Case Report

Left Atrial Myxoma Presenting as Acute Myocardial Infarction

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A 58-year-old man with left atrial myxoma complicated with acute myocardial infarction is presented. On coronary arteriography, 50% stenosis and a saccular aneurysm was found just below the stenosis at segment 1 of the right coronary artery. Ergonovine provocation study was negative. It was proposed that the myocardial infarction probably was caused by coronary embolization from the left atrial myxoma.

Key words: Coronary embolization, Coronary arteriography

Left atrial myxoma frequently presents with embolic manifestations, but coronary artery embolism has been rarely reported. We encountered a case with acute myocardial infarction, which was thought to most probably be caused by coronary embolization from the left atrial myxoma.

CASE REPORT

A 58-year-old man visited our hospital on March 14, 1989 with the complaint of anterior chest oppression lasting approximately 1 h occurring in the night one week prior to the visit. He had no history of smoking, diabetes mellitus, hypertension or hyperlipidemia. On physical examination, the pulse was 72/min and irregular, and the blood pressure was 108/70 mmHg. Thoracic auscultation revealed no heart murmur and no extra-heart sound. Other physical findings, including a neurological examination, were normal. The chest X-ray showed mild cardiomegaly (CTR 53%) and the ECG showed an abnormal Q wave, ST segment elevation and coronary T wave in II, III, aV_F, high R wave in V_2-3, and ST segment depression in V_2-6. These ECG findings were compatible with acute myocardial infarction (infero-posterior). On admission, the laboratory data showed elevation of the erythrocyte sedimentation rate (20 mm/h), positive CRP and elevation of LDH (1409 IU/l) and CPK (162 IU/l), the peak of which was just after admission.

As one week had already elapsed after the onset of acute myocardial infarction, the patient was treated with conservative therapy without acute phase coronary arteriography. He had no complications except for sporadic monofocal premature ventricular contractions.

Echocardiographic study showed hypokinesis, an increase in the density of the left ventricular posterior wall, and a moderate amount of pericardial effusion. Moreover, an elliptical and mobile tumor attached to the interatrial septum was found in the left atrium (Fig. 1).

On cardiac catheterization performed on the 23rd hospital day, left ventriculogram showed akinesis of the infero-posterior wall, 63% of the left ventricular ejection fraction and aneurysm formation at the membranous portion of the interventricular septum. A left atrial tumor was confirmed by the levophase of the pulmonary arteriography (Fig. 2). Coronary arteriography showed no abnormality of the left coronary artery, but a tumor brush from the atrioventricular branch of the right coronary artery was observed. It is noteworthy that the saccular aneurysm formation occurred just below the 50%
Fig. 1. Echocardiography (upper panel: two dimensional; left, end-diastole; right, end-systole, lower panel: M-mode) shows hypokinesis, an increase in the density of the left ventricular posterior wall, and a moderate amount of pericardial effusion. Moreover, an elliptical and mobile tumor (arrow) attached to the interatrial septum can be seen in the left atrium.

Fig. 2. Left ventriculogram (upper panel: RAO view, middle panel: LAO view. Left: end-diastole, right: end-systole) shows akinesis of infero-posterior wall and aneurysm formation (white arrow) at the membranous portion of the interventricular septum. Left atrial tumor (black arrow) is confirmed by the levophase of pulmonary arteriography (lower panel).

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Fig. 3. The intracoronary ergonovine provocation test did not show any significant spasm of either coronary artery.

On the 35th hospital day, surgery was performed for the removal of the tumor in the left atrium. Histologically, the tumor was consistent with the findings of a myxoma. Aneurysm formation of the interventricular septum was due to closure of the ventricular septal defect caused by adhesion of the septal cusp of the tricuspid valve. The post-operative course was uneventful.

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Fig. 3. Coronary arteriography (upper panel: RAO view, middle panel: LAO view, lower panel: lateral view) shows the saccular aneurysm formation (arrow) just below the 50% stenotic portion of segment 1 of the right coronary artery.

**DISCUSSION**

Cardiac myxoma, the most frequent benign primary cardiac tumor, comprises 30 to 50% of all primary cardiac tumors (1, 2). Fifty to 80% of these tumors occur at the left atrium (3–6). The first symptoms frequently originate from the embolic complications of the cerebral or other peripheral arteries.

However, acute coronary embolism or myocardial infarction complicated with left atrial myxoma is rare, and only 21 cases have been reported in the literature as far as we know. Among these 21 cases, only 8 cases underwent selective coronary angiography and were diagnosed during their lifetime (7–14) (Table 1). Their ages ranged from 11 to 55 years old, and the common site of myocardial infarction was the inferior area. Although some cases were considered to simply be accidental coincidence of the left atrial myxoma and acute myocardial infarction (12, 13), it is noteworthy that both stenosis and aneurysmal dilatation of coronary artery were found in 2 cases which underwent the coronary angiography more than one month after the onset of acute myocardial infarction. The present case, also, had 50% stenosis and aneurysmal findings of the right coronary artery. The characteristics of the embolic manifestation from the cardiac myxoma is that 1) the fragment of the tumor emboli forms the foci of metastasis at the embolization site, and 2) the emboli, once lodged in the arteries, subsequently penetrates the endothelium and invades the vessel wall itself, where it continues to grow. There it causes destruction to the wall leading to aneurysmal formation, and gives rise to intravascular masses of varying size (15). These changes have already been reported in the cerebral arteries not only by the histopathological findings but also by cerebral arteriographic findings (16, 17).

On the other hand, there have also been reports on saccular coronary arterial aneurysms (18–21) and the various factors which contribute to aneurysmal formation including arteriosclerosis, trauma, intervention by percutaneous transluminal angioplasty, periarteritis nodosa and mucocutaneous lymph node syndrome. The present case had no previous history of illness and had few risk factors for ischemic heart disease. Saccular aneurysms due to arteriosclerosis are very rare and are generally accompanied by severe obstructive multiple lesions (19). This case had no stenotic lesions other than the mild stenotic lesion of segment 1 of the right coronary artery.

These findings taken together, it can be proposed that the myocardial infarction in this case was likely due to the coronary embolization from the left atrial myxoma. In addition, the negative results of the ergonovine provocation test, also, support this hypothesis.

Although there have been a few reports on the association of atrial septal defect and left atrial myxoma (22, 23), an association with ventricular septal defect has not been reported. The rarity of the coexistence of ventricular septal defect and left atrial myxoma, and the present surgical findings indicate that this combination is a clinical coincidence rather than a distinct clinical syndrome.
Table 1. Reported cases of left atrial myxoma with acute myocardial infarction.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Age</th>
<th>Sex</th>
<th>Site of MI</th>
<th>CAG findings</th>
<th>Interval from MI to CAG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tanabe et al (7)</td>
<td>11</td>
<td>M</td>
<td>inferior RCA</td>
<td>RCA filling defect</td>
<td>10 days</td>
</tr>
<tr>
<td>Balk et al (8)</td>
<td>29</td>
<td>M</td>
<td>anterior LAD, LCx</td>
<td>LAD, LCx stenosis, aneurysmal dilatation</td>
<td>3 months</td>
</tr>
<tr>
<td>Rath et al (9)</td>
<td>55</td>
<td>M</td>
<td>inferior RCA</td>
<td>RCA filling defect</td>
<td>&lt;10 days</td>
</tr>
<tr>
<td>Lehrman et al (10)</td>
<td>43</td>
<td>M</td>
<td>anterior LAD</td>
<td>LAD sharp cut-off</td>
<td>2 weeks</td>
</tr>
<tr>
<td>Tatsukawa et al (11)</td>
<td>42</td>
<td>F</td>
<td>anterior LAD</td>
<td>LAD stenosis, aneurysmal dilatation</td>
<td>32 days</td>
</tr>
<tr>
<td>Hoad et al (12)</td>
<td>68</td>
<td>F</td>
<td>inferior LAD, LCx, RCA</td>
<td>LAD stenosis</td>
<td>2 months</td>
</tr>
<tr>
<td>Usui et al (13)</td>
<td>52</td>
<td>M</td>
<td>inferior RCA</td>
<td>RCA stenosis</td>
<td>?</td>
</tr>
<tr>
<td>Doi et al (14)</td>
<td>34</td>
<td>M</td>
<td>inferior normal</td>
<td></td>
<td>18 days</td>
</tr>
</tbody>
</table>

MI, myocardial infarction; CAG, coronary arteriography; RCA, right coronary artery; LAD, left anterior descending artery; LCx, left circumflex artery

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