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Case Report

Late stent collapse identified with OCT – An underdiagnosed mechanism of restenosis?



IHJ

Nimit C. Shah^{a,*}, Peter D. O'Kane^b, Jehangir N. Din^b

^a Sir H.N. Reliance Foundation Hospital and Research Centre, Mumbai, India ^b Dorset Heart Centre, Royal Bournemouth Hospital, Bournemouth BH7 7DW, UK

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ABSTRACT

Very late stent recoil is a rare albeit recognized phenomenon leading to subsequent in-stent restenosis. Angiography alone may not be adequate in making the diagnosis, and intravascular imaging with optical coherence tomography (OCT) is far superior in confirming the diagnosis and guiding subsequent management.

We describe a case with interesting coronary angiogram and OCT images demonstrating very rare diagnosis of the late stent collapse. These images provide a valuable insight into a novel mechanism responsible for late target lesion failure.

These images highlight the importance of modern intra-coronary imaging techniques in understanding the mechanisms underlying target-lesion failure, and guiding appropriate management.

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A 76-year-old gentleman presented with an acute coronary syndrome. He had been asymptomatic since percutaneous coronary intervention to the right coronary artery (RCA) with a bare metal stent two years earlier (Fig. 1A and B).

Emergent coronary angiography demonstrated that the RCA stent had an abnormal hazy appearance at the distal edge (Fig. 2A). Optical coherence tomography (OCT) demonstrated collapse and circumferential mal-apposition of the distal stent segment from the vessel wall, presumably secondary to complete loss of radial strength (Fig. 3). The minimal luminal area of the stent outflow was reduced to 1 mm² with neointimal hyperplasia within the stent, including the collapsed segment. After sequential predilatation, a 3.75×20 mm drug eluting stent (Promus Element, Boston Scientific, USA) was deployed overlapping the distal end of the original stent. Repeat OCT confirmed satisfactory stent apposition (Fig. 4).

Very late stent recoil is a rare albeit recognized phenomenon leading to subsequent in-stent restenosis. We cannot exclude early stent recoil. However, the stent collapse is unlikely to have occurred at the time of stent insertion. It was a straightforward noncalcific lesion, and we would not expect any significant immediate recoil. The stent was inserted at a hinge point in the artery, and we have hypothesized that this has led to stent fracture with subsequent loss of radial strength of the distal segment and collapse at an undetermined time point after stent insertion. Although stent fracture is widely recognized, complete collapse with circumferential mal-apposition as seen in this case is extremely rare.

Late or very late restenosis is often due to neoatherosclerosis, which was seen in our case. Generally, neointima on OCT can be heterogeneous or homogenous. This can be identified more

* Corresponding author.

E-mail address: drnimitcshah@yahoo.co.uk (N.C. Shah).

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Fig. 1 – (A) RCA angiogram shows tight lesion in the mid vessel (2 years ago). (B) Final result after PCI to RCA with 4.0×18 mm Tsunami (bare metal) stent (2 years ago).



Fig. 2 – (A) Recent RCA angiogram shows abnormal haziness and filling defect in the distal end of the stent. (B) Final result after OCT guided PCI to the RCA with 3.75 × 20 mm Promus Element (drug eluting) stent.



Fig. 3 – OCT shows complete loss of radial strength of the distal stent edge with minimal luminal area of within the stent of 1 mm^2 in a 3.5 mm² vessel with evidence of neointimal hyperplasia.



Fig. 4 - Final OCT result shows that stent struts are well apposed.

readily using OCT compared with IVUS. Typical OCT features of late or very late restenosis due to neoatherosclerosis include instent lipid laden neointima +/– necrotic core, in-stent calcification and macrophage accumulation, more likely heterogeneous appearance (although this is variable), and occasionally neointimal plaque rupture. While some of these features can be visible on IVUS, tissue characterization with IVUS should be interpreted with caution as the technology lacks sufficient resolution to allow reliable determination of plaque composition. OCT has superior axial resolution (10–20 μ m) enabling better characterization of neointimal tissue.

This highlights the importance of contemporary intravascular coronary imaging in understanding the mechanisms underlying angiographic anomalies and guiding subsequent management.

Conflicts of interest

The authors have none to declare.