
CASE REPORT

Very Late Thrombosis of an Undersized Bare-Metal Stent

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ABSTRACT

A 52-year-old gentleman was admitted with anterior non ST elevation myocardial infarction. He had a history of stenting of the left anterior descending (LAD) – first diagonal (D₁) bifurcation with two bare-metal stents (BMS) according to the provisional T-technique ten years earlier. He was also submitted to a simple balloon angioplasty for focal LAD in-stent restenosis 14 months ago. The urgent coronary angiography this time showed a very late stent thrombosis of the LAD BMS fortunately with preserved distal flow. He was initially treated successfully with aspiration thrombectomy combined to few days of aspirin, prasugrel and enoxaparin to enable complete thrombus dissolution. Five days later the LAD stent was examined with optical coherence tomography (OCT) which revealed severe malapposition proximally to the D₁ due to initial BMS undersizing. The stent was expanded and a kissing-balloon inflation was performed at the LAD – D₁ bifurcation with appropriately sized balloons. Finally, after verifying the correct stent expansion and apposition by OCT, a drug-eluting balloon inflation was performed in-stent in order to minimize the risk of restenosis. Subsequent clinical course was uneventful. Details and images concerning these procedures are presented and discussed herein.

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KEY WORDS: *very late stent thrombosis; stent undersizing; stent malapposition; bare metal stent; optical coherence tomography.*

ABBREVIATIONS

BMS = bare-metal stent
D1 = first diagonal branch
DES = drug-eluting stent
IVUS = intravascular ultrasound
LAD = left anterior descending
OCT = optical coherence tomography
PCI = percutaneous coronary intervention

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Stent thrombosis, unlike restenosis which has a more benign course, constitutes a dire complication after percutaneous coronary intervention (PCI) having a severe clinical impact due to a high risk of acute myocardial infarction and death.¹ The incidence of this alarming complication might be increased after implantation of drug-eluting stents (DES), compared to bare metal stents (BMS), although this has been recently refuted.¹⁻³ Based on the elapsed time after stent implantation, stent thrombosis can be classified as early (0-30 days post stent implantation), late (>30 days) and very late (>12 months). Again, studies have indicated that the incidence of late and very late stent thrombosis is increased after DES implantation. We herein report a case of very late stent thrombosis observed after implantation of a BMS.

CASE REPORT

A 52-year-old male patient was admitted to our hospital after a diagnosis of non ST elevation myocardial infarction with electrocardiographic signs of ongoing anterior ischemia. He had a history of non ST elevation acute coronary syndrome 10 years earlier treated by PCI of the left anterior descending (LAD) – first diagonal (D₁) bifurcation

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according to the provisional T-stenting technique with implantation of 2 BMS; a 2.75x12 mm Driver stent was placed in the LAD and a 2.5x8 mm Pixel stent in the D₁ ostium. Fourteen months ago – about 9 years after the initial PCI and after a positive myocardial scintigraphy – the patient was submitted to a new PCI for focal LAD in-stent restenosis just distal to the D₁ origin. A simple 3x9 balloon dilatation at 14 Atm gave an excellent angiographic result, but no intracoronary imaging was performed. He was treated with clopidogrel 75 mg/d on top of aspirin 75 mg/d for a month and he was on aspirin without interruption until his new admission for the acute coronary syndrome. Regarding his risk factors he had quit smoking at the time of his first coronary event 10 years ago and was under high-dose statin for dyslipidemia.

Despite treatment on admission with prasugrel 60 mg loading dose, IV nitrates and heparin, angina and anterior wall ischemia persisted and thus an urgent coronary angiography was decided. This revealed an in-stent LAD heterogeneous lesion compatible with thrombus but fortunately with preserved TIMI III flow in the distal LAD segment (Figure 1A). The patient was treated with two runs of simple aspiration thrombectomy with a good angiographic result (Figure 1B). Of note, chest pain and ischemia disappeared after the first thrombectomy run which retrieved small fragments of white thrombus. He was treated aggressively with aspirin, prasugrel and enoxaparin in order to enable thrombus dissolution and a new coronary procedure was scheduled five days later with optical coherence tomography (OCT) guidance.

The initial LAD stent was a 2.75 mm BMS dilated a year ago with a 3 mm balloon because of focal restenosis. With StentViz (a stent visualization enhancement technique) the stent proximal to D₁ seemed undersized and somehow deformed (Figure 2A). At OCT imaging the stent distal to the

diagonal was well apposed and all its struts well covered by intima, while good apposition was also noted at the diagonal level with opening at the D₁ ostium (Figure 2 B and C). Further pullback revealed that the stent was quite undersized proximal to the D₁ branch, which probably consisted the most important contributing factor for the very late BMS thrombosis (Figure 2 D, E and F). The mean reference lumen diameter proximal to D₁ and just proximal to the stent was measured at 3.62 mm (Figure 3A). At the stent malapposition zone, the minimal in-stent area was measured 6.35 mm², while the minimal lumen area was 8.65 mm², creating a 2.30 mm² area of malapposition due to undersizing (Figure 3B).

In order to expand the proximal part of the stent, PCI was performed by a balloon dilatation with a 4x8 mm non-compliant balloon intra-stent but proximally to the D₁ (at 16 Atm). A kissing-balloon inflation at the bifurcation LAD - D₁ followed with two non-compliant balloons (3.5x12 mm and 2.5x12 mm respectively, both at 12 Atm). The stent now seemed better expanded with StentViz (Figure 4 A) and with OCT good expansion without malapposition was noted throughout its length (Figure 4 B-F). Proximal to the D₁ there was no malapposition with minimal lumen diameter (MLA) at 9.3 mm² and distal to the D₁ the MLA was now at 7.3 mm² from 4.5 mm² before PCI (Figure 5 A and B). After confirming the correct stent expansion, a 4x15 mm SeQuent Please paclitaxel-eluting balloon (B. Braun Melsungen AG, Germany) was positioned under StentViz control so that it covered the LAD stent and slightly exceeded its proximal and distal edges (Figure 5 C). It was inflated at nominal pressure for 1 minute with a good final angiographic result (Figure 5D). Treatment with aspirin 75 mg/d for life and prasugrel 10 mg/d for a year was recommended and the clinical course thereafter was uneventful.

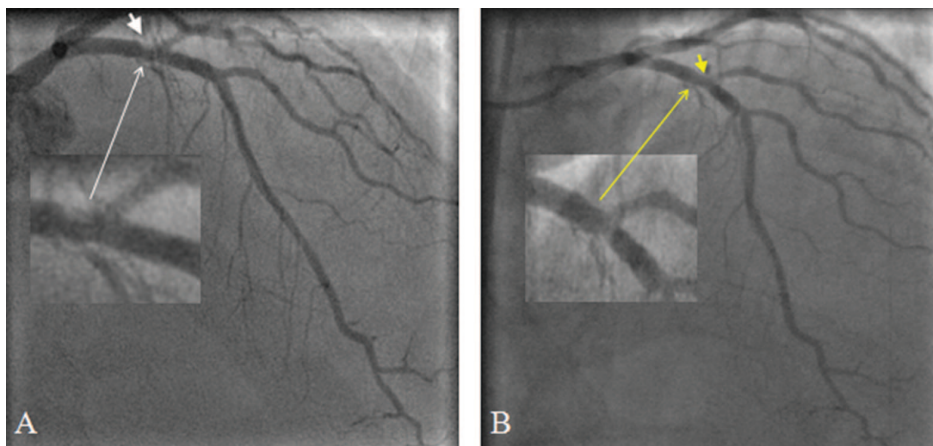


FIGURE 1. Urgent PCI the day of admission. **A.** Haziness compatible with thrombus at the stent level in the LAD, mainly just proximal to the D₁ origin. **B.** After aspiration thrombectomy, haziness proximal to the D₁ disappears. LAD = left anterior descending (coronary artery); PCI = percutaneous coronary intervention.

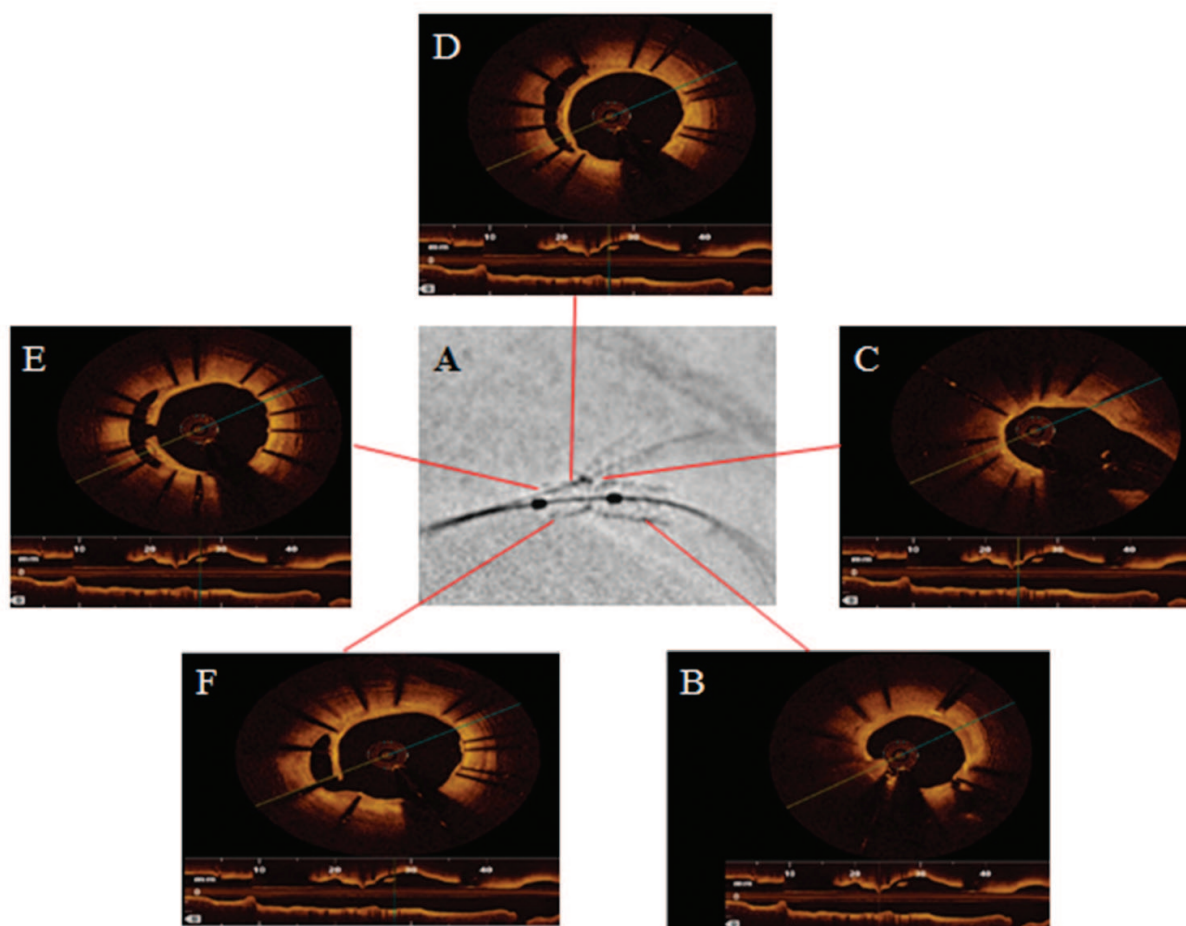


FIGURE 2. Second coronary procedure, imaging before PCI. **A.** With StentViz, the stent proximal to the diagonal seems undersized and somehow deformed. **B.** At OCT distal to the diagonal, the stent is well apposed and all its struts well covered by intima. **C.** Good apposition and strut coverage at the diagonal level with opening at the diagonal ostium and guidewire shadows seen from 3 to 6 o' clock. **D, E and F.** Apparent stent undersizing and malapposition of the stent at consecutive frames and for approximately 2 mm in length proximally to D₁. The malapposition extends from 7 to 12 o' clock, while the struts are covered by fibrin and some thrombus debris. OCT = optical coherence tomography; PCI = percutaneous coronary intervention.

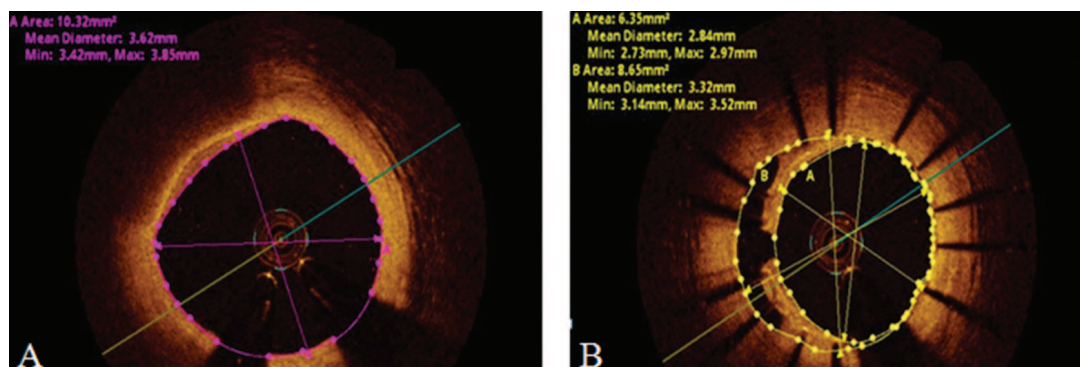


FIGURE 3. A. The mean reference diameter proximal to the diagonal and just proximal to the stent was measured at 3.62 mm. Notice the high intensity line seen from 9 to 12 o' clock which corresponds to macrophage infiltration that creates loss of signal and shadowing of deeper vessel wall tissues. **B.** At this frame of the stent malapposition zone the minimal in-stent area was measured 6.35 mm² while the minimum lumen area was 8.65 mm², creating a 2.30 mm² area of malapposition due to undersizing.

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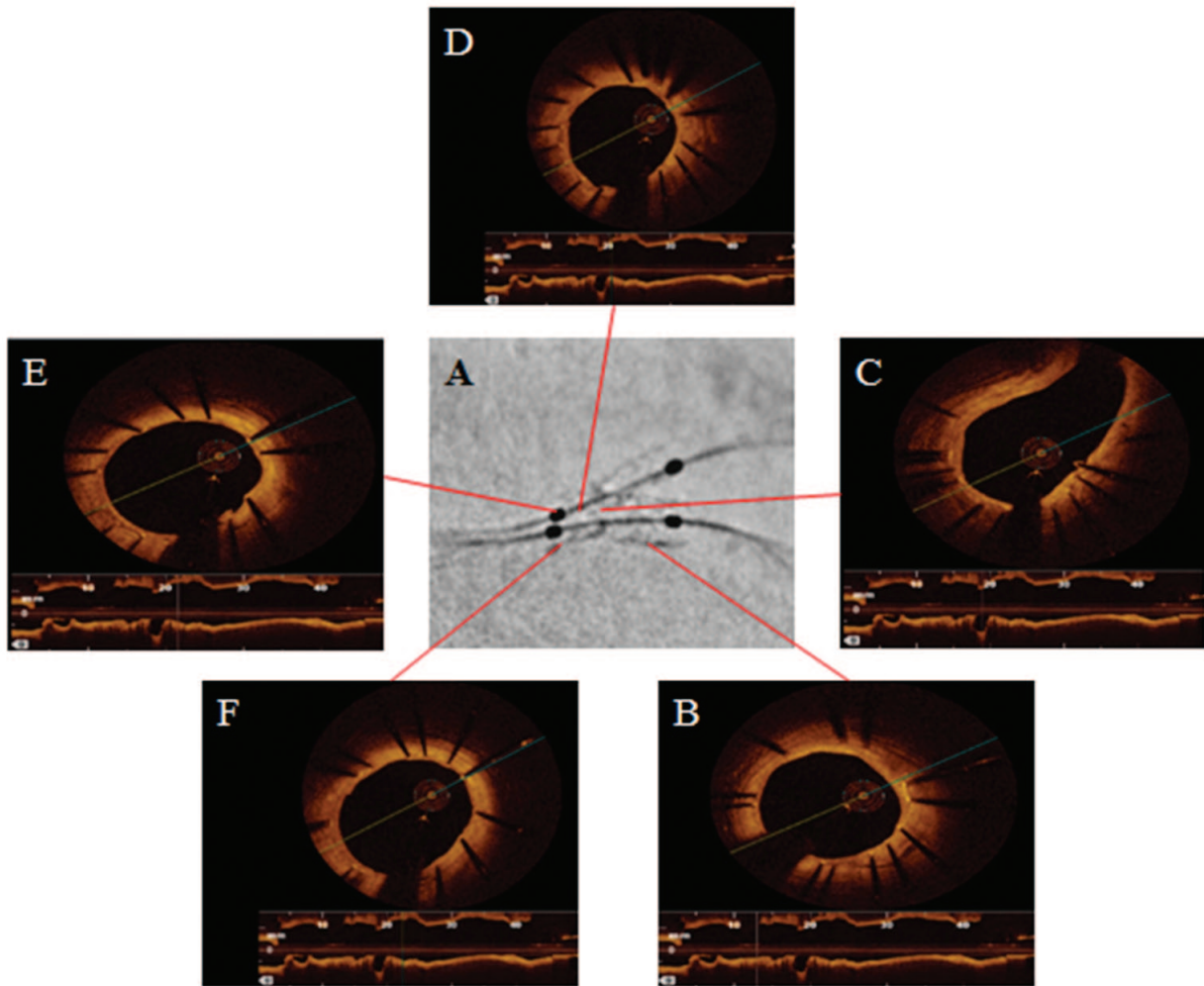


FIGURE 4. Second coronary procedure, OCT after kissing - balloon at the LAD – D₁ bifurcation. **A.** With StentViz, the stent seems better expanded. **B-F.** At corresponding frames compared to Figure 2: the stent is better expanded distal to the bifurcation (B) and at the diagonal level (C), while it is well expanded without malapposition proximal to the bifurcation at frames corresponding to the initial malapposition zone (D, E and F). D1 = first diagonal (branch); LAD = left anterior descending (coronary artery); OCT = optical coherence tomography.

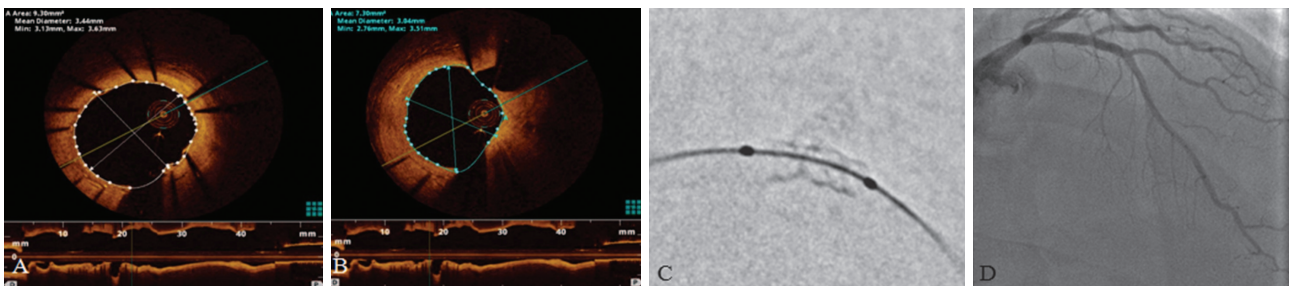


FIGURE 5. OCT after kissing - balloon at the LAD – D₁ bifurcation. **A.** Proximal to D₁ there is no malapposition with minimal lumen area of 9.3 mm². **B.** Distal to the diagonal the minimal lumen area is now 7.3 mm² from 4.5 mm² before PCI. **C.** Positioning of the 4x15 mm paclitaxel-eluting balloon under StentViz control so that it covers the LAD stent and slightly exceeds its proximal and distal edges. It was inflated at nominal pressure for 1 minute. **D.** Final angiographic result. LAD = left anterior descending (coronary artery); OCT = optical coherence tomography; PCI = percutaneous coronary intervention.

DISCUSSION

The case presented is one of a very late BMS thrombosis (10 years after the initial implantation and 14 months after simple balloon angioplasty for focal in-stent restenosis) discovered by OCT to be probably due to stent undersizing and malapposition. Despite the fact that neointima was traumatized from the balloon angioplasty 14 months ago, OCT before further balloon dilations did not show edge dissections, intimal prolapse, excessive neointimal thickness or neoatheroma with rupture or erosion that could otherwise explain the stent thrombosis. Guidance by OCT during PCI further helped to verify optimal stent expansion with appropriately sized balloons. Importantly, this imaging confirmation was sought before the final use of a drug-eluting balloon in order to ensure appropriate circumferential contact and drug delivery to the vessel wall during its inflation. A drug-eluting stent (DES) was not implanted since the problem was not restenosis or neoatherosclerosis but seemingly just the initial BMS undersizing, while enough stent metal was already present at the bifurcation.

Despite remaining an uncommon complication of PCI, when stent thrombosis occurs, it can be catastrophic, commonly presenting as acute myocardial infarction or sudden cardiac death. Very late stent thrombosis is a complication feared mainly after DES implantation even after optimal technique and uninterrupted dual antiplatelet therapy. There are concerns regarding chronic inflammation related to the DES polymer and late acquired malapposition that could contribute to stent thrombosis.¹ Late stent malapposition, despite being rare, is more often met after DES compared with BMS implantation and associates with late stent thrombosis.^{2,3} The development of new DES technologies such as bioabsorbable polymers, polymer-free DES and fully bioabsorbable DES aims to solve these problems and eradicate stent thrombosis risk related to intrinsic DES properties.¹

However, as shown in this case, the stent thrombosis risk exists with any stent, especially in case of suboptimal technique. Concerning late and very late stent thrombosis, although extremely rare with BMS, it is apparently possible and has been reported up to 13 years after stenting.^{4,5} It has been shown that stent undersizing is the main technical problem of PCI related to stent thrombosis.⁶ Studies with intravascular ultrasound (IVUS) from the BMS era have shown that 94% of stent thrombosis cases demonstrated at least one abnormal IVUS finding (stent under-expansion, malapposition, inflow/outflow disease, dissection, or thrombus), while angiography demonstrated an abnormality in only 32% of cases.⁷

Currently OCT, which has 10 times better resolution compared to IVUS, is the gold standard for intravascular imaging and can guide decision making in PCI.^{8,9} Studies with OCT have shown that beyond technical problems with stent implantation, a second mechanism for stent thrombosis could

also be in some cases the formation of neointima within the BMS that often transforms into lipid-laden tissue during an extended period of time with expansion of neovascularization into the neointima that contributes to atherosclerotic progression.¹⁰ The neo-atherosclerotic tissue seems to form earlier after DES compared to BMS implantation and may rupture leading to acute coronary syndromes for both BMS and DES.¹¹ There has been however a recent report of DES very late thrombosis without evidence of uncovered struts or restenosis while the patient was on dual antiplatelet therapy.¹² This suggests that despite we gradually gain insight into stent thrombosis mechanisms by the use of intravascular imaging techniques, there is still research to be done in order to fully elucidate the secrets regarding the etiology of stent thrombosis.

Nevertheless, concerning our case the most important contributing factor seems to have been initial stent undersizing and malapposition. Therefore, it should be emphasized that meticulous PCI technique is important and in cases of uncertainty, intracoronary imaging can give the information needed in order to guide angioplasty and optimize stenting. As shown in this case, suboptimal technique echoes even in the distal future as undersizing and malapposition can be responsible for very late not only DES but also BMS failure and thrombosis.

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