Early Detection of Acute Myocardial Infarction by Routine Computed Tomography of the Chest: a case report

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Evaluation of chest pain in the emergency department (ED) is a formidable challenge. It is established that multidetector computed tomography (MDCT) can detect the changes of myocardial perfusion after acute myocardial infarction (MI). However, there were few studies about MDCT changes before ECG and cardiac biomarkers confirmed acute myocardial infarction. We report a case presented to the ED with sharp chest pain. MDCT performed for suspected aortic dissection showed decrease myocardial enhancement, suggestive of acute MI before ECG changes and rises of cardiac enzymes, but no evidence of aortic dissection. Acute myocardial infarction was subsequently confirmed by both the ECG changes and increased levels of cardiac biomarkers.

Chest pain, a common presenting complaint of patients in the emergent department (ED) is diagnostically challenging since the etiologies include both benign and catastrophic causes. The initial clinical workup should focus on the immediate recognition and exclude acute life-threatening conditions, such as acute ischemic heart disease, aortic dissection, and pulmonary embolism. Initial normal electrocardiograms (ECG) and cardiac markers cannot exclude myocardial infarction in an ED patient with chest pain, and additional diagnostic tests may be required to rule out other life-threatening diseases.

Contrast-enhanced multidetector computed tomography (MDCT) is widely accepted and routinely used as a primary emergent tool to assess the presence of pulmonary embolism or aortic dissection [1, 2]. We report an acute MI case depicted by urgent 4-detector non-ECG-gated MDCT which was requested initially with clinical suspicion of aortic dissection.

CASE REPORT

A 71-year-old male patient presented to the ED with an abrupt onset of first-time chest pain during manual labor on a farm. The patient’s chest pain was sharp, radiated towards the back, and was associated with dizziness, cold sweating, and blurred vision. The pain had lasted for 30 minutes, and the patient arrived in the ED one and a half hours after the onset. He denied past histories of previous surgeries, medications, and drug allergies.

The patient’s vital signs on initial clinical presentation included a temperature of 36°C, a heart rate of 44 beats / minute, a respiratory rate of 16 breaths / minute, and a blood pressure of 74/56 mmHg. The physical examinations revealed no abnormal findings in the neurological systems and abdomen. Lungs were normal on auscultation with...
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no cardiac murmurs or accessory heart sounds. The 12-lead ECG revealed the absence of P-waves, narrow QRS complexes (0.74 ms), and no marked ST segment elevation (Fig. 1). The chest radiograph showed no evidence of cardiomegaly or focal lung lesions. Routine laboratory studies of hemograms, electrolytes, liver and renal function tests were normal. An initial panel of cardiac enzymes revealed normal results, with CK-MB value of 5.6 ng/mL (normal 0.6-6.3 ng/mL) and troponin-I value of 0.025 ng/mL (normal <0.5 ng/mL). The patient was placed on a cardiac monitor in the ED. Initial treatments included 2L/minute oxygen administration by nasal cannulation, intravenous fluid supplementation and then intravenous morphine (3 mg) for pain relief, atropine and dopamine infusion for the bradycardia.

Due to clinical suspicion of aortic dissection, a thoracic computed tomography (CT) scan with contrast was performed. The thoracic CT scan did not reveal any evidence of aortic dissection, but demonstrated decreased myocardial enhancement at the inferior wall, the inferior-septal wall and posterior papillary muscle of the left ventricle without ventricular wall thinning, suggesting acute myocardial infarction (Fig. 2). Serial followed ECGs revealed hyperacute T wave changes (Fig. 3a) and evolutional ST segment elevations (Fig. 3b) in leads III and aVF three and four hours later respectively. Besides, right ventricular infarction was also demonstrated in the right side ECG. The follow-up cardiac makers four hours apart were positive for myocardial infarction with CK-MB value of 27.5 ng/mL (normal 0.6-6.3 ng/mL) and troponin-I value of 0.621 ng/mL (normal <0.5 ng/mL).

With the impression of acute inferior wall and right ventricle ST segment elevation myocardial infarction (STEMI), a coronary angiography was performed. It revealed a completely occlued proximal right coronary artery with massive thrombi formation (Fig. 4a), as well as a 69% stenosis at the middle portion of left anterior descending artery, an 80% stenosis of the first diagonal (D1) vessel,
Figure 3. a. ECG revealed hyperacute T waves in lead III and aVF 3 hours after arrival in the emergency department. b. ECG revealed evolution changes with ST segment elevations in lead III and aVF four hours after arrival in the emergency department.

Figure 4. Coronary angiography revealed a completely occluded proximal right coronary artery (a), which was initially balloon-dilated and followed by 2.75-mm stent placement (b).
and a 67% stenosis of the middle circumflex artery. Besides, balloon-dilation and a 2.75-mm stent placed in the right coronary arterial lesion were performed for primary percutaneous coronary interventions (Fig. 4b). The patient was discharged one week later in a stable condition.

**DISCUSSION**

The assessment of chest pain in the ED remains a significant challenge because of extensive etiologies ranging from benign to potentially lethal. Classic cardiac and non-cardiac etiologies can be differentiated by conventional diagnostic methods, including detailed histories, specific physical findings, ECG, and chest radiographs. Sometimes, the definitive diagnosis frequently requires additional imaging-based testing, particularly in cases of inconclusive chest pain. We reported an acute MI case detected early by urgent 4-detector non-ECG-gated MDCT which was ordered originally for suspected aortic dissection.

The acute MI was defined by a clinical history of ischemic type chest pain, a rise in serum cardiac biomarkers, and typical ST segment changes at initial ECG. However, acute MI in very early stages is prone to several diagnostic challenges. The sensitivity of 12-lead ECG in identification of ischemia can be as low as 50%, and the creatine kinase-MB fraction and troponin levels usually do not rise above normal ranges until more than four hours after the onset of MI [3, 4]. Moreover, aortic dissection may or may not occlude the coronary arteries, presenting with similar clinical features as acute MI, is one of the key differential diagnoses of chest pain in the ED.

In the present case, it was rational to arrange urgent contrast enhanced thoracic MDCT scan for suspected aortic dissections as the presentation with sharp chest pain and initial negative findings of ECG and cardiac biomarkers. The thoracic CT showed no evidence of aortic dissection but decreased myocardial enhancement in inferior wall incidentally, suggestive of acute myocardial infarction. The diagnosis was subsequently confirmed by ECG changes and elevations of cardiac biomarkers.

ECG-gated MDCT can identify infarcted myocardial tissue and differentiate recent and chronic MI based on myocardial wall thickness and CT attenuation. In acute MI, early-phase ECG-gated MDCT may identify myocardial perfusion defects with 94% sensitivity as mentioned by Ko SM. et al [5]. In the chronic infarct stage, the wall thickness was significantly thinner and CT attenuation value was lower than those in the recent infarct stage (< 7 days) [6-8]. Due to substantially improved temporal resolution of CT scanners, ECG-gated 64-MDCT angiography of the entire chest has been available in the differential diagnosis of acute chest pain. It revealed very good image quality for accurate diagnostic and therapeutic decisions [9]. However, for either imaging myocardium or coronary arteries, the optimal morphology of contrast-enhanced MDCT scans requires cooperations of patients with breath holding, a thorough scanning of the entire heart, retrospective ECG gating and reconstruction in the motionless phases.

Though 64-MDCT systems are the current state of the art for cardiac CT as high-resolution imaging of the myocardium and coronary arteries, it is not popularly applied in the ED in Taiwan. In this case, the non-ECG-gated 4-slice MDCT scan was performed for clinically suspected aortic dissection. While without both the ECG gating and retrospective reconstruction, the image quality of the present case is fair enough to identify the thickness and decreased enhancement of myocardium, suggestive of acute myocardial infarction. Gosalia et al. had showed that acute MI is detectable on non-ECG-gated contrast-enhanced thoracic CT as decreased left ventricular enhancement in a specific coronary artery territory, related to the clinical infarct. 83% of AMI patients had a focal decrease in left ventricular myocardial enhancement of 20 Hounsfield units or more in the coronary artery distribution, and as much as 50% of patients had visually detected decreased myocardial enhancement [10]. However, the true sensitivity and specificity of non-ECG-gated MDCT on acute MI is not known, because CT is rarely performed on patients with acute MI.

Patients with inferior wall MI are excluded in Gosalia et al. study, because the inferior wall is usually poorly imaged in the axial plane of MDCT [10]. Motion artifacts at the heart in non-ECG-gated MDCT may make cardiac images interpretation difficult. Both the inferior wall MI and motion artifacts were challenges for image interpretation in the present case. However, decrease myocardial enhancement at the inferior wall and the inferior-septal wall compatible with right coronary artery distribution was the important clue to differentiate myocardial infarction from motion artifacts. Multiplanar reformations in MDCT can be performed with minimal artifact, which may enable us to evaluate the inferior wall of the heart [10]. The
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The occurrence of acute MI after blunt chest trauma is a rare complication, and is often missed and masked by multiple traumas. Traumatic MI could be caused by coronary artery dissection or thrombosis and potentially fatal [11]. Chest contusions can cause a rise in cardiac biomarkers and electrocardiographic abnormalities making it difficult to differentiate a myocardial contusion from a posttraumatic myocardial infarction. The ECG-gated MDCT performed for patients after blunt chest trauma could depict intimal flap or coronary artery dissection which causes posttraumatic MI. In addition, the MDCT could show the decreased myocardial enhancement, corresponding to infarcted area of the myocardium [12]. In the patients with blunt chest trauma, MDCT is useful in evaluating traumatic MI, which is potentially fatal and often an unrecognized complication.

Although the non-ECG-gated MDCT could provide clues suggestive of acute MI, the diagnosis of acute MI still depends on clinical history, serum cardiac biomarkers, and ECG changes. The present case report underscores we should not ignore the important information of myocardial perfusion by MDCT even without ECG-gating, and miss the morphologic consequences of acute myocardial infarction in clinical practice, particularly in cases of inconclusive chest pain.

REFERENCES
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早期檢測AMI的胸部CT

胸部電腦斷層早期發現急性心肌梗塞：病例報告

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胸痛在急診是一個重要的主訴，具有臨床評估上的挑戰性。電腦斷層已經被證實可以發現
心肌梗塞後心肌灌流的變化，然而針對心肌電腦斷層影像變化於心電圖與心肌酵素證實心肌梗
塞前的報告卻很少。我們報告一位七十一歲男性因尖銳性胸痛來急診就診。因為懷疑主動脈
剝離而接受胸部電腦斷層檢查。電腦斷層證實沒有主動脈剝離，但在心電圖與心肌酵素變化前
已發現心肌梗塞的心肌顯影缺損。病人後來的心電圖與心肌酵素證實病人有急性心肌梗塞。