

Precision PCI in the Contemporary Era: NIRS-IVUS Optimized PCI

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Case Presentation

A 68-year-old man with hypertension and hyperlipidemia (untreated) presented with crescendo angina over the past week and resting chest pressure radiating to the jaw and left arm. Physical exam and electrocardiogram were unremarkable.

Pertinent labs included an LDL of 139 mg/dL, lipoprotein(a) of 10 mg/dL, hemoglobin A1C of 5.8%, and a troponin I peak of 1.48 ng/mL (normal <0.04 ng/mL). An echocardiogram showed normal left ventricular ejection fraction with no regional wall motion abnormalities. The patient was initiated on aspirin, ticagrelor, atorvastatin, and an intravenous heparin infusion, and urgent cardiac catheterization in the setting of non-ST elevation myocardial infarction (NSTEMI) was performed.

Diagnostic Evaluation

Coronary angiography showed 90% proximal stenosis of the obtuse marginal 2 (OM2) consistent with acute plaque rupture and 70-80% severe diffuse mid-OM2 disease distal to the culprit lesion. The proximal-mid

left circumflex (LCx) had moderate 50-60% disease, while OM1 was a moderate-caliber vessel without significant disease (Figure 1A). The left anterior descending (LAD) (Figure 1B) and right coronary artery (RCA) (Figure 1C) had moderate disease.

Percutaneous Coronary Intervention (PCI)

Access: 6 French right radial artery

Wire: Sion Blue (Asahi Intecc)

Pre-dilation: 2.5 x 20 mm semi-compliant balloon

Near infrared spectroscopy-intravascular ultrasound (NIRS-IVUS) pre-stent: Proximal and distal reference vessel diameter (media-media) were 4.0 mm and 2.7 mm, respectively. No significant calcium was observed. The culprit lesion had a plaque burden of 78% and minimum lumen area (MLA) of 1.6 mm² (Figure 2, left-hand panel); the lipid core burden index (LCBI 4-mm segment) was 892, and there was additional lipid-rich plaque in the proximal LCx (Figure 2, right-hand panel).

The Makoto HD NIRS-IVUS (Nipro Medical) supported a more precise and optimized PCI strategy.

Stents: Overlapping 3.0 x 28 mm and 2.5 x 33 mm drug-eluting stents (proximal to distal) were placed from the ostial LCx to the mid OM2, post-dilated from 2.5 mm distally gradually to 3.75 mm proximally.

Post-PCI Data

Post-PCI IVUS showed stents were well-expanded and well-apposed. Final angiography demonstrated no residual stenosis in the LCx-OM2 system (Figure 3). Physiologic assessment of the non-culprit vessels showed a fractional flow reserve (FFR) of 0.89 in the LAD and 0.91 in the RCA.

The Impact of NIRS-IVUS on Clinical Decision-Making

The Makoto HD NIRS-IVUS (Nipro Medical) supported a more precise and optimized PCI strategy in several ways:

1. Its exceptional resolution and visualization provided enhanced operator confidence in balloon and stent sizing.

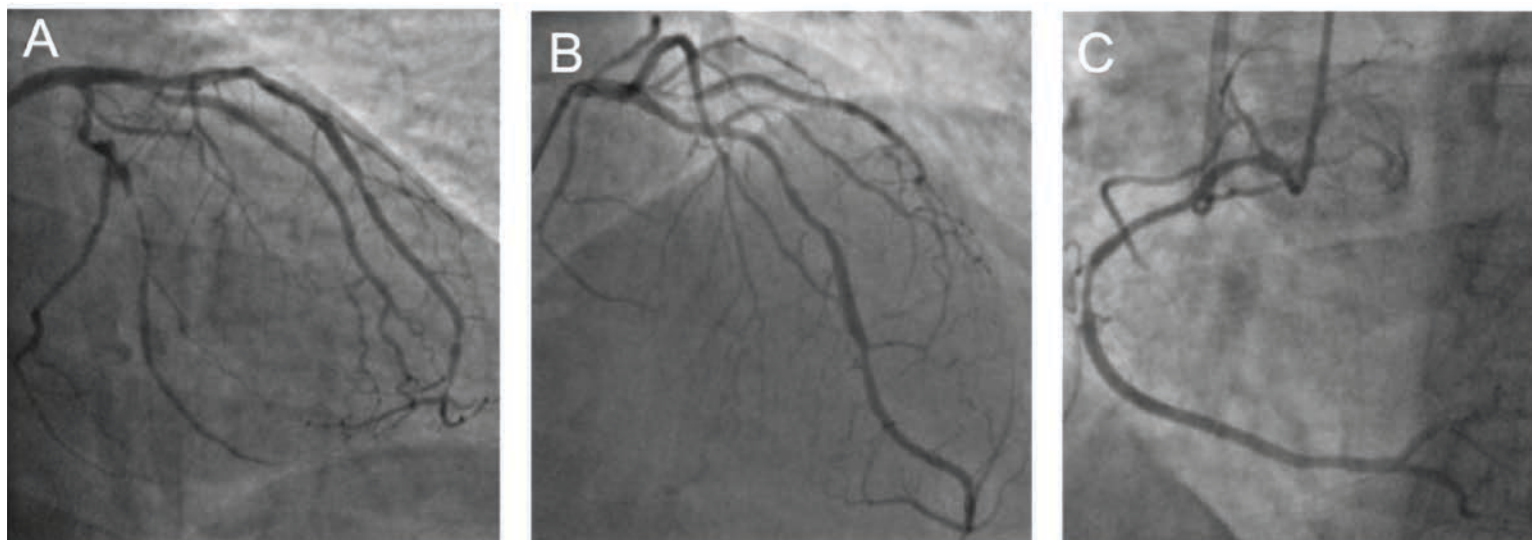


Figure 1. Baseline coronary angiography. (A) 50-60% disease can be seen in the proximal-mid LCx and no significant disease in OM1. Moderate disease is shown in the (B) LAD and (C) RCA.

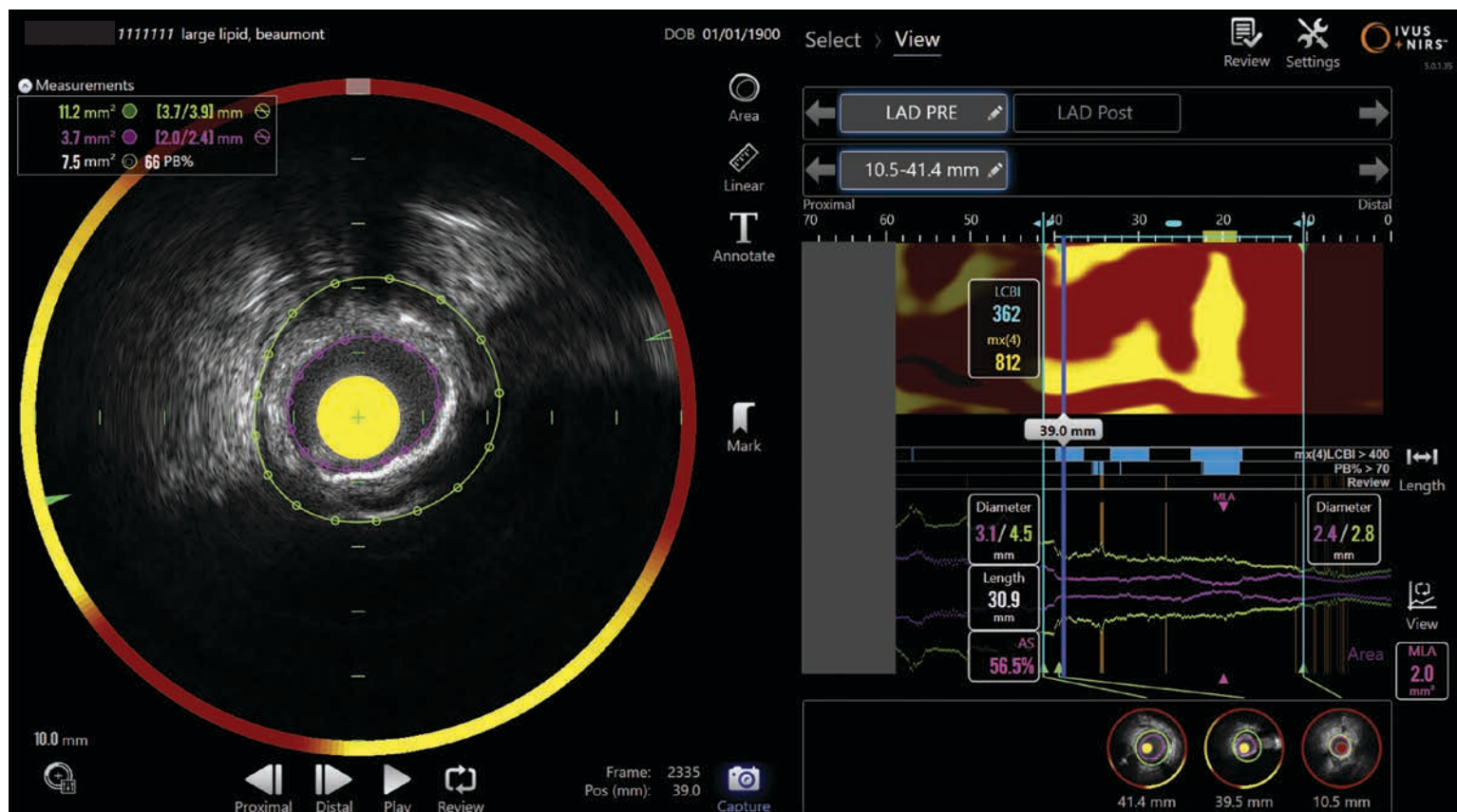


Figure 2. NIRS-IVUS of the LCx-OM2. (Left) Culprit lesion had a plaque burden of 78% and minimum lumen area (MLA) of 1.6 mm². (Right) Lipid core burden index (LCBI 4-mm segment) is 892. Additional lipid-rich plaque can be seen in the proximal LCx.

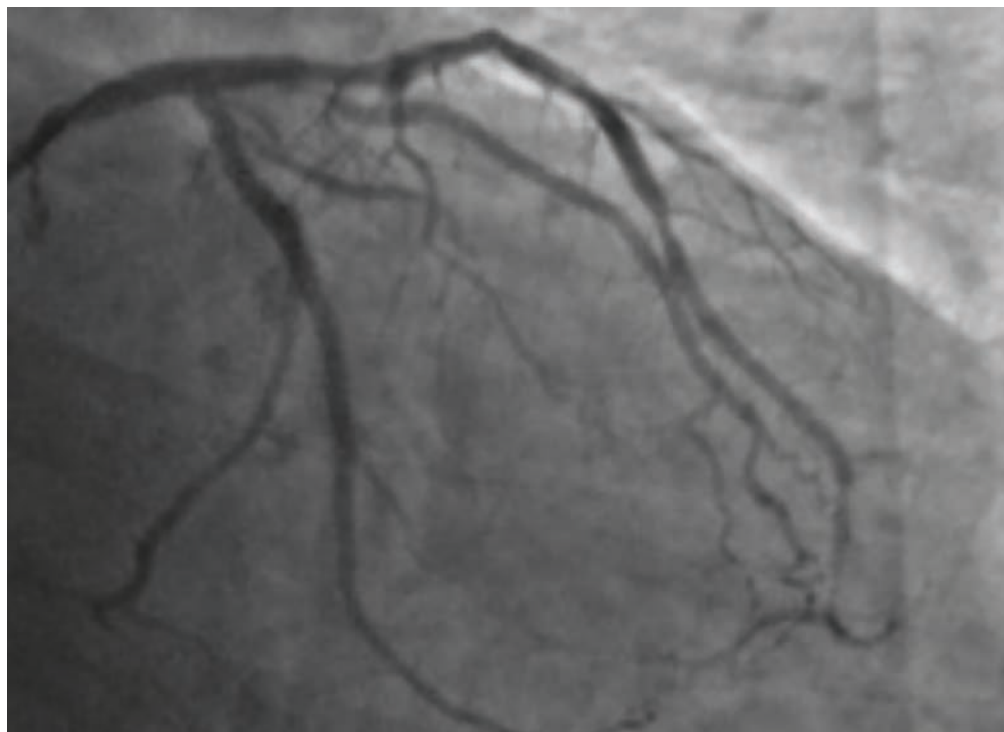


Figure 3. Post-PCI coronary angiography demonstrates no residual stenosis.

2. Optimized stent landing zones (i.e., covering lipid-rich plaque, if feasible): Lipid-rich plaque in the stent margins is associated with a higher risk of in-stent restenosis. Thus, the overlapping proximal stent was adequately sized to ensure coverage of the lipid-rich plaque in the proximal LCx.

3. Selective escalation of anti-lipid therapy: Given the patient's very high LCBI and evidence of moderate plaque throughout the entire coronary tree, aggressive lipid-lowering therapy with a PCSK9 inhibitor in addition to a statin was initiated. ■

This case is supported by Nipro Medical.

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