

and then decreased at 24 h (1.5 ± 0.14 ; $p < 0.05$). The areas of myocardial edema decreased from 3 h ($25.8 \pm 7.4\%$) to 6 h ($21.5 \pm 8.9\%$; $p < 0.05$) and then stayed the same after 6 h (6 h $21.5 \pm 8.9\%$; 12 h $21.7 \pm 7.7\%$; 24 h $22.3 \pm 8.4\%$; $p = \text{NS}$). Infarction areas at 24 h were significantly reduced, compared with those at 6 h ($18.1 \pm 6.1\%$ vs. $20 \pm 3.9\%$; $p < 0.05$), whereas salvaged myocardial areas increased from 6 h to 24 h ($1.5 \pm 1.2\%$ vs. $4.3 \pm 1.1\%$; $p < 0.05$).

Ischemia with reperfusion will induce reperfusion injuries that consequently provoke fluid overload and swelling, which result in an abrupt increase in T2 that then progressively returns to baseline within days. Without reperfusion, T2 will increase further (1). This can explain our findings that nonreperfusion was associated with higher T2 values and T2 values of early post-reperfusion (3, 6, and 12 h) have been much higher than those at 24 h.

The infarcted areas were larger in the non-reperfusion group than the reperfusion group, and infarcted areas of reperfusion heart decreased from 6 to 24 h, from which we may conclude that early reperfusion during the evolution of myocardial infarction is essential for myocardial salvage. However, there was no difference in salvaged myocardial areas between the reperfusion and nonreperfusion group in our study. This may be explained by reperfusion injuries and short delay between occlusion and reopening of the infarct-related artery (2).

In conclusion, compared with nonreperfusion ischemia, early reperfusion may not change salvaged myocardial areas, which should be used cautiously as an endpoint in clinical trials investigating the success of reperfusion strategies.

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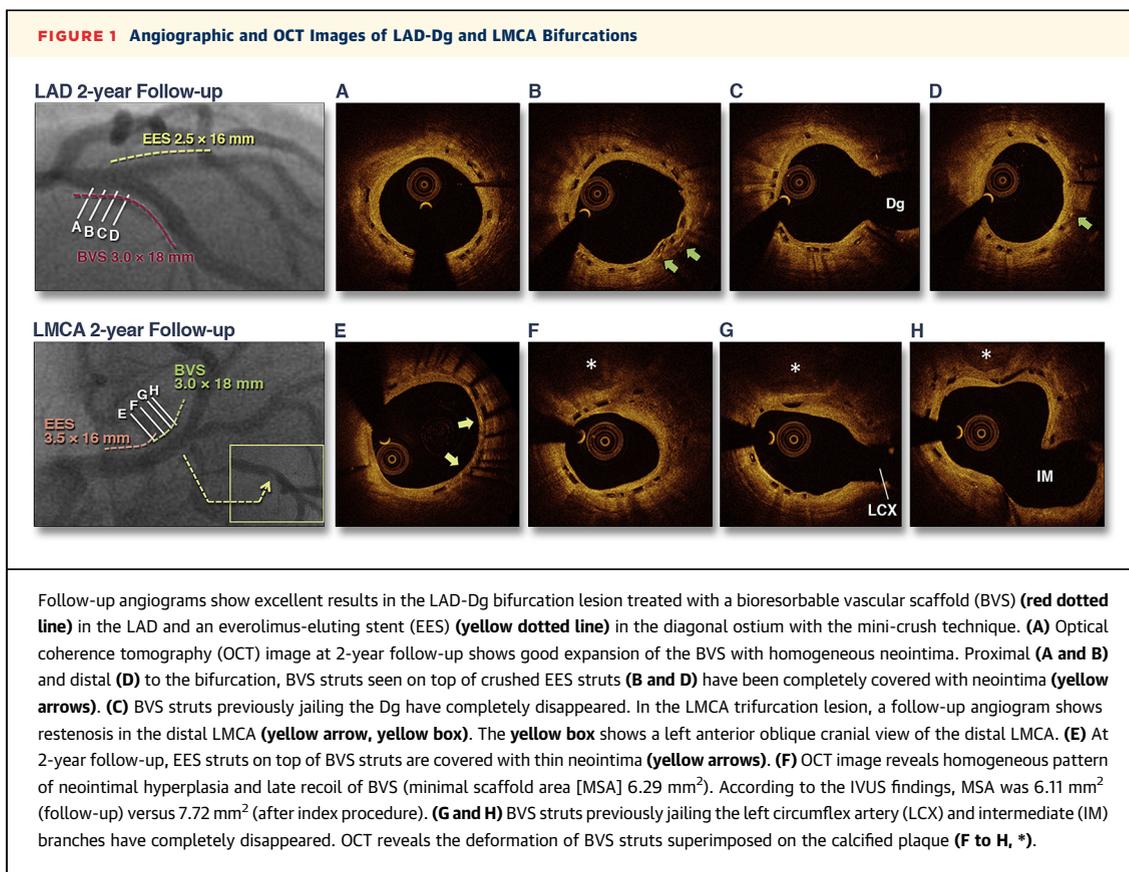
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Two-Year Follow-Up OCT Images of 2 Bifurcation Lesions Treated With Bioresorbable Vascular Scaffolds



Diagnostic angiogram of a 66-year-old man presenting with stable angina revealed significant lesions in the left main coronary artery (LMCA) trifurcation and the left anterior descending artery (LAD) diagonal bifurcation (Figure 1). In the latter, after deployment of a 2.5×16.0 mm Promus element (Boston Scientific, Natick, Massachusetts) with mini-crush technique in the diagonal with initial kissing balloon inflation (KBI), a 3.0×18.0 mm ABSORB bioresorbable vascular scaffold (BVS) (Abbott Vascular, Santa Clara, California) was implanted in the proximal LAD without final KBI. In the LMCA trifurcation lesion, a 3.5×18.0 mm ABSORB BVS was implanted with single crossover technique, jailing the left circumflex and intermediate arteries. This was followed by deployment of a 3.5×15.0 mm Xience PRIME (Abbott Vascular) in the proximal to mid LMCA.

Coronary angiography and optical coherence tomography (OCT) (Illumien Optis, St. Jude Medical, St. Paul, Minnesota) were performed at 24 months' follow-up because of recurrent angina. Coronary angiography showed in-scaffold restenosis in the distal LMCA. OCT showed BVS struts previously jailing the side branch ostia to have completely disappeared in both bifurcation lesions. The fact that BVS struts were still visible on the wall of the distal LMCA but not over the ostia of the side branches could be explained by either a more rapid reabsorption of BVS in the free flowing bloodstream or bulk scaffold erosion and remnant embolization. Despite the absence of symptoms since the index procedure and a current angiogram showing Thrombolysis In Myocardial Infarction flow grade 3 with no evidence of distal emboli, sporadic embolization of small scaffold segments cannot be excluded. In the LAD bifurcation, BVS struts on top of the crushed metal struts have been completely covered with neointima (Figures 1B and 1D). In the LMCA lesion, OCT revealed late recoil of the BVS overlying an area of calcified plaque (1) with scaffold deformation and homogeneous neointimal hyperplasia (Figures 1F to 1H).



Follow-up OCT images clearly revealed the mechanism of in-scaffold restenosis (combination of neointimal hyperplasia and late scaffold recoil) and provided further insight in the treatment of bifurcation lesions with the single crossover technique. Disappearance of BVS struts jailing the side branch ostia may reduce very late restenosis in this location and facilitate very late side branch access should new disease develop in the jailed vessel. This case also demonstrates the importance of meticulous lesion preparation with rotational atherectomy or scoring balloon in case of BVS implantation for calcified lesions.

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Accurate Conductance-Based Post-Dilation Balloon Catheter Sizing



Balloon underexpansion during percutaneous coronary intervention is a major reason for stent underdeployment, which occurs in part because the predicted relationship between balloon pressure and diameter is not always realized in vivo (1). We herein describe a new device that accurately measures balloon cross-sectional area (CSA) during inflation.

The CB catheter provides an accurate, real-time digital display of the balloon CSA based on continuous electrical conductance recordings made inside the balloon. Within each balloon, there are 4 radiopaque platinum-iridium electrodes mounted on the catheter body. The 2 outer electrodes inject a small and alternating electrical current (136 μ A_{pp},