

Optical Coherence Tomography Analysis of Des In-Stent Restenosis Presenting As Stable and Unstable Angina: A Comparison – A Case Series

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ABSTRACT

Rationale: Drug-eluting stents (DES) have been demonstrated to successfully reduce the rate of instant restenosis and Target Vessel Revascularization (TLR) at least during short term follow up, and instant restenosis (ISR) is generally considered to be a stable process. However, recent studies have reported 1/3rd of patients with ISR present with Acute coronary syndrome (ACS).

Objective: We report in this case series, two patients, one presenting with stable angina and another presenting with unstable angina. We compared Optical Coherence Tomography (OCT) analysis of these two subsets of patients.

Findings: In case of stable angina patients the Optical Coherence Tomography (OCT) findings suggested homogenous character of restenotic plaque with high back scatter in majority of tissue, regular lumen shape, no micro vessels, no intraluminal material. There was nothing suggestive of intimal rupture, no Thin Cap Fibrous Atheroma (TCFA) containing neo intima.

In case of unstable angina patients, OCT characterization further reveals both homogenous layered and heterogeneous characteristic of restenotic plaque with mixed high and low backscatter along with irregular lumen shape in between. Presence of micro vessels was also noted, and areas of lipid core were noted. There are areas of intimal rupture, TCFAs and presence of macrophages.

Conclusion: In patients of DES instant restenosis, the presentation as stable or unstable patient can be analysed by OCT characterization of the resultant plaque.

Keywords

Ocular Coherence Tomography (OCT), In stent restenosis, Stable angina, Unstable Angina.

Case Presentations

Case 1

59 years old female, a case of post percutaneous transluminal coronary angiography (PTCA) to left anterior descending artery (LAD) done with second generation DES (Cobalt-chromium everolimus eluting stent; Expedition; Abbott Vascular, Santa Clara, California) previously done one year back. Patient is a known case of hypertension, diabetes, severe left ventricular (LV) dysfunction (Ejection Fraction (EF) - 20%), came with stable angina on exertion since one month (class 3).

Coronary angiography was done which revealed long diffuse restenosis starting from native proximal LAD and involving the stent. Pre PTCA-OCT run was taken which revealed that the diameter of previously deployed stent was from 1.9mm to 2mm in proximal part. The possible cause of ISR was under expansion in mid part and landing in proximal diseased segment. The OCT findings (Figure 1B) revealed homogenous character of restenotic plaque with high back scatter in majority of tissue, regular lumen shape, no micro vessels, no intraluminal material. There was no intimal rupture, no thin Cap Fibrous Atheroma (TCFA) containing neo intima. The lesion was crossed with Balance Middle Weight (BMW) wire. Then distal DES (Promus Element Everolimus Eluting Coronary Stent, Boston Scientific, Massachusetts, USA) 3*36mm deployed at 10 atm. Post dilatation done with 3*15 mm NC voyager balloon at 18 atm. Then a proximal DES (Promus

Element Everolimus Eluting Coronary Stent, Boston Scientific, Massachusetts, USA) 3.5*23mm was deployed from proximal to mid dilated and procedure completed with post dilatation with 3.5*15 mm NC voyager balloon at 20 atm. (Figure 1A)

Case 2

On the other hand, second case presented to us with rest angina since 15 days, diagnosed as unstable angina. He was a post PTCA patient, LAD PTCA was done 3 years back with onyx DES (Resolute onyx Zatarolimus-Eluting coronary stent, Medtronic, Dublin, Ireland) resolute 3 years back. Patient is a known case of hypertension, non-diabetic, LV dysfunction (EF 35%). Coronary Angiography (CAG) revealed tight diffuse in-stent restenosis of LAD. Pre PTCA OCT was done. OCT run revealed (Figure 2B) diameter of proximal and distal stent was between 3-3.5mm, which was adequately expanded. The OCT characterization further reveals both homogenous layered and heterogeneous characteristic of restenotic plaque with mixed high and low backscatter. There was irregular lumen shape in between. Presence of micro vessels was also noted, and areas of lipid core were noted. There are areas of intimal rupture, TCFA and presence of macrophages.

The procedure was completed with predilatation followed by two DES (Cobalt-chromium everolimus eluting stent; Expedition; Abbott Vascular, Santa Clara, California) and (Promus Element Everolimus Eluting Coronary Stent, Boston Scientific, Massachusetts, USA). One in proximal to mid LAD, another from LAD to left main. After stent deployment post dilatation in left main with 5*8mm NC balloon (Mozec NC; Meril) at 24 atm was done to complete the procedure. (Figure 2A).

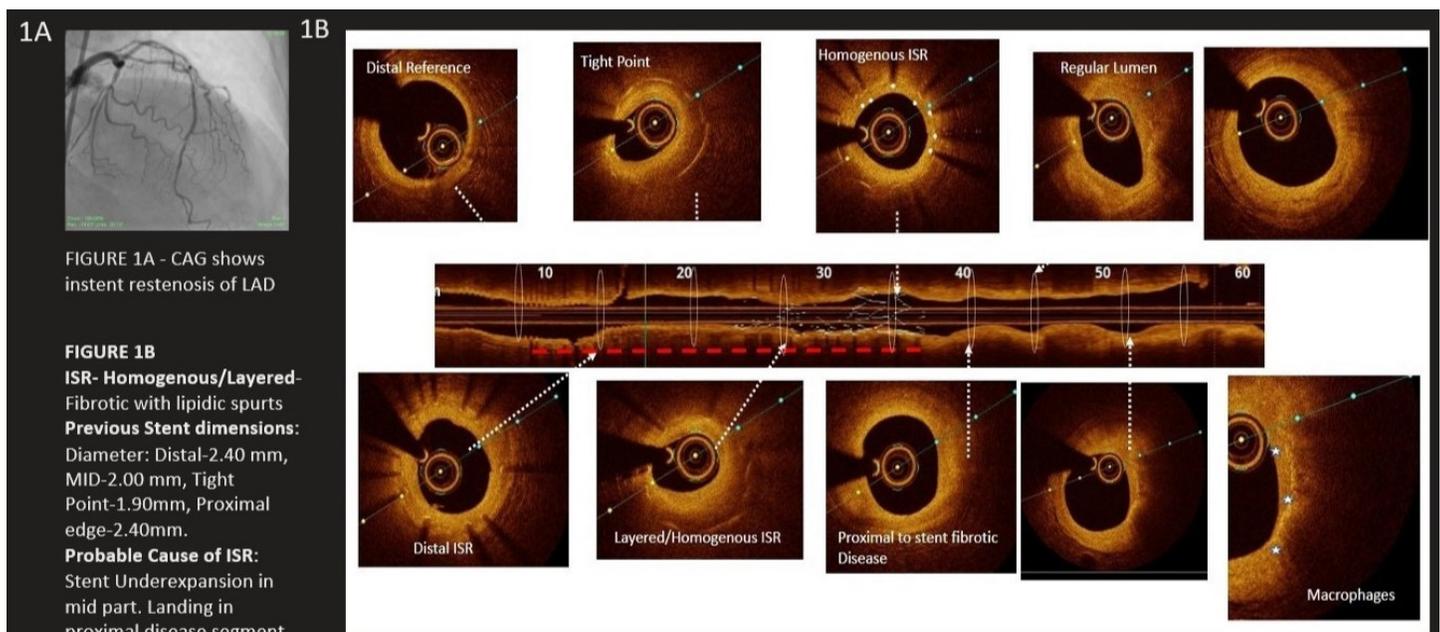


Figure 1a: CAG shows in-stent restenosis of LAD.

Figure 1b: ISR – Homogenous/Layered- Fibrotic with lipidic spurts.

Previous Stent dimensions: Diameter: distal- 2.40mm, MID-2.00mm, Tight point – 1.90mm, proximal edge- 2.40mm.

Probable cause of ISR; Stent Underexpansion in mid part. Landing in proximal disease segment.

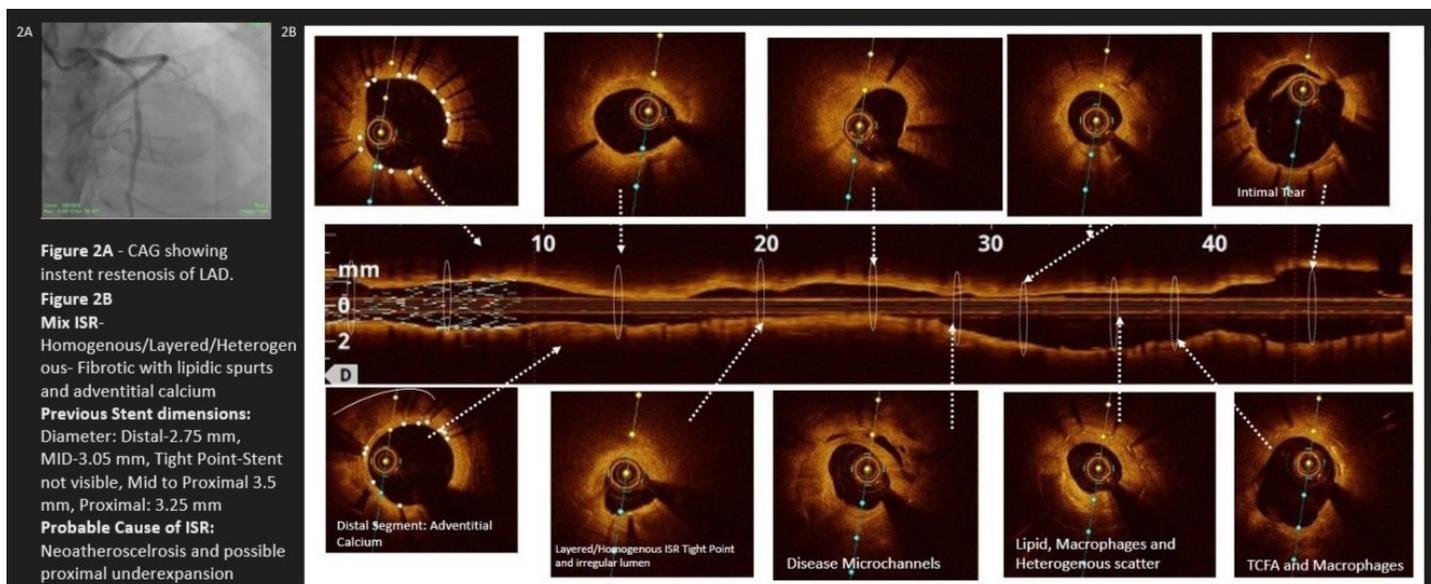


Figure 2a: CAG showing in-stent restenosis of LAD.

Figure 2b: Mix ISR – Homogenous/Layered/heterogeneous- fibrotic with lipidic spurts and adventitial calcium.

Previous Stent dimensions: Diameter; distal- 2.75mm, MID- 3.05mm, Tight Point- Stent not visible, Mid to Proximal 3.5mm, Proximal: 3.25mm.
 Probable cause of ISR: Neoatherosclerosis and possible proximal under expansion.

Discussion

1/3rd of patients with in-stent restenosis present with acute coronary syndromes [1,2]. Furthermore, there is emerging histological and angiographic evidence of late de novo in-stent new atherosclerosis [3-7]. Recently Takano et al suggested that TCFA neointimal disruption and thrombus are detected by OCT were more frequently found late, greater than 5 years in comparison with earlier less than 6 months. In our patients also, the second case presented with unstable angina had stent implanted 3 years back with OCT features of neoatherosclerosis. The higher incidence of in-stent TCFA containing neo intima macrophages, neo intimal rupture in unstable angina patient supported the concept that these OCT findings were similar to vulnerable plaque in the native coronary artery [8,9]. In our case, we do not have histopathological correlation but we can hypothesize that the patients who present as unstable angina have heterogeneous character of restenotic plaque as it correlates with aging plaque. Also characters like irregular lumen shape, micro vessels, TCFAs and areas of intimal rupture with presence of macrophages are more frequent in patients who present with unstable angina.

Conclusion

The present case report identifies the ability of the OCT to identify differential patterns of restenotic tissue after stenting. It also generated a hypothesis that with time in-stent neo atherosclerosis produces unstable characteristics into the plaque and that can be possible cause of late stent thrombosis seen in DES patients. This

hypothesis needs to be confirmed in larger studies comparing unstable with stable patients.

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Supplementary Material

[Link to the PowerPoint presentation](#)