

ACUTE MYOCARDIAL INFARCTION SUSPECTED ON CONTRAST-ENHANCED COMPUTED TOMOGRAPHY : A CASE REPORT

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Abstract

A 69-year-old man with severe chest pain was admitted to our hospital. No evidence of acute myocardial infarction (AMI) was seen on laboratory data, electrocardiogram (ECG), and ultrasonic echocardiography (UCG). Contrast-enhanced computed tomography (CECT) was performed to rule out aortic dissection and pulmonary embolism (PE). CECT images showed the hypo-enhancement of posterior wall in the left ventricle. AMI was suspected based on the symptoms and CECT imaging. Coronary angiography (CAG) was performed, and the left coronary angiogram showed total occlusion of segment 13. Percutaneous cardiac intervention (PCI) was immediately performed, and the patient was discharged 15 days after admission with no complications. It is important to check the myocardial enhancement when we interpret CT images of patients with chest pain.

Keywords : acute myocardial infarction, contrast-enhanced computed tomography (CECT)

Introduction

Since AMI is a fatal disease, early diagnosis and immediate PCI are necessary. Though it is well known that AMI can be detected based on symptoms, laboratory data, the ECG findings, and ultrasonic echocardiographic findings (UCG), it can sometimes be difficult to diagnose. A case of AMI that was suspected on CECT is presented.

Case Report

A 69-year-old man was admitted to our emergency and

critical care center with severe anterior chest pain. Pre-hospital delay time was within 2 hours. He had a history of diabetes mellitus, dyslipidemia, hypertension, and an old cerebral infarction. He was taking aspirin, candesartan, amlodipine, simvastatin, and voglibose. He had smoked 30 cigarettes a day for 30 years and drank about 1.8 L per day of Japanese sake. On admission, his blood pressure was 221/120 mmHg (no difference between the left and right arms), heart rate was 88 beats per minute, body temperature was 36.7°C, and respiratory rate was 18 per minute, with SpO₂ of 96% (room air). On examination, he was in obvious distress and diaphoretic. On chest auscultation, no crackles or murmurs were heard. Table 1 shows the patient's laboratory data on admission. White blood cells (WBC), lactate dehydrogenase isozyme (LDH), aspartate transaminase (AST), creatine kinase (CK), creatine kinase MB (CK-MB) and C-reactive protein (CRP) were 11,000/ μ L, 247 IU/L, 48 IU/L, 264 IU/L, 34 IU/L and 0.34 mg/dL, respective-

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Acute Myocardial Infarction on CT

Table 1. Laboratory data on admission

BUN	12.2 mg/dL	ALT	34 IU/L	WBC	11,000/ μ L
Cre	0.56 mg/dL	CK	264 IU/L	RBC	560 104/ μ L
eGFR	110.9 mL/min	CK-MB	34 IU/L	Hb	17.4 g/dL
Na	138 mEq/L	CRP	0.34 mg/dL	Hct	51.20%
K	4.4 mEq/L			Plt	21.8 *10 ⁴ / μ L
Cl	97 mEq/L				
T-Bil	0.9 mg/dL			APTT	31.0 sec
LDH	247 IU/L			PT-INR	1.06
γ -GTP	118 IU/L			D-dimer	0.29 μ g/mL
AST	48 IU/L			FDP	1.4 μ g/mL

BUN : blood urea nitrogen, Cre : creatinine, eGFR : estimated glomerular filtration rate, T-Bil : total bilirubin, LDH : lactic dehydrogenase, γ -GTP : γ -glutamyltransferase, AST : aspartate aminotransferase, ALT : alanine aminotransferase, CK : creatine kinase, CK-MB : creatine kinase MB, CRP : c-reactive protein, WBC : white blood cells, RBC : red blood cells, Hb : hemoglobin, hct : hematocrit, Plt : platelets, APTT : activated partial thromboplastin time, PT-INR : prothrombin time, international normalized ratio, FDP : fibrinogen degradation products

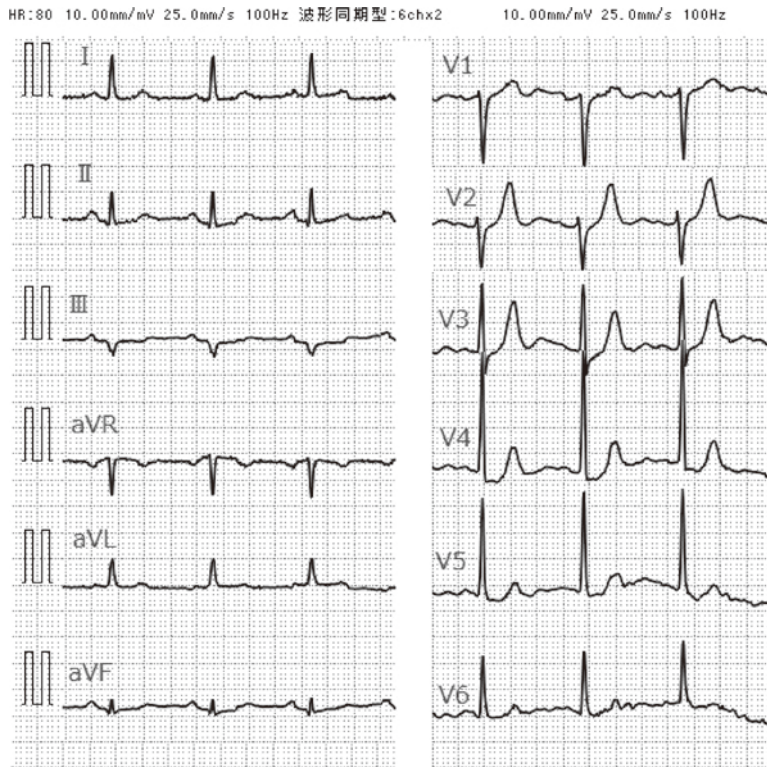


Fig. 1. ECG on admission shows regular sinus rhythm and no ST-segment elevation. Mild ST segment depression is seen in II and V2-6, and a high T is seen in V2-V4, but there are no changes compared with the previous ECG before this episode. It is difficult to diagnose AMI.

ly. Heart type fatty acid-binding protein (H-FABP) was negative. The ECG on admission showed a regular sinus rhythm with no ST segment elevation. Mild ST segment depression was seen in II and V2-6, but there were no changes compared with a previous ECG (Fig. 1). The admission chest X-ray showed no abnormalities. Ultrasonic echocardiography performed by a cardiologist showed poor images, and, thus, it was difficult to detect asynergy. Though there was no evidence of acute coronary syndrome (ACS), given his severe symptoms, aortic dissection and PE were considered in the differential diagnosis, and CECT was performed. CECT was not ECG-gated, and nonionic iodinated contrast medium was administered intravenously. There was no evidence of aortic dissection or PE. However, as shown in Fig. 2, the posterolateral wall of the left ventricle showed hypoenhancement. Posterolateral wall AMI was suspected based on his symptoms and CECT images, and CAG was performed. The left coronary angiogram showed total occlusion of segment 13 (Fig. 3). PCI was performed immediately, and the occlusion was improved (Fig. 4). Insertion of a guide wire into segment 15 was attempted, but it was difficult, because segment 15 was thin as segment 4PD was dominant. The patient



Fig. 2. CECT image on admission shows a hypoenhancing area of the posterolateral left ventricular wall (arrows).

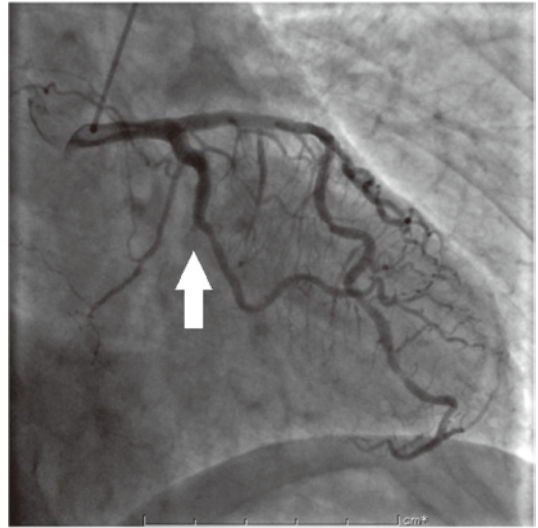


Fig. 3. Left coronary angiogram shows total occlusion of left circumflex artery segment 13 (arrow).

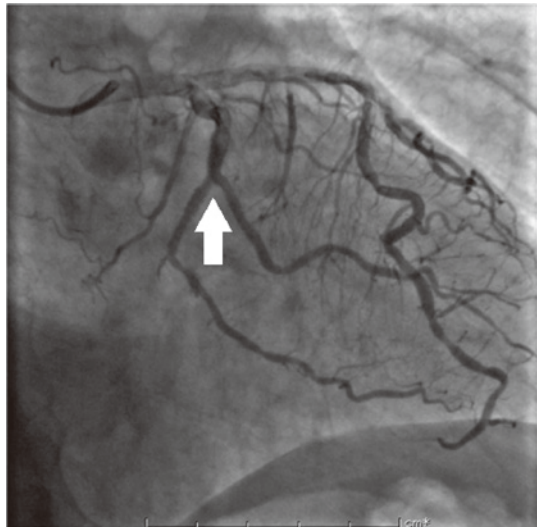


Fig. 4. Left coronary angiogram image after PCI shows the recanalization of occlusion of segment 13 (arrow). Segment 15 is too thin to insert a guide wire because segment 4PD is dominant. Segment 4PD is not shown in this figure.

was discharged 15 days after admission with no complications.

Discussion

When patients with prolonged chest pain or discomfort are first seen, the possibility of AMI must be considered. ECG is an important tool for diagnostic and triage tool for AMI¹⁾. Based on the results of the ECG, patients with AMI are divided into two groups : those presenting with ST-segment elevation, called ST-segment elevation AMI (STEMI), and those not presenting with ST-segment elevation, called non-ST-segment elevation AMI (NSTEMI). NSTEMI is sometimes difficult to diagnose, and the clinical hallmark of NSTEMI is severe chest pain, typically located in the substernal region or sometimes in the epigastrium²⁾. However, these clinical features are also seen with aortic dissection and PE, so that these patients sometimes undergo CECT to diagnose or rule out these conditions.

As CT scanning times have shortened, the quality and timing of contrast enhancement have improved³⁾. Several reports have shown the usefulness of CECT for making the diagnosis of AMI⁴⁻¹⁰⁾. These studies reported that AMI could be detected on CECT as a hypo-enhanced area of the left ventricular wall correlated with the clinically infarcted coronary artery distribution. Naito et al. reported that early-phase myocardial enhancement is considered to reflect vascular bed perfusion, and a hypo-enhanced area of the cardiac wall in the early phase was highly predictive of ischemia or ischemic damage to the myocardium in ischemic heart disease⁴⁾. In our institution, contrast medium is injected for 2-3 minutes and scanning is started immediately after finishing the injection. Compared with several reports in which early phase images were obtained when the contrast medium first passed or 30 to 60 seconds after contrast medium bolus injection^{4,5)}, our CECT phase is slightly delayed. To the best of our knowledge, no report has compared the sensitivity of detection between early phase CECT and delayed phase CECT. Though the present case suggests that it is possible to detect AMI on delayed phase CECT and this is thought to be related to the lack of development of collateral arteries, further more investigations of the relationship between CECT scan timing and sensitivity are needed.

Amy et al. retrospectively evaluated the ability of CT

to detect AMI in a larger group of patients, and they concluded that AMI is detectable on CECT⁶⁾. When we interpret CT images in chest pain patients, we have to evaluate if there are any hypo-enhanced areas in the cardiac walls. If there are such findings, the diagnostic radiologist needs to suggest to the cardiologists the possibility of AMI.

In the present case, it was difficult to confirm AMI or to exclude aortic dissection and PE with laboratory data, initial ECG, and UCG. Only the symptoms and CECT images suggested AMI. Although it is important to diagnose AMI with the initial ECG and UCG because these examinations can be done quickly, when it is difficult to confirm or rule out AMI, as in the present case, CECT is useful.

Conclusion

The possibility of diagnosing AMI with CECT was reported. It is important to check cardiac wall enhancement when we interpret CT images in patients with severe chest pain.

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