Manifestation of latent left ventricular outflow tract obstruction caused by acute myocardial infarction: An important complication of acute myocardial infarction

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A B S T R A C T

Background: Although transient left ventricular outflow tract (LVOT) obstruction is reported as a complication with acute myocardial infarction (AMI), the mechanisms and features of LVOT obstruction in AMI are unclear.

Methods and results: Herein, we present two cases of transient LVOT obstruction with anteroseptal AMI. The mechanisms and features of the appearance of LVOT obstruction in AMI are unclear. We report two cases of transient LVOT obstruction in AMI that reoccurred with dobutamine (DOB) provocation in the chronic phase. We consider the mechanisms and features of LVOT obstruction in AMI with these two cases in addition to another case which we have previously reported [9].

Conclusions: Latent LVOT obstruction would be manifested in the acute phase of AMI. 1-VD of LAD and the maintenance of major SB blood flow are important factors with respect to the manifestation of latent LVOT obstruction.

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Introduction

Dynamic left ventricular outflow tract (LVOT) obstruction is a rare complication in acute myocardial infarction (AMI), and about 20 cases have been reported to date [1–10]. While LVOT obstruction is a potentially reversible complication of AMI, some patients with transient LVOT obstruction during AMI have experienced myocardial rupture and died [3,6]. The mechanisms and features of the appearance of LVOT obstruction in AMI are unclear. We report two cases of transient LVOT obstruction in AMI that reoccurred with dobutamine (DOB) provocation in the chronic phase. We consider the mechanisms and features of LVOT obstruction in AMI with these two cases in addition to another case which we have previously reported [9].

Case 1

A 53-year-old man with hypertension (HT) was admitted to our hospital for severe chest pain. His blood pressure (BP) was 180/110 mmHg, and heart rate (HR) was 95 beats/min. A grade 2/6 ejection systolic murmur was detected at the apex. Electrocardiogram (ECG) revealed sinus rhythm (80 beats/min) and ST elevation in the precordial leads (Fig. 1A). Transthoracic echocardiogram (TTE) showed anterior–apical hypokinesis, compensatory hyperkinesis of other segments including basal intraventricular septum and a sigmoid-shaped septum with a diminished aorto-septal angle of 89° (Fig. 1B1). This examination also demonstrated a peak Doppler velocity of 3.9 m/s corresponding to a peak pressure gradient (PG) of 61 mmHg across the LVOT (Fig. 1B2). Systolic anterior movement of mitral valve (SAM) and mild level of mitral regurgitation (MR) were noted. There were no findings of left ventricular hypertrophy (LVH). Emergent coronary angiography (CAG) was immediately performed and revealed one-vessel coronary heart disease (1-VD) with total occlusion of the mid-left anterior descending artery (LAD) (Fig. 1C1). The blood flow of the major septal branch (SB) was maintained because the LAD was occluded distal from the branching point of the major SB. Direct percutaneous coronary intervention (PCI) was performed for the occluded LAD. A metal stent was implanted into the occluded lesion, and recanalization was achieved (Fig. 1C2). Left ventriculography (LVG) demonstrated anteroseptal–apical akinesia and hypercontractility of other regions (Fig. 1C3). His peak creatine
phosphokinase (CK) level was 1022 IU/L. On the next day, his ejection systolic murmur was diminished, and TTE revealed complete resolution of the LVOT obstruction. After completion of cardiac rehabilitation without any complications, exercise stress TTE (ExTTE) using the Bruce protocol was performed to examine the LVOT obstruction. The PG across the LVOT was 5 mmHg at rest, and a PG of 66 mmHg was evoked by Bruce test with 8 min 00 s of exercise. Therefore, he was treated with atenolol (25 mg/day), and follow-up ExTTE revealed a PG 14 mmHg PG after provocation using the Bruce test with 9 min 00 s of exercise. He was discharged a few days later.

After 1 year, he returned to our hospital for follow-up cardiac catheterization with no symptoms. Restenosis of the LAD lesion was not apparent by CAG. There was no PG between the left ventricular apex and the ascending aorta following interruption of atenolol administration, and intravenous infusion of DOB (15 μg/kg/min) increased the PG to 110 mmHg (Fig. 1D). BP and HR before and after DOB infusion were 170/96 mmHg and 60 beats/min, 168/82 mmHg

Fig. 1. (A) Electrocardiogram upon admission in Case 1. ST elevation was shown in the precordial leads. (B1) Transesophageal echocardiography findings upon admission in Case 1. Parasternal long-axis view of two-dimensional echocardiography showed a sigmoid-shaped septum with diminished aorto-septal angle of 89°. There were no indications of left ventricular hypertrophy. (B2) Spectral imaging of continuous wave Doppler of the left ventricular outflow tract from the apical position revealed the peak velocity value of 3.92 m/s indicating a peak gradient of 61 mmHg. (C) The findings of coronary angiography (CAG) in the right anterior oblique (RAO) cranial view (1), RAO view (2), and left ventriculography (LVG) in the RAO view in Case 1. Emergent CAG revealed one-vessel coronary heart disease with total occlusion of the mid left anterior descending artery (LAD) (arrow) and blood flow maintained in the major septal branch (1). A metal stent was implanted into the occluded lesion and recanalization of the LAD was achieved (2). LVG findings, in diastole (3, left) and systole (3, right), revealed anteroseptal–apical akinesia (arrows in 3, right) and hypercontractility of other regions. (D) The findings of the left ventricular apex (LVA) and the ascending aorta (AAo) pressure before (left) and after (right) dobutamine (DOB) infusion in Case 1. DOB infusion provoked a 110 mmHg pressure gradient between the LVA and the AAo.
and 75 beats/min, respectively. Thereafter, he continued to take atenolol for latent LVOT obstruction.

**Case 2**

A 61-year-old man with HT and dyslipidemia was admitted to our hospital for chest pain with cold sweating. His BP was 168/96 mmHg, and HR was 80 beats/min. A grade 3/6 ejection systolic murmur was detected at the apex. ECG revealed sinus rhythm (70 beats/min) and ST elevation in the precordial leads with incomplete right bundle branch block (Fig. 2A). TTE showed apical hypokinesis, hyperkinesis of other segments, and protrusion of the basal part of the intraventricular septum with an aorto-septal angle of 88° (Fig. 2B1). This examination also demonstrated a peak Doppler velocity of 5.0 m/s corresponding to a peak PG of 101 mmHg across the LVOT (Fig. 2B2). There were SAM and moderate level of MR. The LV wall thickness was within normal limits. Emergent CAG revealed 1-VD with total occlusion of the mid LAD and maintenance of blood flow via the major SB (Fig. 2C1). Direct PCI was performed, and recanalization of LAD was achieved with implantation of a metal stent to the occluded lesion (Fig. 2C2). LVG demonstrated apical akinesia and hypercontractility of other regions (Fig. 2C3). His peak CK level was 6083 IU/L. On the next day, his ejection systolic murmur was diminished, and TTE revealed complete resolution of the LVOT obstruction. After completion of cardiac rehabilitation without complications, ExTTE

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**Fig. 2.** (A) Electrocardiogram (ECG) upon admission in Case 2. The ECG showed ST elevation in the precordial leads and incomplete right bundle branch block. (B) Transesophageal echocardiography findings upon admission in Case 2. Parasternal long-axis view of two-dimensional echocardiography showed apical hypokinesis and protrusion of the basal part of the intraventricular septum with an aorto-septal angle of 88° (1). There were no indications of left ventricular hypertrophy. Spectral imaging of continuous wave Doppler of the left ventricular outflow tract from the apical position revealed the peak velocity value of 5.0 m/s indicating a peak pressure gradient of 101 mmHg (2). (C) The findings of coronary angiography (CAG) in the right anterior oblique (RAO) cranial view (1), RAO view (2) and left ventriculography (LVG) in the RAO view in Case 2. Emergent CAG revealed one-vessel coronary heart disease with total occlusion of the mid left anterior descending artery (LAD) (arrow) and maintained the blood flow in the major septal branch (1). A metal stent was implanted into the occluded lesion and recanalization of the LAD was achieved (2). LVG findings, in diastole (3, left) and systole (3, right), revealed apical akinesia (arrows in 3, right) and hypercontractility of other regions. (D) The findings of the left ventricular apex (LVA) and the ascending aorta (AAo) pressure before (left) and after (right) dobutamine (DOB) infusion in Case 2. DOB infusion provoked a 110 mmHg pressure gradient between the LVA and the AAo.
using the Bruce protocol was performed to examine the LVOT obstruction. The PG across the LVOT was 14 mmHg at rest, and a PG of 148 mmHg was evoked by the Bruce protocol testing with 6 min 00 s of exercise, resulting in shortness of breath. Therefore, he was treated with atenolol (25 mg/day), and follow-up ExTTE revealed a PG of 24 mmHg following provocation by Bruce test with 7 min 30 s of exercise. He was discharged a few days later.

After 1 year, he returned to our hospital for follow-up cardiac catheterization with no symptoms. Restenosis of LAD lesion was not apparent by CAG. There was no PG between the left ventricular apex and the ascending aorta following interruption of atenolol administration, and intravenous infusion of DOB (15 µg/kg/min) increased the PG to 110 mmHg (Fig. 2D). BP and HR before and after DOB infusion were 132/76 mmHg and 75 beats/min, 126/66 mmHg and 107 beats/min, respectively. He continued to take atenolol for latent LVOT obstruction thereafter.

**Discussion**

Herein, we report two cases of transient LVOT obstruction in the acute phase of aorto-septal AMI. Twenty cases of transient LVOT obstruction with AMI have been reported to date [1–10] (Table 1). Seven of these 20 patients (35%) had 1-VD of LAD. However, seven cases (35%) did not have LAD disease, and the remaining six patients (30%) did not undergo CAG. Reperfusion therapy was performed in seven patients (35%). There were some cases in which cardiogenic enzymes did not increase or were not measured. A few cases have referred to the findings of the longitudinal para-sternal view of TTE. Therefore, there is the potential that these 20 cases could include patients without aorto-septal AMI, e.g., those with Takotsubo cardiomyopathy. We cannot consider the mechanisms and features of dynamic LVOT obstruction in AMI from these cases.

In this report, we describe the features of LVOT obstruction in AMI; two cases that are reported herein, and another that has been reported previously [9]. First, all cases were 1-VD of LAD. This is one of the important factors causing LVOT obstruction, as has been reported previously [2,7]. Aorto-septal and apical akininesis with compensatory hyperdynamic contraction of the residual normally perfused basal segments is shown in aorto-septal AMI, and this compensatory basal hyperkinesis narrows the systolic LVOT, resulting in the acceleration of blood flow through the LVOT [9]. This would create Ventri effect and occur SAM, resulting in further LVOT obstruction in both Cases 1 and 2 [4].

Next, the major SB arose from the LAD, proximal to the occlusion site, and the blood flow via the major SB was maintained in all three cases. Percutaneous transluminal septal myocardial ablation (PTSMA) is effective in reducing the PG of the LVOT in patients with HOCM [11]. PTSMA is achieved through the creation of permanent septal necrosis by injecting alcohol into the SBs supplying the myocardium of the basal septum. Therefore, the maintenance of blood flow through the major SB would be indispensable to cause LVOT obstruction in AMI. Unfortunately, there are few reports describing the major SB in these reports [1–8,10].

Finally, in the subacute phase of AMI with disappearance of the resting LVOT obstruction, exercise stress test provoked LVOT PG in these two cases. Moreover, DOB infusion revealed marked LVOT obstruction 1 year after the onset of AMI (Figs. 1D and 2D). We previously reported one case in which DOB provocation caused a LVOT PG of 30 mmHg during the subacute phase of AMI with disappearance of the LVOT obstruction, as in these two cases [9]. LVOT obstruction can be induced by stressors, such as exercise or DOB infusion, in patients with latent LVOT obstruction who do not exhibit LVOT obstruction at rest [12]. Therefore, the above-mentioned three cases (including these two current cases and the previously reported case [9]) were thought to have latent LVOT obstruction, and the occurrence of aorto-septal AMI would manifest as LVOT obstruction in the acute phase. Although no significant LVH was observed in those three cases, a lower aorto-septal angle was revealed in the TTE findings. The aorto-septal angle was reported to be smaller in the cases with sigmoid-shaped septum compared with normal controls, 93 ± 14° vs. 145 ± 7°, respectively [13]. The degree of aorto-septal angle correlated negatively with the LVOT PG provoked by DOB infusion in the latent LVOT obstruction patients [14]. In general, a sigmoid-shaped septum is considered to be a normal part of the aging process and to have little clinical and pathophysiologic importance [15,16]. Several reports have demonstrated the clinical importance of a sigmoid-shaped septum as a potential cause of LVOT obstruction [9,17–24]. The lower aorto-septal angle in these three cases might be strongly related to the cause of latent LVOT obstruction.

**Table 1**

Reported cases of left ventricular outflow tract obstruction in acute myocardial infarction.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age</th>
<th>Sex</th>
<th>CAG</th>
<th>PCI or PTCR</th>
<th>Max CK (IU/L)</th>
<th>Severe prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Armstrong et al. [1]</td>
<td>78</td>
<td>F</td>
<td>Not performed</td>
<td>N</td>
<td>Normal</td>
<td>Dead, malignancy</td>
</tr>
<tr>
<td>Joffe et al. [2]</td>
<td>79</td>
<td>F</td>
<td>@6, 90%</td>
<td>Y</td>
<td>272</td>
<td></td>
</tr>
<tr>
<td>Bartunek et al. [3]</td>
<td>51</td>
<td>M</td>
<td>@7, 100%; @9, 50%</td>
<td>Y (failure)</td>
<td>1734</td>
<td></td>
</tr>
<tr>
<td>Bartunek et al. [3]</td>
<td>72</td>
<td>F</td>
<td>Not performed</td>
<td>N</td>
<td>Moderate size MI</td>
<td></td>
</tr>
<tr>
<td>Hartley et al. [4]</td>
<td>71</td>
<td>F</td>
<td>Normal</td>
<td>N</td>
<td>883</td>
<td></td>
</tr>
<tr>
<td>San Roman Sanchez et al.</td>
<td>70</td>
<td>M</td>
<td>Mid-LAD 80%, D1 90%, OM 90%</td>
<td>Y (failure)</td>
<td>985</td>
<td>Dead, VF</td>
</tr>
<tr>
<td>Hrovat et al. [6]</td>
<td>55</td>
<td>F</td>
<td>Not significant lesion</td>
<td>N</td>
<td>724</td>
<td></td>
</tr>
<tr>
<td>Ozaki et al. [9]</td>
<td>70</td>
<td>F</td>
<td>Not performed</td>
<td>N</td>
<td>Two-fold increment</td>
<td>Dead, rupture</td>
</tr>
<tr>
<td>Möller et al. [10]</td>
<td>74</td>
<td>F</td>
<td>@7, 100%</td>
<td>Y</td>
<td>1775</td>
<td></td>
</tr>
<tr>
<td>Hollander et al. [11]</td>
<td>76</td>
<td>M</td>
<td>LAD acute in-stent thrombosis</td>
<td>Y</td>
<td>1751</td>
<td></td>
</tr>
</tbody>
</table>

F: female; M: male; CAG: coronary angiography; LCCX: left circumflex coronary artery; LAD: left anterior descending coronary artery; D1: first diagonal branch; OM: obtuse marginal branch; PCI: percutaneous coronary intervention; PTCR: percutaneous transluminal coronary recanalization; N: no; Y: yes; CK: creatine kinase; MI: myocardial infarction; VSP: ventricular septal perforation.
After the diagnosis of latent LVOT obstruction, each of these three cases underwent treatment with beta-blockers. The effectiveness of beta-blockers to relieve LVOT obstruction with HCM is well known, and their effectiveness to reduce LVOT PG due to a sigmoid-shaped septum has been reported [21,24]. In the two cases reported herein, administration of a beta-blocker improved the LVOT obstruction that was provoked by exercise. Moreover, beta-blockers should be administered to patients with AMI, when they are not contraindicated [25]. Therefore, beta-blocker administration is the first-line medication for patients with LVOT obstruction complicated with AMI. In our three patients, there have been no clinical events for at least 3 years since the onset of AMI following administration of the beta-blocker.

The present two cases demonstrate that the occurrence of anteroseptal AMI would manifest as latent LVOT obstruction. 1-VD of LAD and the maintenance of major SB blood flow are important factors with respect to the manifestation of latent LVOT obstruction. Furthermore, the sigmoid-shaped septum might be related to the development of latent LVOT obstruction in these present cases.

Limitations

This study was based upon a small number of affected individuals. More studies are needed to elucidate the mechanism of LVOT obstruction with AMI.

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Conflict of interest

The authors declare that there is no conflict of interest.

References