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Short Communication

Transient Global Left Ventricular Dysfunction in a Localized Myocardial Infarction Related to Occlusion of the Distal Left Anterior Descending Artery

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Summary: In some patients with acute myocardial infarction (MI), wall motion in the noninfarcted area declines globally despite localized myocardial damage. In most, an infarct-related lesion is the proximal part of the left anterior descending artery (LAD). Previous studies have reported that hypokinesis of remote myocardium may be related to multivessel disease, impaired coronary flow, or coronary flow reserve in nonculprit arteries. This report describes the case of a 53-year-old man who presented with severe global left ventricular (LV) dysfunction after an acute MI associated with distal LAD occlusion. Follow-up echocardiographic examination revealed nearly normalized LV function 5 days after the episode. We discuss a plausible mechanism of dysfunction of noninfarcted myocardium.

Key words: ventricular dysfunction, myocardial infarction

Introduction

Previous studies have identified hyperkinetic wall motion of noninfarcted myocardium early after myocardial infarction (MI). In some patients, however, myocardial function in the remote region and global left ventricular (LV) systolic function

declines,^{1,2} and these patients usually have multivessel coronary disease or proximal left anterior descending artery (LAD) occlusion.^{2,3}

We report the case of a 53-year-old man who presented with severe but transient global LV dysfunction after an acute MI associated with distal LAD occlusion. We discuss the remote myocardial function in patients with acute MI and present a plausible mechanism of dysfunction of noninfarcted myocardium.

Case Report

A 53-year-old man presented at the emergency department with chest pain and diaphoresis of 1 h duration. The patient had a history of diabetes, dyslipidemia, and smoking, but no history compatible with angina pectoris or congestive heart failure. He had been under considerable emotional stress at work over the previous few days. At the time of admission, his physical examination results were remarkable for hemodynamic instability with cardiogenic shock. Twelve-lead electrocardiogram (ECG) demonstrated ST elevation of 7 mm in leads V₂, V₃, and V₄, of 3 mm in leads I and aVL, and ST depression of 3 mm in leads II, III, and aVF. Creatine kinase MB and troponin I were 2.7 ng/ml (290.2 ng/ml 10 h after admission) and 0.15 ng/ml (66.6 ng/ml 10 h after admission), respectively. Two-dimensional echocardiography showed severely reduced LV systolic function with an ejection fraction of 23% (Fig 1. A, B).

Urgent coronary angiography was performed and revealed an occlusion of the distal part of the LAD after the emergence of several septal branches (Fig 1.C). No luminal stenoses were seen in the left circumflex or right coronary arteries. Percutaneous coronary intervention of the LAD was thus performed using a 2.0 × 15 mm balloon catheter; however, blood flow was not restored after the procedure. The patient was subsequently treated conservatively with aspirin (100 mg/day), captopril (37.5 mg/day), carvedilol (12.5 mg/day), atorvastatin (10 mg/day), and insulin, and the patient made a gradual functional recovery during his 8-day hospitalization

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FIG. 1 End-diastolic (A) and end-systolic (B) frames of baseline echocardiography showing global hypokinesia of the left ventricle. Coronary angiography (C) demonstrating occlusion (arrow) of the distal left anterior descending artery in the right anterior oblique-cranial view. End-diastolic (D) and end-systolic (E) frames of 5-day follow-up echocardiography showing nearly normalized left ventricular systolic function.

period. Follow-up echocardiography 5 days after the episode exhibited an LV ejection fraction of 61% and normalization of wall motion, except that of the apex (Fig 1.D.E). Seven days after admission, a coronary spasm provocation test using intravenous ergonovine infusion proved negative. When discharged on Day 8 of his hospital stay, the patient was doing well and was asymptomatic.

Discussion

The described case was of a localized MI associated with distal LAD occlusion and global LV dysfunction. Severe dysfunction of myocardium remote from the infarct region and its rapid resolution are remarkable features in this case.

Although noninfarct myocardium may become hyperdynamic early after acute MI, wall motion in the affected region and global LV systolic function have been reported to decline in some reports.^{1,2} Homans *et al.*³ demonstrated that, in patients with multivessel disease, large wall motion abnormalities may increase the oxygen consumption by the remaining myocardium, and the dysfunction of this myocardium may be the result of relative hypoperfusion. Recent studies have reported that hypokinesia of remote myocardium may be associated with reduced coronary flow, reduced coronary flow reserve, increased remote loading on the noninfarcted region, or

mechanical tethering of the noninfarcted region to the infarcted myocardium. Other suggested mechanisms include delayed nonculprit artery flow associated with extensive necrosis in shared microvasculature and vasoconstriction mediated by local neurohormonal reflexes. In addition, Yoshino *et al.*² have shown that in anterior wall MI, decreased wall motion of the inferior wall is related to infarct-related narrowing or occlusion proximal to the origin of the first diagonal branch of the LAD. Nonetheless, our case exhibited severe global myocardial dysfunction in a patient with occlusion in the distal part of the LAD. Furthermore, other epicardial arteries revealed no luminal narrowing in this case. Thus, although LV dysfunction in our patient may be partly accounted for by several of the mechanisms mentioned above, the precise cause is not known.

In the described case, it was notable that wall motion and LV ejection fraction recovered rapidly within a week. A small number of studies have been performed on early LV ejection fraction changes in patients with acute MI and remote wall motion abnormalities. Grines *et al.*¹ observed noninfarct zone functional recovery during the first week post infarction; however, the presented data are discordant with the present case. In particular, baseline LV function was mildly depressed and ejection fraction improvement was modest in this earlier study, whereas LV ejection fraction dramatically improved from 23 to 61% in our case. Although reversible LV systolic dysfunction is frequently observed in cases of acute ischemia induced

by coronary artery disease, this abnormality can complicate critically ill patients in the absence of significant coronary narrowing. Sharkey *et al.*⁴ reported the diagnoses in such patients included central nervous system injury, sepsis, acute pulmonary disease, metabolic abnormality, and postnoncardiac surgery. Moreover, transient LV dysfunction in patients under emotional or physical stress, known as LV apical ballooning, is a recently recognized condition. This syndrome is characterized by acute substernal chest pain with ST-segment or T-wave changes, the absence of significant coronary arterial narrowing, marked but transient LV systolic dysfunction (especially of the mid and distal LV), and profound psychological or physical stress preceding the cardiac event.⁵ The mechanism of this syndrome remains uncertain, but may involve ischemia due to an epicardial coronary arterial spasm, a microvascular spasm, or catecholamine-mediated direct myocyte injury. Some features of our case, that is, coronary arterial occlusion and global LV dysfunction, are not typical of LV apical ballooning, while other features, such as concomitant psychological and physical stress, and the rapid recovery of LV systolic function are in keeping with this syndrome. It is difficult to conclude that the present case is representative of

LV apical ballooning; however, the underlying mechanism of this syndrome may be similar to that of the present case.

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