Supplementary Figure 3). Chest pain is common but may be overshadowed by nonspecific symptoms such as hypertension or vague discomfort, making early detection challenging; intraoperative TEE can help identify dissections promptly.[22]

Pseudoaneurysms, affecting 2%–3% of aortic repairs, often form at anastomotic sites due to technical imperfections, infection, or graft dehiscence, posing risks of rupture, thrombosis, or compression. On CTA, true aneurysms are broad-necked dilations of all three vessel layers with smooth or scalloped walls and peripheral calcifications, whereas pseudoaneurysms are narrow-necked, irregular saccular outpouchings with wall disruption, periaortic hematoma, and lobulated margins. CTA is the preferred diagnostic tool for both conditions, distinguishing pseudoaneurysms from true aneurysms and delineating dissection flaps, lumina, and branch vessel involvement (Figure 8). Cardiac MRI improves the evaluation of chronic aortic disease by providing cine images for precise measurement of lumen and flap motion; phase-contrast flow quantification to detect false lumen thrombosis or branch compromise; T1/T2 black-blood and mapping sequences to distinguish thrombus or hematoma; LGE to identify wall inflammation, fibrosis, or pseudoaneurysm integrity; and simultaneous assessment of aortic regurgitation and ventricular function.[3-5,10] Management involves urgent surgical repair or endovascular stenting, combined with stringent blood pressure control to prevent progression. Early imaging and timely intervention are critical to avert hemodynamic instability and life-threatening hemorrhage.[23]

d. Aortic vascular graft complications

Aortic vascular graft complications–graft rupture, thrombosis, pseudoaneurysms, infection, and endoleaks–pose major risks after aortic aneurysm repair or dissection surgery.[3,5,24-26] Though rare, graft rupture can trigger sudden hemodynamic collapse, requiring emergency surgery. Thrombosis, presenting as limb pain, pulse deficits, or organ dysfunction from distal ischemia, is

tempered by improved anticoagulation and endovascular thrombectomy. Pseudoaneurysms often arise from anastomotic leaks or graft degradation and carry a high rupture risk, but endovascular stent-graft placement offers a minimally invasive alternative to open repair.[26] Graft infections, frequently involving *Staphylococcus* species, require combined medical and surgical management, including antibiotics, debridement, and possible graft replacement (Figure 9).[24,25]

Endoleaks–a unique complication of endovascular aneurysm repair–persist in 20%–30% of cases and are classified into 5 types based on etiology: type I (inadequate proximal/distal seal), type II (collateral retrograde flow), type III (graft junction failure), type IV (graft material porosity), and type V (endotension with sac expansion).[27] Unaddressed endoleaks risk aneurysm sac enlargement and rupture, necessitating vigilant surveillance. CTA is pivotal for diagnosis, offering high-resolution visualization of graft integrity, thrombus formation, pseudoaneurysm morphology, and endoleak flow dynamics. Endoleak evaluation requires a three-phase CTA protocol: an unenhanced scan to distinguish calcifications or thrombus from contrast, an arterial-phase acquisition at 30–40 s post-injection to detect high-flow (type I/III) endoleaks and active contrast blush, and a delayed (venous) phase at 60–120 s to identify low-flow (type II) endoleaks that may only opacify on later images (Figure 10). Early imaging-guided intervention–whether endovascular coil embolization for type II endoleaks, stent relining for pseudoaneurysms, or antimicrobial therapy for infections–is critical to avert life-threatening sequelae. Structured postoperative surveillance protocols, including serial CTA, ensure timely identification of complications, underscoring the synergy between radiologists and surgeons in optimizing patient outcomes.[26,27]

2.3. Infectious complications

Non-pulmonary infectious complications pose critical threats to recovery after cardiothoracic surgery, driving morbidity and mortality through conditions such as mediastinitis, surgical site infections (SSIs), bloodstream infections (BSIs), and device-associated infections (e.g., prosthetic valve endocarditis). Risk factors span preoperative (e.g., diabetes, obesity), intraoperative (e.g., prolonged surgery duration, immunosuppression), and postoperative (e.g., inadequate wound care) variables.

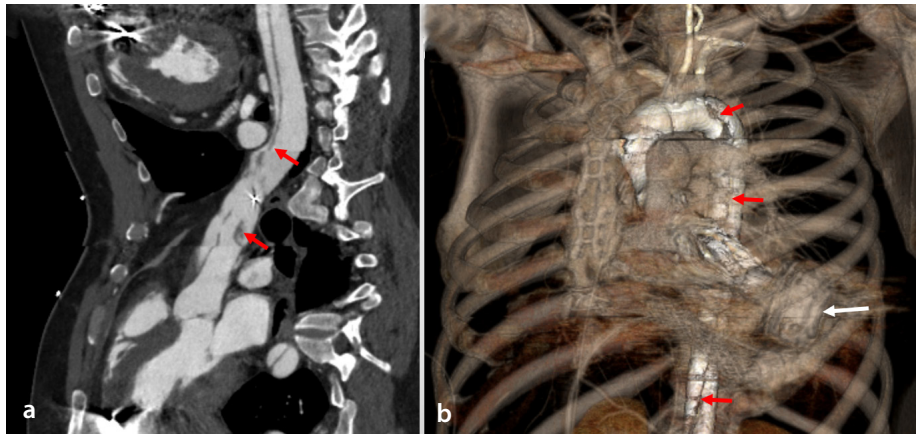

Figure 7. A 57-year-old male status post-left ventricular assist device placement (white arrow). **(a)** Aortic multiplanar reformatted chest CT angiography and **(b)** three-dimensional volume-rendered CT image demonstrate a dissection flap extending through the upper ascending aorta and descending thoracic aorta (red arrows). CT, computed tomography.

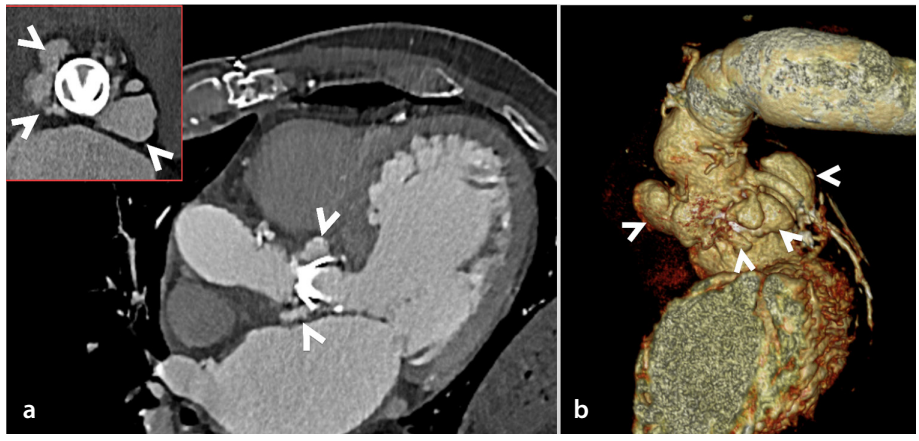

Figure 8. A 63-year-old man with a history of infective endocarditis and composite aortic valve graft with aortic root replacement (Bentall procedure). **(a)** Chest CT angiography images in a left ventricular outflow tract view and corresponding axial view through the aortic valve demonstrate multiple aortic pseudoaneurysms (arrowheads). **(b)** A three-dimensional volume-rendered thoracic CT angiography image also highlights the aortic pseudoaneurysms (arrowheads). CT, computed tomography.

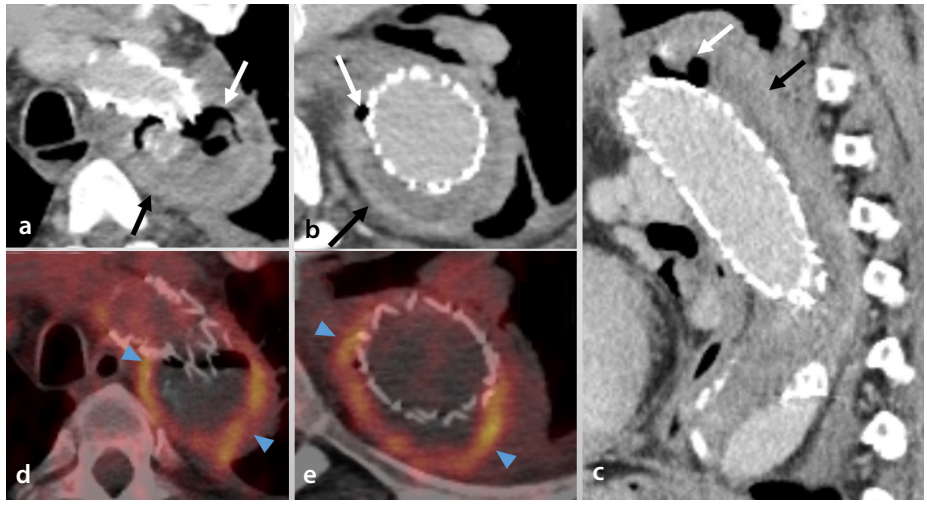

Figure 9. A 73-year-old male status post-aortic endovascular repair. **(a, b)** Axial and **(c)** sagittal CT angiography images demonstrate air surrounding the endograft (white arrows) within the excluded lumen, which exhibits an irregular thickened wall and pathological contrast-enhancement (black arrows). **(d, e)** Axial FDG-PET/CT images show corresponding FDG uptake (arrowheads), consistent with endovascular graft infection. CT, computed tomography; FDG-PET, fluorodeoxyglucose positron emission tomography.

a. Mediastinitis

Mediastinitis is a rare but life-threatening complication of cardiothoracic surgery, affecting 0.4%–5% of cases and occurring more frequently after median sternotomy, such as in CABG or valve replacement. Risk factors include diabetes, obesity, prolonged mechanical ventilation, and *Staphylococcus aureus* colonization.[28] Clinical signs often appear 2–4 weeks postoperatively, featuring fever, sternal instability, purulent drainage, and chest pain. Delayed diagnosis increases mortality beyond 20%.[29] Imaging is key for early detection, and chest radiography can show mediastinal widening, air–fluid levels, or new air collections, but it lacks specificity. CT is preferred, revealing mediastinal fluid collections, gas bubbles, soft tissue fat stranding, rim-enhancing abscesses, and possible sternal wire displacement or osteomyelitis (Figure 11).[3,5,28] Management requires prompt intravenous broad-spectrum antibiotics, surgical debridement, and drainage of any abscesses. Negative-pressure wound therapy aids in wound management, and early diagnosis and aggressive treatment are essential to lower mortality.

b. Surgical site infections

SSIs arise in 1%–8% of cardiothoracic surgeries, with rates influenced by procedure complexity and patient risk factors. They range from superficial infections–limited to skin and subcutaneous tissue–to deep infections involving muscle, fascia, or internal structures. Superficial SSIs typically present with localized redness, pain, swelling, and purulent discharge, whereas deep SSIs may exhibit systemic signs such as fever, leukocytosis, and hemodynamic instability.[29] Imaging is critical in identifying deep infections. Ultrasound effectively detects fluid collections suggestive of abscesses, whereas CT scans provide detailed views of deep-seated infections, including fluid collections, gas, and inflammatory changes (Supplementary Figure 4).[3,5] Treatment depends on infection severity: superficial infections often respond to local wound care and targeted antibiotics, whereas deep infections usually require surgical debridement, drainage, and systemic antimicrobial therapy.[29]

c. Endocarditis and bloodstream infections

Endocarditis is a serious complication of BSIs, especially in patients who undergo cardiothoracic surgery with extended hospital stays or invasive procedures.[30,31] The risk of infective endocarditis (IE) differs by pathogen, with certain *Streptococcus* species (e.g., *S. sanguinis, S. mutans*) linked to higher susceptibility. BSIs affect 2%–5% of postoperative patients, commonly stemming from catheter-related infections or mediastinitis, with *S. aureus*, coagulase-negative staphylococci, and gram-negative bacilli as frequent culprits. Clinically, IE may present with new murmurs, embolic events, or heart failure, requiring swift recognition and management.[30] TEE is the gold standard for diagnosing IE, providing high-resolution visualization of vegetations, abscesses, and prosthetic valve involvement. When echocardiography is inconclusive, cardiac MRI serves as the second-line test, particularly valuable in native valve IE or suspected myocardial/septic embolic extension. Guideline-endorsed fluorodeoxyglucose positron emission tomography/CT is preferred for prosthetic valve or device-related IE, culture-negative cases, and whole-body embolic surveys, with peri-annular hyperuptake beyond 3 months post-surgery confirming infection and extracardiac foci guiding antibiotic duration.[30,31] Cardiac CT–beyond identifying extracardiac septic emboli–directly visualizes perivalvular complications (abscesses, pseudoaneurysms, fistulae) and valve vegetations on both native and prosthetic valves, thereby enhancing diagnostic confidence when echocardiography is inconclusive (Figure 12).[31] Treatment involves a prolonged course of pathogen-targeted intravenous antibiotics. Surgical intervention may be necessary for prosthetic valve endocarditis, substantial valvular dysfunction, or abscess formation to avert further complications.[30,31]

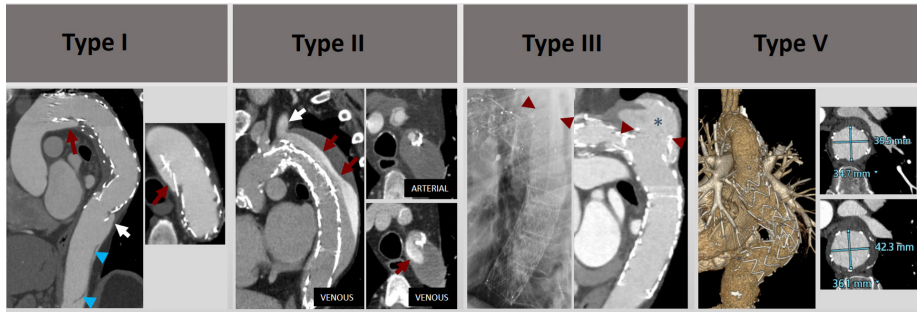

Figure 10. The images illustrate the common types of endoleaks and their corresponding CT findings.

• Type I (inadequate proximal/distal seal): Contrast extravasation into the excluded lumen at the proximal (red arrows) and distal (white arrow) stent margins. A residual dissection flap is noted in the distal descending segment (arrowheads).

• Type II (collateral retrograde flow): Retrograde contrast flow from the proximal left subclavian artery (white arrow) into the excluded false lumen (red arrows) with layering of bright contrast.

• Type III (graft junction failure): Outpouching of contrast (asterisk) extending into the excluded lumen across angulated and separated stent components (arrowhead).

• Type V (endotension with sac expansion): Mildly increased cross-sectional stent dimensions on follow-up. CTA (bottom right image), consistent with progressive sac expansion. CT, computed tomography; CTA, CT angiography.

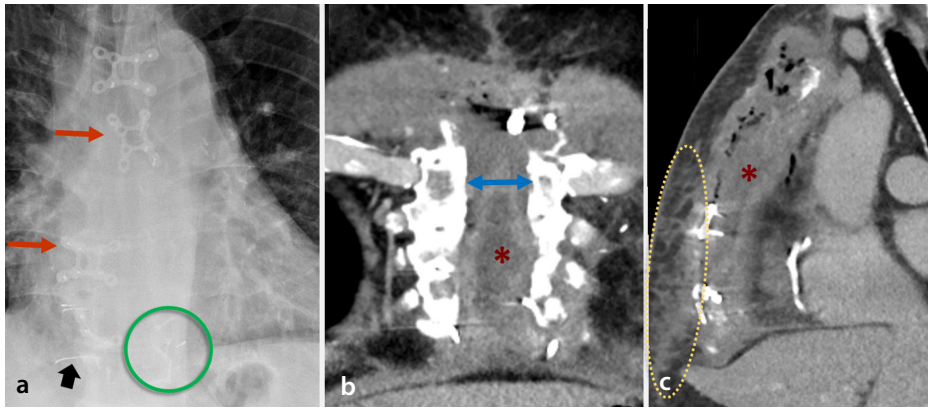

Figure 11. A 62-year-old male status post-coronary artery bypass grafting. **(a)** Chest radiograph demonstrates misaligned sternotomy hardware (arrows), fractured wires (short arrow), and dislodged screws (circle). **(b, c)** Coronal and sagittal chest CT images show a thick-walled air and fluid collection (asterisk) centered on the diastatic sternotomy (double arrow), consistent with an abscess. Associated inflammatory changes in the overlying anterior chest wall are also noted (dotted circle). CT, computed tomography.

2.4. Sternotomy complications

Sternotomy complications considerably affect patient recovery and pose a challenge due to their potential to cause severe morbidity and prolong hospital stay.

a. Sternal dehiscence

Sternal dehiscence, seen in 1%–3% of cardiothoracic surgeries, involves partial or complete separation of the sternum.[32] Common causes include inadequate fixation, infection (e.g., mediastinitis), or poor wound healing, and it often arises within weeks of surgery, presenting with instability, crepitus, pain, or swelling. Untreated cases can progress to mediastinitis, sepsis, or respiratory compromise. Imaging is crucial for early detection. Chest radiographs may show wire or plate displacement, sternal malalignment, or widening between sternal edges. CT offers a more detailed assessment of sternal alignment, wire integrity, and complications such as soft tissue edema, fluid collections, or abscesses (Figure 11 and Supplementary Figure 5).[32,33] Treatment depends on severity. Mild dehiscence may be managed conservatively with chest binders and physical therapy, whereas severe cases often require surgical re-closure with reinforced fixation, vacuum-assisted closure, and targeted antibiotic therapy.[32]

b. Sternal wound infection and osteomyelitis

Sternal osteomyelitis, a serious complication occurring in 0.5%–1% of median sternotomies, typically arises from deep sternal wound infections or mediastinitis.[29,32,34] Risk factors include prolonged operative times, diabetes, obesity, immunosuppression, and bilateral internal mammary artery grafting. Patients may present with persistent chest pain, localized erythema, swelling, purulent discharge, and systemic signs such as fever or leukocytosis. In advanced cases, sternal instability and fistula formation can lead to systemic sepsis or chronic osteomyelitis.[34] Early imaging is essential. Chest radiography may reveal sternal separation, and ultrasound can detect fluid collections or abscesses. CT provides detailed views of bone destruction, periosteal reactions, and soft tissue involvement, whereas MRI is highly sensitive for early marrow edema (Figure 11 and Supplementary Figure 6). Nuclear imaging, including bone scintigraphy, helps clarify ambiguous findings.[32,33] Management involves prolonged, culture-targeted intravenous antibiotics and extensive surgical debridement to remove necrotic tissue and infected bone. In cases of considerable bone loss or nonunion, reconstructive surgery with muscle or omental flaps may be necessary to restore stability.[35]

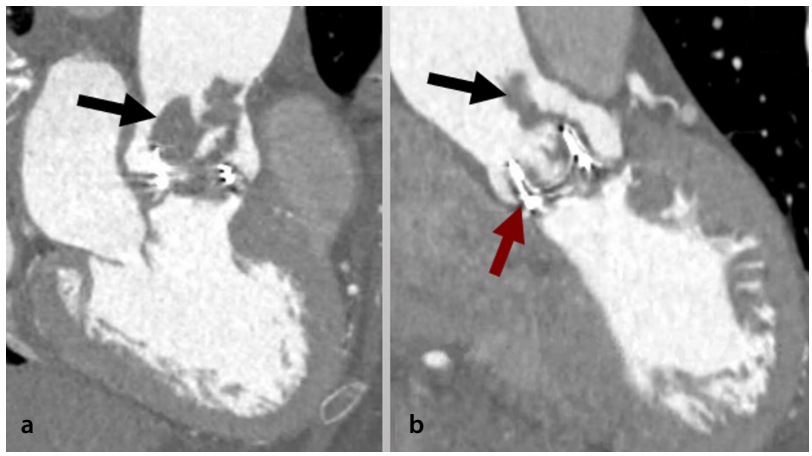

Figure 12. A 54-year-old female status post-surgical aortic valve repair. **(a, b)** Multiplanar reformatted CT angiography images demonstrate a sizeable filling defect (black arrows) adjacent to the metallic aortic valve (red arrow), consistent with a vegetation and perivalvular density and indicative of infective endocarditis. CT, computed tomography.

c. Hardware failure (wire, plates, and screws) and malposition

Hardware failure and malposition of sternal fixation devices occur in 1%–5% of post-sternotomy cases, often caused by mechanical stress, improper placement, or patient-related factors such as osteoporosis, obesity, infection, or inadequate surgical technique.[32,33] Fractured or displaced wires, loose screws, or misaligned plates can weaken sternal stability, increasing the risk of cardiac injury, sternal dehiscence, poor healing, chronic pain, or soft tissue irritation. Chest radiography helps identify broken or migrated wires, whereas CT offers a more detailed view of wire placement and related complications such as dehiscence or infection (Figure 11 and Supplementary Figure 5).[32,33] Management varies according to severity. Symptomatic broken or migrated wires may need removal, and substantial sternal instability may require re-wiring or alternative fixation to restore structural integrity and support healing.[35]

2.5. Device-related complications

Device-related complications can occur due to malfunction, migration, infection, or mechanical issues associated with medical devices utilized during or after cardiothoracic surgery. Early identification and appropriate management are crucial to ensuring patient safety and recovery.

a. Valve prosthesis complications

Prosthetic valve dysfunction, which may present as stenosis, paravalvular leaks, or dehiscence, can arise from mechanical failure, thrombosis, pannus formation, or IE. Mechanical valves are particularly prone to thrombosis if anticoagulation is suboptimal, whereas bioprosthetic valves are more susceptible to structural deterioration (e.g., leaflet tearing, calcification) and non-structural issues (e.g., paravalvular leaks from tissue ingrowth).[31,36] Distinguishing between structural and non-structural dysfunction via imaging is critical to prevent heart failure or thromboembolic events. Echocardiography is the primary diagnostic tool, with TEE offering enhanced visualization of small thrombi and leaks. CT cine reconstructions depict leaflet excursion, opening angle, and restricted mobility, enabling the detection of structural degeneration, flail segments, or stuck leaflets. Density analysis further differentiates pannus (mixed tissue >145 HU) from thrombus (low attenuation <90 HU), whereas high-resolution 3D maps reveal paravalvular abscesses, pseudoaneurysms, fistulae, and dehiscence and quantify cusp, leaflet, and annular calcification–an early marker of bioprosthetic failure (Figures 8, 9).[31] Cardiac MRI offers a radiation-free problem-solving tool that can accurately measure paravalvular leak regurgitant volume and jet direction when echocardiography is equivocal, and LGE with T1/T2 mapping characterizes peri-annular inflammation or abscess when CT findings are indeterminate.[31,36,37] Treatment depends on the underlying problem: anticoagulation for thrombosis, targeted antibiotics for IE, and surgical revision or valve

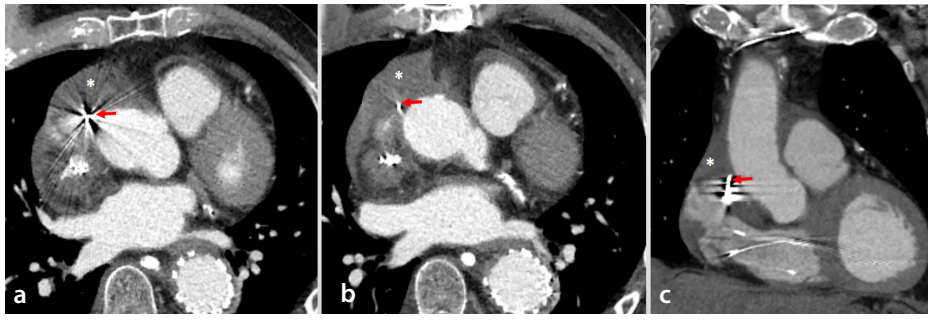

Figure 13. A 66-year-old man with a history of type B aortic dissection, status post-implantable cardioverter-defibrillator placement. **(a, b)** Axial and **(c)** coronal CT images demonstrate right atrial perforation by the pacer lead (red arrow) with associated peri-atrial hemorrhage and fat stranding (*). CT, computed tomography.

replacement for severe dysfunction, substantial leaks, or ongoing infection.[36]

b. Pacemaker or implantable cardioverter defibrillator complications

Pacemaker or implantable cardioverter defibrillator malfunction poses considerable risks for patients who rely on these devices for arrhythmia management. Common causes include lead displacement, battery depletion, infection, and pulse generator failure. Lead displacement may cause syncope, dizziness, or heart block, and infections typically present with local erythema, swelling, or fever. Battery depletion, often identified during routine device interrogation, can lead to sudden pacing or defibrillation failure if left unresolved.[37,38] Prompt recognition and intervention are vital to avert life-threatening arrhythmias, sepsis, or total device failure. Chest radiography is the first-line imaging modality to assess lead positioning, detect fractures, and identify generator or lead migration. Echocardiography helps evaluate intracardiac lead placement and potential complications such as pericardial effusion or hemopericardium. CT provides a more detailed view of device and lead placement, as well as related issues such as hematomas or infections (Figure 13).[37,38] Management depends on the underlying problem. Displaced or fractured leads often need repositioning or replacement, whereas infections require antibiotic therapy and, in severe cases, device removal.[38]

c. Left ventricular assist device complications

Acute complications, including bleeding, infection, right ventricular failure, or thrombosis, following left ventricular assist device (LVAD) implantation can affect up to 15%–30% of patients within the first month. Thrombosis is a key concern, often triggered by biodebris–microscopic particles from mechanical components–that cause microemboli, endothelial damage, and clot formation. These factors increase the risk of thromboembolic events, pump thrombosis, and device malfunction.[39] Clinically, patients may present with hemodynamic instability, worsening heart failure, or neurologic deficits. Sudden power surges or suction alarms often suggest pump thrombosis or biodebris accumulation; elevated D-dimer further supports the diagnosis. Although aortic root thrombi usually appear later, they can emerge early, warranting vigilant monitoring.[39,40]

Imaging is crucial for diagnosing LVAD-related complications. Echocardiography evaluates device flow, detects thrombus formation, and assesses ventricular function and pump integrity in real time. Although CT offers a detailed view of device positioning, structural connections, and associated issues such as biodebris, thrombosis, infection, or mechanical failure, CT images are often limited by beam-hardening and streak artifacts from the device components (Figure 14 and Supplementary Figures 7, 8).[39] Management depends on the underlying cause: anticoagulation therapy for pump thrombosis, targeted antibiotics (and possible device removal) for infection, and transfusion or surgical intervention for bleeding. Device exchange or repair may be necessary for mechanical failures. Current protocols emphasize a multidisciplinary approach–optimizing anticoagulation, employing endovascular interventions, and conducting structured imaging surveillance–ensuring close collaboration between radiologists and cardiologists for timely diagnosis and treatment.[39]

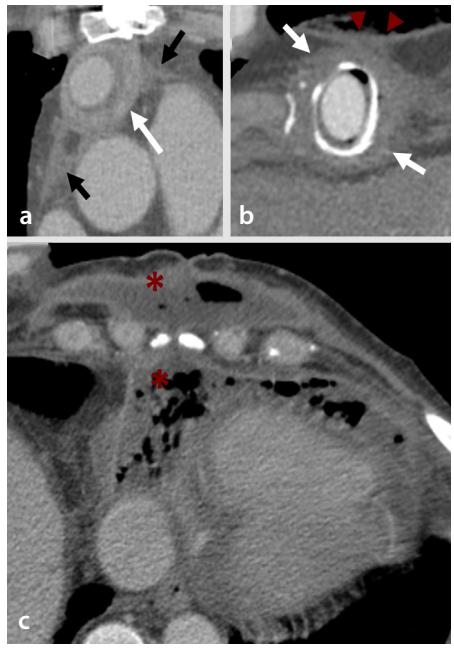

Figure 14. A 65-year-old man with left ventricular assist device (LVAD)-specific infection. **(a, b)** Axial chest CT images demonstrate irregular wall thickening of the LVAD outflow cannula with surrounding fat stranding (white arrows), ill-defined fluid collection (black arrows), and overlying skin thickening (arrowheads), consistent with an LVAD-related infection. **(c)** Status post-LVAD explant and heart transplant, with recurrent air and fluid collections in the mediastinum and chest wall (asterisk), compatible with an abscess. CT, computed tomography.

2.6. Postoperative syndromes

Postoperative syndromes encompass various symptoms and conditions following cardiothoracic surgery, affecting recovery and quality of life. Early recognition and management are crucial for optimizing outcomes.

a. Post-pericardiotomy syndrome

Post-pericardiotomy syndrome (PPS) typically develops days to weeks after cardiothoracic surgery involving pericardial manipulation, triggered by an autoimmune or inflammatory response. Affecting 9%–29% of adult patients, PPS presents with fever, pleuritic chest pain, pericardial or pleural effusions, and elevated inflammatory markers.[41] Imaging is key, and chest radiographs may reveal an enlarged cardiac silhouette or pleural effusions, whereas echocardiography confirms effusions and rules out tamponade. CT and MRI can further detail pericardial edema and complex fluid collections (Figure 15).[42] Management centers on reducing inflammation and symptoms, with non-steroidal anti-inflammatory drugs (NSAIDs) as the first-line therapy and colchicine often added to prevent recurrence. Corticosteroids may be used in refractory cases, and pericardiocentesis is indicated for large or symptomatic effusions.[41]

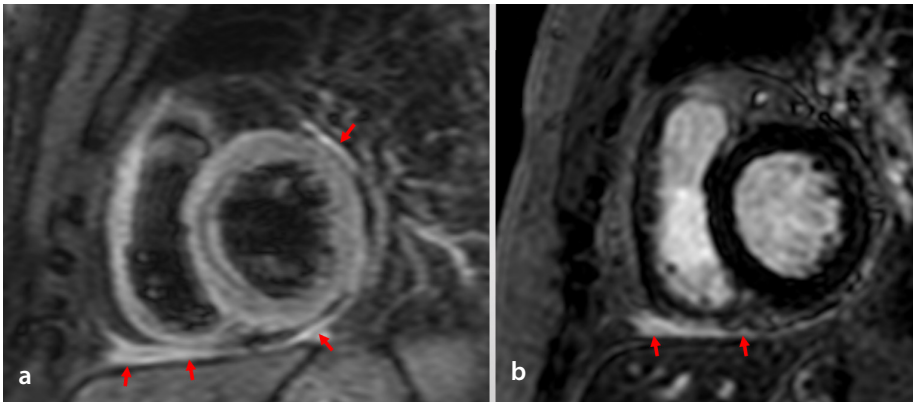

Figure 15. A 62-year-old male status post-coronary artery bypass grafting 2 months ago, presenting with prolonged pleuritic chest pain and mild fever. **(a)** Short-axis short tau inversion recovery image demonstrates patchy pericardial thickening with hyperintensity, suggestive of edema (arrows). **(b)** Late gadolinium enhancement images reveal patchy pericardial enhancement within the basilar inferior pericardium (arrows), consistent with pericardial inflammation.

b. Post-sternotomy syndrome

Post-sternotomy syndrome is a complex complication after median sternotomy, marked by chronic pain, inflammation, and delayed wound healing that impair recovery and quality of life. It affects about 30% of patients, with chronic pain often stemming from surgical trauma to the sternum, intercostal nerves, or soft tissues, whereas inflammation may result from tissue irritation, infection, or reactions to sternal hardware.[43] Imaging–using chest radiography, CT, bone scans, or MRI–is essential for assessing sternal integrity and detecting issues such as fractures, hardware loosening, dehiscence, osteomyelitis, or mediastinitis.[32] Treatment focuses on pain control and functional recovery through medications (NSAIDs, opioids, gabapentin, or pregabalin), physical therapy, rehabilitation, and psychological support.[43]

2.7. Neurological complications

Neurological complications after cardiothoracic surgery, including stroke, transient ischemic attacks, cognitive dysfunction, seizures, and nerve injuries, can severely affect recovery and quality of life. Early detection and prompt management are crucial for optimal outcomes.

a. Phrenic nerve injury

Phrenic nerve palsy complicates 1%–5% of cardiothoracic surgeries, especially those with extensive pericardial manipulation. It typically results from direct nerve injury, thermal damage, or traction, leading to diaphragmatic dysfunction. Patients may experience dyspnea, orthopnea, or reduced exercise tolerance, especially if they have underlying cardiopulmonary issues.[44] Chest radiographs usually show an elevated hemidiaphragm, and a fluoroscopic sniff test reveals paradoxical movement. Ultrasound provides real-time evaluation of diaphragm motion, whereas CT can detect secondary effects such as atelectasis or reduced lung volume, and MRI offers detailed soft tissue assessment in complex cases.[3] Most patients benefit from conservative measures such as breathing exercises and physical therapy, though non-invasive ventilation may be needed for considerable respiratory compromise. Surgical diaphragmatic plication is reserved for refractory cases to improve respiratory mechanics and quality of life.[44]

b. Recurrent laryngeal nerve injury

Recurrent laryngeal nerve injury complicates 3%–10% of neck and upper mediastinal surgeries, especially in oncologic and reoperative cases.[45] It most commonly occurs during thyroidectomy, parathyroidectomy, esophagectomy, or thoracic aortic procedures. Unilateral injury leads to hoarseness, vocal fatigue, and aspiration, whereas bilateral damage can cause severe airway obstruction requiring urgent care. Diagnosis is based on clinical evaluation and direct vocal cord visualization via flexible laryngoscopy, with CT and MRI used to detect compressive lesions. Management ranges from voice therapy and speech rehabilitation to injection laryngoplasty, and in persistent cases, advanced surgical options such as medialization thyroplasty, arytenoid adduction, or reinnervation may be necessary.[45]

c. Brachial plexus injury

Brachial plexus injury is a rare but serious complication of cardiothoracic surgery, often due to excessive arm traction, poor positioning, or direct trauma during sternal retraction. Prolonged hyperextension or aggressive retraction increases the risk.[46] Patients may experience weakness, paresthesia, or pain in the shoulder, arm, or hand, and severe cases can lead to paralysis, muscle atrophy, and limited motion. Diagnosis relies on electromyography, nerve conduction studies, and MRI–the gold standard for detecting nerve damage. CT myelography and ultrasound can also be useful. Preganglionic (root-level) injury manifests as root avulsion, empty nerve root sleeves, or pseudomeningocele; post-ganglionic injury shows nerve thickening, discontinuity, or neuroma with focal T2 hyperintensity. Chronic denervation is reflected by muscle edema progressing to fatty atrophy of the paraspinal and shoulder girdle muscles.[46,47] Treatment includes physical therapy, pain management, and surgery for severe cases, with orthotic devices providing additional support.[46]

In conclusion, non-pulmonary postoperative complications after cardiothoracic surgery can considerably affect recovery and morbidity if not identified and treated promptly. Imaging is essential for detecting and monitoring these conditions, including cardiovascular issues (e.g., graft failure, pericardial effusions), infections (e.g., mediastinitis, sternal osteomyelitis), and device-related complications (e.g., lead displacement, anastomotic leaks) (Table 1). Advanced techniques such as cardiac MRI and CTA have transformed non-invasive evaluation, enabling precise anatomical and functional assessments of vascular conduit stenosis and prosthetic valve dysfunction. These insights facilitate timely, targeted interventions.

Footnotes

Conflict of interest disclosure

Furkan Ufuk, MD, is Section Editor in Diagnostic and Interventional Radiology. He had no involvement in the peer-review of this article and had no access to information regarding its peer-review. Other authors have nothing to disclose.

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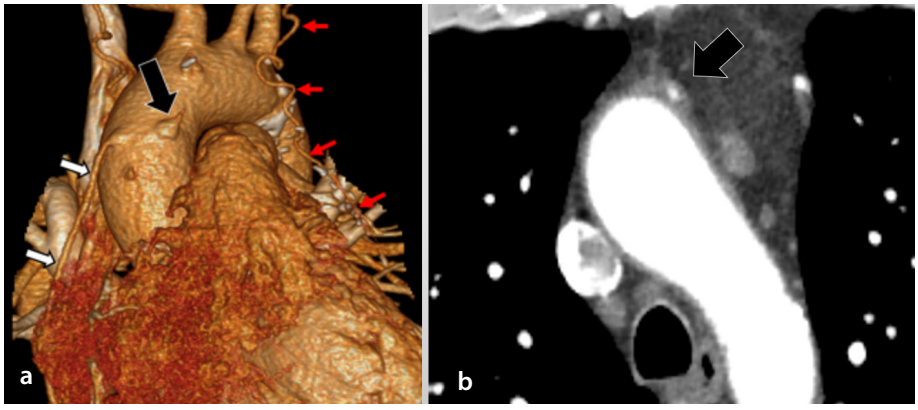
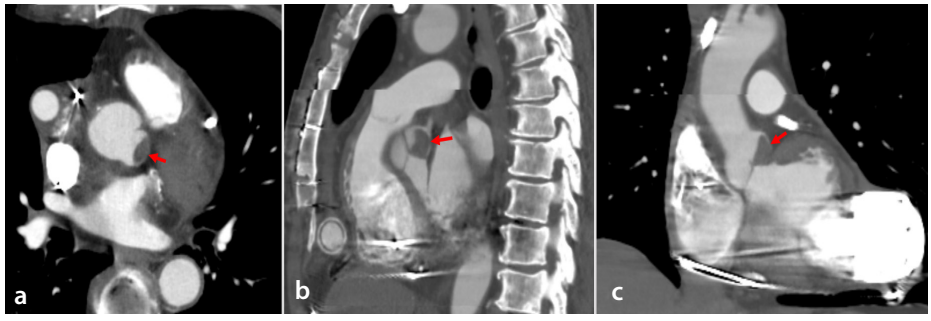
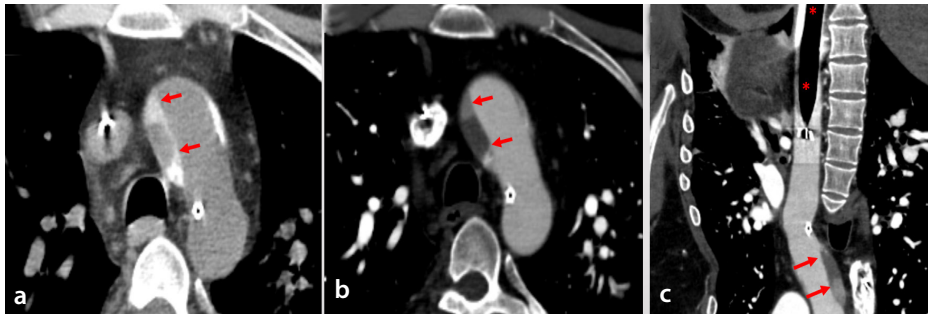


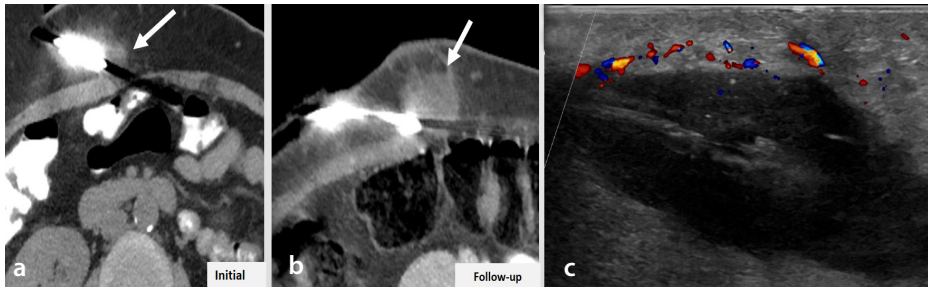
Supplementary Figure 1. A 73-year-old male status post-coronary artery bypass grafting. **(a)** Coronal three-dimensional (3D) and **(b)** axial computed tomography angiography images demonstrate a completely thrombosed saphenous vein graft (SVG) (white arrows), with only the residual most proximal portion of the graft remaining (black arrow). A patent SVG graft to the right coronary artery is seen arising from the right side of the ascending aorta (white arrows), along with a partially imaged left internal mammary artery graft (red arrows).



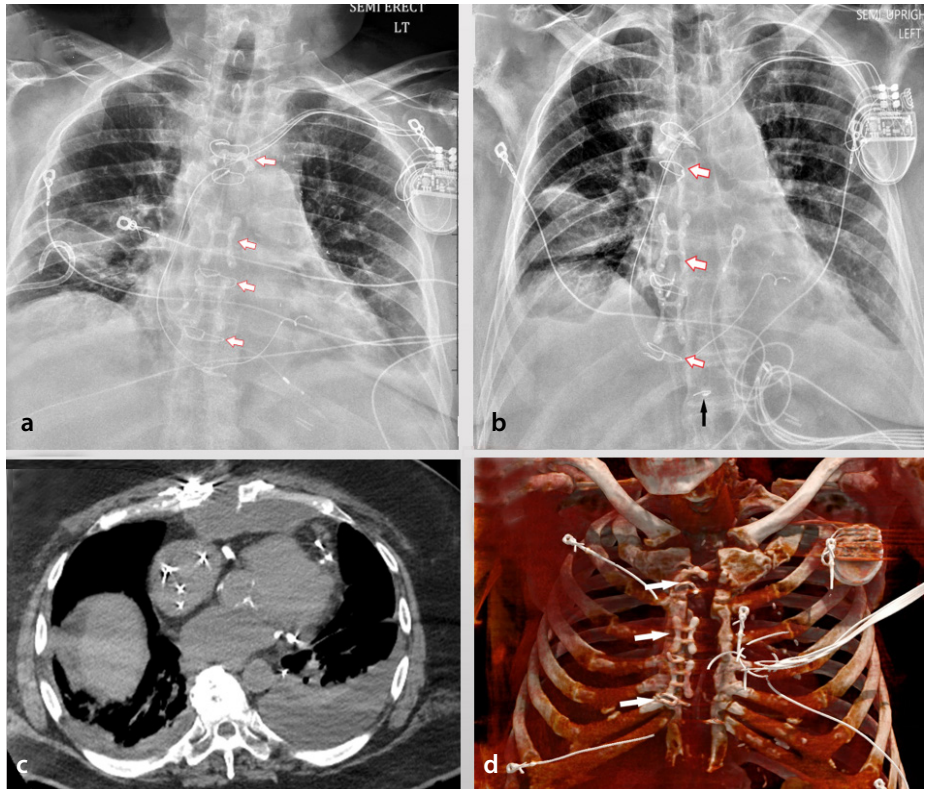
Supplementary Figure 2. A 55-year-old male status post-left ventricular assist device placement. **(a)** Axial, **(b)** sagittal, and **(c)** coronal CT angiography images demonstrate a hypodense lesion within the left sinus of Valsalva, consistent with thrombus (red arrows). CT, computed tomography.



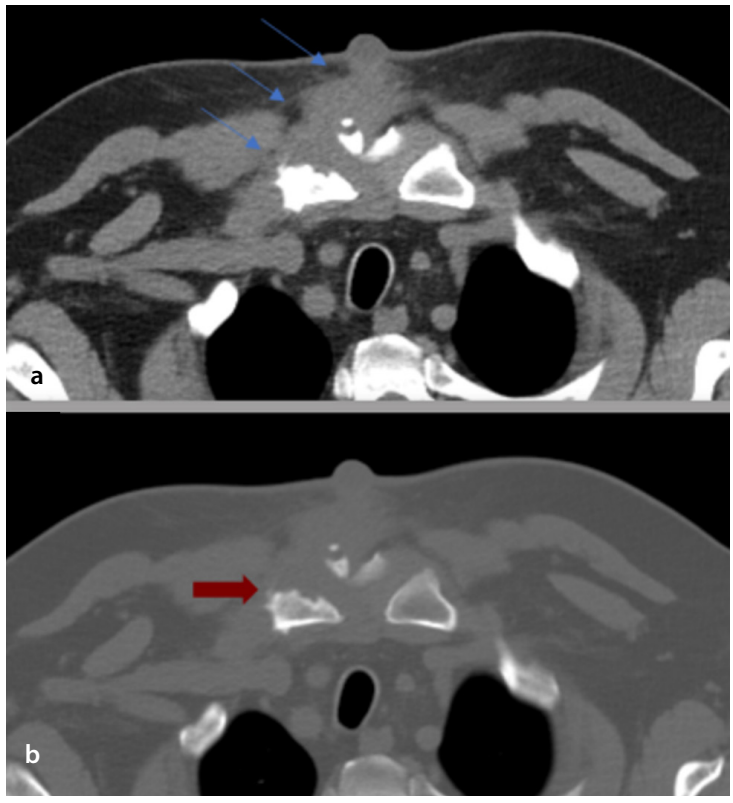
Supplementary Figure 3. A 65-year-old female status post-intra-aortic balloon pump placement. **(a)** Axial non-contrast, **(b)** axial arterial phase, and **(c)** aortic multiplanar computed tomography angiography images demonstrate an intimal defect within the left aortic arch, consistent with a mural hematoma (red arrows).



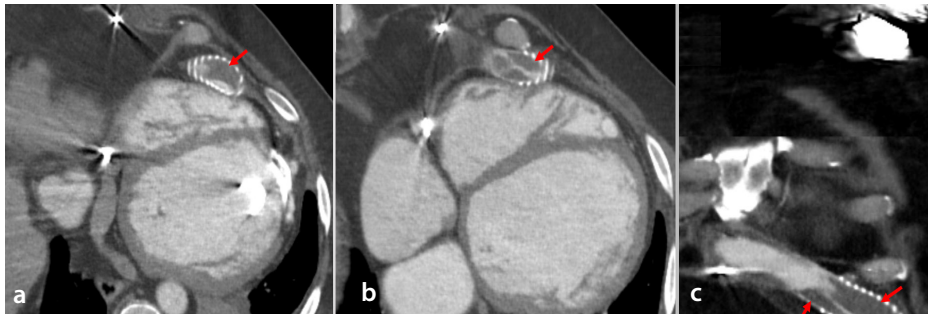
Supplementary Figure 4. A 65-year-old female status post-left ventricular assist device (LVAD) placement. **(a)** The initial axial chest computed tomography (CT) image demonstrates an unremarkable appearance of the LVAD driveline. **(b)** Follow-up axial chest CT image reveals increased soft tissue density (arrows) surrounding the LVAD driveline in the anterior abdominal wall, and **(c)** corresponding ultrasound image shows a fluid collection with surrounding echogenic fat and hyperemia, consistent with an abscess.



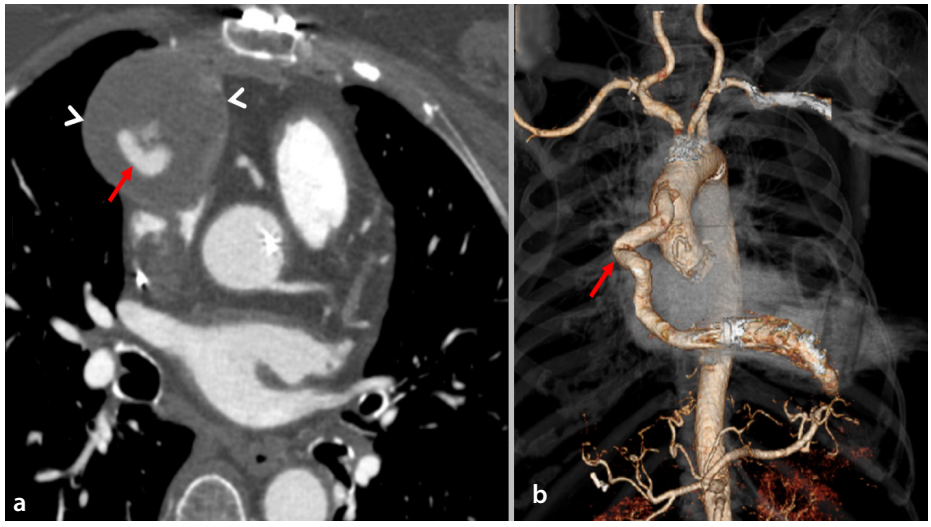
Supplementary Figure 5. A 79-year-old male status post-coronary artery bypass grafting. **(a)** Early postoperative chest radiograph demonstrates aligned sternotomy wires and plates (arrows) with expected postoperative changes and devices. **(b)** Chest radiograph obtained 1 day postoperatively shows marked rightward displacement of the sternal wires and plates (white arrows), representing a change from the initial postoperative radiograph. A fractured lower wire is also noted (black arrow). **(c)** Axial chest computed tomography (CT) image reveals bilateral pleural effusion, sternal dehiscence, and a peristernal fluid collection. **(d)** A three-dimensional (3D) volume-rendered chest CT image demonstrates sternal dehiscence with displaced sternal wires and plates (arrows).



Supplementary Figure 6. A 49-year-old male status post-heart transplantation. **(a)** Axial chest computed tomography image with mediastinal window and **(b)** bone window settings demonstrates a localized abscess surrounding the sternal manubrium, with an anterior sinus tract extending to the skin (thin blue arrows). Sclerosis and erosive changes involving the right clavicular head (red arrow), and to a lesser extent, the sternal manubrium, are better visualized on the bone window image, suggesting osteomyelitis.



Supplementary Figure 7. A 61-year-old man with left ventricular assist device (LVAD) outflow cannula stenosis due to biodebris. **(a, b)** Axial and **(c)** coronal computed tomography images demonstrate considerable luminal stenosis of the LVAD outflow cannula caused by large biodebris (arrows).



Supplementary Figure 8. A 63-year-old woman with left ventricular assist device (LVAD) outflow cannula stenosis due to kinking. **(a)** Axial computed tomography (CT) angiography and **(b)** three-dimensional volume-rendered CT angiography images demonstrate luminal stenosis of the LVAD outflow cannula caused by kinking (arrows). Surrounding fluid around the outflow cannula is also noted (arrowheads), which may suggest associated inflammation or serous collection.