

## Correspondence

## Biovascular scaffolds and reversible coronary aneurysm

Bernardo Cortese<sup>a,\*</sup>, Pedro Silva Orrego<sup>a</sup>, Kazuyuki Yahagi<sup>b</sup>, Renu Virmani<sup>b</sup><sup>a</sup> Interventional Cardiology, A.O. Fatebenefratelli Milano, Italy<sup>b</sup> CVPath Institute, Gaithersburg, MD, USA

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A 54-year-old male underwent coronary angiography for worsening unstable angina at our institution. The examination showed a tight stenosis of the marginal branch (Fig. 1A). After the lesion was predilated with a 2.5 mm semicompliant balloon, an Absorb 2.5/18 biovascular scaffold (BVS, Abbott Vascular, CA) was deployed and postdilated with a 3 mm noncompliant balloon at 10 atm, with good final angiographic result (Fig. 1B, Movie 1). After 11 months the patient experienced atypical effort angina and underwent control angiography, that showed an irregular and ectatic vessel at the site of previous BVS implantation, without signs of restenosis (Fig. 1C, Movie 2). Optical-frequency-domain imaging (OFDI, Terumo, Japan), performed at 0.2 mm intervals, confirmed the angiographic findings, with diffuse vessel ectasia (maximum diameter 4.1 mm) and localized areas of scaffold malapposition in the middle segment of the BVS. Both the proximal and distal segments appeared normal without signs of malapposition (Fig. 2, Movie 3). Our decision was to follow up the patient prolonging his dual antiplatelet therapy. After 23 months from index procedure the patient was asymptomatic and underwent control angiography, that showed reduced signs of ectasia at previous BVS implantation site (Fig. 1D, Movie 4). OFDI confirmed such findings, with the total disappearance of malapposed struts and a maximal lumen area of 7.3 mm<sup>2</sup>, compatible with a 2.5 mm scaffold postdilated with a 3.0 mm noncompliant balloon (Fig. 2, Movie 5).

To the best of our knowledge, this is the first report showing the size reduction of late coronary aneurysm resulted from organizing thrombus within the area of localized malapposition after BVS implantation [1]. BVS are constituted of a polymer backbone of poly L-lactide (PLLA) and have a strut thickness of 150 μm. Due to their conformation these

devices have shown to induce greater neointimal response early within the first months, and given their total reabsorption within 3 to 4 years after implantation in the porcine model, mild to moderate inflammation peaks at 12 months, which is observed at peak degradation time [2]. Therefore, it is not surprising that the malapposed area is now filled by an organized thrombus and size of aneurysm (ectasia) diminishes at 23 months. In the Absorb Cohort B study the rate of malapposition was described in 4% of cases at the time of implantation, however only 15% of the malapposed struts persisted after 6 months [3]. Conversely, late acquired (and irreversible) malapposition has been described with first and second generation DES due to vessel and lumen enlargement. Recently, Gori et al. investigated the incidence of coronary evaginations and peri-scaffold aneurysms following implantation of BVS [4]. Surprisingly, 54% (55 out of 102 lesions) had at least one evagination at 12 months. The presence of evaginations was strongly associated with malapposition ( $p = 0.003$ ) and strut fractures ( $p = 0.01$ ). Peristrut low-intensity areas were more frequently observed in the lesions with evaginations than without (53% vs. 26%,  $p = 0.005$ ), suggesting the presence of inflammatory cells around struts. During the first description of this phenomenon we hypothesized that the drug eluted by the BVS (everolimus), the inflammatory reaction resulted from scaffold degradation, or a scaffold fracture could be the cause for late BVS malapposition and aneurysm formation [1]. After 12 months, at intravascular imaging the areas of malapposition had totally disappeared and the ectasia of the vessel was reduced at angiography and OFDI analysis, showing a probable reversibility of this phenomenon thanks to the advanced stage of scaffold reabsorption which also showed greater neointimal formation.

In conclusion, late acquired scaffold malapposition is a possible complication, whose mechanism and clinical consequences are not yet definitely addressed. However, the natural fate of resorption with inflammation resulting in aneurysm and ectasia with subsequent healing of the currently available BVS could be responsible for the temporary nature of this phenomenon.

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.ijcard.2016.03.167>.

## Conflicts of interest, disclosures

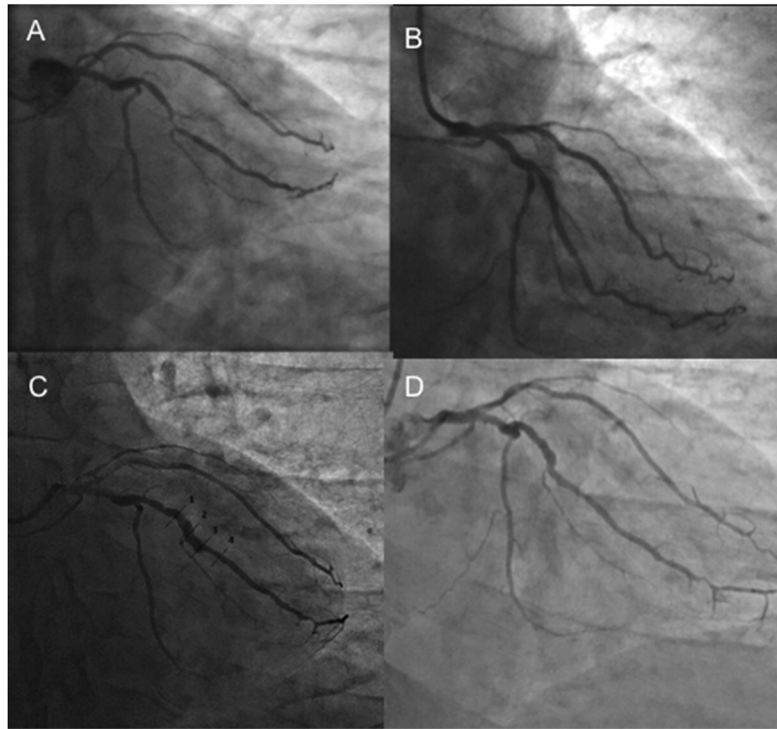
None.

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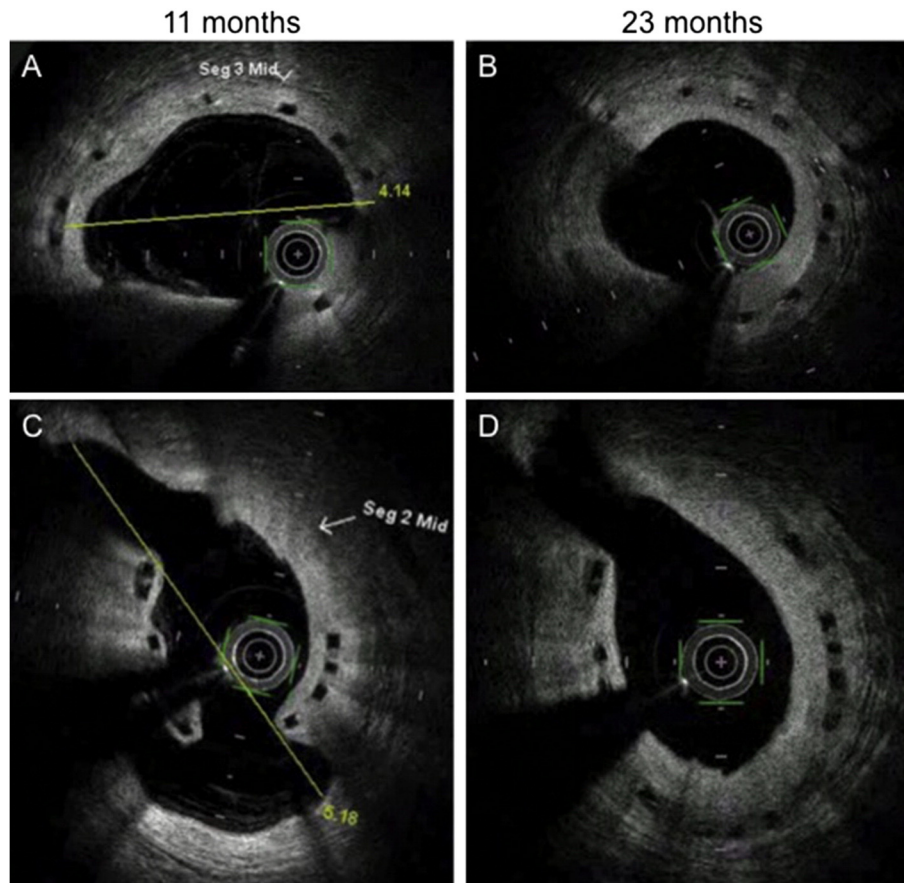
None.

\* Corresponding author at: Interventional Cardiology, A.O. Fatebenefratelli Milano, Bastioni di Porta Nuova 21, 20100 Milano, Italy.

E-mail address: [bcortese@gmail.com](mailto:bcortese@gmail.com) (B. Cortese).



**Fig. 1.** A: coronary angiography showing a severe stenosis at the middle segment of the marginal branch. B: final coronary angiography after BVS implantation. C: coronary angiography after 11 months, showing the broad ectasia at the site of BVS implantation. D: Twenty-three-month angiography showing the reduction of ectasia at BVS implantation site.



**Fig. 2.** OFDI images at 11 months and 23 months after implantation. A: ectatic lesion was observed at 11 months. B: corresponding section to A showing a decrease in lumen area by neointimal growth at 23 months. C: ectatic lesion with malapposed struts at the bifurcation observed at 11 months. D: corresponding section to C showing fully covered struts and the malapposed site is filled by an organizing thrombus. (Figure A and C reproduced from Cortese B et al. *Catheter Cardiovasc Interv.* 2015;86:678–81.

## References

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