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REVIEW

Cardiovascular findings on cross-sectional imaging: spectrum of incidental and critical findings and clinical relevance for the abdominal radiologist

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Abstract
Although not the primary focus of the exams, cardiovascular structures are included to some extent on all abdominal or whole-body cross-sectional studies. Cardiovascular findings often present incidentally and may range from chronic to acute and emergent pathologies. Among the most common cardiovascular findings are the presence of cardiac calcifications, most commonly coronary, which correlate with the presence of coronary artery and valvular disease. Signs of myocardial ischemia, both acute and chronic, and its complications may also be visualized. Cardiac filling defects most commonly represent thrombus and are associated with systemic arterial embolic complications. Pericardial findings often manifest as effusion or thickening, which may lead to hemodynamic consequences visible at imaging. Incidental pulmonary emboli and systemic venous thrombi may be incidentally detected, particularly in hospitalized and oncologic patients, and warrant immediate attention. This review will highlight the appearance of common and important incidental cardiovascular findings and related pitfalls and discuss reporting and follow-up recommendations relevant to the abdominal radiologist.

Keywords Cardiac · Incidental · Cross-sectional

Introduction

Abdominal radiologists routinely encounter the heart and other thoracic cardiovascular structures, whether on imaging that includes the entire chest (i.e. through oncologic staging exams, trauma assessment, etc.) or via dedicated abdominal cross-sectional imaging. While a vast majority of these studies are ordered to investigate pathologies outside the cardiovascular system, these structures are still included in the field of view of the images. As with studies in any part of the body, incidental findings may arise that may warrant emergent or routine follow-up and potentially cause uneasiness and confusion to those not familiar with those findings and their management.

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Reportable incidental cardiac findings are frequently encountered on routine chest CTs with approximately 60% of scans demonstrating at least one cardiac or pericardial finding. Most commonly, these findings involve direct or indirect signs of coronary artery disease occurring in up to 50% of patients in one series [1]. Other commonly encountered findings include valvular calcifications, cardiac devices and post-surgical changes, chamber enlargement, and pericardial disease. Outside of the heart, pathologies involving the thoracic aorta, pulmonary arteries, and systemic veins may also be seen. Although most data regarding the incidence of these findings are derived from dedicated chest CTs, it is plausible that similar rates of findings occurring in the lower chest could be extended to abdominal CTs.

Despite their high prevalence, cardiovascular findings are frequently underreported, particularly among those with less dedicated cardiovascular imaging training [2, 3]. While multifactorial, this is in part due to the challenge of detecting findings on routine studies not optimized for the evaluation of cardiovascular structures. For example, routine CTs are acquired without electrocardiographic (ECG) gating and thus image acquisition is not synchronized to the cardiac cycle. This may produce considerable motion

artifact, particularly of the heart and aortic root. Furthermore, contrast administration in most single-phase, routine cross-sectional imaging is timed for the portal venous phase to optimize enhancement of the abdominal organs. However, cardiac structures are often best evaluated in the arterial phase with higher injection rates and contrast timing (i.e., timing bolus or bolus tracking at the structure of interest) to optimize contrast opacification [4, 5]. Lastly, assessment of cardiovascular structures is often performed in specialized planes other than traditional axial, coronal, or sagittal planes. For example, the heart is traditionally better evaluated in cardiac short axis or four chamber views while double-oblique-, through-plane images are necessary to accurately assess the aorta. While specialized multiplanar reconstructions can still be performed with routine CT data sets, this may be time consuming and lack of ECG-gating and image sets of greater slice thicknesses may produce considerably degraded reformatted images.

While routine CTs may accurately detect incidental cardiovascular findings, further imaging evaluation may be warranted for better characterization. Further assessment may be performed with repeat non-gated CT in a different contrast phase (i.e. timed for opacification of the pulmonary arteries or systemic veins), ECG-gated CTA, cardiac MRI, echocardiography, or angiography. While complete discussion of these modalities is beyond the scope of this article, their utility, strengths and weaknesses are summarized in Table 1 and discussed as appropriate throughout this article.

The remainder of this article will illustrate the appearance of common and important incidental cardiovascular findings on routine cross-sectional imaging with potential pitfalls and discuss reporting and follow-up recommendations and management. These are summarized in Table 2.

Cardiac calcifications

Coronary calcifications

Coronary artery calcifications, a marker of atherosclerotic disease, are among the most commonly described incidental cardiovascular findings on routine CT. Depending on the patient cohort and exam indication, coronary artery calcium has been observed in up to half of patients undergoing routine chest CT for non-cardiovascular indications [1, 6]. Although a high degree of coronary calcium does not necessarily translate to the presence of a significant stenosis, the value of coronary calcium is in its prediction of cardiovascular events and mortality beyond traditional clinical cardiovascular risk factors [7]. Greater degrees of coronary calcium have been associated with higher rates of cardiovascular events and all-cause mortality [8]. Conversely, the

Table 1 Modalities for follow-up of cardiovascular findings

Modality	Pathologies evaluated	Strengths	Weaknesses
Echocardiography (TTE and TEE)	– Chamber size and function – Valvular function – Pericardial disease	– No ionizing radiation – Portable/lower cost – Wide availability – Assess cardiac function and hemodynamics – High temporal resolution – Non-invasive (TTE)	– Poor image quality in poor acoustic windows – Operator dependence – Limited evaluation of extra-cardiac structures – Invasive (TEE)
Angiography	– Coronary disease (left heart) – Pulmonary hypertension (right heart)	– Assess hemodynamics – Excellent spatial resolution/accurately – Ability to intervene/treat	– Invasive – Contrast exposure – Limited evaluation of extra-coronary structures – Ionizing radiation
Coronary/cardiac computed tomography	– Aortic disease – Pulmonary embolism – Systemic veins – Coronary disease	– Evaluation of extra-cardiac structures – Excellent spatial resolution – Non-invasive – Assess hemodynamics (i.e. CT-FFR)	– Low temporal resolution – Ionizing radiation – Contrast exposure
Magnetic resonance imaging	– Chamber size and function – Cardiomyopathy assessment – Valvular function – Pericardial disease – Aortic disease – Systemic veins	– Soft tissue and myocardial/scar assessment – Assess cardiac function and hemodynamics – No ionizing radiation – Non-invasive	– MRI contraindications (devices, claustrophobia) – Long exam time – Limited spatial resolution – Limited availability/high cost

TTE Transthoracic echocardiography, *TEE* transesophageal echocardiography, *CT-FFR* computed tomography fractional flow reserve

Table 2 Summary of incidental cardiovascular findings at cross-sectional imaging

Finding	Significance	Management/follow-up
Coronary artery calcifications	– Presence of cardiovascular events and mortality – Absence suggests against presence of significant obstructive coronary disease	– Thorough cardiovascular risk assessment – Risk factor modification – Possible further coronary disease workup (coronary CTA, stress testing, angiography)
Aortic valve calcifications	– Association with aortic stenosis	– Echocardiography for aortic valve function
Mitral calcifications	– *Mitral annulus* correlation with coronary disease; associated with worse cardiovascular outcome – *Mitral valve leaflet* associated with mitral valve sclerosis/stenosis	– *Mitral annulus* cardiovascular risk assessment – *Mitral valve leaflet* echocardiography for mitral valve function
Heart/chamber enlargement	– Sign of underlying cardiac pathology (e.g. right heart failure, valvular dysfunction, cardiomyopathy)	– Echocardiography for chamber and valvular function assessment
Thoracic aorta dilatation	– Increased risk of rupture with increasing aorta size	– 6–12 month CTA/MRA follow-up or referral for surgical repair depending on size
Myocardial ischemia (myocardial hypoenhancement, thinning/aneurysm, fat deposition, calcification)	– Presence of underlying coronary artery disease ± myocardial infarction	– Risk factor modification/secondary prevention – Possible angiography or coronary CTA for coronary assessment – Possible CMR for possible viability assessment – Anticoagulation for thrombus
Cardiac mass	– *Thrombus* potential source of embolism – *Metastasis* sign of advanced metastatic disease	– Echocardiography or CMR to confirm presence of mass – CMR for further characterization – Anticoagulation for thrombus – Possible surgical referral for mass
Pericardial effusion/thickening	– *Pericardial effusion* tamponade physiology and hemodynamic compromise if large – *Pericardial thickening* possible constrictive physiology	– Echocardiography or CMR to assess tamponade or constrictive physiology
Pulmonary artery enlargement	– Marker of pulmonary arterial hypertension	– Echocardiography to estimate pulmonary pressures, assess left heart and valvular function – Possible angiography to confirm pulmonary artery pressures
Pulmonary artery filling defect	– Presence of pulmonary embolism	– Confirmatory CT or VQ scan in equivocal cases – Anticoagulation
Systemic venous filling defect	– Presence of venous thrombus	– Confirmatory ultrasound or CT/MRA in equivocal cases – Anticoagulation – Consider central catheter removal

CTA computed tomography angiography, *MRA* magnetic resonance angiography, *CMR* cardiac magnetic resonance imaging, *VQ* ventilation perfusion

absence of detectable coronary calcium is associated with very low risk of future cardiovascular events in asymptomatic individuals [9].

Traditionally, coronary artery calcium is detected using dedicated non-contrast ECG-gated CT of the heart. Coronary calcium is quantitatively assessed by a semi-automated Agatston score, which is calculated based on the density and extent (i.e. number of pixels) in which calcium is detected. Based on the Agatston score, patients can be stratified into coronary artery disease risk categories. However, quantification of coronary calcium on routine non-gated CTs has also shown high correlation with gated calcium score CTs and maintains strong prognostic value in the prediction of cardiovascular events and mortality (Fig. 1) [10].

Given the demonstrated prognostic information of coronary calcium on routine studies, it is recommended that the presence of coronary calcium be described on all CTs regardless of the patient's risk status [11, 12]. While

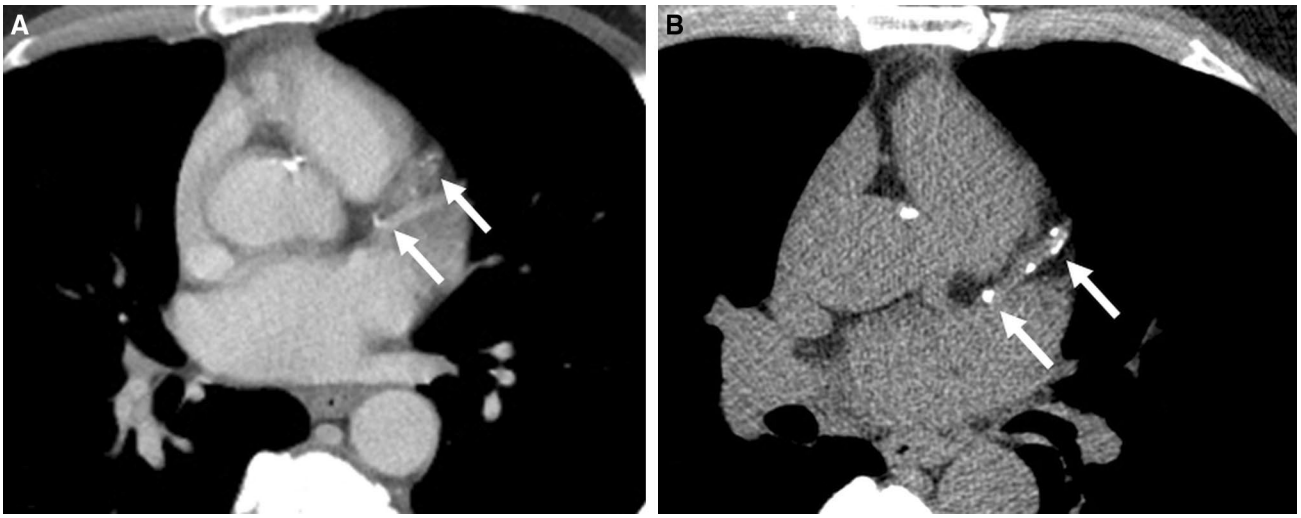

Fig. 1 Coronary artery calcifications. **a** Contrast enhanced CT performed for generalized abdominal pain demonstrates moderate scattered coronary calcifications (arrows) within the visualized left anterior descending artery. **b** Subsequently performed non-contrast ECG gated coronary calcium score CT better demonstrates these calcifications (arrows). The patient's Agatston score was 158, compatible with a moderate amount of coronary calcium

Agatston scores can be calculated from routine studies, this is time consuming in a high-volume practice and requires specialized computational software. Rather, visual assessment of the degree of coronary calcium (i.e. none, mild, moderate, or severe) has been shown to be accurate, reproducible, and still provide useful prognostic assessment [13, 14]. Additionally, the presence of severe coronary calcifications should prompt evaluation for other signs of myocardial ischemia, discussed later. While precise management algorithms are lacking regarding next steps after incidental detection of coronary calcium, its detection is likely beneficial in identifying patients for possible lifestyle and other risk factor modifications. The need for additional stress testing or coronary angiography should only be pursued after a complete clinical assessment is made.

Aortic valve calcifications

While not as prevalent as coronary calcifications, aortic valve calcifications, a potential marker of aortic stenosis, are still a common finding on routine CT studies, particularly in elderly patients, occurring in up to 18–26% of patients [1, 15]. Aortic valve calcifications may be uniform throughout the valve or may spare certain leaflets or commissures. Calcifications may also be seen with non-calcified valvular thickening, which further contributes to reduced valvular function (Fig. 2). Valvular calcification should not be mistaken for root calcifications which do not involve valve leaflets but occur about the periphery at the annulus, aortic root, or proximal tubular ascending aorta and have less impact on valvular function. In the presence of functionally significant aortic stenosis, secondary signs of left ventricular hypertrophy and ascending aortic dilatation may also be seen.

While the presence of aortic valve calcifications does not necessarily equate to the presence of clinically significant aortic valve stenosis, greater degrees of calcium have been shown to correlate with increased pressure gradients across the aortic valve at echocardiography [15, 16]. Similar to coronary calcium, various measurements of calcium have been reported, both quantitatively using the Agatston score and by visual assessment, with both means correlating with valvular function. Although formal reporting guidelines are lacking, valve calcium should be routinely mentioned given its potential clinical implication. Transthoracic echocardiography, particularly in severe cases, may be warranted to assess valvular function.

Mitral calcifications

Calcifications of the mitral valve apparatus occur at either the mitral annulus, a ring-shaped structure at the base of mitral valve between the left atrium and ventricle, or mitral valve leaflets that are in direct connection with the chordae tendineae and papillary muscles. Mitral annular calcifications (MACs) are more commonly seen than leaflet calcifications [1]. MAC may range from finely linear and spotty to mass-like and conform to C-shape of the mitral annulus with sparing of the mitral valve leaflets (Fig. 3a). MAC are typically degenerative in nature and asymptomatic although can be associated with mitral valve dysfunction (either stenosis or regurgitation) in severe cases [17]. Rather, the presence of MACs should be reported due to its association with

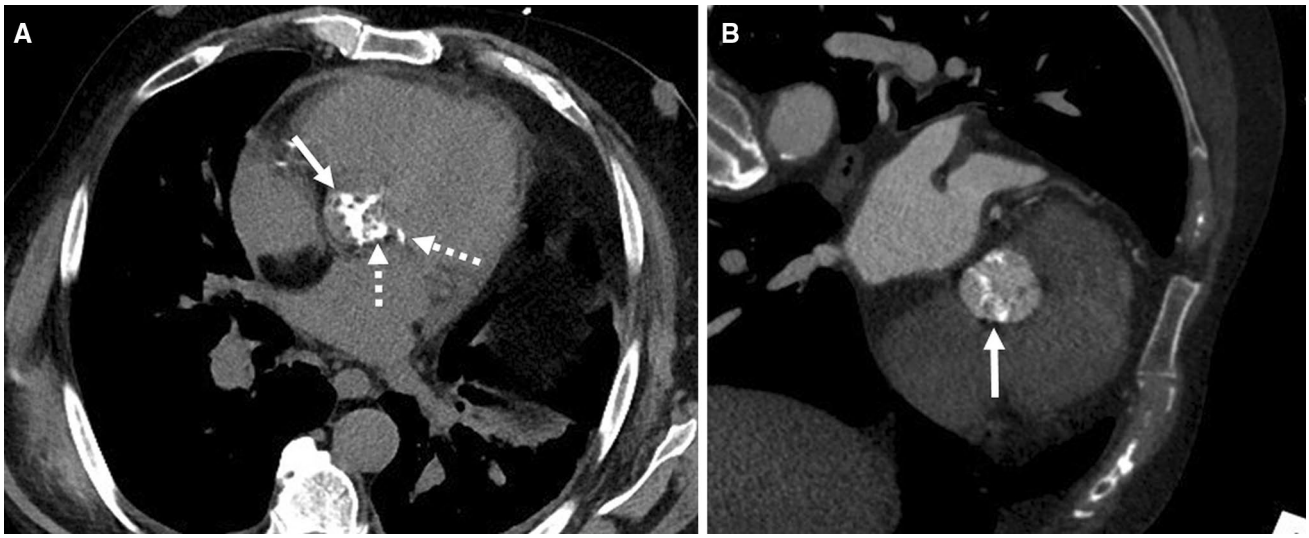

Fig. 2 Aortic valve calcifications. **a** Non-contrast abdominal CT performed for evaluation of leukocytosis demonstrates a heavily calcified aortic valve (solid arrow). Adjacent calcifications not conforming to the valve leaflets represent aortic root and annular calcifications (dashed arrows). **b** Subsequent contrast enhanced ECG gated CT image obtained during peak systolic phase more clearly demonstrates extensive calcification and thickening of the aortic valve (arrow) with reduced valve opening consistent with aortic stenosis

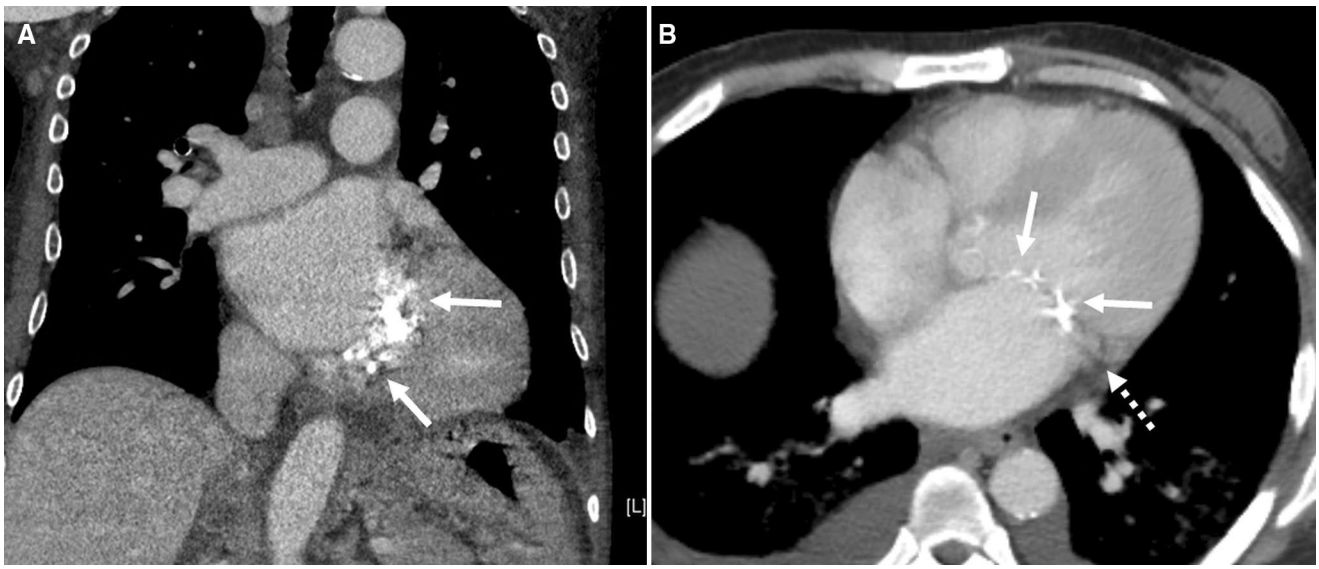

Fig. 3 Mitral calcifications. **a** Contrast-enhanced coronal CT performed for malignancy work-up demonstrates several mitral annular calcifications conforming the entirety of the mitral annulus (arrows). **b** Contrast-enhanced axial CT performed for sepsis evaluation demonstrates calcifications of the mitral valve leaflets (solid arrows) with relative sparing at the annulus (dashed arrow)

coronary artery disease and prognostic implications including prediction of stroke and cardiovascular death [18].

Mitral valve leaflet calcifications (Fig. 3b) can be seen in patients with prior history of rheumatic fever, non-inflammatory calcific disease, and chronic renal failure [19]. Mitral leaflet calcifications have shown to correlate with the presence of mitral valve sclerosis and stenosis with greater degrees of calcification associated with increasing severity of mitral valve disease [20, 21]. Unlike with MACs, echocardiography is more useful to evaluate for associated valvular dysfunction.

Heart chamber and aortic enlargement

Heart chambers

Similar to radiography, heart size can be assessed at cross-sectional imaging by the computed cardiothoracic ratio (CTR), defined as the greatest cardiac diameter divided by the greatest transverse thoracic diameter from inner to inner chest wall on axial images. However, using a CTR cutoff of 0.5, CT has only shown moderate predictive ability of cardiomegaly when referenced to echocardiography, although high negative predictive value (96%) has been shown [22]. It should also be noted than heart size varies with inspiration with greater transverse cardiac diameter observed during expiratory phase images [23]. Individual chamber size can also be assessed at routine CT scans. A recent study by Eifer et al., using cardiac MRI as a reference standard, suggested the following linear dimension criteria for chamber enlargement (male/female): right atrium (transverse): 6.7 cm/6.4 cm, right ventricle (transverse): 6.0 cm/5.7 cm, left atrium (anteroposterior): 5.0 cm/4.5 cm, left ventricle (transverse): 5.8 cm/5.3 cm [24]. While these values have shown overall strong predictive ability for chamber enlargement, they should be considered relative benchmarks rather than absolute cut-offs. Heart size and chamber measurements face some inconsistency on routine cross-sectional imaging due the presence of cardiac motion and variability of size during different phases of the cardiac cycle.

Assessment of cardiac and chamber sizes is important as this may reveal underlying cardiac pathology and be the underlying source of pertinent findings within the abdomen. For example, dilatation of the right atrium and ventricle is commonly seen with right heart overload secondary to left heart failure, pulmonary hypertension, tricuspid regurgitation, or intracardiac or pulmonary shunting. Signs of right heart dysfunction should prompt search for sequelae within the abdomen including hepatic parenchymal abnormalities (ranging from mottled/nutmeg enhancement pattern in early stages to nodular, cirrhotic liver in chronic stages), dilated IVC and hepatic veins, ascites, and other signs of volume overload (Fig. 4). Left atrial enlargement may indicate mitral valve or left ventricular diastolic dysfunction and can be predictive of atrial fibrillation. Thus, a dilated left atrium should prompt search for complications of atrial fibrillation including atrial/atrial appendage filling defects, illustrated later in this article, and sequelae of systemic embolization. The presence of left ventricular dilatation may indicate an underlying cardiomyopathy or volume overload from underlying valvular dysfunction. In the presence of heart or chamber dilatation, echocardiography is often a useful screening tool to evaluate chamber and valve function. Subsequently, further investigation may be warranted such as cardiac MRI to characterize an underlying cardiomyopathy or intracardiac shunt. Right

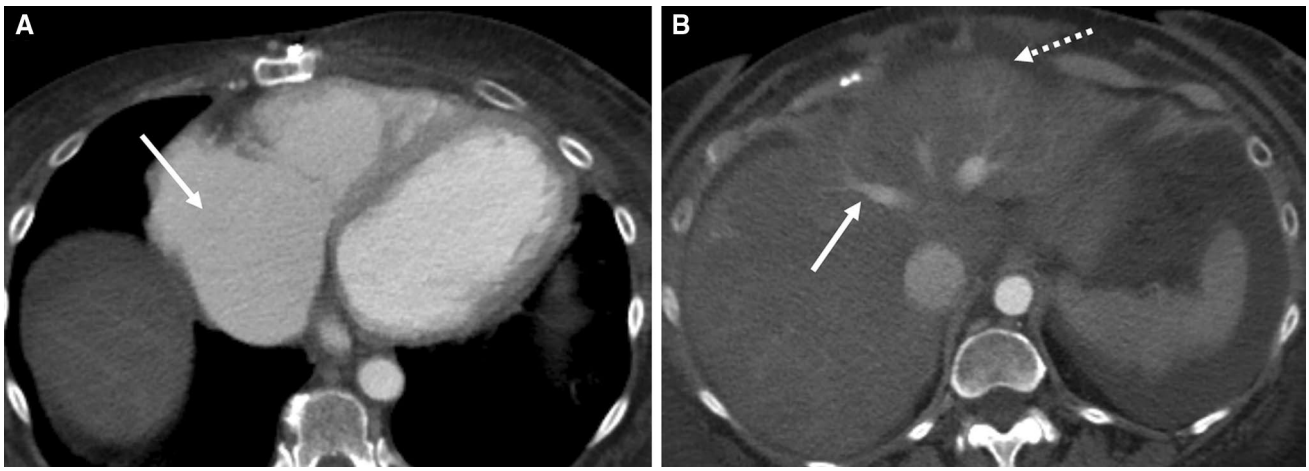

Fig. 4 Right heart failure. **a** Contrast-enhanced axial CT performed for evaluation of abdominal pain and distension demonstrates a dilated right atrium (arrow), a sequela of right heart failure. **b** Axial image through the upper abdomen demonstrates reflux of contrast into the inferior vena cava and hepatic veins (solid arrow), a cirrhotic appearance of the liver (dashed arrow), and ascites secondary to chronic hepatic congestion

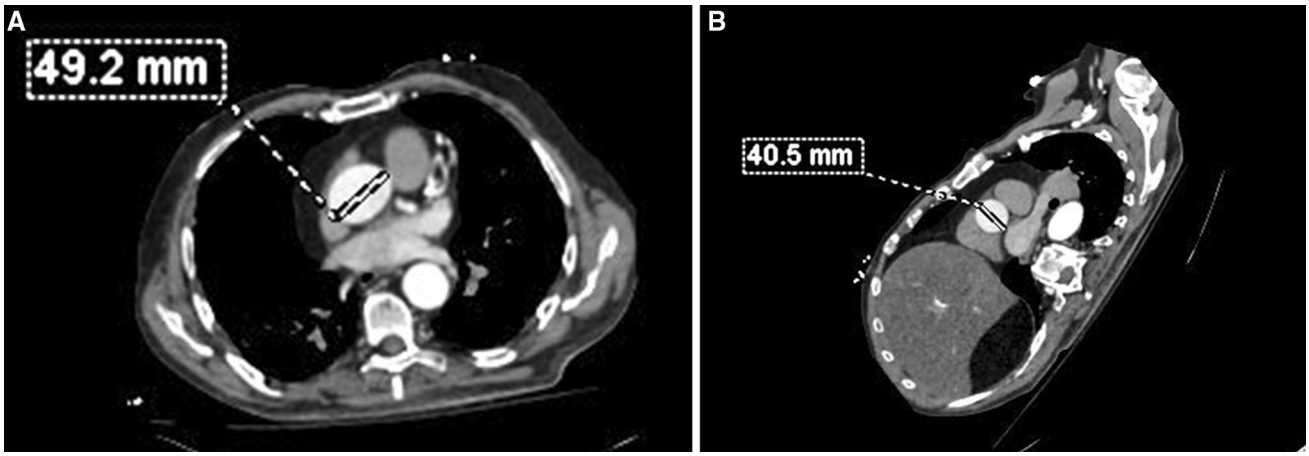

Fig. 5 Thoracic aorta measurement. **a** Contrast enhanced axial CT with the proximal ascending thoracic aorta measured in the true axial plane revealed an apparent diameter of 4.9 cm. **b** When images are reformatted in the plane orthogonal to the aorta at this level, repeat measurement demonstrates true aortic morphology only measuring 4.1 cm

heart catheterization can assess for pulmonary hypertension and right heart pressures.

Thoracic aorta

Incidental thoracic dilatation has been detected in up to 3% of adult patients between 55 and 80 years old undergoing routine chest CT [25]. Thoracic aortic diameter normally depends on age, sex, body surface area, and location along the course of the aorta. For example, average diameter of the ascending and descending thoracic aorta for men are 34.1 ± 3.9 mm and 25.8 ± 3.0 mm, respectively, while average diameters for women are 31.9 ± 3.5 mm and 23.1 ± 2.6 mm, respectively [26]. The term "aneurysm" is used to describe an aortic diameter greater than 150% of normal, which generally occurs for an ascending aorta greater than 5.0 cm and descending aorta greater than 4.0 cm [27]. Caution should be made regarding performing aortic measurements in the conventional axial plane, particularly in an area of aortic tortuosity, as this will lead to overestimation of aortic diameter (Fig. 5). Measurements should be obtained perpendicular to the direction of flow within the aorta, preferably using double-oblique multiplanar reformatted images.

For patients with incidentally discovered thoracic aortic aneurysms not meeting criteria for repair (repair criteria: diameter greater than 5.5 cm and 6.5 cm in the ascending and descending thoracic aorta, respectively, rapid growth, or symptomatic), annual or biannual surveillance is recommended depending on the aneurysm size [27]. Any follow-up imaging should be performed using ECG gating, whether with CT or MRI, as this has shown to reduce pulsation motion artifacts [28]. Although not specifically addressed in clinical guidelines, one should consider immediate follow-up of a dilated aorta partially visualized on abdominal CT for full assessment of the entire thoracic aorta.

Ischemic heart disease

CT may detect the downstream sequelae of coronary disease within the myocardium. Ischemic findings occur in predictable coronary distributions: anterior and septal (left anterior descending artery territory), lateral (left circumflex artery), and inferior (right coronary artery). Affected myocardium will demonstrate hypoenhancement, either subendocardial (partial thickness) or transmural (full thickness), which is best appreciated during arterial phase images (Fig. 6). Myocardial hypoenhancement may indicate ischemic or infarcted myocardium. The use of a narrower window accentuates differential enhancement between normal and ischemic myocardium. True myocardial hypoenhancement should be differentiated from beam hardening artifact from nearby osseous and high density structures that presents in a non-coronary distribution and often is seen extending into adjacent structures. In protocols utilizing delayed images (i.e. cirrhosis, urogram, and most MRI protocols), delayed hyperenhancement may be seen in both acute and chronic ischemia (Fig. 7). Findings suggesting remote myocardial infarction include: subendocardial fat deposition, calcification and myocardial thinning (Fig. 8). Myocardial thinning may be associated with aneurysm or pseudoaneurysm formation. However, focal myocardial thinning confined to

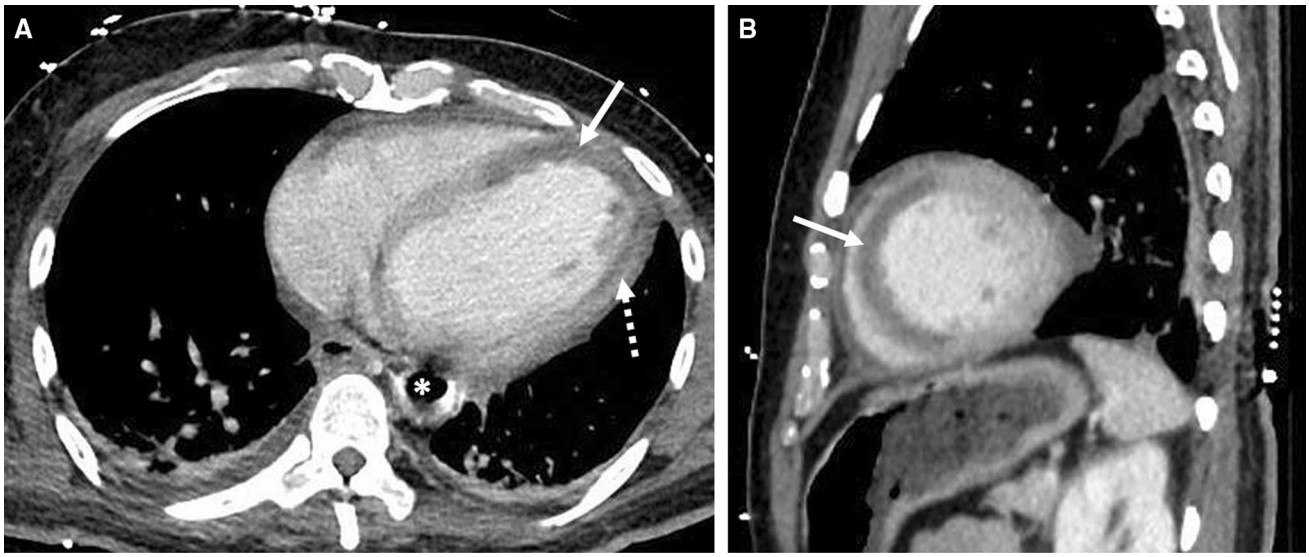

Fig. 6 Acute myocardial infarction. Contrast enhanced axial (**a**) and sagittal (**b**) CT images performed for malignancy staging demonstrate myocardial hypoenhancement, transmural in the mid to apical interventricular septum (solid arrow) and subendocardial within the apex and apical lateral myocardium (dashed arrow). An intra-aortic balloon pump is seen within the descending aorta (asterisks). The patient was recently treated for an ST-elevation myocardial infraction

the left ventricular apex without other findings of ischemia should be considered a normal finding [29]. In the presence of ischemic myocardium, close inspection of nearby areas should be made for thrombus, most commonly located at the left ventricular apex [30].

As ischemia may be clinically silent, signs of ischemia should be reported as these may be the first diagnosis of myocardial infarction or coronary disease in general. Thus, alerting providers of these findings can initiate appropriate cardiovascular work-up and secondary prevention measures. While myocardial thinning is often associated with non-viable myocardium, these areas can potentially be salvageable and therefore further clinical and potential viability assessment with cardiac MRI may be warranted [31]. Myocardial scar may also present clinically as an arrhythmogenic focus. The presence of left ventricle thrombus warrants consideration for anticoagulation [32].

Cardiac masses

Although an uncommon incidental finding, cardiac masses may also be found incidentally on routine studies [1]. The most common cardiac "mass" is thrombus [33]. Thrombi may be found in each of the cardiac chambers with each location associated with various clinical scenarios. As discussed previously, left ventricular thrombi are most often associated with prior myocardial infarction and myocardial scarring or aneurysm. Left atrial thrombi, particularly within the left atrial appendage, can be seen in the patients with atrial fibrillation. However, contrast mixing artifact due to circulatory stasis within the left atrial appendage is also commonly seen in patients with atrial fibrillation and enlarged left atria and may mimic a thrombus. A mixing artifact appears as a more linear and geometric "filling defect" that disappears on more delayed images as opacified contrast circulates through the appendage, while a true appendage thrombus will be more rounded and persist and delayed images (Fig. 9). Thrombi within the right atrium and ventricle are often associated with the central venous catheters and other cardiac devices. However, a prominent crista terminalis, a vertically oriented muscular ridge located at the lateral right atrial wall extending from the inferior to superior vena cava, is a normal anatomic structure and should not be confused for thrombus. Rather, a true right atrial thrombus will appear more rounded and mass-like and will have a shorter craniocaudal extent than a crista terminalis (Fig. 10). Mass-like lesions may be associated with cardiac valves, most commonly vegetations in the setting of infective endocarditis, although are often more difficult to appreciate on routine CT and MR secondary to their small size coupled with cardiac valve motion.

Cardiac neoplasms are most likely to be metastatic and are 20–40 times more common than primary cardiac tumors. Primary malignancies with the highest rates cardiac metastases include mesothelioma, melanoma, lung, lymphoma, and breast [34]. The appearance of cardiac metastases varies with the route of spread. For example, metastases occurring via direct extension will be seen in contiguity with a primary thoracic mass (i.e. pleural mesothelioma and lung)

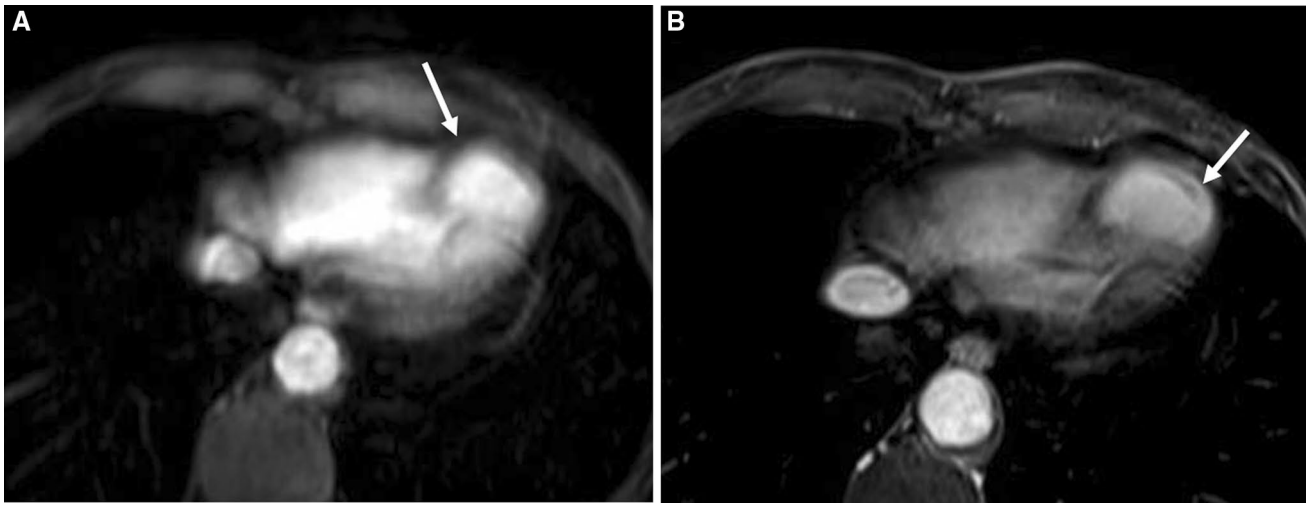

Fig. 7 Delayed myocardial hypoenhancement. **a** Axial MRI image during the late arterial phase performed for the assessment of pancreatitis demonstrates aneurysmal enlargement of the left ventricular apex with associated hypoenhancement (arrow). **b** Corresponding image during the delayed phase demonstrates associated transmural hyperenhancement (arrow) corresponding to the region of scar

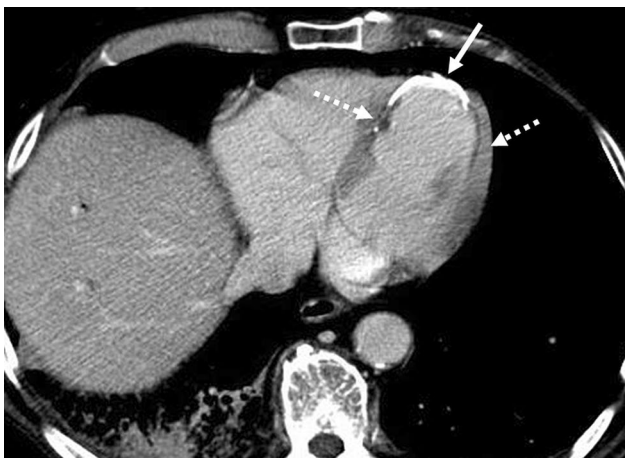

Fig. 8 Chronic myocardial infraction. Contrast-enhanced axial CT performed for trauma assessment demonstrates apical myocardial thinning and calcification of the left ventricular apex and apical septum (solid arrow). Subendocardial fat is seen in the immediately adjacent septal, apical, and lateral myocardium (dashed arrows). Aspiration is seen in the dependent right lower lobe

or via the inferior vena cava into the right heart (i.e. renal cell and hepatocellular carcinomas). Metastases with hematogenous spread will appear as singular or multiple enhancing masses potentially located anywhere within the heart (Fig. 11). Primary cardiac neoplasms are rare, although benign neoplasms account for 75% of primary tumors. In adults, the most common primary cardiac mass in adults is a myxoma, which appears as a circumscribed, heterogenous mass most commonly within left atrium classically attached at the fossa ovalis.

Signs of systemic embolus such as visceral organ infarct or arterial embolism should prompt search for a potential cardioembolic source. While these are most likely to originate from the left heart, emboli from the right heart may still produce systemic embolus via intracardiac or intrapulmonary shunt such as an atrial septal defect (Fig. 12). Given a classic appearance and appropriate setting, routine CT alone may be diagnostic for the evaluation of cardiac masses. However, further imaging either with echocardiography (transthoracic or transesophageal) or cardiac MRI may be needed to confirm the presence of a mass. Due to its superior soft tissue characterization, cardiac MRI is better suited to differentiate cardiac thrombus from neoplasm. Cardiac thrombi warrant consideration for anticoagulation. Depending on symptomatology and effect on cardiac or valvular function, larger masses may need evaluation for surgical resection.

Pericardium

Pericardial effusion and thickening

The normal pericardium appears as a thin, barely perceptible, circumferential structure between the epicardial and mediastinal fat measuring up to 2 mm in thickness without

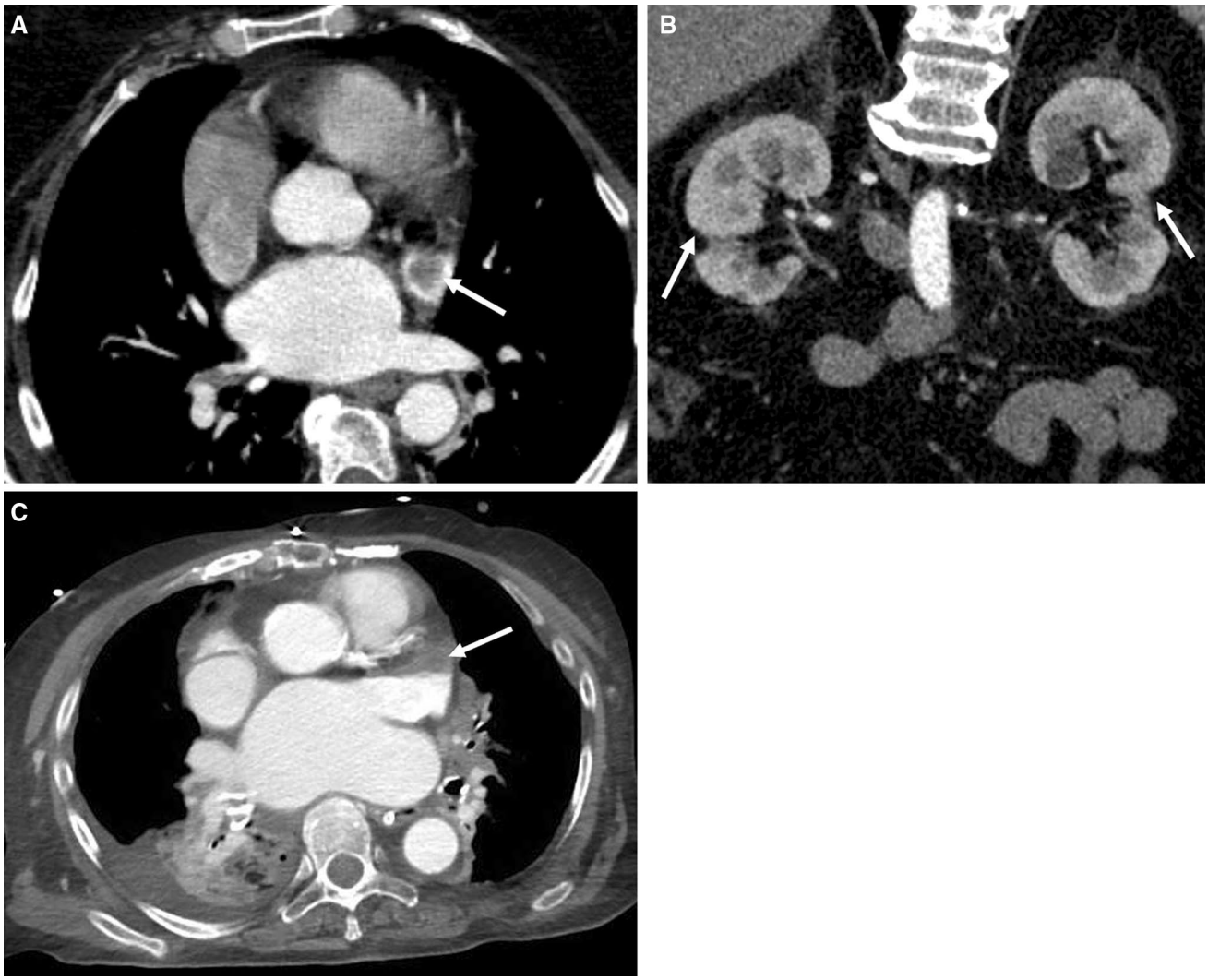

Fig. 9 Left atrial appendage thrombus and pitfall. **a** Contrast-enhanced axial CT performed for abdominal pain demonstrates a rounded filling defect within the left atrial appendage (arrow) consistent with thrombus. **b** Image through the abdomen demonstrates bilateral renal scarring (arrows), likely ischemic from prior emboli. **c** Contrast-enhanced axial CT performed for trauma assessment demonstrates a markedly enlarged left atrium with geometric area of low attenuation within the left atrial appendage (arrow). As no thrombus was seen on subsequent echocardiogram, this finding was likely secondary to underfilling and circulatory stasis

appreciable enhancement. In certain locations, pericardial reflections form pericardial recesses and may become focally distended with physiologic pericardial fluid, possibly mimicking a lymph node or other mass (Fig. 13). These can be distinguished from true mass lesions by their classic locations and isoattenuation to low-density pericardial fluid elsewhere [35].

Pericardial thickness measuring more than 3–4 mm is considered abnormal and may be due to fluid in the pericardium or thickening of the pericardial lining itself. Pericardial fluid is among the most common incidental cardiac finding on routine studies and is the most common pericardial finding [1]. Pericardial effusions may result from a variety of conditions including heart or renal failure, connective tissue disease, trauma, radiation, myocardial infarction, or malignancy. Effusions measuring simple fluid density (< 20 HU) are more likely to be transudative. Alternatively, complex or exudative effusions demonstrate higher density and may be accompanied by pericardial hyperenhancement (Fig. 14). Although there is overlap in density with exudative

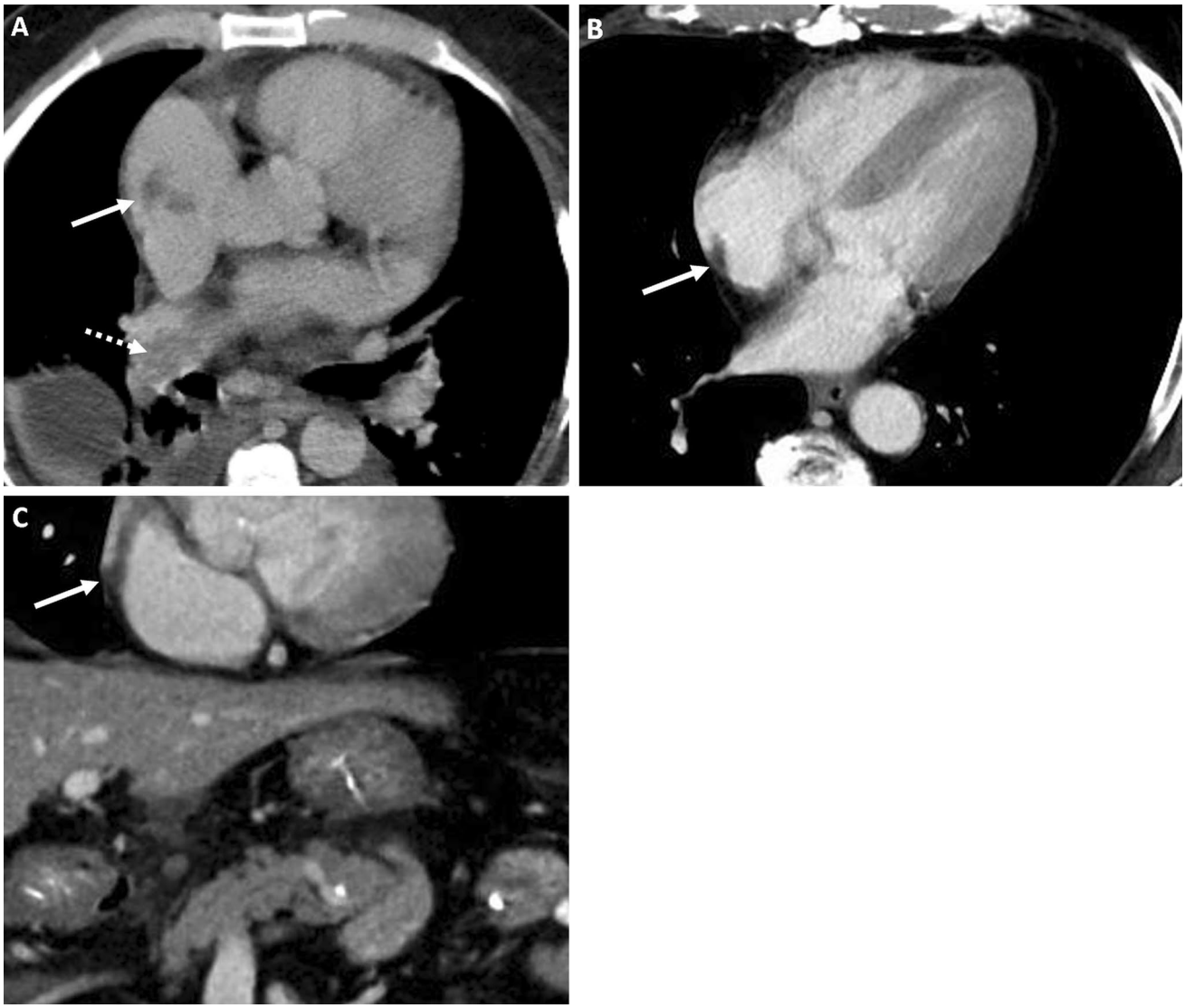

Fig. 10 Right atrial thrombus and pitfall. **a** Contrast-enhanced axial CT performed for evaluation of leukocytosis demonstrates an irregular, mass-like filling defect within the right atrium (solid arrow) with associated right lower lobe pulmonary embolism (dashed arrow). Contrast-enhanced axial (**b**) and coronal (**c**) CT performed for gastrostomy tube assessment demonstrate a linear, vertically oriented filling defect at the lateral right atrium (arrows) consistent with a prominent crista terminalis

effusions, hemorrhagic effusions are hyperdense and most often secondary to trauma or malignancy. Pericardial thickening may also be secondary to thickening of the pericardial tissue itself and may be calcified.

Pericardial fluid volume measuring up to 50 mL is considered normal, although there is no reliable way to determine this volume on cross-sectional studies, and fluid volume is often assessed subjectively [12]. Trace to small-sized simple effusions likely do not need further work-up, particularly if they can be explained by the etiologies described previously. However, moderate to large effusions, especially if accumulating rapidly, may be hemodynamically significant with the most feared complication being cardiac tamponade. Effects of cardiac tamponade include dilation of the vena cavae and hepatic veins with reflux of contrast and bowing of the interventricular septum secondary to ventricular interdependence. If any concerning findings are present, an echocardiogram should be obtained immediately to more reliably determine hemodynamic effects of the effusion as tamponade may be fatal. Pericardial thickening, especially if calcified, may be associated with constrictive physiology, which is better assessed by echocardiography or cardiac MRI.

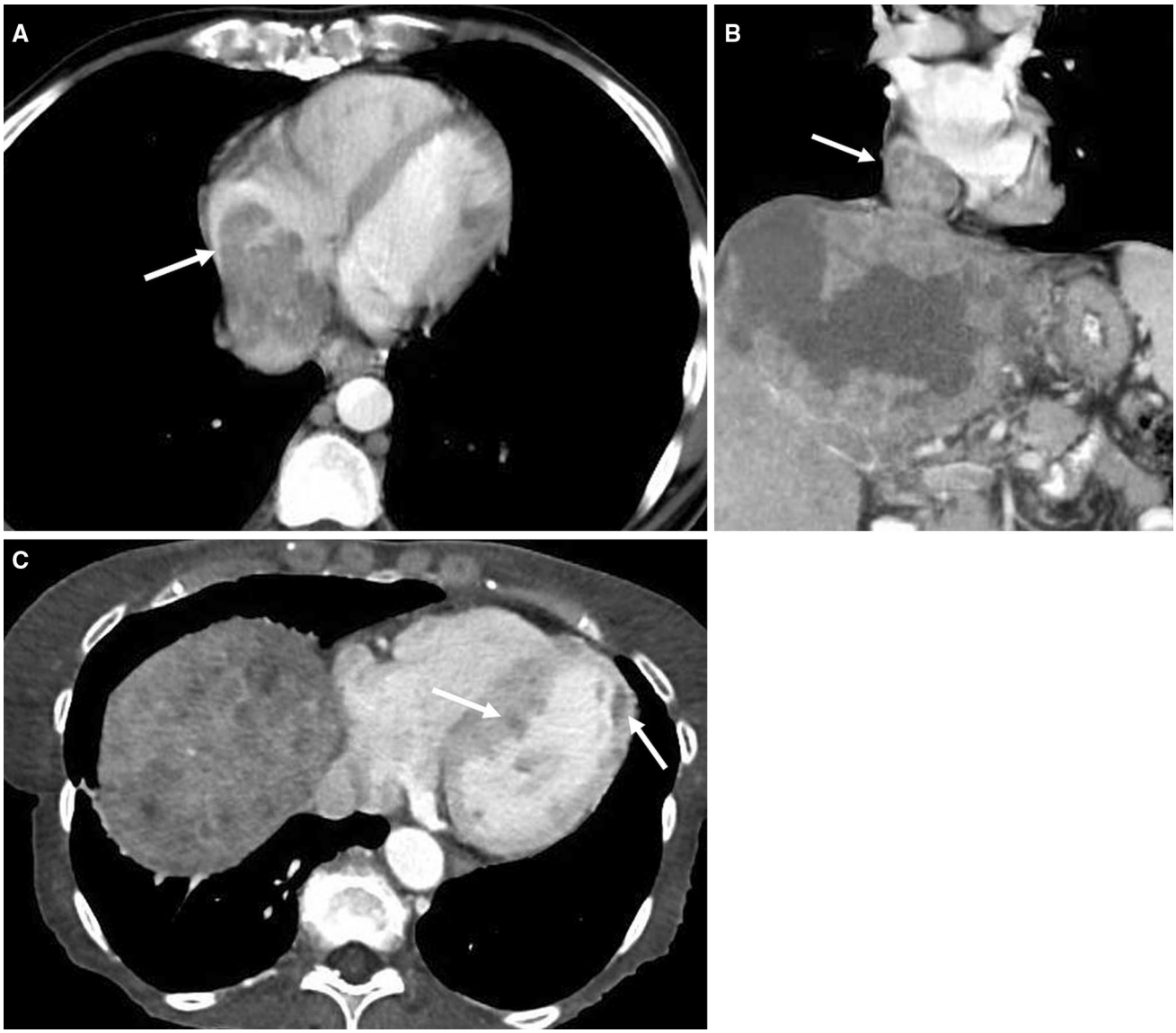

Fig. 11 Cardiac metastases. Contrast-enhanced axial (**a**) and coronal (**b**) CT in a patient with known hepatocellular carcinoma demonstrates a large, heterogenous liver mass directly invading into the inferior vena cava and extending into the right atrium (arrows). **c** Contrast-enhanced axial CT in a patient with known neuroendocrine tumor demonstrates multiple hypoattenuating metastases within the left ventricular myocardium (arrows). Metastases are also seen within the liver

Pericardial masses

True pericardial masses should be differentiated from a loculated pericardial or pleural effusion or focal pericardial thickening. The most common benign pericardial lesion is a pericardial cyst which is recognized by its classic location, most commonly at right cardiophrenic angle, and circumscribed, cystic appearance [36] (Fig. 15). Solid lesions, especially if multiple, should raise concern for metastases especially in the setting of a known primary malignancy. Primary pericardial malignancies are rare. Cardiac MRI is useful in providing more definitive characterization of pericardial masses.

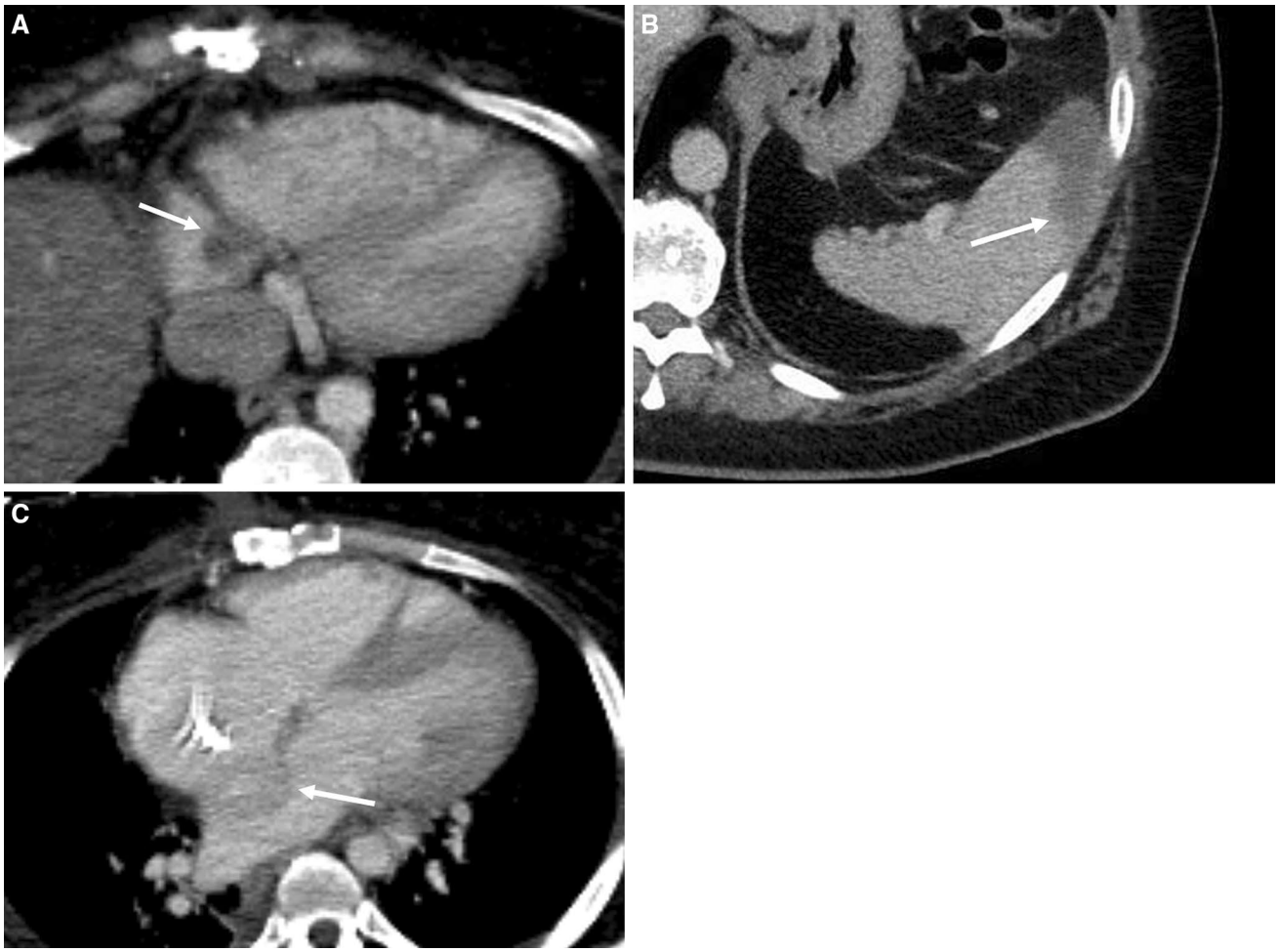

Fig. 12 Right heart thrombus with paradoxical embolism. **a** Axial contrast enhanced CT demonstrates a rounded filling defect with in the inferior right atrium (arrow) consistent with thrombus. **b** Axial image through the upper abdomen demonstrates infarction of the posterolateral spleen (arrow). **c** Axial CT image more superiorly through the heart demonstrates leftward bulging of the interatrial septum (arrow). Subsequent echocardiogram demonstrated an atrial septal aneurysm with associated interatrial shunt allowing a route for paradoxical embolism

Pulmonary arteries

Pulmonary artery enlargement

Pulmonary arterial enlargement is a sign of pulmonary arterial hypertension. Dilation of the main pulmonary artery has shown to correlate with elevated pulmonary artery pressures [37]. Pulmonary size can be assessed via absolute diameter measurement or as a ratio with respect to the ascending aorta. A main pulmonary artery diameter above 29.5 mm demonstrates a sensitivity of 71% and specificity of 79% for the diagnosis of pulmonary hypertension, while a main pulmonary artery-ascending aorta diameter ratio greater than 1.0 is 71% sensitive and 75% specific for pulmonary hypertension [37]. It is recommended that pulmonary artery dilation meeting either of these criteria should be reported as dilated [12]. While there are many underlying etiologies for pulmonary hypertension, common causes include left heart disease (either ventricular or valvular), pulmonary disease such as COPD or other interstitial lung disease, and chronic thromboembolic disease [38]. Signs of pulmonary hypertension on cross sectional imaging should prompt search for associated findings within the heart or lungs such as findings of left heart dysfunction or emphysema or fibrotic lung disease to explain a causative etiology. Given concerns for pulmonary hypertension, echocardiography is a useful initial screening tool of pulmonary artery pressures as well as assessment of left ventricular and valvular function [39].

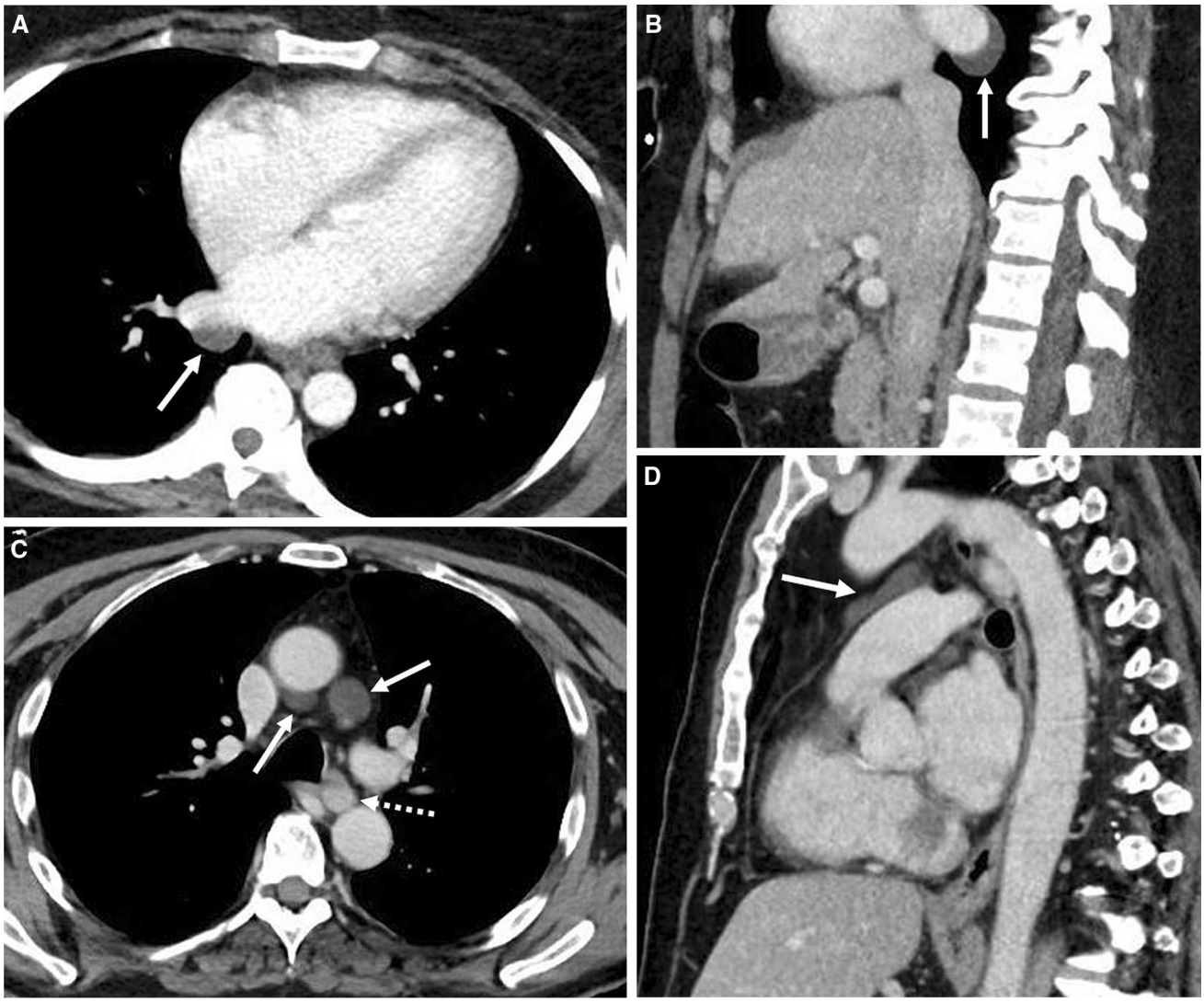

Fig. 13 Pericardial recesses. Contrast-enhanced axial (**a**) and sagittal (**b**) CT performed for assessment of right lower quadrant pain demonstrates a semi-circumferential fluid-attenuating collection (arrows) about the right inferior pulmonary vein. Based on location and appearance, this is consistent with a right pulmonary vein pericardial recess. Contrast-enhanced axial (**c**) and sagittal (**d**) CT performed for neuroendocrine tumor staging demonstrates a pair of fluid-attenuating collections (solid arrows) posterior to the ascending aorta consistent with a superior aortic recess. This is contrasted with a soft tissue density, pathologic mediastinal lymph node (dashed arrow)

However, right heart catheterization is needed to confirm the diagnosis and assess the severity of pulmonary arterial hypertension.

Pulmonary embolism

Incidental pulmonary embolism (PE) is not an uncommon incidental finding on routine cross-sectional studies. In patients undergoing routine chest CT without preexisting suspicion for PE, incidental PE was seen in 3.4% of patients ranging from subsegmental to involving the main pulmonary artery [40]. Risk factors for PE include recent surgery or trauma, immobilization, active malignancy, hormonal therapy, and inherited hypercoagulable conditions. At times, particularly in inpatient settings, patients may have several of these risk factors. Oncologic patients have been shown to have overall slightly higher rates of incidentally detected PE (4.4%) with higher rates seen in inpatient settings, the presence of metastatic cancer, and those undergoing chemotherapy [41]. The relatively higher incidence of PE in the lower lobes makes it especially important to carefully evaluate the basilar pulmonary arties on abdominal studies [42].

With adequate contrast opacification of the pulmonary arteries, typically achieved during arterial phase images and less likely during the portal venous phase, the diagnosis of acute PE is generally straightforward appearing

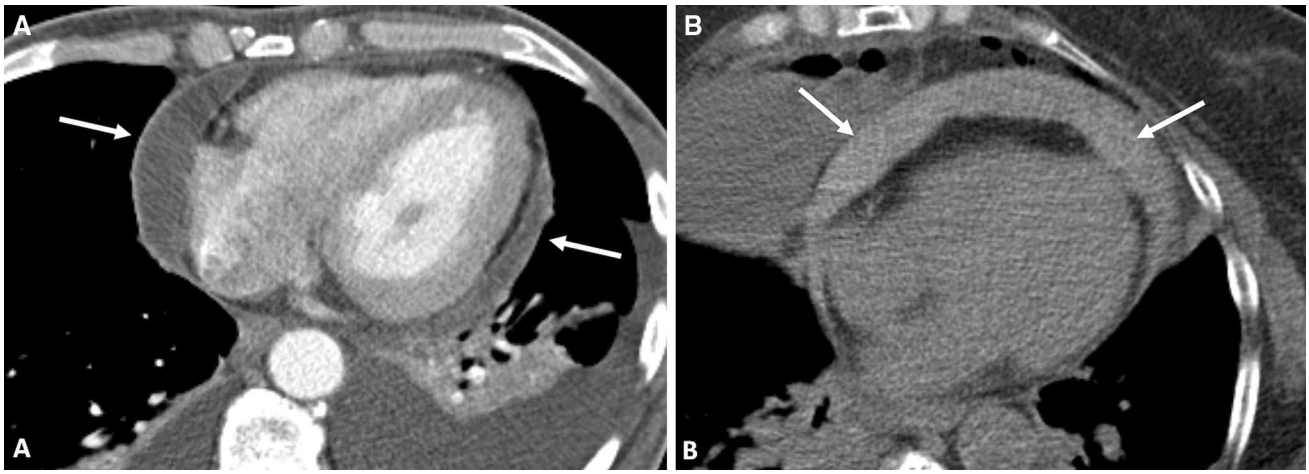

Fig. 14 Complex pericardial effusion. **a** Contrast-enhanced axial CT performed for assessment of pain after endoscopy demonstrates two areas of loculated pericardial effusion with associated pericardial thickening and hyperenhancement (arrows). **b** Noncontrast axial CT performed for pain after hernia repair demonstrates a hyperattenuating pericardial effusion (arrows) consistent with hemopericardium

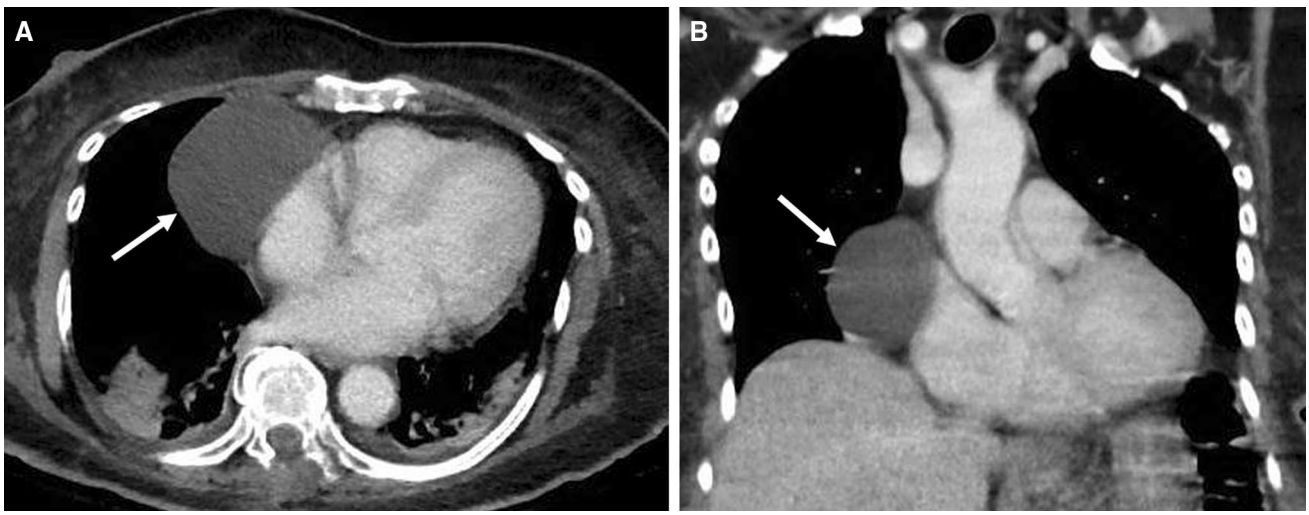

Fig. 15 Pericardial cyst. Contrast-enhanced axial (**a**) and coronal (**b**) CT performed for assessment of fever demonstrate a circumscribed, fluid-attenuating collection (arrows) at the right cardiophrenic angle characteristic of a pericardial cyst

as a centralized filling defect within the pulmonary arterial branch. However, technical factors that may reduce PE detection or artificially simulate PE include inadequate contrast opacification, motion artifact, slow flow artifact, and adjacent streak artifact (Fig. 16). The diagnosis of PE should prompt search for potential complications including right heart strain (right ventricle: left ventricle width > 1, leftward bowing of the interventricular septum), pulmonary hypertension (enlarged pulmonary artery), and pulmonary infarct (peripheral wedge-shaped pulmonary opacity distal to the PE).

The presence of PE and any associated complications should be promptly reported [12]. If the diagnosis of PE is confidently made on routine cross-sectional imaging, no further confirmatory imaging is necessary [43]. However, further imaging either with PE protocol CT or ventilation perfusion scan may be needed in equivocal cases. The treatment of choice for PE is anticoagulation, which should be considered for all incidental PEs, particularly with underlying risk factors, including malignancy [44]. A potential exception is in the treatment of subsegmental

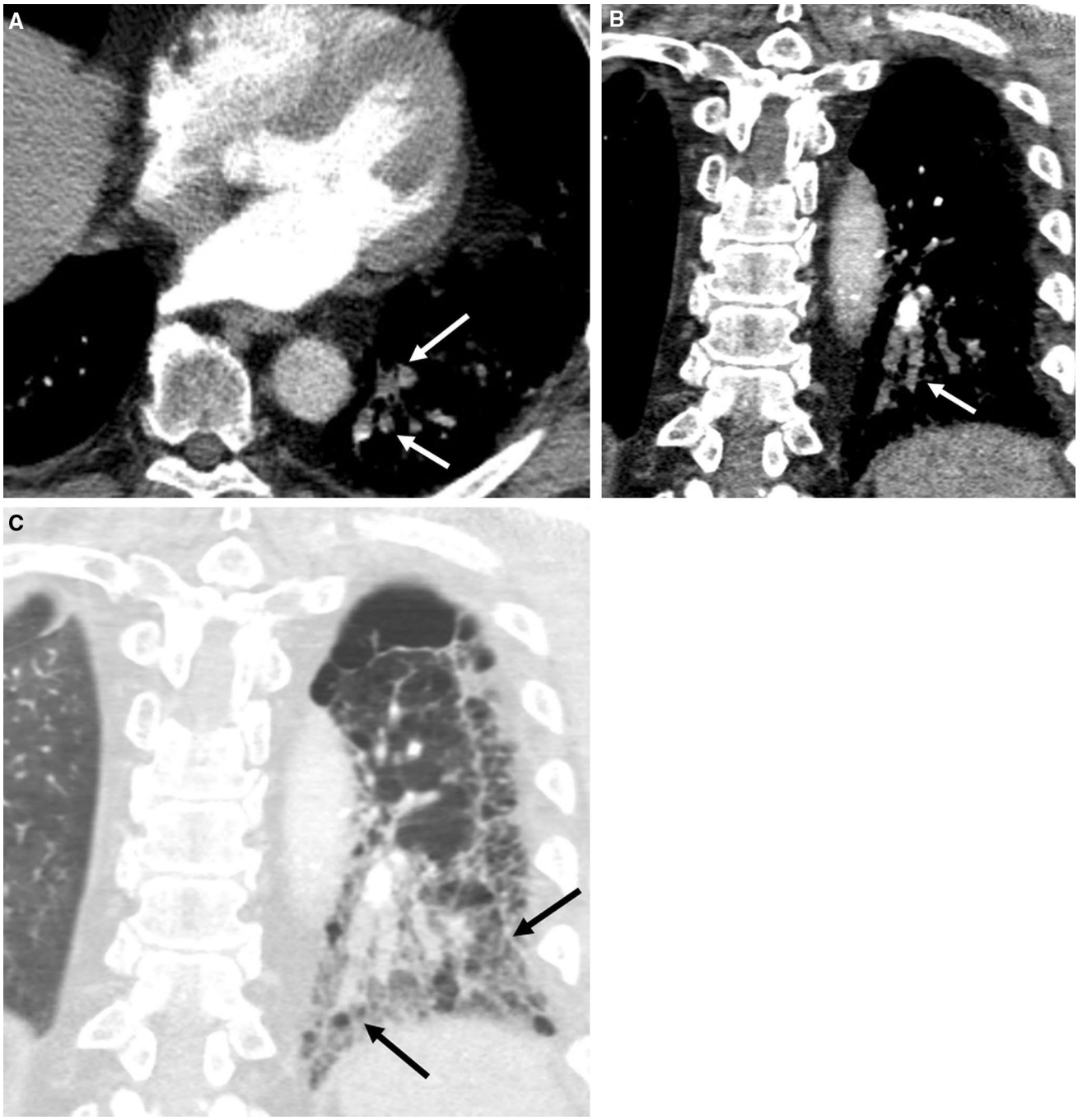

Fig. 16 Slow flow pulmonary embolism mimic. Contrast enhanced axial (**a**) and coronal (**b**) images demonstrate apparent filling defects within the left lobe segmental branches (arrows). Corresponding coronal CT (**c**) demonstrates severe pulmonary fibrosis within the left lower lung (arrows). These filling defects are artifactual secondary to lack of contrast opacification due to slow flow in the setting of increased pulmonary resistance from severe pulmonary fibrosis

PE, which is more controversial with conflicting evidence regarding the benefits of anticoagulation compared to potential bleeding risks [45].

Systemic veins

Images acquired in the portal venous phase can often provide adequate thoracic systemic vein opacification and, therefore,

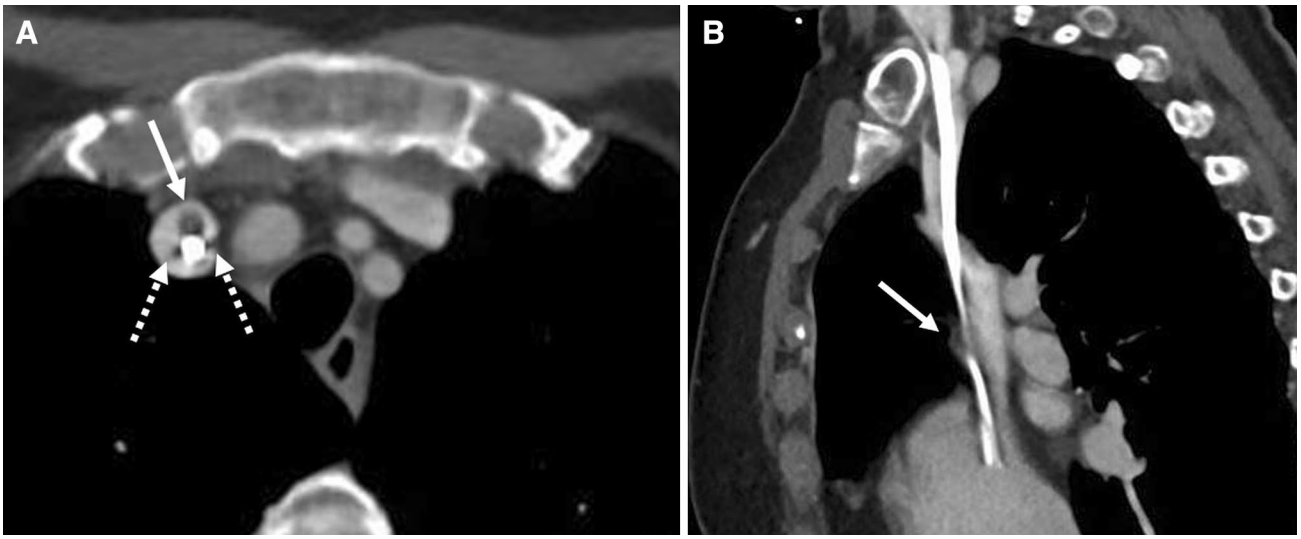

Fig. 17 Catheter-associated thrombus. Contrast enhanced axial (**a**) and sagittal (**b**) images performed for cholangiocarcinoma restaging demonstrate a small, rounded catheter associated thrombus (solid arrows) at the anterior aspect of the catheter. Smaller, more linear hypoattenuating areas at the left and right aspects of catheter (dashed arrows) are secondary to streak artifact

assessment of patency. Acute thrombus may be encountered in any of the central thoracic veins and appears as well-defined filling defect within the vein. Indwelling venous lines such as central venous catheters and cardiac pacing leads are commonly encountered and place patients at higher risk for thrombus [46]. Catheter-associated thrombus may be visualized anywhere about the catheter course while it is still in place or after it has been removed (Fig. 17). Bland thrombi are usually easily distinguished from tumor thrombi, which demonstrate enhancement and are usually secondary to direct invasion from the primary tumor. Several pitfalls including streak artifact from dense structures and inflow of unopacified venous blood can simulate thrombus (Figs. 17, 18). Chronic occlusion can be inferred due to the presence of narrowed, diminutive (rather than expanded), and possibly calcified vein with surrounding intrathoracic or chest wall collaterals. Signs of chronic central venous occlusion may also manifest within the abdomen such as the presence of abdominal wall collaterals or hyperenhancement of the medial segment left hepatic lobe via collaterals ("hot quadrate sign") (Fig. 19).

Although no further work-up is necessary if the diagnosis can be confidently made at routine imaging, confirmatory imaging is warranted in equivocal cases. For more peripheral vessels such as the internal jugular vein, ultrasound should be obtained. However, delayed CT or MRA focused on the venous phase maybe needed for more central vessels such as the SVC or brachiocephalic veins to minimize mixing artifact. The treatment of choice for acute thrombus is anticoagulation. Catheter removal should also be considered for catheter-associated thrombus depending on ongoing need for the catheter. The presence of chronic thrombus is important to note as this may impact future central venous access options for future hemodialysis, chemotherapy, or parenteral nutrition.

Conclusion

Cardiovascular findings are frequently encountered on routine abdominal or whole-body cross-sectional imaging. Sometimes, these findings may be unexpected and may offer the first window into an underlying cardiovascular

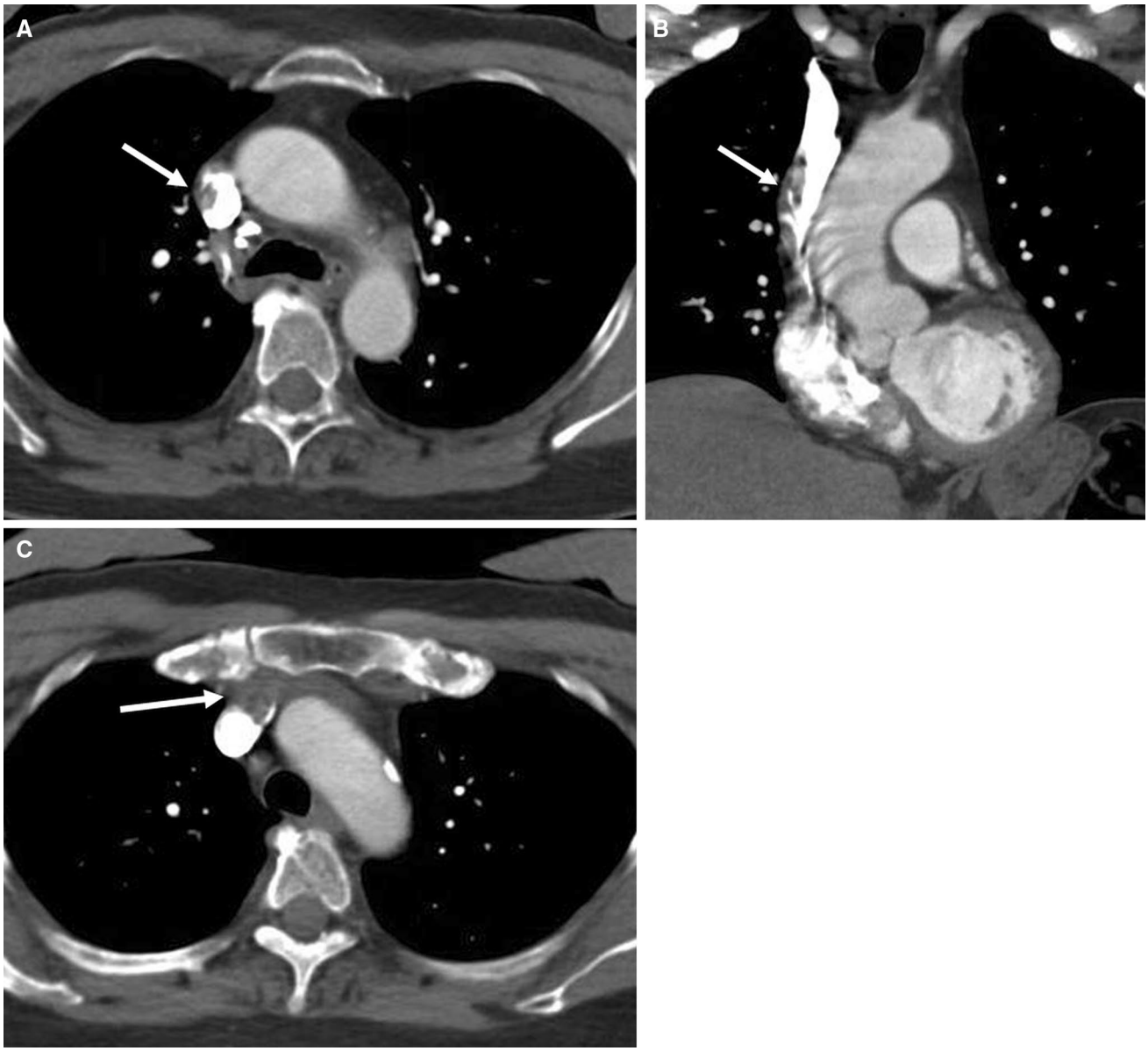

Fig. 18 Venous inflow mimic SVC thrombus. Contrast enhanced axial (**a**) and coronal (**b**) images demonstrates an apparent eccentric filling defect within the lateral superior vena cava (arrows) mimicking thrombus. Axial CT image more superiorly (**c**) demonstrates inflow of unopacified blood from the left brachiocephalic vein (arrow) opposite the side of injection causing mixing artifact within the proximal SVC

pathology for the patient. Some findings are emergent and require immediate work-up or management, while others may a predictor for cardiovascular events and warrant close follow-up and preventative measures. Although focus of most imaging is on non-cardiovascular structures, abdominal imagers should be aware of the imaging patterns, potential pitfalls, and basic work-up and management of these findings to provide comprehensive care for their patients.

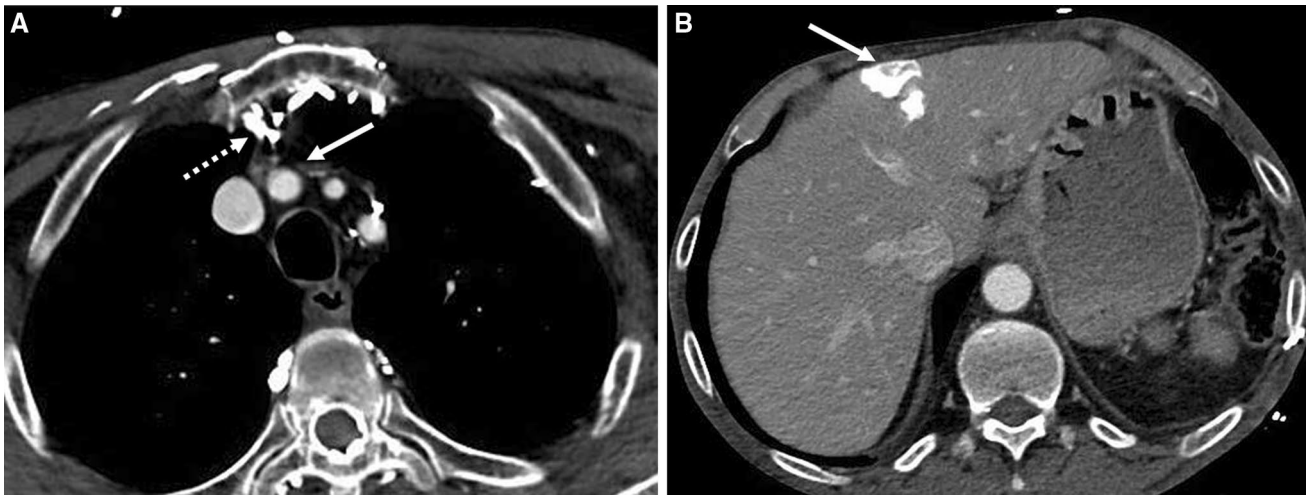

Fig. 19 Chronic brachiocephalic vein thrombus. Contrast enhanced axial image (**a**) performed for malignancy assessment demonstrates a non-opacified, markedly diminutive brachiocephalic vein (solid arrow) with numerous venous collaterals (dashed arrow). Axial image through the liver (**b**) demonstrates hyperenhancement of the segment 4 of the liver (arrow) secondary to enhancement via venous collaterals

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