Clinical Investigation

JOURNAL OF ENDOVASCULAR THERAPY
A SAGE Publication
An official publication of the ISEVS

Journal of Endovascular Therapy
2018, Vol. 25(3) 313–319

DOI: 10.1177/1526602818771345
www.jevt.org
SAGE

Edge Stenosis After Covered Stenting for Long Superficial Femoral Artery Occlusive Disease: Risk Factor Analysis and Prevention With Drug-Coated Balloon Angioplasty

Ting-Chao Lin, MD[1,2], Chun-Yang Huang, MD[3], Po-Lin Chen, MD[2,3], Chiu-Yang Lee, MD[3], Chun-Che Shih, MD, PhD[3,4], and I-Ming Chen, MD, PhD[2,3]

Abstract

Purpose: To report a retrospective analysis of risk factors for edge restenosis after Viabahn stent-graft treatment of superficial femoral artery (SFA) occlusive disease and determine any protective effect of drug-coated balloons (DCBs) used at the time of stent-graft implantation. **Methods:** Between October 2011 and July 2016, 110 patients (mean age 73.3±7.6 years; 78 men) were treated with the Viabahn stent-graft for long SFA occlusions. Thirty-eight (34.5%) patients had DCB reinforcement at the distal edge of the stent-graft. For analysis, the population was divided into groups of no edge stenosis patients (n=88; mean lesion length 22.4±4.2 cm) and edge stenosis patients (n=22; mean lesion length 23.5±5.7 cm). The clinical outcomes, ankle-brachial indices, computed tomography angiography findings, and patency were compared at a minimum of 12 months. Logistic regression analysis was employed to determine risk factors for edge stenosis; the results are presented as the odds ratio (OR) and 95% confidence interval. **Results:** No differences in clinical or procedural characteristics were identified except the higher incidence of diabetes (p=0.008) and greater need for retrograde access (p=0.033) in the edge stenosis group. DCB reinforcement reduced the incidence of edge stenosis (p=0.021) and target lesion revascularization (TLR; p=0.010) and resulted in a significantly higher 1-year primary patency rate (92.1% vs 76.4%, p=0.042). However, multivariate analysis revealed only poor distal runoff (OR 0.31, 95% CI 0.11 to 0.83, p=0.020) as a predictor of edge stenosis. **Conclusion:** The risk of edge stenosis after Viabahn implantation was higher in patients with poor distal runoff. DCB reinforcement over the distal edge reduced edge stenosis, decreased 1-year TLR, and improved 1-year primary patency.

Keywords

angioplasty, covered stent, drug-coated balloon, edge stenosis, occlusion, patency, peripheral artery disease, restenosis, runoff vessels, stent-graft, superficial femoral artery, target lesion revascularization

Introduction

The self-expanding Viabahn covered stent (W.L. Gore & Associates, Flagstaff, AZ, USA) has been shown to have better patency rates and long-term outcomes than bare metal stents (BMS) or balloon angioplasty for long superficial femoral artery (SFA) occlusive disease [TransAtlantic Inter-Society Consensus II (TASC) C and D].[1–6] These advantages of the Viabahn stem from the ability of the polytetrafluoroethylene covering to prevent ingrowth of neointimal hyperplasia, which is the largest disadvantage of BMS in long SFA occlusive disease.[7–10] However, neointimal hyperplasia does occur at the edges of a Viabahn, causing edge stenosis that results in lumen thrombosis, which accounts for the majority of Viabahn failures.[11,12] Nevertheless, the risk factors of such edge stenosis are not completely understood.

Drug-coated balloons (DCBs) have been recently introduced to inhibit smooth muscle proliferation through the delivery of antiproliferative drugs directly to the arterial

[1]Division of Cardiovascular Surgery, Heart Center, Cheng Hsin General Hospital, Taipei, Taiwan
[2]Department of Medicine, School of Medicine, National Yang-Ming University, Taipei, Taiwan
[3]Division of Cardiovascular Surgery, Department of Surgery, Taipei Veterans General Hospital, Taipei, Taiwan
[4]Institute of Clinical Medicine, School of Medicine, National Yang-Ming University, Taipei, Taiwan

Corresponding Author:
I-Ming Chen, Division of Cardiovascular Surgery, Department of Surgery, Taipei Veterans General Hospital, No. 201, Sec. 2, Shih-Pai Road, Taipei, 112, Taiwan.
Email: imchen@vghtpe.gov.tw

wall.[13] This study sought to identify the risk factors for edge stenosis after Viabahn implantation and investigate the application of DCB angioplasty over the distal edge of a Viabahn to determine whether such a strategy can prevent edge stenosis and improve outcomes.

Methods

Patient Population

From October 2011 to July 2016, 110 patients (mean age 73.3±7.6 years; 78 men) underwent non–heparin-bonded Viabahn deployment for de novo, long (>15 cm), TASC C and D[14] SFA lesions (the heparin-bonded Viabahn was unavailable in our country). The indications for intervention included severe claudication (Rutherford category 3) or critical limb ischemia (CLI; Rutherford categories 4–6). Patients with dissected SFA lesions and any previous SFA intervention (eg, angioplasty, stenting, or bypass surgery) were excluded. Demographic and clinical data were collected from the medical charts for this retrospective analysis, which was approved by the institutional review board.

Intervention

A 150-mg loading dose of clopidogrel was administered before the procedures, which were performed by vascular surgeons. Vascular access was obtained through the contralateral limb with the support of a long sheath (Flexor; Cook Medical Inc, Bloomington, IN, USA). If an iliac lesion was present, it was stented before the SFA occlusion was addressed. Intraluminal passage was tried initially, and if this failed, a subintimal approach or retrograde access through the popliteal or below-the-knee (BTK) arteries was used. After wire cannulation, the lesion was predilated along its length with a balloon smaller than the diameter of the chosen stent-graft, which was ~10% larger than that of the native SFA to discourage edge stenosis. The size of the native SFA was estimated using preoperative computed tomography angiography (CTA) because intravascular ultrasound (IVUS) was not available in our institution to determine the size of the native SFA. If >1 Viabahn was required, the second Viabahn was overlapped at least 10 mm with the first.

Although every patient was a potential candidate for DCB reinforcement, these balloons are not reimbursed in our country, so the patient made the decision after the benefits and risks were explained. In the study population, 38 patients selected DCB reinforcement. In these cases, a DCB [IN.PACT Admiral (Medtronic Cardiovascular, Santa Rosa, CA, USA; n=37) or Ranger (Boston Scientific, Würselen, Germany; n=1)] matched in diameter to the Viabahn was inflated over the distal edge of the implanted stent-graft for 3 minutes, overlapping ~1 cm into the Viabahn. In patients without DCB reinforcement, plain balloons of the same diameter as the Viabahn were used for postdilation. To prevent localized trauma to the native vessel, angioplasty across the Viabahn edge was avoided in the group without DCB reinforcement. In patients with stenotic or occlusive BTK lesions, angioplasty was also performed if possible.

Follow-up, Outcome Measures, and Definitions

Clopidogrel (75 mg daily) and aspirin (100 mg daily) were continued for at least 12 months. Clinical evaluation, ankle-brachial index assessment, and lower limb CTA were performed at 6, 12, and 24 months postoperatively.

The procedure was technically successful if the Viabahn was deployed at the target SFA lesion with a residual stenosis <30%. Edge stenosis was defined as >50% stenosis around the Viabahn edge, which was assessed using CTA or traditional angiography. Patients with symptom recurrence, including claudication, gangrene, and ulcer formation, and evidence of restenosis >50% underwent diagnostic angiography. Angioplasty, thrombolysis, thrombectomy, or additional stent insertion was performed according to the clinical status. Some edge stenosis was observed using angiography after thrombus removal through thrombectomy or thrombolysis. In such circumstances, the lumen loss was assumed to be related to the edge stenosis.

Adverse events included stroke, myocardial infarction, acute renal insufficiency, and procedure-related complications. Loss of primary patency was defined as angiographic evidence of stenosis >50% or occlusion, which was treated with target lesion revascularization (TLR) if clinically needed. Loss of secondary patency was defined as a failure of revascularization through an endovascular intervention or thrombectomy.

Statistical Analysis

Categorical variables are expressed as frequencies (percentages) and were compared using the chi-squared test. Continuous variables are expressed as mean ± standard deviation (range) and were compared using the Student *t* test. The Kaplan-Meier method was used to estimate patency and freedom from TLR up to 24 months for the 2 groups; the log-rank test was employed to compare the curves. Multiple logistic regression analysis was used to determine factors influencing edge stenosis; variables achieving $p<0.1$ in the univariate analysis were entered into the regression model in a stepwise fashion. Results are presented as the odds ratio (OR) and 95% confidence interval. The threshold of statistical significance was $p<0.05$. All statistical analyses were performed using IBM SPSS software (version 22.0; IBM Corporation, Armonk, NY, USA).

Table 1. Demographics and Clinical Characteristics of Patients With vs Without Edge Stenosis.[a]

	No Edge Stenosis (n=88)	Edge Stenosis (n=22)	p
Age, y	72.8±7.8 (49–92)	75.0±7.0 (64–90)	0.230
Men	63 (71.6)	15 (68.2)	0.753
Smoking	57 (64.8)	14 (63.6)	0.921
Hypertension	52 (59.1)	11 (50.0)	0.441
Diabetes mellitus	54 (61.4)	20 (90.9)	0.008
Hyperlipidemia	33 (37.5)	9 (40.9)	0.768
Carotid stenosis	28 (31.8)	7 (31.8)	>0.999
COPD	16 (18.2)	6 (27.3)	0.340
Prior stroke	18 (20.5)	6 (27.3)	0.489
CAD	38 (43.2)	14 (63.6)	0.086
Hemodialysis	26 (29.5)	7 (31.8)	0.835
Gastric ulcer	13 (14.8)	1 (4.5)	0.198
Rutherford category			0.853
3	8 (9.1)	1 (4.5)	
4	31 (35.2)	8 (36.4)	
5	34 (38.6)	8 (36.4)	
6	15 (17.0)	5 (22.7)	
ABI	0.51±0.13 (0.12–0.70)	0.48±0.13 (0.21–0.67)	0.386
Distal runoff vessels	1.8±0.6	1.5±0.5	0.072
1	22 (25.0)	10 (45.5)	
2	56 (63.6)	12 (54.5)	
3	10 (11.4)	0 (0.0)	
Lesion length, cm	22.4±4.2 (15–32)	23.2±5.7 (16–33)	0.537

Abbreviations: ABI, ankle brachial index; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease.
[a]Continuous data are presented as the means ± standard deviation (range); categorical data are given as the counts (percentage).

Results

Of the 110 patients assessed, 22 (20%) developed edge stenosis (20 at the distal edge) vs 88 who did not. The demographics and medical history (Table 1) were similar, though patients in the edge stenosis group had a significantly higher prevalence of diabetes (p=0.008). Of note, 80 (91.9%) patients without edge stenosis and 21 (95.5%) of 22 in the edge stenosis group had CLI. More patients in the edge stenosis group required retrograde access for successful wire passage through the SFA lesions (p=0.033; Table 2).

Access-site complications included puncture site hematoma (n=8) and pseudoaneurysm formation (n=2); the incidences did not differ between the groups (Table 3). Five cases of thrombosis required intervention; edge stenosis was diagnosed in 3 by arteriography (p=0.022). All patients in the edge stenosis group underwent a TLR (p<0.001), which resulted in a lower 1-year primary patency rate (p<0.001). However, both groups had similar 1-year secondary patency rates (Table 3).

Since the application of DCBs was intended to prevent edge stenosis, these 38 patients were removed from the risk factor analysis. In the univariate analysis of the remaining 72 patients, the factors achieving p<0.1 were diabetes, coronary artery disease, retrograde access, and adequacy of distal runoff. After stepwise selection methods were applied, only diabetes and adequacy of distal runoff were selected for multivariate analysis. The adequacy of distal runoff emerged as the sole predictor of edge stenosis (OR 0.31, 95% CI 0.11 to 0.83, p=0.020).

Analyzing the efficacy of DCBs in this setting, only 3 (7.9%) of the 38 patients who had DCB reinforcement developed edge stenosis (p=0.021; Figure 1), which lowered the need for TLR and led to superior 1-year primary patency in these patients (92.1% vs 76.4%, p=0.042; Figure 2A). Freedom from TLR at 24 months was 62.2% (95% CI 47.9% to 73.6%) in the group without DCB use vs 91.2% (95% CI 75.1% to 97.1%) for the group with DCB reinforcement (p=0.018; Figure 2B).

Discussion

Viabahn failure is not related to SFA disease severity or lesion length but rather to restenoses occurring at the ends of the covered stent.[15,16] However, few studies have analyzed the risk factors for this edge stenosis or have distinguished this phenomenon from other forms of lumen narrowing. Previous studies have stated that edge stenosis accounts for 52.2% to 100% of all stenoses and occlusions.[3,11,12,17] In our study, the vast majority of Viabahn failures that required intervention were due to edge stenosis; only 2 patients presented with acute occlusion and thrombosis that could have

Table 2. Procedure Characteristics and Outcomes of Patients With vs Without Edge Stenosis.[a]

	No Edge Stenosis (n=88)	Edge Stenosis (n=22)	p
Procedure variables			
Contralateral access	88 (100)	22 (100)	>0.999
Retrograde access	17 (19.3)	9 (40.9)	0.033
Number of stents			0.307
1	2 (2.3)	0 (0.0)	
2	70 (79.5)	15 (68.2)	
3	16 (18.2)	7 (31.8)	
Size of stent, mm			0.252
5	5 (5.7)	0 (0.0)	
6	83 (94.3)	22 (100)	
Contrast volume, mL	60.8±15.6 (24–99)	62.9±32.1 (24–130)	0.777
DCB reinforcement	35 (39.8)	3 (13.6)	0.021
Technical success	88 (100)	22 (100)	>0.999
BTK intervention	68 (77.3)	21 (95.5)	0.052
Iliac stenting	23 (26.1)	8 (36.4)	0.340
Outcomes			
Complications	8 (9.1)	2 (9.1)	>0.999
Stent fracture	0 (0.0)	2 (9.1)	0.004
Death	8 (9.1)	1 (4.5)	0.487
PAD-related death	4 (4.5)	1 (4.5)	>0.999
Major amputation	4 (4.5)	0 (0.0)	0.308
AMI	4 (4.5)	1 (4.5)	>0.999
Stroke	2 (2.3)	0 (0.0)	0.475
Dialysis	2 (2.3)	0 (0.0)	0.475
TLR	2 (2.3)	22 (100)	<0.001
Acute thrombosis	2 (2.3)	3 (13.6)	0.022
1-Year ABI	0.99±0.08 (0.79–1.21)	0.94±0.08 (0.79–1.07)	0.022
1-Year primary patency	80 (90.9)	10 (45.5)	<0.001
1-Year secondary patency	80 (90.9)	21 (95.5)	0.487

Abbreviations: ABI, ankle-brachial index; AMI, acute myocardial infarction; BTK, below the knee; DCB, drug-coated balloon; PAD, peripheral artery disease; TLR, target lesion revascularization.
[a]Continuous data are presented as the means ± standard deviation (range); categorical data are given as the counts (percentage).

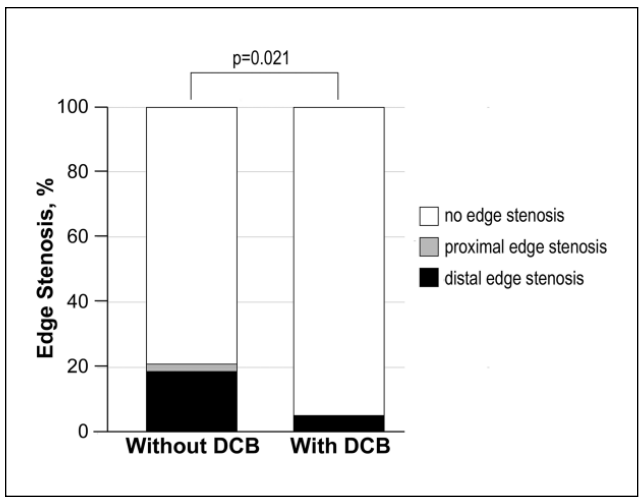

Figure 1. Incidence of edge stenosis after Viabahn implantation with or without drug-coated balloon (DCB) reinforcement.

been related to poor compliance with the antiplatelet regimen or poor distal runoff.

In our study, 20 of 22 edge stenoses were distal, but other studies have had different findings. The VIPER study reported that 50% of patients had proximal stenosis, 11% had distal stenosis, and 33% had both.[3] Golchehr et al[18] reported that 63% of edge stenosis events occurred at the proximal end and 37% at the distal end. Thus, the accurate incidence of edge stenosis and its distribution at the proximal or distal end remain unclear. In addition, it is unknown whether the different types of edge stenosis result in different clinical presentations and risks of acute thrombosis and total occlusion.

Our results revealed that diabetes, coronary artery disease, retrograde access, and adequacy of distal runoff are predictors of edge stenosis after Viabahn implantation. However, only poor outflow remained as the independent predictor of edge stenosis after multivariate analysis. There

Table 3. Outcomes and Follow-up of DCB Reinforcement at the Stent-Graft Distal Edge.[a]

	Without DCB (n=72)	With DCB (n=38)	p
Complications	7 (9.7)	3 (7.9)	0.751
Stent fracture	2 (2.8)	0 (0.0)	0.300
Edge stenosis	19 (26.4)	3 (7.9)	0.021
Proximal	2 (2.8)	0 (0.0)	0.300
Distal	17 (23.6)	3 (7.9)	0.042
Death	8 (11.1)	1 (2.6)	0.123
PAD-related death	4 (5.6)	1 (2.6)	0.484
Major amputation	4 (5.6)	0 (0.0)	0.139
AMI	4 (5.6)	1 (2.6)	0.484
Stroke	1 (1.4)	1 (2.6)	0.643
Dialysis	2 (2.8)	0 (0.0)	0.300
TLR	21 (29.2)	3 (7.9)	0.010
1-Year ABI	0.97±0.08 (0.79–1.21)	0.99±0.08 (0.79–1.18)	0.232
1-Year primary patency	55 (76.4)	35 (92.1)	0.042
1-Year secondary patency	64 (88.9)	37 (97.4)	0.123

Abbreviations: ABI, ankle brachial index; AMI, acute myocardial infarction; DCB, drug-coated balloon; PAD, peripheral artery disease; TLR, target lesion revascularization.
[a]Continuous data are presented as the means ± standard deviation (range); categorical data are given as the counts (percentage).

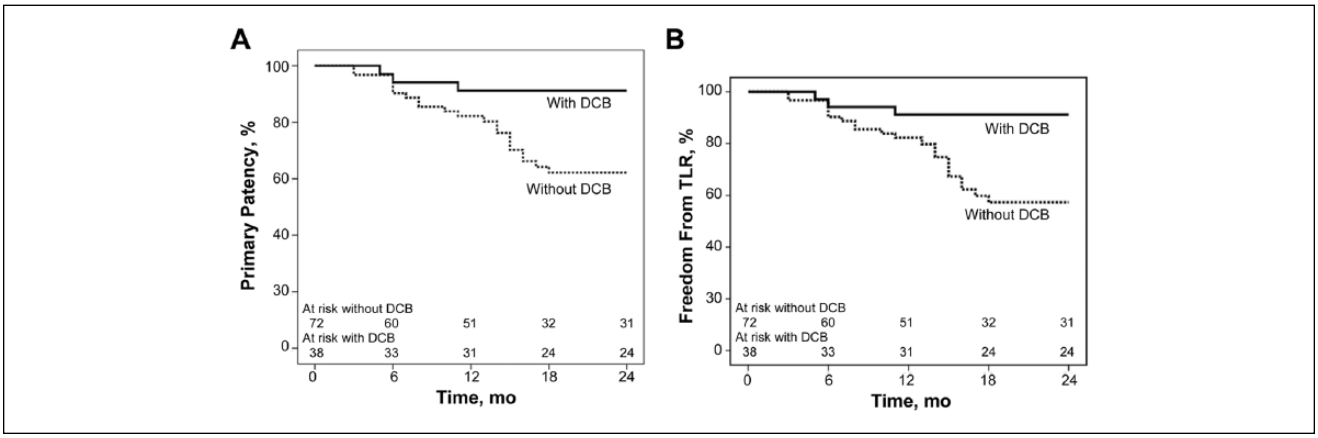

Figure 2. Kaplan-Meier curves of (A) primary patency and (B) freedom from target lesion revascularization (TLR). The standard errors did not exceed 10% at 24 months for all curves.

was a signal that diabetes might be a predictor for edge stenosis (p=0.066), but it was not significant in our study, likely due to the small sample size. Future studies should investigate this association.

Our use of the Viabahn stent-graft in the SFA began in 2011, and DCBs were not available in our institution until 2013. From 2011 to 2013, 24 patients received a Viabahn device. A third of these patients developed edge stenosis, all at the distal verge. As a result, a decision was made to use DCB to reinforce the distal edge of the Viabahn, and the outcome has supported this decision. Accordingly, a significantly higher 1-year primary patency rate was observed in the DCB reinforcement group. The patients without DCB reinforcement had 1-year primary and secondary patency estimates (76.4% and 88.9%, respectively) that were comparable to those published in a recent review article (58% to 80% and 57% to 93.4%, respectively)[5] as well as the Viabahn 25-cm trial for long SFA lesions.[19] Thus, DCB reinforcement at the edge can significantly improve the outcome of SFA treatment with the Viabahn stent-graft.

A heparin-bonded Viabahn came to the market in September 2007 but was not available in our country during the course of this study. The VIPER, VIASTAR, and other recent studies have used a heparin-bonded Viabahn exclusively.[2,3,7,20] Some investigations, including VIBRANT,[1,21] have combined heparin-bonded and non–heparin-bonded models. Kruse et al[21] analyzed the factors predicting the 5-year outcome of Viabahns in the SFA and

discovered that a heparin-bonded Viabahn does not outperform a non–heparin-bonded Viabahn in terms of patency. The benefit of heparin bonding may lie in its ability to improve the outcomes of the 5-mm-diameter Viabahn to a level comparable with larger-diameter devices.[3] This was suggested by the VIPER study. Although several studies concluded that the patency of the 5-mm Viabahn is poorer than that of larger-diameter devices, this was not observed in the VIPER study.[11,22,23] Otherwise, no evidence exists that a heparin-bonded Viabahn has lower restenosis or edge stenosis rates than does a non–heparin-bonded Viabahn.

Using CTA as a surveillance tool brought the concern of renal impairment and radiation exposure. Although duplex ultrasound is regarded as a standard lower limb imaging technique in most institutions, multisegment stenosis, as occurred in this study, was shown to have a significant negative impact on the qualification of native artery stenosis in the femoropopliteal segment.[24] Thus, this study used CTA owing to its availability, operator independence, and sensitivity.

Limitations

To the best of our knowledge, no one has until now analyzed the edge stenosis characteristics of a Viabahn stent-graft. However, our study had several limitations. First, this was a nonrandomized, retrospective study and the sample size was small. Second, the non-reimbursement of DCBs in our country could have introduced bias. However, the baseline underlying condition in both groups was comparable, and our intervention strategy may provide a promising solution. Third, some of the patients did not have their 2-year follow-up at the time this report was written; therefore, only the 1-year results were analyzed. Furthermore, the period of protection offered by DCB application and the corresponding long-term outcomes warrant further investigation.

Conclusion

Poor distal runoff was a predisposing factor for the development of edge stenosis after Viabahn implantation to treat long SFA occlusions. DCB reinforcement over the distal Viabahn edge may prevent edge stenosis, resulting in fewer TLRs and superior primary patency at 1 year vs Viabahn implantation alone.

Acknowledgments

We thank the Taiwan Association of Cardiovascular Surgery Research for their assistance.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

ORCID iD

I-Ming Chen https://orcid.org/0000-0001-5532-7957

References

1. Geraghty PJ, Mewissen MW, Jaff MR, et al. Three-year results of the VIBRANT trial of VIABAHN endoprosthesis versus bare nitinol stent implantation for complex superficial femoral artery occlusive disease. *J Vasc Surg*. 2013;58:386–395.e4.
2. Lammer J, Zeller T, Hausegger KA, et al. Sustained benefit at 2 years for covered stents versus bare-metal stents in long SFA lesions: the VIASTAR trial. *Cardiovasc Intervent Radiol*. 2015;38:25–32.
3. Saxon RR, Chervu A, Jones PA, et al. Heparin-bonded, expanded polytetrafluoroethylene-lined stent graft in the treatment of femoropopliteal artery disease: 1-year results of the VIPER (Viabahn Endoprosthesis with Heparin Bioactive Surface in the Treatment of Superficial Femoral Artery Obstructive Disease) trial. *J Vasc Interv Radiol*. 2013;24:165–173.
4. Saxon RR, Dake MD, Volgelzang RL, et al. Randomized, multicenter study comparing expanded polytetrafluoroethylene-covered endoprosthesis placement with percutaneous transluminal angioplasty in the treatment of superficial femoral artery occlusive disease. *J Vasc Interv Radiol*. 2008;19:823–832.
5. Zhang L, Bao J, Zhao Z, et al. Effectiveness of Viabahn in the treatment of superficial femoral artery occlusive disease: a systematic review and meta-analysis. *J Endovasc Ther*. 2015;22:495–505.
6. Kedora J, Hohmann S, Garrett W, et al. Randomized comparison of percutaneous Viabahn stent grafts vs prosthetic femoral-popliteal bypass in the treatment of superficial femoral arterial occlusive disease. *J Vasc Surg*. 2007;45:10–16.
7. Lammer J, Zeller T, Hausegger KA, et al. Heparin-bonded covered stents versus bare-metal stents for complex femoropopliteal artery lesions: the randomized VIASTAR trial (Viabahn endoprosthesis with PROPATEN bioactive surface [VIA] versus bare nitinol stent in the treatment of long lesions in superficial femoral artery occlusive disease). *J Am Coll Cardiol*. 2013;62:1320–1327.
8. Capek P, McLean GK, Berkowitz HD. Femoropopliteal angioplasty. Factors influencing long-term success. *Circulation*. 1991;83:I70–I80.
9. Scheinert D, Scheinert S, Sax J, et al. Prevalence and clinical impact of stent fractures after femoropopliteal stenting. *J Am Coll Cardiol*. 2005;45:312–315.
10. Johnston KW. Femoral and popliteal arteries: reanalysis of results of balloon angioplasty. *Radiology*. 1992;183:767–771.
11. Saxon RR, Coffman JM, Gooding JM, et al. Long-term patency and clinical outcome of the Viabahn stent-graft for femoropopliteal artery obstructions. *J Vasc Interv Radiol*. 2007;18:1341–1349.
12. Golchehr B, Lensvelt MM, Fritschy WM, et al. Outcome of thrombolysis and thrombectomy for thrombosed endografts inserted in the superficial femoral artery for occlusive disease. *J Endovasc Ther*. 2013;20:836–843.

13. Steinfeld DS, Liu AP, Hsu SH, et al. Comparative assessment of transient exposure of paclitaxel or zotarolimus on in vitro vascular cell death, proliferation, migration, and proinflammatory biomarker expression. *J Cardiovasc Pharmacol*. 2012;60:179–186.
14. Norgren L, Hiatt WR, Dormandy JA, et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). *J Vasc Surg*. 2007;45(suppl S):S5–S67.
15. Deutschmann HA, Schedlbauer P, Berczi V, et al. Placement of Hemobahn stent-grafts in femoropopliteal arteries: early experience and midterm results in 18 patients. *J Vasc Interv Radiol*. 2001;12:943–950.
16. Verta MJ, Schneider JR, Alonzo MJ, et al. Percutaneous Viabahn-assisted subintimal recanalization for severe superficial femoral artery occlusive disease. *J Vasc Interv Radiol*. 2008;19:493–498.
17. Mohr PJ, Oyama JK, Luu JT, et al. Clinical outcomes of endovascular treatment of TASC-II C and D femoropopliteal lesions with the Viabahn endoprosthesis. *Cardiovasc Revasc Med*. 2015;16:465–468.
18. Golchehr B, Holewijn S, Kruse RR, et al. Efficacy of treatment of edge stenosis of endografts inserted for superficial femoral artery stenotic disease. *Catheter Cardiovasc Interv*. 2015;86:492–498.
19. Zeller T, Peeters P, Bosiers M, et al. Heparin-bonded stent-graft for the treatment of TASC II C and D femoropopliteal lesions: the Viabahn 25-cm trial. *J Endovasc Ther*. 2014;21:765–774.
20. Golchehr B, Kruse R, van Walraven LA, et al. Three-year outcome of the heparin-bonded Viabahn for superficial femoral artery occlusive disease. *J Vasc Surg*. 2015;62:984–989.
21. Kruse RR, Poelmann FB, Doomernik D, et al. Five-year outcome of self-expanding covered stents for superficial femoral artery occlusive disease and an analysis of factors predicting failure. *J Endovasc Ther*. 2015;22:855–861.
22. Schneider JR, Verta MJ, Alonzo MJ, et al. Results with Viabahn-assisted subintimal recanalization for TASC C and TASC D superficial femoral artery occlusive disease. *Vasc Endovascular Surg*. 2011;45:391–397.
23. McQuade K, Gable D, Pearl G, et al. Four-year randomized prospective comparison of percutaneous ePTFE/nitinol self-expanding stent graft versus prosthetic femoral-popliteal bypass in the treatment of superficial femoral artery occlusive disease. *J Vasc Surg*. 2010;52:584–590.
24. Macharzina RR, Schmid SF, Beschorner U, et al. Duplex ultrasound assessment of native stenoses in the superficial femoral and popliteal arteries: a comparative study examining the influence of multisegment lesions. *J Endovasc Ther*. 2015;22:253–260.