Correspondence

Letter by Foin et al Regarding Article, "Edge Effect From Drug-Eluting Stents as Assessed With Serial Intravascular Ultrasound: A Systematic Review"

To the Editor:

We read with great interest the article by Wakabayashi et al[1] recently published in *Circulation: Cardiovascular Interventions*. Wakabayashi et al reviewed the mechanisms of edge restenosis, bringing insights into the possible underlying causes of this phenomenon analyzing results from several drug-eluting stent (DES) trials.

They suggest that the principal mechanism of edge restenosis in bare metal stent as in DES is through plaque shift and intimal hyperplasia proliferation beyond the stent edges, within the first millimeter from the stent.

Interestingly, the authors noted from several trials, including paclitaxel-, sirolimus-, zotarolimus-, and everolimus-eluting stents that despite significant reduction in restenosis with DES compared with bare metal stent control, stent edge restenosis was nearly equally frequent in both DES and bare metal stent and could not be resolved at the proximal edge of the stents by drug elution from the stent.

The authors suggest that drug distribution downstream of the stent may produce a beneficial stent effect on the lumen distal from DES compared with bare metal stent. They propose this drug diffusion distal to the edge of the stent as a possible explanation of the difference between proximal and distal restenosis, which also might explain the relatively high restenosis rate at the proximal edge of DES observed in trials.

Based on our experience with optical coherence tomography,[2,3] we strongly believe that another important phenomenon may explain the difference: incomplete stent apposition is a particularly frequent problem, and incomplete stent apposition assessed by intravascular ultrasound has been reported in up to one third of treated segments after DES implantation,[4] affecting more frequently the proximal edge of the stent compared with the distal edge of the stent.[2,4,5] Stents deployed across the origin of a side branch or covering long segments of tapering vessels (left anterior descending) are at particular risk, and postdilation with larger balloon is only effective if the balloon matches the proximal vessel diameter and is also covering the proximal edge. If the stent is incompletely expanded with its struts not in contact with the vessel wall, drug will diffuse from the coating within the bloodstream, instead of diffusing to the intima and, therefore, prevent smooth muscle proliferation. Postprocedure malapposition may resolve by the rapid proliferation of neointimal tissue filling the gap between the stent strut and the vessel wall,[6] which may thereby limit the risk of stent thrombosis but at the same time facilitate edge restenosis. If on the opposite, the stent struts are well apposed and embedded in the tissue (such as almost systematically observed at the distal edge of the stent), the drug can diffuse from the strut to the vessel and thereby prevent intimal hyperplasia.

The incidence of proximal edge incomplete stent apposition and its direct impact of edge restenosis should be assessed using high-resolution intravascular optical coherence tomography. Comparison of stent segment postimplantation and at follow-up may identify the respective propensity of partially deployed stent, its role on restenosis, and whether complete stent apposition at the proximal edge can actually limit such edge effect.

Disclosures

None.

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