Case Report

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Identification of Vulnerable Plaque in a Stented Coronary Segment 17 Years after Implantation Using Optical Coherence Tomography

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A patient presented with exertional chest pain two months prior to admission. Coronary angiography revealed a subocclusive stenosis within the boundaries of the stent. Optical coherence tomography showed remarkable intimal growth inside the stent, which demonstrated a heterogeneous appearance including low-intensity areas. These findings were congruent with the morphology of fibroatheroma in the native coronary artery and suggested that new atherosclerotic progression of the intima within the stent had occurred over 17 years following bare metal stent implantation. To the best of our knowledge, this is one of the most delayed instances of a bare metal stent restenosis described in the medical literature.

Key Words: Atherosclerosis, stent, percutaneous coronary intervention, restenosis

INTRODUCTION

Although the bare metal stent (BMS) has been widely used for at least 15-20 years, long-term clinical events associated with BMS implantation have not yet been fully elucidated. In general, in-stent restenosis after BMS replacement has been known to be caused by a neointimal hyperplasia that is irritated chronically by a foreign body granulomatous reaction after stenting.[1] Yokoyama, et al.[2] reported that white neointima in the BMS can often change into yellow plaque over an extended period, and that atherosclerotic progression inside the BMS might contribute to late luminal narrowing. We report herein a case of a vulnerable plaque in a stented coronary segment 17 years after implantation using optical coherence tomography (OCT). These findings suggest that *de novo* atherosclerotic plaque formation can also occur within the boundaries of previously implanted stents.

CASE REPORT

A 73-year-old woman was admitted to our hospital because of exertional chest pain two months before admission. She had been hypertensive for 20 years. No history of diabetes, smoking, dyslipidemia, or family history of early coronary ar-

tery disease was noted. When she was 56 years old, she had been implanted with a BMS (Palmaz-Shatz, Cordis, Johnson & Johnson, Warren, NJ, USA) in the proximal left anterior descending artery (LAD) to treat stable angina pectoris. Her blood pressure at admission was 122/75 mm Hg, pulse rate was 59/min and heartbeat was regular. Physical examinations of her cardiovascular and other systems showed normal. Resting electrocardiography revealed nonspecific ST-T wave abnormalities. Because an exercise stress test showed a positive result, coronary angiography was performed. Baseline coronary angiography showed a subocclusion of the LAD within the boundaries of the previously placed BMS (Fig. 1A) and impaired blood flow distal to the lesion. The right coronary artery showed mild stenosis in the mid region. A guidewire was advanced into the LAD through a 6-Fr Judkin's Left 3.5 guide catheter (Cordis, Johnson and Johnson Co., Miami Lakes, FL, USA). Then, the OCT balloon catheter was advanced proximal to the lesion over a coronary guidewire, which was then replaced with the OCT imaging wire (ImageWire TM, LightLab Imaging Inc., Westford, MA, USA), and it was advanced only just distal to the lesion to prevent injury by the OCT balloon catheter. It was then withdrawn proximally, and regions distal and proximal to the lesion were analyzed using an automated pullback system at 1.0 mm/s. Cross-sectional images were acquired at 20 frames/s. Analysis of contiguous cross sections was performed at 1-mm intervals. OCT revealed remarkable intimal growth inside the stent, demonstrating a heterogeneous appearance including a signal-poor area, a bright reflective fibrous cap (Fig. 2A), and a homogeneous high signal band at the inner luminal border in a more proximal segment within the BMS (Fig. 2B). Longitudinal OCT view showed layered pattern with inner high signal and outer low signal layers (Fig. 2). After imaging, the lesion was predilated with a 2.0×20 mm Ikazuchi 10™ balloon (Kaneka, Osaka, Japan) at 12 atmospheres. Then, a 2.75×28 mm Promus element stent (Boston Scientific Corp., Natick, MA, USA) was placed successfully at 16 atmospheres over the proximal portion of the LAD, covering the previous stent. Final angiographic findings were acceptable and did not show any complications such as dissection, distal embolization or slow flow (Fig. 1B). The patient recovered well after the intervention and was discharged the next morning.

DISCUSSION

Several recent reports have raised the issue of late and very late stent thromboses after BMS implantation, whereas this issue was poorly described while the BMS technology was being developed. Generally, in-stent neointimal hyperplasia following BMS implantation peaks in the early restenosis phase (6-12 months), and then the stented lesion stabilizes.[3] Another study demonstrated a triphasic luminal response characterized by an early restenosis phase up to 6 months, an intermediate term regression phase from 6 months to 3 years and a late renarrowing phase beyond 4 years after coronary stent placement in native coronary arteries.[4] Usually, such an in-stent neointima is observed as a homogeneous structure; however, in our case, the neointima showed a heterogeneous morphology congruent with a fibroatheroma in the native coronary. These features suggest that atherosclerotic progression developed in the neointima over 17

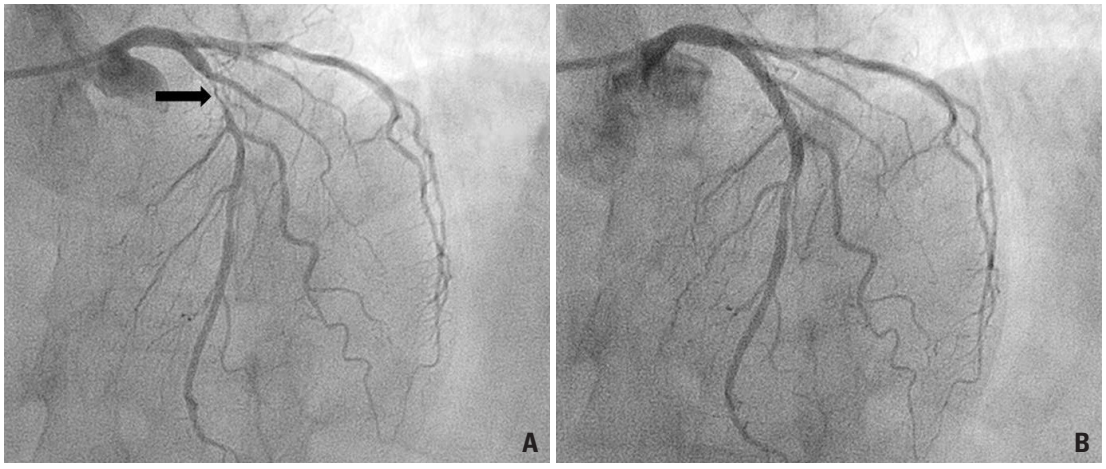

Fig. 1. (A) Baseline coronary angiography showing in-stent restenosis at the proximal segment of the left anterior descending (LAD) coronary artery (black arrow). (B) A 2.75×28 mm Promus element stent (Boston Scientific Corp., Natick, MA, USA) was placed successfully at 16 atmospheres within the previous stent of the LAD.

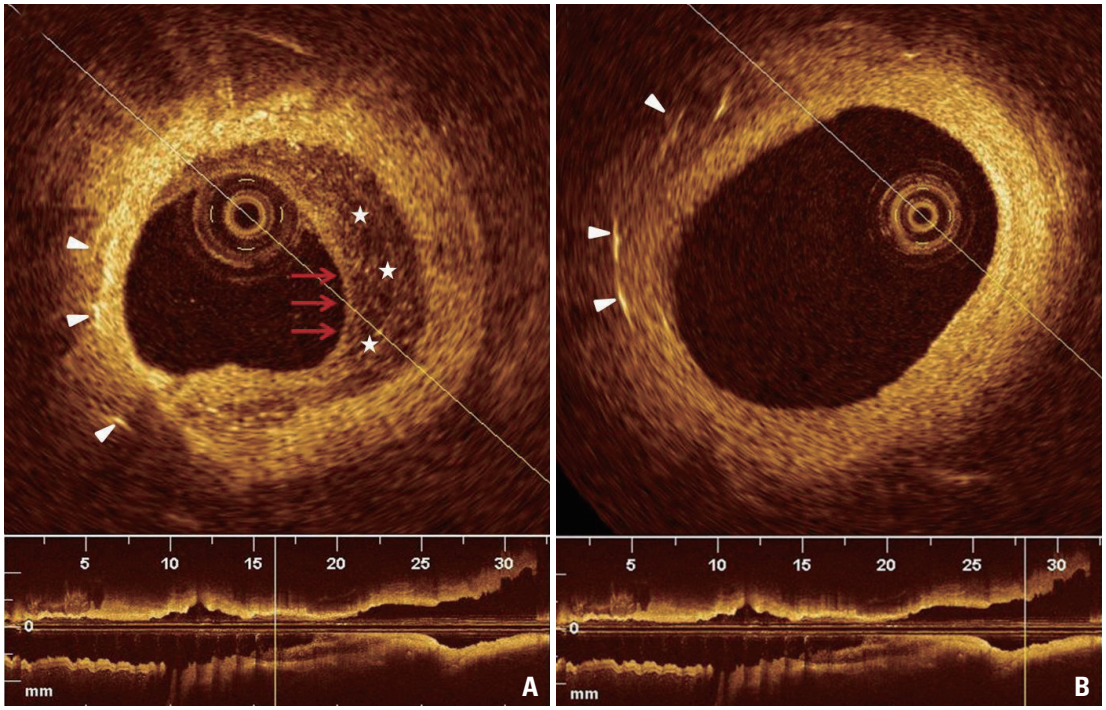

Fig. 2. (A) A thin-cap fibroatheroma assessed using optical coherence tomography (OCT) overlying a large lipid-rich plaque (1 to 5 o'clock position). The OCT shows the presence of lipid-rich intima as a signal-poor area (★); a bright reflective fibrous cap (red arrow) and stent struts (white arrowheads) are also observed. (B) OCT reveals a homogeneous high signal band at the inner luminal border in a more proximal segment within the BMS. Longitudinal OCT view shows layered appearance with inner high scattering and outer low scattering layer. BMS, bare metal stent.

years following BMS implantation, which is consistent with previous reports.[3,5] Also, a study using serial angioscopic observations of this type of feature inside a BMS showed that a white appearance occupied mainly by neointima at a 6- to 12-month follow-up often changed to a partially yellow and red lumen composed of atherosclerotic plaque and thrombus beyond 4 years.[2]

OCT is a promising tool for the genesis of biomedical images. It enables tissue pathology to be imaged *in situ* and in real time, and the histopathology hallmark of vulnerable plaque is known as a thin-cap fibroatheroma. These pathological findings were consistent with the OCT imaging of this case of late luminal narrowing after coronary stenting. Actually, in our case, the OCT demonstrated the presence of lipid-rich intima as a signal-poor area and a bright reflective fibrous cap. Similar to previous study, the radial axis of the neointima showed the layered appearance with inner high scattering and outer low scattering layer as variations in tissue composition, cell density, and orientation.[6] Our case suggests that atherosclerotic plaques can develop *de novo* within the boundaries of previously implanted stents and that these plaques can have a natural history similar to the history of plaques occurring in native untreated coronary arteries. Similar to our case, a previous study suggests that stainless steel stent evokes a remarkable foreign-body inflammatory reaction to metal and these peri-strut chronic inflammatory cells may accelerate new indolent atherosclerotic changes and consequent plaque vulnerability.[7]

In summary, we treated a patient with a vulnerable plaque in a stented coronary segment 17 years after implantation using OCT. To the best of our knowledge, this is one of the most prolonged bare metal stent restenosis described in the medical literature. We suggest that *de novo* atherosclerotic progression should be considered as a cause of very late in-stent restenosis after BMS implantation.

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