Letter by Pérez de Prado et al Regarding Article, “Delayed Coverage in Malapposed and Side-Branch Struts With Respect to Well-Apposed Struts in Drug-Eluting Stents: In Vivo Assessment With Optical Coherence Tomography”

To the Editor:

We read with great interest the article by Gutiérrez-Chico et al., which reports delayed coverage of incompletely apposed and non-apposed side-branch struts with respect to well-apposed struts in drug-eluting stents as assessed by optical coherence tomography. The relationship between incomplete stent apposition and delayed strut coverage has previously been explored. However, the study of fate of nonapposed side-branch struts is quite original.

These struts, placed across the origin of side branches, have systematically been excluded from analysis in most of the published optical coherence tomography studies. Intuitively, they were deemed not suitable to be covered. These new results abrogate that theory, demonstrating by optical coherence tomography analysis that up to 78.1% of these struts appear covered 9 to 13 months after drug-eluting stent implantation. This finding is indeed relevant, but a question arises: What is covering those struts? As the authors acknowledge, optical coherence tomography covering is not a surrogate for neointimal healing. The vascular healing process involves the participation of the coagulation system and many types of cells in different phases, all of them covering the stent struts. Optical coherence tomography is unable to distinguish between fibrin, giant cells, granulomatous reaction, and degree of endothelialization. Until we can confirm recent findings with optical density analysis that claims being able to distinguish between neointima and fibrin/thrombus, we can only search for clues. Can the authors offer any extra information that helps in this endeavor? Is there any difference in the thickness of the coverage between apposed and nonapposed struts?

We have previously reported that these nonapposed side-branch struts show a high reendothelialization rate in a swine coronary model. Our data coincide with the results presented by Gutiérrez-Chico et al. Nonapposed side-branch struts show delayed endothelialization but a parallel time course to the rest of struts. The endothelial cells that cover these nonapposed struts necessarily come from circulating endothelial progenitor cells because there is no chance of repopulation from the surrounding area. The animal models use young, healthy individuals as opposed to human patients with defective healing mechanisms. Whether these findings predict a similar behavior in the clinical setting is still unknown.

It is common to observe nonapposed struts placed over the side-branch origin; up to 41% of the analyzed stents were nonapposed. In daily practice, the rate should be even higher because the analysis refers to randomized studies with moderate stent lengths. Therefore, the clinical relevance of the status of these struts is not negligible. Whatever the coverage of these struts is, some issues must be considered. One of them emerges from the use of the new biodegradable scaffolds. The struts of these prostheses, also those located over the side-branch takeoff, finally disappear as expected. Do they show a strut coverage similar to that observed in metallic struts? Can the breakdown of the scaffold cause clinically relevant side-branch embolism? We guess this topic will prompt further analyses of these floating struts in different situations.

Disclosures

None.

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References


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Circulation. 2012;125:e458
doi: 10.1161/CIRCULATIONAHA.111.064493
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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