

Neovascular Microchannels in Early Bioresorbable Vascular Scaffold Failure

Diego Arroyo, Serban Puricel and Stéphane Cook*

Department of Cardiology, University & Hospital Fribourg, Switzerland

Received: April 08, 2015; Accepted: May 11, 2015; Published: May 17, 2015

*Corresponding author: Stéphane Cook, Department of Cardiology, University & Hospital Fribourg, 1708 Fribourg, Switzerland, Tel: +41-26-4268140; Fax: +41-26-4268135; E-mail: stephane.cook@unifr.ch

Abstract

Bioresorbable Vascular Scaffold (BVS) are the latest devices used for percutaneous coronary lesion treatment that undergo polymer dissolution over 2 to 3 years. There is hope that by restoring early physiological processes, late stent complications may be diminished. We present a hypothesis-generating case of microchannel development after BVS implantation and subsequent stent failure. Microchannels are indicators of enzymatic plaque activity and vulnerability and have been documented in cases of neoatherogenesis and plaque rupture after drug eluting stent implantation.

Case Presentation

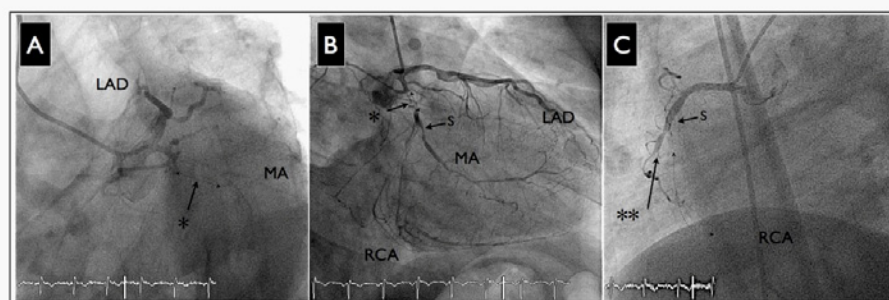
A 48-year-old male smoker with dyslipidemia and angina episodes upon mild exertion was diagnosed with a coronary three-vessel disease with Chronic Total Occlusions (CTO) of the mid Right Coronary Artery (RCA) and the proximal Left Circumflex Artery (LCx) (Figure 1). His global left ventricular function was preserved despite severe hypokinesis of the inferobasal wall. The patient refused surgical revascularization and underwent Percutaneous Coronary Intervention (PCI) of the RCA with implantation of 3 bioresorbable vascular scaffolds

(BVS; Two 3.0/28mm Absorb distally and one 3.5/28mm Absorb proximally, Abbott Vasc) (Figure 2A-2C).

The patency of the RCA was confirmed angiographically and by intravascular optical coherence tomography (OCT, Optis, St. Jude Medical, Figure 2D and 2E) one month later at the time of the LCx staged PCI. Interestingly, abundant microvessels were observed in the media and adventitia of the RCA at scaffold location. The patient was prescribed a lifelong treatment of aspirin 100mg/day, prasugrel 10mg daily for 12 months and rosuvastatin 10mg daily.

Repeat coronary angiography was performed at 7 months due to recurrent exercise induced chest pain. The angiogram showed an 80% RCA inscaffold restenosis where microchannels had previously been documented (Figure 2F). The lesion was treated with one Drug Eluting Stent (DES) implantation (3.5/18mm Xience Xpedition, Abbott Vasc) (Figure 2G and 2H). At 12 months, the patient is free from symptoms with a normal left ventricular function and absence of ischemia at exercise electrocardiographic testing.

Bioresorbable vascular scaffolds use a poly-L-lactide polymer that undergoes dissolution over 2 to 3 years via the Krebs cycle.



Arroyo et al., Figure 1

Figure 1: Baseline Coronary Angiogram.

Occlusion (*, dotted arrow) of Ramus Circumflex (RCx), stenosis (S) of the Marginal Branch (MA) and collaterals to right coronary artery (RCA) in "spider" view (Panel A) and RAO/cranial view (Panel B), Panel C: Occlusion (*, dotted arrow) of RCA.

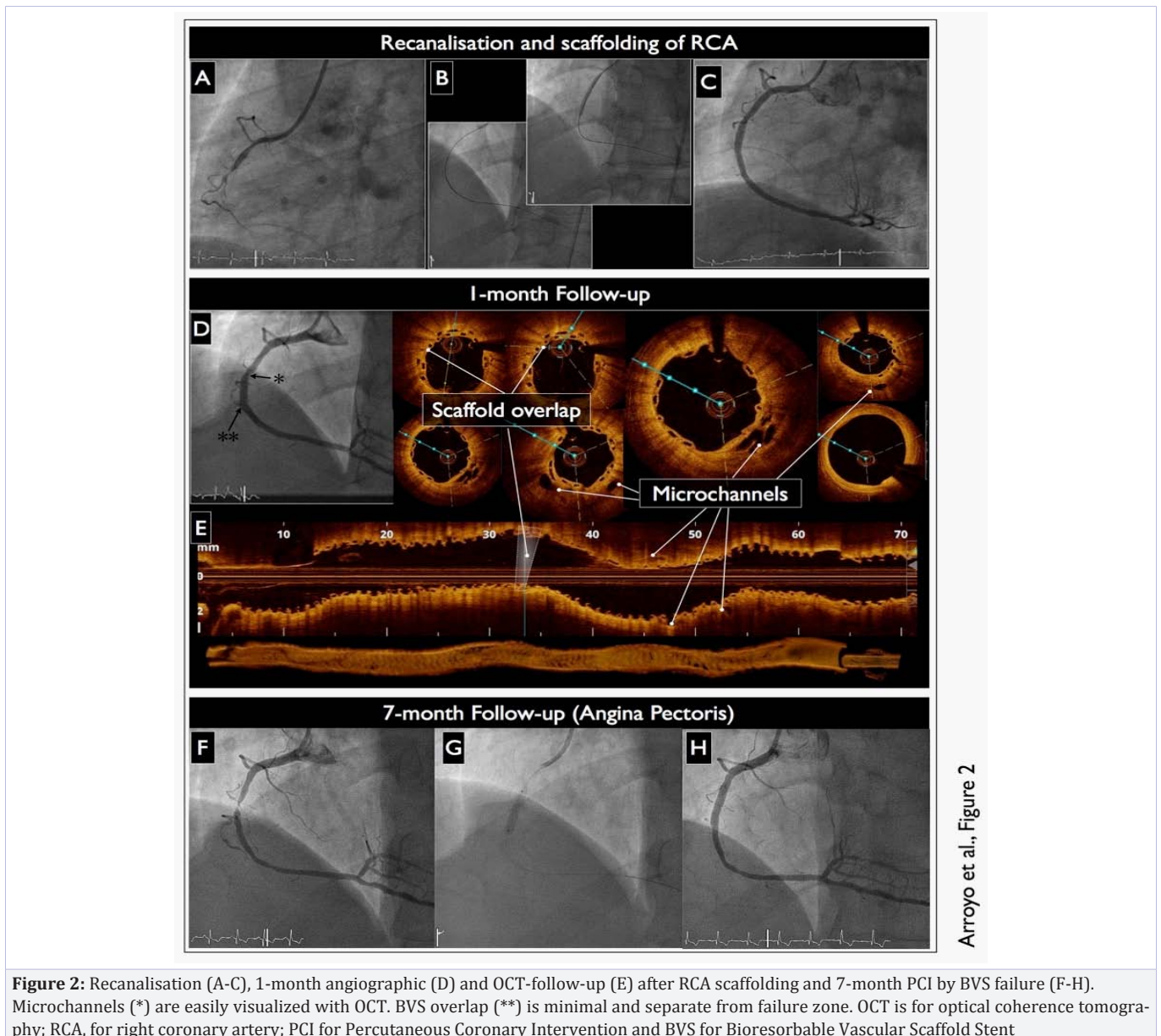


Figure 2: Recanalisation (A-C), 1-month angiographic (D) and OCT-follow-up (E) after RCA scaffolding and 7-month PCI by BVS failure (F-H). Microchannels (*) are easily visualized with OCT. BVS overlap (**) is minimal and separate from failure zone. OCT is for optical coherence tomography; RCA, for right coronary artery; PCI for Percutaneous Coronary Intervention and BVS for Bioresorbable Vascular Scaffold Stent

Putative advantages over conventional DES are early restoration of physiological processes, superior conformability, and beneficial edge-vascular response and suppression of late stent-related complications. We know that intraplaque microvessels are indicators of enzymatic plaque activity and vulnerability. Microhemorrhages via microchannels may enlarge the necrotic core and promote macrophage infiltration leading to lumen narrowing [1]. Vasa vasorum may further promote plaque inflammation [2]. Microchannels have been documented in cases of neoatherogenesis and plaque rupture after DES implantation [3,4].

In our case, BVS overlap was minimal and separate from the failure zone as depicted in figure 2. The OCT imaging revealed no underexpansion and there was no discontinuation of double anti-platelet therapy. The CTO procedure was straightforward

and recanalization performed on first attempt; some of the microchannels visualized one month after BVS implantation could have been induced by the procedure. However, we believe that the abundant microchannels (either neovessels or remnant microvessels from the CTO) are associated with increased plaque activity, and contributed to BVS failure several months after.

Conflict of Interest

There are no conflicts of interest with any of the authors relating to the present report.

References

1. Virmani R, Kolodgie FD, Burke AP, Finn AV, Gold HK, Tulenko TN, et al. Atherosclerotic plaque progression and vulnerability to rupture: angiogenesis as a source of intraplaque hemorrhage. *Arterioscler Thromb Vasc Biol.* 2005; 25(10): 2054-61.

2. O'Brien ER, Garvin MR, Dev R, Stewart DK, Hinohara T, Simpson JB, et al. Angiogenesis in human coronary atherosclerotic plaques. *Am J Pathol.* 1994; 145(4): 883-94.
3. Stadelmann M, Valentin J, Giraud M-N, Cook S. Ruptured neoatherosclerosis as source of late stent thrombosis. *Cardiovascular Medicine.* 2012; 15: 293-4.
4. Kitabata H, Tanaka A, Kubo T, Takarada S, Kashiwagi M, Tsujioka H, et al. Relation of microchannel structure identified by optical coherence tomography to plaque vulnerability in patients with coronary artery disease. *Am J Cardiol.* 2010; 105(12): 1673-8. doi: 10.1016/j.amjcard.2010.01.346.