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Original Research

Early and Late Optical Coherence Tomography Findings Following Everolimus-Eluting Bioresorbable Vascular Scaffold Implantation in Myocardial Infarction: A Preliminary Report

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Key words:
Bioresorbable vascular scaffold, optical coherence tomography, ST-elevation myocardial infarction, non-ST-elevation myocardial infarction.

Introduction: Although bioresorbable vascular scaffolds (BVS) have been used with promising results in patients with stable and unstable angina, little is known about the acute vascular response following BVS implantation in myocardial infarction. We present angiographic and OCT findings from the first patients undergoing bioresorbable vascular scaffold (BVS) implantation for non-ST-elevation myocardial infarction (NSTEMI) or ST-elevation myocardial infarction (STEMI) in our institution.

Methods: The first 5 patients with NSTEMI and the first 5 patients with STEMI who underwent BVS implantation in our institution, followed by optical coherence tomography (OCT) imaging of the treated culprit vessel, were included in this series. All patients underwent angiographic analysis pre- and post- BVS implantation, as well as OCT analysis, including qualitative and quantitative assessment.

Results: Implantation was successful in all cases, as assessed by angiography and OCT. There were no cases with coronary spasm, distal embolization or no-reflow. No adverse clinical events were recorded in any patient at the 6-month follow up. Specific illustrative cases demonstrating the challenges of BVS implantation in myocardial infarction are presented.

Conclusions: BVS implantation can potentially be used in the setting of thrombotic lesions encountered in myocardial infarction; however, the role of this treatment approach warrants systematic evaluation in prospective studies.

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Implantation of metallic platform drug-eluting stents constitutes the mainstay of revascularization in acute myocardial infarction.[1] However, their use has been associated with an impaired vascular healing response, while concerns have been raised over their long-term performance. Bioresorbable vascular scaffolds (BVS) could help overcome such long-term pitfalls of metallic platforms.[2] Although bioresorbable scaffolds have shown promising results in stable and unstable angina,[3,4] the acute vascular response following BVS implantation in myocardial infarction has not been extensively studied.[5-7]

As of September 1st, 2012, the ABSORB™ BVS (Abbott Vascular, Santa Clara, CA, USA) has been commercially available in the Netherlands. Based on available evidence,[3,4] our department selected these devices as the first option for younger patients presenting for percutaneous coronary intervention (PCI) in every-

day clinical practice. As lesions in these patients might be more complex compared to those of trial investigational patients, the BVS-EXPAND registry was initiated. In this registry, follow-up data are collected as part of the hospital routine for monitoring outcomes after PCI and the introduction of a different generation of stent or scaffold. The BVS-EXPAND includes patients with stable disease, but also patients with unstable angina or non-ST-elevation myocardial infarction (NSTEMI). After the first experience with acute patients and an interim analysis, a decision was made to extend BVS utilization to the treatment of ST-elevation myocardial infarction (STEMI).

To assess the safety and procedural success of a BVS strategy in STEMI, optical coherence tomography (OCT) imaging was performed, according to clinical judgment, in patients with STEMI and BVS implantation, for a more comprehensive evaluation of the acute procedural outcome by the operator. OCT is a high-resolution intravascular imaging modality that enables visualization of the acute vascular response after stent implantation.[8-11] Specifically, OCT can accurately evaluate scaffold expansion and apposition, and can also assess vascular trauma and residual thrombotic burden.[11-13]

We previously reported on a systematic analysis of the OCT findings post BVS implantation in STEMI.[6] In the current report, we present patient-level angiographic and OCT findings from the first five patients of BVS-EXPAND presenting with NSTEMI and imaged by OCT, and the first five patients who underwent BVS implantation for STEMI followed by OCT imaging, in our institution, in an attempt to illustrate the main challenges of BVS implantation in thrombotic lesions.

Methods

Study population

The current series comprised 1) the first five patients of BVS-EXPAND presenting with NSTEMI and imaged by OCT, and 2) the first five patients who underwent BVS implantation for STEMI followed by OCT imaging, in our institution. The STEMI patients were not a part of BVS-EXPAND.

Procedure and OCT image acquisition

Patients with NSTEMI typically underwent coronary angiography within 24-72 hours from symptom onset, according to the regional protocol, followed by urgent PCI after discussion in the HeartTeam, while patients with STEMI underwent primary PCI upon hospital arrival. Interventional management, including the use of thrombectomy, pre-dilation and post-dilation, was performed according to the operator's discretion. OCT was performed after BVS implantation using the C7™ imaging system and the Dragonfly™ catheter (both St. Jude Medical, St. Paul, Minnesota, USA), as previously described.[11] In several cases, OCT findings guided further procedural optimization (i.e. additional scaffold implantation and/or post-dilation). In these cases, a new OCT pullback was performed at the end of the procedure. The patients were contacted by telephone 6 months after the procedure, and adverse events (death, myocardial infarction, any unplanned revascularization), according to the Academic Research Consortium definitions, were recorded.[14]

Angiographic and OCT analysis

Quantitative coronary angiography (QCA) analyses were performed offline by an experienced observer (TM) using CAAS 5.10 (Pie Medical Imaging, Maastricht, Netherlands) according to a previously reported methodology.[13] Intracoronary thrombus was identified angiographically and scored in five grades, as previously described.[13] Complications such as dissection, spasm, distal embolization and no-reflow were also recorded. OCT analysis was performed offline by an experienced observer in fixed 1-mm longitudinal intervals within the treated culprit segment, after exclusion of frames with <75% lumen contour visibility. Quantitative analysis included measurement of the minimal lumen and the minimal scaffold area, according to a previously described methodology.[15] Qualitative assessment included evaluation of in-scaffold and edge dissections, tissue prolapse and in-scaffold thrombus. Dissections were defined as the presence of intimal discontinuity, with or without flap formation, either within the scaffolded segment (intra-scaffold dissection), or within 5-mm-long proximal or distal edge segments (edge dissections).[16] Tissue prolapse was defined as the projection of tissue into the lumen between stent struts after implantation.[16] Incomplete strut apposition (ISA) was defined as a clear lack of contact between scaffold strut and vascular wall;[15] apposition was assessed at a scaffold-level basis, using a definition of malapposed scaffold as a scaffold with >5% malapposed struts.

Ethics

This was an observational study, performed according to the privacy policy of Erasmus MC and the Erasmus MC regulations for the appropriate use of data in patient-oriented research. These are based on international regulations, including the declaration of Helsinki. A waiver from the Ethical Committee of Erasmus MC was obtained for written informed consent, as—according to Dutch law—written consent is not required if patients do not undergo procedures other than as part of their regular treatment. Invasive follow up in two of the patients was performed as part of an ethical committee-approved, single-center, investigator-driven, observational study (BVS-STEMI first) for which written informed consent was obtained from the patients.

Results

The median patient age was 56 years (range 40-75 years). Baseline characteristics are presented in Table 1, while angiographic data are presented in Table 2. There were no cases with spasm, distal embolization or no-reflow. No in-hospital events were recorded. Table 3 summarizes the OCT findings. Clinical follow-up at 6 months was available for all patients, showing an absence of adverse clinical events. Specific illustrative cases demonstrating the challenges of BVS implantation in NSTEMI and STEMI are presented below.

Patient 2 (Figure 1)

A 56-year-old man with no prior cardiovascular history, who presented with NSTEMI, had a total occlusion of the proximal left circumflex artery (LCx) with collateral filling from the left anterior descending artery (LAD), as documented by coronary angiography. OCT imaging performed after thrombus aspiration and predilation with a 2.0 × 20 mm compliant Trek™ balloon (Abbott Vascular) revealed a thrombosed, severely stenotic lesion with plaque rupture and intimal tears, probably induced by balloon predilation (Panels A-D). After additional predilation with a 3.0 × 20 mm Trek™ compliant balloon inflated at 14 atm (maximum diameter 3.11 mm; just below the maximum luminal diameter by QCA), a 3.0 × 18 mm BVS was implanted in the culprit lesion (inflation pressure 16 atm). Following this strategy, post-implantation OCT showed no edge dissections or residual stenosis. There were, however, several sites with moderate tissue prolapse/thrombus within the scaffold (panels A'-C'), for which no additional treatment was applied.

Patient 5 (Figures 2-3)

A 56-year-old man was admitted with NSTEMI and referred for PCI after HeartTeam consensus. Coronary angiography had demonstrated three-vessel disease: a lesion in the proximal LCx, a diffuse calcified lesion of the mid LAD, and a diffusely diseased right

Table 1. Baseline and procedural characteristics.

Patient	Age	Sex	Clinical syndrome	Lesions treated (n)	BVS implanted (n)	Thrombus aspiration	Balloon predilation	BVS size	Balloon post-dilation	Side-branch dilation
Patient 1	59	♀	NSTEMI	1	4	No	3 × 15	3 × 28, 3 × 28, 3 × 18, 2.5 × 28	3 × 12, 3.5 × 15	No
Patient 2	56	♂	NSTEMI	1	1	Yes	3 × 20	3 × 18	No	No
Patient 3	75	♂	NSTEMI	1	3	No	3 × 15	3.5 × 18, 3 × 18, 2.5 × 18	No	1.5 × 8
Patient 4	57	♀	NSTEMI	1	1	No	2.5 × 20	3 × 28	No	No
Patient 5	56	♂	NSTEMI	2	3	No	3 × 15 (LCx) 3 × 20 (LAD)	3.5 × 18, 3.5 × 12 (LCx) 3.5 × 28 (LAD)	No (LCx) 3.75 × 15 (LAD)	No
Patient 6	56	♀	STEMI	1	1	Yes	2.5 × 20	3 × 28	3.25 × 15	No
Patient 7	40	♂	STEMI	1	1	Yes	2.5 × 15	3.5 × 18	No	No
Patient 8	53	♂	STEMI	1	1	No	2 × 8	2.5 × 18	No	No
Patient 9	65	♂	STEMI	1	2	Yes	No	3.5 × 12, 3.5 × 12	4 × 8	No
Patient 10	58	♂	STEMI	1	2	Yes	3 × 12	3.5 × 18, 3.5 × 18	3.5 × 15	1.5 × 15

BVS – bioresorbable vascular scaffold; NSTEMI – non-ST-elevation myocardial infarction; STEMI – ST-elevation myocardial infarction; LCx – left circumflex artery; LAD – left anterior descending artery.

Table 2. Angiographic characteristics.

Patient	Culprit vessel	Pre procedure		Pre BVS implantation				Post implantation (in-scaffold)				
		Total Occlusion	Thrombus burden	Dmax (mm)	RVD (mm)	MLD (mm)	DS (%)	RVD (mm)	MLD (mm)	DS (%)	Final TIMI flow	Dissection
Patient 1	RCA	Yes	5	3.6	2.64	1.04	61	2.76	2.22	20	II	Yes
Patient 2	LCx	Yes	5	3.22	2.74	2.1	23	3.15	3	5	III	No
Patient 3	LAD	No	2	3.16	2.18	1.56	28	2.51	2.32	8	III	No
Patient 4	LAD	No	1	3.09	2.74	1.22	55	2.79	2.46	12	III	No
Patient 5, Lesion 1	LCx	No	0	3.44	2.96	1.39	53	2.71	2.36	13	III	No
Patient 5, Lesion 2	LAD	No	0	3.28	3.27	1.27	61	2.81	2.54	10	III	No
Patient 6	RCA	Yes	5	3.44	2.89	0.96	67	2.85	2.42	15	III	Yes
Patient 7	LAD	No	2	3.16	2.68	2.16	19	2.92	2.92	0	II	Yes
Patient 8	LD	Yes	5	2.55	2	0.51	74	2.47	2.05	17	III	No
Patient 9	RCA	Yes	5	3.97	3.52	1.4	60	3.17	2.39	25	III	No
Patient 10	LAD	No	3	3.63	2.29	0.9	61	2.58	1.93	25	III	No

Dmax – maximum diameter; RVD – reference vessel diameter; MLD – minimum lumen diameter; DS – diameter stenosis; RCA – right coronary artery; LD – diagonal branch. Other abbreviations as in Table 1.

coronary artery (RCA). It was decided to proceed with LCx and LAD revascularization, and staged revascularization of the RCA one month later.

The proximal LCx lesion (Panel 2I) was treated by 3.5 × 18 mm BVS implantation and post-dilation with a 3.0 × 15 mm compliant Trek™ balloon (Panel 2II). Subsequently, lumen narrowing with haziness was observed proximally, corresponding to an edge dissection by OCT (Panels 2A-B). The dissection was treated by implantation of an overlapping 3.5 × 12 mm BVS with a good angiographic result (panel 2III). A final OCT pullback showed focal under-expansion due to calcification, as well as sites with mild tissue prolapse and in-stent thrombus (Panels 2A'-D').

The LAD lesion (Panel 3I) was treated by 3.0 × 20 mm Trek™ balloon predilation, 3.5 × 28 mm BVS implantation and post-dilation with a 3.75 × 15mm Trek™ non-compliant balloon (Panels 3II-III). OCT following predilation revealed a heavily calcified vessel without intracoronary thrombus. Post-implantation OCT demonstrated good scaffold apposition, moderate pinching of the ostium of the diagonal (Panel 3A'), focal under-expansion at sites with heavy calcification (Panel 3C'), lack of tissue prolapse, and a small distal edge dissection (Panel 3D').

Patient 7 (Figure 4)

A 40-year-old man with diabetes and no known cardiovascular history was admitted with anterior STEMI. Angiography demonstrated a stenotic LAD lesion with angiographic haziness (Panel I). The lesion was treated by thrombus aspiration, 2.5 × 15 mm Trek™ compliant balloon pre-dilation and 3.5 × 18 mm BVS implantation (Panels II-III). A small edge dissection was visible by angiography after implantation. OCT confirmed the diagnosis of distal edge dissection extending for ~5 mm, which was left untreated (Panels D-E). The scaffold was well-expanded and apposed with moderate tissue prolapse and in-scaffold thrombus at the middle segment (Panels A-C). The patient underwent invasive follow up, including OCT imaging, 9 months post implantation for study purposes (Panel IV). The follow-up OCT showed a good healing response, without late ISA, high strut coverage (Panels A'-C') and complete healing of the dissection with integration of the dissection flap in the vessel wall (Panels D'-E').

Patient 8 (Figure 5)

A 52-year-old man with an unremarkable cardiovascular history was admitted with anterolateral STEMI. Angiography demonstrated total occlusion of the second diagonal branch (Panel I). The lesion was treated by 2.0 × 8 mm Trek™ compliant balloon dilation and 2.5 × 18 mm BVS implantation (Panels II-III). OCT showed a well-expanded and apposed scaffold with no vascular trauma at the middle and distal segments, and mild tissue prolapse and thrombus near the vessel ostium (Panels C-D). Scaffold struts were visible at the polygon of confluence of

Table 3. Optical coherence tomography (OCT) findings.

Patient	ISA	Minimal lumen area (mm^2)	Minimal scaffold area (mm^2)	Intraprocedural findings	Final OCT findings
Patient 1	Yes	Proximal BVS: 6.63	Proximal BVS: 7.85	Extensive intra-scaffold dissections with ISA in proximal scaffold, treated with post-dilation	• Improvement of scaffold apposition after post-dilation. • Minimal tissue prolapse.
Patient 2	No	7.60	8.34	-	• Good expansion and apposition. • No edge dissections • Moderate tissue prolapse/thrombus.
Patient 3	Yes	4.42	4.97	Side-branch compromise, treated with balloon dilation	• Distal and middle scaffolds well-expanded without tissue prolapse or thrombus. • ISA in the proximal and middle scaffolds due to eccentric calcium and intra-scaffold dissections. • Mild tissue prolapse in the proximal scaffold. • No evident scaffold fracture at the level of the side-branch.
Patient 4	No	6.05	6.57	-	• Good expansion and apposition without edge dissections. • Short segment with moderate tissue prolapse/thrombus
Patient 5	LCx: Yes LAD: No	LCx: 6.28 mm^2 LAD: 4.98 mm^2	LCx: 6.28 mm^2 LAD: 5.36 mm^2	Proximal dissection in LCx, treated with 2nd BVS	• LCx: mild tissue prolapse/thrombus in both scaffolds. • Sub-optimal expansion and ISA focally. • LAD: good apposition with sub-optimal expansion focally due to calcium. • Small distal edge dissection.
Patient 6	Yes	6.27	7.07	Extensive intra-scaffold dissection with ISA, treated with post-dilation.	• Reduction of dissection cavity size and malapposition distance after post-dilation. • Mild tissue prolapse and in-scaffold thrombus ISA.
Patient 7	No	7.55	8.33	-	• Distal edge dissection ~5 mm long. • Sites with moderate tissue prolapse/in-scaffold thrombus proximally.
Patient 8	No	4.21	4.76	-	• Well-expanded and apposed scaffold with minimal tissue prolapse/thrombus. • Scaffold protrusion into the main branch for ~1 mm.
Patient 9	Yes	5.72	7.6	Stenosis in distal RCA after BVS implantation in RPL, treated with 2nd BVS.	• ISA proximally due to scaffold-lumen dimensions mismatch. • Minimal overlap at the crux with moderate tissue prolapse. • Presence of struts at the RPD ostium and residual red thrombus.
Patient 10	No	6.77	7.15	Small edge dissection and residual lesion distally (MLA: 1.71 mm^2), treated with 2nd BVS - Side-branch compromise, treated with balloon dilation	• Good expansion and apposition with minimal overlap. • Mild tissue prolapse/thrombus in both scaffolds. • No evident scaffold fracture at the level of the side-branch.

ISA – incomplete scaffold apposition; RPL – right posterolateral branch; RPD – right posterior descending branch; MLA – minimal lumen area. Other abbreviations as in Tables 1 and 2.

the LAD-diagonal bifurcation, with minimal protrusion of the scaffold into the LAD (Panels A, B, G). Invasive follow up with OCT imaging was performed 1 year post implantation for study purposes (Panel IV). The follow-up OCT revealed a good healing response without ISA and a low number of uncovered struts (Panels A'-D'). Dense tissue coverage was also observed at the side-branch-related struts located in the vessel ostium, resulting in the development of a neo-carina (Panel H).

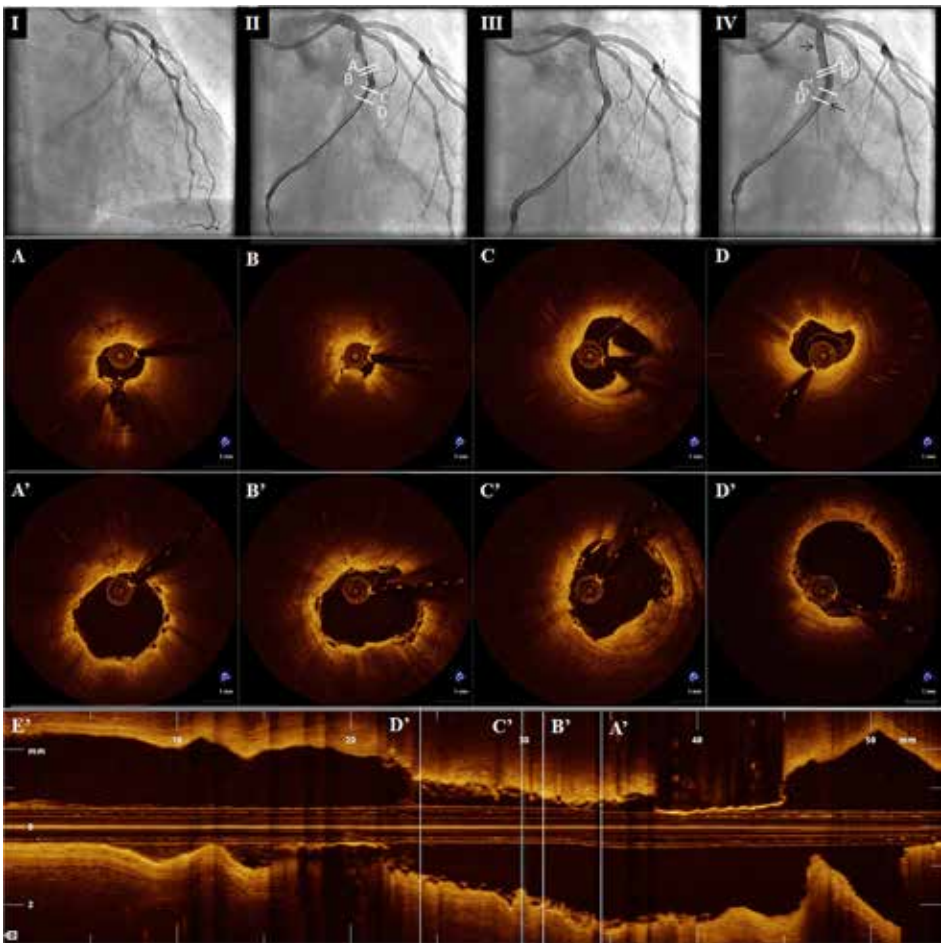

Figure 1. Top panels: Coronary angiography demonstrating the culprit lesion of the proximal left circumflex artery (I) pre-intervention, (II) after 2.0 mm balloon predilation, (III) after 3.0 mm balloon predilation, and (IV) after 3.0 × 18 mm bioresorbable vascular scaffold (BVS) implantation. Black arrows indicate scaffold markers, and white lines the sites corresponding to the bottom panels. Bottom panels: Culprit lesion optical coherence tomography images of matched sites (A-D) after 2.0 mm balloon dilation and (A'-D') after BVS implantation. Pre-implantation images demonstrate (A) plaque rupture with cavity, (B) minimal lumen area with red thrombus, (C) dissection flap, and (D) distal segment with macrophage infiltration. Findings after BVS implantation include (A') mild tissue prolapse at the cavity site, (B') residual compressed red thrombus, (C') tissue prolapse at the site of the flap, and (D') good expansion and apposition without tissue prolapse/thrombus.

Discussion

The current series of patients provides real-life insights into the acute vascular response after BVS implantation in myocardial infarction. In order to assess the efficacy of BVS implantation, OCT imaging was employed. OCT-derived parameters can potentially be used as surrogate markers for assessing the acute outcome of BVS implantation in patients with myocardial infarction. In particular, in addition to the assessment of luminal dimensions and scaffold expansion, OCT allows a comprehensive assessment of the scaffold-vessel wall interaction, providing detailed information about scaffold apposition, vascular trauma (intra-scaffold and edge dissections, tissue prolapse), and residual thrombotic burden. The clinical impact of such OCT parameters has not been established; however, their evaluation in studies of BVS implantation in myocardial infarction could be of significance, considering i) the increased incidence of incomplete apposition after metal stent implantation in myocardial infarction,[17] ii) the need for more aggressive lesion preparation with BVS, and iii) the association of increased residual thrombus with impaired reperfusion after metal stent implantation in myocardial infarction.[18]

In the ABSORB B study, in a relatively non-complex lesion subset of patients with stable and unstable

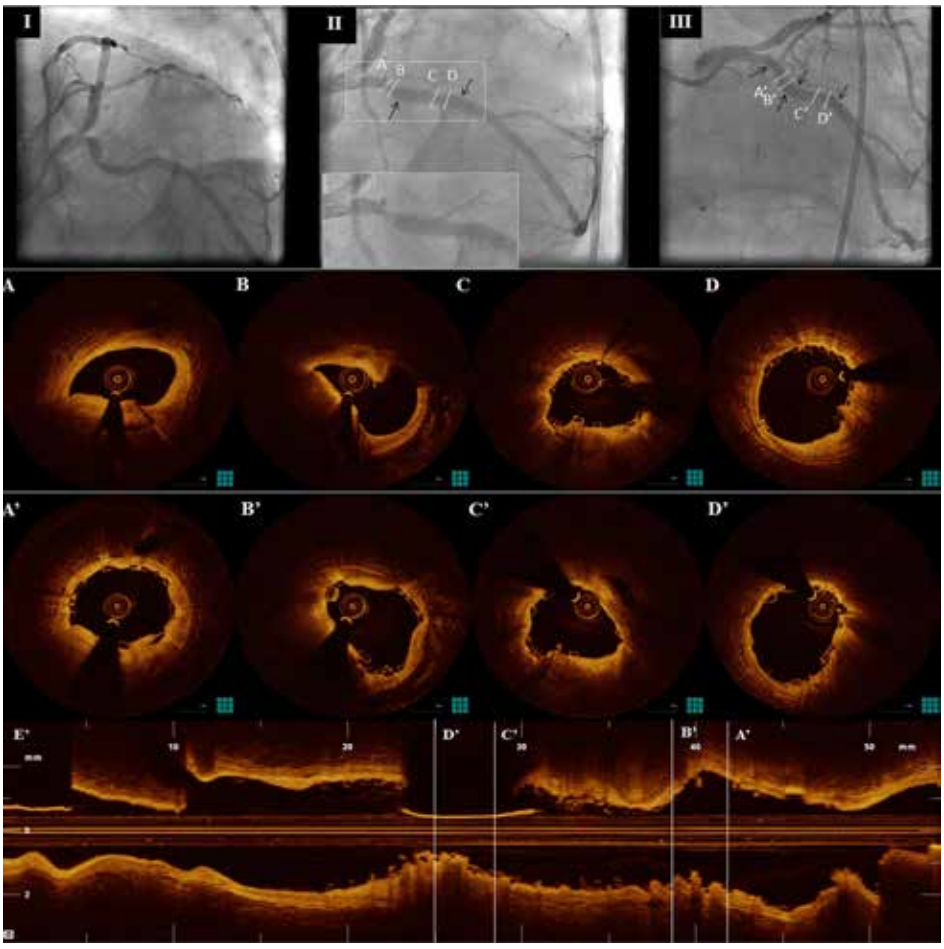

Figure 2. Top panels: Coronary angiography demonstrating the left circumflex artery lesion (I) pre-intervention, and (II) after 3.5 × 18 mm bioresorbable vascular scaffold (BVS) implantation. Because of proximal dissection a 3.5 × 12 mm scaffold was implanted (III). Black arrows indicate scaffold markers, and white lines the sites corresponding to the bottom panels. Bottom panels: Culprit lesion optical coherence tomography images of matched sites after (A-D) first and (A'-D') second BVS implantation. In the proximal scaffold, there is tissue prolapse at the sites of the dissection flaps with a small amount of intraluminal thrombus (A-A', B-B'). Mild under-expansion over a calcified plate (C-C') and mild tissue prolapse/in-stent thrombus (D-D') are observed at the distal scaffold.

angina, the BVS has shown adequate expansion, with a minimum scaffold area of 6.31 ± 1.25 mm^2.[15] In our series of patients with myocardial infarction, good scaffold expansion was observed in all but one case, in which case the implanted scaffolds were focally under-expanded because of heavy calcification (Panels 2C' and 3C'). In all other patients the implanted scaffolds were well expanded, including a case with direct BVS implantation in STEMI (Patient 9).

Apposition was not optimal in all cases, with causes for incomplete apposition including extensive intra-scaffold dissections and in one case mismatch of lumen and scaffold dimensions after bail-out BVS implantation in a proximal segment (Patient 9). The selection of scaffold size during the acute phase of myocardial infarction can be challenging, as the increased thrombus burden and the enhanced vascular tone can hamper evaluation of the true vessel size.[12] Post-dilation guided by OCT imaging – always in agreement with scaffold compliance and respecting the recommended maximum scaffold diameter – helped improve scaffold apposition in most cases. Still, the future course of scaffold apposition remains elusive. In the two patients in our series who underwent invasive follow up, no late incomplete scaffold apposition was observed. Although implantation of metal stents during primary PCI is associated with higher incomplete apposition at follow up compared to stable angina,[8,17] possibly due to resolution of vasoconstriction and thrombus,[12] no such data are currently available for the follow up of BVS implanted in myocardial infarction. Thus, longer-term follow up and prospective

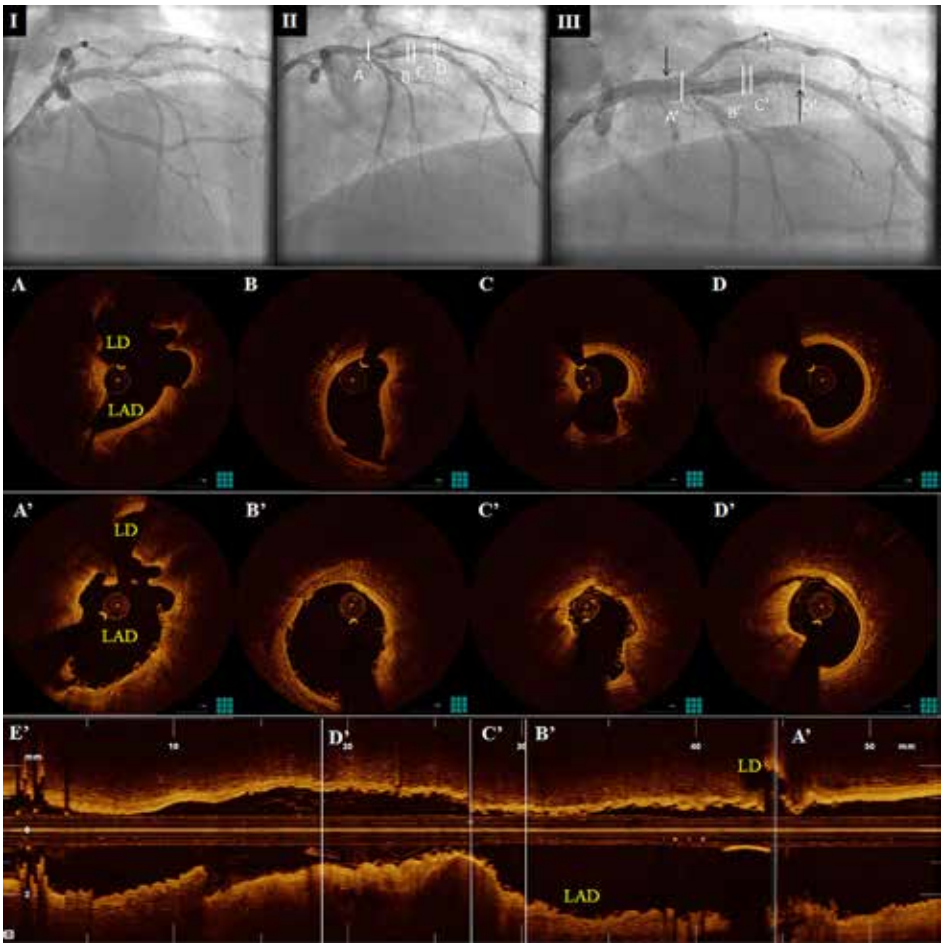

Figure 3. Top panels: Coronary angiography demonstrating the LAD lesion (I) pre-intervention, (II) after 3.0 mm balloon dilation, and (III) after 3.5 × 28 mm bioresorbable vascular scaffold (BVS) implantation and post-dilation with 3.75 mm non-compliant balloon. Black arrows demonstrate scaffold markers and white lines the sites corresponding to the bottom panels. Bottom panels: Culprit lesion optical coherence tomography images of matched sites after (A-D) pre-dilation and (A'-D') BVS implantation. (A-A') Small thrombus formation and pinching of the diagonal ostium post-implantation. (B-B') Good expansion and apposition at the site of a dissected eccentric calcified plaque. (C-C') Suboptimal scaffold expansion due to the presence of two protruding calcified plates at a site without dissection. (D-D') Small distal edge dissection at the site of a calcified nodule. LAD – left anterior descending branch; LD – diagonal branch.

studies are warranted to assess the true extent of incomplete scaffold apposition in myocardial infarction and its clinical implications at follow up.

Despite their similar pathophysiological mechanisms, NSTEMI and STEMI exhibit differences in lesion morphology and disease severity.[19,20] In our series, high disease heterogeneity was observed in NSTEMI. Two of the cases with NSTEMI (Patients 1 and 2) presented with total occlusion. Implantation was successful in both cases with a good final result, demonstrating the potential of BVS implantation in a highly thrombogenic setting, similar to STEMI. Indeed, the vascular response after BVS implantation in STEMI resembled these NSTEMI cases. The other end of the NSTEMI spectrum also includes patients with multiple lesions, increased calcification and minimal thrombus, as in Patient 5. Conversely, all STEMI cases in our series were associated with angiographic thrombus, dictating the use of thrombus aspiration in all but one case.

The different lesion substrate could have implications for the extent of vascular trauma and residual thrombotic burden. Both lesion morphology and unstable presentation have been associated with periprocedural vascular trauma.[9] In cases with heavy calcification, as in Patient 5, aggressive lesion preparation could help optimize scaffold expansion. However, lesion preparation should be performed with caution, as it may as well induce extensive vascular trauma, as in Patients 1 and 6.[21] Predilation has been associated with increased distal embolization in the setting of metal stent implantation for myocardial in-

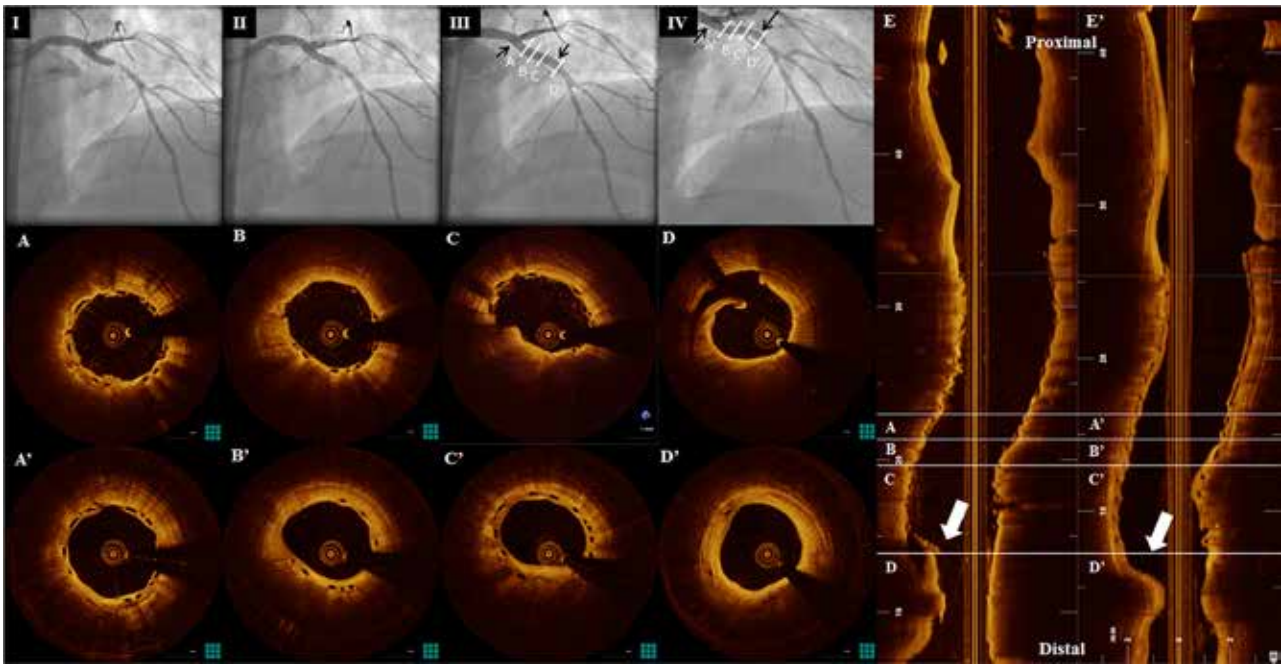

Figure 4. Top panels: Coronary angiography demonstrating the culprit left anterior descending artery lesion (I) pre-intervention, (II) post thrombus aspiration/2.5 mm balloon predilation, (III) post 3.5 × 18 mm BVS implantation, and (IV) at 9-month follow up. Black arrows indicate scaffold markers and white lines the sites corresponding to the bottom panels. Bottom panels: Optical coherence tomography of the treated lesion. Panels A-C disclose the presence of varying degrees of tissue prolapse/intra-scaffold thrombus, and Panel D an intra-scaffold dissection. Nine-month follow-up images show a favorable healing response with the absence of late incomplete apposition, symmetric coverage of the bioresorbable vascular scaffold (A'-C'), and the complete healing of the edge dissection (D'). L-mode images at baseline (E) and follow up (E') demonstrate the longitudinal morphology of the healed dissection (arrow).

farction,[22] however such data are not available for BVS. In our series, although distal embolization was not angiographically documented, OCT revealed varying degrees of tissue prolapse and/or residual thrombus in all cases. Importantly, increased tissue prolapse and/or residual thrombus have been associated with impaired reperfusion following stenting for STEMI.[18] Therefore, it is important to evaluate such findings systemically in BVS and to assess their significance and differences with metal platform stents.[6]

Another important aspect is the presence of culprit bifurcation lesions. In cases of ostial side-branch disease, as in Patients 8 and 9, BVS implantation constitutes an appealing option, given the favorable response observed in side-branch-related scaffold struts, with their gradual replacement by tissue bridges and development of a neo-carina,[23] Conversely, in metal stents, the permanent stent layer jailing side-branches is often characterized by impaired healing,[24] providing a potential substrate for thrombosis. In the case of culprit lesions with large side-branch involvement, however, a high thrombus burden can cause underestimation of the side-branch involvement, potentially resulting in side-branch compromise post-procedurally, as in Patients 3 and 10. The good angiographic result after side-branch dilation and the absence of scaffold fracture by OCT suggest a potential role for this approach in the treatment of side-branch compromise.

It is important to note that the current series represents a preliminary report of a single-center experience with BVS implantation in myocardial infarction. The current report did not aim to conduct a systematic analysis of the OCT findings, but instead gives patient-specific descriptions of the acute vascular response, aiming to provide initial observations and demonstrate challenges and differences with stable disease. The impact of a thrombotic substrate on the vascular response following BVS implantation still needs to be systematically evaluated in larger patient cohorts with a longer follow up.

Conclusions

In the current series, we presented angiographic and OCT findings following BVS implantation in myocardial infarction. Implantation was successful both in thrombotic culprit lesions of NSTEMI patients and in culprit lesions of STEMI patients, with a similar acute vascular response by OCT. The incidence of incom-

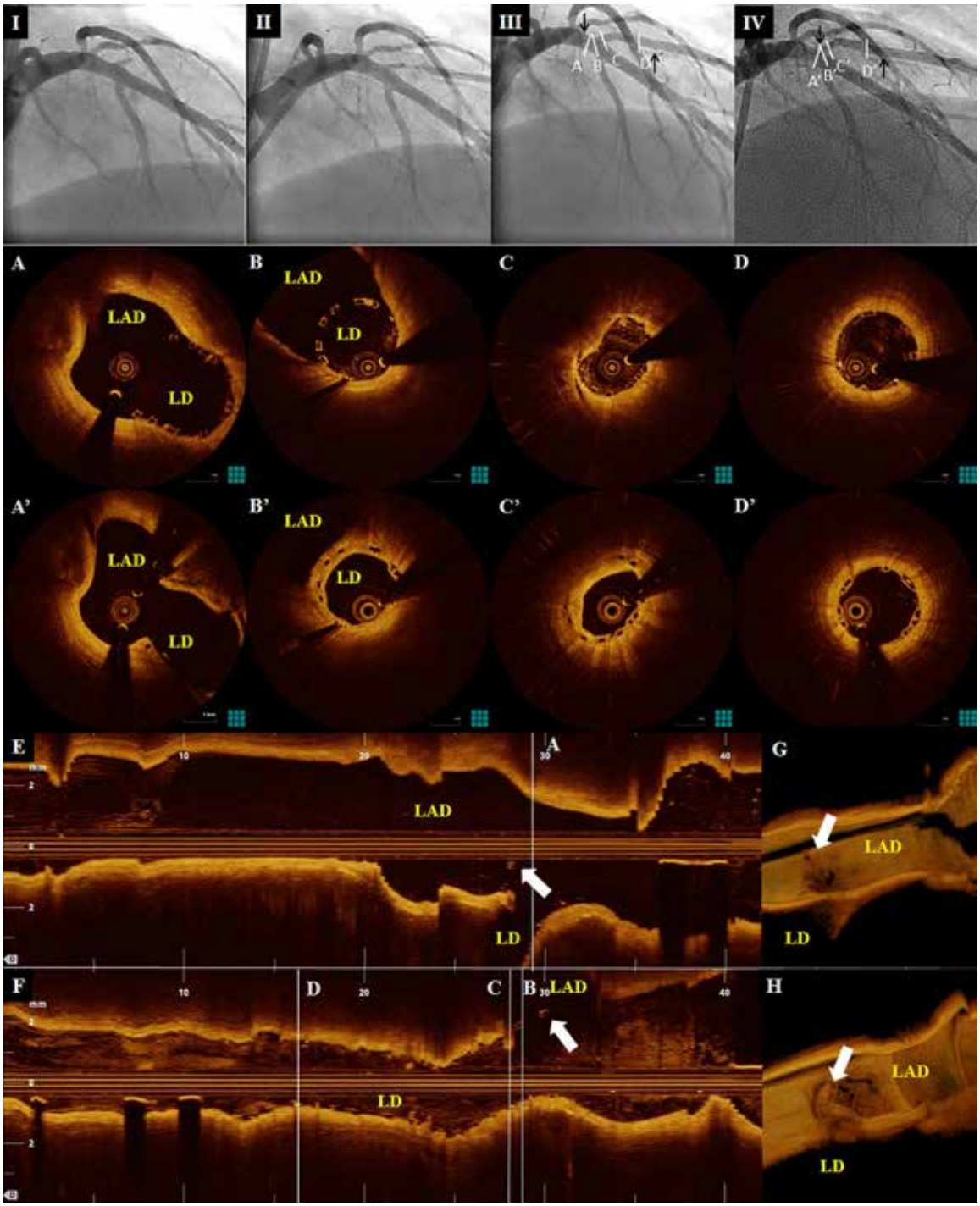

Figure 5. Top panels: Coronary angiography demonstrating (I) total occlusion of the LD, and the angiographic result after (II) 2.0 mm balloon predilation and (III) 2.5 × 18 mm bioresorbable vascular scaffold implantation. Panel IV shows the angiography at 12-month follow up. Black arrows indicate scaffold markers and white lines the sites corresponding to the bottom panels. Bottom panels: Optical coherence tomography (OCT) of the culprit diagonal branch lesion. Scaffold struts are protruding into the polygon of confluence of the LAD-diagonal bifurcation, as can appreciated from both the LAD (A, E) and the diagonal pullback (B, F). There is mild tissue prolapse and thrombus near the vessel ostium (C), but minimal prolapse in the middle and distal scaffold segments, with good expansion and apposition (D). Follow-up OCT images at matched sites are presented in panels A'-D', showing coverage of the implanted scaffold, absence of incomplete apposition, and the development of tissue bridges in relation to side-branch-related struts. Three-dimensional renderings illustrate the development of a neo-carina at the site of scaffold protrusion into the LAD (E, E'). LAD – left anterior descending branch; LD – diagonal branch.

plete scaffold apposition at follow up, the role of the pathological substrate in the selection of lesion preparation strategy, and the impact of periprocedural trauma and residual thrombus post-intervention remain open questions regarding BVS implantation in myocardial infarction that warrant systematic evaluation in prospective studies.

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