

Acute myocardial infarction found by multi-detector computed tomography ordered for suspected aortic dissection

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Urgent requests for contrast computed tomographic scans of the thorax are often performed for suspected aortic dissections and pulmonary embolisms, both of which have clinical features similar to those seen in acute myocardial infarction. We present two cases with computed tomographic scans showing decreased myocardial enhancement, suggestive of acute myocardial infarction, but no evidence of aortic dissection. Acute myocardial infarction was subsequently confirmed by coronary angiography.

Introduction

Urgent requests for contrast computed tomographic (CT) scans of the thorax are performed for suspected aortic dissections and pulmonary emboli, both of which have clinical features similar to those seen in acute myocardial infarction (MI). Thoracic CT scans are frequently requested for patients presenting with sudden onset of chest discomfort. We have encountered several patients with acute MI who were evaluated with contrast CT for other indications. Computed tomographic findings showed an area of decreased myocardial enhancement in the distribution of particular coronary arteries, findings compatible with infarction. Coronary angiography was performed in these patients to confirm the diagnosis.

Case reports

Case 1

A non-smoking 83-year-old man with good past health was admitted for sudden onset of chest discomfort. An electrocardiogram (ECG) showed tachycardia with subtle ST depression in the V4-6 leads. An urgent contrast CT (Aquillon 16, Toshiba, Otawara-Shi, Tochigi, Japan) of the thorax was requested to rule out dissection. No evidence of dissection was found but an area of decreased myocardial enhancement was noted in the area supplied by the left anterior descending artery (LAD), with no evidence of myocardial thinning (Fig 1). The creatinine kinase level was raised but the troponin level was normal on admission. After excluding aortic dissection, the patient was treated as having ischaemic atrial fibrillation with cardiogenic shock. He was put on amiodarone, aspirin, and low-molecular-weight heparin. Three weeks after the CT scan, a coronary angiogram showed 80% blockage of the LAD (Fig 1).

Case 2

A 63-year-old male smoker with a family history of ischaemic heart disease presented with back pain and shortness of breath. An ECG showed ST elevation over the inferior leads and ST depression over V4-5. His troponin and creatinine kinase levels were raised. An urgent contrast CT of the thorax (Aquillon 16) was performed to rule out aortic dissection before starting a streptokinase infusion. No evidence of dissection was detected. An area of decreased myocardial enhancement was noted in the area supplied by the LAD with no evidence of myocardial thinning (Fig 2). An urgent coronary angiogram performed later the same day showed a 90% stenosis of the right circumflex artery and the LAD, and 50% blocking of the first diagonal artery of the LAD (Fig 2). A percutaneous coronary angioplasty was performed to manage his coronary artery disease.

Discussion

These cases demonstrate that acute MI is detectable using a contrast multi-detector CT (MDCT) of the thorax, even when using a protocol designed for a different purpose. Multi-

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對疑患主動脈夾層病者進行多探測器電腦X線斷層攝影術而發現患上急性心肌梗塞

主動脈夾層和肺栓塞的臨床特徵與急性心肌梗塞相似，對懷疑患上這兩種疾病的病者，通常須馬上接受胸廓相位視度電腦X線斷層攝影術檢查。本文報告的兩個案例經電腦X線斷層攝影術掃描發現心肌強化程度下降，反映出病人所患可能是急性心肌梗塞，但沒有證據指向是主動脈夾層。之後經冠狀動脈成像，確診病人患上急性心肌梗塞。

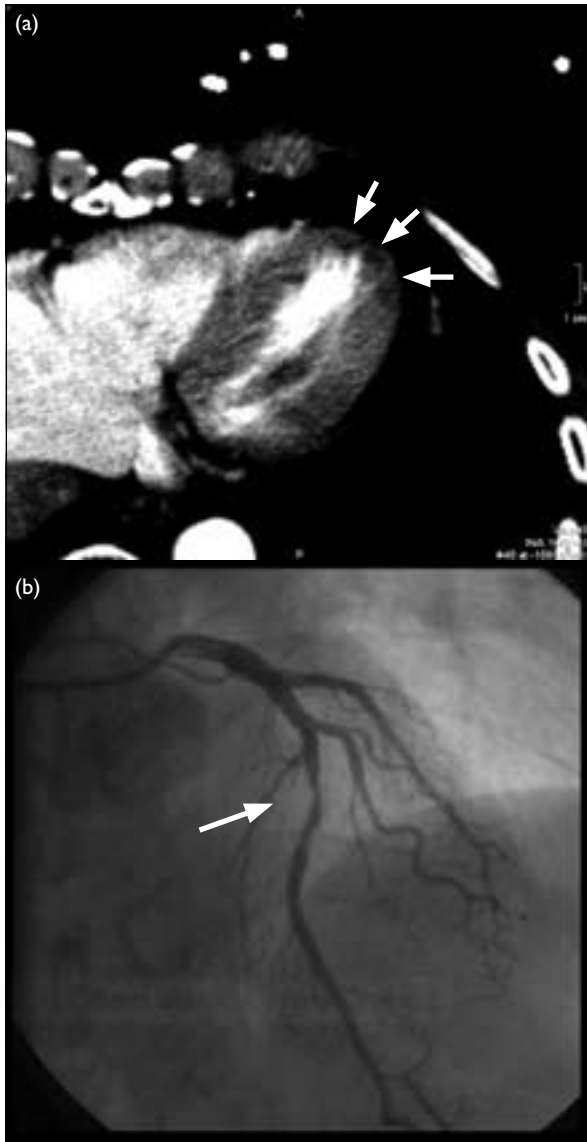


FIG 1. (a) Computed tomogram showing an area of decreased myocardial enhancement in the left anterior descending artery territory (arrows). (b) Corresponding coronary angiogram showing stenosis in the left anterior descending artery (arrow)

detector CT has a reported 83% sensitivity and 91% specificity for the detection of MIs.¹ Acute MI causes a myocardial enhancement defect in the distribution

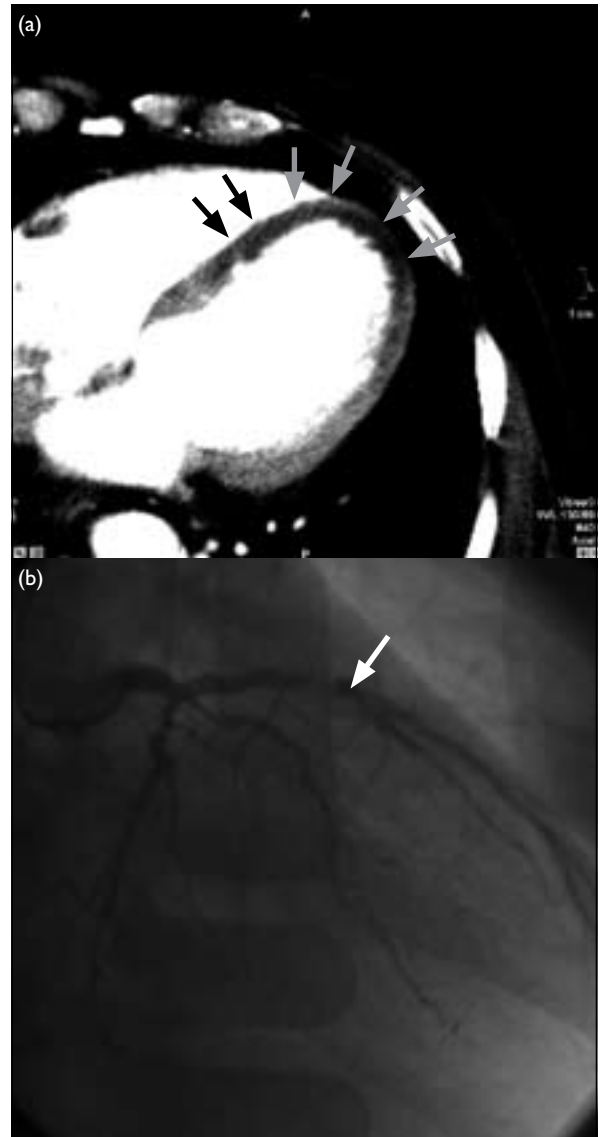


FIG 2. (a) Computed tomogram showing an area of decreased myocardial enhancement in the cardiac apex and ventricular septum suggestive of infarction (arrows). (b) Corresponding coronary angiogram showing stenosis in the left anterior descending artery (arrow)

of the occluded coronary artery. This diminished contrast medium uptake, attributed to hypoperfusion, usually involves the endocardium and may extend transmurally to the epicardium. Ischaemic changes in the myocardium after occlusion of the coronary artery consist of disruption to cell membrane function and integrity along with increased permeability of small vessel walls. The area of low attenuation seen on CT primarily reflects myocardial oedema. Necrotic myocardium is later replaced by fibrous and/or fatty tissue.² Animal studies done on porcine models have demonstrated that MDCT can detect acute MI via a lower measured CT attenuation in infarct areas relative to normal reference areas.³ As CT scanning times have diminished, especially when using MDCT,

the quality and timing of contrast enhancement have both improved.⁴ There are a number of case reports describing detection of MI using MDCT.^{5,6} A perfusion defect, however, is not specific for infarction because similar findings can occur in cases with severe local ischaemia (but no infarction) or other cardiac disease causing non-homogenous perfusion, such as hypertrophic cardiomyopathy. Independent variables such as the contrast injection protocol and the cardiac output can also affect myocardial contrast enhancement. Therefore, an absolute Hounsfield unit measurement of myocardium to detect the presence of infarcted tissue is difficult. Instead, a relative change in enhancement is a more practical measure. Indirect findings such as thinning of the left ventricle wall, associated with healing after MI, is another useful sign because it has been shown that at the infarction site the wall thickness decreases significantly over time.²

An urgent CT of the thorax is frequently requested for suspected cases of aortic dissection or pulmonary embolism which may have atypical clinical features overlapping with those of acute MI.⁷ One should therefore also look out for CT evidence of acute MI, especially, as in our patients, where there is no evidence of aortic dissection. Our cases had their thoracic CT scans performed with aortic dissection

protocols; the post-contrast scan was done in phase with the thoracic aorta without ECG gating and no thin slices of the heart were included. A pulsation artefact can affect the image quality when evaluating the myocardium and coronary arteries. Therefore, further evaluation with a cardiac CT protocol is suggested. Intravenous contrast-enhanced CT can readily identify the cardiac chambers, grossly assess their volumes and, using ECG gating, can assess left ventricular wall motion and thickening.⁸ Therefore, in a patient with acute chest pain, a dedicated cardiac MDCT with ECG gating should be performed, after non-cardiac causes have been excluded.

In conclusion, we have shown that acute MI is detectable using a contrast-enhanced CT of the thorax. It is indicated by decreased myocardial enhancement in the distribution of a specific coronary artery, related to the clinical infarct. To achieve better image quality, a dedicated cardiac MDCT with ECG gating should be performed. Multi-detector CT can provide comprehensive imaging of MI, offering a combined morphological and angiographic assessment. A fast and non-invasive examination with MDCT can provide information that cannot be obtained via standard clinical evaluation. This could substantially improve the clinical care of patients with acute chest pain.

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