One-year follow-up after active aortic aneurysm sac treatment with shape memory polymer devices during endovascular aneurysm repair

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ABSTRACT

Objective: To determine the safety and efficacy of treating abdominal aortic aneurysm (AAA) sacs with polyurethane shape memory polymer (SMP) devices during endovascular aneurysm repair (EVAR), using a technique to fully treat the target lumen after endograft placement (aortic flow volume minus the endograft volume). SMP devices self-expand in the sac to form a porous scaffold that supports thrombosis throughout its structure.

Methods: Two identical prospective, multicenter, single-arm studies were conducted in New Zealand and the Netherlands. The study population was adult candidates for elective EVAR of an infrarenal AAA (diameter of ≥55 mm in men and ≥50 mm in women). Key exclusion criteria were an inability to adequately seal a common iliac artery aneurysm, patent sac feeding vessels of >4 mm, and a target lumen volume of <20 mL or >135 mL. Target lumen volumes were estimated by subtracting endograft volumes from preprocedural imaging-based flow lumen volumes. SMP devices were delivered immediately after endograft deployment via a 6F sheath jailed in a bowed position in the sac. The primary efficacy end point was technical success, defined as filling the actual target lumen volume with fully expanded SMP at the completion of the procedure. Secondary efficacy outcome measures during follow-up were the change in sac volume and diameter, rate of type II endoleak and type I or III endoleaks, and the rate of open repair and related reinterventions, with data collection at 30 days, 6 months, and 1 year (to date). Baseline sac volumes and diameters for change in sac size analyses were determined from 30-day imaging studies. Baseline and follow-up volumes were normalized by subtraction of the endograft volume.

Results: Of 34 patients treated with SMP devices and followed per protocol, 33 patients were evaluable at 1 year. Preprocedural aneurysm volume was 181.4 mL (95% confidence interval [CI], 150.7-212.1 mL) and preprocedural aneurysm diameter was 60.8 mm (95% CI, 57.8-63.9 mm). The target lumen volume was 56.3 mL (95% CI, 46.9-65.8 mL). Technical success was 100% and the ratio of SMP fully expanded volume to estimated target lumen volume was 1.4 ± 0.3. Baseline normalized sac volume and diameter were 140.7 mL (95% CI, 126.6-154.9 mL) and 61.0 mm (95% CI, 59.7-62.3 mm). The adjusted mean percentage change in normalized volume at 1 year was −28.8% (95% CI, −35.3 to −22.3%; $P < .001$). The adjusted mean change in sac diameter at 1 year was −5.9 mm (95% CI, −7.5 to −4.4 mm; $P < .001$). At 1 year, 81.8% of patients (95% CI, 64.5%-93.0%) achieved a ≥10% decrease in normalized volume and 57.6% of patients (95% CI, 39.2%-74.5%) achieved a ≥5 mm decrease in diameter. No device- or study procedure-related major adverse events occurred through 1 year after the procedure.

Conclusions: Treatment of AAA sacs with SMP devices during EVAR resulted in significant sac volume and diameter regression at 1 year with an acceptable safety profile in this prospective study. (J Vasc Surg 2024;■:1-11.)

Keywords: Abdominal aortic aneurysm; Endovascular aneurysm repair; Aortic endograft; Aneurysm regression; Shape memory polymer

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Shape memory polymer (SMP) is a novel material designed to treat large vascular volumes with an intention to support thrombosis in the short term and support the conversion of thrombus to collagen over the long term. The polyurethane SMP in IMPEDE-FX RapidFill (Shape Memory Medical, Santa Clara, CA) is manufactured in an open-cell structure, that is then crimped for storage and catheter delivery (Fig 1). On deployment of SMP devices into the warm, aqueous environment of a vessel, the SMP remembers its manufactured shape and the material self-expands (~15×) to a porous structure that supports thrombus formation throughout its structure. The open-cell, porous structure of SMP also contributes to its radiolucency, low radial force, and conformability. These properties formed the basis of an investigation into the potential of SMP devices to support abdominal aortic aneurysm (AAA) sac regression, when used to treat the flow lumen volume outside an endograft during endovascular aneurysm repair (EVAR). AAA sac regression has been associated with improved mortality and morbidity compared with stable or expanding sacs post EVAR.[1-12] The decisive factors in sac regression after EVAR are mostly unknown to date, but type II endoleaks seem to play an important role. They have been linked to more AAA-related complications and reinterventions, but also unfavorable sac remodeling with more growth and less regression.[13] The preprocedural analysis and intraprocedural techniques developed during this investigation to fully treat AAA sacs with SMP devices have previously been described.[14] Here, 1-year outcomes are presented.

ARTICLE HIGHLIGHTS

- **Type of Research:** Multicenter prospective study
- **Key Findings:** At 1 year after treatment of the abdominal aortic aneurysm (AAA) sac with shape memory polymer devices during endovascular aneurysm repair, 81.8% of 33 patients achieved a ≥10% decrease in sac volume and 57.6% achieved a ≥5 mm decrease in sac diameter. No device- or study procedure-related major adverse events occurred through 1 year.
- **Take Home Message:** Treatment of the AAA sac with shape memory polymer devices during endovascular aneurysm repair is safe and resulted in significant AAA sac regression at 1 year.

METHODS

Study design. Two identical prospective, multicenter, single-arm studies in New Zealand (NCT04227054) and the Netherlands (NCT04751578) were undertaken and approved by Northern-A Health and Disability Ethics Committee (20/NTA/4) and Commissie Mensgebonden Onderzoek Regio Arnhem-Nijmegen (2021-7370). Participants were adult (≥18 years) candidates for elective EVAR of an infrarenal aortic aneurysm (≥55 mm in men and ≥50 mm in women[15,16]). Key exclusion criteria were an aortoiliac aneurysm or concomitant iliac artery ectasia or aneurysm close to the aortic bifurcation that could not be adequately sealed in the proximal common iliac artery, patent feeding vessels >4 mm in diameter, <20 mL or >135 mL target lumen volume, and off-label endograft use (Supplementary Table I, online only provides a complete list, including allowed endografts, which were selected by the treating investigator on a case-by-case basis). The 135-mL upper limit was imposed as a safety factor based on available data from the manufacturer at the time of study initiation, although this parameter is subject to change based on ongoing device development. Patients gave written informed consent before study procedures.

The primary efficacy end point of the studies was technical success, defined as filling the actual target lumen volume with expanded SMP material. The primary safety end point was the incidence of device/study procedure-related major adverse events (MAEs) at 30 days. MAEs were defined as all-cause mortality, bowel ischemia, myocardial infarction, paraplegia, renal failure, respiratory failure, stroke, or procedural blood loss of >1000 mL. Relatedness to the study device/procedure was adjudicated by a medical monitor (vascular surgeon, P.D.H.) independent from the study investigators. Primary end points have previously been reported, in which all preprocedural aneurysm size data and endograft sizing were determined by the treating investigators.[14] Secondary efficacy outcome measures during follow-up were the change in sac volume and diameter, rate of type II endoleak and type I or III endoleaks, and the rate of open repair and related reinterventions, with data collection at 30 days, 6 months, and 1 year (to date). Secondary safety outcome measures were device- or study procedure-related MAE and serious adverse event (SAE) rates over time.

Enrolled patients with no devices implanted were followed for 30 days for safety and then terminated from the study. Patients implanted with the study device were evaluated per protocol and will be followed for 5 years. One-year outcomes were determined for this report; data are subject to change based on ongoing data monitoring.

Study device. The porous SMP in each IMPEDE-FX RapidFill expands to occupy up to 6.25 mL (Fig 1). Individual SMP devices contain a radiopaque marker. A 6F flexible sheath (0.070- to 0.090-inch inner diameter) minimizes the likelihood of friction during delivery (SMP devices start to self-expand on contact with blood). The SMP devices are pushable and 0.035- to 0.038-inch guidewires minimize the likelihood of wedging the guidewire between the devices and the delivery sheath.

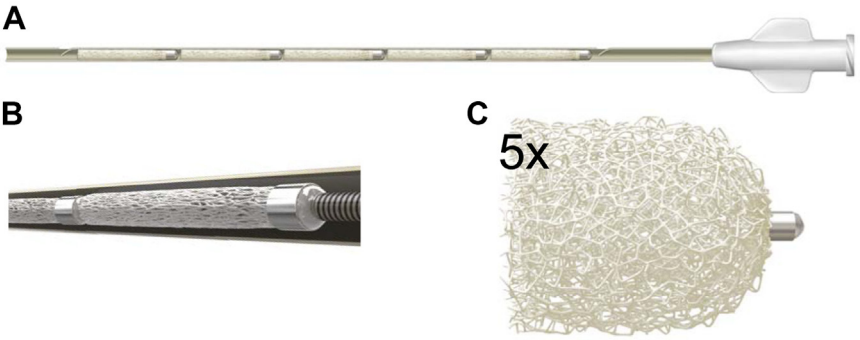

Fig 1. The shape memory polymer (SMP) study device. **(A)** IMPEDE-FX RapidFill with five SMP devices in a crimped form for catheter delivery. **(B)** The SMP devices are pushable. **(C)** The porous SMP in each IMPEDE-FX RapidFill (containing 5 SMP devices) self-expands to occupy up to a total of 6.25 mL for the five devices (1.25 mL each). The individual SMP devices each contain a radiopaque marker.

Procedural planning. Computed tomography angiography (CTA) ≤3 months before the study procedure and acquired per local standard of care imaging protocols was used to determine eligibility and estimate the target lumen volume for treatment. The estimated target lumen volume (flow lumen volume minus the endograft volume [derived from dimensions in instructions for use]) was used to determine the number of IMPEDE-FX RapidFill devices required, as detailed previously.[14]

Intervention. The procedure has been described previously, including a supplemental video with intraprocedural fluoroscopy imaging to illustrate different parts of the procedure.[14] In brief, ipsilateral or contralateral approaches were used; contralateral delivery of the SMP required upsizing the introducer by 2F. Ipsilateral delivery was possible if the ipsilateral endograft limb landed above the aortic bifurcation, and did not require access upsizing.

After deployment of the endograft main body, a hydrophilic guidewire and catheter were positioned around the outer circumference of the aneurysm sac blood lumen, and then replaced by a medium support J-tip 0.035-inch guidewire. The limb was deployed parallel to the guidewire, which was consequently jailed between the endograft and the artery wall. Compliant balloon molding of the endograft except the distal seal zone containing the jailed guidewire was performed.

A compatible flexible 6F delivery sheath was advanced over the jailed guidewire, around the circumference of the blood lumen, to the first caudal quadrant. Approximately 25% of the estimated volume of SMP devices was then slowly distributed into the quadrant, with a 5-minute pause for SMP expansion before moving to the next quadrant. The sheath was retracted circumferentially to deliver the SMP into each of the cephalad quadrants, and finally into the remaining caudal quadrant of the sac. Repeated manual contrast injections (sacograms) monitored treatment progress and confirmed comprehensive sac treatment at case completion.[14] After sheath removal, balloon molding sealed the working endograft limb.

Follow-up imaging schedule and imaging study evaluation. Patients returned for CTA acquired per local standard of care imaging protocols within 30 days, which was the source of baseline data for sac size analyses. Patients underwent follow-up CTA studies at 6 months and 1 year. After enrollment and study treatments were complete, preprocedural and follow-up CTA studies were evaluated by a core laboratory independent from the investigators (Cleveland Clinic Vascular Core Lab, Cleveland, OH). Reference points of the proximal and distal aspects of the sac were used for consistent volume analysis (TeraRecon, Fremont, CA). Sac centerline diameters were determined, corresponding with the widest point of the sac. Preprocedural data determined by the core laboratory were AAA diameter and volume, thrombus burden, flow lumen volume, and the nature of aneurysm sac feeding vessels. Baseline and follow-up data determined by the core laboratory were AAA diameter and volume, endograft volume (in situ), and the presence and characterization of endoleak.

Type II endoleaks were categorized according to an endoleak volume of ≥2.4 mL and <2.4 mL based on a cutoff derived from a published analysis of the need for reinterventions in patients with type II endoleaks after EVAR.[17]

Statistical analyses. The protocols and data acquisition in the two studies were identical; therefore, data were combined for analysis. Baseline and follow-up volumes were normalized by subtraction of the endograft volume (ie, subtraction of the endograft volume from the aneurysm volume) to accommodate differences in endograft volumes and the extent of endograft expansion. Volume and diameter-based aneurysm size change data were reviewed graphically for normality and considered appropriate for parametric testing; means and 95% confidence intervals (CIs) are presented, except where

Table I. Baseline demographics and medical history (n = 34)

Characteristic	Value	
Age, years	75.5	±7.3
Male sex	29	(85.3)
Body mass index, kg/m^2	29.6	±5.6
Hypertension	26	(75.6)
Hyperlipidemia	22	(64.7)
Diabetes	9	(26.5)
Coronary heart disease	10	(29.4)
Atrial fibrillation, rate controlled[a]	2	(5.9)
Angina	4	(11.8)
Myocardial infarction	7	(20.6)
Peripheral vascular disease	16	(47.1)
Chronic obstructive pulmonary disease	4	(11.8)
Renal insufficiency	5	(14.7)
Smoking (current or former)	29	(85.3)
Cerebrovascular accident	4	(11.8)
Transient ischemic attack	1	(2.9)
Cancer	19	(55.9)
ASA grade[b]		
I	0	(0.0)
II	6	(17.6)
III	27	(79.4)
IV	1	(2.9)

ASA, American Society of Anesthesiologists.
Continuous variables are mean ± standard deviation unless stated otherwise. Categorical variables are quoted as count (percentage).
[a]Patients with atrial fibrillation that was not well rate controlled were excluded from the study.
[b]American Society of Anesthesiologists physical status classification system.

noted. Longitudinal analyses were performed using a repeated measures analysis of variance model with time and the baseline end point value as factors in the model; adjusted (least squares) means and 95% CIs are reported. Change in volume data are reported as both the percentage (relative) change from baseline and the absolute change from baseline. Change in diameter data are reported as the absolute change from baseline. Subgroup analyses were performed including the extent of preexisting thrombus on the preprocedural CTA (<50% and ≥50%) as a factor in the repeated measures analysis of variance model. A negative change in sac volume and/or diameter represents sac regression, a positive change represents sac growth.

Change in volume data were categorized as a ≥10% decrease, a <10% decrease to a <10% increase (no change), and a ≥10% increase. Change in diameter data were categorized as a ≥5 mm decrease, a <5 mm decrease to a <5 mm increase (no change), and a ≥5 mm increase.[15,18] Categorical variable 95% CIs were generated using the exact method.

For analyses of groups with different type II endoleak status at 1 year (≥2.4 mL, none, and <2.4 mL), mean and 95% CI or range are reported, as specified with each result; *P* values comparing 1-year and baseline within group were generated by paired *t* test.

SAS V9.4 (SAS Institute, Care, NC) was used for statistical analyses other than descriptive statistics, which were generated in Microsoft Excel. A *P* value of ≤.05 was considered significant.

RESULTS

Study population. Thirty-five consecutive patients were enrolled and treated with the study device September 2020 through August 2022 (enrollment was disrupted by the coronavirus disease 2019 pandemic).[14] Two additional enrolled patients failed intraprocedural eligibility criteria as detailed elsewhere in this article. After study treatment, one patient was discovered to have an inflammatory aneurysm and followed separately. Therefore, 34 patients were followed per protocol for efficacy, of whom 33 reached the 6-month and 1-year follow-ups. The remaining patient died from coronavirus disease 2019 just before the 6-month visit. The study population was typical of elective EVAR patients based on demographics and medical history (Table I).

Preprocedural and intraprocedural data. Preprocedural aneurysm characteristics are shown in Table II. Although the eligibility criteria dictated exclusion of patients with patent feeding vessels of >4 mm in diameter, patients with smaller feeding vessels were included and 23 of 34 (67.6%) had >1 patent feeding vessels in addition to a patent inferior mesenteric artery (IMA), including 10 of 34 patients (29.4%) with a patent IMA of ≥3 mm in diameter (Table II).

Endografts were selected based on investigator preference and the approach was dictated by the nature of the endograft (Table II). The technical success of this analysis population was 100% based on investigator estimates of target lumen volumes, with a mean ± standard deviation SMP volume (expanded)/target lumen volume estimate ratio of 1.4 ± 0.3 (Table II). The mean ± standard deviation additional procedure time for SMP treatment to deliver and expand the material and to perform sacograms was 27 ± 14 minutes. Additional radiation time was 135 seconds (n = 6).

One-year efficacy outcomes. There was a significant decrease in normalized sac volume at 6 months and 1 year relative to baseline, both in terms of relative and absolute change (Table III and Table IV). Furthermore, there was a significant decrease in normalized sac volume between 6 months and 1 year. The proportion of patients with a ≥10% decrease, no change, and a ≥10% increase in normalized volume at 6 months was 60.6%

Table II. Preoperative aneurysm characteristics, endograft details, and study devices (n = 34)

Characteristic[a]		Value
Aneurysm diameter, mm	60.8	(57.8-63.9)
Aneurysm volume, mL	181.4	(150.7-212.1)
Aneurysm flow lumen volume, mL	98.2	(88.4-108.1)
Thrombus volume, mL	83.2	(56.3-110.1)
Thrombus volume,[b] %	41.7	(35.5-47.8)
≥50% thrombus burden	11.0	(32.0)
Patent feeding vessels		
IMA ≥3 mm	10	(29.4)
Accessory renal artery[c]	5	(14.3)
Lumbar arteries[c]	31	(91.2)
Risk of type II endoleak[d]		
High	10	(29.4)
Medium	13	(38.2)
Low	11	(32.4)
Endografts		
Medtronic Endurant II/IIs	17	(50.0)
Gore Excluder	12	(35.3)
Gore Excluder Conformable	5	(14.7)
Target lumen volume,[e] mL	56.3	(46.9-65.8)
Approach		
Contralateral	14	(41.2)
Ipsilateral	20	(58.8)
SMP devices[f]	11	(7-15)
SMP volume/target lumen volume estimate ratio[g]	1.4	±0.3

IMA, Inferior mesenteric artery; *SMP*, shape memory polymer.
Data presented as mean ± standard deviation (95% confidence interval) for continuous variables unless stated otherwise or number (%) for categorical variables.
[a]Aneurysm diameter, volume, flow lumen volume, thrombus volume and burden, and patent feeding vessels determined by a core laboratory.
[b]Thrombus volume/aneurysm volume.
[c]One or more.
[d]High risk was defined as a patent IMA ≥3 mm in diameter, regardless of other feeding vessels. Medium risk was defined as ≥3 patent accessory renal or lumbar arteries. Low risk was defined as <3 patent accessory renal or lumbar arteries. Inferior mesenteric arteries in medium and low risk were <3 mm (patent), nonpatent (any diameter), or absent (not detected). Patients with patent AAA sac feeding vessels (within the sac) >4 mm in diameter (determined by the investigator) were excluded from the study.
[e]Determined by the treating investigator. Target lumen volumes were estimated from measurements determined using EndoSize software, as described previously.[11].
[f]The number of IMPEDE-FX RapidFill devices implanted (occupies up to 6.25 mL when the SMP expanded fully), quoted as median (interquartile range).
[g]Maximum volume occupied by the implanted fully expanded SMP/estimated target lumen volume from preprocedural imaging analysis by the treating investigator, quoted as mean ± standard deviation.

(95% CI, 42.1%-77.1%), 36.4% (95% CI, 20.4%-54.9%), and 3.0% (95% CI, 0.1%-15.8%), respectively (Table III). At 1 year, the proportion of patients with a ≥10% decrease in normalized volume had increased to 81.8% (95% CI, 64.5%-93.0%) along with 15.2% (95% CI, 5.1%-31.9%), with no change in volume and 3.0% (95% CI, 0.1%-15.8%) with a ≥10% increase in volume (Table III).

There was a significant decrease in sac diameter at 6 months and 1 year relative to baseline (Table III and Table IV). Furthermore, there was a significant decrease in sac diameter between 6 months and 1 year. The proportion of patients with a ≥5 mm decrease, no change, and a ≥5 mm increase in diameter at 6 months was 39.4% (95% CI, 22.9%-57.9%), 60.6% (95% CI, 42.1%-77.1%), and 0%, respectively (Table III). At 1 year, the proportion of patients with a ≥5 mm decrease in diameter had increased to 57.6% (95% CI, 39.2%-74.5%) with a consequent decrease in the number of patients with no change in diameter to 42.4% (95% CI, 25.5%-60.8%); no patients had diameter-based growth (Table III).

There were no differences in volume- or diameter-based treatment effects in patients with a <50% or ≥50% preprocedural thrombus ($P = .931$ for percentage change and $P = .772$ for absolute change in normalized volume, and $P = .599$ for diameter).

Fig 2 and Supplementary Fig 1 (online only) show examples of patients with a ≥10% decrease in normalized volume and a ≥5 mm decrease in diameter at 6 months and even further improvement at 1 year. Supplementary Fig 2 (online only) shows an example of a patient with a ≥10% decrease in normalized volume at both 6 months and 1 year, but with no change in diameter at either timepoint.

At 30 days, 15 of 34 patients (44.1%) had a type II endoleak (3 ≥2.4 mL, 12 <2.4 mL), of which 7 (all <2.4 mL) resolved spontaneously over the course of 1 year. One type II endoleak (<2.4 mL) occurred at 6 months. Overall, at 1 year, 9 of 33 patients (27.3%) had a type II endoleak, of which 3 (9.1% overall) were ≥2.4 mL in volume and 6 (18.2% overall) were <2.4 mL in volume. One patient with a type II endoleak <2.4 mL in volume also had a type I endoleak, which is under review by the treating investigator and not intervened to date.

The three type II endoleaks ≥2.4 mL in volume were 12.0 mL (range, 7.5-18.3 mL). The baseline normalized sac volume was 125.1 mL (range, 97.3-146.3 mL); the percentage change in normalized volume from baseline to 1 year was 8.9% (range, 3.4%-18.7%). Categorically, two of the three patients had no change and one of the three had a ≥10% increase in normalized volume. The baseline sac diameter was 60.2 mm (range, 55.1-64.4 mm); the change in diameter was 2.1 mm (range, 0.8-3.0 mm). Categorically, all three patients had no change in diameter at 1 year. Notably, all three patients had a patent IMA ≥3 mm in diameter and two to four patent lumbar arteries on preprocedural imaging.

The six type II endoleaks <2.4 mL in volume were 0.5 mL (95% CI, 0.04-0.9 mL). At 1 year, there was a significant decrease in normalized sac volume over baseline in both patients without any endoleak (n = 24) and those with a type II endoleak <2.4 mL. For patients without

Table III. Aneurysm size outcomes (n = 33)

Parameter	Baseline[a]		6 Months		1 Year	
Relative volume, %[b]	-	-	82.1	(76.1 to 88.1)	71.2	(63.9 to 78.5)
Volume, mL	140.7	(126.6 to 154.9)	115.4	(100.6 to 130.2)	98.6	(86.4 to 110.9)
Aneurysm diameter, mm	61.0	(59.7 to 62.3)	55.6	(51.7 to 59.4)	53.4	(49.5 to 57.4)
Change in volume[b,c]						
≥10% decrease	-	-	20	(60.6)	27	(81.8)
<10% decrease to <10% increase[d]	-	-	12	(36.4)	5	(15.2)
≥10% increase	-	-	1	(3.0)	1	(3.0)
Change in diameter[c]						
≥5 mm decrease	-	-	13	(39.4)	19	(57.6)
<5 mm decrease to <5 mm increase[d]	-	-	20	(60.6)	14	(42.4)
≥5 mm increase	-	-	0	(0.0)	0	(0.0)

Data presented as mean (95% confidence interval) for continuous variables or number (percentage) for categorical variables; 95% confidence intervals for categorical variables are listed in the text. All volume data were normalized by subtraction of the endograft volume.
[a]Baseline values were determined based on the 30-day imaging study.
[b]Follow-up aneurysm volume as a percentage of baseline volume.
[c]Change from baseline.
[d]Also described in the text as no change.

endoleak, the baseline normalized volume was 147.7 mL (95% CI, 129.1-166.3 mL); the percentage change in normalized volume from baseline to 1 year was −36.0% (95% CI, −42.8 to −29.2%; $P < .001$). For patients with a type II endoleak <2.4 mL, the baseline normalized volume was 120.4 mL (95% CI, 108.5-132.3 mL); the percentage change in normalized volume from baseline to 1 year was −18.9% (95% CI, −35.5 to −2.3%; $P = .033$). Categorically, 23 of 24 patients (95.8%) without endoleak at 1 year had a ≥10% decrease and 1 of the 24 (4.2%) had no change in normalized volume; 4 of the 6 patients (66.7%) with type II endoleak <2.4 mL at 1 year had a ≥10% decrease and two (33.3%) had no change in normalized volume. For patients without endoleak, the baseline diameter was 60.6 mm (95% CI, 59.1-62.2 mm); the change in diameter was −7.4 mm (95% CI, −9.2 to −5.5 mm; $P < .001$). For patients with a type II endoleak <2.4 mL, the baseline diameter was 62.8 mm (95% CI, 59.3-66.2 mm); the change in diameter was −4.3 mm (95% CI, −8.7 to −0.2 mm; $P = .057$). Categorically, 16 of 24 patients (66.7%) without endoleak at 1 year had a ≥5 mm decrease and 8 of the 24 (33.3%) had no change in diameter; 3 of 6 patients (50.0%) with type II endoleak <2.4 mL at 1 year had a ≥5 mm decrease and 3 of the 6 (50.0%) had no change in diameter.

One-year safety outcomes. There were no device- or study procedure-related MAEs reported through 1 year. The monitored perioperative (30-day) safety profile included a total of four device- or study procedure-related SAEs reported in four patients in the per-protocol population (Supplementary Table II, online only). Two periprocedural device-related SAEs occurred in the patient with an inflammatory aneurysm being followed separately (Supplementary Table II, online only). From 30 days through 1 year, no additional device- or study procedure-related SAEs were reported. There were no conversions to open repair and no device- or study procedure-related reinterventions through 1 year.

On core laboratory review of 30-day CTA, a few SMP devices were noted outside of the aneurysm wall in one patient. The patient was asymptomatic and the sac perforation was not noted on study procedure completion angiography. At 1 year, the patient had a substantial decrease in sac size from baseline: a −52.5% change in normalized volume and a −14.1 mm change in diameter. No endoleaks were identified in this patient. Although this event was notable, it did not meet the criteria for an SAE because no symptoms occurred and no intervention was necessary. On case review, it was designated an iatrogenic perforation of the aneurysm sac that likely occurred during wire and delivery sheath advancement and not a consequence of SMP device expansion.

It was not possible to place a delivery sheath in two patients. The learnings from these cases were (i) upsizing the access sheath may be necessary if an endograft limb diameter prevents delivery sheath access to the sac, and (ii) upsizing the introducer for contralateral delivery of the SMP facilitates access, which was integrated into the procedure description.

DISCUSSION

In this prospective study, techniques and procedures were developed for treating the flow lumen outside of an endograft during EVAR with SMP devices. Treatment resulted in significant sac regression at 6 months in terms of both volume and diameter, and further improvement at 1 year, evident in both the continuous variables and in the percentage of patients achieving

Table IV. Change in aneurysm size parameters (n = 33)

Parameter	Value		*P* value
Relative volume, %[a]			
Baseline to 6 months	−17.9	(−24.4 to −11.3)	<.001
Baseline to 1 year	−28.8	(−35.3 to −22.3)	<.001
6 months to 1 year	−10.9	(−20.2 to −1.7)	.022
Volume, mL			
Baseline to 6 months	−25.3	(−34.8 to −15.8)	<.001
Baseline to 1 year	−42.1	(−51.6 to −32.6)	<.001
6 months to 1 year	−16.8	(−30.2 to −3.3)	.015
Diameter, mm			
Baseline to 6 months	−3.8	(−5.3 to −2.2)	<.001
Baseline to 1 year	−5.9	(−7.5 to −4.4)	<.001
6 months to 1 year	−2.2	(−4.4 to −.02)	.048

Data from a repeated measures analysis of variance (RMANOVA) model presented as adjusted mean (95% confidence interval). All volume data were normalized by subtraction of the endograft volume.
[a]Follow-up aneurysm volume as a percentage of baseline volume.

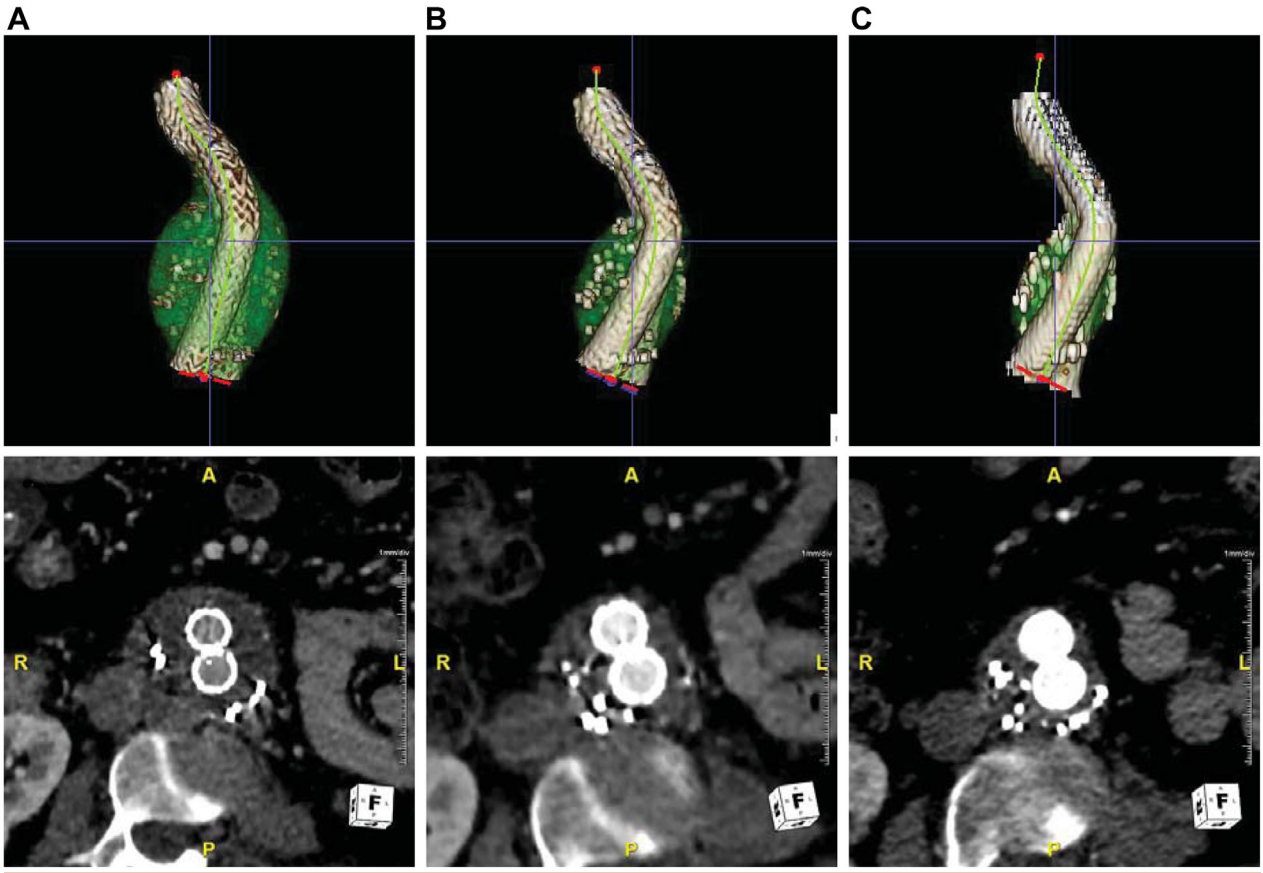

Fig 2. Patient with ≥10% decrease in normalized volume and ≥5 mm decrease in diameter at 6 months and even further improvement at 1 year. The preprocedural target lumen volume estimate was 92.5 mL. The shape memory polymer (SMP) volume/target lumen volume estimate ratio was 1.1. **(A)** The baseline normalized volume was 121.9 mL and the diameter was 58.7 mm. **(B)** At 6 months, the patient showed a −48.2% change in normalized volume and a −11.9 mm change in diameter from baseline. **(C)** At 1 year, the patient showed a −63.7% change in normalized volume and a −14.1 mm change in diameter from baseline, resulting in a normalized volume of 44.2 mL and a diameter of 44.6 mm. No endoleaks were observed in this patient at 6 months and 1 year.

clinically relevant cutoffs defining sac regression. One-year categorical diameter-based regression in this study (57.6%) was numerically greater than the 1-year results observed in large EVAR registries (40% Vascular Quality Initiative,[7] 41% ENGAGE[19]); importantly, no diameter-based growth was observed in this study (25% Vascular Quality Initiative,[7] 4% ENGAGE[19]).

In this study to establish a preliminary understanding of the SMP device treatment effect after fully treating the sac, patients with AAA sac feeding vessels >4 mm in diameter were excluded to limit the scope of the study in the initial stages of technique development. However, 29.4% of the treated patients still had a patent IMA ≥3 mm in diameter, which is frequently considered the primary criterion for patients at high risk of developing type II endoleak.[20-23] Of course, future studies should include analysis of patients with feeding vessels >4 mm in diameter, either by appropriately powered subgroup analyses or in a dedicated study; these patients may also benefit from the approach. It is also possible that pre-embolization of large feeding vessels in combination with SMP treatment may result in better outcomes in certain patient populations; however, this practice should be established in appropriately designed studies.

Although the type II endoleak rate of 27.3% at 1 year is not significantly different from historical rates for EVAR, the use of a core laboratory to analyze follow-up imaging resulted in detailed assessment of type II endoleaks. Type II endoleaks were categorized into an endoleak volume of ≥2.4 mL and <2.4 mL, based on a cutoff derived from an analysis of the need for reinterventions in patients with larger type II endoleaks after EVAR.[17] The mean volume of the type II endoleaks <2.4 mL observed in this study was very small (0.5 mL) and, importantly, significant volume-based sac regression was observed at 1 year, even in the presence of these small endoleaks. The occurrence of type II endoleaks after active sac treatment may be explained by the fact that only the flow lumen can be filled and a persistent gutter may persist from the orifice of the side branches, through the mural thrombus, to the flow lumen. It is also notable that no diameter-based growth occurred in any patient, even those with endoleaks ≥2.4 mL. This finding is consistent with feasibility study results, which also showed significant sac regression in the presence of endoleaks.[24] For comparison, 4.8% of patients with type II endoleak and 2.4% of patients with no endoleak exhibited diameter-based sac growth at 1 year in a contemporary EVAR registry.[13] In future studies, it may be possible to further optimize the SMP treatment procedure to further minimize the potential of even small type II endoleaks and thereby maximize the extent of sac regression. Furthermore, comparative studies in which both arms use core laboratory endoleak analyses are needed to firmly establish the differences between EVAR alone and sac treatment with SMP devices.

Other single-arm studies on AAA sac embolization during EVAR using devices such as coils, fibrin glue, gelfoam, and gelatin sponge have generally also shown preemptive sac treatment results lower endoleak rates (than literature precedents) and either an absence of sac growth or sac regression based on diameter.[25-29] In a retrospective comparative study, Dosluoglu et al[21] reported that perigraft nonselective coil embolization of the sac resulted in a significantly smaller type II endoleak rate and greater diameter-based sac regression than EVAR alone in patients at high risk of developing a type II endoleak. In another retrospective comparative study, Mascoli et al[30] reported a significant decrease in the type II endoleak rate after nonselective coil embolization, but no difference in diameter-based sac regression. In randomized controlled trials (RCTs) in patients at high risk of developing type II endoleak, significantly smaller type II endoleak rates and greater volume-based sac regression were reported in the investigational arms at 1 year; patients in the investigational arms were treated with coils and fibrin glue (Piazza et al) or coils (Fabre et al) and the control arms were EVAR alone.[20,23] In the investigational arm, Piazza et al reported −7.5% (95% CI, −10.5 to −4.5%), −14.2% (95% CI, −18.9 to −9.5%), and −21.6% (95% CI, −26.5 to −16.7%) change in volume relative to preprocedural values at 6 months, 1 year, and 2 years, respectively. In Piazza et al's control arm, the change in volumes were −1.7% (95% CI, −6.0 to 2.6%), −2.1% (95% CI, −9.7 to 5.5%), and −2.9% (95% CI, −9.0 to 3.2%) at 6 months, 1 year, and 2 years, respectively. In our study, the change in normalized volume relative to baseline was −17.9% (95% CI, −24.4 to −11.3%) and −28.8% (95% CI, −35.3 to −22.3%) at 6 months and 1 year, respectively, suggesting that SMP device treatment may result in more rapid regression than coils or fibrin glue.

We reported results based on volume in addition to diameter as the procedure was developed to comprehensively treat the sac with expanded SMP and therefore, analysis of the entire sac was an intuitive choice to assess the treatment effect. The importance of volume as an outcome measure is also gaining traction in the literature, both as a AAA surveillance tool[31-33] and in evaluating outcomes after EVAR.[34-37] Imaging technology development is also advancing to maximize the potential of volume measurement, both in CT[38,39] and three-dimensional ultrasound[40-43] assessments. The notion that volume may be a more sensitive and/or leading indicator of sac volume changes is supported by the results of this study, in which the proportion of patients with a ≥10% decrease in normalized volume was notably larger than the number of patient with a ≥5 mm decrease in diameter at both 6 months and 1 year. The single patient with a ≥10% increase in normalized volume at both 6 months and 1 year was also exhibiting no change in diameter at either timepoint. Although a volume-based

standard to describe sac regression/growth is arguably not yet established, a ≥10% change in volume has been used in a number of studies.[2,34,35,44]

Although the clinical outcomes in the presence of sac regression as compared with stable or expanding sacs is a contemporary subject of discussion in the field of EVAR, observations supporting the current conversation date back to at least 2000, including that the extent of sac regression is associated with improved survival, reduced rate of secondary interventions, and EVAR-related complications.[1-12] Furthermore, the 2023 initiation of the large, randomized ADVANCE trial (NCT05378347) to evaluate sac regression outcomes of the Medtronic (Dublin, Ireland) Endurant II/IIs Stent Graft System and Gore (Flagstaff, AZ) Excluder/Excluder Conformable AAA Endoprosthesis in standard EVAR subjects highlights the current focus on sac regression in the EVAR field in general.

Cost analyses of AAA sac treatment should include consideration of all aspects of postoperative care, including reinterventions. Our study was not designed to perform cost analysis. However, Piazza et al[20] noted the additional cost of sac embolization more than offset the cost of the greater number of reinterventions in the arm without sac embolization in their RCT. Similarly, Fabre et al[23] noted the cost of sac embolization should balance against the cost of follow-up and secondary interventions associated with aneurysm enlargement.

Limitations of this study were its small size and lack of a control arm. Subgroup analyses based on preexisting thrombus and type II endoleaks were exploratory and limited by small sample size in each group. In the EVAR field in general, sac volume measurement methods need to be standardized in prospective trials to facilitate comparison among studies; however, the inclusion of diameter data offers a consistent and comparable measure of study outcomes. The analysis of the type II endoleaks based on volume categories (≥2.4 mL and <2.4 mL) was based on observations in a single report and the analysis of sac regression in the context of endoleak volume may be considered a novel approach that requires further investigation in prospective trials.

As the first prospective study designed to develop the procedure for sac treatment with SMP devices and determine the effect of our approach in terms of sac regression, a combination of target flow lumen volume estimates determined before the procedure and intraprocedural contrast injections (sacograms) were used to monitor device distribution and sac treatment. Although the addition of lateral and oblique fluoroscopic views, cone-beam CT angiography, and contrast-enhanced ultrasound examination were considered as potential methods of monitoring device distribution during the procedure, additional monitoring methods add complexity to the procedure and may also increase time and radiation dose; our practical experience in the initial cases led us to believe that the technique developed and described and the extent and distribution of sac filling was sufficient and balanced risk and benefit in the first study of its kind. Of course, this experience should not deter future studies from considering and developing additional intraprocedural imaging and endovascular techniques to potentially improve the procedure.

Technical success at the completion of a procedure is a common primary efficacy end point in early stage device studies, particularly when a technique is being developed for the first time. With an initial understanding of the treatment effect of the technique in hand, it is likely that future studies on this approach will include sac size measurements in the primary efficacy end point. The list of MAEs in the primary safety end point of this study is commonly used in endograft studies; in the absence of additional information, it was also used in this prospective study. The observation of sac perforation during the follow-up in this study suggests sac rupture should be considered for inclusion in the primary safety end point of future studies evaluating this approach.

Additional considerations for future study design based on experiences in this study include CTA studies in this study were performed per site standard of care imaging protocols; future studies may consider imaging acquisition parameter standardization, within the limits of equipment and software differences between sites. Future studies should also include comprehensive analyses of radiation exposure and contrast use, where the most appropriate study design is likely an RCT to avoid potential inconsistencies on defining the start of the sac treatment part of the procedure. Future studies may include three-dimensional ultrasound assessments for comparison with CTA assessments.

CONCLUSIONS

Treatment of AAA sacs with SMP devices during EVAR resulted in significant sac volume and diameter regression at 1 year with an acceptable safety profile in this prospective study. Comparative studies (including an RCT) are needed to validate the treatment effect and larger studies are required to determine the effectiveness of the approach and optimize the definition of appropriate patient selection.

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AUTHOR CONTRIBUTIONS

Conception and design: AH, MMPJR
Analysis and interpretation: PDH
Data collection: AH, AAH, MK, JMMH, AMW, MMPJR
Writing the article: AH, MMPJR
Critical revision of the article: AH, AAH, MK, JMMH, AMW, PDH, MMPJR
Final approval of the article: AH, AAH, MK, JMMH, AMW, PDH, MMPJR
Statistical analysis: Not applicable
Obtained funding: Not applicable
Overall responsibility: AH

DISCLOSURES

A.H. and M.M.P.J.R. are consultants for Shape Memory Medical. P.D.H. was reimbursed for his role as Medical Monitor during the studies by Shape Memory Medical.

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Additional material for this article may be found online at www.jvascsurg.org.

Supplementary Table I (online only). Eligibility criteria

Study inclusion criteria
1. ≥18 years of age.
2. A candidate for elective EVAR of an infrarenal aortic aneurysm ≥5.5 cm in diameter in men and ≥5.0 cm in women.
Study exclusion criteria
1. An inability to provide informed consent.
2. Enrolled in another clinical study.
3. Aortoiliac aneurysm, or concomitant iliac artery ectasia or aneurysm (common iliac artery diameter >24 mm) close to the bifurcation and/or that cannot be adequately sealed.
4. Patent AAA sac feeding vessels (within the sac) >4 mm in diameter.
5. Volume of AAA sac to be filled after stent graft placement <20 mL or >135 mL, based on pre-procedure CTA (ie, aortic flow volume exclusive of stent graft volume).
6. Use of aortic stent grafts other than the Gore Excluder AAA Endoprosthesis, Cook Zenith Flex AAA Endovascular Graft, Medtronic Endurant II Stent Graft, or Endologix Ovation Alto Abdominal Stent Graft System to treat the AAA.
7. Planned use of the chosen stent graft outside its IFU.
8. Planned use of fenestrated or chimney stent grafts.
9. Study participants in which stent graft placement is abandoned for any reason, and/or in which the investigator decides, during the course of the stent graft placement, that the study procedure may not be appropriate.
10. Planned use of embolic devices other than the investigational product to embolize the AAA sac.
11. Vascular disease and/or anatomy that preclude the safe access and positioning of a catheter to deliver the investigational product into the AAA sac.
12. Ruptured, leaking, or mycotic (infected) aneurysm.
13. Aneurysmal disease of the descending thoracic aorta.
14. Coagulopathy or uncontrolled bleeding disorder.
15. Long-term (>6 months before the procedure) use of direct oral anticoagulant or any vitamin K antagonist anticoagulant use.
16. Serum creatinine level >2.5 mg/dL.
17. Cerebrovascular accident within 3 months before the procedure.
18. Myocardial infarction and/or major heart surgery within 3 months before the procedure.
19. Atrial fibrillation that is not well rate controlled.
20. Unable or unwilling to comply with study follow-up requirements.
21. Life expectancy of <2 years postprocedure.
22. Known hypersensitivity or contraindication to platinum, iridium, or polyurethane.
23. A condition that inhibits radiographic visualization during the implantation procedure.
24. History of allergy to contrast medium that cannot be managed medically.
25. Uncontrolled co-morbid medical condition, including mental health issues, that would adversely affect participation in the study.
26. Pregnant or a lactating female. For females of child-bearing potential, based on a positive pregnancy test within 7 days before the procedure or refusal to use a medically accepted method of birth control for the duration of the study.
27. New Zealand: Prisoner or member of other vulnerable population. The Netherlands: Member of a vulnerable population.

AAA, Abdominal aortic aneurysm; *CTA*, computed tomography angiography; *EVAR*, endovascular aneurysm repair; *IFU*, instructions for use.

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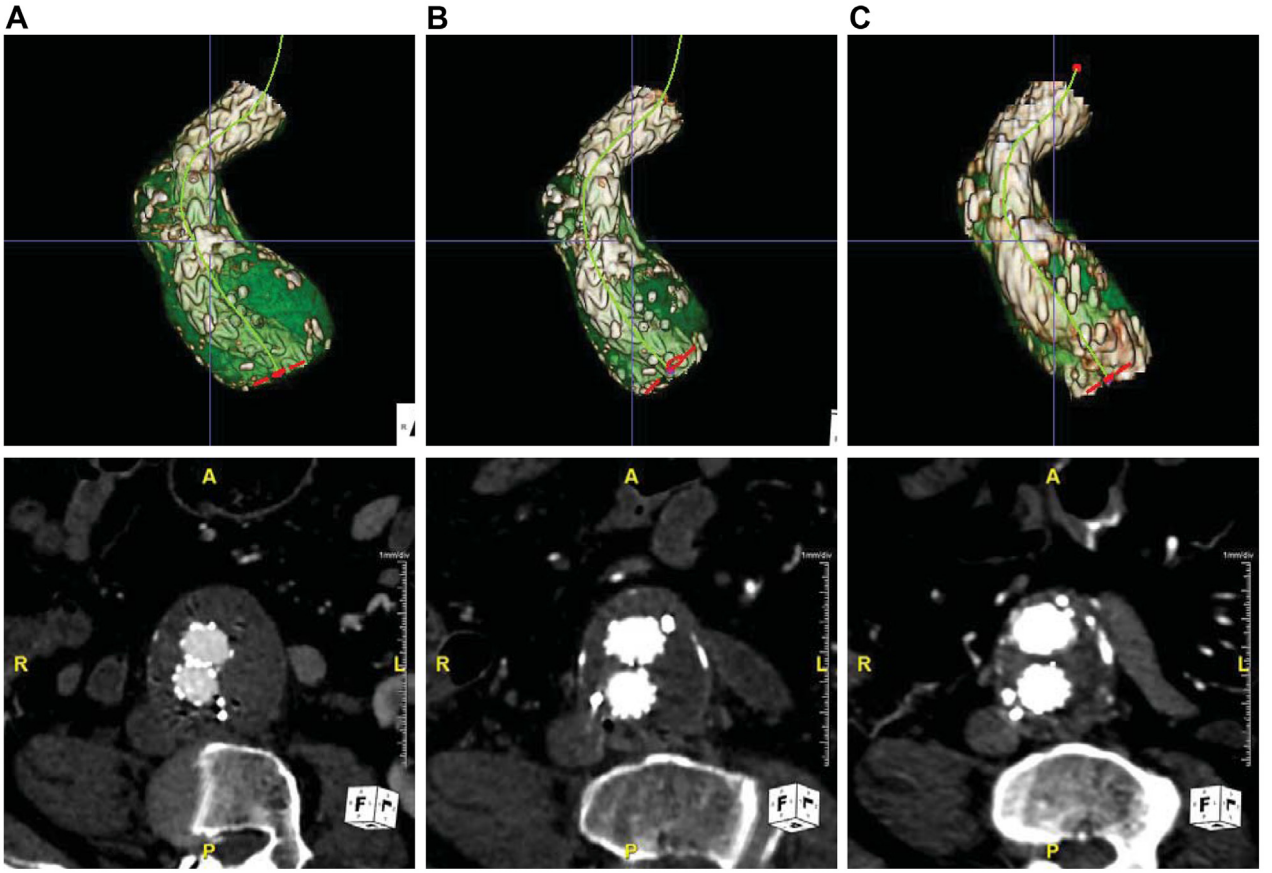
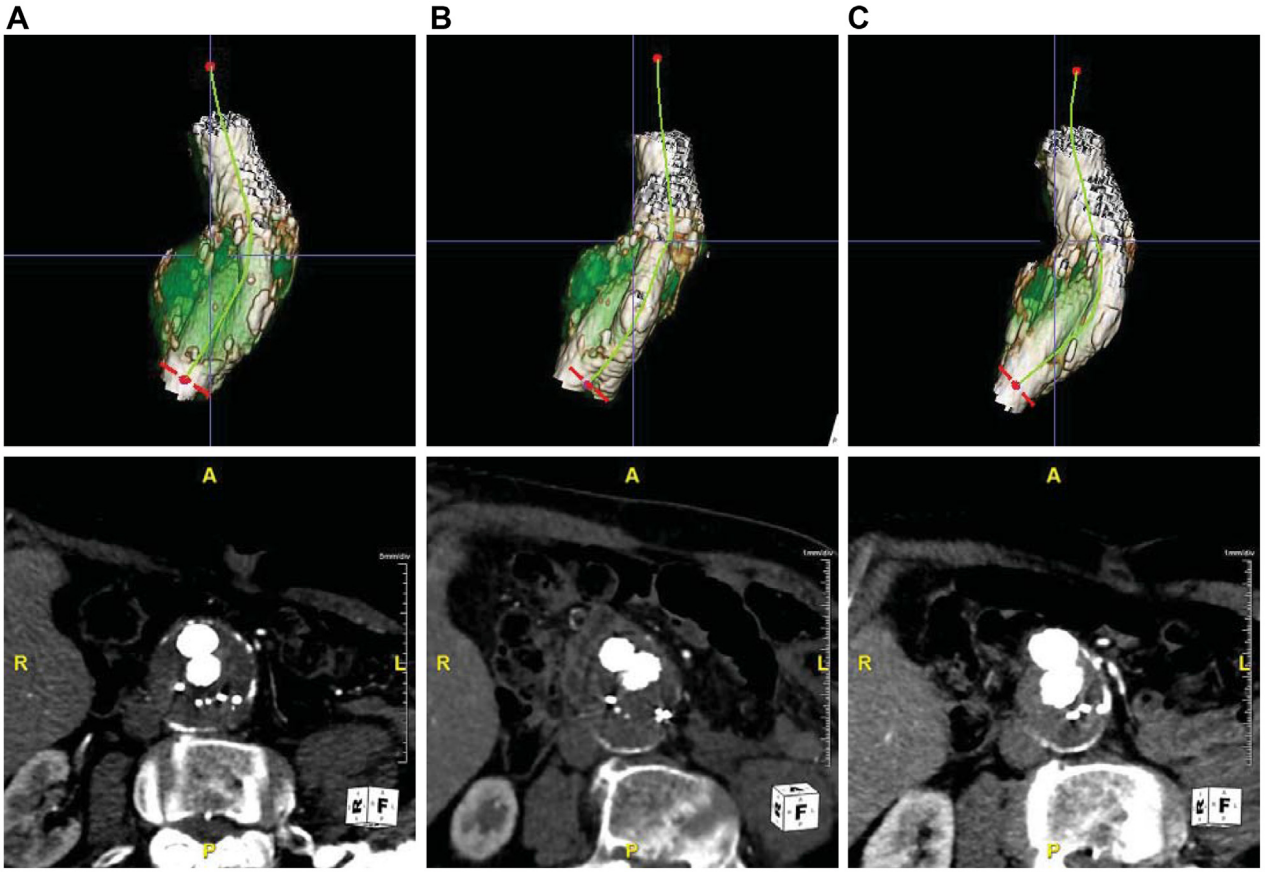
Supplementary Table II (online only). Major and serious adverse events (*SAE*)[a]

Adverse event	Device related[b]	Procedure related[b]	Days postprocedure[c]	Status[d]
MAEs or SAEs — 30 days through 1 year				
-	-	-	-	-
SAEs — through 30 days (in per protocol patients, N = 34)				
Arrythmia	No	Yes	1	Resolved without sequelae
Postimplantation syndrome	Yes	No	1	Resolved without sequelae
Constipation	No	Yes	2	Resolved without sequelae
Abdominal pain	No	Yes	5	Resolved without sequelae
SAEs — through 30 days (in additional intention-to-treat patients, total N = 37)				
Fever[e]	Yes	No	1	Resolved without sequelae
Low hemoglobin[e]	No	Yes	2	Resolved with sequelae

SAE, Serious adverse events.
[a]Serious per ISO 14155:2020 Clinical investigation of medical devices for human subjects—Good clinical practice. Major adverse events were defined as all-cause mortality, bowel ischemia, myocardial infarction, paraplegia, renal failure, respiratory failure, stroke, or procedural blood loss of >1000 mL. Endoleaks are reported separately in the text.
[b]Probably related or greater to the study device or study procedure. Relatedness to the study device or procedure was adjudicated by a medical monitor independent from the study investigators.
[c]Days after the procedure of event onset, 0 = day of procedure.
[d]Status at the time of manuscript submission.
[e]Patient with an inflammatory aneurysm and followed separately.



Supplementary Fig 1 (online only). Patient with ≥10% decrease in normalized volume and ≥5 mm decrease in diameter at 6 months and even further improvement at 1 year. The preprocedural target lumen volume estimate was 61.4 mL. The shape memory polymer (SMP) volume/target lumen volume estimate ratio was 1.4. **(A)** The baseline normalized volume was 128.0 mL and the diameter was 62.6 mm. **(B)** At 6 months, the patient showed a −23.0% change in normalized volume and a −6.3 mm change in diameter from baseline. **(C)** At 1 year, the patient showed a −31.8% change in normalized volume and a −10.2 mm change in diameter from baseline, resulting in a normalized volume of 87.3 mL and a diameter of 52.4 mm. This patient also had a type II endoleak at 6 months (0.2 mL) and 1 year (0.5 mL), based on core laboratory imaging review.



Supplementary Fig 2 (online only). Patient with a ≥10% decrease in normalized volume at 6 months and 1 year, but with no categorical change in diameter at either timepoint. The preprocedural target lumen volume estimate was 46.9 mL. The shape memory polymer (SMP) volume/target lumen volume estimate ratio was 1.3. **(A)** The baseline normalized volume was 129.6 mL and the diameter was 57.9 mm. **(B)** At 6 months, the patient showed a −34.4% change in normalized volume and a −2.5 mm change in diameter from baseline. **(C)** At 1 year, the patient showed a −46.9% change in normalized volume and a −4.1 mm change in diameter from baseline, resulting in a normalized volume of 68.8 mL and a diameter of 53.8 mm. No endoleaks were observed in this patient.