CASE REPORTS

ACUTE MYOCARDIAL INFARCTION DUE TO CORONARY EMBOLIZATION FROM LEFT ATRIAL MYXOMA

HARUHISA HASHIMOTO, M.D., HIROKAZU TAKAHASHI, M.D.
YASUSHI FUJIWARA, M.D.†, TADAUMI JOH, M.D.†
AND TETSUO TOMINO, M.D.**

We encountered a 67-year-old woman with a left atrial myxoma which was discovered during echocardiographic examination and emergency coronary arteriography just after an onset of acute inferior myocardial infarction. Coronary arteriography disclosed an abrupt and total occlusion of the right coronary artery and an abnormally large and tortuous atrial circumflex branch feeding a left atrial mass. These findings were the most useful for diagnosis. Aorto-coronary bypass surgery and excision of the myxoma were performed simultaneously by emergency operation. The postoperative course was uneventful. Myocardial infarction in this patient is believed to have been caused by coronary embolization from the left atrial myxoma. (Jpn Circ J 1993; 57: 1016–1020)

Left atrial myxoma is a common primary tumor which frequently presents systemic embolization1,2. However, acute myocardial infarction due to coronary embolization from the left atrial myxoma is an extremely rare clinical presentation3–12. We report here such a case of left atrial myxoma for which abnormal findings from emergency coronary arteriography were the most useful for diagnosis.

CASE REPORT

A 67-year-old woman visited the Yoshida General Hospital on the 8th of June, 1992, because of a sudden onset of severe epigastralgia which lasted nearly 30 min. An electrocardiogram revealed a normal sinus rhythm of 64 beats per min and ST-segment elevation in Leads II, III, and aVF, which were compatible with acute inferior wall infarction. On auscultation, S1 was increased.

Key words:
Left atrial myxoma
Acute myocardial infarction
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Coronary embolization

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Department of Internal Medicine, Yoshida General Hospital, Yoshida, Ehime 799-37, Japan
*Department of Internal Medicine and **Department of Cardiovascular Surgery, Ehime Prefectural Central Hospital, Matsuyama, Ehime 790, Japan
Mailing address: Haruhisa Hashimoto, M.D., Dept. of Internal Medicine, Yoshida General Hospital 217 Kitakohji, Yoshida, Ehime 799-37, Japan

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but neither S3, S4, systolic or diastolic murmur, nor “tumor plop” was heard. A two-dimensional echocardiographic examination disclosed a large mobile mass in the left atrium (Fig. 1) and hypokinesis of the inferior wall. No evidence of valvular disease or vegetation was detected. During this echocardiographic study, the patient suffered from a sudden ventricular fibrillation which was successfully defibrillated with a DC electrical shock of 200 Joules. Immediately after this episode, xylocaine and recombinant tissue plasminogen activator were administered intravenously. The patient was then transferred to the Ehime Prefectural Central Hospital for further evaluation of the left atrial mass and her coronary artery.

The first coronary angiographic examination, which was performed 5 h after the onset, showed an abrupt and total occlusion of the right coronary artery at its origin and “vascular blush” of an atrial mass, arising from an abnormally large and tortuous atrial circumflex branch of the left circumflex artery (Fig. 2). The rest of the coronary artery was smooth and normal in diameter, and no aneurysmal dilatation was observed. Since recanalization of the right coronary artery had not been achieved despite the administration of a potent thrombolytic agent, i.e., recombinant tissue plasminogen activator, and since the patient had no coronary risk factors or family history of ischemic heart disease, a probable diagnosis of left atrial myxoma complicated with its embolization of the coronary artery was possible before the emergency operation. To prevent further embolization, our patient underwent simultaneous excision of the left atrial tumor and aorto-coronary bypass surgery using a saphenous vein graft on the first day of hospitalization. The excised tumor (58×39×30 mm, Fig. 3) was gelatinous and friable, and histologically was a typical myxoma. No thrombi were found on the surface of the tumor. The tumor was attached by a 6 mm pedicle to the interatrial septum adjacent to the fossa ovalis. The postoperative course was uneventful.

The peak values of cardiac enzymes were observed at 24 h after the onset: creatine phosphokinase (CPK), 1404 IU/L with 46 IU/L of MB isoenzyme fraction; glutamic oxaloacetic transaminase (GOT), 151 IU/L; and lactic dehydrogenase (LDH), 953 IU/L.

The second coronary arteriography performed on July 6, 28 days after the operation, revealed a degradation of the feeding artery and a patent bypass graft, as well as recanalization of the right coronary artery (Fig. 4). The hemodynamic measurements were as follows: pulmonary artery pressure, 29/15 mmHg; mean pulmonary wedge pressure, 11 mmHg; right ventricular pressure, 31/8 mmHg with an end-diastolic pressure of 12 mmHg; aortic pressure, 143/83 mmHg with a mean aortic pressure of 105 mmHg; left ventricular pressure, 139/7 mmHg with an end-diastolic pressure of 22 mmHg, and a cardiac index of 3.21/min/m². An exercise thallium-201 scintigraphic examination disclosed no significant defect or delay in any
area of the myocardium. The patient was discharged on August 4, 1992.

DISCUSSION

The most common clinical manifestations of left atrial myxoma are signs and symptoms resembling mitral valve disease due to mechanical interference with cardiac function. Nonspecific signs and symptoms such as fever, weight loss, arthralgia, and elevated erythrocyte sedimentation rate and serum gammaglobuline, frequently suggest other systemic disorders, and may lead to misdiagnosis as a collagen disease such as polyarteritis nodosa. Another clinical presentation of left atrial myxoma, i.e., embolization to various systemic organs, is often the first and only clinical symptom.

Cerebral and peripheral arteries are the most common sites of embolization. Silvermann et al. in their review of 29 cases of systemic embolization from left atrial myxoma, reported that 15 cases (52%) were cerebral embolization, followed by 11 cases (38%) of embolization of peripheral arteries, 10 cases (34%) of renal embolization, 6 cases (21%) of splenic embolization, and only 3 cases (10%) of coronary embolization. Therefore, coronary embolization from left atrial myxoma is extremely rare.

Regarding coronary embolism, Wenger and Bauer reported in a large scale post-mortem study, that only 11 of 17,469 (0.06%) consecutive cases manifested coronary embolization. In a review of established postmortem studies, they found 74 cases with coronary embolism. The underlying diseases were subacute bacterial endocarditis (39 cases, 53%), acute bacterial endocarditis (8 cases, 11%), intra-cardiac thrombus (8 cases, 11%), luetic aortitis (4 cases, 5%) and paradoxical embolization from a tumor (1 case, 1%). However, no case of coronary embolization was reported from left atrial myxoma. Possible explanations for the rarity of coronary embolization include 1) filling of coronary circulation during diastole occurs after most of the blood volume has entered systemic circulation, 2) coronary arteries arise from ascending aorta at right angles, 3) coronary arteries are protected during systole by leaflets of the aortic valve and 4) coronary arteries have relatively small calibers compared with the ascending aorta.

Regarding the cause of acute myocardial infarction in the present case, coronary embolization from a left atrial myxoma is the most reasonable explanation since she had no coronary risk factors, such as hypertension, smoking, hypercholesterolemia or diabetes mellitus, and because her coronary arteriogram disclosed no atherosclerotic

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changes other than the sharp filling defect at the right coronary artery. In addition, the short-term failure of recanalization of the right coronary artery despite the administration of recombinant tissue plasminogen activator, and the fact that there was no thrombus formation on the surface of the excised tumor, strongly support this possibility. An interesting observation was the complete recanalization of the right coronary artery at the second catheterization study. Rath et al reported a similar case who showed complete disappearance of filling defect at postoperative coronary arteriography. Although typical angiographic findings in embolization from myxoma include narrowing of vessels and aneurysmal dilatations in the chronic stage, the present case did not show such changes at the second coronary arteriography. The precise reason for this phenomenon is unknown because a histological examination was not performed at the embolic site in this case.

As shown in Table I, 11 cases, including the present case, have been reported in which cardiac events were the direct clinical

**TABLE I** REPORTED CASES OF LEFT ATRIAL MYXOMA WHICH WERE DIAGNOSED FOLLOWING AN ONSET OF ACUTE MYOCARDIAL INFARCTION, AND WHICH UNDERWENT EXCISION OF THE TUMOR

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Age/sex</th>
<th>Site of MI</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Tanabe</td>
<td>1979</td>
<td>11/M</td>
<td>inferior</td>
<td>12 days</td>
</tr>
<tr>
<td>2. Balk</td>
<td>1979</td>
<td>29/M</td>
<td>anterior</td>
<td>3 months</td>
</tr>
<tr>
<td>3. Rath</td>
<td>1984</td>
<td>55/M</td>
<td>inferior</td>
<td>10 days</td>
</tr>
<tr>
<td>4. Lehman</td>
<td>1985</td>
<td>43/M</td>
<td>anterior</td>
<td>2 weeks</td>
</tr>
<tr>
<td>5. Tatsukawa</td>
<td>1986</td>
<td>42/F</td>
<td>anterior</td>
<td>89 days</td>
</tr>
<tr>
<td>6. Hoad</td>
<td>1987</td>
<td>68/F</td>
<td>inferior</td>
<td>2 months</td>
</tr>
<tr>
<td>7. Usui</td>
<td>1987</td>
<td>52/M</td>
<td>inferior</td>
<td>44 days</td>
</tr>
<tr>
<td>8. Doi</td>
<td>1988</td>
<td>34/M</td>
<td>inferior</td>
<td>47 days</td>
</tr>
<tr>
<td>9. Namura</td>
<td>1990</td>
<td>58/M</td>
<td>inferior</td>
<td>35 days</td>
</tr>
<tr>
<td>10. Onitsuka</td>
<td>1991</td>
<td>16/F</td>
<td>anterior</td>
<td>32 days</td>
</tr>
<tr>
<td>11. Hashimoto</td>
<td>1993</td>
<td>67/F</td>
<td>inferior</td>
<td>1 days</td>
</tr>
</tbody>
</table>

Duration represents interval from onset of myocardial infarction to excision of the myxoma. M: male, F: female, MI: myocardial infarction.

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manifestation which led to the discovery of left atrial myxoma during the patient's life-time. These 11 cases include 7 males and 4 females with ages ranging from 11 to 68. The site of myocardial infarction was the anterior wall in 4 cases and the inferior wall in 7 cases. All of these cases underwent excision of the tumor and survived. A report by Chamberlin et al. was excluded because coronary embolization occurred during an operation. Other reports by de Morais et al. and Romisher et al. were also excluded because excision of the myxoma was not performed due to the death of their patients soon after the onset of myocardial infarction. The intervals from onset of cardiac events to surgery varied from one day in the present case to three months by Balk et al. Because an unfavorable outcome is not infrequent in patients who are awaiting surgery, we support the view that left atrial myxoma should be removed essentially on an emergency basis to prevent systemic embolization.

In summary, a patient with a left atrial myxoma, which was discovered during echocardiographic examination and emergency coronary arteriography just after an onset of acute myocardial infarction, underwent successful emergency aorto-coronary bypass surgery and excision of the myxoma. The most useful diagnostic information in this case came from the abnormal findings on coronary arteriography.

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