



Contradictory Shear Stress Distribution Prevents Restenosis after Provisional Stenting for Bifurcation Lesions

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Background: Endothelial shear stress is one of the local hemodynamic factors suspected in the development of coronary atherosclerosis in bifurcation lesions. In patients with provisional stenting, the endothelial shear stress (SS) distribution is unknown.

Objective: The aim of this study was to investigate the magnitude and distribution of the SS of coronary bifurcation lesions stenting by the provisional approach.

Methods: Ten consecutive patients were included in this study. Quantitative coronary analysis, flow study, and three-dimensional computational analysis with the aid of the commercial software CD STAR-CCM+ were done before and after the provisional stenting procedure and also 8 months later.

Results: Clinical and angiographic follow-up were available in all patients. No patient had a side branch (SB) stent. At the 8-month follow-up, no major adverse cardiac event (MACE) occurred. There was also no clinical and angiographic restenosis. Before PCI, the distal main vessel (MV)-lateral, and the SB-lateral subsegments had relative nonsignificant lower SS value (4.08 ± 2.78 Pa and 4.35 ± 5.04 Pa, respectively) when compared to other segments. After 8-month follow-up, sustained decreased SS value was shown in the distal MV-lateral segment (4.08 ± 2.78 ? 1.68 ± 1.65 Pa), when compared with significantly increased SS value in the SB-lateral subsegment 4.35 ± 5.04 ? 16.50 ± 40.45 Pa). The explanation is that after stenting in the MV, the flow was redistributed immediately after percutaneous coronary intervention (PCI) and reversed back to its original 8 months later. However, the growth of the fibrous tissue causing in-stent restenosis (ISR) is prohibited by sirolimus on the stent struts. In contrast, in a branch opened up by plain old balloon angioplasty (POBA), the flow did not change much, the flow could even be worse because it is shifted to the MV after the cross-sectional area of the MV improved by stenting. However, thanks to POBA, there is increased fibrous tissue formation, enough to increase the SS and prevent further accumulation of cell and cholesterol needed for more restenosis.

Conclusion: In the provisional approach, low endothelial SS correlated with no restenosis for

patients who underwent stenting of the MV, while a contradictory combination of high SS and no restenosis was seen in the SB after only POBA. The mechanism of prevention of restenosis in the SB is by increasing the SS while in the MV, the mechanism of prevention of ISR is secondary to sirolimus on the stents struts.

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