CASE REPORT

Perfusion Balloon for the Treatment of Very Late Stent Thrombosis

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Summary

A method to manage ST-segment elevated myocardial infarction (STEMI) caused by very late stent thrombosis (VLST) has yet to be established. In this case series, we present several cases of STEMI caused by VLST, which were successfully revascularized using a perfusion balloon. Since the perfusion balloon (Ryusei: Kaneka Medix Corporation, Osaka, Japan) has the unique advantage of maintaining blood flow during balloon inflation, we can keep dilating the target lesion for more than several minutes. Extended inflation might work to prevent acute recoil, and to achieve optimal expansion without an additional stent. Our case series may provide a reasonable option for the treatment of VLST.

Key words: ST-segment elevated myocardial infarction, Percutaneous coronary intervention

Recently, percutaneous coronary intervention (PCI) has been developed with the use of new devices and imaging modalities.1,2 However, some complications still occur during contemporary PCI. Very late stent thrombosis (VLST) is a rare but life-threatening complication after stent deployment.3,4 VLST can result in acute coronary syndrome, especially ST-elevated myocardial infarction (STEMI).5 However, a treatment strategy for STEMI caused by VLST has not been established. The perfusion balloon we used (Ryusei: Kaneka Medix Corporation, Osaka, Japan) is a unique balloon that has a coil reinforcement layer within the inner shaft inside the balloon. The coil reinforcement layer preserves blood perfusion during balloon inflation. Perfusion balloons have been used as a bailout device in cases of coronary perforation or occlusive dissections.6 In this case series, we present 2 cases of STEMI caused by VLST, which were successfully treated using a perfusion balloon.

Case Report

In our institution, 8 cases of STEMI caused by VLST were treated with a perfusion balloon between July 2016 and June 2020. A summary of the 8 cases is shown in Table I. All procedures were successfully completed without deploying additional stents. The target vessels were the right coronary artery (RCA) (n = 5), left anterior descending artery (LAD) (n = 2), and left circumflex artery (LCX) (n = 1). We used a drug-coated balloon in 1 case. The thrombosis related stents were a bare metal stent (BMS) (n = 2), 1st generation drug-eluting stents (DES) (n = 2), and 2nd generation DES (n = 4). All cases presented as Killip class 1, and there were no in-hospital deaths. We describe here 2 representative cases.

Case 1: A 77-year-old man with a history of inferior myocardial infarction was transferred to our medical center because of chest pain. A BMS had been deployed to the RCA 12 years earlier. His antplatelet therapy was single (aspirin) at the onset of VLST. He was diagnosed with STEMI and underwent emergent coronary angiography, which revealed total occlusion in the stent site of the proximal RCA (Figure 1A). We performed PCI to the occluded RCA. A 7-Fr JR 4.0 SH guide catheter was inserted via the right radial artery. We advanced a conventional 0.014 inch guidewire, and obtained TIMI-3 flow after balloon dilatation using a 2.0 mm semi-compliant balloon (Figure 1B). Since the IVUS revealed thrombus and low echoic plaque in the previous stent (Figure 1C), we selected a long inflation using a perfusion balloon. We delivered a 3.5-mm Ryusei balloon catheter to the lesion, and kept inflating for 2 minutes × 3 (max 6 atm) (Figure 1D). Immediately after long inflation, the lesion was successfully revascularized without acute re-coil (Figure 1E). A follow-up angiogram after 8 months revealed good flow without restenosis (Figure 1F). We continued dual anti-platelet therapy (aspirin and clopidogrel) until the follow-up angiogram, and switched to single antiplatelet therapy (clopidogrel) after the follow-up angiogram.

Case 2: A 77-year-old man with a history of inferior myocardial infarction was referred to our medical center because of chest pain and bradycardia. A BMS had been deployed to the RCA 12 years previously. His antplatelet therapy was single (aspirin) at the onset of VLST. He was diagnosed with STEMI complicated with complete atriov-
**Table 1.** Clinical and Procedural Characteristics of Our 8 Cases

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Target lesion</th>
<th>Age</th>
<th>Gender</th>
<th>Perfusion Balloon diameter (mm)</th>
<th>Inflation pressure (atm)</th>
<th>Inflation time (seconds)</th>
<th>DCB use</th>
<th>Duration from stent deployment (months)</th>
<th>Anti-platelet therapy (preprocedure)</th>
<th>Killip classification</th>
<th>In-hospital death</th>
<th>ID-TVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RCA proximal</td>
<td>77</td>
<td>Male</td>
<td>3.5</td>
<td>12</td>
<td>360</td>
<td>No</td>
<td>BMS</td>
<td>141</td>
<td>SAPT</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>RCA mid</td>
<td>77</td>
<td>Male</td>
<td>3.0</td>
<td>8</td>
<td>600</td>
<td>No</td>
<td>BMS</td>
<td>145</td>
<td>SAPT</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>LAD proximal</td>
<td>53</td>
<td>Male</td>
<td>3.0</td>
<td>6</td>
<td>120</td>
<td>Yes</td>
<td>R-ZES</td>
<td>33</td>
<td>DAPT</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>RCA proximal</td>
<td>70</td>
<td>Male</td>
<td>2.5</td>
<td>6</td>
<td>300</td>
<td>No</td>
<td>R-ZES</td>
<td>81</td>
<td>SAPT</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>LAD proximal</td>
<td>82</td>
<td>Male</td>
<td>2.5</td>
<td>6</td>
<td>180</td>
<td>No</td>
<td>BP-EES</td>
<td>14</td>
<td>SAPT</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>RCA proximal</td>
<td>75</td>
<td>Male</td>
<td>3.0</td>
<td>10</td>
<td>300</td>
<td>No</td>
<td>EES</td>
<td>39</td>
<td>DAPT</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>LCX proximal</td>
<td>76</td>
<td>Male</td>
<td>3.0</td>
<td>6</td>
<td>70</td>
<td>No</td>
<td>PES</td>
<td>117</td>
<td>SAPT</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>8</td>
<td>RCA distal</td>
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<td>Male</td>
<td>3.0</td>
<td>6</td>
<td>120</td>
<td>No</td>
<td>PES</td>
<td>102</td>
<td>SAPT</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

LAD indicates left anterior descending artery; RCA, right coronary artery; LCX, left circumflex artery; DCB, drug coated balloon; BP-EES, biodegradable-polymer everolimus-eluting stent; EES, everolimus-eluting stent; ZES, zotarolimus-eluting stent; PES, paclitaxel-eluting stent; BMS, bare metal stent; DAPT, dual antiplatelet therapy; SAPT, single antiplatelet therapy; and ID-TVR, ischemic driven target vessel revascularization.

![Figure 1](image_url)

**Figure 1.** A: Coronary angiography shows total occlusion in the proximal portion of the right coronary artery. B: Dilated using a 2.0 mm semi-compliant balloon. C: Intravascular ultrasound revealed thrombus and low echoic plaque in the previous stent. D: Dilated using a perfusion balloon catheter (3.5 × 20 mm) E: Final angiogram. F: Follow-up angiogram revealed good coronary flow without restenosis.
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Figure 2. A: Coronary angiography shows total occlusion in the proximal portion of the right coronary artery. B: Dilated using a 2.0 mm semi-compliant balloon. C: Intravascular ultrasound revealed neotherosclerosis that contained thrombus and calcification. D: Dilated using a perfusion balloon catheter (3.0 × 20 mm) E: Final angiogram. F: Follow-up angiogram revealed only mild stenosis.

The patient experienced a right bundle branch block, and underwent emergent coronary angiography, which revealed total occlusion in the stent site of the mid RCA (Figure 2A). We performed PCI to the occluded RCA. A temporary pacemaker was inserted via the right femoral vein, and a 6-Fr AL1.0 SH guide catheter was inserted via the right radial artery. We advanced a conventional 0.014 inch guidewire, and performed balloon dilatation using a 2.0 mm semi-compliant balloon (Figure 2B). After checking IVUS (Figure 2C), acute recoil occurred followed by a worsening of coronary flow. IVUS revealed rich and floating thrombosis within the stent. We delivered a 3.0-mm Ryusei balloon catheter to the lesion, and inflated it for 5 minutes × 2 (max 8 atm) (Figure 2D). Immediately after long inflation, the lesion was successfully revascularized without acute re-coil (Figure 2E). A follow-up angiogram after 3 months also revealed good flow without restenosis (Figure 2F). We continued dual antiplatelet therapy (aspirin and clopidogrel) until the follow-up angiogram, and switched to single antiplatelet therapy (clopidogrel) after the follow-up angiogram.

Discussion

In this case series, we present a novel strategy using a perfusion balloon for the treatment of VLST. Compared to conventional balloon dilatation, the advantage of a perfusion balloon is that inflation can be maintained for a longer period of time, which is effective for the prevention of acute recoil.

Stent thrombosis is the most serious concern following coronary stent implantation, and VLST is defined as stent thrombosis that occurred more than 1 year after stent deployment. The use of a second-generation DES reduced the risk of VLST as compared to the use of a first-generation DES or BMS. However, VLST is still observed in contemporary clinical practice. Although a stent-less strategy might be considered as the first-line treatment for VLST, more than half of patients with VLST required additional stent implantation, partly because a treatment strategy for VLST has not yet been established.

A detailed optical coherence tomography (OCT) analysis revealed that the causes of VLST were classified as malapposition, neotherosclerosis, uncovered strut, and underexpansion. If the causes of VLST were malapposition, uncovered strut, or underexpansion, it would not be reasonable to deploy additional stents, because additional stents would not help either malapposition or underexpansion. Additional stents would delay healing after stent deployment, which should be associated with uncovered struts. On the other hand, if the cause of VLST was neotherosclerosis, there would be several options including additional stent deployment.
For the treatment of VLST, there are 3 types of treatment strategy including balloon angioplasty, thrombus aspiration, and additional stent deployment. Our balloon angioplasty using a perfusion balloon is a novel modification of balloon angioplasty. Since the perfusion balloon has the unique characteristic of maintaining blood flow during balloon inflation, we can inflate for a longer period of time as compared to conventional balloons. Earlier studies including PCI or endovascular treatment revealed that longer inflation was associated with less trauma and larger lumen gain.\(^{17,18}\) We suggest two reasons why the long-inflation using a perfusion balloon worked to overcome acute recoil and acute severe dissection for the treatment of VLST. First, long-time balloon inflation may compress the thrombus, and fix the thrombus on the stent, which would achieve the prevention of acute recoil. Second, even if balloon dilation was aggressively performed, acute severe dissection including medial dissection would not occur within the stent, because the media always exists outside of the stent struts. Since the beginning of balloon angioplasty, acute recoil and acute severe dissection have been associated with unsuccessful revascularization.\(^{19,20}\) The long-inflation using a perfusion balloon would overcome acute recoil and acute severe dissection for the treatment of VLST. Regarding thrombus aspiration, the contemporary aspiration device may not have sufficient performance to remove all thrombus in the case of VLST. Moreover, if VLST was caused by neoatherosclerosis, atherosclerotic burden including a necrotic core or lipid pool would not be removed by aspiration. Regarding additional stent deployment, we may achieve a good acute lumen gain following stent deployment. However, additional stent deployment may increase the future risk of stent thrombosis. The advantages and disadvantages of the 3 types of treatment strategy are summarized in Table II.

The clinical limitations of long inflation using a perfusion balloon should be discussed. First, this technique has not been performed widely for the treatment of VLST. Therefore, the risks and benefits of this technique have not been confirmed in clinical practice. Second, since the profile of perfusion balloons is larger than that of conventional balloons, operators may experience difficulty when delivering a perfusion balloon to the culprit lesion. Third, although we propose long-inflation using a perfusion balloon, the ideal inflation time (3-minutes?, 5-minutes?) is still unknown for VLST. Fourth, we did not compare the device performance directly between perfusion balloons and other devices. Even after considering the above limitations, long inflation using a perfusion balloon would have potential advantages for the treatment of VLST.

**Conclusions**

Long inflation using a perfusion balloon worked to prevent acute recoil, and to achieve optimal expansion without an additional stent. Perfusion balloons may be a reasonable option for the treatment of VLST.

**Disclosure**

**Conflicts of interest:** Dr. Sakakura has received speaking honoraria from Abbott Vascular, Boston Scientific, Medtronic Cardiovascular, Terumo, OrbisNeich, Japan Lifeline, Kaneka, and NIPRO; he has served as a proctor for Rotablator for Boston Scientific, and he has served as a consultant for Abbott Vascular and Boston Scientific. Prof. Fujita has served as a consultant for Mehergen Group Holdings, Inc. Other authors have no conflicts of interest to declare.

**References**