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Visceral stent patency in fenestrated stent grafting for abdominal aortic aneurysm repair

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Objective: **Fenestrated endovascular abdominal aortic aneurysm repair (F-EVAR) has been introduced for treatment of aneurysms in which visceral arteries are incorporated. Patency of target vessels has been reported to be excellent. Results of the use of stent grafts to accommodate visceral arteries in F-EVAR are presented in this study, including an overview of factors that affect outcome.**
Methods: **All patients treated with fenestrated stent grafts in a single center between November 2001 and October 2011 were reviewed. Patients treated for suprarenal, juxtarenal, and infrarenal short-necked aortic aneurysms were included. Patients with thoraco-abdominal aneurysms or aneurysms treated with grafts with fixed side branches were excluded. Polytetrafluoroethylene covered stents were used routinely since June 2005. Target vessels and stents were examined using computed tomography angiography reconstructions. Primary end points were primary patency, defined as the absence of occlusion, and loss of renal function. Secondary end points were technical success, stenosis (defined as a ≥50% angiographic diameter reduction), stent fracture, and mortality.**
Results: **A total of 138 patients with a median age of 73 years (range, 50-91 years) met the inclusion criteria. Median computed tomography angiography follow-up was 13 months (range, 1-97 months). In total, 392 target vessels were provided with 140 scallops and 252 fenestrations. Visceral stents (−grafts) were placed in 254 target vessels. Technical success was obtained in 249 arteries (98.0%). Overall stent patency of target vessels was 95.7% at 1 year and 88.6% at 4 years. Renal artery stent patency was 97.4% at 1 year and 91.2% at 4 years (96.8% and 89.1% for uncovered stents; 97.3% and 92.4% for covered stents, respectively). There was no significant difference in patency between covered and uncovered stents in renal arteries ($P = .71$). Renal artery stenosis occurred in 26 stented arteries (11.3%) and occlusion in seven arteries. Renal artery stent stenosis occurred significantly more in uncovered than in covered stents ($P = .04$). Stent fractures occurred more in uncovered than in covered stents ($P = .01$) and was associated with a significantly lower visceral stent patency rate ($P < .01$). During follow-up, 13 patients developed permanent renal function impairment (9.4%), of which two required permanent dialysis (1.4%). Renal dysfunction was significantly associated with renal stent occlusion or stenosis ($P < .01$).**
Conclusions: **Patency rates of visceral artery stent (−grafts) in F-EVAR were 95.7% at 1 year and 88.6% at 4 years. Patency rates were affected by stent fractures, which occurred more in uncovered compared with covered stents. Renal artery stent stenosis occurred more in uncovered compared with covered stents. Renal dysfunction was significantly associated with renal stent occlusion or stenosis. (J Vasc Surg 2014;59:298-306.)**

Endovascular abdominal aortic aneurysm repair (EVAR) was introduced in 1986.[1] Since then, the use of the technique has evolved from treating infrarenal abdominal aortic aneurysms (AAAs) to more complex fenestrated (F-EVAR) and branched stent grafting for juxta- and suprarenal AAAs.[2,3] No randomized controlled trials comparing F-EVAR and open repair have been conducted. Retrospective cohort studies, however, showed that the 30-day mortality has been reduced from 3.6% for open repair to 1.4% for F-EVAR.[4]

Major complications of endovascular repair of juxta- and suprarenal aneurysms include renal failure and mesenteric ischemia.[5] Few reports described details on the outcome of visceral artery stenting in F-EVAR. Overall estimated 1-year patency of visceral target vessels was reported up to 92%, with a renal artery patency rate of 85%.[4] Our group reported a cumulative visceral target vessel patency (275 target vessels in 100 patients) of 93.3% at 5 years.[6]

Few studies focused on renal-related outcome of F-EVAR. In one report, baseline renal dysfunction was a strong predictor for mortality. Adverse renal events, including renal artery stenosis, renal artery occlusion, and hemodialysis, occurred in 40% of the patients within the first year after treatment. Renal artery stenosis (>50%) after treatment was detected in 10 of 142 (7.0%) and occlusion

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in five of 142 (3.5%) treated vessels.[7] One report revealed significantly more stenoses in uncovered stents (10%) compared with covered stents (2.5%).[8] Recently, Mastracci et al showed low branch-related death after F-EVAR for thoraco-abdominal and juxtrarenal aneurysms (three of 650 patients), with freedom of reintervention of 89% at 5 years.[9]

Little is known about factors that may affect outcome of visceral artery stenting in F-EVAR. Possible factors are preprocedural renal function,[7] aneurysm size, visceral artery ostium stenosis, the angle of the target vessel with the aorta, and failure of material.

This study reports on the outcome of visceral artery stenting after F-EVAR of abdominal aneurysms in terms of stent patency and technical failure. Results of covered stents in target vessels are analyzed specifically. Additionally, factors influencing patency are analyzed.

METHODS

Patients. All patients treated with fenestrated and branched stent grafts in our tertiary referral center were enrolled in an investigational device protocol database. Indications for treatment with F-EVAR were abdominal short-necked, juxtarenal, or suprarenal AAAs with a diameter ≥55 mm in men and ≥50 mm in women, as well as AAAs <55 mm in conjunction with an iliac artery aneurysm diameter ≥35 mm. All patients treated for such an aneurysm in a 10-year period (November 2001 to October 2011) were analyzed. In this study, thoraco-abdominal aneurysms were excluded. Demographic details on comorbidity were noted.

Preoperative assessment. Thin-cut (≤1 mm) spiral computed tomography angiography (CTA) was performed to assess aneurysm morphology and determine position and patency of visceral vessels. For all patients, a customized fenestrated device based on the three-part Cook Zenith System (William A. Cook Australia, Ltd, Brisbane, Australia) was constructed.[10] The clock position of the fenestrations was noted in degrees where 0° equals the 12 o'clock position, −90° the 9, 90° the 3, and 180° the 6 o'clock position. Renal artery angulation in relation to the aorta was measured on coronal multiplanar reconstructions after adjustment of the central lumen line of the aorta in the sagittal plane. Grafts were fitted with diameter-reducing ties to allow partial deployment before catheterization of side branches and final orientation of the stent graft. In this way, both rotational and craniocaudal repositioning of the device was made possible to allow catheterization of target vessels. Fenestrations included scallops and small and large fenestrations. In target vessels, three types of stents were used: uncovered stents (Genesis stent; Cordis Corporation, Miami Lakes, Fla) and polytetrafluoroethylene-covered stents (Jostent stent graft; Abbott Laboratories, Abbott Park, Ill), and Advanta V12 stent graft (Atrium Medical, Hudson, NH). Fenestrations had radiopaque markers to enable accurate alignment during implantation.

Procedure. The detailed operative technique has been described before.[10-13] Briefly, patients received heparin (5000 IU) intravenously at the time when the first sheath was introduced, and an additional dose of 2500 IU was administered after 90 minutes. Since 2010, we have been monitoring the activated clotting time to adjust heparin dosage to an activated clotting time of 250 seconds. The stent grafts in the fenestrations were flared with a 12 mm × 20 mm percutaneous transluminal angioplasty balloon to improve apposition and fixation at the level of the fenestration and in most cases also with a compliant balloon (Reliant Stent Graft Balloon Catheter; Medtronic, Minneapolis, Minn) for additional flaring. After stenting of each target vessel, a selective angiography through the flaring balloon catheter was performed to demonstrate patency and outflow. After the procedure, completion angiography was carried out to confirm overall vessel patency and exclusion of the aneurysm. Procedural data including contrast volume and technical features of stent placement were collected.

Follow-up. Patients were followed up with abdominal X rays during admission, CTA at 6 weeks, and abdominal X rays, duplex, CTA, and renal function analysis yearly thereafter to assess (target) vessel patency, stent fracture, endoleak, and migration. X rays were performed in four views, including anteroposterior, lateral, and left and right anterior oblique.

All patients received anti-platelet therapy after the procedure. Acetylsalicylic acid was given on the day of the procedure. Clopidogrel was started after removal of spinal or epidural catheters and for a minimum duration of 2 months. In addition, renal function and blood pressure were monitored.

Three-dimensional volumetric CTA reconstructions were made and analyzed on an Aquarius workflow platform (TeraRecon, Foster City, Calif). In all reconstructions, diameters were measured at the level of the fenestration, in-stent, and at the distal end of the stent. In addition, any changes in the configuration of the stents were noted. Target vessel angulations in relation to the aorta were noted in degrees, before and after the procedure. The aneurysm sac diameter was measured perpendicular to the axis of the aorta, in two directions, before and after the procedure.

Definitions and statistics. Reporting standards for endovascular aortic aneurysm repair and renal artery stenting were used for definitions and analysis of outcome.[14-17] Short-necked aneurysms were defined as having a proximal neck length below the lowest renal artery of 4 to 12 mm. Juxtarenal aneurysms were defined as AAAs with aneurysmal extension up to the inter-renal aorta, or AAAs with normal inter-renal aorta but aneurysmal involvement of one of the renal artery origins. Suprarenal aneurysms were defined as extending above the renal aorta but below the celiac trunk.

Target vessels were defined as vessels for which a scallop or fenestration was needed. Stenting of a target vessel was considered technically successful when there was a <30% residual stenosis, and the stent was positioned within the target vessel. Technical success of F-EVAR was defined as a completed endovascular procedure with patent target

vessels and in absence of conversion or endoleak type I or III.

In follow-up of target vessels and stents, an angiographic diameter reduction of >50%, comparing diameters distal from the stenosis and at the stenosis was considered hemodynamically significant and was therefore appointed as "stenosis."[15,18,19] Additionally, occlusions were noted. Physiologic renal function was monitored using calculated estimated glomerular filtration rate (eGFR), determined by using the abbreviated Modification of Diet in Renal Disease study equation: eGFR (mL/min/1.73 m^2) = 186 × (serum creatinine (μmol/L))$^{-1.154}$ × (age)$^{-0.203}$ (× 0.742; if female).[20] Renal dysfunction was defined as an eGFR lower than 60 mL/min/1.73 m^2. Renal function impairment or decrease was defined as a 30% reduction of eGFR. Transient renal function impairment was defined as a temporary reduction of at least 30% eGFR that was restored during follow-up. Permanent renal function impairment was defined as a permanent >30% reduction of eGFR without improvement.

Primary patency was defined as the uninterrupted patency in absence of occlusion, with no procedures performed on the vessel or stent to prevent occlusion.[15]

Statistical analysis was done with SPSS version 19.0 (Statistical Package for the Social Sciences, Inc., Chicago, Ill). Variables were expressed as mean ± one standard deviation in case of normal distribution, or as median plus range in other distributions.

Patency was estimated using Kaplan-Meier survival analysis. To test for statistically significant survival differences, the generalized Wilcoxon (Breslow) or the log-rank (Mantel-Cox) tests were used. Patency rates were based on a per-artery analysis. Additionally, a per-patient analysis was performed to assess for predictive factors that may affect patency.

Differences between groups were determined using analysis of variance with $P < .05$ considered significant.

Primary end points were primary patency and renal function. Secondary end points were technical success, stenosis, stent fracture, and mortality.

RESULTS

Outcome of F-EVAR. F-EVAR was performed in 138 patients, including 107 primary procedures (77.5%) and 31 F-EVAR procedures (22.5%) after previous open surgical AAA repair (n = 19), EVAR (n = 8), or other aortic repairs (n = 4). One conversion to an open procedure was due to migration of the endograft and occlusion of both renal arteries during the procedure.

Technical success of F-EVAR was obtained in 127 of 138 procedures (92.0%). Within 30 days after the procedure, 69 complications were noted in 31 patients (22.3%; Table I). At completion angiography, there were five type I endoleaks, which were all followed up on. At the first follow-up, two of the five type I endoleaks had persisted but resolved with conservative treatment. Type II endoleaks discovered at completion angiography persisted in 13 of the 18 cases at first follow-up. In one patient, a type II endoleak caused by three large lumbar arteries was treated surgically. Follow-up revealed another type I and eight type II endoleaks, of which one type II endoleak was treated by coil embolization. The reason for treating type II endoleaks was ongoing aneurysm growth.

Table I. Complications

Procedural complications	
Dislocation prosthetic limb (n = 1)	Fem-fem bypass
Disconnection stents prosthetic limb (n = 1)	Plug CIA + fem-fem bypass
Dislocation prosthetic limb (n = 1)	Covered stent placement
Unsuccessful target vessel stenting (n = 4)	Overstented
Endoleak type 1 (n = 5)	Followed up
Dissection (n = 2)	Followed up
Rupture femoral artery (n = 1)	Surgical interposition graft
Migration aortic endograft (n = 1)	Endovascular repositioning graft caudally
Occlusion renal artery (n = 1)	Retrogradely revascularization
Postoperative complications	
Pulmonary (n = 6)	Temporary injury
Cardiac event (n = 12)	One patient sustained damage
Ischemic cerebrovascular accident (n = 2)	Both patients remaining neurologic deficit
Delirium (n = 3)	Temporary impairment
Gastrointestinal (n = 8)	Two patients deceased because of bowel ischemia
Urinary tract infection/ retention (n = 10)	Temporary impairment
Decrease renal function (n = 13)	Five patients permanent renal function loss
Wound infection/ hematoma (n = 6)	Temporary injury
Non-infectious fever (n = 3)	Temporary injury
Other (n = 6)	Temporary injury

CIA, Common iliac artery.

One F-EVAR procedure was converted to an open procedure and therefore not included for analysis. Median age of the patients was 73 years (range, 50-91 years). Demographic details are described in Table II. CTA follow-up was not available for 11 patients (8.0%), and therefore they were considered lost to follow-up. Median CTA follow-up for the other 127 patients was 13 months (range, 1-97 months). CTA follow-up at 1, 2, and 4 years was available for 72, 50, and 15 patients, respectively. Renal function impairment occurred in four patients, and therefore no CTA was performed. Additionally, CTAs performed in other hospitals were of low quality (5-mm slices) and therefore not adequate for three-dimensional reconstruction and stent measurement.

Thirty-day mortality was 1.4%. Two patients died within the first month after the procedure, both due to bowel ischemia. During follow-up, 56 (40.5%) patients died, of whom 47 died (34.1%) within 5 years of the

Table II. Patient characteristics

	Total (N = 138)	
	No.	*%*
Male	123	89.1
Smoking	79	58.5
Hypertension	120	87.6
Diabetes mellitus	21	15.2
Coronary artery disease	89	69.5
Neurologic disease	25	18.1
Pulmonary disease	64	48.9
Renal disease	49	35.5
Age, years (range)	73 (50-91)	

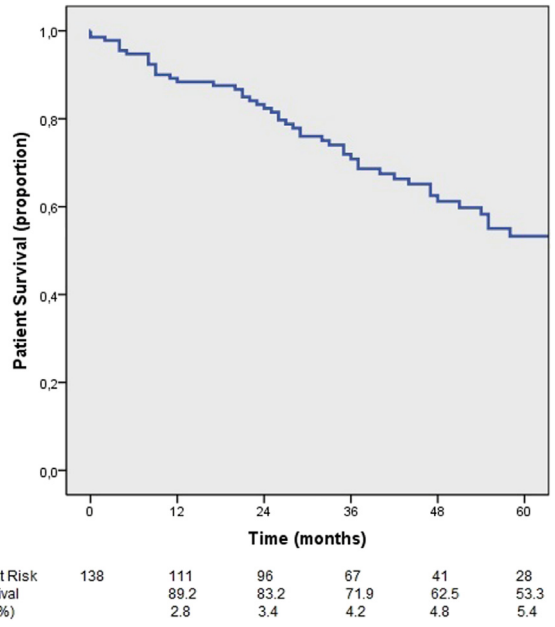


Fig 1. Kaplan-Meier curve showing overall patient survival. *SE,* Standard error.

procedure. Six aneurysm-related deaths were reported (4.3%). In one patient, dislocation of the main body had resulted in a large endoleak. The other patients died as a result of other aneurysm-related problems (infection of the prosthesis, bowel ischemia, and mycotic aneurysm). The cumulative survival probability of patients 1 and 4 years after the procedure was 89.2% and 62.5%, respectively (Fig 1).

Target vessels. In total, 392 target vessels in 138 patients were provided with 140 scallops and 252 fenestrations. Stents were placed in 254 target vessels. Details of target vessels and stent types that were used are described in Table III and F-EVAR configurations in Table IV. Technically successful stent placement was obtained in 249 arteries (98.0%). Overall stent patency (absence of occlusion or intervention) was 95.7% at 1 year and 88.6% at 4 years (Fig 2). Renal artery stenosis occurred in 26 stented

Table III. Characteristics of target vessels

	Total	*Scallop, No.*	*Fenestration, No.*
Renal artery	261	33	228
No stent	31	26	5
Uncovered stent	71	6	65
Covered stent	153	1	152
Combination	1	-	1
Unknown stent type	5	-	5
SMA	110	87	23
No stent	87	86	1
Uncovered stent	6	1	5
Covered stent	9	-	9
Combination	7	-	7
Unknown stent type	1	-	1
Celiac trunk	21	20	1
No stent	20	20	-
Uncovered stent	0	-	-
Covered stent	1	-	1

SMA, Superior mesenteric artery.

Table IV. Fenestrated endovascular aneurysm repair (F-EVAR) configuration

Type	*No.*	*%*
Scallop SMA, fenestrated renal arteries	77	55.8
Scallop celiac trunk, fenestrated SMA, fenestrated renal arteries	20	14.5
Scallop renal arteries	8	5.8
One scallop renal artery, one fenestrated renal artery	7	5.1
Scallop SMA, one fenestrated/scallop renal artery	6	4.3
One fenestrated renal artery	6	4.3
Other	14	10.2

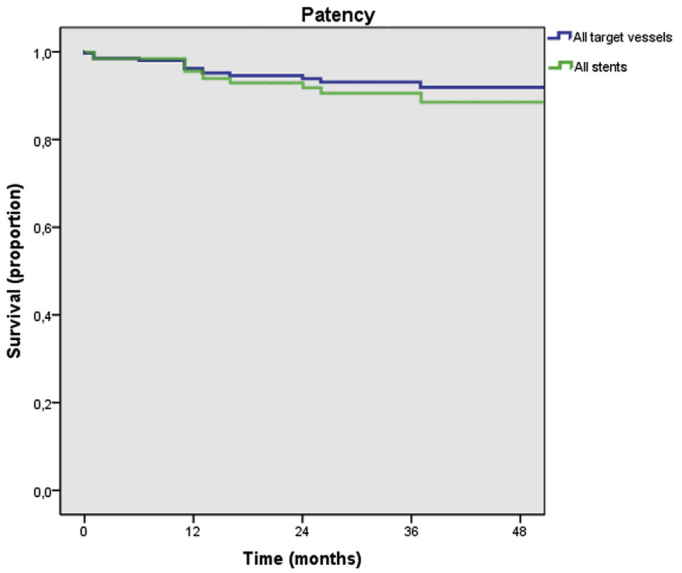
SMA, Superior mesenteric artery.

arteries (11.3%) in 24 patients and occlusion in seven arteries (3.0%) in five patients. Freedom from occlusion in renal artery stents was 97.4% at 1 year and 91.2% at 4 years (96.8% and 89.1% for uncovered; 97.3% and 92.4% for covered stents, respectively). There was no significant difference in renal stent patency between uncovered and covered stents ($P = .70$).

Freedom from stenosis in renal artery stents (occluded stents were excluded in this analysis) was 90.0% at 1 year and 71.4% at 4 years (84.5% and 58.9% for uncovered stents; 92.9% and 86.7% for covered stents, respectively). Stenosis was more prevalent in uncovered than in covered stents ($P = .04$; Fig 3). At 4 years, the combined outcome of freedom from stenosis or occlusion was 80.9% for covered stents and 70.7% for uncovered stents, without significant difference ($P > .05$; Fig 4).

Stent fractures (Fig 5) were seen in eight uncovered (10.3%) and two covered stents (1.2%; $P = .01$).

Stent fractures significantly reduced stent patency rate ($P < .01$). In total, 41 stenoses or occlusions occurred in 254 stents. In 22 cases with stenosis or occlusion, an in-stent kink was present (53.7%), and in five cases, the stent was fractured (12.2%). In the other 14 stents, there were no evident changes in stent configuration.



All target vessels					
No at Risk	361	198	137	92	39
Survival		96.2	94.6	93.1	91.9
SE (%)		1.2	1.5	1.8	2.2
All stented vessels					
No at Risk	198	123	83	54	20
Survival		95.7	91.9	90.7	88.6
SE (%)		1.6	2.4	2.7	3.3

Fig 2. Kaplan-Meier survival curve showing the overall patency rate of all target vessels and the patency rate of stents used in target vessels. *SE,* Standard error.

Stenosis or occlusions of target vessels led to reinterventions in 1.0% of the cases (four target vessels). In one patient, the body of the endoprosthesis migrated, thereby occluding both renal arteries. The body was repositioned surgically. An attempt to recanalize two renal arteries in two patients failed.

Univariate analysis (log-rank) showed no significant influence of diabetes ($P = .95$), smoking ($P = .07$), hypertension ($P = .97$), pulmonary status ($P = .22$), cardiac status ($P = .20$), carotid disease ($P = .12$), renal status ($P = .24$), or aneurysm sac size ($P = .53$) on stent patency.

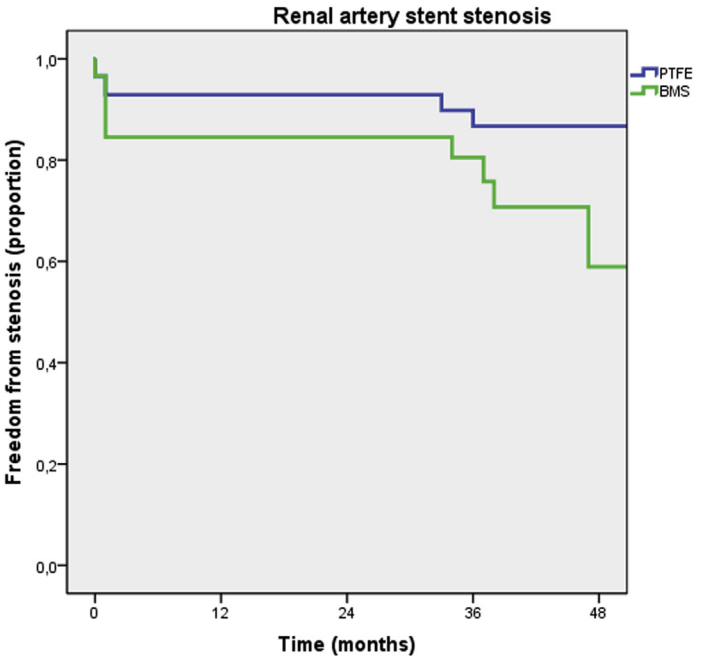
Renal outcome. Preprocedural renal dysfunction was noted in 49 patients (35.5%). There were no patients on hemodialysis before the procedure. The median volume of contrast used (270 mg/mL iodine solution) was 195 mL (range, 80-350 mL). Renal function impairment was not associated with contrast load ($P = .63$).

Permanent renal function impairment occurred in 13 patients (9.4%), within the first week after the procedure in four of them. Temporary renal function impairment was seen in 57 patients (41.3%), within the first week after the procedure in 18 of them (16.8%). Two patients developed dialysis-dependent renal function loss, caused by bilateral occlusion of the stented renal arteries. One of these patients received kidney transplantation. Transient and permanent renal function impairment did not significantly occur more in patients with preoperative renal dysfunction ($P = .58$ and $P = .77$, respectively) and was not significantly associated with mortality ($P = .21$). A pre-procedural eGFR <60 was significantly associated with mortality ($P = .04$). Renal artery stenosis or occlusion lead to a >30% decrease in eGFR in eight patients. Renal function impairment in general was significantly associated with renal stent occlusion or stenosis ($P < .01$).

Renal artery stenosis was seen in eight right renal arteries and 10 left renal arteries in patients before the procedure. Restenosis after stenting was detected in three renal arteries (three patients) and occlusion in three renal arteries (two patients).

Stent patency was not significantly associated with the angulation of the vessel before the procedure ($P = .71$), nor with an increase or decrease of the angulation after the procedure ($P = .86$). The clock position of the renal arteries did not influence stent patency ($P = .60$).

Mesenteric outcome. In total, 131 mesenteric vessels (celiac trunk and superior mesenteric artery [SMA]) were targeted. There were 107 scallops (of which one was stented) and 24 fenestrations (of which 23 were stented; Table III). Stents were placed in 24 mesenteric vessels in 23 patients. Due to this small group, the standard error for calculation of survival was >10%, and therefore no reliable stent patency rates could be calculated. During follow-up in four patients, stenosis was noted in two SMA stents, and occlusion occurred in one stented celiac trunk and in two SMAs. One of these patients died after both stents in celiac trunc and SMA had occluded (as well as both renal



PTFE					
No at Risk	116	56	40	29	6
Survival		92.9	92.9	89.8	86.7
SE (%)		2.4	2.4	3.8	4.8
BMS					
No at Risk	58	33	24	18	10
Survival		84.5	84.5	80.5	58.9
SE (%)		4.7	4.7	6.0	10.3

Fig 3. Kaplan-Meier survival curve showing freedom from stenosis in uncovered (bare metal stent [*BMS*]) and covered (polytetrafluoroethylene [*PTFE*]) stents in renal arteries ($P = .04$). *SE*, Standard error.

artery stents), which led to bowel ischemia, bowel resection, multiorgan failure, and death of the patient.

The other three patients remained symptom-free, and therefore no intervention was performed.

Bowel ischemia occurred in two patients in the follow-up period after the procedure (8.7%). Apart from the one mentioned above, one patient developed bowel ischemia of unknown origin with a patent targeted SMA.

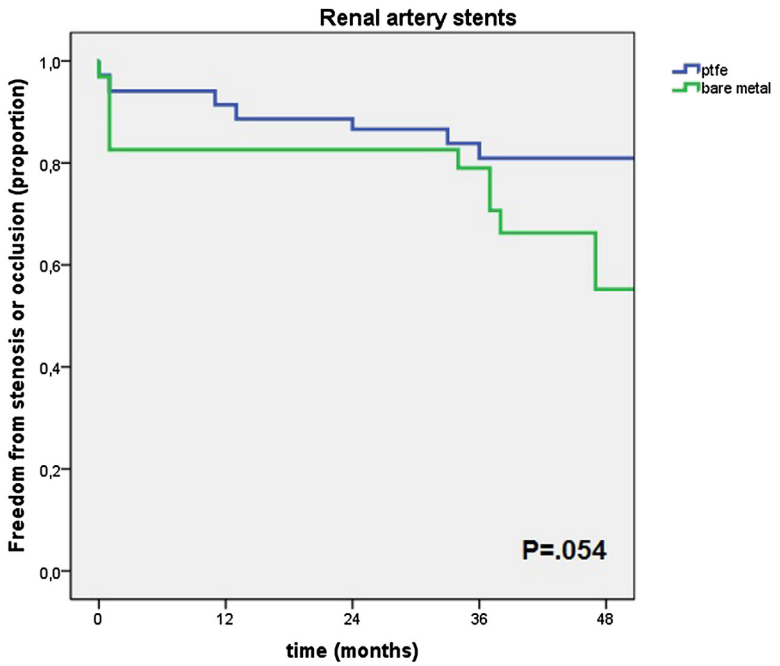
DISCUSSION

Results of F-EVAR in literature are generally good. Mortality rates have been reported to be 1.4% with target vessel patency of 93% at 5 years.[4,6] The effect of F-EVAR on target vessels, however, has not been addressed in many articles.

In 2005, Haddad et al outlined the effect of F-EVAR on target vessels, with special attention to the "renal side of the story."[7] The risk for adverse renal events was 16% in patients without preoperative renal dysfunction but was 39% for patients with preoperative renal dysfunction. In our study, postprocedural renal function impairment (transient as well as permanent) was not influenced by preoperative renal dysfunction. Haddad et al also described that adverse renal events were associated with death, a result we confirmed in our study. The advice of Haddad et al was to meticulously follow-up on patients and to reintervene without hesitation when renal artery stenosis or material failure is suspected.

Stent patency of target vessels was 95.7% at 1 year and 88.6% at 4 years in this study. These figures are similar to target vessel patency rates according to the GLOBAL collaborators on Advanced Stent-graft Techniques for Aneurysm Repair (GLOBALSTAR) Registry (318 patients and 889 target vessels, of which 670 were stented), in which target vessel patency was 97% and 95% at 12 and 42 months, respectively.[5] The GLOBALSTAR Registry, however, did not describe stent patency separately. Therefore patency rates are difficult to compare. Additionally, the GLOBALSTAR figures may be influenced by different follow-up protocol. In our study, all patients received CTA follow-up, and all stents were measured separately, whereas the GLOBALSTAR registry did not differentiate between "target vessel patency" and "stent patency" and did not describe which cohort of patients were followed up with CTA. Therefore, the influence of the use of different definitions is unclear.

Visceral stent patency in our study was 95.7% at 1 year and 88.6% at 4 years. Bowel ischemia after mesenterial stent placement occurred in only two patients (8.6%). However, both patients died as a result of multiorgan



PTFE					
No at Risk	141	67	44	29	6
Survival		91.4	86.6	83.8	80.9
SE (%)		2.7	3.8	4.6	5.2
Bare Metal					
No at Risk	63	36	27	20	17
Survival		82.6	82.6	79.0	70.7
SE (%		4.8	4.8	5.8	7.6

Fig 4. Kaplan-Meier survival curve showing freedom from combined outcome of occlusion and stenosis in uncovered (bare metal stent [*BMS*]) and covered (polytetrafluoroethylene [*PTFE*]) stents in renal arteries. *SE*, Standard error.

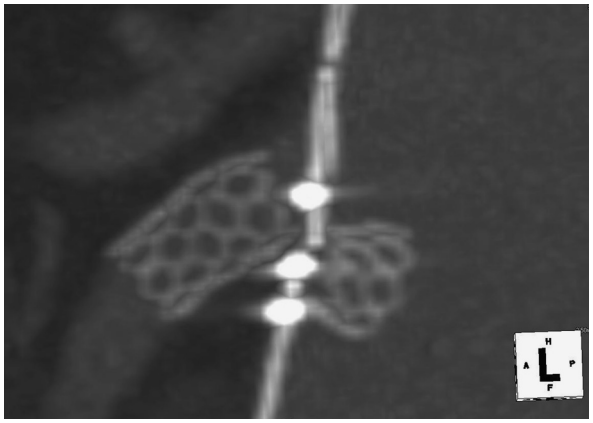


Fig 5. Fractured uncovered stent in superior mesenteric artery (SMA) of 72-year-old man. Since the patient had no abdominal complaints, no intervention was performed.

failure. Hypertension, smoking, and diabetes did not influence outcome of stents, neither did cardiopulmonary comorbidity. Stent patency was, however, affected by material failure (fracture). This underlines the importance of the materials used.

Mastracci et al described freedom from secondary intervention of 89% at 3 years after fenestrated or branched repair, and the authors underlined the importance of meticulous follow-up by imaging.[21]

Estimated patient survival rates are 89.2% at 1 year and 62.5% at 4 years. In the EVAR trials 1 and 2, 4-year all-cause mortality rates were 26% and 64%, respectively.[22,23] Although 30-day mortality rate in this study cohort is 1.3%, all-cause mortality rates at medium term emphasize the need for well-thought consideration for F-EVAR in medically compromised patients.

Natural renal artery motion during cardiac cycles can reach up to 3 mm both near and distant from the aorta. EVAR inhibits proximal renal artery motion, decreasing 31% in maximal movement, but does not influence distal renal artery motion.[24] However, renal stents used in F-EVAR reduce proximal motion to 25% of preoperative value and reduce distal renal artery motion by 20%.[25] Additional to these kinetic forces created by longitudinal movement of the endograft and side branches, forces (though modest) are created solely due to mismatch between stent graft and native anatomy.[26] These forces subject stents to repetitive stress and make them vulnerable to fracture. In our study, covered stents showed significantly less fractures

than uncovered stents ($P = .01$). Possibly, the polytetrafluoroethylene layers in the covered stents create high radial strength by connecting the stent struts, thereby providing strong fixation of the stent graft to the visceral arteries. Flexibility is not compromised, and therefore covered stents might be less prone to fatigue fracture.[27]

Transient renal function impairment was seen in 41.3% and persistent renal function impairment in 9.4%. In contrast to Haddad et al,[7] renal function impairment was not significantly associated with preprocedural renal dysfunction. In patients with renal artery stenosis or occlusion, renal function impairment did significantly occur more often ($P < .01$).

In total, 41 stenoses or occlusions occurred in 254 stents. In 22 of these cases, there appeared to be a kink in the stent (53.7%), and in five cases, the stent was fractured (12.2%). Since stenosis and occlusion are significantly associated with loss of renal function, it could be advised to perform a percutaneous transluminal angioplasty or new stent placement when a change in stent configuration occurs.

This study is limited by the fact that in the first years of F-EVAR, only uncovered stents were used, whereas the more expensive covered stents were used since 2005. Therefore, a learning curve could have influenced stent outcome. Loss of target vessel or stent patency was, however, equally distributed over time from the procedure, suggesting that loss of patency was not influenced by a learning curve. Since 2009, stents at risk for kinking (vessels under a sharp angle) were realigned with self-expanding nitinol stents to support a smoother passage from stent to vessel. In this F-EVAR series, only eight target vessels were treated this way. The effects of this realignment are therefore unknown. The method we used to determine the angulation of target vessels is not a validated test. However, with the use of three-dimensional reconstructions and measurements in the axis of the aorta and visceral arteries, we were able to present reliable measurements. Another limitation is the use of eGFR to address renal function. We do acknowledge that a 24-hour urine collection would have been more precise to address renal function. This has not been performed for logistic reasons.

In literature regarding F-EVAR, different definitions of "loss of patency," "stenosis," and "target vessel" are used, as well as different techniques to assess vessel or stent patency. Therefore it is difficult to compare results.

In our study, we did not measure migration. In theory, migration and therefore crushing of the stent between the fenestration and artery could be related to lower visceral stent patency. We did, however, perform a univariate analysis, which outlined that visceral stent failure did not influence other stent failure within the same patient. Therefore, we considered no influence of migration of endoprosthesis.

Finally, only 52% of the patients had a 1-year CT follow-up and only 11% had a 4-year CT follow-up, which influenced the results of this study.

Occlusions and stenoses of target vessels seem not to be influenced by comorbidity, target vessel angulation, clock position, or aneurysm sac size. However, we cannot rule out the effect of dislocation of the endoprosthesis or the influence of preprocedural miscalculation of the sizing and positioning of fenestrations.

CONCLUSIONS

Patency rates of visceral artery stents (−grafts) in fenestrated EVAR are 95.7% at 1 year and 88.6% at 4 years. Patency rates were affected by stent fractures, which occurred more in uncovered stents compared with covered stents. Renal artery stent stenosis occurred more in uncovered compared with covered stents. Renal dysfunction was significantly associated with renal stent occlusion or stenosis.

AUTHOR CONTRIBUTIONS

Conception and design: FG, CZ, MR, IT
Analysis and interpretation: FG, CZ, IT
Data collection: FG, EV, FB, IT
Writing the article: FG, IT
Critical revision of the article: FG, CZ, EV, FB, MR, IT
Final approval of the article: FG, CZ, EV, FB, MR, IT
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