SFR : Scaffold Failure of BVS

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Although current metallic drug-eluting stents (DES) have remarkable reduction of neointimal proliferation and occurrence of stent thrombosis, late stent failure including late catch-up phenomenon or neoatherosclerosis remained a clinical issue in patients with coronary artery disease. Recently, bioresorbable vascular scaffold (BVS) has been introduced to improve the late complications after metallic DES implantation. It completely disappears two to three years after implantation and shows lumen enlargement and recovery of vasomotion in coronary lesion treated with BVS. With these promising scenario, BVS is being widely used in real-world population with complex lesions. However, several cases of scaffold thrombosis have been reported at early and late clinical phrases that was not seen in the previous clinical trials. The Gauging Coronary Healing With Bioresorbable Scaffolding Platforms in Europe (GHOST-EU) registry reported that definite scaffold thrombosis occurred in 2.1% of cases at 6 months after BVS implantation, which was in a line with other BVS registry data. Thus, there were much concerns regarding a higher rate of scaffold thrombosis compared to metallic DES thrombosis. Intracoronary imaging including optical coherence tomography or intravascular ultrasound enable to evaluate the underlying mechanisms of scaffold thrombosis, which were not detected by angiography, because of its higher resolution in the treated lesions. Previous clinical and imaging studies of metallic DES showed that early stent thrombosis were associated with procedural factor such as underexpansion and undersizing of stent, longer stent length, and STEMI whereas discontinuation of dual antiplatelet therapy, neoatherosclerosis, incomplete vascular healing, and lower left ventricular function were predictors of late stent thrombosis. Although there was not enough data to assess pathophysiologic mechanisms of scaffold thrombosis, a recent single center experience reported that stent underexpansion and undersizing, inadequate antiplatelet

therapy were main factors of early scaffold thrombosis, whereas peristrut low-intensity area, suggesting peristrut vascular edema and inflammation, mainly contributed to the development of late scaffold thrombosis. Similarly, another investigator also showed that suboptimal findings including incomplete lesion coverage, underexpansion, incomplete stent apposition were the main factors for both early and late scaffold thrombosis. Importantly, a meta-analysis reported that scaffold thrombosis is likely to occur between 1 to 30 days after BVS implantation. Another aspect of scaffold failure of BVS is disruption of scaffold implantation. Because of polymeric material in BVS, it can unwind when deployed beyond maximum scaffold expansion limit, which should be paid attention during implantation. From a clinical standpoint, BVS optimization with skillful technique during implantation and continuation of dual antiplatelet therapy might be the best strategy for minimizing the scaffold failure. Further investigation involving Korean patients may be necessary to evaluate these observations after BVS implantation.