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# How to Do It?: Percutaneous Treatment of a Severely Stenosed and Calcified Left Main Stem Bifurcation

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| **Case Summary** | A frail 87 year-old lady presented with rest angina associated with widespread ECG change and troponin release. She failed attempts at medical therapy and therefore was referred for coronary intervention on the basis that she was not a surgical candidate. |
| **Investigation** | Coronary angiography demonstrated heavily calcified coronary arteries with critical disease at the distal left main stem bifurcation extending into the proximal segments of both LAD and circumflex. |
| **Diagnosis** | Acute coronary syndrome with extensive calcific coronary artery disease in the left main stem bifurcation. |
| **Management** | Sequential rotational atherectomy of the left main stem bifurcation followed by 'Y'-stenting using three Xience Prime drug eluting stents. |

**Keywords** Calcification • Left main stem disease • Rotational atherectomy • Bifurcation

## Case Report

### Presentation of the Case

A frail 87 year-old lady of New Zealand European origin was referred for cardiac catheterisation due to refractory angina pectoris preventing hospital discharge. She had been admitted five days previously with rest pain associated and high sensitivity troponin T 199 ng/L (reference range <14).

Our patient had a background of congenital deafness, hypertension, dyslipidaemia and severe osteoarthritis with multiple failing joints. Consequently her mobility was poor and she was considered relatively frail. She had a long documented history of ischaemic heart disease. Coronary angiography had first been performed in 2003 following a non-ST segment elevation myocardial infarct and reported moderate diffuse atheroma which was treated medically. In 2010 due to worsening effort angina, she underwent dobutamine stress echocardiography with no evidence of inducible ischaemia. During the six months prior to her present admission she had worsening angina resulting in several hospital admissions. On each occasion she was treated conservatively.

In the early phase of the present admission she was again treated conservatively. However she had ongoing symptoms despite maximal tolerated medical therapy: aspirin 100 mg o.d., clopidogrel 75 mg o.d., enoxaparin 70 mg b.d., metoprolol CR 47.5 mg o.d., felodipine 5 mg o.d., simvastatin 40 mg o.d. in addition to an intravenous infusion of glyceryl trinitrate. On arrival in the catheter laboratory she was pain free, but had widespread ECG changes affecting multiple territories. Her heart rate was 60 bpm and blood pressure 110/62 mmHg. Her creatinine was 87 (Cockroft-Gault Glomerular Filtration Rate 47 mL/min), haemoglobin 11.9 g/dL and weight 74 kg.

Coronary angiography was performed via the right radial arterial access route after administration of 5000 IU heparin and using a 6 French Radifocus II hydrophilic sheath (Terumo, JP) with 5F JL4 and LCB diagnostic catheters (Boston Scientific, MA, USA). Cineangiography of the left coronary system demonstrated extensive calcification and severe stenosis of the distal left main stem (LMS) with extensive disease in the proximal segments of both left anterior descending (LAD) and left circumflex coronary arteries

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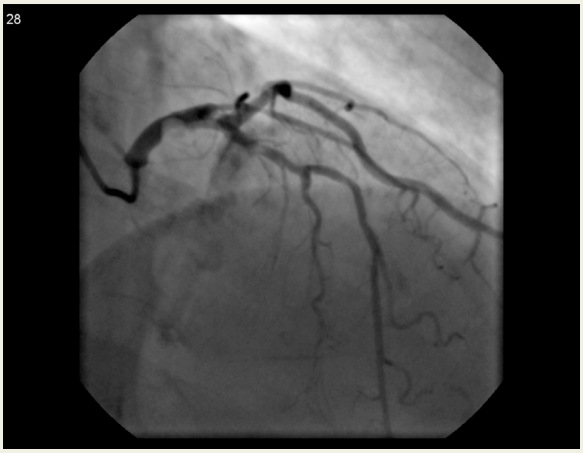

**Figure 1** Cranial 35° projection. Severe calcific stenosis in left main stem and proximal LAD. Note also extensive calcification of mitral valve apparatus.

(Figs. 1 and 2). The right coronary artery was also heavily calcified but had only mild diffuse atheroma. Left ventricular function had not been assessed on the present admission but systolic function six months earlier had only been mildly impaired.

## How Did I Treat?

This lady was clearly very frail, had ongoing evidence of ischaemia and had failed valiant attempts at medical therapy. She was clearly not a surgical candidate; this had been discussed with the patient prior to her arrival in the catheterisation laboratory and she had already indicated that she was agreeable to high risk intervention as deemed appropriate. Our proposed strategy was sequential rotational atherectomy (RA) of the LMS to circumflex, followed by LMS to LAD. It was anticipated that a 'Y'-stenting strategy would be preferable given size mismatch between parent and daughter vessels.

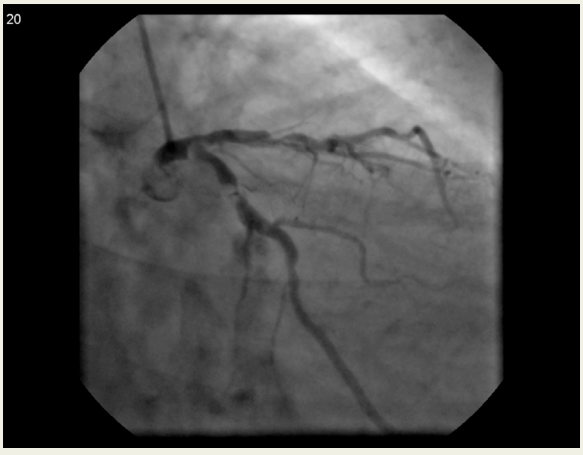

**Figure 2** Caudal 35° projection. Severe calcific disease in left main stem and proximal circumflex. Note also mismatch in calibre between LMS and daughter vessels.

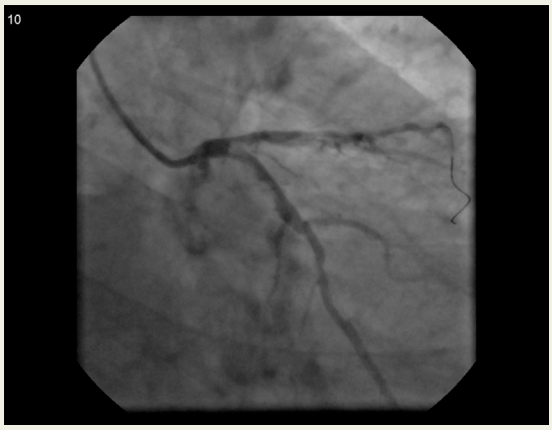

**Figure 3** Caudal 35° projection. Following rotational atherectomy balance wires in both LAD and circumflex. The Rotawire remains in situ. Successful debulking of lesions with preserved flow and without angiographic evidence of dissection. Note Ikari guide position without deep engagement of left main.

A 6-french Ikari left 3.5 guide catheter (Terumo) was chosen to prevent deep engagement and was positioned within the LMS ostium without pressure damping. The circumflex was wired (surprisingly easily) with a 0.009″ floppy Rotawire (Boston Scientific). We proceeded to debulk using a 1.5 mm burr at 190,000 rpm using gentle but constant pressure on the lesion for a total of 90 s in 30 s bursts. Having proven absence of extensive dissection with a repeat cineangiogram, the Rotawire was then withdrawn and resited into the LAD. Rotational atherectomy was again performed using the same 1.5 mm burr at 190,000 rpm using the same technique for a total of 200 s. The patient remained haemodynamically stable throughout both these manoeuvres.

Both LAD and circumflex were then wired with separate High Torque Balance wires (Abbott, IL, USA). Cineangiography confirmed improvement in lesion appearances with preserved distal flow (Fig. 3). Further lesion preparation was undertaken with serial pre-dilatation of LMS and proximal segments of both daughter vessels using a 2.5 mm × 12 mm non-compliant (NC) Trek Balloon (Abbott). Complete angiographic expansion was observed with each inflation.

A 3.5 mm × 12 mm Xience Prime (Abbott) drug eluting stent was advanced on the LAD wire in to the LMS, the distal marker positioned carefully at the bifurcation and deployed at 16Atm (Fig. 4). The jailed circumflex wire was removed and used to rewire the same vessel through the stent. A kissing balloon inflation was performed at the distal end of the LMS stent using 2 mm × 12 mm NC Trek balloons into both LAD and circumflex. The left main stent was then post dilated using a 4.5 mm × 12 mm NC Trek balloon to 26 Atm.

A 3 mm × 23 mm Xience Prime stent was then positioned within the circumflex, the proximal marker being placed precisely at the ostium and a 2.5 mm × 15 mm NC Trek balloon placed in the ostium of the LAD (Fig. 5). The stent