



Very Late ISR in a 4 mm Stent: Mechanism of “Plaque Creep”

A 70 year old male presented with history of class III effort angina since 3 months. He has had angioplasty and stenting to his LAD in 2008 when a 4 mm x 15 mm DES was deployed in proximal LAD. His angiogram revealed total occlusion of LAD in the stented segment (Video 1, Fig 1). The post occlusion LAD was seen to be filling retrogradely via collaterals from RCA (Video 2). The LAD lesion was crossed with a Cross-it 100 guide wire and microcatheter support (Video 3). The occlusion was dilated with a 1.5 mm followed by 2.5 mm balloon. IVUS revealed a well expanded stent with presence of extensive atherosclerosis burden in a fairly long segment beyond the stent which was contiguous with the neoatherosclerotic burden of ISR (Video 4). A well expanded stent was also well visualised by stent boost (Video 5, Fig 2). The stented segment and the diseased portion beyond the stent was then serially dilated with appropriate sized balloons (based on IVUS sizing). Finally a 4 mm x 15 mm cutting balloon (Flextome) was used to cut the neo-atheroma inside the stent (Fig 3) and a long 3.5 mm x 33 mm DES was deployed to cover the previously stented segment and the diseased segment beyond the stent (Video 6). The proximal segment of the stent was then post-dilated by a 4.5 mm NC balloon. The final angiographic result was excellent (Video 7).

Discussion

In-stent restenosis (ISR) in a 4 mm stent is uncommon. Neo-atherosclerosis by “Plaque creep” is the commonest pathology seen in very late (17 years in this case) ISR after the index procedure. Here, the atherosclerosis begins at the edge or well outside the stent and eventually creeps into the stented segment. The following stent edge-related phenomenon are also important.

- Residual plaque burden >50% at stent edges
- Geographic miss
- Positive remodeling outside the stent

These allow continuity of atherosclerotic disease from native vessel to in-stent segment. Factors that favor “plaque creep” into stent are as follows:

- Long stents
- Inadequate lesion coverage
- High plaque burden at edges
- Diabetes
- CKD
- Poor lipid control
- First-generation DES (but also seen with newer DES over time)

Plaque creep is very rare in early DES ISR. It is increasingly recognised as the underlying mechanism in late ISR and is the commonest mechanism in very late ISR.

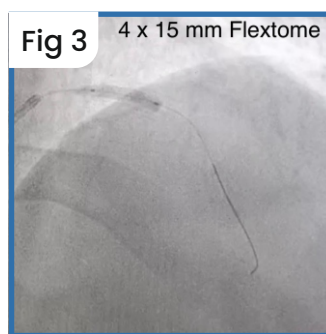
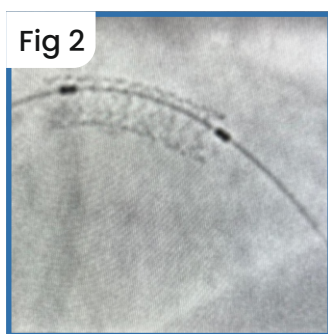
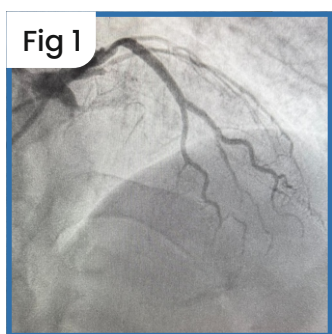
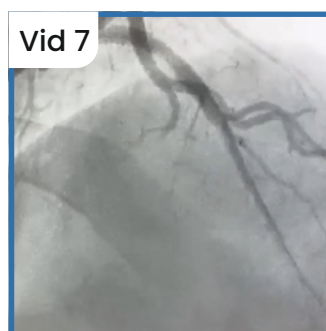
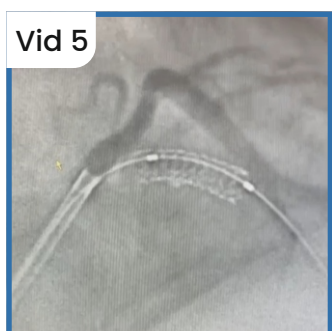
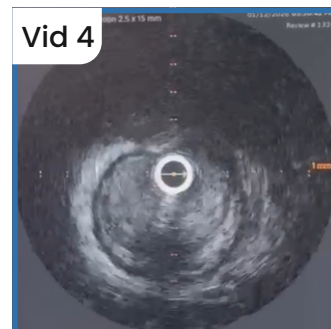
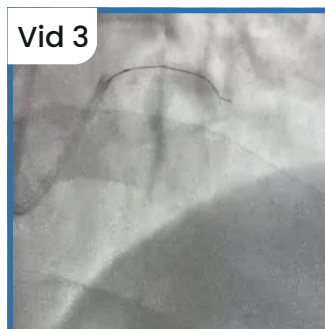
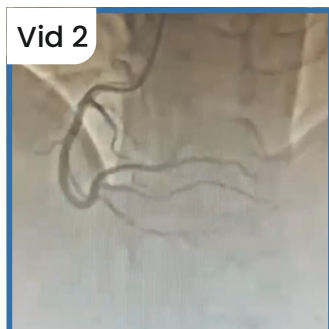
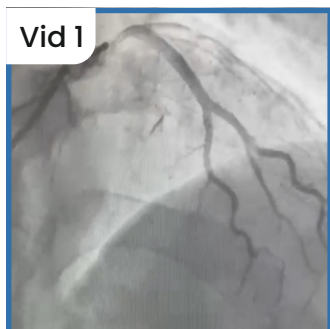
Clinical implications

Prevention

- Adequate lesion preparation
- Avoid geographic miss
- IVUS-guided stent sizing & landing in healthy segments
- Aggressive lipid lowering (LDL < 55 mg/dL)

Treatment

- Imaging-guided therapy
- DCB or DES depending on mechanism
- Treat edge disease adequately
- Address systemic atherosclerosis



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