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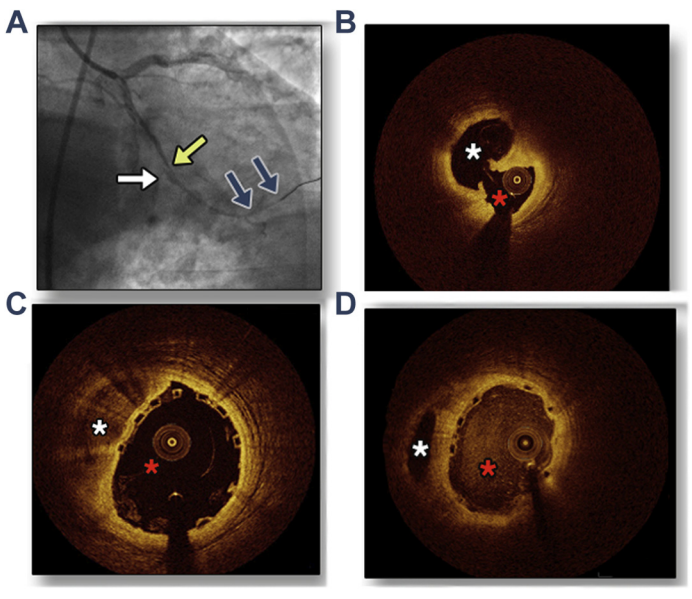
## Fate of Coronary Chronic Total Occlusion Recanalization via Subintimal Tracking With Bioresorbable Vascular Scaffolds: A Temporary Cage for a Permanent New Lumen?

A 62-year-old man was admitted as a result of stable angina Canadian Cardiovascular Society class III. Coronary angiography showed a chronic total occlusion (CTO) of the mid-left circumflex coronary artery (LCX), which was filled by grade III collateral flow from the right coronary artery (RCA), whereas the left anterior descending artery and RCA showed no significant stenoses (SYNTAX score = 9.5). Cardiac magnetic resonance demonstrated the presence of stress-induced ischemia in the posterolateral wall. Therefore, a CTO-percutaneous coronary intervention (PCI) was undertaken. An attempt to cross the lesion antegradely with an Ultimate Bros 3 guidewire (Asahi Intecc, Aichi, Japan) was performed, leading to vessel dissection and advancement of the guidewire in the false lumen and re-entering the true lumen more distally (Figure 1A). Optical coherence tomography (OCT) (Ilumien, St. Jude Medical, St. Paul, Minnesota) after pre-dilation revealed the entry point into the subintimal space and re-entering in the true lumen (Figure 1B). Excellent angiographic and OCT results were obtained with the implantation of 2 (3.0 × 18 mm and 3.5 × 28 mm) overlapping bioresorbable vascular scaffolds (BVS) (Absorb, Abbot Vascular, Santa Clara, California). Notably, subintimal scaffolding leading to true lumen collapse in a short segment (9.7 mm) was well demonstrated by OCT (Figure 1C). Follow-up angiography and OCT were performed 1 year after the index procedure, showing optimal results (Figure 1D). The scaffold was well apposed in the subintimal space, whereas the magnitude of neointimal hyperplasia was comparable between the scaffold located at the subintimal and intimal space.

In the present case, OCT clearly elucidated the mechanism of subintimal wire tracking and scaffolding. A previous study has shown the possible negative impact of subintimal metallic drug-eluting stent implantation, including late stent malapposition and fracture (1). The current case did not demonstrate late scaffold malapposition or exacerbated neointimal hyperplasia in the subintimal space. Although the scaffold enlargement demonstrated in our case was mostly due to hematoma absorption (i.e., healing), scaffold enlargement has recently been demonstrated late after BVS implantation (2). This particular feature of BVS could reduce concerns regarding late malapposition in complex scenarios, such as CTO-PCI (3). Because long-term follow-up is not available, we believe that BVS implantation in the subintimal space should be considered only in cases with short subintimal wiring without large side branch compromise, whereas in cases with long subintimal wiring with important side branch loss (i.e., subintimal tracking and re-entry technique), it would not be recommended because of the possible risk of reocclusion (4).

**FIGURE 1 Baseline, Post-Scaffolding, and 1-Year Follow-Up of BVS Implantation**




Representative pictures of baseline **(A and B)**, post-scaffold implantation **(C)**, and 1-year follow-up **(D)** of bioresorbable vascular scaffold (BVS) implantation are demonstrated. Baseline **(A and B)**: the angiographic image **(A)** illustrates the injection of the left coronary artery after the progression of the guidewire in the false lumen **(yellow arrow)**. The **white arrow** shows a side branch. The **blue arrows** highlight the position of the guidewire in the true lumen of the distal marginal branch. The cross-sectional optical coherence tomography (OCT) image **(B)** illustrates the subintimal space **(red asterisk)**, where the guidewire and the OCT catheter are located, and the true lumen **(white asterisk)**. Post-scaffold implantation **(C)**: in this OCT image, the BVS appears well expanded and apposed in the subintimal space, whereas hematoma is observed in the true lumen. Follow-up **(D)**: OCT image of 1-year follow-up is demonstrated. The scaffold is well expanded, apposed, and covered in the subintimal space.

To our knowledge, this is the first report of follow-up OCT assessments in CTO lesions with BVS including subintimal scaffolding. Although the results herewith demonstrated are promising, we acknowledge that longer-term follow-up imaging evaluations would be able to provide further insights into the assessment of scaffold reabsorption, whereas physiological assessments would be able to test whether vasodilation capabilities are recovered in this setting. Further validation of our findings is warranted.

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## Comparison of Echocardiography and CT for the Assessment of Aortic Stenosis Valve Area

We read with great interest the paper of Clavel et al. (1) in this issue of *iJACC*. The authors focused on the elliptic shape of the left ventricular outflow tract (LVOT) that was first shown by Baumgartner on transthoracic echocardiography in 1990 (2).

Clavel et al. compared the functional area obtained by the substitution of the echocardiographically calculated LVOT area by LVOT planimetry on 64-slice computed tomography (CT) in the continuity equation (aortic valve area [AVA] CT) with CT planimetry of the stenotic aortic valve (AVA plani).

Two important messages are provided in their paper: the threshold found for predicting mortality in patients with medically treated aortic stenosis; and the demonstration that some discrepancies between the mean aortic valve gradient and the functional AVA (AVA echo) is not the result of inadequacy in measuring the LVOT area.

However, CT LVOT planimetry should be performed exclusively in systole because, as shown for the aortic annulus (3), the ellipticity index is less in systole compared with that in diastole.

The good correlation of the AVA reached by the continuity equation using the LVOT area calculated by echocardiographic Doppler (AVA echo) with AVA CT is not surprising because the 2 areas share the same ratio (velocity time integral [VTI] outflow tract/VTI aortic valve). This is also true for the mean aortic valve gradient and AVA CT or AVA echo because this gradient is related to $1/AVA^2$ (CT or echo).

The functional AVA (calculated by the continuity equation) is a mean area in systole compared with the anatomic aortic valve area, which is a maximal instantaneous valve area in systole. When the valve is calcified and rigid, as in aortic stenosis, the aperture is delayed and limited, increasing the difference between the anatomic area and the functional area. The flow convergence downstream from the anatomic valve area also explains the lower value of the functional area.

Although this study adds to the reliability of echocardiographic Doppler assessment, CT planimetry of aortic stenosis remains an alternative in cases of poor acoustic window or when the continuity equation is inaccurate (high dynamic gradient of the LVOT). CT adds the incremental prognostic value of valve calcium scoring (4). There is a 0.2 $cm^2$ upward shift of the threshold to define significant stenosis on AVA CT planimetry compared with the AVA echo functional area (5).

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