CLINICAL STUDY

Evaluation of Delayed Endoleak Compared with Early Endoleak after Endovascular Aneurysm Repair

Trong Binh Le, MD, Keun-Myoung Park, MD, Yong Sun Jeon, MD, PhD, Kee Chun Hong, MD, PhD, and Soon Gu Cho, MD, PhD

ABSTRACT

Purpose: To identify prevalence and evaluate outcomes of delayed endoleak (DEL) compared with early endoleak (EEL) after endovascular aortic aneurysm repair (EVAR).

Materials and Methods: Data of 164 patients who underwent elective EVAR at a single center were retrospectively analyzed. DEL was defined as any type of endoleak that was first detected ≥ 12 months after EVAR. Patients who had < 1 year of follow-up were excluded. Endoleak was classified into a more aggressive category if a patient had > 1 type of endoleak. Analysis included 81 patients (82.7% male). Mean age was 73.1 years ± 9.3. Median follow-up duration was 43 months (range, 12–135 months).

Results: Endoleak was present in 32 patients (39.5%), including 21 EEL (25.9%) and 11 DEL (13.6%). DEL consisted of 2 type I, 5 type II, 1 type III, and 3 type V (endotension). Median time to detection was 45 months (range, 15–60 months), and median follow-up duration was 62 months (range, 37–104 months). Compared with EEL, DEL had larger aneurysm diameters and higher rates of non–type II endoleak and reintervention. Type II DEL also required more reintervention procedures than type II EEL.

Conclusions: DEL had a noteworthy incidence and occurred late after EVAR. It predominantly consisted of non–type II endoleak and appeared to have more reinterventions than EEL. Meticulous long-term imaging surveillance to identify and manage DEL is critical.

ABBREVIATIONS

DEL = delayed endoleak, EEL = early endoleak, EVAR = endovascular abdominal aortic aneurysm repair

Short-term survival benefits of endovascular abdominal aortic aneurysm repair (EVAR) versus open repair in the treatment of abdominal aortic aneurysm have been widely accepted (1–4). However, these early advantages usually erode over time (2,4–7). A long-term study found that aneurysm-related deaths increased from 6 months after EVAR (4). The cause of death was prominently aneurysm rupture, which was partly due to sac expansion resulting from uncorrected endoleak (1,3–5). Endoleak is a major concern after EVAR with a reported incidence of 20%–50% of patients (1,8–10); approximately half of these endoleaks are type II (6,7). The natural history of type II endoleaks is poorly understood, and thus management of this type remains controversial, whereas type I and III endoleaks usually require early intervention (1,6,7,9). Spontaneous resolution of type II endoleak was reported in 35.4% of patients over a range of 3 months to 4 years (7,11). However, up to 20% of type II endoleaks persist over time and increase the risk of reintervention and aneurysm rupture (9,11). Although late endoleaks have been recognized in published reports, the frequency and clinical significance of endoleak detected ≥ 12 months after EVAR remains poorly defined (9). In 1 report, the incidence of delayed endoleak (DEL) was 13.1%, and type II DEL was significantly associated with sac enlargement compared with type II early endoleak (EEL) (9). This study aimed to identify the prevalence and evaluate outcomes of DEL compared with EEL.

From the Endovascular Training Center (T.B.L.) and Departments of Vascular Surgery (K.-M.P., K.C.H.) and Radiology (Y.S.J., S.G.C.), Inha University Hospital, Inha University School of Medicine, 7-206 Sinheung-dong 3-ga, Jung-gu, Incheon, Republic of Korea. Received May 13, 2017; final revision received September 22, 2017; accepted September 24, 2017. **Address correspondence to** Y.S.J.; E-mail: radjeon@inha.ac.kr

None of the authors have identified a conflict of interest.

© SIR, 2017

J Vasc Interv Radiol 2017; ■:1–7

https://doi.org/10.1016/j.jvir.2017.09.023

MATERIALS AND METHODS

Definitions

DEL was defined as any type of endoleak that was first detected ≥ 12 months after EVAR with all follow-up

computed tomography (CT) angiograms within the first year being negative for endoleak (9,10). EEL was defined as endoleak detected within 12 months. Endoleaks are stratified into 5 types based on the source of communication between the systemic circulation and aneurysm sac (3). Type V endoleak has also been termed an endotension, which indicates aneurysm enlargement after EVAR without a detectable endoleak (8,12,13). Endoleaks were classified into a more aggressive category if a patient had multiple endoleaks. In particular, type I and III endoleaks are considered more aggressive than type II, IV, or V endoleaks. For example, a patient with both type I and II endoleaks was classified in the type I endoleak category. When endoleak was detected on the completion angiogram after EVAR, the management strategy was (*i*) conservative if the endoleak was considered benign (type II or IV) or (*ii*) aggressive with intraoperative adjunctive procedures, including balloon molding, aortic cuff, limb extension, or additional graft if the endoleak was considered malignant (type I III). If endoleak still existed on 30-day follow-up CT angiography, it categorized as EEL.

Data Collection

This study was approved by the institutional review board. Informed consent was obtained from all patients. Data of 164 patients who underwent elective EVAR using commercially available devices for infrarenal abdominal aortic aneurysms from December 2005 to March 2017 were retrospectively analyzed. The following patients were excluded: (*i*) patients with EVAR for isolated iliac artery aneurysms (n = 12), (*ii*) patients who had < 1 year or loss of follow-up (n = 54), and (*iii*) patients who recently received EVAR and did not have 1 year of follow-up (n = 17). There were 81 patients (82.7% male; mean age, 73.1 y ± 9.3) identified for analysis. The overall median follow-up duration was 43 months (range, 12–135 months). Follow-up CT angiograms were obtained at discharge or during the first 30 days and at 6 months and at 12 months in the first postoperative year. After 12 months, CT angiography was recommended annually with an alternative option for contrast-enhanced ultrasound. Medical records and CT scans of each patient were carefully reviewed. Patient demographics, clinical characteristics, and comorbidities were documented. Type of endoleak, date of detection, type of stent graft, follow-up duration, and reintervention and complications after the procedure were also recorded. Abdominal aortic aneurysm profiles were reviewed from preoperative CT angiography. Sac diameters were documented according to the maximum diameter comparison from axial, sagittal, and coronal images. Clinical data were retrieved from hospital electronic medical records.

The CT angiography protocol included a detector coverage of 40 mm, gantry rotation time of 0.6 seconds, scan thickness of 1.25 mm, and image reconstruction interval of 2.5 mm using a 64-slice multidetector CT. Helical scan images were acquired from the xyphoid process to the feet in the supine position. Three separate imaging examinations were performed: (*i*) scan before contrast enhancement to identify opacities, (*ii*) contrast scan after infusion of nonionic contrast medium (BONOREX IOHEXOL 300; Central Medical Services, Seoul, Korea) with average dose of 2 mL/kg of body weight at 5 mL/s, and (*iii*) delayed phase scanned at 180–210 seconds after injection with slice thickness of 5 mm. Computer-assisted bolus-tracking software was used to determine the optimal scan delay for the arterial phase in each patient. All contrast-enhanced ultrasound scans were performed by 1 interventional radiologist (Y.S.J.) using a convex array probe (Philips iU22; Philips Healthcare, Andover, Massachusetts). Position, shape, internal echo, and diameter of the aneurysm were recorded. Color Doppler scan was performed to examine the blood flow and its signal within and around the graft. To fully evaluate size, location, direction, and sources of flow and phases of a detected endoleak, 4.8 mL of contrast agent (SonoVue; Bracco Imaging, Milan, Italy) was given intravenously, divided into 2 sessions.

Four types of stent grafts were used in this study, including 35 Zenith (Cook, Inc., Bloomington, Indiana), 21 Endurant (Medtronic, Minneapolis, Minnesota), 13 GORE EXCLUDER (W.L. Gore and Associates, Inc, Flagstaff, Arizona), and 12 Seal (S&G Biotech Inc, Seongnam, Korea). EVAR was indicated after multidisciplinary discussions and consensus between vascular surgeons and the interventional radiologist, considering each patient's age, clinical condition, imaging findings, and instructions for use of specific stent grafts. All procedures were performed by 1 interventional radiologist (Y.S.J.) with 15 years of experience in EVAR.

Table 1. Baseline Demographics and Comorbidities of Patients

Variables	No-Endoleak Group (n = 49)	EEL Group (n = 21)	DEL Group (n = 11)	*P* Value
Demographics				
Age, y, mean ± SD	72.1 ± 8.9	74 ± 10.6	76.1 ± 8.5	.386
Sex, male, n (%)	42 (85.7)	16 (76.2)	9 (81.8)	.625
Hostile neck, n (%)	22 (44.9)	9 (42.8)	7 (63.6)	.483
Comorbidity (%)				> .05
Smoking	17 (34.7)	10 (47.6)	6 (54.5)	
Hypertension	35 (71.4)	16 (76.2)	6 (54.5)	
Coronary artery disease	6 (12.2)	3 (14.3)	1 (9.1)	
Diabetes mellitus	15 (30.1)	9 (42.3)	3 (27.3)	
Cerebrovascular disease	2 (6.1)	1 (4.8)	0 (0)	
Hyperlipidemia	11 (22.4)	7 (33.3)	2 (18.2)	
Device				
Zenith	17	10	8	.147
Endurant	17	2	2	< .01
Excluder	10	3	0	.052
Seal	5	6	1	.174

DEL = delayed endoleak; EEL = early endoleak.

Table 2. Factors Associated with Delayed Endoleak

Variables	Univariate Analysis, OR (95% CI)	*P* Value	Multivariate Analysis, OR (95% CI)	*P* Value
Sac diameter before EVAR	1.12 (1.05–1.2)	< .01*	1.23 (1.06–1.43)	< .01*
Neck diameter	1.22 (1.03–1.44)	.809	1.03 (0.82–1.3)	.808
Neck length	0.96 (0.92–1.01)	.655	0.98 (0.91–1.06)	.645
Neck angulation	1.004 (0.98–1.03)	.942	0.99 (0.96–1.04)	.942
Age	1.24 (0.85–1.81)	.458	1.23 (0.72–2.1)	.449
Sex	0.93 (0.18–4.86)	.667	1.72 (0.15–20.03)	.663

CI = confidence interval; EVAR = endovascular aneurysm repair; OR = odds ratio.
*Indicates statistical significance.

The primary endpoint was incidence and timing of endoleak after the initial EVAR. Secondary endpoints were rates of sac growth, aneurysm rupture, and reintervention of DEL compared with EEL.

Statistical Analyses

Continuous data were presented as mean ± SD if the variables were normally distributed or as median and range if the variables were not normally distributed. Categorical data were given as counts and percentages. Independent 2-sample *t* test and Mann-Whitney *U* test were used to compare means and medians between 2 groups, respectively, whereas one-way analysis of variance was used to compare continuous data among DEL, EEL, and no-endoleak groups. Pearson χ^2 test and Fisher exact test were used to compare percentages of categorical variables. Logistic regressions were performed to ascertain the effects of age, sex, aneurysm diameter before EVAR, and neck morphology on the likelihood that a patient would develop DEL. All analyses were performed using R software version 3.1.2 (R Foundation for Statistical Computing, Vienna, Austria). *P* values < .05 indicated statistical significance for all comparisons.

RESULTS

Endoleaks were detected in 32 of 81 patients (39.5%), including 21 with EEL (25.9%) and 11 with DEL (13.6%). Across all baseline demographics, neck morphology, comorbidities, and types of device, no statistically significant differences were found among the no-endoleak, EEL, and DEL groups except for a higher proportion of the Endurant device used in the no-endoleak group (Table 1). Logistic regression analyses showed aneurysm sac diameter before EVAR was associated with DEL ($P <$ .01), whereas neck morphology, age, and sex were not associated with DEL (Table 2).

The DEL group included 2 (18.2%) type I, 5 (45.4%) type II, 1 (9.1%) type III, and 3 (27.3%) type V endoleaks (Table 3). All patients with DEL had no evidence of endoleaks on follow-up CT angiography during the first year after EVAR. Median time to detection was 45 months

Table 3. Comparison of Early Endoleak and Delayed Endoleak

Variables	EEL (*n* = 21)	DEL (*n* = 11)	*P* Value
Types of endoleak, n (%)			
Non–type II	4 (19)	6 (54.6)	.042
Type I	4 (19)	2 (18.2)	
Type III	0 (0)	1 (9.1)	
Type V	0 (0)	3 (27.3)	
Type II	17 (80.9)	5 (45.4)	.043
Time to detection, months, median (range)	1 (1–9)	45 (15–60)	N/E
Follow-up duration, months, median (range)	40 (13–135)	62 (37–104)	.016
Aneurysm diameter, mm			
Preprocedure, mean ± SD (range)	59.8 ± 8.6 (41–75.9)	73.4 ± 10.9 (60–97)	< .01
At time of detection, mean ± SD (range)	59.5 ± 10 (42.8–79.9)	77.8 ± 24.7 (41.6–126)	< .01
Last follow-up, mean ± SD (range)	55.9 ± 14.2 (30.1–86)	75.4 ± 22.2 (41.6–117.3)	< .01
Average diameter change*, mm	−3.9	+2.0	.283
Aneurysm rupture, n (%)	1 (4.8)	1 (9.1)	.639
Reintervention, n (%)	9 (42.8)	9 (81.8)	.038

DEL = delayed endoleak; EEL = early endoleak; N/E = not evaluated.
*Average diameter change = Last diameter − preprocedure diameter.

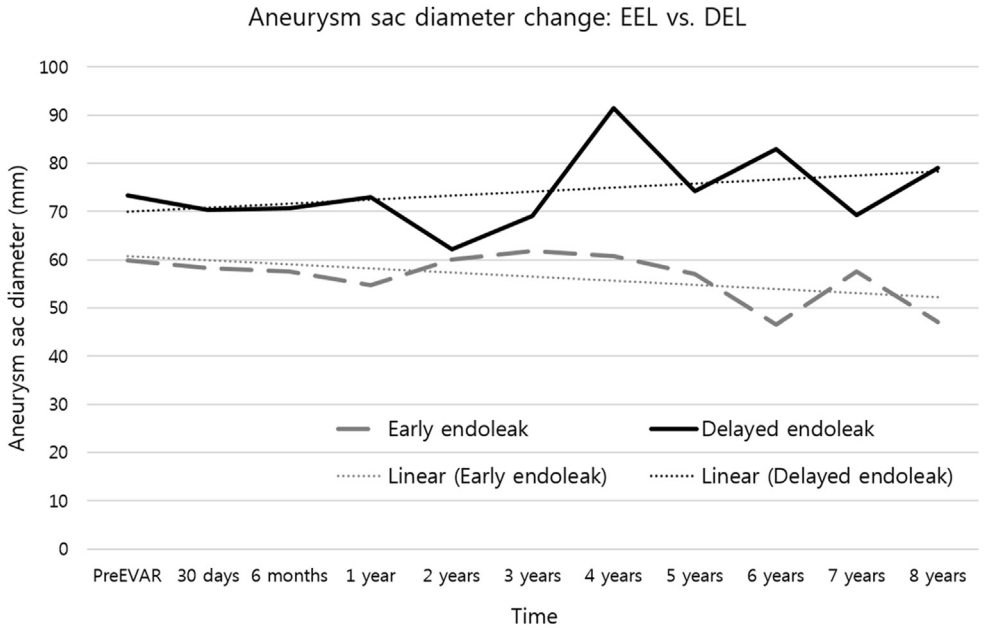

Figure 1. Comparison of aneurysm sac diameter between the EEL group and DEL group. Across any checkpoints, the DEL group had a higher mean aneurysm sac diameter than the EEL group. At the latest follow-up, the average sac diameter of the DEL group slightly increased 2 mm, whereas the average sac diameter of the EEL group decreased 3.9 mm. Dotted lines indicate trends of sac diameter changes over time.

(range, 15–60 months), and median follow-up duration was 62 months (range, 37–104 months). The DEL group comprised more non–type II endoleaks than the EEL group (56.4% vs 19%, $P = .042$), whereas the EEL group comprised more type II endoleaks than the DEL group (81% vs 45.5%, $P = .043$). In the DEL group, the maximum sac diameter had an average increase of 14.3 mm at the time of diagnosis. At any checkpoints (before procedure, detection timing, last follow-up), the DEL group had higher mean aneurysm sac diameters than the EEL group (73.4 mm ± 10.9 vs 59.8 mm ± 8.6, 77.8 mm ± 24.7 vs 59.5 mm ± 10, and 75.4 mm ± 22.2 vs 55.9 mm ± 14.2). Throughout the cohort, average sac diameter of the DEL group slightly increased 2 mm, whereas average sac diameter of the EEL group decreased 3.9 mm, suggesting different trends of sac diameter change between 2 groups (Fig 1). However, the difference was not statistically significant ($P = .283$). Patients in the DEL group appeared to have more reinterventions and longer follow-up duration than patients in the EEL group. Two patients, 1 in each group, experienced aneurysm ruptures during follow-up. No EVAR-related deaths were recorded.

The subgroups of type II EEL (n = 17) and type II DEL (n = 5) were further analyzed (Table 4). In the type II DEL subgroup, the median sac diameter before the procedure was larger (76.1 mm in type II DEL vs 58.5 mm in type II EEL, $P = .003$). However, no inter-subgroup differences were found in sac diameter (at detection and last follow-up), average diameter change, and follow-up duration. During follow-up of type II endoleak, 47.1% of EEL and 0% of DEL resolved spontaneously, although the difference did not reach statistical significance ($P = .06$). Furthermore, patients in the type II DEL subgroup were probably more prone to reinterventions than patients in the

Table 4. Comparison of Subgroups of Type II Early Endoleak and Type II Delayed Endoleak

Variables	Type II EEL (n = 17)	Type II DEL (n = 5)	*P* Value
Aneurysm diameter, mm			
Preprocedure, median (range)	58.5 (41–75)	76.1 (64–79.7)	.003
At time of detection, median (range)	58.1 (43.5–77.7)	65 (51.1–82.3)	.256
Last follow-up, median (range)	55.7 (35.2–86)	65 (51.1–80.2)	.256
Time of detection, months, median (range)	1 (1–4)	28 (15–60)	N/E
Follow-up duration, months, median (range)	48 (13–135)	62 (37–92)	.210
Spontaneous resolve, n (%)	8 (47.1)	0 (0)	.060
Reintervention, n (%)	5 (29.4)	4 (80%)	.048

DEL = delayed endoleak; EEL = early endoleak; N/E = not evaluated.

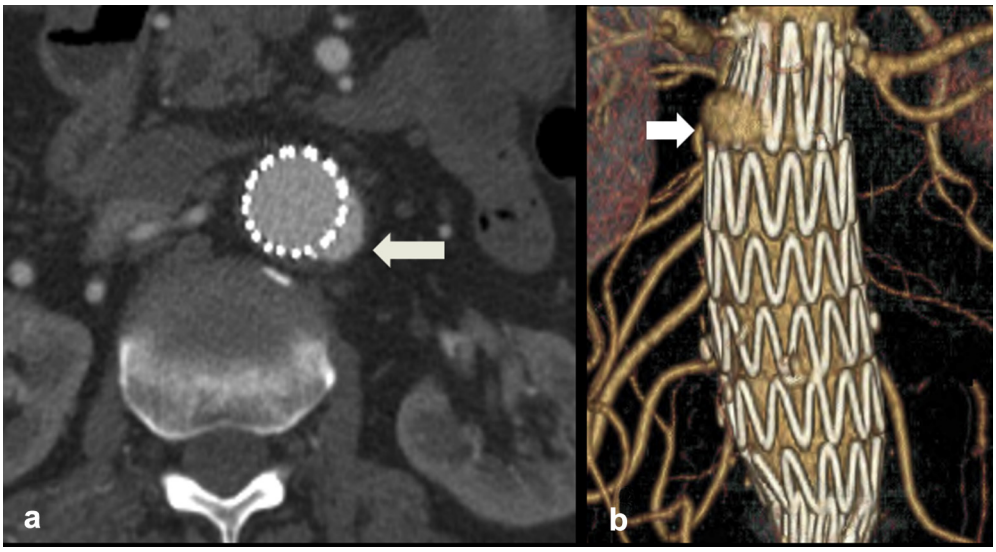

Figure 2. An 80-year-old man received elective EVAR for a hostile neck abdominal aortic aneurysm using the Zenith stent graft. No demonstrable endoleak was detected on the completion angiogram and serial follow-up CT angiograms thereafter. Axial CT angiogram **(a)** and a three-dimensional reconstruction image **(b)** obtained 57 months later demonstrated a small amount of contrast leakage (arrow) at the proximal level of the graft, indicating a type Ia endoleak. Reintervention was not indicated because the aneurysmal sac was not enlarged. The patient died of multiple hepatocellular carcinomas 2 years later with no EVAR-related events.

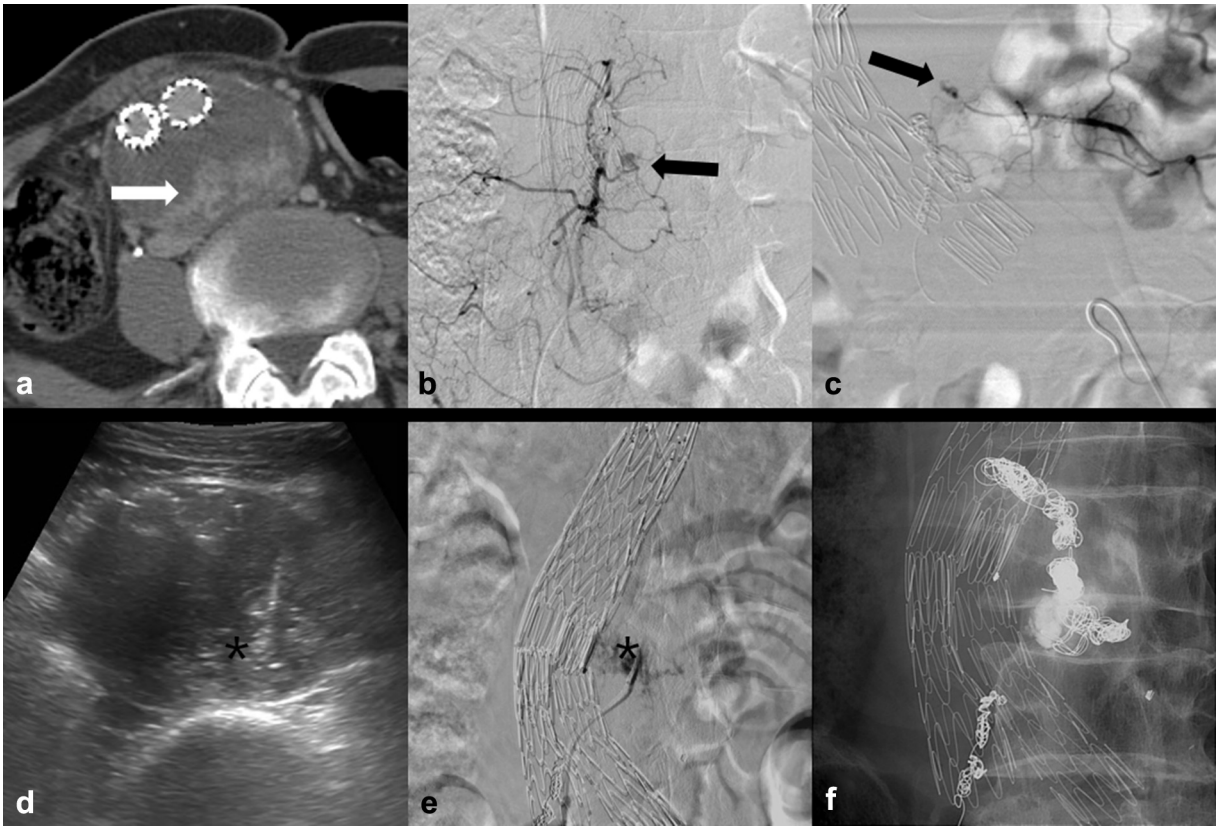

Figure 3. Persistent type II DEL in a 66-year-old man after EVAR. There was no endoleak on either completion angiogram or serial follow-up CT angiograms during the first year. **(a)** A type II DEL (white arrow) was evident on CT angiography 26 months after EVAR. Reintervention was ultimately indicated because of significant sac enlargement noted on serial CT angiograms afterward. **(b, c)** Bilateral internal iliac arteriograms confirmed type II endoleak (black arrow) from lumbar arteries. Selective embolization of the feeding arteries with coils and glue was performed. Endoleak and sac expansion recurred at 84 months. **(d)** Transabdominal ultrasound–guided puncture of the endoleak nidus (asterisk) was performed. **(e)** Contrast medium injection through a 4-F microsheath confirmed the endoleak cavity (asterisk). **(f)** Embolization of the nidus with detachable coils and glue was subsequently performed. However, persistent type II DEL was still evident on contrast-enhanced ultrasound at the latest 90-month visit (not shown).

Table 5. Summary of Delayed Endoleak Group

Sex/Age (y)	Sac Diameter (mm)	Neck Morphology	Stent Graft	DEL			Follow-up Duration (months)	Last Follow-up
				Type	Detection (months)	Management		
M/80	62	Hostile	Zenith	Ia	57	Follow-up	81	Persistent DEL*
M/71	82	Favorable	Zenith	Ia	48	Aortic cuff	50	No endoleak
M/66	79	Hostile	Zenith	II	26	Embolization	92	Persistent DEL
M/64	79.7	Hostile	Endurant	II	28	Embolization	89	Persistent DEL
M/78	64	Hostile	Zenith	II	15	Follow-up	37	Persistent DEL
M/66	65	Hostile	Zenith	II	60	Follow-up	62	Persistent DEL
F/73	76	Favorable	Endurant	II	38	Follow-up	40	Persistent DEL
F/84	50.2	Favorable	Seal	III	34	Graft insertion	60	No endoleak
M/81	71.1	Favorable	Zenith	V	57	Aspiration and relining	104	No endoleak
M/87	60	Hostile	Zenith	V	45	Aspiration	48	No endoleak
M/87	97	Hostile	Zenith	V	57	Follow-up	76	No endoleak

DEL = delayed endoleak; F = female; M = male; N/A = not available.
*Died of hepatocellular carcinoma.

type II EEL subgroup (80% vs 29.4%, $P = .048$). Similarly, different trends of sac diameter change were observed between these 2 subgroups, although no statistical difference was seen.

Overall reinterventions were 18 of 81 (22.2%), including 9 of 21 (42.8%) for EEL and 9 of 11 (81.8%) for DEL. Two patients with non–type II DEL, 1 with minimal type Ia and 1 with suspected endotension, were managed conservatively because of their advanced age, unfavorable general condition, and low life expectancy. The patient with type Ia DEL died of hepatocellular carcinoma 2 years later without evidence of either sac enlargement or EVAR-related events (**Fig 2a**, **b**). The patient with endotension is currently under close monitoring with regular CT angiograms. The latest CT angiogram demonstrated a slight sac regression without a discernible endoleak. One patient developed a type III DEL with 31% sac enlargement 34 months after the initial EVAR. An additional tubular graft and an aortic cuff were promptly placed, and no endoleak has been detected by CT angiography up to 26 months after reintervention. Five patients developed type II DEL during follow-up. Two of these patients presented with progressive sac growth over time, and embolization was ultimately indicated to correct the leakage (**Fig 3a–f**). Of these 2 patients, 1 required 3 embolization procedures. The other 3 patients continued to be followed owing to minimal amount of leakage. **Table 5** summarizes information on DEL.

DISCUSSION

The overall incidence of endoleak (39.5%) in the present study fell in the range of published data (1,8–10). The incidence of DEL was also in keeping with that of other reports (9,10,14,15). In contrast to previous studies, we stratified endoleaks into 2 distinct groups for comparison and clarified the clinical significance of DEL. During > 10 years of follow-up, this study demonstrated that DEL accounted for more than one third (34.3%) of all endoleaks. Furthermore, more than half (54.6%) of DELs were non–type II endoleaks, whereas 80.9% of EELs were type II endoleaks. Correspondingly, the DEL group included more non–type II endoleaks and consequently seemed to have higher rates of reintervention than the EEL group. Basically, non–type II endoleaks require early diagnosis and treatment to exclude risk of aneurysm rupture, whereas type II endoleaks are usually benign and can be managed conservatively if the aneurysm sac is not remarkably enlarged (1,8,14–17). Advocating this strategy, non–type II endoleaks were promptly treated whenever they were identified. Except for 1 patient without reintervention, the overall outcomes were good, as no endoleak was seen, and the aneurysm size remained stable on the latest follow-up images.

As DEL was detected as late as 45 months, a trend of late sac enlargement was observed in the DEL group. The maximum sac diameter of patients with non–type II DEL markedly increased at the time of detection (14.3 mm) requiring prompt and aggressive treatment. This implied not only that type II DEL contributed to aneurysm progression as mentioned by previous works (2,9,10), but that non–type II DEL also played an important role. This finding underscored the necessity of regular imaging follow-up for early diagnosis and management of DEL.

Type II DEL may be considered a marker for high risk and warrant more aggressive subsequent surveillance and management (10). It is postulated that patients with DEL have higher arterial collateral pressure from the inferior mesenteric arteries and lumbar arteries or weaker aneurysm walls prone to dilation (9). However, in the present study, late sac growth of the type II DEL subgroup was not seen as described by other studies (9,10). This was probably due to the small number of type II DELs as well as the fact that long-term imaging follow-up was not available in some patients of this subgroup.

Three cases of endotension were identified at a relatively late median time of 57 months. The exact cause of endotension after EVAR is undefined at the present time (13). However, many theories exist, such as the presence of small, radiologically undetectable endoleaks; pressure transmission from the adjacent endograft lumen or through thrombotic "seals"; and fluid shifts caused by osmosis, fibrinolysis, or enzymatic activity (8,12,13,18). In patients with endotension, continued aneurysm sac growth increases the risk of rupture, leads to enlargement of landing zones, and precipitates stent migration and formation of type I or III endoleaks (8).

The overall reintervention rate (22.2%) of this study was in keeping with previous data (15,17,19). The DEL group tended to have a higher reintervention rate than the EEL group (81.8% vs 42.8%, $P = .038$), which was in accordance with the higher incidence of non–type II endoleaks. Moreover, 47.1% of type II EELs, but 0% of type II DELs, resolved spontaneously during follow-up, which resulted in more reintervention procedures being performed to treat type II DEL than type II EEL (80% vs 29.4%, $P = .048$). Hence, it is hypothesized that type II DELs, once they appear, usually persist and require more reinterventions than type II EELs. Similarly, other studies demonstrated that EEL had earlier and higher rates of spontaneous resolution than late and persistent type II endoleak (10,14). Persistent endoleak may cause continuous pressurization of the aneurysm sac and ultimately lead to sac expansion and possible rupture (16,17). Accordingly, frequent and long-term imaging surveillance of DEL is strongly recommended.

Limitations of the present study include its retrospective design and relatively small population. The rate of follow-up loss was relatively high (32.9%), although still comparable with other reports (5,9,20). This might be partially attributed to the narrow and strict inclusion criteria. With respect to the definition of DEL, patients who received EVAR within 1 year and patients without $\geq$ 12 months of imaging follow-up were excluded despite their continuous visits at the outpatient clinic. Potentially, this dropout may have a negative impact on the estimation of endoleak prevalence.

In conclusion, this study highlighted the frequency and clinical significance of DEL among patients undergoing EVAR who had long-term follow-up. The prevalence of DEL was noteworthy, and the average time of detection was relatively late. More than half of DELs were non–type II, which necessitated early diagnosis and treatment. DEL appeared to have a trend of late sac growth and more reinterventions. Meticulous long-term imaging surveillance to identify and manage DEL, even in the endoleak-free setting (patients without a history of endoleak and patients with treated endoleak), is crucial.

ACKNOWLEDGMENTS

This study was supported by an Inha University Research Grant.

REFERENCES

1. Pippin K, Hill J, He J, Johnson P. Outcomes of type II endoleaks after endovascular abdominal aortic aneurysm (AAA) repair: a single-center, retrospective study. Clin Imaging 2016; 40:875–879.
2. Okada M, Handa N, Onohara T, et al. Late sac behavior after endovascular aneurysm repair for abdominal aortic aneurysm. Ann Vasc Dis 2016; 9:102–107.
3. Ilyas S, Shaida N, Thakor AS, Winterbottom A, Cousins C. Endovascular aneurysm repair (EVAR) follow-up imaging: the assessment and treatment of common postoperative complications. Clin Radiol 2015; 70:183–196.
4. Patel R, Sweeting MJ, Powell JT, Greenhalgh RM. Endovascular versus open repair of abdominal aortic aneurysm in 15-years' follow-up of the UK endovascular aneurysm repair trial 1 (EVAR trial 1): a randomised controlled trial. Lancet 2016; 388:2366–2374.
5. Antoniou GA, Georgiadis GS, Antoniou SA, et al. Late rupture of abdominal aortic aneurysm after previous endovascular repair: a systematic review and meta-analysis. J Endovasc Ther 2015; 22:734–744.
6. Brown A, Saggu GK, Bown MJ, Sayers RD, Sidloff DA. Type II endoleaks: challenges and solutions. Vasc Health Risk Manag 2016; 12:53–63.
7. Sidloff DA, Stather PW, Choke E, Bown MJ, Sayers RD. Type II endoleak after endovascular aneurysm repair. Br J Surg 2013; 100:1262–1270.
8. Green N, Sidloff DA, Stather PW, Bown MJ, Sayers RD, Choke E. Endoleak after endovascular aneurysm repair: current status. Rev Vasc Med 2014; 2:43–47.
9. Zhou W, Blay E Jr, Varu V, et al. Outcome and clinical significance of delayed endoleaks after endovascular aneurysm repair. J Vasc Surg 2014; 59:915–920.
10. Lal BK, Zhou W, Li Z, et al. Predictors and outcomes of endoleaks in the Veterans Affairs Open Versus Endovascular Repair (OVER) trial of abdominal aortic aneurysms. J Vasc Surg 2015; 62:1394–1404.
11. Nakai M, Ikoma A, Sato H, Sato M, Nishimura Y, Okamura Y. Risk factors associated with late aneurysmal sac expansion after endovascular abdominal aortic aneurysm repair. Diagn Interv Radiol 2015; 21:195–201.
12. Chaikof EL, Blankensteijn JD, Harris PL, et al. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg 2002; 35:1048–1060.
13. Blackwood S, Mix D, Chandra A, Dietzek AM. A model to demonstrate that endotension is a nonvisualized type I endoleak. J Vasc Surg 2016; 64:779–787.
14. Sidloff DA, Gokani V, Stather PW, Choke E, Bown MJ, Sayers RD. Type II endoleak: conservative management is a safe strategy. Eur J Vasc Endovasc Surg 2014; 48:391–399.
15. Walker J, Tucker LY, Goodney P, et al. Type II endoleak with or without intervention after endovascular aortic aneurysm repair does not change aneurysm-related outcomes despite sac growth. J Vasc Surg 2015; 62:551–561.
16. Silverberg D, Baril DT, Ellozy SH, et al. An 8-year experience with type II endoleaks: natural history suggests selective intervention is a safe approach. J Vasc Surg 2006; 44:453–459.
17. Lo RC, Buck DB, Herrmann J, et al. Risk factors and consequences of persistent type II endoleaks. J Vasc Surg 2016; 63:895–901.
18. Buth J, Harris PL, van Marrewijk C, Fransen G. The significance and management of different types of endoleaks. Semin Vasc Surg 2003; 16:95–102.
19. Sakata M, Takehara Y, Katahashi K, et al. Hemodynamic analysis of endoleaks after endovascular abdominal aortic aneurysm repair by using 4-dimensional flow-sensitive magnetic resonance imaging. Circ J 2016; 80:1715–1725.
20. AbuRahma AF, Yacoub M, Hass SM, et al. Compliance of post-endovascular aortic aneurysm repair imaging surveillance. J Vasc Surg 2016; 63:589–595.