A Case Report of Acute Myocardial Infarction Induced by Coronary Spasm

Intravascular Findings

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SUMMARY

A 53-year-old male complaining of chest pain was admitted to our hospital with suspected acute myocardial infarction (AMI). Emergent coronary angiography (CAG) determined a totally occluded middle right coronary artery (RCA). Thrombus aspiration was conducted, followed by intravascular ultrasound (IVUS) imaging. Diffuse intima plus media thickness was identified at the obstruction site and a thrombus was observed proximally to the occlusion site on IVUS. Following isosorbide dinitrate (ISDN) administration, dilatation of the RCA was confirmed. IVUS study indicated the luminal dilatation was achieved by the release of the diffuse intima plus media thickening. Of note, plaque volume showed no significant difference after administration of ISDN at any vessel site. These results clearly show that luminal dilatation and vessel dilatation were achieved from the redistribution of plaque volume (intima plus media). A follow-up CAG showed no significant stenosis in the RCA. After a provocation test using methylergometrine maleate, the RCA was totally occluded at the very site of the initial event. The involvement of vasospasm as a cause of AMI in the present case was doubly confirmed with characteristic IVUS images of vasospasm in the acute phase and with a provocation test at follow-up. (Int Heart J 2013; 54: 237-239)

Key words: Intravascular ultrasound, Intima and media thickening

Coronary artery vasospasm has been shown to play an important role in the pathogenesis of ischemic heart disease, variant angina, myocardial infarction, and ventricular arrhythmia. Previous studies have shown that the etiology of spasm is due to transient abnormal or hypersensitive response of vascular smooth muscle to various stimuli and that the atherosclerosis is invariably present at the site of focal vasospasm. Previous intravascular ultrasound (IVUS) and optical coherence tomography (OCT) studies have reported the morphological features of vasospastic lesions. However, most of the data were obtained in a spastic lesion that was artificially induced by the administration of a vasoconstrictor (acetylcholine or ergonovine). In the present report, we report IVUS images from a patient with ST elevation type acute myocardial infarction (STEMI) due to vasospasm.

CASE REPORT

A 53-year-old male complaining of chest pain at rest was admitted to our hospital. The patient had experienced 2-3 minutes of similar episodes once a month for 2 to 3 years. This time, his chest pain had lasted for more than 3 hours prior to the admission. Laboratory data showed a white blood cell count of 14460/mm³, creatine kinase (CK) of 3075 IU/L, CK-MB of 86.9 IU/L, and troponin-I of 16.2 ng/mL. An electrocardiogram (ECG) showed ST elevation in the II, III, and aVF leads and ST depression in the aVL lead. The data indicated an acute myocardial infarction. Emergent coronary catheterization was performed.

Emergent coronary angiography showed a totally occluded RCA (Figure 1A). Immediate percutaneous coronary intervention (PCI) for the RCA was conducted. After a guide wire crossed the lesion, a thrombectomy was performed. Following thrombus aspiration, an IVUS catheter (View It, Terumo Co., Tokyo, Japan) was inserted to visualize the characteristics of the lesion (Figure 1B and a,b,c). The IVUS findings indicated the luminal dilatation was achieved by the release of the diffuse intima plus media thickening. The residual thrombus was still observed proximal to the obstruction site by IVUS imaging. A thrombus was observed proximal to the obstruction site. Five mg of ISDN was injected into the RCA. Following administration of 5 mg ISDN, dilatation of the RCA was confirmed by both coronary angiography and IVUS (Figure 2A and a,b,c). The IVUS findings indicated the luminal dilatation was achieved by the release of the diffuse intima plus media thickening. The residual thrombus was still observed proximal to the obstruction site by IVUS imaging.

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the obstruction site. The residual intimal thickness of the vessel surface was also observed at this site.

Based on these images, we concluded that stent implantation was not necessary and finished PCI after only thrombectomy (Figure 2B).

A follow-up angiography was performed 2 weeks after emergent PCI. Baseline coronary angiography showed no significant stenosis in the right coronary artery. B: After a provocation test using ergonovine, total occlusion of the RCA was triggered at the very site of the occlusion site in the initial event.

## Table. Comparison of IVUS Findings Between Control and Post ISDN Administration Using Volumetric Analysis

<table>
<thead>
<tr>
<th>Component (mm³)</th>
<th>Control proximal</th>
<th>mid</th>
<th>distal</th>
</tr>
</thead>
<tbody>
<tr>
<td>lumen volume</td>
<td>58.0</td>
<td>30.6</td>
<td>22.0</td>
</tr>
<tr>
<td>vessel volume</td>
<td>175.3</td>
<td>89.1</td>
<td>83.2</td>
</tr>
<tr>
<td>plaque volume</td>
<td>117.3</td>
<td>58.5</td>
<td>61.2</td>
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</table>

<table>
<thead>
<tr>
<th>Component (mm³)</th>
<th>Post ISDN administration proximal</th>
<th>mid</th>
<th>distal</th>
</tr>
</thead>
<tbody>
<tr>
<td>lumen volume</td>
<td>65.0</td>
<td>142.2</td>
<td>122.8</td>
</tr>
<tr>
<td>vessel volume</td>
<td>180.6</td>
<td>203.1</td>
<td>191.3</td>
</tr>
<tr>
<td>plaque volume</td>
<td>115.6</td>
<td>60.9</td>
<td>68.6</td>
</tr>
</tbody>
</table>

The volume of each vascular component was calculated as a sum of the cross-sectional area of each vascular component from 20 consecutive IVUS images. The pull back speed was 0.5mm per second.

The obstruction site. The residual intimal thickness of the vessel surface was also observed at this site.

Based on these images, we concluded that stent implantation was not necessary and finished PCI after only thrombectomy (Figure 2B).

A follow-up angiography was performed 2 weeks after emergent PCI. Baseline coronary angiography showed no significant stenosis in the RCA (Figure 3A). After a provocation test using ergonovine (48 µg intracoronary injection), total occlusion of the RCA was triggered at the very site of the initial occlusion (Figure 3B).

A comparison of the IVUS findings both before and after ISDN administration is presented in the Table. The IVUS images of the coronary artery before ISDN administration showed diffuse intima plus media thickness at the obstruction site. In contrast, after ISDN administration, the vascular lumen was dilated and intimal shrinkage was released. However, intima plus media thickness was still observed at the site of a high echoic area on the intimal surface. This segment was thought to be the place where the damage to endothelial function was more profound.

We further performed a volumetric analysis of IVUS data and compared it to before and after ISDN administration. The distal site showed more robust changes in luminal and vessel volumes compared to those of the proximal site after the administration of ISDN. Of note, the plaque volume (intima plus...
media) showed no significant difference after the administration of ISDN at any vessel site.

**Discussion**

In this report, we present IVUS images from a STEMI case caused by coronary spasm and confirmed the diagnosis of STEMI with a provocation test conducted during follow-up. To the best of our knowledge, this report is the first to describe IVUS findings from a STEMI case caused by coronary spasm.

Previous studies demonstrated that coronary spasm is caused by local hyper-reactivity to a generalized constrictor stimulus on the vessel. The characteristics of the lesion that is the culprit of coronary spasm are the existence of minimal atherosclerosis, with relatively low-echo intensity, less calcification, and negative remodeling on IVUS images. 

In our case, the IVUS image at the occlusion site shared some of the above previously reported IVUS characteristics of a spasm-induced vessel site: minimal atherosclerosis and less calcification.

On top of the above findings, the IVUS images did not demonstrate plaque rupture, which is a common finding at the culprit lesion in patients with acute coronary syndrome, although there was an image of a thrombus at the proximal site of the coronary spasm. Furthermore, the IVUS image at the same site showed diffuse intima plus media thickening immediately after thrombectomy, which disappeared after ISDN administration. These IVUS findings corresponded to the OCT findings from a previous case report of acute coronary syndrome caused by coronary spasm. Additionally, the laboratory data (CK-MB, Troponin-I, etc) and ECG findings on admission without other suspicious coronary lesion support that this case was an acute coronary syndrome (STEMI in this case) caused by coronary spasm at this site.

Volumetric analysis of IVUS clarifies that dilatation of the lumen and vessel by intracoronary administration of ISDN was obtained without reduction of total plaque volume. (There was hardly any plaque in the patient’s coronary artery. Therefore, intima plus media volume account for most of the plaque volume.) Tanaka, et al in an OCT study reported that intimal thickness and medial thickness are significantly different between the time of a provocation test and after nitroglycerine administration only in patients with coronary spasm. Furthermore, they concluded that luminal narrowing during spasm is associated with intimal gathering without alteration of intimal area. Taken together, the result of volumetric analysis in our study strongly supports the mechanism of coronary spasm, in other words, coronary spasm is caused by a change in the distribution of intimal and media volume. These results clearly show that the mechanism of luminal and vessel dilatation was achieved from the redistribution of plaque volume, in other words, the composite of intima and media.

In addition, this case confirmed that the characteristics of coronary spasm that leads to acute coronary syndrome are similar to those of coronary spasm that is caused by a provocation test. We believe that this case report provides clinically important and educational information.

**Conclusion:** The characteristic IVUS images of vasospasm were observed in a patient with acute myocardial infarction. The involvement of vasospasm as a cause of acute myocardial infarction in the present case was doubly confirmed with the characteristic IVUS images of vasospasm in acute phase and with a provocation test at follow-up.

**References**