

A clinical feature of myocardial stunning associated with acute myocardial infarction

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We report a case of myocardial stunning after acute myocardial infarction. In the hyperacute phase of myocardial infarction, the patient's coronary arteries showed normal features on coronary angiography during extensive ST-segment elevation observed on a standard 12-lead electrocardiogram and extensive akinesis observed on a left ventriculogram. Thallium-201 emission computed tomography revealed extensive perfusion abnormality. In the chronic phase, the perfusion abnormality was markedly improved. However, the electrocardiogram demonstrated poor R wave progression, and the left ventriculography revealed slight hypokinesis in the anterolateral wall. The acetylcholine provocation test disclosed coronary vasospasm of the left anterior descending coronary artery. About six months thereafter, left ventricular wall motion became completely normal and no poor R wave progression was observed on the electrocardiogram. The findings in this case indicate that myocardial stunning resulted from brief but severe ischemia due to vasospasm which led to cardiogenic shock, and that the recovery of findings for thallium-201 perfusion might be followed by those of electrocardiography and left ventriculography in the stunned myocardium.

Key words: Stunned myocardium, acute myocardial infarction, thallium-201 scintigraphy, left ventriculography

INTRODUCTION

WE EXPERIENCED a case of myocardial stunning associated with acute myocardial infarction. Perfusion scintigraphy, left ventriculography, and coronary angiography revealed several unique findings which may be characteristic of clinical myocardial stunning.

CASE REPORT

A 68-year-old female who had no coronary risk factors experienced faintness and mild chest discomfort while attending to her father at our hospital. She was immediately taken to the emergency room. On physical examination, her systolic blood pressure was 60 mmHg and pulse was weak but regular at 120 beats/min. A grade 3/6 holosystolic murmur, which radiated to the axilla and was

best heard at the apex, was detected. The electrocardiogram revealed extensive ST-segment elevation possibly indicating acute myocardial infarction (Fig. 1A). Forty minutes later, she was taken to the cardiac catheterization laboratory. Coronary angiography disclosed no significant luminal narrowing (Fig. 2). However, left ventriculography revealed akinesis in the antero-lateral, apical, and septal segments and severe mitral valve regurgitation with a global ejection fraction of 48% (Fig. 3A), although the electrocardiogram still revealed ST-segment elevation similar to that observed on the initial electrocardiogram. It took about 4 days for the electrocardiogram ST-segment elevation on electrocardiogram to return to the baseline (Fig. 1B). Hemodynamics also stabilized within 24 hours after onset. The serum levels of total creatine kinase (normal; 27-191 IU/l) and myosin light chain 1 (normal; < 5 ng/ml) subsequently peaked at 965 IU/l (24 hours after onset) and 19.6 ng/ml (3 days after onset), respectively. There were no obvious data indicating inflammatory diseases. Serial rest-redistribution thallium-201 emission computed tomography was performed on the 3rd, 12th, 20th, and 44th days after onset. Persistent perfusion defects were visible in the anterior wall on the

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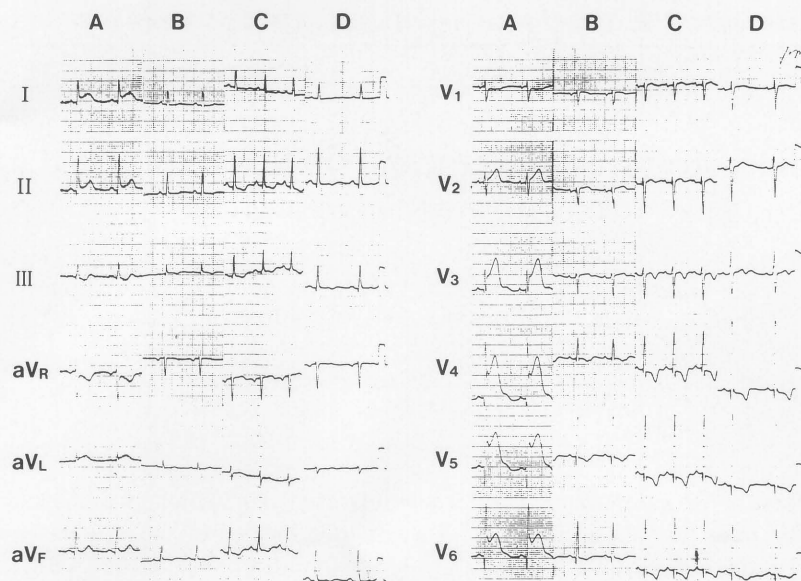


Fig. 1 Serial electrocardiograms. A, during the episode; B, 4th day after onset; C, 58th day after onset; D, 6 months after onset.

3rd day (Fig. 4A), but on the 20th day perfusion defects were considerably improved on the thallium-201 images (Fig. 4C). On the 58th day after onset cardiac catheterization was performed after all drugs were withdrawn for 24 hours. At this time, the electrocardiogram disclosed ST-T changes in II, III, aV_F, and V₃–V₆ leads, but increases in R waves in precordial leads (Fig. 1C). Left ventriculography revealed slight hypokinesis in the antero-lateral segment but marked improvement of regional wall motion, with a global ejection fraction of 65% (Fig. 3B). After control coronary angiograms were obtained, 100 μ g of acetylcholine was injected into the left coronary artery (Fig. 5). The mid to distal portion of the left anterior descending coronary artery and the diagonal branches subsequently revealed diffuse spasm with ST-segment elevation and chest pain. Six months later, the patient underwent left ventriculography, which revealed quite normal wall motion with a global ejection fraction of 78% (Fig. 3C). The electrocardiogram demonstrated left ventricular hypertrophy without any decrease in R waves in precordial leads (Fig. 1D).

DISCUSSION

The final diagnosis of acute myocardial infarction in the patient was made on the basis of serial changes in enzymes. In addition, the observation of acetylcholine-induced spasm of the coronary artery supplying the territory displaying abnormal wall motion supported this diagnosis. However, several possible mechanisms for the development of acute myocardial necrosis with cardiogenic shock when the coronary arteries are normal have been proposed.¹ It is particularly difficult to exclude a possible diagnosis of acute myocarditis, but the laboratory values,

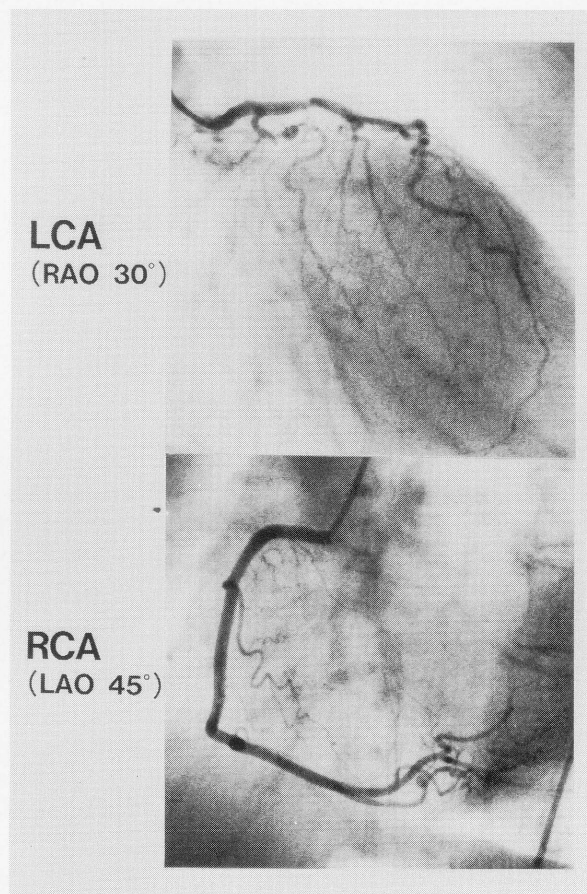


Fig. 2 Emergent coronary angiogram. Both left and right coronary arteries revealed almost normal findings.

including those for C-reactive protein and virus titers, were not abnormal in this case.

Among the unique features observed in this case were

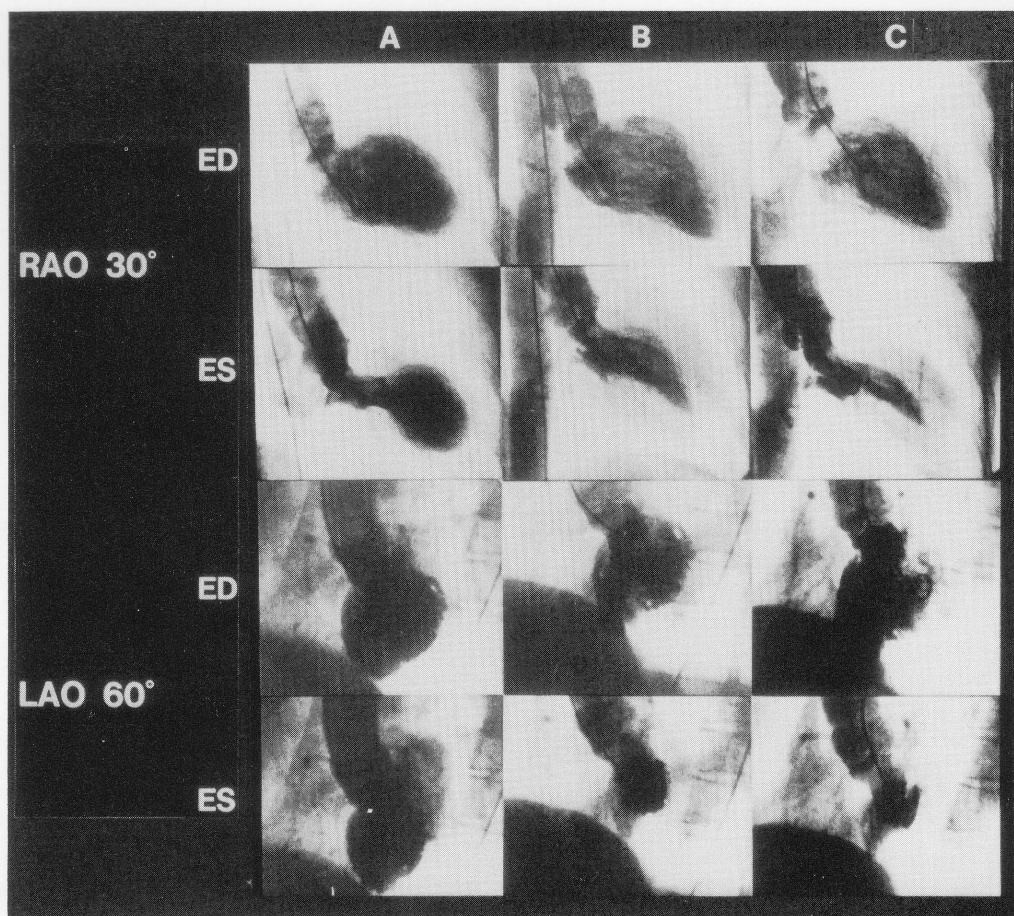


Fig. 3 Serial left ventriculography images. A, at the time of emergency cardiac catheterization; B, during chronic phase (58th day); C, 6 months after onset.

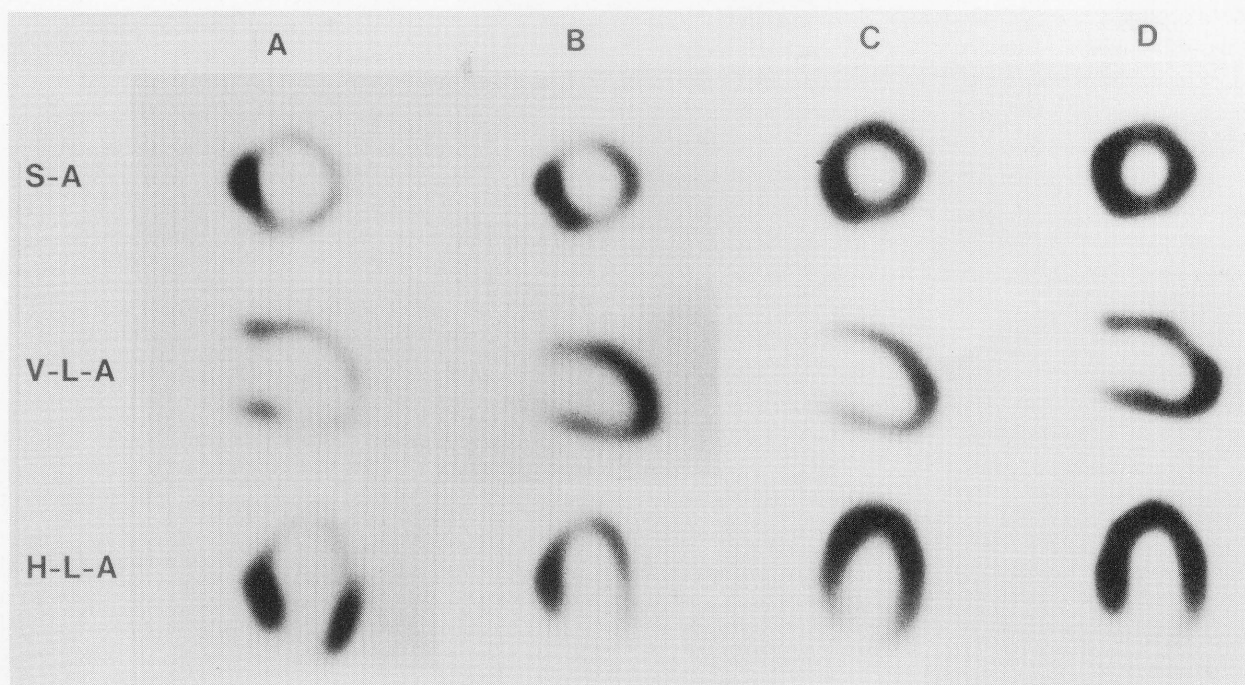


Fig. 4 Serial initial thallium-201 emission computed tomography images. A, 3rd day after onset; B, 12th day after onset; C, 20th day after onset; D, 44th day after onset.

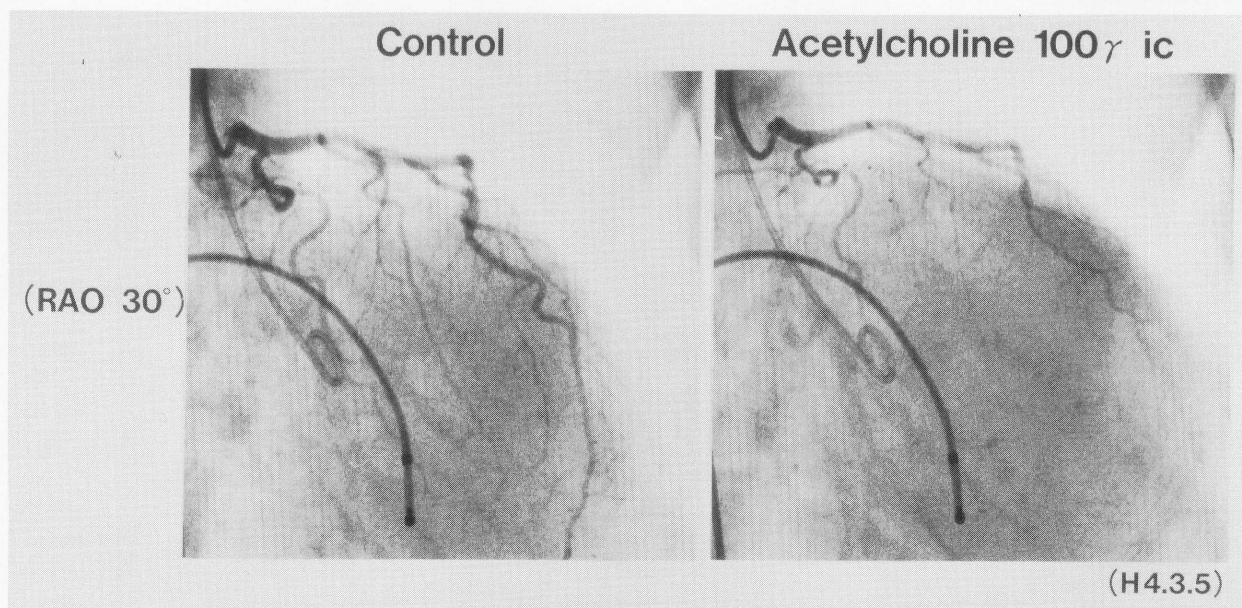


Fig. 5 Acetylcholine provocation test. Control coronary angiography of the left coronary artery is in the right. After the intracoronary injection of 100 μ g acetylcholine, the left anterior descending coronary artery revealed diffuse coronary spasm in the left.

normal coronary arteries during ST-segment elevation and cardiogenic shock at onset due to severe left ventricular dysfunction followed by almost complete recovery of the left ventricular wall motion despite perfusion abnormality (stunned myocardium).

ST-segment elevation in patients with normal coronary arteries and myocardial infarction has been reported.¹⁻³ Although ST-segment re-elevation after reperfusion has been reported as reperfusion injury,⁴ patients presenting this phenomenon have significantly decreased left ventricular function during the recovery phase compared to those without it. However, as left ventricular wall motion returned to normal in this case, reperfusion injury is not considered to be the mechanism of ST-segment elevation. Except during the very early phase of the infarction, the patient had no cardiac symptoms during ST-segment elevation and showed complete recovery of left ventricular wall motion, which might indicate that the ST-segment elevation was caused by neither evolving myocardial infarction nor reperfusion injury. In addition, as it is rare that patients with left ventricular akinesis after acute myocardial infarction have ST-segment elevation,² we therefore considered that the ST-segment elevation with a duration of about 4 days might have reflected the repolarization abnormality resulting from a severe ischemic insult.

A most unique feature in this case is the myocardial stunning. Several mechanisms of stunned myocardium have been proposed.^{3,5-8} In a report of a similar case by Tawarahara et al.,³ it was considered that stunned myocardium might be associated with the no reflux phenomenon or impaired sarcolemmal sodium-potassium

pump, or both. An experimental study demonstrated that thallium-201 extraction was unaltered in the stunned myocardium.⁹ However, in the present case persistent thallium-201 perfusion defects were observed on the images obtained soon after the onset of myocardial infarction. This finding indicates that acute ischemic insult can result in severe metabolic disturbance. Bulkey et al.¹⁰ demonstrated that some postinfarction patients presenting large thallium-201 defects on rest scintigraphy who subsequently died presented significantly smaller areas of necrosis at postmortem analysis than those of thallium-201 defects. The thallium-201 perfusion abnormality in our case is related to ischemia without necrosis. In addition, we were able to document the time course of recovery of myocardial function and of myocardial thallium-201 uptake. Left ventricular function returned to normal with a slight delay after thallium-201 uptake returned to almost normal. This finding might indicate that recovery from functional impairment (mechanical stunning) followed that from impairment of the Na^+ - K^+ pump (metabolic stunning) after the onset of ischemic insult. With regard to the changes in the 12-lead electrocardiograms, the time course of the recovery of the R wave in precordial leads (electrical stunning) was similar to that of left ventricular wall motion.

We have described the case of myocardial stunning associated with acute myocardial infarction. As a pathogenesis, coronary vasospasm may play an important role. Although the prognosis of this case was good, the patients with this type of acute myocardial infarction must be treated carefully because of the cardiogenic shock resulting from myocardial stunning at the onset of acute

myocardial infarction.

REFERENCES

1. Yasuda T, Molina-murphy IL, Vandervoot PM, Souther JF. Acute onset of cardiogenic shock associated with normal coronary arteries, diffuse myocardial necrosis, and rapid clinical recovery. *J Nucl Med* 3: 1558–1565, 1992.
2. Arvan S, Varat MA. Persistent ST-segment elevation and left ventricular wall abnormalities: A 2-dimensional echocardiographic study. *Am J Cardiol* 53: 1542–1546, 1984.
3. Tawarahara K, Kurata C, Okayama K, Watanabe H, Kobayashi A, Yamazaki N. Reversal of perfusion defect on resting thallium-201 emission computed tomography in a patient with stunned myocardium and coronary spasm. *Am Heart J* 125: 1425–1427, 1993.
4. Kondo M, Tamura K, Tanio H, Shimono Y. Is ST segment re-elevation associated with reperfusion as indicator of marked myocardial damage after thrombolysis? *J Am Coll Cardiol* 21: 62–67, 1993.
5. Braunwald E, Kloner RA. The stunned myocardium: prolonged, post-ischemic ventricular dysfunction. *Circulation* 62: 1146–1149, 1982.
6. Kim MS, Akera T. O₂ free radical: cause of ischemia-reperfusion injury to cardiac Na⁺-K⁺ ATPase. *Am J Physiol* 252: H252–H257, 1987.
7. Kloner RA, Ganote CE, Jennings RB. The no reflow phenomenon after temporary coronary occlusion in dog. *J Clin Invest* 51: 1496–1508, 1971.
8. Kloner RA, Przyklek K, Patel B. Altered myocardial states. The stunned and hibernating myocardium. *Am J Med* 86 (suppl): 14–22, 1989.
9. Moore CA, Cannon J, Watson DD, Kaul S, Beller GA. Thallium-201 kinetics in stunned myocardium characterized by severe postischemic systolic dysfunction. *Circulation* 81: 1622–1632, 1990.
10. Bulkey BH, Silverman K, Weisfeldt ML, Buroww R, Pond M, Becker LC. Pathologic basis of thallium-201 scintigraphic defects in patients with fatal myocardial injury. *Circulation* 60: 785–792, 1979.