

Modification

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[Target Lesion] A 72-year-old gentleman with diabetes mellitus, hypertension, previous anterior wall myocardial infarction and CABG (2001) presented with recurrent angina despite PCI to the SVG-LAD performed eight months earlier for an anterior wall myocardial infarction. Echocardiography demonstrated moderate left ventricular systolic dysfunction (LVEF 42%) with moderate mitral regurgitation. Coronary angiography demonstrated native triple vessel disease with severe calcification involving the left main coronary artery and proximal LAD, together with a heavily calcified mid-LAD chronic total occlusion. The previously stented SVG-LAD remained patent, without native collateral filling, due to competitive graft flow. Dual injection demonstrated a blunt proximal cap, lesion length >20 mm, severe calcification, tortuosity (>45°) and a septal branch adjacent to the proximal cap. IVUS demonstrated extensive circumferential calcium extending from the left main into the LAD with a calcific left main nodule. The lesion was classified as a J-CTO score of 4. Considering the patient's previous CABG and the limited long-term durability of SVG intervention, PCI to the native LAD CTO was planned.

[Strategy] The procedure was performed through bilateral femoral arterial access. The left main coronary artery and SVG-LAD graft were engaged with 7 Fr guiding catheters. A hybrid antegrade-retrograde strategy was planned from the outset because the heavily calcified proximal cap was anticipated to make antegrade cap penetration challenging. Finecross microcatheters with Fielder FC wires were advanced simultaneously to the proximal and distal CTO caps through the native LAD and SVG-LAD, respectively. Selective tip injections through both microcatheters delineated the CTO anatomy and confirmed lesion length. Although a hybrid strategy was planned, the initial crossing attempt was performed retrogradely through the SVG. Sequential retrograde wire escalation was unsuccessful. Antegrade wire escalation was then continued. During manipulation, the antegrade Fielder FC wire tracked into the subintimal plane and advanced to within approximately 3–5 mm of the distal CTO cap. With the antegrade wire positioned close to the distal CTO cap, reverse CART was prepared as the anticipated re-entry strategy. However, continued antegrade escalation, Miracle wire successfully entered the lumen of the retrograde Finecross microcatheter, achieving antegrade tip-in before reverse CART became necessary. The antegrade wire was balloon-trapped within the SVG guide catheter to facilitate wire exchange and device support. Despite successful guidewire crossing, neither the Finecross microcatheter nor a 1.0-mm balloon could be advanced across the CTO because of the extreme calcific burden, confirming a microcatheter- and balloon-uncrossable lesion. A Rotawire was exchanged with the Finecross advanced to its maximum achievable position. Rotational atherectomy was initially attempted using a 1.5-mm burr, which failed to cross the resistant calcified segment. The burr was downsized to 1.25 mm. During advancement, true burr entrapment occurred. Initial attempts to activate the burr were unsuccessful because of repeated stalling, and en bloc traction failed to disengage the burr. The distal wire position was changed, the guide catheter was disengaged to alter the traction angle, repeated burr activation attempts were performed, and the drive-shaft connector was withdrawn to reduce system tension before the burr was successfully retrieved. Haemodynamic stability was maintained throughout because myocardial perfusion was preserved by the patent SVG-LAD graft. Rotational atherectomy was subsequently completed successfully using the 1.5-mm burr. IVUS confirmed circumferential calcium involving the proximal LAD, Mid LAD and calcific nodule in the left main with evidence of Rotablation. Lesion preparation was continued with a cutting balloon, followed by high-pressure non-compliant balloon dilatation. Although the critical mid-LAD CTO segment expanded satisfactorily, persistent dog-boning of the non-compliant balloon in the proximal LAD indicated resistant deep calcium. Intravascular lithotripsy using a 3.0-mm Shockwave balloon was therefore performed, resulting in satisfactory plaque modification. A drug-eluting stent was implanted in the mid-LAD with the proximal landing zone just distal to the major septal branch. A second drug-eluting stent was implanted from the left main into the proximal LAD with overlap. Proximal optimisation technique was performed using a 4.0-mm non-compliant balloon under IVUS guidance. Final IVUS demonstrated satisfactory stent expansion and apposition. Following successful native vessel revascularization, the SVG-LAD was coil embolized using a VortX Diamond coil to eliminate competitive graft flow.

[Final Result] Final angiography demonstrated TIMI III flow through the native LAD without residual stenosis, perforation or distal embolization. Final IVUS demonstrated a left main minimum stent area of 10.52 mm², proximal LAD minimum stent area of 7.09 mm², and mid-LAD minimum stent area of 5.25 mm², confirming satisfactory stent expansion and apposition. The patient remained haemodynamically stable throughout the procedure and was discharged in stable condition on guideline-directed medical therapy.