Transit Severe Mitral Regurgitation Complicating Myocardial Stunning due to Coronary Vasospasm

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As in papillary muscle dysfunction complicating mitral prolapse, dyskinesis of the left ventricular wall underlying the papillary muscles has been shown to cause mitral regurgitation following myocardial infarction. Myocardial stunning has been experimentally evidenced to cause mitral regurgitation due to a wall motion abnormality, but it has not yet been clinically defined. We report a clinical case of transient severe mitral regurgitation complicating myocardial stunning caused by coronary vasospasm. Transient wall motion abnormality beneath the anterolateral papillary muscle was considered to be responsible for the mitral regurgitation.

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Introduction

Mitral regurgitation complicating myocardial infarction has been well recognized due to mitral valve prolapse associated with tendon or papillary muscle rupture, or mitral annular dilation secondary to left ventricular enlargement. In addition to the pathologic conditions mentioned above, dyskinetic wall motion beneath one of the papillary muscles of the left ventricle per se has been shown to cause mitral regurgitation following myocardial infarction via incomplete mitral leaflet closure (1). Meanwhile, hypokinesis not dyskinesis of the left ventricular segment overlying the papillary muscles has been experimentally shown to produce mitral regurgitation during myocardial stunning (2), but it has not been clinically demonstrated until the present case.

Case Report

A 72-year-old woman was hospitalized for chest discomfort lasting over 2 hours. Physical examination revealed a systolic blood pressure of 106/60 mmHg and a regular pulse of 86 beats/min. A grade 3/6 holosystolic murmur was noted at the apex, and fine crackles were heard at the bases of both lungs. An electrocardiogram showed ST-segment elevation in leads V3 to V6. A chest radiograph showed pulmonary congestion without cardiomegaly. Arterial blood gas analysis showed hypoxemia with PO2 of 62 torr. Serum value of creatine kinase and white blood cell count were normal. Two-dimensional echocardiography revealed hypokinesis of the anteroseptal and anterolateral walls of the left ventricle without ventricular enlargement, mitral valve prolapse, or tendon or papillary muscle rupture (Fig. 1A). Color Doppler echocardiography showed severe mitral regurgitation without lateralization of the regurgitant jet (Fig. 2A).

Coronary angiography revealed no coronary artery stenosis but showed myocardial squeezing of segment 7 of the left anterior descending coronary artery with delayed distal filling. Right-sided cardiac catheterization demonstrated a slightly elevated mean pulmonary capillary wedge pressure (17 mmHg) with prominent v waves and a cardiac index of 2.54 l/min/m2. T-wave inversion in the left precordial leads appeared after completion of the catheterization. Neither myocardial enzyme leakage nor electrocardiographic Q waves were noted during hospitalization. The ST-segment elevation and T-wave inversion disappeared by the second hospital day. Echocardiography on the third hospital day revealed resolution of both left ventricular asynergy (Fig. 1B) and mitral regurgitation (Fig. 2B), leading to a diagnosis of myocardial stunning. The coaptation point of the mitral leaflets at systole was closer to the mitral annulus at the third hospital day compared with that on admission (Fig. 1A, B).
Mitral Regurgitation by Myocardial Stunning

Figure 1. A: Two-dimensional echocardiography from an apical four chamber view on admission showed hypokinesis of the anteroseptal and anterolateral wall. B: The wall motion abnormality disappeared on the third hospital day. The coaptation point of the mitral leaflets at systole was located closer to the apex on admission compared with that at the third hospital day. Left panel: end-diastole; Right panel: end-systole.

Follow-up coronary angiography 1 month later showed normal coronary arteries at the baseline. To examine the association of coronary spasm with myocardial stunning, intracoronary injection of acetylcholine was performed. An injection of 100 µg acetylcholine into the left coronary artery provoked diffuse spasm of the left anterior descending coronary artery with chest pain and electrocardiographic ST-segment depression in V1 to V6. An injection of 50 µg acetylcholine into the right coronary artery elicited diffuse constriction of the artery without chest pain and electrocardiographic changes. Right-sided cardiac catheterization showed normal pulmonary capillary wedge pressure without prominent v waves. Left ventriculography revealed slightly reduced motion of the segments 3 and 6 without mitral regurgitation. Thallium myocardial scintigraphy showed no perfusion defect.

Discussion

The present case exhibited severe transient mitral regurgitation associated with myocardial stunning. Coronary spasm was provoked in the left anterior descending coronary artery whose perfusion area was identical to the asynersic area on myocardial stunning. Thus, we considered that coronary spasm of the left anterior descending coronary artery was responsible for the myocardial stunning. Myocardial squeezing of the proximal left anterior descending coronary seemed unlikely to have caused the myocardial stunning, because it decreases coronary blood flow only at systole.

Papillary muscle or tendon rupture resulting in mitral valve prolapse and mitral annular dilation, which frequently cause mitral regurgitation in left ventricular infarction, were not shown in this patient. Papillary muscle ischemia as a cause of mitral regurgitation seems unlikely in this patient. As the anterolateral papillary muscle is perfused by both the diagonal and obtuse marginal branches of the coronary artery, its ischemic dysfunction infrequently occurs by only the occlusion of the left anterior descending coronary artery shown in the present case (3). Moreover, previous studies have experimentally shown that
isolated infarction of the papillary muscle without wall motion abnormality of the left ventricle does not lead to mitral regurgitation (4, 5).

Godley et al (1) reported that mitral regurgitation complicating prior myocardial infarction is caused by dyskinesis of the left ventricular myocardium beneath one of the papillary muscles, which produces retraction of the anterior mitral leaflet toward the apex at systole and results in incomplete mitral leaflet closure. Kono et al (2) have experimentally shown that left ventricular hypokinesis following occlusion of the left circumflex coronary artery causes transient mitral regurgitation. They described that hypokinesis of the wall motion underlying the papillary muscle also led to a retraction of the mitral leaflets toward the apex at systole which resulted in incomplete mitral leaflet closure and mitral regurgitation. The resolution of mitral regurgitation with concurrent disappearance of the wall motion abnormality shown by Kono et al (2) coincided with the finding in the present patient. Although incomplete mitral leaflet closure failed to be identified in our patient, hypokinesis of the anterolateral wall associated with myocardial stunning was considered to likely cause incomplete mitral leaflet closure and to result in transient mitral regurgitation. This is supported by the fact that the mitral coaptation point was deviated toward the apex on admission compared with that on the third hospital day, which was also noted in the transient mitral regurgitation in Kono’s report.

This is the first clinical report to present transient mitral regurgitation due to myocardial stunning that was caused by coronary spasm. It is also noteworthy that coronary vasospasm can produce myocardial stunning that causes transient but severe mitral regurgitation resulting in pulmonary congestion.

References