Case Report

Multislice coronary computed tomographic angiography in emergency department presentations of unsuspected acute myocardial infarction

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**KEYWORDS:**
- Acute myocardial infarction;
- CCTA;
- Coronary artery disease;
- Emergency department;
- Transmural hypodensity

**BACKGROUND:** Coronary computed tomographic angiography (CCTA) is not indicated in the setting of acute myocardial infarction in the emergency department (ED). Nonetheless, acute coronary syndromes may have atypical presentations, and CCTA may be inadvertently performed in this setting.

**OBJECTIVES:** This study was designed to determine the frequency and characteristics of CCTA imaging of unsuspected acute myocardial infarction in the ED.

**METHODS:** All CCTAs performed in the ED at Lenox Hill Hospital were reviewed for clinical indications and subsequent course; patients with documented acute myocardial infarction were identified.

**RESULTS:** Of the 500 CCTAs performed on ED patients in the Lenox Hill laboratory, 5 patients (1%) were imaged during the initial phase of an unsuspected acute myocardial infarction; in all cases the CCTAs were key to the diagnosis. The imaging characteristics were (1) total or subtotal occlusion and (2) transmural hypodensity in the infarct area.

**CONCLUSION:** Although acute myocardial infarction on CCTA in ED patients is an infrequent event, proper and prompt recognition is critical for appropriate patient care, particularly as applications to the ED increase.

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**Introduction**

The introduction of multidetector coronary computed tomographic angiography (CCTA) into the emergency department (ED) has provided a new tool for the rapid and accurate evaluation of patients presenting with a stable symptom complex, a normal set of cardiac enzymes, and an electrocardiogram that does not show acute changes attributable to coronary disease. Patients with acute myocardial infarction may present to the ED with atypical symptoms, electrocardiographic changes that are not specific for an acute myocardial infarction, and a normal initial troponin and creatinine phosphokinase or enzyme elevations thought to be secondary to myopericarditis. This report characterizes the frequency and appearance of inadvertent CCTA imaging of acute myocardial infarction.

**Cases**

All CCTAs performed on ED patients at Lenox Hill Hospital (n = 500) were reviewed for clinical indications and subsequent course. Five patients (1%) with documented acute myocardial infarction by elevated enzymes were
identified; they were acquired during the initial phase of an unsuspected acute myocardial infarction. CCTA and catheter-based coronary angiography were acquired and analyzed by previously described standard techniques. All CCTAs were retrospectively gated without dose modulation. Myocardial density was visually classified as normal or hypodense and was verified by a ratio of infarct HU/normal HU of <0.75 within a constant region of interest. Hypodense areas were classified as either subendocardial (<50% of the myocardial thickness) or transmural (≥50% of the myocardial thickness). The study was approved by the institutional review board of Lenox Hill Hospital.

Case 1

A 74-year-old woman, with a prior myocardial infarction and drug-eluting stent placement 2 months before, presented with sudden onset of a severe headache and left shoulder pain, an electrocardiogram without acute changes, and a normal troponin. CCTA showed total occlusion within and at the distal edge of the left anterior descending (LAD) coronary stent with distal arterial filling, presumably by collateral vessels (Fig. 1B); a large area of apical, septal, and anterior akinesia; and transmural hypodensity consistent with a recent infarction (Fig. 1C). Cardiac catheterization confirmed the CTA findings (Fig. 1D).

Case 2

A 61-year-old woman presented with a 24-hour history of chest pain radiating to the back; an electrocardiogram with diffuse, 2-mm, ST-segment elevation in the anterior leads; a troponin of 2.25 ng/mL; and a creatinine phosphokinase level of 1329 U/L. CCTA, ordered primarily to exclude aortic dissection, showed occlusion of the mid LAD artery as well as distal arterial filling, either retrograde by presumed collaterals or antegrade through a subtotal occlusion (Fig. 2A left, right). An anteropapical/septal transmural hypodensity consistent with an acute myocardial infarction was noted (Fig. 2C left).

Figure 1  CTA and catheter-based angiography: acute in-stent thrombosis. Curved multiplanar reconstruction of the left circumflex (A) shows a 50%-75% ostial stenosis (arrow) secondary to calcified and noncalcified plaque (the mean luminal area was <4 mm²). Curved multiplanar reconstruction of the left anterior descending artery (B) shows severe hypodensity in the proximal and mid vessel stents (arrow). The hypodensity at the distal edge of the stent is consistent with total occlusion. The distal vessel fills by collaterals. Axial imaging of the left ventricle (C) shows anteropapical and apical transmural hypoperfusion (arrows) consistent with acute myocardial infarction. Of note, the thickness of the hypodense area is the same as normal myocardium. Coronary angiography (D) confirmed the CTA findings of a totally occluded stent.
Cardiac catheterization showed a totally occluded mid LAD coronary artery without visible collaterals (Fig. 2B). On readmission 1 month later with dyspnea on exertion, CCTA showed an apical ventriculoseptal defect (Fig. 2C). Thinning with subendocardial hypodensity (Fig. 2C right) replaced the previously noted full-thickness hypodensity, consistent with evolution of the infarct.

Figure 2  CCTA and catheter-based angiography: totally occluded left anterior descending (LAD) artery. Curved multiplanar reconstruction shows total occlusion of the mid LAD artery (A left, circle), and the magnified image shows hypodensities consistent with both lipid and thrombus (A right). Angiography confirmed the CTA findings of a totally occluded LAD artery (B left). Stenting restored flow to the LAD artery (B right). (C left) Axial image shows anteroapical myocardial transmural hypoperfusion consistent with acute myocardial infarction. Note the labeled components of the intraarterial hypodensity are presumptive. (C right) Axial images on CTA performed 1 month later show interval development of an aneurysm and a small ventriculoseptal defect in the distal anteroseptal.
Case 3

A 74-year-old man presented with right shoulder pain, a normal electrocardiogram, and normal serum troponin level. CCTA showed subtotal occlusion of a large diagonal branch of the LAD coronary artery (Fig. 3A) accompanied by apical transmural hypoperfusion consistent with acute myocardial infarction (B). Cross-sectional analysis of the straightened multiplanar reconstruction (C) shows areas of negative Hounsfield units adjacent to the lumen, consistent with a thin cap fibroatheroma. The luminal densities are consistent with small amounts of iodinated contrast as noted in subtotal occlusions. Invasive coronary angiography (D) confirmed the subtotally occluded diagonal (arrow).

Figure 3  CCTA and catheter-based angiography: subtotally occluded diagonal. Curved multiplanar reconstruction shows subtotal occlusion of the diagonal branch of the LAD artery (A), accompanied by apical transmural hypoperfusion consistent with acute myocardial infarction (B). Cross-sectional analysis of the straightened multiplanar reconstruction (C) shows areas of negative Hounsfield units adjacent to the lumen, consistent with a thin cap fibroatheroma. The luminal densities are consistent with small amounts of iodinated contrast as noted in subtotal occlusions. Invasive coronary angiography (D) confirmed the subtotally occluded diagonal (arrow).

Case 4

A 54-year-old man presented with the sudden onset of constant, left-sided chest pain, normal electrocardiogram, and normal serum troponin level. CCTA showed an occlusion of the first marginal branch of the left circumflex coronary artery (Fig. 4A arrow) with distal filling, presumably by collaterals, accompanied by transmural anterolateral hypoperfusion consistent with an acute myocardial infarction (Fig. 4B arrows). Cross-sectional analysis (Fig. 4C top) showed a variety of densities in the totally occluded segment (Fig. 4C right). Six hours later, the troponin was 0.48 ng/mL, and the creatinine phosphokinase level was 237 U/L with a 10.5% MB fraction. Cardiac catheterization confirmed the CCTA findings (Fig. 4D).

Case 5

A 43-year-old man presented with nonradiating chest burning for 24 hours, an electrocardiogram with diffuse ST-segment elevation, a troponin level of 16.1 ng/mL, and a creatinine phosphokinase level of 555 U/L with a 9% MB fraction. The presumed diagnosis was myopericarditis. However, CCTA showed multiple branch vessel subtotal occlusions (Fig. 5A), confirmed by invasive angiography (Fig. 5B and C). Cardiac magnetic resonance imaging did not show evidence of myocarditis.
Urgent catheter-based angiography, rather than CCTA, is indicated in patients with acute myocardial infarction. However, the inevitable consequence of CCTA evaluation of chest pain in the ED will be the imaging of patients with acute myocardial infarction with atypical presentations. The 5 patients in this case series presented with presumptive diagnoses ranging from aortic dissection to myopericarditis to nonspecific musculoskeletal pain. The observations from the CCTA examinations in these patients highlight the potential for CCTA to aide in the diagnosis of unrecognized acute myocardial infarction based on diagnostic arterial and myocardial findings.

Arterial findings

A severe stenosis, typically a total or subtotal occlusion, was noted in each case. Distal filling by collaterals was noted in cases 1, 2, and 4, highlighting the ability of CCTA to show the vessel distal to a total occlusion. The presence of collaterals may have mitigated the severity of the presentation, explaining the initial absence of electrocardiographic changes and myocardial biomarker elevations in cases 1 and 4. Identification of a thrombus or ruptured plaque is problematic and cannot be a prerequisite. Although thrombus has a density of 40 HU in vitro, the measured density in vivo may be dramatically altered by adjacent, more dense structures such as calcified plaque or iodinated contrast. Consequently, there are no atherosclerotic lesion characteristics that can reliably distinguish acute from chronic arterial occlusion. Areas of negative Hounsfield units consistent with lipid adjacent to the lumen were noted adjacent to the hypodense subtotally or totally occluded culprit vessel lesion in 2 patients (Fig. 3 and Fig. 4). These are suggestive of thin cap fibroatheromas, which are associated with a higher risk of an acute coronary syndrome. However, corroboration by radiofrequency backscatter intravascular ultrasound, optical coherence tomography, or histology are needed to confirm this diagnosis. The thin cap itself is beyond the resolution of CT. A limitation of the spectrum of findings in this case series is cases showing the absence of significant stenosis, as sometimes noted in the catheterization laboratory in

![Figure 4](image-url)
patients with acute myocardial infarction and spontaneous thrombolysis.

**Myocardial findings**

Myocardial transmural hypodensity consistent with acute microvascular hypoperfusion in the distribution of the affected artery\(^7,8\) is characteristic (cases 1–4) and was always associated with a severe wall motion abnormality, but it may not be present in the setting of small infarcts (case 5). The transmural hypodensity in the setting of collateral filling suggests the presence of insufficient collateral flow. Over time, thinning and, rarely, a ventricular septal defect occur (case 2). In the experience at our center of >5000 CCTAs, transmural hypodensity has been clinically identified in only the 4 patients described here and in 1 other patient with an acute myocardial infarction. If present, it may be pathognomonic for an acute myocardial infarction, but it is not a prerequisite.

**Clinical implications**

When a diagnosis of acute myocardial infarction is clinically apparent, CCTA is contraindicated, and rapid intervention (“door to balloon time”) is of the essence. However, in atypical presentations of acute myocardial infarction, CCTA may provide the first clues to the correct diagnosis; thus, recognition of CCTA patterns of acute myocardial is of increasing importance as use of CCTA in the ED increases. As a corollary, interpretation of chest pain studies of patients immediately after their completion, no matter how stable the patients may appear, is necessary for rapid therapeutic implementation.

**References**


