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【 CASE REPORT 】

Potential Utility of Non-gated Enhanced Computed Tomography for an Early Diagnosis of Myocardial Infarctions

Riku Arai, Daisuke Fukamachi, Yasunari Ebuchi, Naotaka Akutsu and Yasuo Okumura

Abstract:
The diagnosis of acute myocardial infarctions (MIs) is challenging when no significant ischemic ST-segment changes are noted on a 12-lead electrocardiogram (ECG). We herein report two patients suffering from chest pain in whom non-gated enhanced computed tomography (CT) images were used to rule out aortic dissection and pulmonary embolism, aiding in the early diagnosis of an acute MI. Subsequently, urgent revascularization was successfully performed in these patients. In non-gated enhanced CT imaging, the infarcted myocardium is initially visible as a focal myocardial perfusion defect.

Key words: chest pain, diagnosis, myocardial infarction, non-gated enhanced computed tomography

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Introduction

A careful diagnosis is important for patients suffering from chest pain, as we must rule out lethal chest pain, such as that due to an acute myocardial infarction (MI), aortic dissection (1), pulmonary embolism (2), or tension pneumothorax (3). However, the diagnosis of an acute MI is sometimes challenging when a 12-lead electrocardiogram (ECG) does not show any significant ST-segment changes, especially when the culprit vessel is the left circumflex artery (4, 5) and/or the side branch of a main coronary artery (6). In such cases, a delayed diagnosis leads to increased heart damage.

We herein report two patients with acute MIs in whom non-gated enhanced computed tomography (CT) images were used for the differential diagnosis of an aortic dissection and pulmonary embolism, helping in the early diagnosis and revascularization.

Case Reports

Case 1

A 42-year-old man without any major medical history experienced sudden persistent chest pain. A synthesized 18-lead ECG during the initial examination showed no significant ST-segment changes except for ST-depression only in lead III (Fig. 1). Transthoracic echocardiography revealed a normal left ventricular function without asynergy, D-shape, echo-free space, or valvular disease. Chest X-ray revealed no notable findings, including pneumothorax. The laboratory data showed that the creatinine kinase (CK), troponin I, and D-dimer levels were within normal limits, although the low-density lipoprotein (LDL) cholesterol level was high during the initial examination. To rule out aortic dissection and pulmonary embolism, non-gated enhanced CT imaging was conducted but yielded no signs of those diseases. However, there was a focal myocardial perfusion defect in the left myocardium along the posterolateral region and posterior papillary muscle, suggestive of a focal coronary vessel occlusion (Fig. 2). Because of persistent chest pain in addition to several coronary risk factors, including a smoking habit

Division of Cardiology, Department of Medicine, Nihon University School of Medicine, Japan
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Correspondence to Dr. Riku Arai, riku.arai@icloud.com

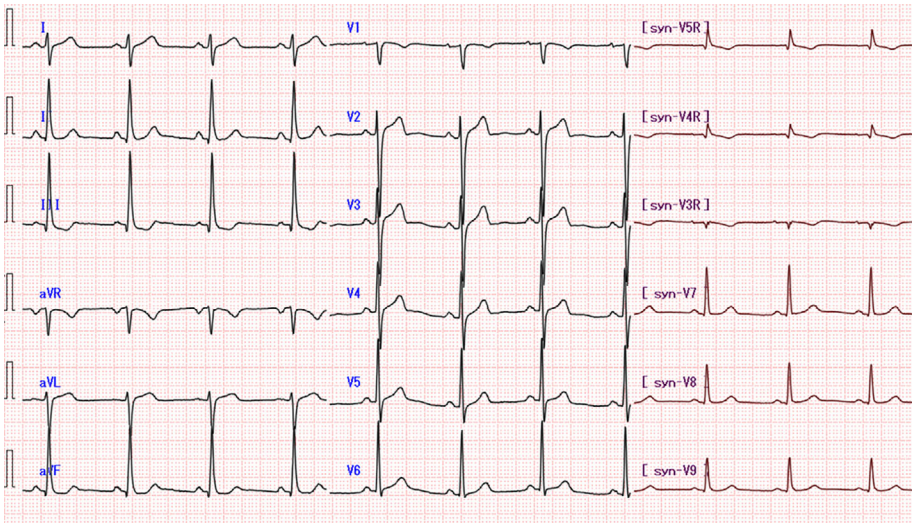

Figure 1. **Synthesized 18-lead electrocardiogram during the initial examination in case 1. No significant ST-segment changes except for ST-depression only in lead III were noted.**

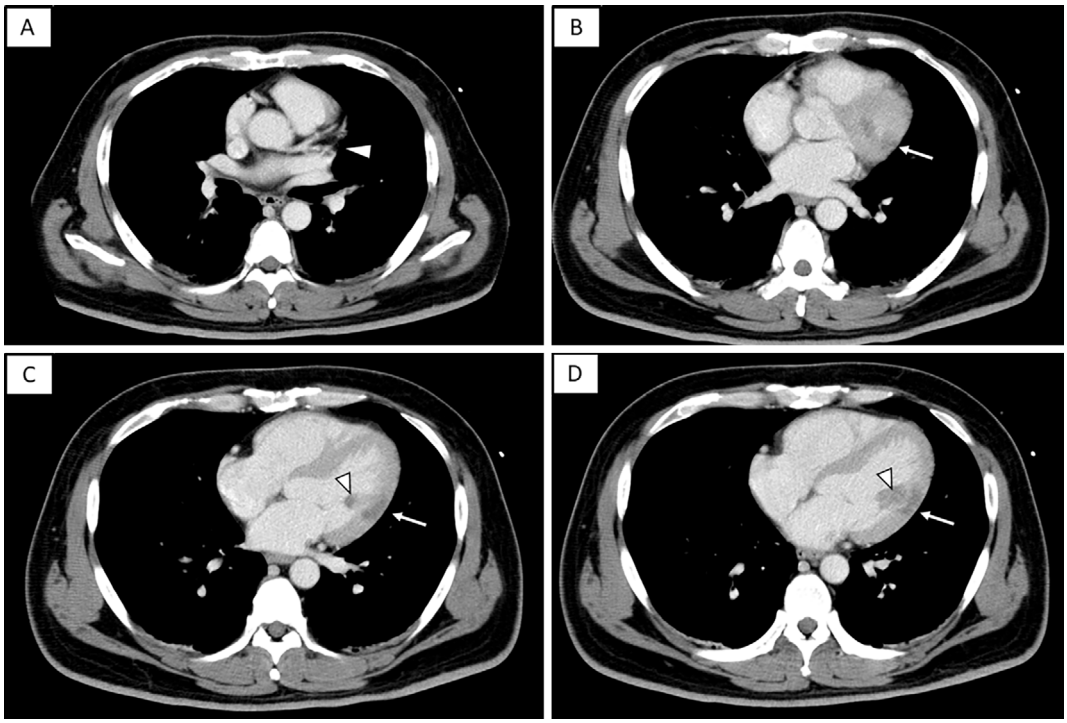

Figure 2. **Non-gated enhanced computed tomography (CT) during the initial examination in case 1. Non-gated enhanced CT imaging showed that the focal myocardial perfusion defect was located in the posterolateral region of the myocardium (B, C, D: arrow) and the posterior papillary muscle (C, D: arrow), along where the left circumflex artery travels (A: arrow).**

and high LDL level, coronary angiography (CAG) was performed, revealing a subtotal occlusion of the high lateral branch of the left circumflex artery (Fig. 3A), which completely matched with the damaged myocardium in the non-gated enhanced CT imaging. The culprit vessel was successfully restored with percutaneous coronary intervention (Fig. 3B). Thereafter, the CK level peaked at 955 IU/L after revascularization without any complications.

Case 2

We subsequently experienced another similar case in a 48-year-old man with hypertension and a smoking habit who experienced the sudden onset of chest pain. A synthesized 18-lead ECG during the initial examination showed no significant ST-segment changes except for T-wave flattening in leads III and aVF (Fig. 4). Transthoracic echocardiography revealed a normal left ventricular function without any asynergy, D-shape, echo-free space, or valvular disease.

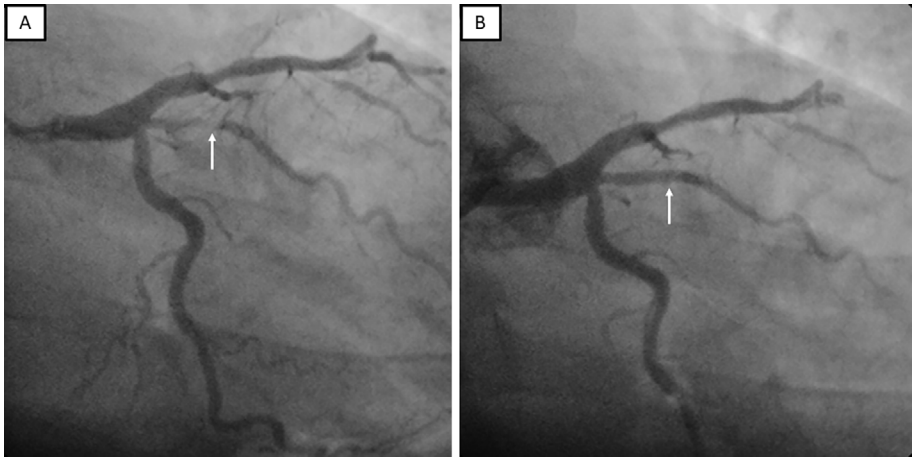

Figure 3. **Coronary angiography and percutaneous coronary intervention in case 1. Coronary angiography showed subtotal occlusion in a high lateral branch (branch of the left circumflex artery) (A: arrow). Subsequently, successful revascularization was performed with a drug-eluting stent (B: arrow).**

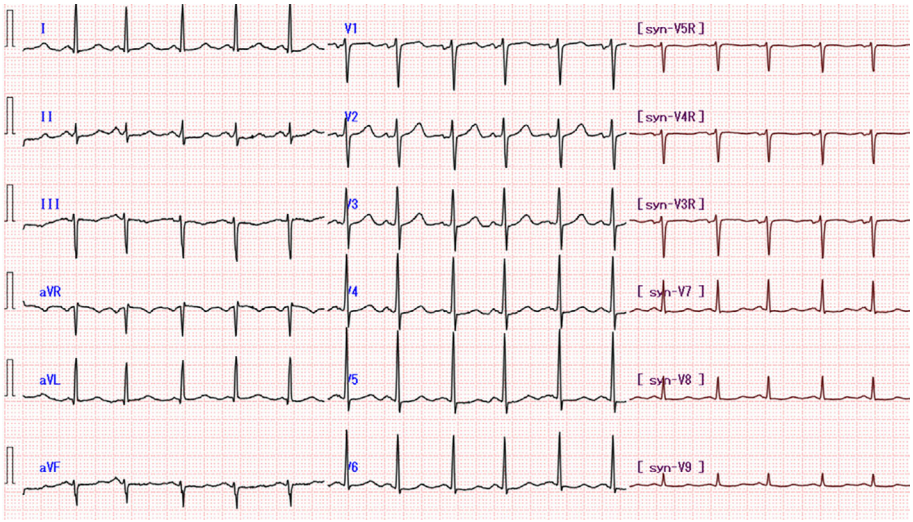

Figure 4. **Synthesized 18-lead electrocardiogram during the initial examination in case 2. T-wave flattening in leads III and aVF was noted.**

Chest X-ray revealed no notable findings, including pneumothorax. Non-gated enhanced CT also showed a focal myocardial perfusion defect in his left myocardium along the lateral region, which suggested a focal coronary vessel occlusion (Fig. 5). Based on the experience obtained in case 1, we highly suspected this case to be one of MI and therefore carried out urgent CAG before all of the laboratory data results had been obtained. CAG revealed total occlusion of the first diagonal branch of the left anterior descending artery (Fig. 6A), which matched the damaged myocardium from the non-gated enhanced CT image. The culprit vessel was successfully restored with percutaneous coronary intervention (Fig. 6B). Thereafter, we obtained all of the laboratory data results, but they showed that the CK, troponin I, and D-dimer levels were within normal limits. The CK level peaked at 426 IU/L after revascularization without any complications. He was treated with early revascularization due to the early diagnosis based on non-gated enhanced CT images.

Informed consent was obtained from both patients for the publication of this study.

Discussion

The guidelines by the Japanese Circulation Society suggest ECG-gated coronary CT angiography as a “triple role out” [JCS Joint Working Group (JCS 2009)] in patients with acute chest pain and a low-to-intermediate risk (Class 2b). However, ECG-gated CT imaging is not available in the emergency room (ER) in most hospitals. Furthermore, it has limited utility in cases of arrhythmias, such as atrial fibrillation and extrasystoles, or tachycardias that often require beta-blockers to reduce the heart rate for ECG-gated imaging. Non-gated CT imaging has potential advantages over ECG-gated CT imaging in several aspects. First, non-gated CT imaging is often performed in patients with chest pain to

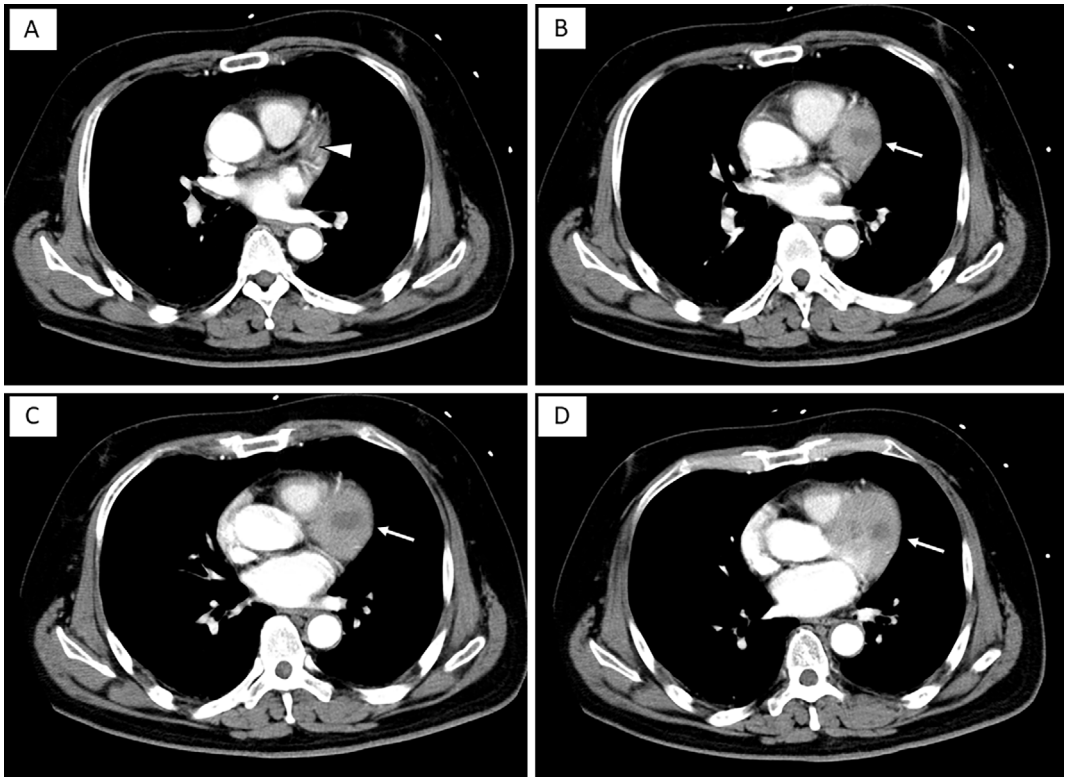

Figure 5. **Non-gated enhanced computed tomography (CT) during the initial examination in case 2. Non-gated enhanced CT imaging showed that the focal myocardial perfusion defect was located in the lateral region of the myocardium (B, C, D: arrow), along where the diagonal branch (branch of the left anterior descending artery) travels (A: arrow).**

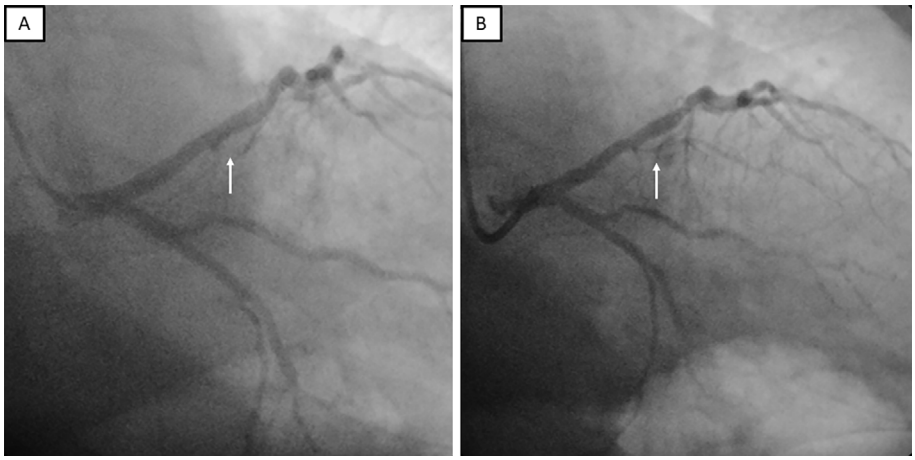

Figure 6. **Coronary angiography and percutaneous coronary intervention in case 2. Coronary angiography showed total occlusion in the first diagonal branch (branch of the left anterior descending artery) (A: arrow). Subsequently, successful revascularization was performed with a drug-eluting balloon (B: arrow).**

differentiate acute coronary syndrome (ACS), aortic dissection, and pulmonary embolism. Second, this approach can be performed in most hospitals, even in the ER, although we must consider the possibility of motion artifacts with non-gated CT imaging.

When the enhanced CT findings are negative for aortic dissection or pulmonary embolism, if the pre-test probability does not indicate a low likelihood of acute ischemia, patients should undergo an early risk stratification that focuses on angina symptoms, physical and ECG findings, and biomarkers of cardiac injury, and a temporal follow-up should be carefully performed. Through this process, when symptoms suggestive of angina, significant ischemic ECG changes, and/or biomarker elevations occur, we can eventually arrive at a diagnosis of ACS. When the risk stratification is high or intermediate, CAG can be performed, accord-

ing to the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines (7) for patients with non-ST elevation ACS (NSTE-ACS).

We experienced two similar cases in which enhanced CT was useful for diagnosing ACS of culprit vessels involving a high lateral branch and the first diagonal branch, both of which are unlikely ECG changes. In both cases, we were unable to diagnose ACS immediately due to the lack of any ECG changes or a troponin level elevation during the initial visit. Therefore, we ruled out aortic dissection and pulmonary embolism in such cases by non-gated CT imaging. After excluding these diseases in the present two cases, the risk stratification of ACS was performed. Case 1 had a high risk because of persistent chest pain, while case 2 had a low risk because the Thrombolysis in Myocardial Ischemia (TIMI) risk score, which is often used for risk stratification of NSTE-ACS, was 2. However, for case 2, we recalled the potential diagnostic usefulness of CT imaging based on our experience with case 1, and early CAG was performed in a prospective fashion immediately after enhanced CT imaging (before all of the laboratory data had been obtained), which helped minimize the myocardial damage.

Non-gated CT imaging would be effective in patients with cardiovascular risk who present with acute chest pain without any significant ischemic ECG changes or a troponin level elevation and therefore cannot be diagnosed with ACS during the initial examination. Given the findings in our two cases, non-gated CT imaging can potentially be used to evaluate the myocardial damage from focal myocardial perfusion defects while carefully differentiating aortic dissection and pulmonary embolism, leading to an early diagnosis of ACS before the progression of myocardial damage in cases conventionally evaluated with follow-up ECGs and troponin level measurements several hours later. An early diagnosis of ACS with a myocardial perfusion defect during the initial examination might reduce the myocardial damage by allowing for early revascularization (8, 9).

Author's disclosure of potential Conflicts of Interest (COI).
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