Computed Tomography Angiographic Assessment of Acute Chest Pain

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Abstract: Acute chest pain is a leading cause of Emergency Department visits. Computed tomography angiography plays a vital diagnostic role in such cases, but there are several common challenges associated with the imaging of acute chest pain, which, if unrecognized, can lead to an inconclusive or incorrect diagnosis. These imaging challenges fall broadly into 3 categories: (1) image acquisition, (2) image interpretation (including physiological and pathologic mimics), and (3) result communication. The aims of this review are to describe and illustrate the most common challenges in the imaging of acute chest pain and to provide solutions that will facilitate accurate diagnosis of the causes of acute chest pain in the emergency setting.

Key Words: acute chest pain, challenges, pulmonary angiography, aortography, computed tomography

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LEARNING OBJECTIVES

After completing this SA-CME activity, physicians should be better able to:

1. Recognize the most common challenges of computed tomography (CT) angiography performed for acute chest pain and describe steps that can be taken to optimize image quality and interpretation.
2. Identify the most common physiological mimics in acute chest pain imaging and describe strategies to identify them.
3. Accurately identify and differentiate acute causes of chest pain from their pathologic mimics.
4. Describe the optimal methods of communication related to acute thoracic imaging.

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Acute chest pain leads to 6 million Emergency Department visits per year in the United States.[1] Evaluation of acute chest pain often leads to a prolonged inpatient assessment, with assessment duration often exceeding 12 hours. The estimated cost of a negative inpatient chest pain assessment amounts to $8 billion per year in the United States.[2,3]

The main challenge to diagnosis is the broad range of pathologies that can cause chest pain. Vascular causes include pulmonary embolism (PE), traumatic and spontaneous aortic syndromes including aortic transection, dissection, intramural hematoma, and penetrating atherosclerotic ulcer, aortitis, and coronary artery disease. The latter will not be discussed in detail because of the complexity and breadth of this topic alone. CT angiography is playing an increasing role in the assessment of acute chest pain because of its ability to rapidly identify vascular causes of chest pain[4–8] with the aim of improving patient outcomes.

There are, however, several common challenges associated with the imaging of acute chest pain, which, if unrecognized, could lead to an incorrect diagnosis. These potential challenges lie in image acquisition, image interpretation, and result communication.

In this review, we describe the most common radiologic challenges encountered in acute chest pain assessment and suggest solutions to facilitate accurate diagnosis of vascular causes of chest pain in the emergency setting.

OPTIMIZING IMAGE ACQUISITION

Suboptimal image acquisition can be avoided by the use of carefully selected imaging parameters, particularly avoiding motion and other artifacts. These parameters are of paramount importance to the acquisition of diagnostic quality images.

CT Pulmonary Angiography

CT is a sensitive tool for the evaluation of pulmonary arteries and for detection of PE.[9] However, suboptimal studies account for up to 10.8% of all CT pulmonary angiograms performed.[10] Suboptimal image quality can arise when motion artifact obscures the pulmonary arteries or nonopacification of the pulmonary arteries mimics or obscures a PE.[11]

Motion Artifact

Respiratory Motion

Challenge. Respiratory motion can result in artifactual duplication of normal physiological structures or overlapping of adjacent structures—for example, a pulmonary artery and an airway. Volume averaging of these 2 adjacent structures may then produce the appearance of a filling defect or termination of the vessel mimicking a PE

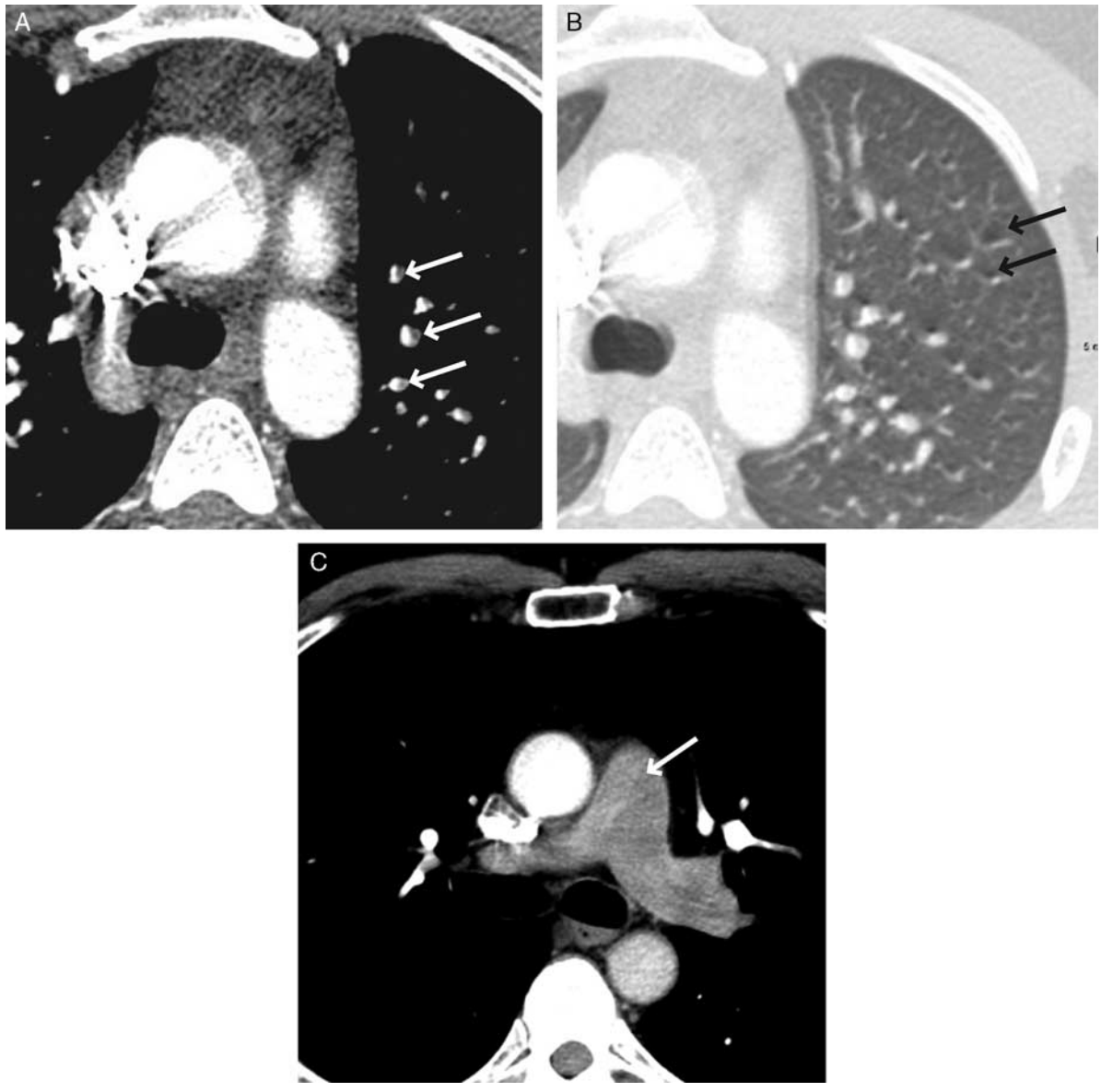

FIGURE 1. Challenges of image acquisition, pulmonary arteries. A and B, Motion artifact creating apparent filling defects in all arteries on 1 slice (white arrows), which can mimic PE. Comparison with lung windows demonstrates blurring of all bronchial structures on the same slice (black arrows), confirming motion artifact. C, Suboptimal opacification in the main pulmonary artery (arrow) due to transient interruption of contrast from deep inspiration during contrast injection, which can lead to missed findings.

(Fig. 1A). This may result in overdiagnosis of segmental and subsegmental PE.[12]

Solution. Steps to help prevent this challenge include (1) coaching the patient before CT on the importance of a sustained breath-hold and (2) using a scan direction from the lung bases toward the apices. High-pitch dual-source CT pulmonary angiography can lead to good-quality images, where motion artifact is a particular challenge.[13] These techniques enhance the visualization of the lower-lobe pulmonary arteries where motion artifact and pulmonary emboli are most likely to occur. After these steps have been taken, any artifacts arising from respiratory motion can be recognized by cross-referencing soft tissue and lung window images. On soft tissue windows, artifactual filling defects will often be present throughout all of the pulmonary arteries on the same image, and if these apparent filling defects are cross-referenced with lung window images, blurring or duplication of the bronchovascular bundle is often evident on the same image (Figs. 1A, B).

Cardiac Motion

Challenge. Pulsation artifacts arising from cardiac motion can also result in volume averaging and apparent filling defects in the pulmonary arteries adjacent to the heart, mimicking PE.

Solution. Identification of duplicated physiological structures in the lung adjacent to the heart may raise

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a suspicion of pulsation artifact. If pulsation artifact is considered a major limiting factor to achieving a diagnosis, prospective electrocardiogram (ECG)-gated CT pulmonary angiography can be performed; images are typically acquired at 70% of the R-R interval.[14]

Patient Motion

Challenge. Motion artifact due to patient motion on the scanning table is a particular challenge in the acutely ill or confused patient.

Solution. Patient coaching may reduce the impact of patient motion during pulmonary CT angiography. In the case of an intubated or confused patient, however, this strategy may not be sufficient. High-pitch imaging can minimize the impact of patient motion. If, however, CT pulmonary angiography is nondiagnostic because of unpreventable patient motion, alternative imaging modalities such as lower-limb Doppler ultrasound to exclude an associated deep venous thrombosis can be considered.

Suboptimal Vascular Opacification

Adequate main pulmonary arterial opacification is defined as good if it is ≥ 210 HU and acceptable if it ranges between 170 and 209 HU. Focal nonopacification can mimic PE, whereas global nonopacification can obscure a PE.

Deep Inspiration

Challenge. Deep inspiration immediately before image acquisition is a recognized cause of artifactual focal nonopacification of the pulmonary arteries.[15,16] Deep inspiration leads to decreased intrathoracic pressure, which draws unopacified blood into the right atrium from the inferior vena cava (IVC) and causes transient interruption of the contrast bolus as it flows into the right heart from the superior vena cava (SVC). As this unopacified blood enters the right atrium, right ventricle, and pulmonary arteries, it can create a low-attenuation filling defect in the pulmonary arteries that mimics PE (Fig. 1C). If a large volume of unopacified blood is drawn into the pulmonary arteries, global hypoenhancement due to dilution of the contrast bolus can also occur.

Solution. The primary preventative measure to avoid transient interruption of the contrast bolus is patient coaching before imaging to ensure shallow held inspiration. This has been shown to eliminate this error in image acquisition.[17] Once imaging has been performed, characteristic indicators of transient interruption of contrast due to deep inspiration include decreased opacification of multiple vessels at the same level bilaterally and the presence of nonopacified blood in the right heart arising from the IVC, which then moves to the left heart on subsequent imaging, if delayed sequences are acquired. The following equation calculates the fraction of blood flow contributed by the IVC to the right side of the heart (KIVC):

$$\mathrm{KIVC} = (\mathrm{CSVC} - \mathrm{CRA\ or\ RV}) / (\mathrm{CSVC} - \mathrm{CIVC}).$$

In this equation, the relative IVC contributions to the right atrium (RA) and right ventricle (RV) are calculated by equating attenuation in HU (C) in these chambers to a weighted average of the attenuations of the SVC and IVC. It is assumed in this equation that the SVC and IVC are the sole contributors of blood flow to the right side of the heart.[18] When IVC contribution to pulmonary arterial blood flow is excessive, KIVC ranges from 87% to 91%, and when optimal it ranges from 42% to 62%.[19] If a questionable filling defect is detected, attention should also be paid to secondary signs of PE, such as vessel dilation, pulmonary infarction, right heart strain, or pleural effusion. If these secondary signs of PE are absent and KIVC is elevated, transient interruption of the contrast bolus may have occurred.

Shunt Physiology

Challenge. A right-to-left intracardiac shunt or an intrapulmonary shunt can allow a portion of injected contrast to bypass the pulmonary arterial tree, thereby leading to decreased opacification of the pulmonary arteries.[20] A rarer left-to-right intracardiac shunting can also contribute to suboptimal pulmonary arterial opacification by allowing nonopacified blood from the left heart to mix with and dilute the bolus of contrast as it enters the right heart.

Solution. A right-to-left shunt can be recognized by the presence of pulmonary arterial hypoenhancement coupled with early contrast entry into the left heart and aorta. Diagnosis of a right-to-left shunt can also be facilitated by visualization of the jet of contrast from the right to the left heart. Left-to-right shunt is suggested by decreased pulmonary arterial opacification and a prolonged time to peak enhancement during bolus tracking. A left-to-right connection may also be visible, if large.[21] If shunt physiology is considered a limiting factor to CT pulmonary angiography, a test bolus can be injected to determine peak pulmonary arterial enhancement comprising a 20 mL bolus of contrast injected at 4.5 mL/s before the main injection. The volume of contrast medium used for image acquisition is then calculated by adding the time to peak to the predicted scan time and multiplying by the injection rate.

Global Pulmonary Arterial Hypoenhancement

SVC Obstruction

Challenge. Obstruction of the SVC due to stenosis, thrombosis, or external compression can lead to global suboptimal or nonopacification of the pulmonary arteries by delaying the transit of the contrast bolus to the right heart.

Solution. If SVC obstruction is present and venous collaterals have not developed, contrast injection through a lower-limb intravenous cannula may better opacify the pulmonary arteries.

Bolus Timing Error

Challenge. Inappropriate bolus timing is a common cause of global nonopacification of the pulmonary arteries.[10] This is particularly true in the clinical setting of chest pain, for which imaging of both the aorta and pulmonary arteries is often indicated. To evaluate the pulmonary arteries and aorta simultaneously, referred to as a "double rule-out" CT, imaging must occur during a narrow window of time in which the aorta has reached sufficient opacification and the contrast bolus has not yet left the pulmonary arteries.

Solution. Double rule-out CT can be performed using bolus tracking at the left atrium with a threshold trigger of 100 absolute Hounsfield units (HU) during administration of 350 mg/mL iodinated contrast material injected at a rate of 4 mL/s, followed by a 30 mL saline chaser, with image acquisition during shallow held inspiration in the craniocaudal direction at 100 kVp, for the majority of patients. In our

institution, we calculate the total volume of contrast by using the following formula: (estimated scan time + 3 s × injection rate + 6 s fudge factor).[22] Imaging parameters have also been described for ECG-gated "triple rule-out" studies, which seek to achieve adequate opacification and imaging of the coronary arteries in addition to the aorta and pulmonary arteries.[23–26]

Pregnancy

Challenge. Pulmonary arterial opacification can be affected by the altered physiology of pregnancy, including a hyperdynamic circulation, increased cardiac output, increased body mass index, and increased plasma volume, the latter leading to hemodilution. Transient interruption of the contrast bolus by unopacified blood from the IVC is also more common in this patient group because of increased intra-abdominal pressure.

Solution. To adjust for earlier and lower peak enhancement associated with increased cardiac output, the delay between injection and acquisition can be shortened, and the contrast injection rate should be increased.[27] A suggested protocol comprises intravenous injection of 95 mL of high-density contrast (eg, 370 mg/mL Iodine) at a flow rate of 6 mL/s, followed by a 50 mL intravenous saline flush. Patient coaching to avoid deep inspiration is important.

CT Aortography

CT aortography allows for quick and sensitive evaluation of the aorta in a wide range of clinical scenarios. When the image quality is suboptimal, this is most frequently due to pulsation, streak, or beam-hardening artifacts.

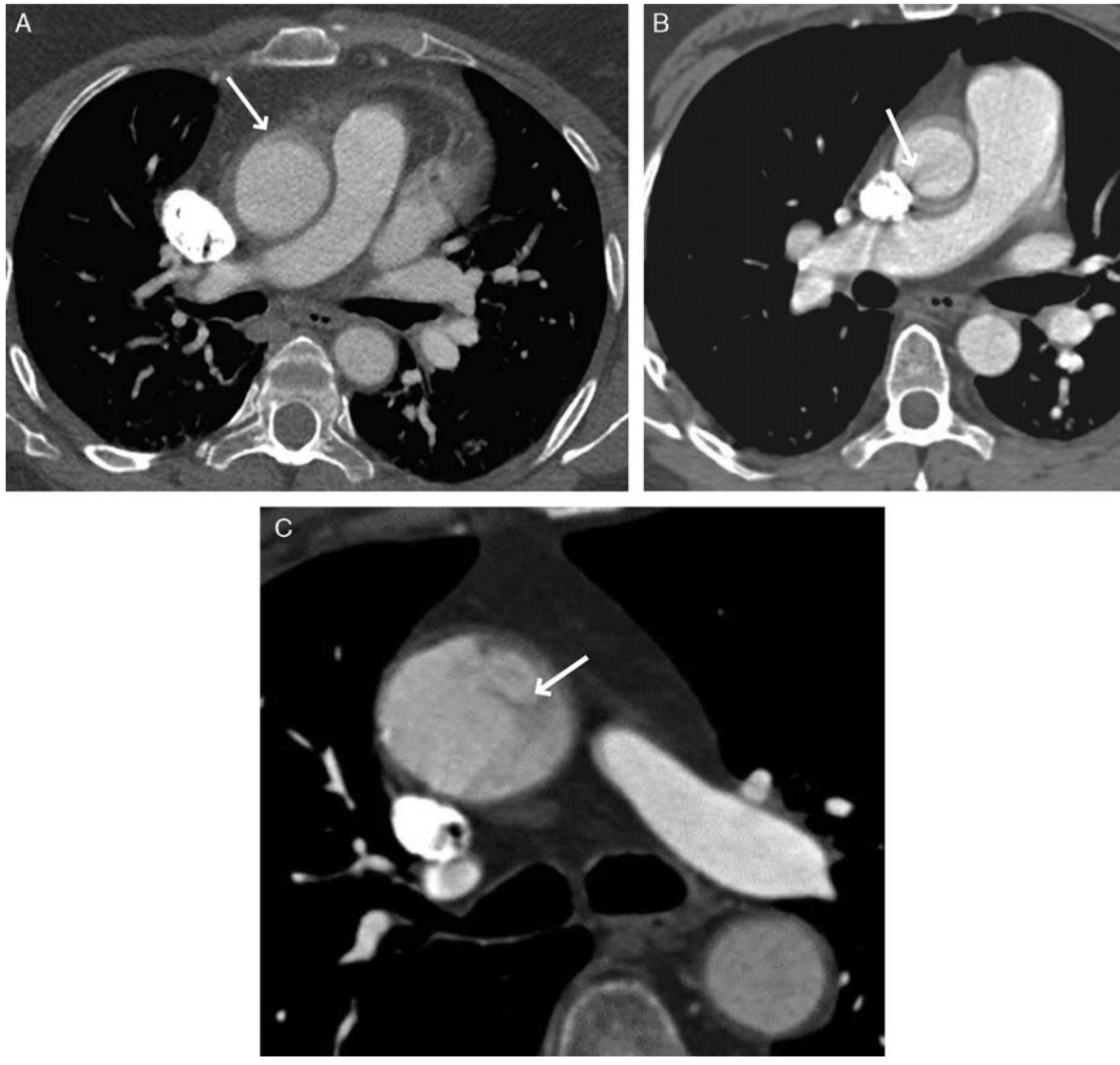

FIGURE 2. Challenges of image acquisition, aorta. A, Pulsation artifact creating a pseudo-flap in the aorta (arrow), which can mimic dissection. B, Streak artifact (arrow) from concentrated contrast in the SVC, which can mimic dissection or other pathology. C, Flow-related phenomenon creating ill-defined, smoke-like filling defects in the aorta (arrow), which can mimic cobwebbing seen in aortic dissection (courtesy of Dr Bhalla, St Louis).

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Motion Artifact

Pulsation Artifact

Challenge. Cardiac pulsation creates motion artifact on nongated CT imaging. This is most pronounced in the ascending aorta. The result is a double curvilinear hypodensity, in the anteromedial aortic wall, which mimics an aortic dissection (Fig. 2A).

Solution. Pulsation artifact may not affect the aorta exclusively but may simultaneously affect the pulmonary arteries within the same axial image. If pulsation artifact in the aorta is suspected, the presence of pulsation artifact in the adjacent pulmonary arteries can confirm this suspicion. In studies in which careful evaluation of the ascending aorta is indicated, pulsation artifact from cardiac motion can be eliminated or reduced by using ECG-gating or ultrafast pitch imaging.[28,29]

Streak and Beam Hardening Artifacts

Streak artifact due to dense contrast in the SVC and beam-hardening artifact due to x-ray beam attenuation by radiodense structures can also result in suboptimal imaging of the aorta, particularly when they arise from structures directly adjacent to the aorta.

SVC Contrast

Challenge. The presence of dense contrast in the SVC from the injected contrast bolus can lead to stellate radiodense artifact arising from the SVC. This streak artifact can obscure adjacent structures, including the aorta (Fig. 2B).

Solution. Streak artifact from SVC contrast can be minimized by using a saline chaser after contrast injection and a longer delay between injection and acquisition to allow more mixing and/or clearing of contrast in the SVC before imaging.

Patient Arm Positioning

Challenge. Beam hardening is the attenuation of the x-ray beam by a highly attenuating structure in the region of interest (eg, contrast, bone, or metal). This results in a dark line traversing and potentially obscuring the tissues adjacent to the attenuating structure. Arm position at either side of the body is a common cause of beam-hardening artifact.

Solution. This type of artifact can be avoided by positioning the arms above the head, or, if this is not possible, the patient may hug a pillow in front of the chest.[12] Although not all patients will be able to assume one of these positions, adopting this positioning where possible will greatly reduce beam-hardening artifacts.

Focal Nonopacification

Flow-related Phenomenon

Challenge. A smoke-like appearance within the aortic lumen due to sluggish mixing of opacified and unopacified blood can be observed immediately after contrast injection. This mimics "cobwebbing" seen in the false lumen of an aortic dissection (Fig. 2C).[30] This flow-related artifact is especially common in patients with a reduced ejection fraction.

Solution. Flow-related artifacts can be reduced by using bolus tracking to ensure the aorta is well opacified before image acquisition.[31] Bolus tracking in the descending thoracic aorta is typically performed with image acquisition when the absolute attenuation in the descending aorta reaches 100 HU with a scan delay time of 5 to 7 seconds. A saline chaser can be used to maximize the concentration of the contrast bolus within the aorta. If needed, delayed image acquisition can also be performed to troubleshoot any suspected flow-related artifacts.

ERRORS IN IMAGE INTERPRETATION: MISSES

After high-quality images have been acquired, errors in image interpretation become the next challenge. Image interpretation errors can be categorized into 3 groups: misses and physiological and pathologic mimics.

CT Pulmonary Angiography

Missed findings are rare in CT pulmonary angiography. The most common reasons for missed findings in the pulmonary arteries are suboptimal image quality, as discussed previously, an incomplete search, or chronic PE.

Missed Pulmonary Artery Findings

Chronic PE

Challenge. Chronic PE may be subtler than acute PE because of its linear and nonocclusive nature. The sensitivity for chronic PE in one small series was reported to be 47.8%, compared with 62% using single-photon emission CT perfusion scintigraphy.[32] Subtle pulmonary arterial webs may be particularly difficult to detect in the presence of high-attenuation intravascular contrast medium.

Solution. Secondary signs of chronic PE such as pulmonary arterial dilation, tortuosity, and calcification should be sought if there is a high clinical suspicion. Using a wider window setting (eg, width: 1100 HU; level: 100 HU) in the setting of high-attenuation contrast medium may better elucidate subtle arterial webs observed in chronic PE.[33]

Aortic CT Angiography

The aorta is an uncommon site for missed findings, primarily because of its large size relative to adjacent structures and its predominantly perpendicular orientation relative to the axial imaging plane. Missed findings such as a localized intimal tear may still occur.

Missed Aortic Findings

Incomplete Search/Satisfaction of Search

Challenge. An incomplete search/satisfaction of search is the primary contributor to missed aortic findings. Among the most commonly overlooked aortic findings are minor traumatic aortic injuries, which include small intimal flaps and shallow pseudoaneurysms.[34]

Solution. Careful examination of the aorta on axial and multiplanar reconstructed (MPR) images will greatly reduce the chanced of missed aortic findings. Minor traumatic aortic injuries may be detectable by the presence of a small periaortic mediastinal hematoma, which can facilitate detection.

CHALLENGES IN IMAGE INTERPRETATION: MIMICS

Mimics include physiological and pathologic radiologic findings that resemble a pathologic cause of chest pain. The most commonly mimicked acute chest pain etiologies include PE, aortic dissection, and intramural hematoma.

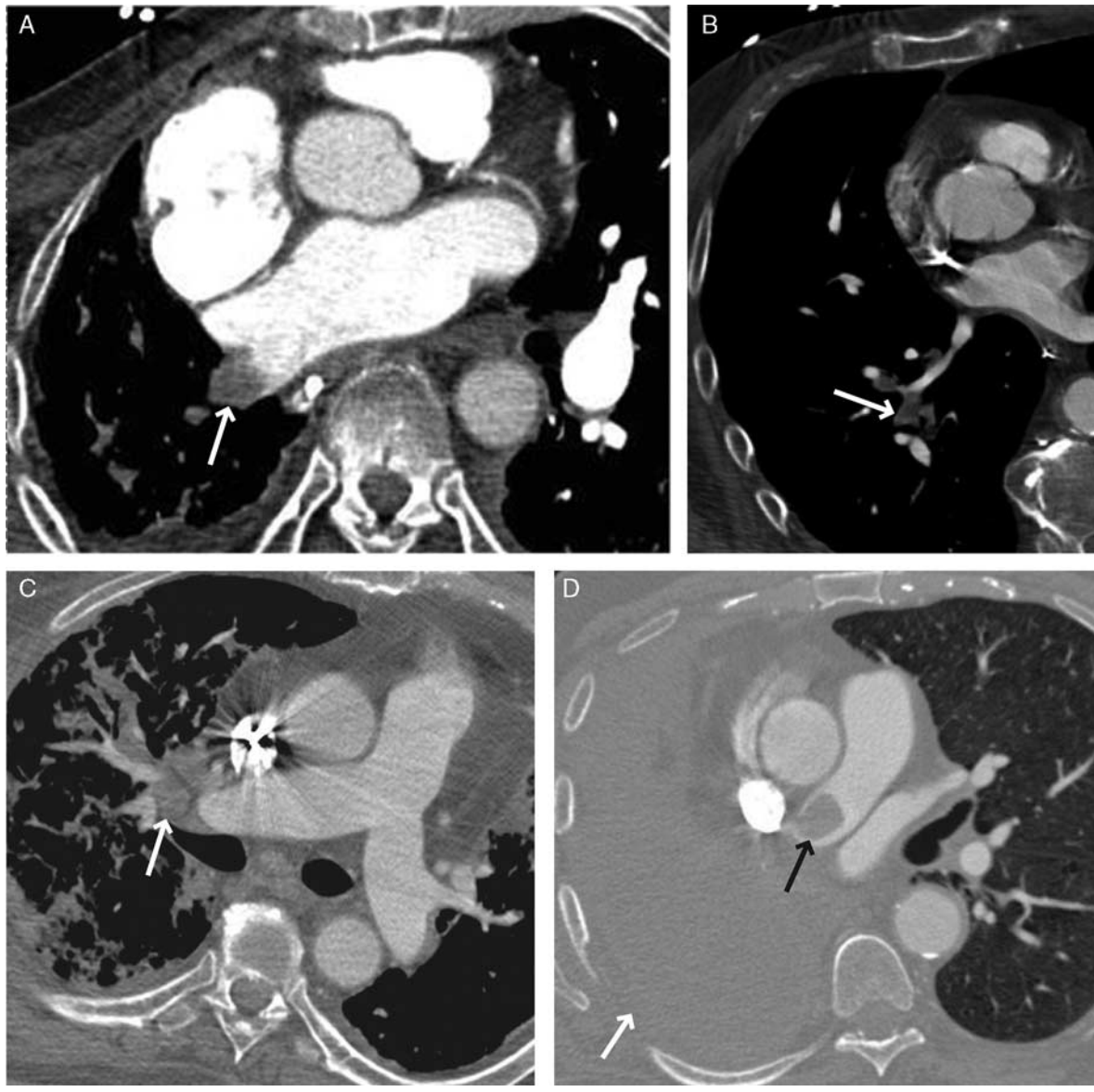

FIGURE 3. Challenges in image interpretation: mimics of PE. A, Pulmonary vein thrombus creating a filling defect within a vascular structure in the lungs (arrow), which can mimic PE. B, Mucoid bronchial impaction creating a soft tissue density within a tubular structure in the lungs (arrow), which can mimic PE. C, Lymph nodes appearing as soft tissue masses adjacent to pulmonary arteries (arrow), which can mimic PE, especially when located near bifurcations. D, Thrombus in the pulmonary artery stump (black arrow) after pneumonectomy (white arrow), which can mimic PE.

PE Mimics

Physiological Mimics

Pulmonary Artery Bifurcation Versus PE

Challenge. Artifactual hypoenhancement at pulmonary arterial branch points, either due to volume averaging with adjacent low-density structures or due to mixing of unopacified blood with contrast, may be misinterpreted as a small PE.[35]

Solution. Careful assessment of MPR images may help distinguish pulmonary arterial branch points from branch point emboli. Accompanying findings to support a diagnosis of PE, PEs at other locations, pulmonary infarct, right heart strain, and evidence of pulmonary hypertension may help distinguish the 2 entities.

Pulsation Artifact Versus Pulmonary Artery Dissection

Challenge. Pulsation artifact in the pulmonary artery can create an intraluminal linear hypodensity adjacent to the vessel wall that can be misinterpreted as a pulmonary artery dissection (termed a "pseudoflap").

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Solution. Identification of intraluminal linear hypodensity adjacent to the vessel wall in the aorta and the pulmonary arteries would support the presence of a "pseudoflap" and help exclude a dissection.[36] If further clarification is needed, ECG-gated CT can also be used for problem solving.

Pathologic Mimics

Pulmonary Vein Thrombus

Challenge. Thrombus located in a pulmonary vein can manifest as a filling defect within an opacified vascular structure in the lungs and therefore can closely mimic a pulmonary artery embolus (Fig. 3A).

Solution. This challenge can be avoided by carefully following the abnormal structure back toward the heart to determine whether it drains into the left atrium with the other pulmonary veins or whether it connects to the main pulmonary artery.

Mucoid Impaction of Bronchi

Challenge. Mucoid impaction of the small airways produces a soft tissue density within a tubular structure in the lungs, which can mimic PE (Fig. 3B). Congenital bronchial atresia can cause additional diagnostic difficulty, as it may contain soft tissue density from mucoid impaction while also demonstrating an abrupt cutoff, which can closely resemble a PE.

Solution. Mucoid impaction or atresia of bronchi can be differentiated from pulmonary emboli by carefully following the abnormal structure. Bronchi will demonstrate a high degree of branching, whereas arteries will demonstrate only bifurcation branching. By following the structures centrally, it may also be possible to demonstrate air in the bronchi or a connection to the main bronchus. Identification of a normally enhancing pulmonary artery running parallel to the opacified structure can also help identify the abnormal structure as a bronchus with mucoid impaction.

Lymph Nodes, Peribronchial Connective Tissue, and Tumor Involvement

Challenge. Hilar, interlobar, and bronchial lymph nodes as well as peribronchial connective tissue can all appear as soft tissue attenuation masses at the branch points of pulmonary arteries. In addition, tumor involvement of the pulmonary arteries (eg, primary lung carcinoma or, rarely, angiosarcoma) can also create areas of soft tissue attenuation abutting or invading the walls of the pulmonary arteries, mimicking PE (Fig. 3C).

Solution. Familiarity with the normal location of pulmonary lymph nodes is essential to differentiating true PE from periarterial soft tissue structures. The cross-referencing of lung windows to soft tissue windows can facilitate the differentiation of soft tissue density adjacent to the pulmonary arteries from soft tissue density within the pulmonary arteries. A key differentiator between lymph nodes and chronic PE is that lymph nodes generally do not invaginate the vessel lumen, whereas chronic pulmonary emboli are within the vessel lumen, with an obtuse interface with the long axis of the lumen.[37]

Pulmonary Artery Stump Thrombosis

Challenge. In patients who have undergone pneumonectomy, the residual stump of the resected pulmonary artery may normally contain thrombus.[38,39] This thrombus has no physiological implication, but it can raise concern for an acute PE (Fig. 3D).

Solution. Awareness of this physiological phenomenon in the presence of pneumonectomy will help avoid misdiagnosis of postsurgical thrombus as a PE.

Acute Versus Chronic PE

Challenge. Acute and chronic PE may be difficult to distinguish (Fig. 4A). Acute PE can occur on a background of chronic PE, further complicating diagnosis in the setting of multiple filling defects.

Solution. An acute PE typically forms an acute angle with the vessel wall, and the distal edge of the thrombus often creates a concave filling defect.[35] Chronic PE, in contrast, usually forms an obtuse angle with the vessel wall, and the distal edge of the thrombus often creates a convex filling defect. Expansion of the vessel is a common finding in acute PE that is absent in chronic PE. Webs, central recanalization, and calcification can be seen in chronic PE but are not observed in acute PE.[35]

Tumor Thrombus Versus PE

Challenge. Tumor thrombus in the pulmonary arterial tree presents a diagnostic challenge, as it can closely resemble PE (Fig. 4B). In addition, PE is more common in oncology patients, which further complicates interpretation.

Solution. Clues for identifying tumor thrombus include a chronic appearance on CT, enhancement of the filling defect, and proximity to a coexisting tumor. Tumor thrombus may also give rise to a beaded appearance of the distal pulmonary arteries.[40]

Aortic CT Angiography

Aortic pathology, most commonly aortic dissection and intramural hematoma, can be mimicked by several normal physiological entities, as well as by extravascular pathology.

Acute Aortic Syndrome Mimics

Physiological Mimics

Left Superior Intercostal Vein

Challenge. The left superior intercostal vein passes adjacent to the arch of the aorta as it drains from the accessory hemiazygos vein posteriorly to the left brachiocephalic vein anteriorly. The close approximation of these 2 vessels can resemble a double lumen in the aorta and can be mistaken for an aortic dissection.

Solution. Careful analysis of the opacified vessels along their full course and targeted arterial phase imaging can prevent misdiagnosis of this normal structure as aortic dissection. In addition, a right-arm contrast injection would also help minimize opacification of the left superior intercostal vein.

Normal Pericardial Recesses

Challenge. The normal pericardial recesses are situated adjacent to the aortic arch at or just above the level of the left pulmonary artery. Both the retroaortic and preaortic portions of the superior pericardial recess can be fluid-filled particularly in the presence of a large pericardial effusion and can mimic an intramural hematoma (Fig. 5A).

Solution. Familiarity with the appearance and location of the pericardial recesses and the use of precontrast and

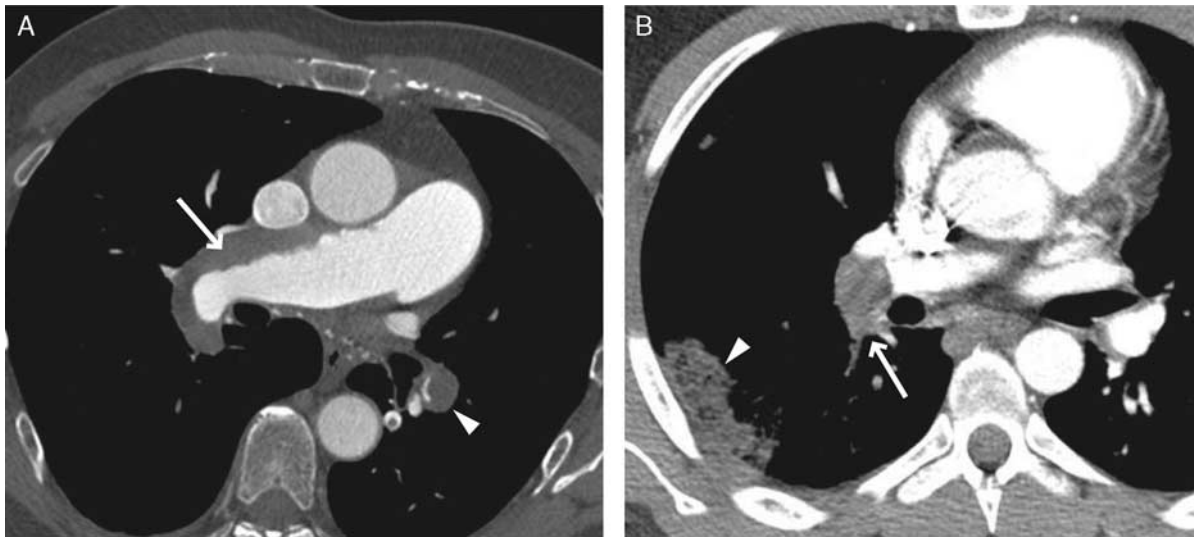

FIGURE 4. Errors in image interpretation: PE mimics. A, Acute PE (arrowhead) in a background of chronic PE (arrow). The presence of these entities is not mutually exclusive, and they can often coexist. B, Tumor thrombus (arrow) with associated right lower-lobe infarct (arrowhead), which can mimic PE causing pulmonary infarct.

postcontrast thin-section CT reduces the likelihood of mistaking this mimic for intramural hematoma.

Normal Thymus

Challenge. In young patients, residual normal thymus situated immediately anterior to the ascending aorta may resemble aortic wall thickening suggesting an intramural hematoma.

Solution. Thymic tissue can be confidently identified by its typical triangular shape, smooth outer contour, and the presence of interdigitating fat within its parenchyma. The young age of these patients is an additional clue that a normal residual thymus rather than an intramural hematoma may be present.

Pathologic Mimics

Aortitis

Challenge. Aortitis is the inflammation of the aortic wall arising from infection or inflammatory aortopathy, most commonly giant cell arteritis and Takayasu arteritis.[41,42] Thickening of the aortic wall due to aortitis can mimic the appearance of an intramural hematoma (Fig. 5B).

Solution. A clinical history of chronic abdominal and back pain or fever and a prior medical history of connective tissue disease can suggest a diagnosis of aortitis, whereas acute aortic syndrome typically manifests as acute chest pain.[42,43] Radiologically, aortitis is usually diffuse and circumferential as it reflects a systemic aortopathy, whereas intramural hematoma is usually focal and crescentic. An intramural hematoma will also demonstrate high attenuation on noncontrast imaging, which is not observed in aortitis. The presence of secondary signs of aortic fibrosis or vasculitis, such as periaortic lymphadenopathy or intramural calcification, can also help distinguish aortitis from dissection.[42]

Atelectasis

Challenge. Periaortic atelectasis in the medial left lung can closely appose the descending aorta and create the appearance of an enhancing crescentic second aortic lumen or intramural hematoma.[44] This appearance can mimic aortic dissection, with the normal aortic wall appearing to represent the dissection flap.

Solution. Familiarity with the appearance and location of periaortic atelectasis on soft tissue and lung windows enables differentiation of this entity from aortic dissection. Atelectatic lung may also be recognized on lung windows, which frequently contains small bubbles of air. Prone CT could be used as a problem-solving tool to improve aeration of the periaortic lung parenchyma.

Severe Anemia

Challenge. Severe anemia reduces the density of the blood in the aortic lumen relative to the aortic wall on CT.[45] This can create a false appearance of hyperdense blood products in the aortic wall, mimicking intramural hematoma (Fig. 5C).

Solution. The apparent hyperdensity of the aortic wall seen in anemia will be diffuse and circumferential, whereas a true aortic wall hyperdensity seen with intramural hematoma will typically be focal and crescentic.

Coexistence of Multiple Pathologies

Multiple aortic pathologies often exist simultaneously because of the interrelationship of the several acute pathologies, including intramural hematoma, penetrating atherosclerotic ulcer, dissection, and pseudoaneurysm.

Intramural Hematoma Versus Dissection

Challenge. Intramural hematomas can coexist with aortic dissection (Figs. 6A, B). In some cases intramural hematomas are the direct precursors to dissection. Although intramural hematoma is optimally visualized on noncontrast imaging, dissection is better seen on contrast-

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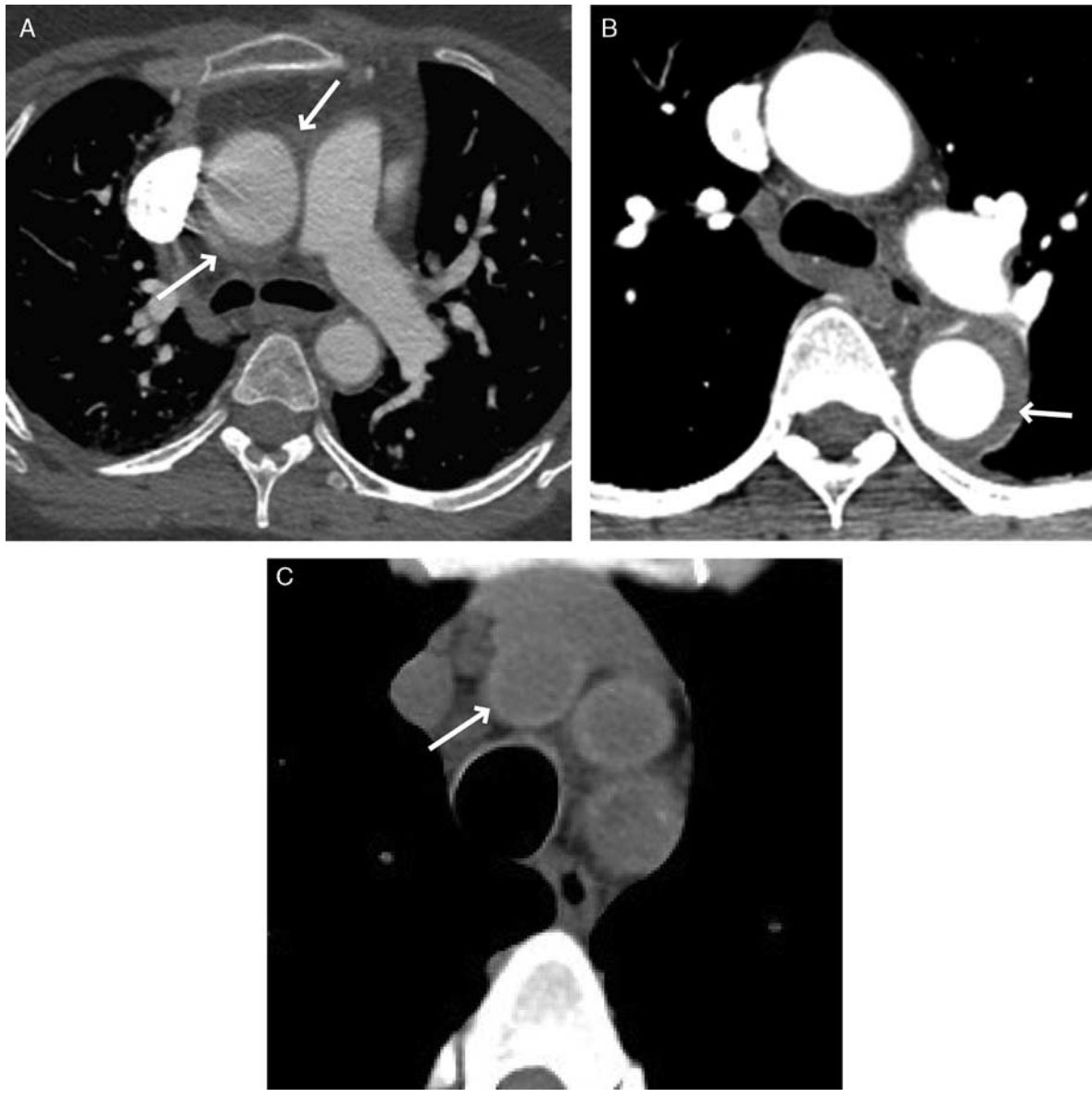

FIGURE 5. Challenges of image interpretation: mimics of acute aortic syndrome. A, Fluid-filled superior pericardial recesses (arrows), which can mimic intramural hematoma. B, Aortitis causing aortic wall thickening (arrow), which can mimic intramural hematoma. Sagittal imaging in this case demonstrated diffuse wall thickening, consistent with aortitis. C, Severe anemia causing a relatively hyperdense aortic wall (arrow) due to reduction in blood density, which can mimic intramural hematoma.

enhanced imaging. Therefore, coexisting intramural hematoma and aortic dissection can be underappreciated.

Solution. The coexistence of aortic dissection with intramural hematoma is best detected using a combination of nonenhanced and contrast-enhanced CT. Careful attention to radiation dose is important when following this dual-scan protocol.

Aortic Pseudoaneurysm Versus Aortitis

Challenge. Aortic pseudoaneurysm can coexist with aortitis (Figs. 6C, D). "Satisfaction of search" may lead to the detection of only 1 of these entities on examinations where both pseudoaneurysm and aortitis are present.

Solution. Satisfaction of search can be avoided in patients with pseudoaneurysm by remembering to fully evaluate the aorta for diffuse wall thickening, which could represent an underlying aortitis. Laboratory values, clinical history, and clinical time of onset of symptoms and comparison with prior imaging, if available, may also provide clues as to whether an infectious or inflammatory aortitis preceded the development of the pseudoaneurysm.

CHALLENGES OF RESULT COMMUNICATION

After accurate and complete interpretation of the images has been performed, the remaining challenge is effective result communication. Ensuring that the

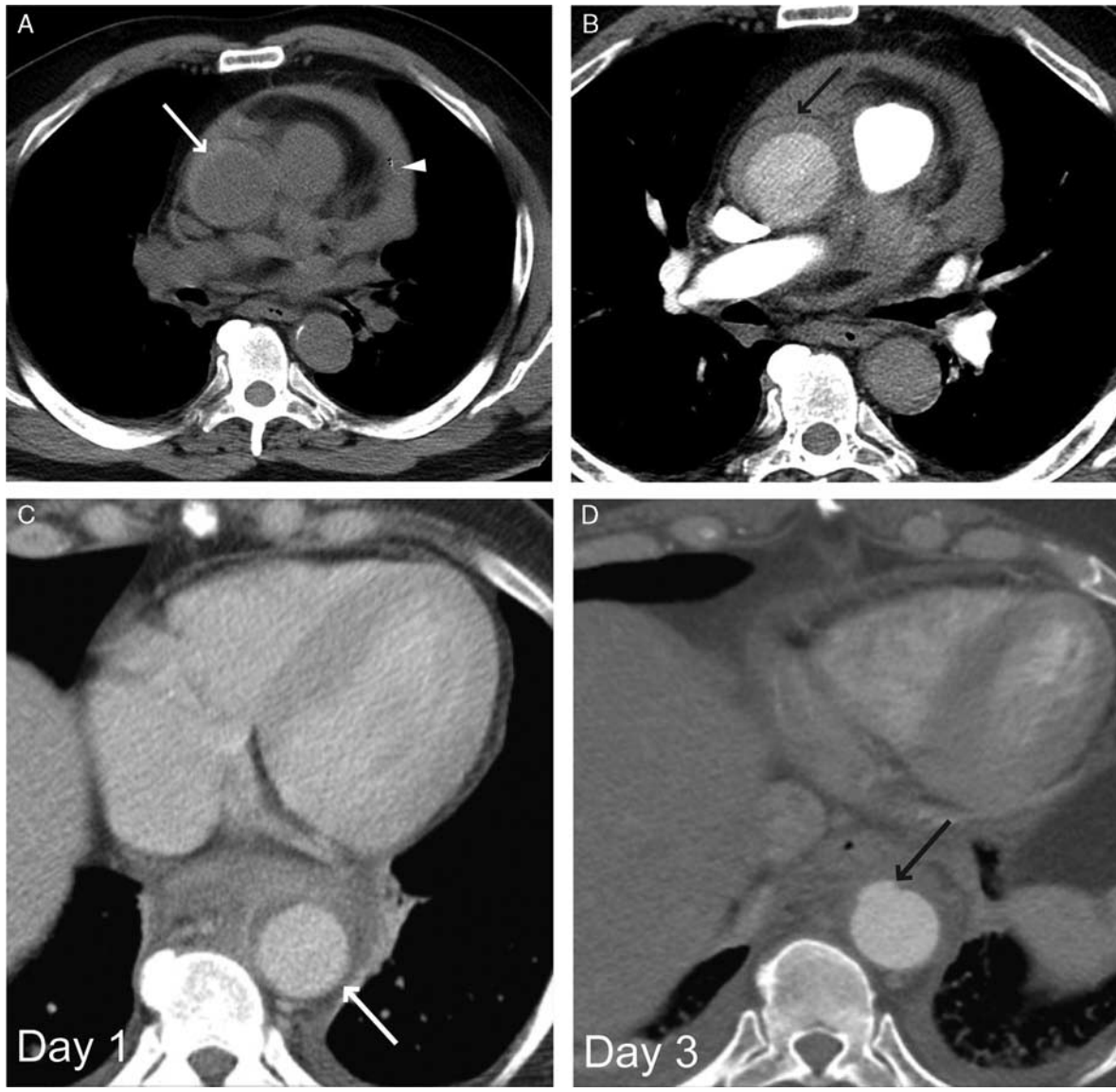

FIGURE 6. Errors in image interpretation: coexisting aortic pathologies. A and B, Intramural hematoma (white arrow) evident on noncontrast imaging with a coexisting aortic dissection (black arrow) evident on contrast-enhanced images. Hemopericardium is also present (white arrowhead). C and D, Aortitis (white arrow) progressing over 3 days to cause a pseudoaneurysm (black arrow), leading to the coexistence of both pathologies on imaging.

radiologist's interpretation is conveyed to the ordering physician and patient in a complete and timely manner is the final step in radiologic evaluation. Best practice guidelines regarding the communication of diagnostic imaging findings have been published by the American College of Radiology (ACR) and other societies. A specific diagnosis should be given where possible. A differential diagnosis may be given where appropriate. Abbreviations or acronyms should be avoided to avoid misinterpretation of the report by the reader.[46]

Preliminary reports can be used to communicate findings needed to direct immediate patient management. However, these reports should be finalized and communicated to the ordering physician in a timely manner. The Society of Cardiovascular Computed Tomography (SCCT) guidelines recommend that all potentially life-threatening findings be reported on the same date of the study, all emergency studies be reported within 24 hours, and all elective studies be reported within 2 working days.[47] A delay in direct communication of acute PE diagnosis, for example, has been shown to correlate with a higher risk for delayed treatment initiation and death within 30 days.[48] Documentation of the communication of results to the ordering physician should be placed in the report.

The ACR guidelines also point out the particular importance of nonroutine communications.[46] Nonroutine communication may be used to report findings needed for urgent or emergent intervention, a significant difference between preliminary and final reports that may adversely affect patient health if not acted upon promptly, or nonemergent findings that may worsen over time without intervention (eg, possible malignancy, infection, unanticipated findings), possibly resulting in an adverse patient outcome.

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Specific guidelines pertaining to CTA studies, practice guidelines issued by the ACR in collaboration with the North American Society of Cardiovascular Imagers (NASCI), Society for Pediatric Radiology (SPR), and Society of Interventional Radiology (SIR), have emphasized the importance of uniform reporting of quantitative imaging data. Specific recommendations are given, for instance, that vessel diameter measurements should be made perpendicular to the median vessel centerline using multiplanar reformation (MPR) images rather than CT source images transverse to the patient.[49]

Appropriate and complete communication regarding a diagnosis of PE will also include information regarding the following: (1) the probable acuity of the PE (acute vs. chronic), (2) the location of the PE (main, lobar, segmental, or subsegmental arteries), and (3) the presence or absence of CT signs of right heart strain. Appropriate communication regarding acute aortic syndrome includes the following: (1) a clear articulation of the urgency associated with the described findings, (2) the presence, size, and location of any fenestrations in dissections, and (3) evidence of end organ injury.

CONCLUSIONS

CT angiography plays a vital role in the evaluation of acute chest pain. Recognizing the common challenges in image acquisition, interpretation, and communication is essential for accurate and timely diagnosis. This review describes and illustrates the most common imaging challenges, including physiological and pathologic mimics, and provides solutions to facilitate accurate diagnosis of the noncoronary causes of chest pain in the emergency setting.

REFERENCES

1. Niska R, Bhuiya F, Xu J. National Hospital Ambulatory Medical Care Survey: 2007 emergency department summary. *Natl Health Stat Report*. 2010;26:1–31.
2. Swap CJ, Nagurney JT. Value and limitations of chest pain history in the evaluation of patients with suspected acute coronary syndromes. *Jama*. 2005;294:2623–2629.
3. May JM, Shuman WP, Strote JN, et al. Low-risk patients with chest pain in the emergency department: negative 64-MDCT coronary angiography may reduce length of stay and hospital charges. *Am J Roentgenol*. 2009;193:150–154.
4. Ozakin E, Kaya FB, Acar N, et al. An analysis of patients that underwent computed tomography pulmonary angiography with the prediagnosis of pulmonary embolism in the emergency department. *ScientificWorldJournal*. 2014;2014:1–5.
5. Tresoldi S, Kim YH, Baker SP, et al. MDCT of 220 consecutive patients with suspected acute pulmonary embolism: incidence of pulmonary embolism and of other acute or non-acute thoracic findings. *Radiol Med*. 2008;113:373–384.
6. Bastarrika G, Thilo C, Headden GF, et al. Cardiac CT in the assessment of acute chest pain in the emergency department. *Am J Roentgenol*. 2009;193:397–409.
7. Hulten E, Pickett C, Bittencourt MS, et al. Outcomes after coronary computed tomography angiography in the emergency department: a systematic review and meta-analysis of randomized, controlled trials. *J Am Coll Cardiol*. 2013;61:880–892.
8. Samad Z, Hakeem A, Mahmood SS, et al. A meta-analysis and systematic review of computed tomography angiography as a diagnostic triage tool for patients with chest pain presenting to the emergency department. *J Nucl Cardiol*. 2012;19:364–376.
9. Patel S, Kazerooni EA. Helical CT for the evaluation of acute pulmonary embolism. *Am J Roentgenol*. 2005;185:135–149.
10. Jones SE, Wittram C. The indeterminate CT pulmonary angiogram: imaging characteristics and patient clinical outcome. *Radiology*. 2005;237:329–337.
11. Shahir K, Goodman LR, Tali A, et al. Pulmonary embolism in pregnancy: CT pulmonary angiography versus perfusion scanning. *Am J Roentgenol*. 2010;195:W214–W220.
12. Hutchinson BD, Navin P, Marom EM, et al. Overdiagnosis of pulmonary embolism by pulmonary CT angiography. *Am J Roentgenol*. 2015;205:271–277.
13. Bucher AM, Kerl MJ, Albrecht MH, et al. Systematic comparison of reduced tube current protocols for high-pitch and standard-pitch pulmonary CT angiography in a large single-center population. *Acad Radiol*. 2016;23:619–627.
14. Nikolaou K, Thieme S, Sommer W, et al. Diagnosing pulmonary embolism: new computed tomography applications. *J Thorac Imaging*. 2010;25:151–160.
15. Gosselin MV, Rassner UA, Thieszen SL, et al. Contrast dynamics during CT pulmonary angiogram: analysis of an inspiration associated artifact. *J Thorac Imaging*. 2004;19:1–7.
16. Tay KL, Ridley LJ. Contrast column interruption artefact in computed tomography pulmonary angiography. *Australas Radiol*. 2005;49:338–341.
17. Ridge CA, Mhuircheartaigh JN, Dodd JD, et al. Pulmonary CT angiography protocol adapted to the hemodynamic effects of pregnancy. *Am J Roentgenol*. 2011;197:1058–1063.
18. Wittram C, Yoo AJ. Transient interruption of contrast on CT pulmonary angiography: proof of mechanism. *J Thorac Imaging*. 2007;22:125–129.
19. Ridge CA, McDermott S, Freyne BJ, et al. Pulmonary embolism in pregnancy: comparison of pulmonary CT angiography and lung scintigraphy. *Am J Roentgenol*. 2009;193:1223–1227.
20. Henk CB, Grampp S, Linnau KF, et al. Suspected pulmonary embolism: enhancement of pulmonary arteries at deep-inspiration CT angiography—influence of patent foramen ovale and atrial-septal defect. *Radiology*. 2003;226:749–755.
21. Oyama-Manabe N, Sugaya T, Yamaguchi T, et al. Noncoronary cardiac findings and pitfalls in coronary computed tomography angiography. *J Clin Imaging Sci*. 2011;1:1–7.
22. Yuan R, Shuman WP, Earls JP, et al. Reduced iodine load at CT pulmonary angiography with dual-energy monochromatic imaging: comparison with standard CT pulmonary angiography—a prospective randomized trial. *Radiology*. 2012;262:290–297.
23. Vrachliotis TG, Bis KG, Haidary A, et al. Atypical chest pain: coronary, aortic, and pulmonary vasculature enhancement at biphasic single-injection 64-section CT angiography. *Radiology*. 2007;243:368–376.
24. Litmanovich D, Zamboni GA, Hauser TH, et al. ECG-gated chest CT angiography with 64-MDCT and tri-phasic IV contrast administration regimen in patients with acute non-specific chest pain. *Eur Radiol*. 2008;18:308–317.
25. Halpern EJ. Triple-rule-out CT angiography for evaluation of acute chest pain and possible acute coronary syndrome. *Radiology*. 2009;252:332–345.
26. Mitsumori LM, Wang E, May JM, et al. Triphasic contrast bolus for whole-chest ECG-gated 64-MDCT of patients with nonspecific chest pain: evaluation of arterial enhancement and streak artifact. *Am J Roentgenol*. 2010;194:W263–W271.
27. Schaefer-Prokop C, Prokop M. CTPA for the diagnosis of acute pulmonary embolism during pregnancy. *Eur Radiol*. 2008;18:2705–2708.
28. Fleischmann D, Mitchell RS, Miller DC. Acute aortic syndromes: new insights from electrocardiographically gated computed tomography. *Semin Thorac Cardiovasc Surg*. 2008;20:340–347.
29. Ko SF, Hsieh MJ, Chen MC, et al. Effects of heart rate on motion artifacts of the aorta on non-ECG-assisted 0.5-sec thoracic MDCT. *Am J Roentgenol*. 2005;184:1225–1230.
30. Godwin JD, Webb WR. Contrast-related flow phenomena mimicking pathology on thoracic computed tomography. *J Comput Assist Tomogr*. 1982;6:460–464.
31. Salvolini L, Renda P, Fiore D, et al. Acute aortic syndromes: role of multi-detector row CT. *Eur J Radiol*. 2008;65:350–358.

32. Soler X, Kerr KM, Marsh JJ, et al. Pilot study comparing SPECT perfusion scintigraphy with CT pulmonary angiography in chronic thromboembolic pulmonary hypertension. *Respirology*. 2012;17:180–184.
33. Castaner E, Gallardo X, Ballesteros E, et al. CT diagnosis of chronic pulmonary thromboembolism. *Radiographics*. 2009; 29:31–50; discussion 50–33.
34. Forman MJ, Mirvis SE, Hollander DS. Blunt thoracic aortic injuries: CT characterisation and treatment outcomes of minor injury. *Eur Radiol*. 2013;23:2988–2995.
35. Wittram C, Maher MM, Yoo AJ, et al. CT angiography of pulmonary embolism: diagnostic criteria and causes of misdiagnosis. *Radiographics*. 2004;24:1219–1238.
36. Pua U, Tan CH. CT diagnosis of pulmonary artery dissection—potential pitfall of multidetector CT. *Br J Radiol*. 2009;82:82–83.
37. Filipek MS, Gosselin MV. Multidetector pulmonary CT angiography: advances in the evaluation of pulmonary arterial diseases. *Semin Ultrasound CT MR*. 2004;25:83–98.
38. Kim SY, Seo JB, Chae EJ, et al. Filling defect in a pulmonary arterial stump on CT after pneumonectomy: radiologic and clinical significance. *Am J Roentgenol*. 2005;185: 985–988.
39. Kwek BH, Wittram C. Postpneumonectomy pulmonary artery stump thrombosis: CT features and imaging follow-up. *Radiology*. 2005;237:338–341.
40. Shepard JA, Moore EH, Templeton PA, et al. Pulmonary intravascular tumor emboli: dilated and beaded peripheral pulmonary arteries at CT. *Radiology*. 1993;187: 797–801.
41. Gornik HL, Creager MA. Aortitis. *Circulation*. 2008;117:3039–3051.
42. Cabero Moyano J, Andreu Magarolas M, Castaner Gonzalez E, et al. Nonurgent aortic disease: clinical-radiological diagnosis of aortitis. *Radiologia (Panama)*. 2013;55:469–482.
43. Loricera J, Blanco R, Hernandez JL, et al. Non-infectious aortitis: a report of 32 cases from a single tertiary centre in a 4-year period and literature review. *Clin Exp Rheumatol*. 2015;33:S-19–S-31.
44. Batra P, Bigoni B, Manning J, et al. Pitfalls in the diagnosis of thoracic aortic dissection at CT angiography. *Radiographics*. 2000;20:309–320.
45. Kamel EM, Rizzo E, Duchosal MA, et al. Radiological profile of anemia on unenhanced MDCT of the thorax. *Eur Radiol*. 2008;18:1863–1868.
46. Radiology, A.C.o. *ACR Practice Parameter for Communication of Diagnostic Imaging Findings*. 2014 [cited 2016 July 3]; Available at: http://www.acr.org/~/media/C5D1443C9EA4424AA12477D1AD1D927D.pdf.
47. Tomography, T.S.o.C.C. *SCCT Guidelines for the Interpretation and Reporting of Coronary Computed Tomographic Angiography* 2010 [cited 2016 July 3]; Available at: http://www.scct.org/advocacy/coverage/PubGuidelines.pdf.
48. Kumamaru KK, Hunsaker AR, Kumamaru H, et al. Correlation between early direct communication of positive CT pulmonary angiography findings and improved clinical outcomes. *Chest*. 2013;144:1546–1554.
49. American College of Radiology, N.A.S.o.C.I., Society for Pediatric Radiology, and Society of Interventional Radiology. *ACR/NASCI/SIR/SPR Practice Parameter for the Performance and Interpretation of Body Computed Tomography and Angiography (CTA)*. 2014 [cited 2016 July 3]; Available at: http://www.acr.org/~/media/ACR/Documents/PGTS/guidelines/Body_CTA.pdf.

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SA-CME EXAMINATION QUESTIONS

*1) Which of the following does not typically mimic pulmonary embolism?
- (a) Left atrial appendage thrombus
- (b) Impacted bronchi
- (c) Perihilar lymph nodes
- (d) Atherosclerotic plaque

Please see the following references for further study:

1. Wittram C, Maher MM, Yoo AJ, Kalra MK, Shepard JA, McLoud TC. CT angiography of pulmonary embolism: diagnostic criteria and causes of misdiagnosis. *Radiographics*. 2004;24:1219-1238.

*2) Which of the following does not typically mimic aortic dissection?
- (a) Aortitis
- (b) Severe anemia
- (c) Atherosclerosis
- (d) Flow-related phenomenon

Please see the following references for further study:

1. Batra P, Bigoni B, Manning J, et al. Pitfalls in the diagnosis of thoracic aortic dissection at CT angiography. Radiographics. 2000;20:309-320.

*3) Which of the following is not a part of appropriate and complete communication regarding a diagnosis of pulmonary embolism?
- (a) Timely communication of the findings
- (b) Description of the location of the lesions to the subsegmental level
- (c) Description of acuity of the lesions
- (d) Description of the presence or absence of pericardial effusion

Please see the following references for further study:

1. Araoz PA, Gotway MB, Harrington JR, Harmsen WS, Mandrekar JN. Pulmonary embolism: prognostic CT findings. *Radiology*. 2007;242:889-897.

*4) Which of the following statements is false?
- (a) Chronic pulmonary embolism typically forms an acute angle with the vessel wall and demonstrates a concave trailing edge.
- (b) Webs, central recanalization, and calcification are characteristic findings of chronic pulmonary embolism.
- (c) Tumor thrombus may give rise to a beaded appearance of the distal pulmonary arteries.

(d) If an apparent pulmonary artery flap is related to pulsation artifact, there will typically be a similar pseudo-flap in the aortic root on the same image.

Please see the following references for further study:
1. Wittram C, Maher MM, Yoo AJ, Kalra MK, Shepard JA, McLoud TC. CT angiography of pulmonary embolism: diagnostic criteria and causes of misdiagnosis. *Radiographics*. 2004;24:1219-1238.

*5) Which of the following is false regarding the differentiation of intramural hematoma from aortitis?
(a) Acute chest pain is more indicative of aortitis than intramural hematoma.
(b) Crescentic wall thickening is more often seen with intramural hematoma than aortitis.
(c) High attenuation within the aortic wall on non-contrast imaging is more consistent with intramural hematoma than aortitis.
(d) Periaortic lymphadenopathy is more commonly seen with aortitis than intramural hematoma.

Please see the following references for further study:
1. Gornik HL, Creager MA. Aortitis. *Circulation*. 2008;117:3039-3051.
2. Chao CP, Walker TG, Kalva SP. Natural history and CT appearances of aortic intramural hematoma. *Radiographics*. 2009;29:791-804.

*6) Which of the following is not used to differentiate pulmonary embolism from mucoid impaction of bronchi?
(a) Following the opacified structure centrally
(b) Diameter of opacified structure
(c) Adjacent structures
(d) Branching pattern of opacified structure

Please see the following references for further study:
1. Wittram C, Maher MM, Yoo AJ, Kalra MK, Shepard JA, McLoud TC. CT angiography of pulmonary embolism: diagnostic criteria and causes of misdiagnosis. *Radiographics*. 2004;24:1219-1238.
2. Aviram G, Levy G, Fishman JE, Blank A, Graif M. Pitfalls in the diagnosis of acute pulmonary embolism on spiral computer tomography. *Current problems in diagnostic radiology*. 2004;33:74-84.

*7) Which of the following is true regarding artifacts encountered on acute chest pain imaging?
(a) Pulsation artifact is a concern in aortic imaging but does not affect pulmonary artery imaging.
(b) Streak artifact is due to the attenuation of the x-ray beam by a highly attenuating structure in the region of interest, such as bone or metal.
(c) Beam hardening artifact can be reduced or avoided by appropriately positioning the patient.
(d) Respiratory motion artifact can be reduced by asking the patient to perform a shallow breath hold

Please see the following references for further study:
1. Barrett JF, Keat N. Artifacts in CT: recognition and avoidance. *Radiographics*. 2004;24:1679-1691.

*8) Which of the following statements regarding vessel opacification during CTA imaging is false?
(a) In pulmonary artery CTA, good main pulmonary arterial opacification is defined as greater than or equal to 100 HU.
(b) In double rule out CTA, imaging is performed using bolus tracking at the left atrium with a threshold trigger of 100 absolute HU.
(c) In aortic CTA, reduction of flow-related artifacts is typically achieved by initiating image acquisition when the absolute attenuation in the descending aorta reaches 100 HU.
(d) In pulmonary artery CTA, using a wider window setting with the level set at 100 HU may improve visualization of subtle arterial webs observed in chronic PE.

Please see the following references for further study:
1. Jones SE, Wittram C. The indeterminate CT pulmonary angiogram: imaging characteristics and patient clinical outcome. *Radiology*. 2005;237:329-337.

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