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Usefulness of Delayed Enhancement Magnetic Resonance Imaging for Detecting Cardiac Rupture Caused by Small Myocardial Infarction in a Case of Cardiac Tamponade

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Delayed enhancement magnetic resonance imaging (DE-MRI) has excellent spatial resolution and compared with other cardiac imaging techniques it can detect a small myocardial infarction (MI) or a subendocardial infarction. A 76-year-old man was admitted for loss of consciousness because of cardiac tamponade. The cause of tamponade was unknown, but electrocardiography and blood test suggested a recent MI. The removal of 100 ml bloody fluid by immediate pericardiocentesis normalized his hemodynamics, and he regained consciousness. Neither echocardiography nor scintigraphy could determine the location of the MI or rupture, but DE-MRI clearly demonstrated a transmural enhancement in a very narrow range of the lateral wall of the left ventricle. Coronary angiography revealed a severely stenotic lesion in the obtuse marginal branch of the left circumflex artery. DE-MRI is a powerful tool for diagnosing small MI that are undetectable with other imaging. Therefore, DE-MRI should be applied in cases with cardiac tamponade by unknown causes. (*Circ J* 2005; **69:** 1556–1559)

Key Words: Cardiac rupture; Magnetic resonance imaging; Myocardial infarction

Delayed enhancement magnetic resonance imaging (DE-MRI) has excellent spatial resolution and compared with other cardiac imaging techniques, such as echocardiography and single photon emission computed tomography (SPECT), it can detect a small or a subendcadial myocardial infarction (MI)! Here we report a case of cardiac tamponade in which the cause was initially unknown and DE-MRI was useful to detect a cardiac rupture due to a small transmural infarction.

Case Report

A 76-year-old man was admitted to hospital after losing consciousness during exercise. He had complained of back pain that continued all night for 1 week before the admission. His blood pressure was 66/30 mmHg and heart rate was regular and 101 beats/min. The echocardiogram showed an echo-free space in the cardiac circumference, although the global left ventricular wall motion was almost normal and there was no segmental wall motion abnormality (Fig 1). His electrocardiogram recorded a counter-clockwise rotation of the QRS complex, an abnormal Q wave only in the aVL lead, and ST elevation in the inferior and anterior leads (Fig 2). The patient was diagnosed as having cardiac tamponade of unknown causes and underwent pericardiocentesis immediately. After the removal of 100 ml of bloody fluid, his systolic blood pressure rose to 130 mmHg and he regained consciousness. His leukocyte count was 6,800 cells/μl, and the serum concentrations of aspartate aminotransferase, creatine kinase and lactate dehydrogenase were 54 IU/L, 234 IU/L and 243 IU/L, respectively. Additionally, the qualitative troponin T test was positive. From the results, it was suspected that the cardiac tamponade was the result of a cardiac rupture caused by a recent MI. He was then treated with angiotensin-converting enzyme inhibitor, angiotensin II receptor blocker and β-blocker, and his systolic blood pressure was controlled between 100 and 120 mmHg.

After 4 weeks, he underwent both DE-MRI and dipyridamole stress ^{201}Tl myocardial SPECT. The magnetic resonance imaging (MRI) used a 1.5 T MR system with the combined use of 8-channel cardiac phased array coil (Signa Infinity Twinspeed, GE Medical Systems, WI, USA). DE-MRI was based on the inversion recovery prepared fast gradient echo sequence. Five to 9 slices of 10 mm in thickness were used to cover the entire heart. Contrast agent (0.2 mmol/kg Gd-DTPA-BMA; Daiichi Pharma, Tokyo, Japan) was intravenously injected, and DE-MRI was performed after 15 min. A transmural enhancement was clearly detected in a very narrow range of the lateral wall of left ventricle (Fig 3), although dipyridamole stress ^{201}Tl myocardial SPECT indicated only a slight hypoperfusion in the posterolateral segment (Fig 4). Re-distribution of ^{201}Tl was observed in the rest image, suggesting a significant coronary stenosis in a branch of the left coronary artery. Coronary angiography revealed a severely stenotic lesion in the obtuse marginal branch of the left circumflex artery. There were no significant stenotic lesions in the left anterior descending artery or the right coronary artery. Left ventriculography demonstrated only mild hypokinesis in the lateral wall of the left ventricle.

The patient was discharged without chest pain, and

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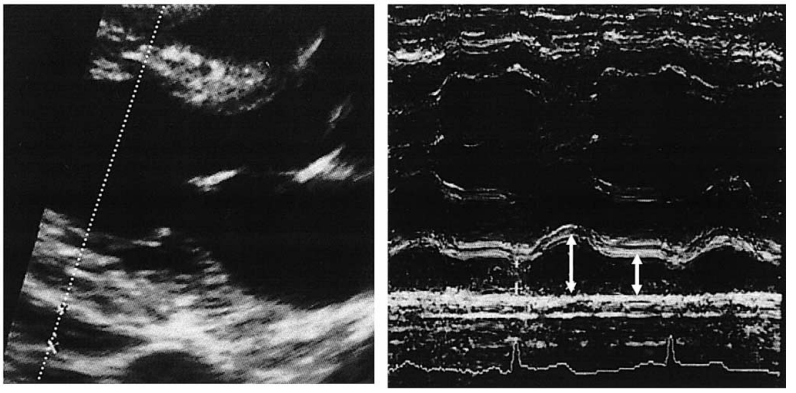

Fig 1. Echocardiography showing a circumferential echo-free space in the 2-dimensional image (Left). In the M-mode image (Right), the posterior echo-free space was 10 mm at systole and 8 mm at diastole (arrows). The left ventricular wall motion was almost normal and the ejection fraction was 72%.

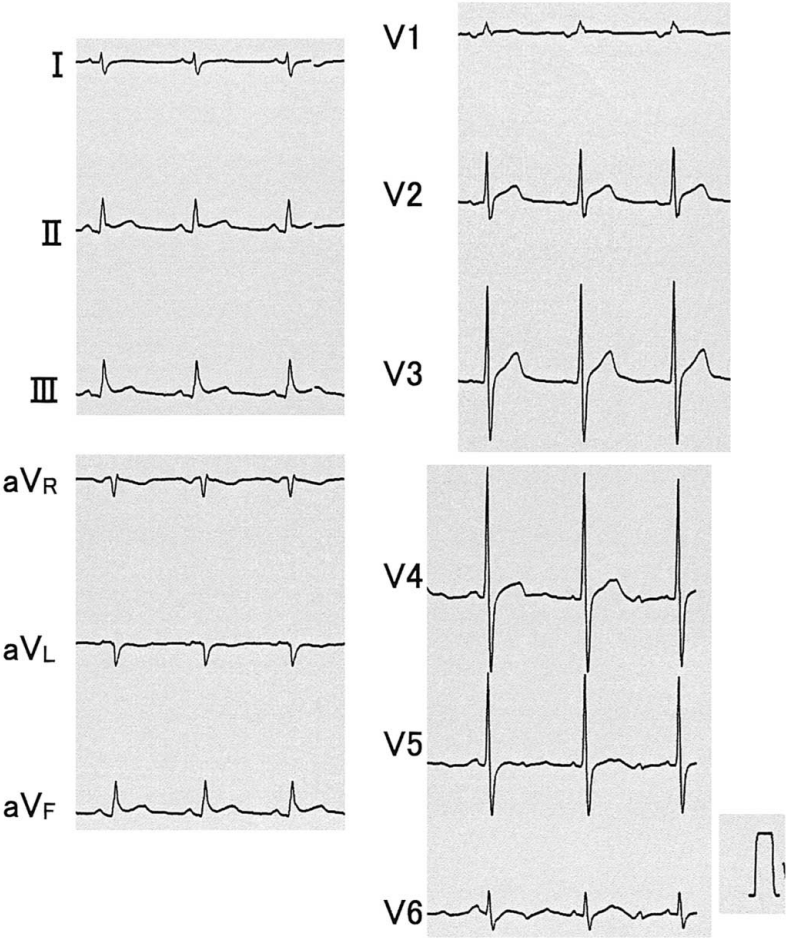

Fig 2. Electrocardiography showing normal sinus rhythm with heart rate of 101 beats/min, counterclockwise rotation of the QRS complex, an abnormal Q wave only in the aVL lead, and ST elevation in the inferior and anterior leads.

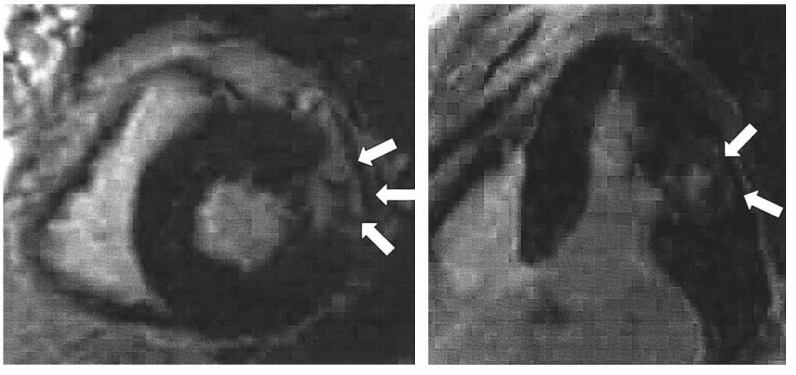

Fig 3. Delayed enhancement magnetic resonance imaging in the short-axis view (Left) and horizontal axis view (Right). Transmural enhancement can be clearly seen in a very narrow range of the lateral wall of left ventricle (arrows).

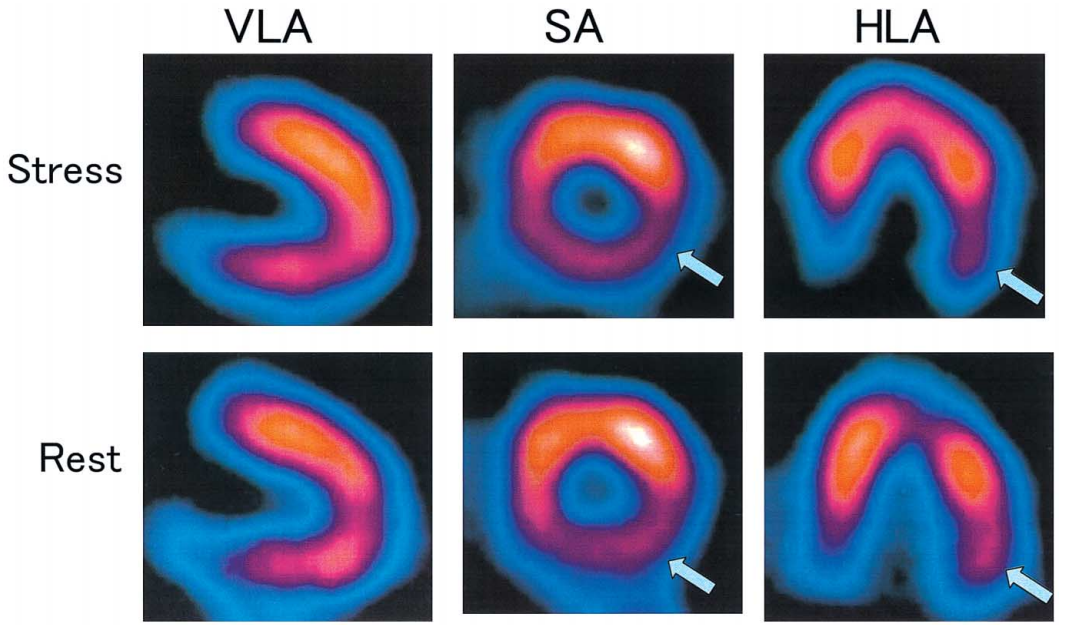

Fig 4. Dipyridamole stress myocardial ^{201}Tl single photon emission computed tomography. In the stress images (Upper), there is only slight hypoperfusion in the inferolateral segment (arrows). In the rest images, there is re-distribution of ^{201}Tl in the segment. VLA, vertical long-axis view; SA, short-axis view; HLA, horizontal long-axis view.

after a follow-up period of 12 months, remains alive and asymptomatic under treatment with angiotensin II receptor blocker, β-blocker and aspirin.

Discussion

In this case of cardiac tamponade caused by a cardiac rupture after a very small transmural MI in the lateral wall of the left ventricle, DE-MRI clearly showed the location and depth of the MI, whereas the findings of the other non-invasive examinations (ie, electrocardiography, echocardiography and SPECT) were equivocal.

Delayed cardiac enhancement in MRI requires intravenous gadolinium chelate, which is a biologically inert tracer that freely distributes in the extracellular space but does not cross the intact cell membrane. Because of a combination of increased extracellular volume and slower washout kinetics, there is a relative accumulation of gadolinium in damaged myocardium (eg, areas of fibrosis) in the late washout phase[2,3] DE-MRI can detect small and focal myocardial abnormalities because of its improved spatial resolution compared with ^{201}Tl and ^{99m}Tc myocardial SPECT[1,4–6] and is used to detect the site of cardiac rupture, and also to diagnose impending rupture[7,8]

Cardiac rupture accounts for 7–10% of early death after MI. The risk factors for cardiac rupture are older women, first MI with 1-vessel disease, anterior wall infarction, poor collateral branches, and a history of hypertension[9–12] The present case was initially diagnosed as cardiac tamponade of unknown cause, as the patient had not experienced the typical chest pain before losing consciousness. Serological findings suggested a recent MI, but electrocardiography, echocardiography and stress ^{201}Tl myocardial SPECT failed to provide precise information about an infarct or a ruptured lesion.

Ikeda et al reported that most cases of early-phase rupture are the blow out type, whereas late-phase rupture is mainly the oozing type[13] Saffitz et al also noted that the prevalence of cardiac rupture might not be related to the size of the MI[14] As the present case appeared to cardiac tamponade that had presumably occurred 1 week after the onset of MI, the oozing type of cardiac rupture was suspected. DE-MRI revealed the small transmural MI in the lateral wall of the left ventricle, which was consistent with the perfusion area of the obtuse marginal branch that had a significant stenotic lesion.

In this case, DE-MRI was performed 4 weeks after admission, and therefore, we can not apply the findings to the treatment in the acute phase. Cardiac rupture is a life-threatening complication of MI, even if the infarct is relatively small, so DE-MRI should be routinely performed in the acute phase in patients with cardiac tamponade of unknown cause.

In conclusion, DE-MRI is powerful tool for diagnosing small MI that are undetectable with other examinations. It should be used in the acute phase of cases of cardiac tamponade of unknown cause because early diagnosis is necessary for making the correct therapeutic decision and thus improving the prognosis.

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