CT diagnosis of nonspecific acute chest pain in the emergency department: from typical acute coronary syndrome to various unusual mimics

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Learning objectives

The purpose of this article is

1. to present an overview of how to choose an appropriate CT protocol in patients with nonspecific acute chest pain,

2. and to provide specific CT findings of acute coronary syndrome (ACS) and various mimics.
Background

Acute coronary syndrome (ACS) consists of ST-elevated myocardial infarction (STEMI), non-ST elevated myocardial infarction (NSTEMI), and unstable angina (UA). Diagnosis of ACS is straight forward in patients with any combination of typical substernal chest pain lasting more than 30 minutes, elevation of cardiac enzyme levels, and ischemic ECG change. However, substantial proportion of the patients who eventually turn out to have ACS present with atypical chest pain, normal initial cardiac enzyme levels, or nonspecific ECG change. In addition, there is a substantial overlap of clinical and laboratory findings between ACS and the various mimics of ACS, resulting in diagnostic difficulty.\(^1\) Thus, it is a challenging task to establish a precise diagnosis based on clinical findings in patients with nonspecific acute chest pain. In this situation, CT angiography often provides important clue to the diagnosis. Thus, the purpose of this poster is to provide specific CT findings of ACS and various mimics.
Findings and procedure details

Various CT protocols in the emergency department

In general, five different types of CT protocols are used for the evaluation of acute chest pain in the emergency department (ED). These consist of dedicated coronary CT angiography (CTA), triple rule out (TRO), pulmonary CTA, aortic CTA, and routine chest CT. In terms of z-axis coverage, dedicated coronary CTA has the smallest coverage extending from the tracheal carina to the upper half of the diaphragm including the entire heart. In contrast, aortic CTA has largest z-axis coverage from the thoracic inlet to both femoral heads. TRO, pulmonary CTA, and routine chest CT have similar z-axis coverage that includes the entire chest. Thus, if we consider only z-axis coverage, aortic CTA appears to have the greatest radiation exposure. However, if TRO is performed with retrospective gating, it has the greatest radiation exposure due to ECG gating (i.e., use of a low pitch) with 64-slice MDCT. If the heart rate is regular and slow, prospective gating can be performed with the advantage of lower radiation exposure compared with retrospective gating. Prospective gating should be considered primarily in younger patients due to their relatively higher risk of radiation induced cancer. However, disadvantages of prospective gating should be also be considered, including the absence of an alternative cardiac phase to reconstruct in the event of motion and the lack of functional information.

Challenges in selecting an appropriate CT protocol in emergency department

The main difficulty in selecting an appropriate ED chest CT protocol is the substantial overlap of clinical symptoms and signs. Thus, cardiothoracic radiologists often encounter occasions in which the inappropriate CT protocol is used in daily practice. The simplest way to avoid this issue is to enumerate possible causes of acute chest pain based on objective pretest probability, rather than personal experience. As an example, Well's criteria can be used to provide a more objective pretest probability of pulmonary embolism. Using these criteria, investigators found that a score supported by a negative d-dimer is associated with a low 3-month incidence of thromboembolic events (0.5%). As a more objective assessment of pretest probability of aortic dissection, one can use three variables suggested by Kodolitch et al. Their study indicated that the possibility of aortic dissection is low (4%) in patients lacking the following three variables: aortic pain (immediate onset or ripping nature, or both), mediastinal widening on chest radiography, and a substantial pulse or blood pressure differential, defined in the study as a difference in the blood pressure #20 mm Hg between both arms. The presence of one
of the three variables indicates moderate pretest probability, whereas any combination of two of the three variables denotes a high pretest probability in the study.  

For an objective assessment of pretest probability of ACS, if a patient has typical substernal chest pain lasting more than 30 minutes and evidence of ischemia on ECG or a significant coronary stenosis (#50%) on a previous coronary angiography, pretest probability is high. In contrast, if a patient has atypical chest pain or no evidence of ischemia on ECG, the pretest probability is low to intermediate. Coronary CTA is only indicated in patients with a low to intermediate risk for ACS. In summary, the appropriate CT protocol should be selected based on objective assessment of pretest probability of the three major diagnoses (ACS, pulmonary embolism, and aortic dissection).

How to choose best CT option in patients with nonspecific acute chest pain

To explain how to select the optimal CT protocol, clinical settings are divided into four scenarios. Scenarios 3 and 4 below are not typically applicable in the ED setting because CT in these conditions is performed after a negative coronary angiography. However, as most diseases related to these scenarios may present with nonspecific acute chest pain that mimics ACS, these scenarios are discussed in this section.

1. Clinical suspicion of acute coronary syndrome (low-to-intermediate pretest probability for ACS) and normal initial troponin

Unfortunately, there are several mimics of ACS in this situation (Table 1).

Table 1. Mimics of acute coronary syndrome

<table>
<thead>
<tr>
<th>Aorta</th>
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<tr>
<td>Intramural hematoma (IMH)</td>
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<td>Penetrating aortic ulcer</td>
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<td>Aortic dissection</td>
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Pulmonary
Pulmonary embolism
Pneumonia
Pneumothorax
Pleuritis
Cardiac

Stress induced cardiomyopathy
Pericarditis/myocarditis
Cardiac tamponade
Paracardial fat necrosis

Musculoskeletal

Rib fracture/costochondritis

Gastrointestinal

Pancreatitis
Peritonitis
Cholecystitis
Renal stone
Peptic ulcer
Gastrointerstial reflux
Esophageal perforation
Esophgeal spasm
In this scenario, dedicated coronary CTA or TRO should be indicated to differentiate ACS from the various mimics. One study reported that diagnostic tests for at least two of three major diagnoses were performed in 21.6% of patients with nonspecific acute chest pain. Thus, approximately one-fifth of ED patients with nonspecific acute chest pain may potentially undergo TRO. However, the 2010 ACCF guideline classified TRO in the uncertain category for appropriateness. Thus, TRO should be used with caution, especially in younger patients due to its high radiation dose. In addition, it should be remembered that most often pulmonary embolism and aortic dissection can be diagnosed on dedicated coronary CTA.

2. High suspicion for aortic dissection or pulmonary embolism

In a patient with a high probability of pulmonary embolism or aortic dissection, dedicated pulmonary embolism or aortic CTA should be performed because these protocols have a lower radiation dose (i.e., no ECG-gating). In addition, TRO is not specified for the evaluation of these disease entities. For example, the field of view of the TRO does not include the abdomen and pelvis. Thus, the entire extent of aortic dissection cannot be assessed on TRO. If second or third generation dual source CT is available, aortic CTA with a high pitch mode rather than conventional pitch aortic CTA can be performed with an advantage of lower radiation dose and potential increase in the detail of coronary arteries. One study suggested that using a high pitch mode, even a non-gated acquisition is associated with a decreased radiation exposure and increased detail of the aorta, coronary arteries, and pulmonary artery compared with conventional pulmonary CTA.

3. Increase in troponin and normal coronary angiography

The third situation occurs in patients with increase in troponin and normal coronary angiography. In many patients with increased troponin and nonspecific acute chest pain, coronary angiography is performed as the initial study, but coronary artery evaluation is often negative. In this scenario, Stanford type A aortic dissection with coronary artery involvement and pulmonary embolism with myocardial injury as well as nonobstructive myocardial infarction, myocarditis and stress induced cardiomyopathy should be included in the differential diagnosis. Therefore, the CT protocol in this situation should cover the entire chest, and good contrast enhancement of both the pulmonary arteries and aorta is required to identify aortic dissection with coronary artery involvement and pulmonary embolism with myocardial injury. Severe dilatation of the right ventricle in patients with massive or submassive pulmonary embolism may itself lead to an increase in troponin levels. Delayed CT with a low kV can be performed additionally to identify delayed enhancement in patients with myocarditis or nonobstructive myocardial infarction.
4. Normal troponin and normal coronary angiography

In this scenario, it is also necessary to differentiate various mimics of ACS. Acute chest pain secondary to abdominal and esophageal causes should be included in the differential diagnosis in this clinical setting. Thus, z-axis coverage should include upper abdomen to exclude abdominal causes mimicking ACS.

*Typical CT findings of acute coronary syndrome

The main role of coronary CTA in the ED is to exclude the presence of ACS in patients with low to intermediate risk due to its high negative predictive value to expedite early discharge. However, coronary CTA also has the potential to diagnose ACS with moderate specificity. The less than perfect specificity of coronary CTA in identifying significant coronary stenosis is mainly caused by blooming or motion artifact. Coronary CT findings of acute myocardial infarction consist of critical stenosis (#70%) by mixed or noncalcified plaque suggesting rupture of vulnerable plaque and/or hypoperfusion in the myocardial segments corresponding to the occluded artery (Fig. 1-3).27
Fig. 1: A case of acute myocardial infarction demonstrated on coronary CT angiography. A 55-year-old man presented with nonspecific acute chest pain. Coronary CT angiography was performed in this patient as initial troponin and ECGs were negative. Abrupt total occlusion (arrows) probably due to plaque rupture at mid portion of left anterior descending coronary artery (LAD) on curved multiplanar reformatted (MPR). Contrast enhancement at the distal LAD after the occlusion is probably due to collateral flow.

References: - Bundang/KR
Fig. 2: Same patient with Fig.1. Subendocardial hypoperfusion (arrowheads) is noted at the antero-septal wall of mild portion of the left ventricle on a short-axis view, indicating acute myocardial infarction of LAD territory.

References: - Bundang/KR
Fig. 3: Same patient with Fig. 1. Subsequent coronary angiography confirms the total occlusion of mid LAD (arrow).

References: - Bundang/KR However, one study reported that prevalence of nonobstructive myocardial infarction (i.e., <50% coronary stenosis on coronary angiography) is not negligible. Nonobstructive myocardial infarction may occur as a result of embolic infarction, sustained coronary spasm, or spontaneous thrombolysis. The presence of wall motion abnormalities in the left ventricle should be assessed in patients presenting with acute chest pain if coronary CTA was performed with retrospective gating. Investigators have shown that analysis of wall motion abnormality in the left ventricle on CT is helpful to identify nonobstructive myocardial infarction, and to determine the culprit artery (Fig. 4-5) that resulted in a cardiac event, particularly
in patients with multi-vessel coronary artery disease or a nondiagnostic scan due to motion or blooming artifact.29

Fig. 4: A case of acute myocardial infarction with akinesia in the territory of the left circumflex coronary artery. A 55-year-old man presented with nonspecific acute chest pain. Emergent coronary CTA was performed. Multiple ≥ 50% stenoses or motion blurring are noted in the left anterior descending coronary, right coronary, and left circumflex coronary artery (not demonstrated here). Thus, it is difficult to determine which coronary artery is the culprit one only based on coronary CTA. Diastolic (Fig.4) and systolic (Fig.5) cine image of the left ventricle shows akinesia (i.e., no increase in the myocardial thickness) in the territory of the left circumflex coronary artery (arrows), indicating culprit artery.

References: - Bundang/KR
Fig. 5: A case of acute myocardial infarction with akinesia in the territory of the left circumflex coronary artery. A 55-year-old man presented with nonspecific acute chest pain. Emergent coronary CTA was performed. Multiple $\geq 50\%$ stenoses or motion blurring are noted in the left anterior descending coronary, right coronary, and left circumflex coronary artery (not demonstrated here). Thus, it is difficult to determine which coronary artery is the culprit one only based on coronary CTA. Diastolic (Fig. 4) and systolic (Fig. 5) cine image of the left ventricle shows akinesia (i.e., no increase in the myocardial thickness) in the territory of the left circumflex coronary artery (arrows), indicating culprit artery.

References: - Bundang/KR On occasion, an occluded proximal coronary arterial segment can be identified even on non-gated routine chest CT (Fig. 6-8).
Fig. 6: Acute myocardial infarction demonstrated on non-gated aortic CT angiography in a 42-year-old man. Left anterior descending coronary artery (LAD) occlusion by thrombus (arrow) is noted on an axial CT image at the level of pulmonary conus, even though ECG-gated was not performed. This case may indicate that radiologists should carefully evaluate the proximal coronary arteries even in non-gated routine chest CT. 

References: - Bundang/KR
Fig. 7: Same patient with Fig. 6. Typical hypoperfusion (arrowheads) is noted in the LAD territory on an axial image at the level of left ventricle.

References: - Bundang/KR
**Fig. 8**: Same patient with Fig. 6. Emergent coronary angiography demonstrates the total occlusion of LAD (arrow), confirming the CT findings.

**References**: Bundang/KR Thus, it is recommended that radiologists carefully evaluate the proximal coronary arteries even on routine chest CT.

In summary, coronary CT findings of ACS are

1. **Significant stenosis (>70%) with noncalcified or mixed plaque**
2. **Hypoperfusion without myocardial thinning**
3. **Wall motion abnormality in the corresponding arterial territory**
Mimics of acute coronary syndrome with increase in troponin

*Mimics with increase in troponin

Aortic dissection with coronary involvement

Pulmonary embolism with myocardial injury

Myocarditis

Stress induced cardiomyopathy

1. Aortic dissection with coronary involvement

Stanford type A aortic dissection is defined as an aortic dissection involving the ascending aorta, irrespective of extension of dissecting flap into the descending aorta. If an intimal flap extends into a coronary artery wall, acute myocardial infarction (Fig. 9-10) may occur as a complication.
Fig. 9: Stanford type A aortic dissection as a mimic of acute coronary syndrome complicated by acute myocardial infarction. 48-year-old man presented with acute chest pain of abrupt onset. Subendocardial hypoperfusion (arrowheads) is noted in the territory of left anterior descending coronary artery on an axial CT image at the level of the left atrium, indicating acute myocardial infarction.

References: - Bundang/KR
Fig. 10: Same patient with Fig.9. Severe narrowing of the true lumen (black arrows) of the left main coronary artery caused by thrombosis in the false lumen (white arrows) (F) is demonstrated on a coronal reformatted image.

References: - Bundang/KR It is possible to misinterpret secondary ACS complicated by Stanford type A aortic dissection as ACS per se, leading to a recommendation for coronary stent insertion. In this situation, administration of antiplatelet agents or tissue plasminogen activators may result in disastrous consequences. If ECG-gating is used in patients with Stanford type A aortic dissection, the presence of coronary artery involvement by intimal flap extension can be precisely determined.

2. Pulmonary embolism with myocardial injury

One study reported that approximately one-third of patients with pulmonary embolism have the triad of acute chest pain, increase in troponin levels, and ECG changes simulating myocardial ischemia.30 Thus, a precise diagnosis of pulmonary embolism can be quite difficult. Increase in troponin in patients with massive or sub-massive pulmonary
embolism (Fig. 11-12) may result from myocardial injury secondary to severe stretching or ischemia.

Fig. 11: A case of massive pulmonary embolism with right ventricular dilatation and increase in troponin due to myocardial injury in a 62-year-old man. Multiple pulmonary emboli (arrows) are demonstrated on an axial CT image at the level of the main pulmonary artery.

References: - Bundangan
Fig. 12: Same with Fig. 11. Marked enlargement of the right ventricle (RV) is noted on an axial CT image at the level of the left ventricle (LV). First impression of the emergency physician was acute myocardial infarction due to increase in troponin in this case.

References: - Bundang/KR

3. Stress induced cardiomyopathy

The pathophysiology of stress induced cardiomyopathy (Takotsubo cardiomyopathy) remains uncertain, although a marked increase in catecholamines is believed to play an important role.\textsuperscript{31 -33} Stress induced cardiomyopathy typically occurs in postmenopausal woman with a triggering emotional or physical stressor. There is a substantial overlap of clinical, ECG, and laboratory findings between stress-induced cardiomyopathy and ACS. One study reported that more than 80% of patients with stress induced cardiomyopathy have an increase in troponin levels and ECG changes such as ST-elevation or T wave inversion, leading to coronary catheterization as a frequent downstream test.\textsuperscript{31} Although there is no diagnostic criteria that are universally accepted, the following conditions should be present to make the diagnosis of the apical type of stress induced cardiomyopathy: 1. apical ballooning of the left ventricle that extends beyond a coronary
artery territory and preserved or hyperdynamic contraction in the base of the heart on echocardiography, MRI, or cardiac CT (Fig. 13-14); 2. absence of an explanatory occlusive coronary lesion on coronary angiography; 3. full recovery of wall motion abnormality within several weeks. However, according to recent studies, the presence of concomitant obstructive coronary artery disease does not exclude the diagnosis of stress induced cardiomyopathy.31-33

Fig. 13: A case of apical stress-induced cardiomyopathy in a 65-year-old woman. 4-chamber view on diastolic phase shows apical thrombus (arrowhead).

References: - Bundang/KR
Fig. 14: Same patient with Fig. 13. 4-chamber view on systolic phase shows typical apical ballooning (arrows) and compensatory hypercontractility of basal wall (*).

References: - Bundang/KR Importantly, nonobstructive myocardial infarction and myocarditis should be excluded to make the diagnosis of stress induced cardiomyopathy because these entities can also show cause an increase in troponin and nonobstructed coronary arteries on coronary angiography. Cardiac MR is important to differentiate stress induced cardiomyopathy from nonobstructive myocardial infarction and myocarditis. In contrast to nonobstructive myocardial infarction and myocarditis, most stress induced cardiomyopathies do not show delayed enhancement on MR, although <5% cases of those may show patchy enhancement on delayed enhancement MR. In addition, on cine MR, wall motion abnormality of the left ventricle in patients with stress induced cardiomyopathy does not conform a specific
vascular territory as demonstrated in acute myocardial infarction. If cardiac CT is performed with retrospective ECG-gating, CT has the potential to precisely evaluate both coronary anatomy and left ventricular wall motion abnormality. As a potential initial diagnostic tool for this entity, CT may obviate the need to perform both MR and coronary angiography. Thus, it is essential for radiologists to analyze cine imaging to identify stress induced cardiomyopathy on cardiac CT. Although it is relatively rare compared with the apical ballooning type, radiologists should be aware of the existence of basal and mid-ventricular types of stress induced cardiomyopathy with preserved apical contractile function.

4. Cardiac inflammatory disease; Myocarditis/ pericarditis

The diagnosis of myocarditis can be difficult because of substantial overlap of clinical symptoms and signs with other causes of acute chest pain. The principal imaging tools for the diagnosis of myocarditis have been MRI and echocardiography rather than CT because delayed CT scan would need to be performed to identify the typical subepicardial patch delayed enhancement, leading to increased radiation exposure and longer stay in the CT room. However, delayed CT, typically at 5 minutes can be obtained with a minimal radiation dose (80 kV). Studies have shown that diagnostic accuracy to identify delayed enhancement on CT is comparable to that of delayed enhancement MR. Thus, delayed CT scanning may be a valuable option to identify myocarditis in patients with an increase in troponin levels and negative or nonobstructive coronary angiography. Typical CT findings of pericarditis are pericardial effusion with enhancement of pericardium.

Mimics of acute coronary syndrome with normal troponin

* Mimics with normal troponin

Aortic dissection without coronary involvement

Pulmonary embolism without myocardial injury

Paracardial (epipericardial) fat necrosis

Musculoskeletal causes such as rib fracture and costochondritis

Various abdominal causes such as esophageal or peptic ulcer perforation, cholecystitis, renal colic, and pancreatitis
1. Epipericardial fat necrosis

The pathophysiologic mechanism of this entity has not been elucidated clearly. A possible cause of paracardial fat necrosis is ischemia by torsion or hemorrhagic necrosis due to increased intrathoracic pressure related to a Valsalva maneuver.\textsuperscript{34, 35} The term "epipericardial fat necrosis" is a misnomer because the place where the event occurs is not pericardial but paracardiac fat. Epipericardial fat necrosis is a benign entity with a self-limiting course. However, this condition can be misinterpreted as ACS, leading to coronary catheterization and its associated morbidity and mortality. Thus, a precise CT diagnosis of epipericardial fat necrosis is important. The typical triad of CT findings of epipericardial fat necrosis (Fig. 15) is an encapsulated fatty lesion with inflammatory changes, including paracardial fat stranding, pericardial thickening, and a small left pleural effusion.\textsuperscript{34, 35} Pericardial thickening and small amount of the left pleural effusion are presumably secondary findings of paracardial fat inflammation.
Fig. 15: A 45-year-old woman with paracardial fat necrosis: a mimic of acute coronary syndrome unaccompanied by increase in troponin. An axial CT image at the level of the left atrium shows encapsulated fatty lesion with inflammatory changes, including paracardial fat stranding (large arrows), mild pericardial thickening (small arrow) and small amount of the left pleural effusion (arrowheads). These CT findings are a typical triad of paracardial fat necrosis.

References: - Bundang/KR

2. Musculoskeletal causes of acute coronary syndrome; acute rib fracture

It is important to identify the presence of acute rib fracture (Fig. 16) because it may be an alternative cause of acute chest pain, and its recognition may obviate invasive coronary catheterization. It should be stressed that rib fractures can occur without a history of blunt trauma (e.g., chronic cough or stress-related injury).

Fig. 16: A case of acute rib fracture initially misinterpreted as acute coronary syndrome in a 45-year-old woman. The patient presented with nonspecific acute chest pain. The attending physician did not consider rib fracture as a differential diagnosis initially. As acute coronary syndrome cannot be reliably excluded, coronary angiography was performed, but coronary arteries were negative. Routine chest CT
was performed to identify alternative cause of acute chest pain. Buckling (arrow) indicating rib fracture is noted at the left 6th rib on an axial CT image with bone window setting.

References: - Bundang/KR

3. Abdominal causes of mimics of acute coronary syndrome

Various abdominal and esophageal diseases can mimic ACS. Thus, radiologists should carefully evaluate abdominal organs and the esophagus so as not to miss important mimics of ACS. These include peritonitis secondary to rupture of peptic ulcer (Fig. 17), reflux esophagitis, esophageal spasm, esophageal perforation or dissection (Fig. 18-19), cholecystitis, pancreatitis, or spontaneous dissection of the celiac or superior mesenteric artery.

Fig. 17: A case of peritonitis mimicking acute aortic syndrome in a 48-year-old woman. As the primary concern of emergency physician was acute aortic syndrome, aortic CT angiography was performed in this case. Free air (arrows) in the intraperitoneal space
is noted on an axial CT image at the level of liver. Note wall thickening at the gastric antrum (arrowheads) due to gastric ulcer.

References: - Bundang/KR

Fig. 18: A case of esophageal rupture as a mimic of acute coronary syndrome in a 44-year-old woman. The patient presented with sudden acute chest pain during meal. Primary concern of emergency physician was acute aortic syndrome. Thus, aortic CT angiography was performed in this case. Retro-esophageal air collection (arrows) suggesting esophageal perforation or dissection is noted on an axial CT image at the level of left atrial appendage.

References: - Bundang/KR
**Fig. 19:** Same patient with Fig. 17. Retro-esophageal air collection (arrowheads) suggesting esophageal perforation or dissection is noted on sagittal reformatted image. E in the Fig. B indicates esophageal air.

**References:** - Bundang/KR
Images for this section:

**Fig. 7:** Same patient with Fig. 6. Typical hypoperfusion (arrowheads) is noted in the LAD territory on an axial image at the level of left ventricle.

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Fig. 4: A case of acute myocardial infarction with akinesia in the territory of the left circumflex coronary artery. A 55-year-old man presented with nonspecific acute chest pain. Emergent coronary CTA was performed. Multiple # 50% stenoses or motion blurring are noted in the left anterior descending coronary, right coronary, and left circumflex coronary artery (not demonstrated here). Thus, it is difficult to determine which coronary artery is the culprit one only based on coronary CTA. Diastolic (Fig.4) and systolic (Fig.5) cine image of the left ventricle shows akinesia (i.e., no increase in the myocardial thickness) in the territory of the left circumflex coronary artery (arrows), indicating culprit artery.

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Conclusion

1. List up any possible causes of acute chest pain based on an objective assessment of pretest probability.

2. Do not hesitate to perform appropriate imaging if clinically suspected.

3. CT often provides specific causes of nonspecific acute chest pain.