Uncovered non-apposed side-branch struts in a bifurcation lesion: a nidus for late stent thrombosis

A 67-year-old patient was admitted with acute lateral ST-elevation myocardial infarction. The patient had a history of a bifurcation procedure to his left anterior descending (LAD) and 1st diagonal (D1) vessels 5 years ago, because of stable angina symptoms. The technique used was main branch (LAD) cross-over stenting with a Firebird-2\textsuperscript{TM} (Microport Shanghai, China) 3.5 $\times$ 13 mm drug-eluting stent without post stent kissing balloon inflation (KBI). The angiographic results were excellent (online video 1). The patient received dual antiplatelet treatment (DAPT) for 14 months – when he had an unremarkable routine follow-up angiogram (online video 2); and then, he continued aspirin treatment until the event. Urgent coronary angiography at the event demonstrated a filling defect at the ostium of D1 (Panels B\textsubscript{1}-\textsubscript{3}, online video 3). To further assess this defect, optical coherence tomography (OCT) was performed in both LAD and D1. OCT in the LAD demonstrated a well-expanded stent; there was no strut malapposition and all the struts were well covered – apart from these at the orifice of D1 (Panels A\textsubscript{1}-\textsubscript{3}, online video 4). OCT in D1 showed a disease-free vessel with a thrombus at its ostium starting from the struts of the stent implanted in the LAD (Panels C\textsubscript{1}-\textsubscript{3}, online video 5). In view of these findings, kissing balloon inflation was performed in LAD and D1 (3.75 $\times$ 10 mm in LAD with 2.0 $\times$ 15 mm in D1), to open the orifice of D1 and appose the struts over the side branch to the vessel wall. The patient was discharged with dual antiplatelet therapy for one year and standard secondary prevention therapy; up to now, he is symptom-free at three years follow-up.

To elucidate the pathophysiological mechanisms leading to this event, anatomical model reconstruction of the LAD-D1 vessels was performed from the OCT and angiographic data acquired at the time of the event (Panel D). The model was then used to perform a computational blood flow simulation, from which the shear stress and shear rate distribution were estimated (Panels E\textsubscript{1}-\textsubscript{4}). As shown in Panels E\textsubscript{3}-\textsubscript{4}, the uncovered struts at the ostium of D1 caused flow separation that resulted in increased shear rate (>5000 s\textsuperscript{-1}) in this area; while distally to the carina, at the D1, there was an area of recirculation zones and low shear stress (<1 Pa) values (online videos 6–8).

A high shear rate leads to the accumulation of platelets and adhesion molecules toward the vessel wall – creating a prothrombotic environment and changing the globular structure of the von Willebrand factor, leading to its elongation that facilitates platelet adhesion. Moreover, a low shear stress environment causes endothelial dysfunction and acute inflammation that promotes platelet aggregation and thrombosis.\textsuperscript{1}

In our case, the struts at the orifice of D1 appear to create flow disturbances and high shear rate with further recirculation zones at the vicinity of this segment that resulted in low shear stress values at the wall of D1, near the carina. We hypothesize that this hemodynamic environment and possible external triggers that increased blood thrombogenicity led to an event at 5 years post stent implantation.\textsuperscript{2}

The optimal treatment strategy of this patient is open to debate. Prolonged dual antiplatelet therapy can be considered in patients with acute coronary syndrome; but not in this case, as the index procedure was performed for stable angina symptoms.\textsuperscript{3,4} In addition, registry data have failed to demonstrate a prognostic benefit of KBI after single stent implantation in the main vessel and its major side branch.\textsuperscript{5} Based on this evidence, the 15\textsuperscript{th} consensus document from the European Bifurcation Club does not recommend routine KBI in this setting.\textsuperscript{6} Moreover, bench studies have shown that the recrossing location of the side branch guidewire has significant implications on stent apposition post KBI. Recrossing through the proximal cell at the orifice of the side branch is associated with strut deflection leading to an unfavorable hemodynamic milieu, while distal cell recrossing is related to better vessel coverage and strut apposition.\textsuperscript{7} The OPTIMUM study that randomized 110 patients to angiography and distal wire recrossing under the OCT guidance showed a better stent apposition at the bifurcation site in the OCT-guided group.\textsuperscript{8} Further research is required to examine whether this approach improves short- and long-term outcomes before advocating OCT-guided KBI after single stent percutaneous coronary intervention in a bifurcation lesion.

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Conflicts of interest

All authors have no conflicts of interest to declare related to this article.

Appendix A. Supplementary data

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