JACC: CASE REPORTS VOL. 28, 2023

IMAGING VIGNETTE

CLINICAL VIGNETTE

Very Late Stent Thrombosis Due to Neoatherosclerosis After Implantation of an Ultra-Thin Strut Stent

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ABSTRACT

A 69-year-old man developed very late stent thrombosis (VLST) 3 years after Orsiro stent implantation in the proximal left anterior descending coronary artery. Intravascular imaging evaluations before and after the onset of VLST allowed us to document neoatherosclerosis as the etiology of VLST. (J Am Coll Cardiol Case Rep 2023;28:102121) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Three years before admission, a 69-year-old man had the first acute antero-septal myocardial infarction. An Orsiro (Biotronik) ultra-thin strut bioabsorbable-polymer sirolimus-eluting stent (O-SES) (3.0 × 22 mm) was implanted in the left anterior descending coronary artery.

One year later, coronary angiography (CAG) showed no in-stent restenosis, intravascular ultrasound (IVUS) also showed no significant restenosis, and coronary angioscopy (CAS) showed thin neointima on part of the stent, no thrombus, and no yellow plaque. However, 3 years after O-SES implantation, he was transferred to our hospital for chest pain at rest. Blood pressure was 130/72 mm Hg, and heart rate was 64 beats/min sinus rhythm. Twelve-lead electrocardiogram showed ST-segment elevation in V_1-V_3, and troponin T was slightly elevated.

INVESTIGATIONS

CAG showed an obstruction in the stent, and VLST was diagnosed. After thrombus aspiration, IVUS revealed marked intimal thickening inside the O-SES. Recanalization was achieved by a 3.0 × 15-mm drug-coated balloon at the O-SES site.

After recanalization, optical coherence tomography (OCT) and CAS were performed to investigate the cause of VLST. OCT showed marked neointimal thickening in the previously implanted stent and plaque rupture (Video 1). CAS showed thick neointima and ulcerative lesions in the stent and some thin neointima in the proximal lesion (Video 2).

We compared the images obtained at 1 year and 3 years after the O-SES implantation. Compared with the first evaluation, integrated backscatter intravascular ultrasound (IB-IVUS) showed lower fibrous content but a larger lipid pool at 3 years (**Figures 1A1 and 1A2**), CAS showed ulcerative lesions and a covering of thick

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received August 21, 2023; revised manuscript received October 16, 2023, accepted October 18, 2023.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2023.102121

ABBREVIATIONS AND ACRONYMS

CAG = coronary angiography

CAS = coronary angioscopy

IB = integrated backscatter

IVUS = intravascular ultrasound

OCT = optical coherence tomography

O-SES = Orsiro sirolimus-eluting stent

VLST = very late stent thrombosis

neointima (Figures 1B1 and 1B2), and OCT showed plaque rupture and progression of new in-stent atherosclerotic lesions (Figure 1C).

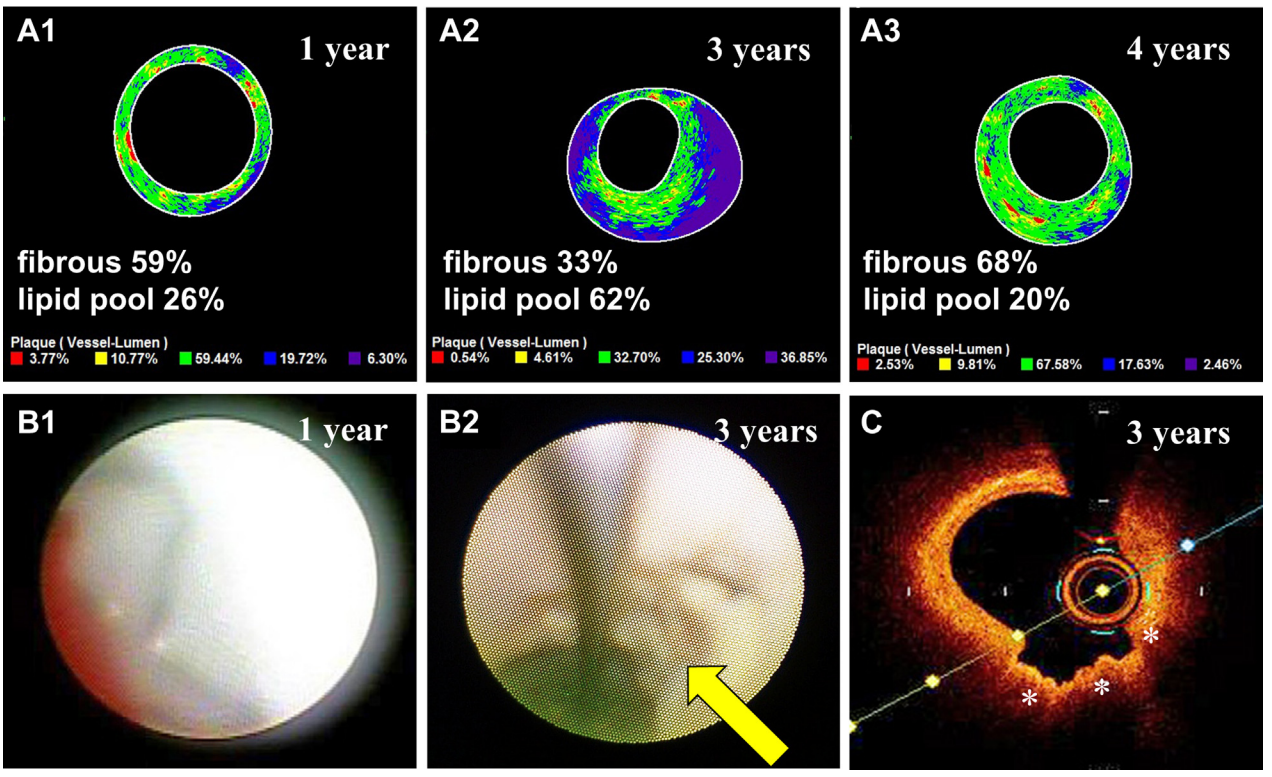
MANAGEMENT

After the onset of VLST, more strict lipid management was performed. Follow-up CAG and IVUS were performed 9 months after onset of VLST. CAG showed a moderately restenotic lesion in the stent, but follow-up IB-IVUS showed a marked increase in the fibrous content and reduction of the lipid pool (Figure 1A3).

DISCUSSION

The BIOFLOW-V trial ((Biotronik Prospective Randomized Multicenter Study to Assess the Safety and Effectiveness of the Orsiro Sirolimus Eluting Coronary Stent System in the Treatment of Subjects with Up to Three De Novo or Restenotic Coronary Artery Lesions V), which compared the long-term clinical outcomes of the O-SES with the Xience stent (Abbott Cardiovascular), found that in O-SES, late stent thrombosis and VLST were extremely rare.[1] The following causes of VLST have been reported: 1) stent malapposition; 2) neoatherosclerosis[2]; and 3) abnormal vascular reactions associated with local chronic inflammation and hypersensitivity reactions. A previous registry study found that malapposition, underexpansion, and an uncovered strut were the most common causes of VLST with a drug-eluting stent and that neoatherosclerosis was

FIGURE 1 Comparisons of the Images at 1, 3, and 4 Years After O-SES Implantation



Integrated backscatter intravascular ultrasound showed that at 3 years, the fibrous content (green) had decreased and the lipid pool (blue and purple) had increased (A1-A2). Coronary angioscopy showed ulcerative lesions (arrow) and thick neointima at 3 years (B1-B2). Optical coherence tomography at 3 years showed plaque rupture (asterisks) and progression of in-stent neoatherosclerosis (C). Integrated backscatter intravascular ultrasound imaging at 9 months after the onset of very late stent thrombosis (4 years after Orsiro sirolimus-eluting stent [O-SES] implantation) showed a marked increase in the fibrous content and a marked reduction in the lipid pool (A3).

a less common cause than a bare-metal stent.[3] In the present case, no stent malapposition was observed, but IB-IVUS showed an increased lipid pool outside the stent. Based on OCT and CAS findings, we presume that VLST was caused by development of atherosclerosis.

This case is of great interest because we were able to evaluate images of VLST at 3 different time points: before, during, and after the onset of the VLST.

CONCLUSIONS

We experienced a rare case of VLST 3 years after implantation of an O-SES.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS coronary angioscopy, integrated backscatter intravascular ultrasound, ultra-thin strut bioabsorbable-polymer sirolimus-eluting stent, very late stent thrombosis

APPENDIX For supplemental videos, please see the online version of this paper.