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Original Article

Chronological Change of the Sac after Endovascular Aneurysm Repair

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Purpose: The purpose of this study was to evaluate the potential risk factors of type II endoleak and sac growth after endovascular aneurysm repair (EVAR) and the outcomes of secondary interventions.

Materials and Methods: Ninety seven patients underwent elective EVAR for infrarenal abdominal aortic aneurysms in two tertiary centers between April 2005 and July 2013. Clinical and imaging parameters were compared among sac growth (>5 mm) and non-growth groups. Risk factors associated with sac growth and persistent type II endoleak were analyzed. The outcomes of reinterventions for persistent type II endoleak were determined.

Results: Sac growth was observed in 20 cases (20.6%) and endoleak was found in 90% of them compared to 28.6% (22/77) in the non-growth group (P<0.001). The majority of endoleaks were type II (36/40) and 80.5% were persistent. Sac diameter, neck diameter and number of patent accessory arteries were also statistically significant for sac growth. On multivariate analysis, grade of calcification at the neck, grade of mural thrombus at the inferior mesenteric artery and number of patent accessory arteries were risk factors of persistent type II endoleak. Twenty six reinterventions were done for 16 patients with persistent type II endoleak, with a technical success rate of 88.5%, yet 55.5% showed sac growth regardless of technical success. There were no ruptures during the follow-up period.

Conclusion: Sac growth after EVAR was mostly associated with persistent type II endoleak. Secondary interventions using transarterial embolization is partially effective in achieving clinical success.

Key Words: Abdominal aortic aneurysm, Sac enlargement, Endoleak

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INTRODUCTION

Endovascular aneurysm repair (EVAR) is a less invasive alternative method of repairing abdominal aortic aneurysms (AAA) to open repair. A common complication after EVAR is endoleak which is linked to adverse outcomes such as sac growth or rupture. Type I and III endoleaks are generally regarded candidates for urgent reinterventions due to the high pressurization of the aneurysm sac and are normally caused by the mismatch of stent dimensions and aneurysm morphology. This problem can potentially be overcome with arrival of newer generation devices allowing better fit in complex aneurysms. Type II endoleaks on the other hand, are caused by the collateral retrograde flow into the

sac from the aortic branches. Though initially thought to be benign, the presence of persistent type II endoleaks (>6 months) have been shown to be associated with an increased incidence of aneurysm sac growth, secondary reinterventions, open repair, and rupture [1,2]. While some groups adapt a more aggressive prophylactic approach to embolize potential sources in selective high risk patients, it is generally regarded that secondary intervention is indicated in the presence of type II endoleak with sac growth (>5 mm in diameter) [3]. However the outcomes of secondary interventions outcomes vary in terms of success rate and sac size changes.

Here, we report our experiences of sac growth after EVAR, with relationship to persistent type II endoleak. The purpose of this study was to evaluate the potential risk factors of type II endoleak and sac growth along with the outcomes of secondary interventions.

MATERIALS AND METHODS

1) Study population

We retrospectively reviewed the data of patients who had undergone EVAR for infrarenal AAA at two tertiary care hospitals and parameters such as persistent endoleak, sac growth, outcome and risk factors were analyzed. The study was approved by the Institutional Review Board of Seoul National University Bundang Hospital (IRB no. B-1408/264-101) and the requirement for informed consent from the patients was waived. By searching the database from the two institutions (Seoul National University Hospital and Seoul National University Bundang Hospital), 138 patients were identified who underwent EVAR for infrarenal type AAA between April 2005 and July 2013. The inclusion criteria were as follows: (a) patients treated with EVAR with a favorable anatomy (within the instructions for use); (b) available radiologic images for at least one year after the procedure. Of these, a total of 97 patients were analyzed. Procedures were done with the collaboration of interventional radiologists and vascular surgeons in an operation room setting equipped with fluoroscopic unit. The surgical approach and technique varied and were determined according to lesion location, type, and clinical features.

2) Assessment of clinical and imaging features

All patients received a clinical examination, laboratory tests including serum creatinine, and a measurement of ankle-brachial index when necessary at baseline. Clinical characteristics of the patients included age, sex, height, weight, body mass index, smoking history, and comorbidities.

On contrast-enhanced computed tomography (CT), an independent reader assessed aneurysm characteristics including the following: sac diameter (mm), neck length (mm), presence of a reverse tapered neck, presence of a short neck, grade of mural thrombus, calcification grade, suprarenal and infrarenal aortic neck angulation (degree). Aneurysm morphologic characteristics were defined according to the standards and grading factors suggested by the Society for Vascular Surgery [4,5]. Sac diameter was defined as the short diameter of the maximal aneurysmal plane on axial image shown in CT scans. A reverse tapered neck was defined as a proximal neck with more than 2 mm dilatation of diameter within 10 mm from the lowest renal artery [6]. A short neck was defined as an aneurysm starting less than 15 mm from the lowest renal artery. The grade of mural thrombus was measured at the neck and was graded from 0-3, each being 0%, 0%-25%, 25%-50%, >50%. Calcification grade was measured at the neck and was graded 0-2, each being <25%, 25%-50%, >50%. The sac diameter, neck length, and aortic neck angulation were measured using electronic calipers on picture archiving and communication system. The inferior mesenteric artery (IMA) was also assessed, which included the patency and diameter of the IMA. Mural thrombus at the level of the IMA was additionally measured and was graded 0-2, each being <50%, >50%, 100% of the aortic circumference, respectively [7]. The number of patent accessory arteries, which included the lumbar arteries, median sacral artery, and accessory renal arteries visible on CT images, were also counted.

3) Outcome measurement

Image follow-up interval of patients was at 1 week, 1 month, 6 months, 12 months and annually thereafter. If there were signs of sac enlargement with presence of endoleak, the clinical follow-up schedule was shortened. Sac growth was defined as a sac diameter change of more than 5 mm compared with the most recent follow-up image study. Endoleak was defined as persistence of blood flow between the graft and the aneurysmal sac wall as determined by imaging studies. Persistent endoleak was described as an endoleak proven to be existent on follow-up images for at least 6 months. All other parameters were defined according to the reporting standards suggested by the Society for Vascular Surgery [5].

4) Secondary interventions

The primary method was transarterial embolization with

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either coil embolization (n=13) and/or *n*-butyl-2-cyanoacrylate (NBCA)-glue:lipiodol mixture (1:1-5, n=19). Technical success was defined as the occlusion of the feeding artery and/or endoleak sac confirmed by angiography performed immediately after the embolization process. Clinical success was defined as no sac growth irrespective of the existence of residual endoleaks during continued follow-up (minimum 3 months follow-up).

5) Statistical analysis

Means and standard deviations were used for continuous variables and frequencies and percentages were used for categorical variables. All continuous variables were initially assessed for normality using the Kolmogorov-Smirnov test.

For identification of the characteristics of sac growth, all patients were divided into two groups: sac growth group (sac diameter of at least 5 mm growth compared to initial diameter) and non-growth group. To assess between the 2 groups, the Student's t-test was used for continuous variables and the Fisher's exact test for categorical variables.

Clinical and imaging features were analyzed as possible risk factors for their association with sac growth by univariate analysis. A Cox proportional hazards model was used for multivariate analysis of sac growth by including variables that were statistically significant (P<0.05) on univariate analysis. Univariate and multivariate analysis was done among morphological features of the sac for persistent type II endoleak. Additionally, the cases which underwent a secondary procedure after EVAR were reviewed in regards to their sac growth, follow-up period, and outcome.

All statistical analyses were performed using IBM SPSS Statistics ver. 20.0 (IBM Co., Armonk, NY, USA), and statistical significance was defined as P-value<0.05.

RESULTS

1) Sac changes

A summary of patient demographics and clinical factors of the studied population is summarized in Table 1. The mean sac diameter at baseline was 53.5±10.5 mm (range, 30.4-93.2 mm) and showed a mean decrease of −4.16±11.2 mm after the procedure (average period: 45.8±23.3 months; range, 12-114 months). Sac growth was observed in 20 cases (20.6%) with an average increase of 10.9±5.28 mm in diameter. Sac regression (more than 5 mm decrease in diameter) was observed in 45 patients (46.4%) with an average change in diameter of −14.1±5.98 mm, and no change of diameter was observed in 32 patients (33.0%, average change +0.46±2.6 mm). There were no statistically significant differences between the patients with and without sac growth in terms of age, sex, height, weight, smoking history, medical comorbidities, or device used. A comparison of the clinical and imaging characteristics of the patients with and without sac growth is summarized in Table 2.

Open repair after EVAR was done in 4 patients. The average time to open repair was 46±21 months (range, 25-74 months). The respective causes of open repair were as follows: Type I endoleak with sac growth (73.2 mm→89.6 mm), endotension with sac growth (67.5 mm→89.6 mm), failed secondary intervention for type II endoleak with sac growth, and graft infection following 4 prior secondary interventions.

2) Sac growth and association with endoleaks

Endoleaks that appeared at least once during the follow-up period were found in 41 (42.3%) patients, the majority being type II endoleak. Endoleaks occurred in 90% of the patients with sac growth in contrast to 28.6% without sac growth. Type II endoleaks were observed in 15 of the 18 endoleaks in patients with sac growth, 14 (93.3%) of which were persistent type II endoleaks. Persistent type II endoleaks accounted for only 15 (out of 21 type II endoleaks, 71.4%) cases in the non-sac growth group. One type I endoleak was found in the non-growth group and was treated within one week post-EVAR without complications. A summary of the endoleaks relative to each group is noted in Table 2.

Table 1. Patient demographics and clinical factors

Characteristic	Value
No. of patients	97
Age (y)	72.2±8.1
Male	89 (91.8)
Smoking history	33 (34.0)
Comorbidities	
Diabetes mellitus	17 (17.5)
Hypertension	67 (69.1)
Coronary artery disease	28 (28.9)
Cerebrovascular disease	8 (8.2)
Chronic renal failure[a]	12 (12.4)
Device	
Zenith	68 (70.1)
Endurant	22 (22.7)
Excluder	7 (7.2)

Values are presented as number only, mean±standard, or number (%).
[a]Serum creatinine >1.5 mg/dL.

Table 2. Comparison of the parameters of patients with/without sac growth

Parameter	No sac growth (n=77)	Sac growth (n=20)	P-value
Clinical findings			
Age (y)	71.8±8.19	73.7±7.81	0.365
Male	72 (93.5)	17 (85.0)	0.355
Height (cm)	167.1±6.63	166.9±6.66	0.907
Weight (kg)	67.0±9.50	68.1±9.20	0.649
Body mass index	23.95±2.85	24.53±3.64	0.445
Smoking history	28 (36.4)	5 (25.0)	0.432
Comorbidities			
Diabetes mellitus	13 (16.9)	4 (20.0)	0.747
Hypertension	52 (67.5)	15 (75.0)	0.597
Coronary artery disease	20 (26.0)	8 (40.0)	0.270
Cerebrovascular disease	5 (6.5)	3 (15.0)	0.355
Chronic renal failure	8 (10.4)	4 (20.0)	0.262
Stent manufacturer			0.845[a]
Cook Zenith	53 (68.8)	15 (75.0)	
Medtronic endurant	18 (23.4)	4 (20.0)	
Gore excluder	6 (7.8)	1 (5.0)	
Initial lesion characteristics			
Sac diameter (mm)	52.4±10.4	57.6±10.1	0.048
Neck diameter (mm)	22.5±2.28	24.2±2.11	0.004
Neck length (mm)	39.8±16.4	32.8±13.6	0.083
Suprarenal angulation	30.2±22.0	38.9±20.9	0.118
Infrarenal angulation	54.6±19.6	59.0±16.3	0.359
Presence of reverse tapered neck	10 (13.0)	1 (5.0)	0.451
Presence of short neck	4 (5.2)	1 (5.0)	1.00
Presence of patent IMA	61 (79.2)	18 (90.0)	0.349
Patent IMA diameter (mm)	3.43±0.73	3.77±0.63	0.085
Patent accessory arteries (n)	5.38±2.22	6.96±1.79	0.004
Follow-up (mo)	42.9±22.4	56.7±24.1	0.018
Sac diameter change (mm)	-8.07±8.71	10.9±5.28	<0.001
Endoleaks	22 (28.6)	18 (90.0)	<0.001
None	55	2	
I	1	1	
II (persistent type II, n)	21 (15)	15 (14)	
III	0	1	
IV	0	0	
V	0	1	

Values are presented as mean±standard, number (%), or number only.
Reverse tapered neck indicates a proximal neck with more than 2 mm dilatation of diameter within 10 mm from the lowest renal artery; Short neck indicates neck less than 1.5 cm.
IMA, inferior mesenteric artery.
[a]Pearson chi-squared analysis.

3) Risk factors of sac growth and persistent type II endoleak

By univariate analyses, neck diameter, grade of calcification at neck, and number of patent accessory arteries were significantly associated with sac growth (Table 3). In the multivariate analyses, all three parameters were significant risk factors of sac growth (hazard ratio [HR]=1.44, P=0.006;

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Table 3. Logistic regression analysis for sac growth

Variable	Univariate analysis		Multivariate analysis	
	HR (CI)	P-value	HR (CI)	P-value
Age (y)	1.03 (0.97-1.10)	0.362		
Male	0.39 (0.09-1.81)	0.231		
Body mass index	1.07 (0.90-1.26)	0.441		
History of smoking	0.58 (0.19-1.78)	0.343		
Diabetes mellitus	1.23 (0.35-4.23)	0.744		
Hypertension	1.44 (0.47-4.42)	0.521		
Coronary artery disease	1.90 (0.68-5.32)	0.222		
Cerebrovascular disease	2.54 (0.55-11.69)	0.231		
Chronic renal failure	2.16 (0.57-8.05)	0.253		
Sac diameter (mm)	1.05 (1.00-1.09)	0.057		
Neck diameter (mm)	1.38 (1.09-1.74)	0.007	1.44 (1.11-1.87)	0.006
Presence of reverse tapered neck	0.35 (0.04-2.93)	0.335		
Presence of short neck	0.96 (0.10-9.10)	0.972		
Grade of mural thrombus at neck	1.26 (0.78-2.04)	0.337		
Grade of calcification at neck	2.37 (1.08-5.21)	0.032	3.51 (1.38-8.96)	0.009
Suprarenal angulation (°)	1.02 (1.00-1.04)	0.123		
Infrarenal angulation (°)	1.01 (0.99-1.04)	0.356		
Presence of patent IMA	2.36 (0.50-11.24)	0.281		
Patent IMA diameter (mm)	1.92 (0.90-4.06)	0.090		
Grade of mural thrombus at IMA	0.66 (0.32-1.35)	0.257		
Patent accessory arteries (n)	1.49 (1.12-2.00)	0.007	1.59 (1.14-2.20)	0.006

Reverse tapered neck indicates a proximal neck with more than 2 mm dilatation of diameter within 10 mm from the lowest renal artery; Short neck indicates neck less than 1.5 cm; Grade of mural thrombus at neck was defined as grade 0: 0%, grade 1: 0%-25%, grade 2: 25%-50%, and grade 3: >50% of aortic circumference; Grade of calcification at neck was defined as grade 0: <25%, grade 1: 25%-50%, and grade 2: >50% of aortic circumference; Grade of mural thrombus at IMA was defined as grade 0: <50%, grade 1: >50%, and grade 2: 100% (totally encircling) of aortic circumference.
HR, hazard ratio; CI, confidence interval; IMA, inferior mesenteric artery.

HR=3.51, P=0.009; HR=1.59, P=0.006; respectively).

Similarly by using the significant morphological parameters in univariate analysis for persistent type II endoleak, grade of calcification at neck (HR=3.65, P=0.006), grade of mural thrombus at IMA level (HR=0.43, P=0.041), and number of patent accessory arteries (HR=1.48, P=0.010) were independent risk factors in multivariate analysis (Table 4).

4) Secondary interventions and sac growth

There were 16 patients with a total of 26 interventions who received secondary interventions for type II endoleak during the observed period of 54.6±26.1 months (range, 19-114 months). The average time to first intervention was 27.3±21.5 months (range, 2.8-77.0 months). Six patients (37.5%) required more than one reintervention (2 interventions in 3 patients, three interventions in 2 patients and 4 interventions in 1 patient). Six cases were targeted solely to the IMA, 5 were targeted to the IMA plus lumbar/accessory arteries, and 15 were targeted to the lumbar and/or accessory arteries. Simultaneous transarterial embolization of the sac was done in 9 cases. The primary cause of intervention was sac growth (n=23, 88.5%). There were two cases where angiography was done to differentiate type II from a more aggressive type (I or III) endoleak in which both cases proved to be type II and embolization was done for preventive measures. In 11 patients (68.8%), the endoleaks that were targeted were present from the immediate post-operative period. A summary of cases are presented in Table 5.

Technical success was achieved in 88.5%, and clinical success was achieved in 5 out of 11 patients (45.5%) irrespective of technical success. Chronological representation of the sac diameters showed two distinct patterns of behavior after the first reintervention (Fig. 1). Six patients had a tendency for sac growth regardless of technical success or residual endoleak status. Eventually, all patients with this behavior underwent multiple reinterventions. For

Table 4. Logistic regression analysis of selective parameters for persistent type II endoleak

Variable	Univariate analysis		Multivariate analysis	
	HR (CI)	P-value	HR (CI)	P-value
Sac diameter (mm)	1.01 (0.97-1.05)	0.773		
Neck diameter (mm)	1.06 (0.88-1.28)	0.551		
Presence of reverse tapered neck	0.21 (0.03-1.70)	0.143		
Presence of short neck	0.57 (0.06-5.35)	0.624		
Grade of mural thrombus at neck	1.10 (0.71-1.71)	0.669		
Grade of calcification at neck	2.18 (1.03-4.60)	0.042	3.65 (1.44-9.27)	0.006
Suprarenal angulation	1.02 (1.00-1.04)	0.112		
Infrarenal angulation	1.02 (0.99-1.04)	0.219		
Presence of patent IMA	9.33 (1.18-73.87)	0.034	6.19 (0.57-66.9)	0.134
Patent IMA diameter (mm)	2.41 (1.19-4.86)	0.014		
Grade of mural thrombus at IMA	0.41 (0.21-0.81)	0.010	0.43 (0.19-0.97)	0.041
Patent accessory arteries (n)	1.47 (1.14-1.89)	0.003	1.48 (1.10-1.99)	0.010
Stent manufacturer (vs. Cook Zenith)				
Cook Zenith	1.00	-		
Medtronic Endurant	1.20 (0.43-3.41)	0.727		
Gore Excluder	1.93 (0.40-9.47)	0.415		

Reverse tapered neck indicates a proximal neck with more than 2 mm dilatation of diameter within 10 mm from the lowest renal artery; Short neck indicates neck less than 1.5 cm; Grade of mural thrombus at neck was defined as grade 0: 0%, grade 1: 0%-25%, grade 2: 25%-50%, and grade 3: >50% of aortic circumference; Grade of calcification at neck was defined as grade 0: <25%, grade 1: 25%-50%, and grade 2: >50% of aortic circumference; Grade of mural thrombus at IMA was defined as grade 0: <50%, grade 1: >50%, and grade 2: 100% (totally encircling) of aortic circumference.
HR, hazard ratio; CI, confidence interval; IMA, inferior mesenteric artery.

patients with multiple interventions (n=6), the mean time from the first reintervention to the second reintervention was 32.1±14.8 months (range, 9-54 months) with an average number of 3.2±1.3 (range, 1-5) follow-ups before the next reintervention. Of the patients receiving reinterventions, there were 4 patients where endoleaks were not detected on the first or second subsequent follow-ups following reintervention, however developed another type II endoleak thereafter. Eventually, 3 of the 4 patients underwent additional reinterventions.

Residual type II endoleak after secondary intervention was present in 14 of the 20 cases (excluding cases of follow-up loss). There were 9 cases where an initial regression was followed by growth resembling a 'dipper' shape in the graph (Fig. 1B-E, G-K). This initial regression was irrespective of the residual endoleak status.

DISCUSSION

The results of this study confirm the high prevalence of sac growth and type II endoleak after EVAR in a mid-to-long term follow-up. 20.3% of the study population was observed to have sac growth, and 42.3% had type II endoleak. This is in concordance to previous reports where the incidence of growth of the aneurysm sac after EVAR varied between 0.2% and 41% and the incidence of type II endoleak between 0% to 47.7% [8]. Cases of rupture after type II endoleak have been described [1,9]. Despite sac growth, there were no recorded cases of rupture after EVAR in our population.

Presence of a patent IMA is known to be an independent risk factor of type II endoleak [10]. In our population, this was only shown to be significant in univariate analysis (HR=9.33, P=0.034) and was not so in multivariate analysis (P=0.134). Also the number of patent lumbar arteries detected on preoperative images is an independent risk factor for developing type II endoleak [11]. In an analysis of 308 patients, Couchet et al. [12] found that permeability of the IMA (83% vs. 69%, P=0.01), IMA diameter (3.49 mm vs. 2.71 mm, P<0.001), number of patent lumbar arteries higher than or equal to 4 (P<0.001), mean lumbar artery diameter greater than 2.4 mm (P<0.001), and median sacral artery diameter (2.28 mm vs. 1.94 mm, P<0.01) were predictive morphologic factors of type II endoleak. Our results showed a hazard ratio of 1.48 (95% confidence interval, 1.10-1.99; P=0.010) for each number increase in patent accessory arteries. The amount of circumferential mural thrombus in the sac has shown to be a protective factor in the

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Table 5. Summary of patients undergoing secondary interventions

Sex/ age (y)	Case	Indication	Time (mo)	Source	Embolization material	Target	Technical success	Residual type II endoleak
M/82	1	Sac growth	68.5	IMA	Coiling	Main feeder	Yes	NA
M/67	2	Sac growth	30.6	IMA	Coiling	Main feeder	Yes	Yes
M/75	3	Differentiate type II vs. III	12.5	Right circumflex iliac	Coiling	Main feeder	Yes	No
M/62	4	Sac growth	77.0	IMA	NBCA (1:3)	Endoleak sac	Yes	Yes
M/86	5	Sac growth	8.2	Lumbar	Coiling and NBCA (1:5)	Main feeder & endoleak sac	Yes	No
M/66	6-1	Signs of sac growth	7.5	IMA	Coiling and NBCA (1:5)	Endoleak sac	Yes	No
	6-2	Sac growth	61.5	IMA+lumbar	NBCA (1:5)	Main feeder	Yes	Yes
M/73	7	Sac growth	7.8	Lumbar and midsacral	Coiling and NBCA (1:5)	Endoleak sac	Yes	No
	7-1	Residual IMA endoleak	38.8	IMA	NBCA (1:5)	Main feeder	Yes	No
	7-2	Sac growth	44.9	Lumbar	NBCA (1:5)	Main feeder	Yes	NA
M/80	8-1	Sac growth	9.6	Branches of right iliolumbar	Sac-coiling and NBCA (1:1)	Endoleak sac	Yes	Yes
	8-2	Sac growth	42.6	Lumbar	NBCA (1:5)	Main feeder	Yes	Yes
F/72	9-1	Sac growth	21.4	IMA+lumbar	Lumbar-NBCA (1:4)	Main feeder	Partial[a]	Yes
	9-2	Sac growth	30.4	IMA	NBCA (1:2)	Main feeder	Yes	Yes
	9-3	Sac growth	41.4	Lumbar	NBCA (1:4, 1:5)	Main feeder	Yes	Yes
	9-4	Sac growth	51.8	Right circumflex iliac	PVA particle	Main feeder	Yes	Yes
M/74	10	Sac growth	26.3	Lumbar and midsacral	Coiling and NBCA (1:6)	Endoleak sac	Yes	No
M/65	11-1	Sac growth	21.1	IMA+lumbar	IMA-coiling	Main feeder	Partial[b]	Yes
	11-2	Sac growth	60.4	Lumbar	Feeder-coiling sac-coiling and NBCA	Main feeder & endoleak sac	Yes	NA
F/81	12-1	Differentiate type I vs. II	2.8	IMA+lumbar	IMA, lumbar-coiling sac-coiling and NBCA (1:4)	Main feeder & endoleak sac	Yes	Yes
	12-2	Sac growth	29.2	Median sacral	NBCA (1:5)	Main feeder	Yes	Yes
	12-3	Sac growth	46.9	Lumbar	NBCA (1:3)	Main feeder	Yes	Yes
F/72	13	Sac growth	34.3	Lumbar	Gelatin foam	Main feeder	No	NA
M/87	14	Sac growth	44.5	Lumbar	Inflow-NBCA (1:2) outflow-coiling	Main feeder & draining vessel	Yes	Yes
M/73	15	Sac growth	31.7	Lumbar	NBCA (1:5)	Main feeder & endoleak sac	Yes	NA
M/69	16	Sac growth	33.1	IMA+lumbar	IMA-coiling lumbar-gelatin foam	Main feeder	Yes	NA

All cases were transarterial embolization.
M, male; F, female; IMA, inferior mesenteric artery; NBCA, *n*-butyl-2-cyanoacrylate; PVA, polyvinyl alcohol; NA, no available computed tomography images after embolization.
[a]IMA selection failed, [b]Lumbar artery selection failed.

development of endoleaks [7,11,13]. This was shown to be significant for our study population as well.

The treatment strategy for type II endoleak varies and the optimal threshold for secondary interventions is insufficient [14,15]. However, interventions are generally indicated when there is sac growth. A systematic review done by Sidloff et al. [9] analyzed 21,744 patients from 32 studies with 393 reinterventions, of which 281 (71.5%) were

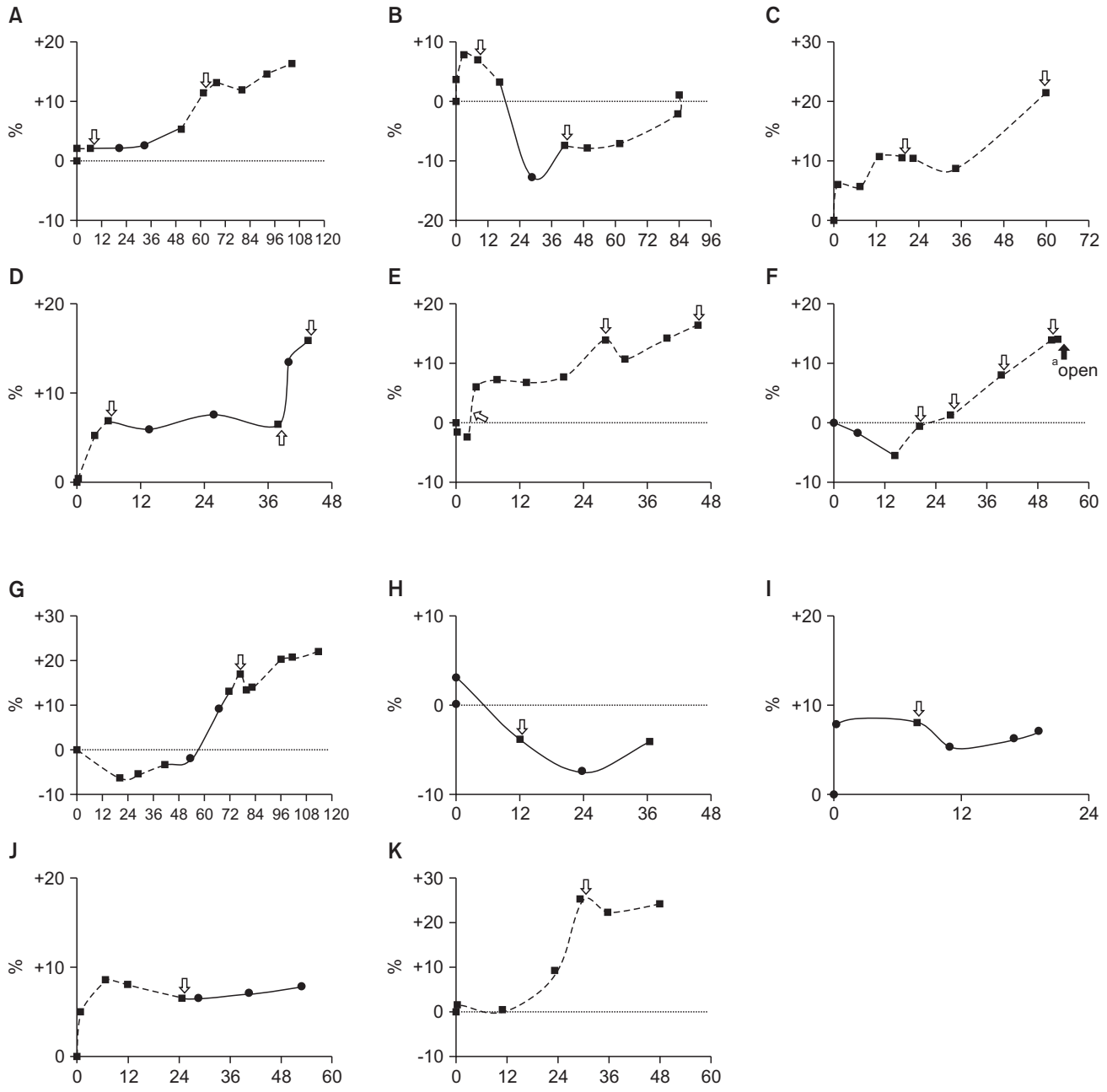

Fig. 1. Chronological change of sac diameters for selective patients with follow-up data of at least 3 months after first reintervention. X-axis, months; Y-axis, percentage diameter difference from initial diameter. Square points represent endoleaks existent on computed tomography image. Circular points represent no endoleaks. Dotted lines are representative of the diameter change when endoleaks are present. White arrows indicate the time at which an intervention was performed. Black arrows with [a]open, open repair. (A-F) Cases where growth was shown irrespective of initial reintervention. (G-K) Cases where reinterventions were regarded sufficient enough to prevent sac growth.

technically successful. Transarterial approach was done in 120 cases, of which 57 (47.5%) were reported as having a stable or decreasing sac after reintervention. The largest transarterial embolization series reported was done by Sarac et al. [16] where 95 patients underwent 140 embolization procedures. Twenty percent required more than one procedure, and the freedom from sac growth was 81.5% at 1 year and 43.7% at 5 years. Our results showed technical success in 88.5% and clinical success in 45.5% of cases. However, 6 out of 11 patients showed sac growth despite multiple reinterventions. Aziz et al. [17] reported that there was no significant effect of reintervention in the rate of

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sac growth/regression. They reported that in 42 patients who underwent reintervention, the mean diameters of the aneurysm were 6.1±1.6 cm preoperatively, 6.6±1.5 cm at the initial reintervention, and 6.9±1.7 cm at the last follow-up.

Although not statistically significant, there was a tendency for higher use of NBCA glue mixture alone (compared to coiling+NBCA glue mixture) in the group showing sac growth following secondary interventions. This was not evident on immediate or short term follow-ups, but rather after two to three years of follow-up. This may suggest that the effect of NBCA glue may decay over time. However in the results shown by Sarac et al. [16], there were no differences in terms of materials used. Interestingly, patients who underwent only coil embolization were more likely to require a second intervention.

Our primary method for treating type II endoleaks was transarterial embolization, however translumbar direct sac embolization is another alternative technique. Baum et al. [18] was one of the first to report a case series comparing the efficacy of translumbar vs. transarterial embolization, stating that all patients should undergo the translumbar approach. Additionally recent reports have shown the superiority of translumbar approach over transarterial approach, especially with the utilization of Onyx (ev3 Inc., Irvine, CA, USA) [9,19,20]. Due to the unavailability of this material during the study period, translumbar embolization was not done in our cases and may be a limitation to this study. Considering the recent promising treatment results of this method, we believe translumbar embolization using Onyx may be a potentially good option in the treatment of type II endoleaks.

The relatively high follow-up loss rate, partially due to the underlying comorbidities of the patients and the difficulty of tertiary centers in tracking all patients, and the small number of cases, can be considered a limitation to this study. However, some of this was overcome by actively reaching out to the patients to call in their regular follow-ups.

CONCLUSION

This study demonstrated that sac growth after EVAR was mostly associated with persistent type II endoleaks. Morphological factors associated with sac growth were neck diameter, grade of calcification at the neck, and increasing number of patent accessory arteries. Secondary interventions using transarterial embolization for type II endoleaks with sac growth is only partially effective in selective cases, and thus alternative approaches such as translumbar embolization should be considered for improved outcomes.

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