SYSTOLIC ANTERIOR MOTION OF THE MITRAL VALVE ASSOCIATED WITH ACUTE MYOCARDIAL INFARCTION IN A 62-YEAR-OLD PATIENT

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A 62-year-old patient was admitted to our hospital for treatment of early gastric cancer. After total gastrectomy, he suffered from antero-septal myocardial infarction. Systolic anterior motion of the mitral valve which was not observed before surgery was observed during the acute phase of myocardial infarction and completely disappeared 3 weeks after the onset of myocardial infarction. Systolic anterior motion of the mitral valve in this patient may have been caused by changes in left ventricular wall motion during the acute phase of myocardial infarction.
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SYSTOLIC anterior motion of the mitral valve (SAM) is generally observed in patients with hypertrophic obstructive cardiomyopathy (HOCM). Venturi theory seems to be an important factor in the maintenance of SAM¹, but it cannot explain the onset of anterior motion of the mitral valve prior to aortic valve opening. The precise mechanism for the pathogenesis of SAM is not fully understood. We report a patient showing SAM associated with acute myocardial infarction, and discuss factors which may be responsible for SAM.

CASE REPORT

A 62-year-old man was admitted to Ehime University Hospital for the treatment of early gastric cancer on August 30, 1990. He began to notice dizziness after taking a bath or consuming alcohol 2 years before the admission. One year before the admission

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he was suspected of having hypertrophic cardiomyopathy (HCM), due to the existence of a proximal bulge of the septum, a small left ventricular chamber (Fig. 1-A) and chordal slack on the echocardiogram under no medication. There was no family history of HCM. He had been treated with a β -blocker and a calcium antagonist, which eliminated the dizziness. He was diagnosed as having early gastric cancer and gastric polypsis by gastroscopy and biopsy, and was admitted for surgical treatment of gastric cancer. On admission, his electrocardiogram was normal (Fig. 2A). After total gastrectomy on September 25, his electrocardiogram showed ST elevation. At that time, his blood pressure was 110/60 mmHg, pulse rate 80 beats/min and respiratory rate 12/min. Auscultation of the heart revealed a loud fourth sound and grade 2/6 systolic murmur at the third left sternal border. Standard 12-lead electrocardiogram showed ST elevation in leads V₄ and V₅ with a reduction of the R wave in V₂–V₆, as well as ST depression in II, III and aVF (Fig. 2-B). Laboratory data

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Fig.1. Two-dimensional echocardiograms in the parasternal long axis view (upper) and apical 4-chamber view (lower).

- A: Before gastrectomy. The wall motion was normal. The arrow indicates the bulge of the basal interventricular septum.
 B: After acute myocardial infarction. Wall motion in the anteroseptal region showed akinesis (arrows). The origin of SAM is indicated by double arrows.
 C: Three weeks after acute myocardial infarction. Left ventricular wall motion almost returned to normal.

Fig.2. Electrocardiograms.

- A: Pre-operative electrocardiogram.
 B: Electrocardiogram recorded on the day after the onset of acute myocardial infarction.
 C: Two weeks after acute myocardial infarction.
 D: One year after acute myocardial infarction.

showed a red blood cell count of $4.1 \times 10^6/\text{mm}^3$, a hematocrit of 37.8% and a hemoglobin concentration of 13.1 g/dl. Serum levels of creatine kinase and creatine kinase MB were maximally elevated to 1794 IU/l and 10%, respectively. Akinesis of the left ventricular anteroseptal segment (arrows) was observed on the echocardiogram (Fig. 1-B). These findings indicated anteroseptal myocardial infarction.

The acute phase of the clinical course is shown in Fig. 3. His systolic blood pressure was maintained at 100 to 120 mmHg with a

drip infusion of dopamine, $5 \mu\text{g/kg/min}$, and by application of nitroglycerin ointment, 27 mg/day. Cardiac output was 2.89 l/min. His systolic blood pressure gradually decreased to 70 mmHg, and cardiac output fell to 2.09 l/min. Dopamine was increased to $9 \mu\text{g/kg/min}$, but systolic blood pressure only increased to 90 mmHg. Grade 4/6 systolic murmur began to be heard at the third left sternal border. SAM and semiclosure of the aortic valve were observed on M-mode echocardiogram (Fig. 4B). Existence of a significant pressure gradient in the left ventricular

Fig.3. Clinical courses.

Abbreviations: SBP, systolic blood pressure; PG, peak pressure gradient of the left ventricular outflow tract; SAM, systolic anterior motion of the mitral valve. A, B, C and D indicate the days the echocardiograms shown in Fig. 4 were recorded.

Fig.4. M-mode echocardiograms at the mitral level.

- A: Before gastrectomy. Systolic anterior motion of the mitral valve (SAM) was not recognized. Chordal slack was observed.
- B & C: After acute myocardial infarction. Complete SAM was seen. SAM was diminished by volume overload (B to B'). SAM was also diminished after intravenous administration of propranolol (2 mg/hr), (C to C'). The wall motion of the basal interventricular septum showed hyperkinesis.
- D: Three weeks after acute myocardial infarction. SAM had completely disappeared.

TABLE I CHANGES IN THE WIDTH OF THE LEFT VENTRICULAR OUTFLOW TRACT AND EXCURSIONS OF THE LEFT VENTRICULAR WALLS

	Width of outflow tract (mm)	Excursion (mm)			
		Proximal IVS	Middle IVS	Proximal PW	Middle PW
Before AMI	27	7.2	12.2	6.2	13.0
AMI	22	10.5	3.0	11.5	11.0
After AMI	25	6.2	11.0	6.5	11.0

Abbreviations: IVS, interventricular septum; PW, left ventricular posterior wall; AMI, acute myocardial infarction. The width of the outflow tract was calculated as the distance between the C point of the mitral valve and the IVS using M-mode echocardiography. Proximal IVS and PW were recorded at the mitral level. Middle IVS and PW were recorded at the tips of the mitral leaflets.

outflow tract was confirmed by continuous wave Doppler using Bernoulli's equation. The pressure gradient after volume overload (Fig. 4-B') was 22 mmHg. However, as shown in Fig. 4-C, SAM gradually increased again and the maximal pressure gradient reached 92 mmHg on the 4th day after acute myocardial infarction. Moderate mitral regurgitation was also found. Central venous pressure ranged from 40 to 60 mmH₂O. After intravenous administration of propranolol (2 mg/hr), the pressure gradient of the left ventricular outflow tract decreased to 49 mmHg (Fig. 4-C'). Despite the continuous administration of propranolol, the pressure gradient at the left ventricular outflow tract varied from 45 to 65 mmHg during the existence of anteroseptal akinesis. Three weeks after the onset of acute myocardial infarction, SAM and the left ventricular pressure gradient completely disappeared, and the left ventricular anteroseptal motion was almost normalized (Figs. 1-C and 4-D).

Coronary angiography was performed on February 7, 1991, and showed a long segmental 50% narrowing of the left anterior descending artery (AHA classification=7), and a 90% narrowing of the first diagonal branch. Left ventriculogram showed no abnormal left ventricular motion, and left ventricular ejection fraction was 89%. No significant pressure gradient was observed in the left ventricular outflow tract at the basal state (heart rate; 60 beats/min). The pressure gradient was not provoked by the intravenous administration of isoproterenol (0.02 µg/kg/min). By the time the total dose had reached 1.5 µg, the heart rate had increased to 96 beats/min. There was no de-

fect in the ²⁰¹Tl myocardial scintigram. We diagnosed this patient as non-Q wave infarction from the clinical features. Table 1 shows the changes in the width of the outflow tract and the excursions of left ventricular walls. Narrowing of the outflow tract and hyperkinesis of the proximal left ventricular walls in the acute phase of myocardial infarction were confirmed.

DISCUSSION

Before myocardial infarction, the left ventricle of this patient was echocardiographically characterized by a small ventricular size, a bulge at the basal septum and chordal slack. These findings suggested HCM with fixed hypertrophy of the basal septum. However, the patient had no family history of HCM and myocardial biopsy was not performed, so the diagnosis of HCM could not be confirmed. After anterior myocardial infarction, movement of the proximal interventricular septum, which was not involved in the infarction, increased (Fig. 4-C) to compensate for hypokinesis of the mid-to-distal interventricular septum. Movement of the left ventricular posterior wall also increased to maintain the stroke volume. These changes resulted in the following physiological conditions: 1) Narrowing of the left ventricular outflow tract was provoked and a high-velocity ejection flow was generated; and 2) The streamline from the posteroinferior region seemed to be relatively stronger than that from the anteroseptal region, and this condition progressed as the anterior mitral leaflet was drawn anteriorly. Existence of slackened chorda suggests mal-

position of the papillary-mitral apparatus in this patient, and this malposition seemed to be promoted by the damage associated with myocardial infarction. As indicated by Jiang et al², this malposition seemed to play an important role in the onset of SAM. Thus, the appearance of SAM in our patient was considered to be caused by the existence of slackened chorda^{2,3}, narrowing of the left ventricular outflow tract⁴, and hyperkinesis of the left ventricular posterior wall⁵ induced by anterior myocardial infarction. These 3 factors seemed to be indispensable for the appearance of SAM. The disappearance of SAM after inferior myocardial infarction⁶ and the decrease in both SAM and the pressure gradient after the administration of a β -blocker also suggested that wall motion strongly influenced the severity of SAM. Other factors, including dehydration, anemia, tachycardia and administration of inotropic agents, have also been reported to cause SAM⁷. SAM in our patient was temporarily improved after volume overload and initially after discontinuation of an inotropic agent (Fig. 4-B,B'). Therefore, these factors were undeniably involved in the appearance of SAM. Anemia was not observed at the onset of acute myocardial infarction. In the chronic stage of myocardial infarction, the pressure gradient was not provoked by intravenous administration of isoproterenol. This seemed to be due to the normalization of left ventricular wall motion at the chronic phase. Therefore, taking into account the fact that SAM disappeared when the left ventricle returned to its pre-infarction state, these findings suggest that SAM in our case

was mainly due to narrowing of the left ventricular outflow tract and hyperkinesis of the left ventricular posterior wall associated with acute myocardial infarction.

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