

CASE REPORT

Percutaneous Revascularization Strategy for Acute Coronary Syndrome With Two Culprit Arteries and Distal Left Main Disease With Consecutive Bifurcation Lesions

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Abstract

The case of a patient with NSTEMI is presented who was shown to have two culprit thrombotic coronary lesions and underwent successful percutaneous coronary intervention for multivessel coronary artery disease at a staged approach. *Rhythm 2018;13(3):54-58.*

Key words: acute coronary syndrome; multiple culprit arteries; left main disease; bifurcation lesions

Abbreviations: ACS = acute coronary syndrome; D1 = first diagonal branch, DES = drug-eluting stent; LAD = left anterior descending; LCx = left circumflex; LM = left main; NSTEMI = non-ST-elevation myocardial infarction; PCI = percutaneous coronary intervention; RCA = right coronary artery

Introduction

Despite the high reported incidence of multiple severe coronary lesions in patients presenting with acute coronary syndromes (ACS), suggesting a systemic vulnerable plaque activation,^{1,2} multiple culprit lesions with simultaneous thrombosis in different coronary arteries requiring multi-vessel percutaneous coronary intervention (PCI) at the index procedure are encountered less frequently.³ The combination of multi-vessel coronary thromboses with consecutive severe bifurcation lesions involving the left main stem is an even more complex and unusual scenario requiring a stepwise approach and cautious selection of the optimal intervention strategy, amongst a variety of sophisticated techniques invented for the treatment of challenging coronary lesions.

Case Report

A 48-year-old male patient was admitted shortly after a promptly resuscitated out-of-hospital cardiac arrest, which was the initial manifestation of an apparently very high-risk non-ST-elevation myocardial infarction (NSTEMI). Upon admission, the patient was conscious (Glasgow Coma Scale 15) and hemodynamically stable, yet with severe ongoing anginal chest pain. His clinical examination revealed a fourth heart sound but no rales (Killip class I). His 12-lead ECG showed sinus rhythm, diffuse ischemia with ST depression in inferior and precordial leads and ST elevation in aVR, while ECG

monitoring revealed sporadic non-sustained ventricular tachycardia episodes. Past medical history revealed severe depression under heavy medical treatment, whereas his cardiovascular risk factors were obesity and active smoking. Echocardiography showed severe left ventricular systolic dysfunction (ejection fraction 30%) with diffuse hypokinesis and mild mitral regurgitation. Cardiac troponin I was mildly elevated. His TIMI and GRACE risk score calculations were 4 and 167 respectively.

Following his initial clinical evaluation, the patient was pretreated with loading doses of ticagrelor 180 mg and aspirin 250 mg. He was then immediately transferred to the catheterization laboratory for emergency coronary angiography (performed by the right femoral approach due to an abnormal Allen test) which revealed a distal left main (LM) to proximal left anterior descending (LAD) significant (50-70%) stenosis, a very proximal well - sized first diagonal branch (D1) with 90-95% ostial stenosis and a short, thrombotic and occlusive mid - LAD culprit lesion with TIMI II flow distally. Retrograde filling of the distal right coronary artery (RCA) branches was also noted. Left circumflex (LCx) ostium was free of disease, but the distal part of the first marginal branch was diffusely infiltrated. The RCA was dominant with a long thrombotic occlusion at its mid-segment and TIMI I flow distally (Fig. A).

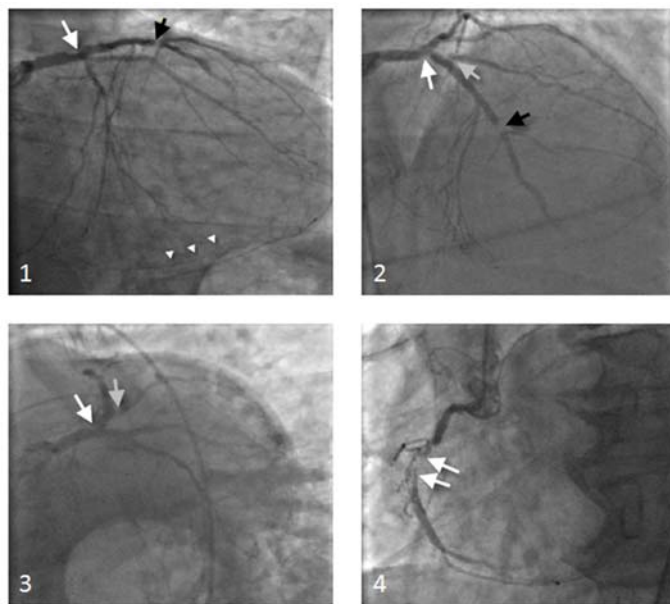


Figure A. (1, 2 & 3: RAO caudal, RAO cranial & spider views respectively): Distal LM to proximal LAD significant lesion, 50-70% stenosis (white arrow). Very proximal well - sized D1 with 90-95% ostial stenosis (grey arrow). Short, thrombotic and occlusive mid - LAD culprit lesion with TIMI II flow distally (black arrow). Retrograde filling of the distal RCA branches (white arrowheads, 1). LCx ostium free of disease, diffusely infiltrated distal first marginal. (4, LAO view): Dominant RCA with thrombotic occlusion at its mid-segment (white arrows) and TIMI I flow distally.

Despite the presence of multi-vessel disease with distal LM involvement coronary artery bypass surgery apparently was not an appropriate option for revascularization due to ongoing ischemia, electrical instability and a history of serious psychiatric disorder. Therefore, PCI at two stages was decided: 1) *Ad hoc PCI* of the 2 culprit flow-limiting lesions (mid-LAD and mid-RCA) to restore normal coronary flow and 2) *Scheduled PCI before discharge* to treat the lesions of distal LM, ostial-proximal LAD and D1 ostium.

I. Double culprit lesions primary PCI

The primary PCI consisted of direct stenting of the LAD lesion first with a 3x13mm drug-eluting stent (DES) followed by stenting of the RCA lesion with a 3.5x28mm DES resulting in prompt restoration of TIMI III flow in both vessels (Figure B). Before RCA stenting manual thrombectomy of the mid-RCA thrombotic occlusion was performed, but its effectiveness was limited because of a heavy burden of thrombus which was well organized and with most of its volume steadily attached to the vessel wall. Intra-coronary bolus of abciximab (0.25 mg/kg) had also been administered with no benefit before stenting. After treating the LAD and RCA thrombotic lesions the patient became asymptomatic with resolution of electrocardiographic signs of ischemia documented after PCI. Peak troponin I was measured at 38.19 ng/ml at day 2 after admission with gradual regression afterwards.

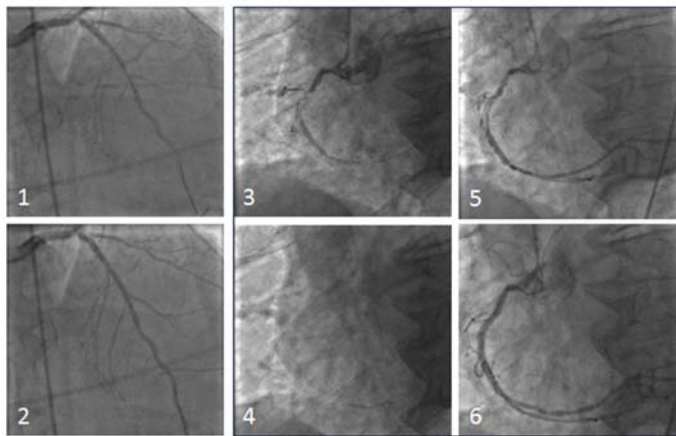


Figure B. (1,2): PCI of the LAD lesion with direct stenting (with a DES 3x13mm) and prompt restoration of TIMI III flow in distal vessel. (3-6): After minimally effective manual thrombectomy (3) subsequent stenting of the RCA mid-RCA thrombotic lesion (with a DES 3.5x28mm) led to a good angiographic result with restoration of TIMI III flow.

II. Double bifurcation scheduled PCI

The second PCI was performed 3 days later (earlier than initially scheduled due to angina recurrence), in order to achieve complete revascularization by treating the

remaining severe lesions of distal LM to proximal LAD and D1 ostium. The close proximity of the two bifurcation lesions and the LM involvement made the second PCI technically quite challenging. A decision was made to treat with one stent from the distal LM to LAD, beyond the D1 ostium. The LCx ostium was free of disease and a second stent would probably not be needed. However, the D1 ostium obviously needed stenting, thus a complicated PCI strategy with minimum two stents had to be undertaken. The idea of using a dedicated bifurcation stent (such as the Tryton stent) was rejected because of the close proximity of the LCx and D1 ostia (Figure C) which might increase technical complexity, while furthermore such a stent could not be available on the day of the intervention for logistic reasons. Therefore, a standard mini-crush was selected as the most suitable technique in order to effectively treat the LM to proximal LAD lesion and at the same time secure coverage of the D1 ostium.

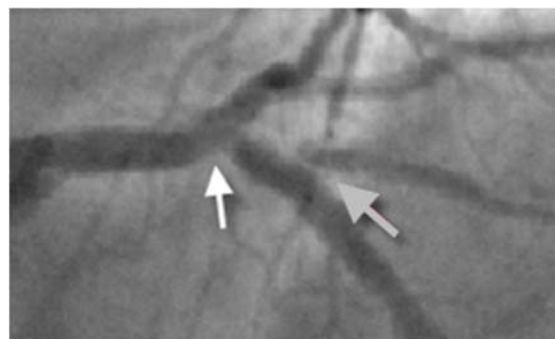


Figure C. Remaining lesions to be treated with a staged second PCI. (1) Distal LM - LAD Medina 1.1.0 bifurcation lesion (white arrow). (2) Consecutive LAD - D1 Medina 0.0.1 bifurcation lesion (grey arrow). Note that the proximal LAD atheroma extends slightly distally to the severely diseased D1 ostium which is very proximal (≤ 8 mm from the LAD ostium).

At the beginning of the second procedure the RCA was controlled to verify the good angiographic result of the initial PCI. With the use of optical coherence tomography the good apposition and expansion of the mid-LAD stent was also verified, the minimal-lumen area at the LAD ostium was measured at 3.42 mm² and the reference diameters of the LM (4.5 mm) and proximal LAD (3.75 mm) were acquired (Figure D). As for the primary PCI unfractionated heparin was administered as anticoagulant during the intervention and three conventional 0.014" guidewires were then positioned in the distal LAD, the D1 and the LCx.

A) First diagonal ostium stenting (mini-crush technique): The D1 ostial lesion was very tight, fibro-calcific and resistant. It was possible to cross initially only with a 1x10 mm balloon. Subsequently it was progressively prepared

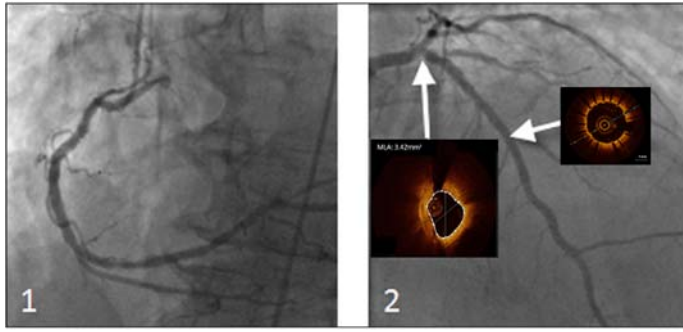


Figure D. (1) Verification of the good angiographic result of RCA-PCI, (2) With the use of optical coherence tomography the good apposition and expansion of the mid-LAD stent is verified and the ostial LAD lesion minimal-lumen area is measured 3.42mm^2 ($<4\text{mm}^2$, thus abnormally low).

by increasing diameter balloon inflations and finally it was pre-dilated with a $2.5 \times 10\text{ mm}$ scoring balloon. A $2.5 \times 13\text{ mm}$ DES was positioned at the D1 ostium, slightly protruding (1-2 mm) in the LAD where a $3.5 \times 12\text{ mm}$ non-compliant balloon waited in order to crush the protruding struts after the D1 stent deployment. Following verification that there was no dissection in the D1 after stenting, the D1 guidewire was withdrawn and the balloon waiting in the ostial – proximal LAD (centered in front of the D1 ostium) was inflated to crush the protruding stent struts (Figure E).

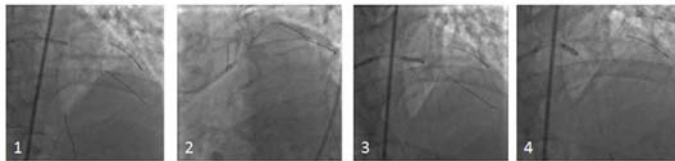


Figure E. Diagonal ostium stenting (mini-crush technique): (1) After initial inflation with a $1 \times 10\text{ mm}$ balloon and subsequent pre-dilatations with balloons of increasing diameter a final pre-dilatation with a $2.5 \times 10\text{ mm}$ scoring balloon is shown, (2,3) Stenting of the D1 ostium (with a DES $2.5 \times 13\text{ mm}$), (4): Balloon inflation in the ostial – proximal LAD to crush the protruding D1 stent struts.

B) LM to proximal LAD stenting and LM bifurcation kissing

Having already obtained a nice angiographic result at the D1 ostium, the same $3.5 \times 12\text{ mm}$ non-compliant balloon was used to pre-dilate the distal LM-ostial LAD lesion. A $4 \times 22\text{ mm}$ DES was then positioned in order to stent the distal LM to LAD lesion, landing just distally to the D1 ostium at a disease-free zone. The stent was deployed with a nice result. The proximal part of the stent at the distal LM level was post-dilated with a $4.5 \times 8\text{ mm}$ non-compliant balloon (proximal optimization technique to avoid malapposition). After rewiring the LCx and opening the stent struts at the LCx ostium with inflation of a $2 \times 12\text{ mm}$ balloon (not

shown), a $4 \times 12\text{ mm}$ and a $2.75 \times 12\text{ mm}$ non-compliant balloons were positioned at the left main bifurcation towards the LAD and the LCx respectively and a kissing balloon inflation was performed with a very good angiographic result at the bifurcation afterwards (Fig. F).

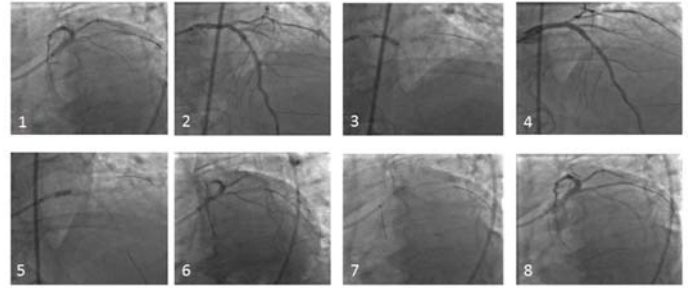


Figure F. LM to proximal LAD stenting and LM bifurcation kissing. (1): Distal LM-ostial LAD lesion pre-dilatation. (2): A $4 \times 22\text{ mm}$ DES was positioned in order to stent the distal LM - proximal LAD lesion, landing just distally to the D1 ostium at a disease free zone. (3,4): Stent deployment with a nice result. (5): Post-dilatation of proximal part of the stent at the distal LM level (proximal optimization technique to avoid malapposition with a $4.5 \times 8\text{ mm}$ non-compliant balloon). (6,7): A $4 \times 12\text{ mm}$ and a $2.75 \times 12\text{ mm}$ non-compliant balloons are positioned at the left main bifurcation towards the LAD and the LCx respectively and a kissing balloon inflation is performed. (8): Very good angiographic result at the bifurcation after kissing.

C) LAD – D1 bifurcation optimization and kissing balloon

The D1 was rewired but it was difficult to cross with a balloon the three layers of struts at its ostium. It became finally possible again with a $1 \times 10\text{ mm}$ balloon. The ostium of the D1 was further dilated with a $1.5 \times 12\text{ mm}$ and $2 \times 12\text{ mm}$ semi-compliant balloons. That made possible to cross through the opened struts into the D1 with a $2.5 \times 12\text{ mm}$ non-compliant balloon and also advance for kissing a $4 \times 12\text{ mm}$ non-compliant balloon in the proximal LAD. At the kissing position each balloon (first in the D1 then in the LAD) was inflated at 20 Atm, before performing the kissing balloon inflation at 12 Atm. Finally, after all the above mentioned technical steps, a quite satisfactory angiographic result was obtained (Fig. G).

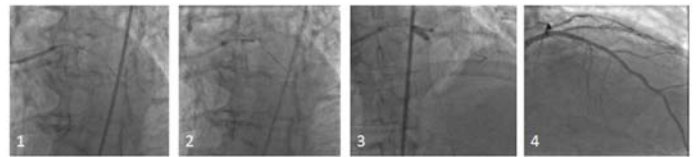


Figure G. LAD – D1 bifurcation optimization and kissing balloon. (1) Crossing the three layers of struts at the D1 ostium with a $1 \times 10\text{ mm}$ balloon, (2) Further dilatation of the D1 ostium with semi-compliant balloons, (3) Kissing balloon with non-compliant balloons ($4 \times 12\text{ mm}$ in the LAD and $2.5 \times 12\text{ mm}$ in the D1), (4) Final angiographic result.

After the second PCI the patient remained asymptomatic without electrocardiographic signs of ischemia or arrhythmia recurrence and troponin I continued to decrease until normalization without any rebound. He was discharged 2 days later and his clinical course was uneventful at 1-year follow up.

Discussion

In the majority of patients undergoing primary PCI only one atherothrombotic plaque is identified as the ACS-related culprit lesion. Despite that, several investigators have found that in some unstable patients multiple angiographically complex or ruptured non-culprit plaques may exist simultaneously.⁴ Multi-vessel coronary thrombosis with flow impairment in more than one major epicardial arteries is a more scarcely encountered clinical scenario.³ Similarly to the presented case, most of these patients have angiographic evidence of simultaneous thrombotic occlusion in LAD and RCA.^{3,5} The second most common combination is the detection of thrombus in both RCA and LCx.⁵ Patients with multi-vessel coronary thrombosis have a high incidence of major complications, such as ventricular arrhythmias and cardiogenic shock, and are therefore more prone to develop out-of-hospital cardiac arrest. This could possibly offer an explanation of the higher rates of multiple coronary thromboses reported by autopsy data (approximately 50% of the STEMI cases) in comparison to the incidence reported by angiographic data (between 1.7 and 4.8%).^{5,6} So far, no recommendations exist regarding the revascularization strategy one should follow in the case of double coronary artery occlusion. The two culprit lesions of the presented patient seemed both very important. During the primary PCI, it was decided to treat first the technically easiest mid-LAD lesion in order to promptly decrease the myocardial ischemic burden before treating the more complex mid-RCA lesion. After TIMI III flow restoration in the LAD the patient remained hemodynamically and electrically stable during the remaining PCI in the RCA.

In the present case a Medina 1.1.0 distal LM bifurcation lesion was closely followed by a 0.0.1 LAD – D1 bifurcation lesion with a very tight, ischemia producing, fibro-calcific and resistant to crossing and balloon pre-dilatation ostial D1 stenosis that could not be left untreated. Sophisticated PCI tools were used to prepare this lesion for stenting (such as a very low-profile balloon for initial crossing and an appropriately sized scoring balloon for final pre-dilatation). A minimum two-stent strategy was chosen with provisional side-branch stenting for the LM bifurcation and mandatory stenting of the D1 ostium with a mini-crush technique. A dedicated side branch stent could have been used alternatively (but only

if available and appropriately designed for the given anatomy).

While single bifurcation lesions are common, cases of two consecutive bifurcation lesions (where both side branches should be wired and treated with final kissing balloon to obtain a technically optimal result) are sporadic. Such a scenario is mainly pertinent to LM plus LAD – D1 bifurcation lesions. Patients with unprotected LM disease are at high risk of major cardiovascular events, such as fatal arrhythmias and cardiogenic shock. Although the evolution of DES and PCI techniques used in such patients seem to offer comparable results with coronary artery bypass surgery, unprotected LM distal bifurcation lesions require technically challenging interventions and can be associated with poor long-term outcomes.⁷ Whereas several reports support a single-stent technique in these cases,⁸⁻¹⁰ a large number of investigators have suggested more complex stenting strategies to improve the angiographic and clinical outcomes of distal LM bifurcation lesions.^{7,11} Zhang et al. comparing single versus double stent interventions, showed that both approaches had comparable results regarding PCI success and safety, reporting though, a lower ostial residual stenosis of LAD with single stent technique and lower ostial residual stenosis of LCx with double stenting.¹² According to a recent review, specific angiographic characteristics, such as the plaque distribution, the ostial LCx disease severity and the bifurcation angle, may be considered as indicators for a best suited double stenting strategy.⁷ In addition, the currently available intracoronary imaging modalities (mainly intravascular ultrasound and optical coherence tomography) offer a better evaluation of the severity of bifurcation lesions and can serve as useful guidance tools to select the appropriate PCI technique.^{13,14}

Among the available bifurcation 2-stent intervention techniques the mini-crush, its variant double-kissing crush and the culotte are the most popular. Although several trials have compared the long-term outcomes of these techniques in non-LM bifurcation lesions, there is a paucity of data regarding the optimal two-stent strategy for distal LM bifurcation disease. The DK-CRUSH III study has shown though that the culotte technique was associated with higher rates of major adverse cardiac events at one year, mostly secondary to increased target vessel revascularization, in comparison to the double-kissing crush technique.¹⁵ Finally, the use of dedicated stents in conjunction with conventional drug-eluting stents has been proven both successful and safe in LM bifurcation lesions,^{16,17} though further prospective trials to assess their long-term outcomes are required.

Conclusion

Multi-vessel coronary thrombosis and flow impairment is an infrequent but sometimes encountered clinical scenario among patients with ACS. So far, no specific recommendations exist regarding the revascularization strategy one should follow during PCI in such cases and the appropriate procedural steps should be decided promptly and on a case-by-case basis by the interventional cardiologist according to his experience and skills.

True coronary bifurcation lesions are associated with poorer angiographic and clinical outcomes while the optimal treatment strategy remains equivocal. In case of two consecutive bifurcation lesions the possible anatomic variations multiply, while PCI techniques and procedural steps for any given variation are not standardized as for single bifurcation lesions.¹⁸ Familiarization with PCI techniques of common bifurcation lesions and a complete toolbox in the catheterization laboratory can contribute to overcome challenging cases of consecutive bifurcation lesions, especially the ones involving the left main stem.

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