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ORIGINAL RESEARCH

Type II Endoleak after Endovascular Aneurysm Repair Using the EXCLUDER Stent Graft System in Patients with Abdominal Aortic Aneurysm

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Abstract:
Purpose: This study aimed to evaluate type II endoleak incidence and its outcome in patients who underwent endovascular aneurysm repair using the EXCLUDER device for abdominal aortic aneurysm.
Material and Methods: One hundred sixty-seven patients who underwent endovascular aneurysm repair for abdominal aortic aneurysm (96 with patent and 71 with occluded inferior mesenteric artery) between 2008 and 2017 were retrospectively evaluated. Type II endoleak incidence and aneurysm enlargement of >5 mm after endovascular aneurysm repair were evaluated. The predictive factors for late type II endoleak identified >6 months after endovascular aneurysm repair and aneurysm enlargement were assessed based on the preoperative patient and anatomical characteristics.
Results: Late type II endoleak incidence was higher in the patent inferior mesenteric artery at 42.7% (41/96; 95% confidence interval, 33.3-52.7), compared with 22.5% (16/71; 95% confidence interval, 13.5-34.0) in the occluded inferior mesenteric artery group ($p = 0.01$). Freedom from aneurysm sac enlargement at 1, 3, and 5 years was 100%, 85.0%, and 68.1% in the patent inferior mesenteric artery and 98.9%, 86.7%, and 73.9% in the occluded inferior mesenteric artery group, respectively ($p = 0.22$). Freedom from aneurysm sac enlargement at 1, 3, 5 years was 100%, 76.9%, 43.5%, and 99.1%, 90.6% and 87.8% in the patients with and without late type II endoleak ($p < 0.01$). Patent inferior mesenteric artery (odds ratio, 3.43; 95% confidence interval, 1.43-8.21) and an increasing number of patent lumbar arteries (odds ratio, 2.14; 95% confidence interval, 1.48-3.08) were risk factors for late type II endoleak.
Conclusions: Patent inferior mesenteric artery was a risk for late type II endoleak without contributing to aneurysm enlargement after endovascular aneurysm repair using the EXCLUDER. Late type II endoleak was associated with aneurysm enlargement. Patent inferior mesenteric artery and an increasing number of patent lumbar arteries were risk factors for late type II endoleak.

Keywords:
type II endoleak, inferior mesenteric artery, aortic aneurysm, endovascular aortic aneurysm repair

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Introduction

Endovascular aneurysm repair (EVAR) is a widely accepted standard treatment for abdominal aortic aneurysm (AAA) owing to the lower incidence of perioperative mortality and complications than open surgery. However, EVAR resulted in higher rates of aneurysm-related complications and mortality during the long-term follow-up than did open repair in a randomized controlled trial [1].

Type II endoleaks (EL-II) are defined as a retrograde backflow into the aneurysm sac from the aortic side branches, such as the inferior mesenteric artery (IMA) or lumbar arteries (LAs), after EVAR. Most cases of early EL-II that occur during the perioperative period resolve spontaneously. However, late EL-II, which is identified >6 months after EVAR, can result in worse outcomes owing to aneu-

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rysm sac enlargement during the follow-up period [2, 3]. Reintervention is recommended if aneurysm enlarges to >5 mm or >10 mm after EVAR [4, 5]. Endovascular treatments, including TAE or translumbar embolization, are recommended for EL-II. However, the efficacy of these treatments remains controversial, as approximately 60% of aneurysms continue to expand even after embolization, requiring reintervention or open conversion [6, 7].

Some researchers advocate preventing EL-II incidence before EVAR rather than treating it after EVAR. Pre-emptive IMA embolization during EVAR is widely accepted owing to its high technical success rate of >85%, prevention of EL-II, and promotion of aneurysmal sac reduction after EVAR [8-12]. However, previous studies on pre-emptive IMA embolization utilized various EVAR devices, which may have influenced the results. The use of EXCLUDER stent graft (W.L. Gore & Associates, Flagstaff, AZ, USA) as an EVAR device yielded a higher incidence of EL-II than did other devices [13-15]. Therefore, evaluating the outcome of pre-emptive IMA embolization using a single EVAR device is essential.

A previous study on EL-II incidence and outcome after EVAR using the ENDURANT stent graft (Medtronic Vascular, Santa Rosa, CA, USA) for AAA revealed that IMA occlusion or embolization can reduce EL-II incidence and aneurysm sac enlargement [16]. However, no similar studies have been conducted using the EXCLUDER system. This study aimed to evaluate EL-II incidence and its outcomes after EVAR using the EXCLUDER system for AAAs. The hypothesis was that an occluded or embolized IMA before EVAR would be associated with fewer EL-II incidence rates and aneurysm sac enlargement after EVAR.

Material and Methods

Patient selection

The medical records and images of 241 patients who underwent EVAR for unruptured AAA using the EXCLUDER device between August 2008 and December 2017 at 2 institutions were retrospectively reviewed. 32 patients who underwent CT without contrast enhancement (CECT) after EVAR, 24 with a follow-up duration of <6 months, 10 with pre-emptive LA embolization, 7 who developed type I or III endoleaks during follow-up, and 1 without pre-EVAR CECT were excluded. Thus, 167 patients were included in this study. Among the 96 patients with a patent IMA before EVAR, 94 did not undergo pre-emptive IMA embolization, and 2 patients underwent embolization unsuccessfully. Among the 71 patients with occluded IMA before EVAR, 17 underwent pre-emptive IMA embolization, and 54 had spontaneous occlusion of the IMA before EVAR. This study has obtained approval from the Institutional Review Board in both institutions. Written informed consent to participate in this study was waived owing to its retrospective nature.

Preoperative patient and anatomical characteristics

Preoperative patient characteristics, including age, sex, medical history, smoking history, and use of antithrombogenic drugs, were extracted from the medical records. Multiple-detector CT scanners (Aquilion ONE™, Canon Medical Systems, Japan or SOMATOM Force™, Siemens Healthcare, Forchheim, Germany) were used to acquire preoperative three-phase CECT. A contrast medium dose of 400 mg I/kg was administered at a flow rate of 3 mL/s using an automatic injection system. Bolus tracking was employed with the ROI set at the descending aorta. Arterial phase images were obtained 5 s after the contrast enhancement within the ROI reached a threshold of 200 Hounsfield Units. Delayed phase images were obtained 120 seconds after the acquisition of arterial phase images. Data from 1-mm slices in the arterial phase, such as the patency and diameter of the aortic side branches, were used to evaluate anatomical eligibility for EVAR. Aneurysm diameter was defined as the maximum short diameter for fusiform aneurysms and the maximum long diameter for saccular aneurysms on axial images. The proximal neck diameter was measured as the shortest diameter in the perpendicular plane. The proximal neck length was measured on oblique coronal images parallel to the proximal neck of the aneurysm. Patent LAs originating from the aneurysmal sac were counted; the maximum short diameter in the axial plane was measured for IMA and LA. Two interventional radiologists with 8 and 15 years of experience performed imaging analysis using Ziosoft 2 software (Ziosoft, Tokyo, Japan).

Pre-emptive IMA embolization before EVAR

Pre-emptive IMA embolization was performed at our institutions between 2014 and 2017. A patent IMA is generally indicated for pre-emptive embolization. However, the patients were selected to undergo IMA embolization at the operator's discretion in this study. A 4-F J-curve-type catheter or a 5-F shepherd-hook-type catheter was used to selectively catheterize IMA. The microcatheter was coaxially inserted into the main trunk of the IMA and embolized immediately proximal to the left colonic artery using a detachable coil, followed by several pushable coils. Embolization was performed until near stasis of the antegrade blood flow was achieved.

Follow-up after EVAR

Follow-up CT was performed at 1 and 6 months after EVAR and subsequently at 6-month intervals. All patients underwent three-phase CECT 1-month post-EVAR. Patients who presented with EL-II at 1 month underwent CECT 6-months after EVAR. Patients without EL-II at 1 month underwent non-CECT at 6-month intervals. However, patients underwent CECT if aneurysm enlargement was identified after EVAR. EL-II was defined as contrast enhancement in the aneurysm sac through the aortic side branches on CECT without signs of any other endoleak type. Early EL-II was defined as EL-II detected 1 month after EVAR. Persistent

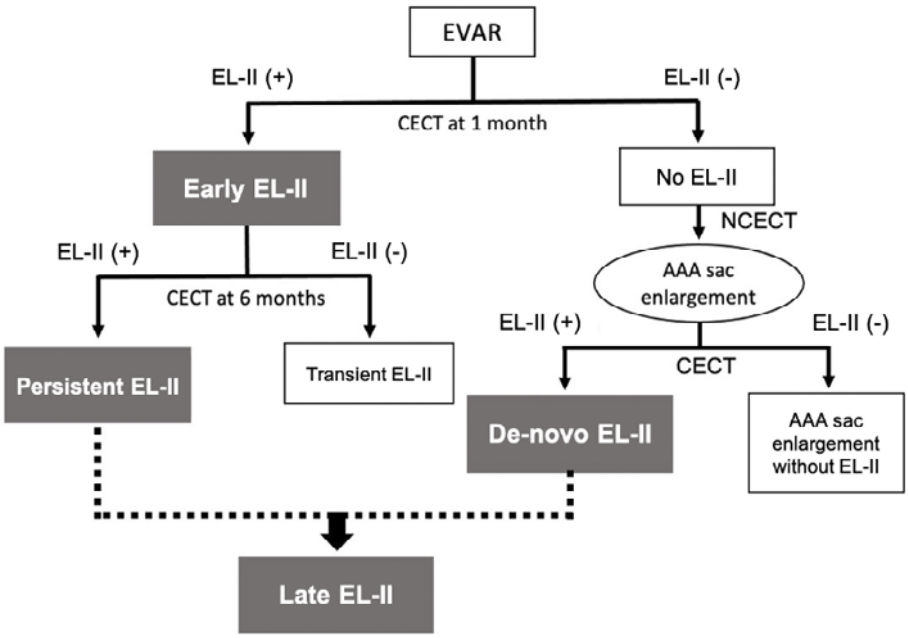

Figure 1. Flowchart showing the follow-up imaging and details of type II endoleaks after EVAR.
CECT: contrast-enhanced computed tomography; EL-II: type II endoleak; EVAR, endovascular abdominal aortic aneurysm repair

EL-II was defined as EL-II detected at 1 month, persisting for >6 months after EVAR. De novo EL-II was defined as EL-II that was not detected at 1 month but detected at >6 months after EVAR. Late EL-II was defined as persistent EL-II or de novo EL-II. The follow-up imaging protocol and definition of EL-II have been depicted in **Fig. 1**.

TAE for EL-II

TAE for EL-II was indicated for the patients with aneurysm sac enlargement >5 mm due to late EL-II after EVAR. However, not all patients with aneurysm sac enlargement underwent reintervention. EL-II via IMA was approached by accessing the middle colic artery through the superior mesenteric artery. EL-II via the LAs was approached by accessing the iliolumbar arteries via the internal iliac artery and retrograde cannulating the LAs. The endoleak nidus and the feeding arteries were embolized using N-butyl-2-cyanoacrylate (Histoacryl; B.Braun, Melsungen, Germany) and iodized oil (Lipiodol; Guerbet, Aulnay-sous-Bois, France) glue or metallic coils. Details of the procedures have been previously described in details [6].

Outcomes

The primary outcome was EL-II incidence. The secondary outcomes were aneurysm sac enlargement of >5 mm due to EL-II after EVAR, reintervention for EL-II including TAE and open conversion. The predictive risk factors for late EL-II were evaluated based on preoperative patient and anatomical characteristics. The reintervention results for EL-II were also reviewed. The success of TAE for EL-II was defined as preventing aneurysm sac enlargement of 5 mm or more after TAE.

Statistical analysis

Preoperative patient characteristics, anatomical characteristics, and operative details are summarized using descriptive statistics. Categorical variables were expressed as raw numbers, proportions, and percentages. Continuous variables are presented as means (± standard deviations). EL-II incidence is presented as a two-sided 95% CI. Freedom from aneurysm sac enlargement was assessed using the Kaplan-Meier analysis, and the log-rank test was used to compare patients with patent and occluded IMA, with and without persistent EL-II, and with and without late EL-II. Univariate logistic regression was performed using preoperative patient and anatomical characteristics to identify risk factors for late EL-II and calculate the ORs and 95% CI. Multivariate analysis with adjustments for confounding variables was not performed owing to the small sample size. Statistical significance was set at $p < 0.05$. Statistical analysis was performed using the EZR software (Saitama Medical Center, Jichi Medical University, Saitama, Japan).

Results

Patient characteristics

Patient characteristics are presented in **Table 1**. The median follow-up period was 59.5 ± 26.2 months in the patent IMA group and 52.9 ± 28.0 months in the occluded IMA group ($p = 0.121$). Patients with patent IMA had a higher number of current smokers (32.3% [31/96] vs. 12.7% [9/71]; $p = 0.003$) and were significantly more likely to receive anticoagulant (19.8% [19/96] vs. 5.6% [4/71]; $p = 0.011$)

Table 1. Patient and Anatomical Characteristics.

Variables	Patent IMA (n = 96)	Occluded IMA (n = 71)	p Value
Age (years)	77.8 ± 6.9	78.6 ± 6.7	0.458
Sex			
Men	70 (70/96; 72.9)	47 (47/71; 66.2)	0.395
Women	26 (26/96; 27.1)	24 (24/71; 33.8)	0.395
Median follow-up, months	59.5 ± 26.2	52.9 ± 28.0	0.121
Hypertension	70 (70/96; 72.9)	46 (46/71; 64.8)	0.309
Hyperlipidemia	40 (40/96; 41.7)	25 (23/71; 35.2)	0.426
Diabetes mellitus	12 (12/96; 12.5)	8 (8/71; 11.3)	1.000
COPD	7 (7/96; 7.3)	8 (8/71; 11.3)	0.420
Coronary artery disease	21 (21/96; 21.9)	8 (8/71; 11.3)	0.098
Cerebrovascular disease	3 (3/96; 3.1)	3 (3/71; 4.2)	0.700
Chronic kidney disease	34 (34/96; 35.4)	16 (16/71; 22.5)	0.088
Current smoker	31 (31/96; 32.3)	9 (9/71; 12.7)	0.003*
Anticoagulant therapy	19 (19/96; 19.8)	4 (4/71; 5.6)	0.011*
Antiplatelet therapy	40 (40/96; 41.7)	18 (18/71; 25.3)	0.033*
Aneurysm sac diameter (mm)	50.5 ± 9.2	50.3 ± 8.0	0.883
Aneurysm shape			
Fusiform	84 (84/96; 87.5)	57 (57/71; 80.3)	0.280
Saccular	12 (12/96; 12.5)	14 (14/71; 19.7)	0.280
Proximal neck diameter (mm)	20.7 ± 3.2	20.6 ± 2.5	0.910
Number of patent LAs	2.5 ± 1.4	2.5 ± 1.5	0.917
Maximum LA diameter (mm)	2.0 ± 0.8	2.2 ± 1.0	0.080
Iliac aneurysm	26 (2/96; 27.1)	15 (15/71; 21.1)	0.468
IIA embolization	33 (33/96; 34.4)	22 (22/71; 31.0)	0.740

COPD: chronic obstructive pulmonary disease; IMA: inferior mesenteric artery; LA: lumbar artery; IIA: internal iliac artery

Qualitative variables are expressed as raw numbers followed by proportions and percentages in parentheses. Quantitative variables are expressed as means ± standard deviations.

*p < 0.05.

Table 2. Incidence of EL-II after EVAR Using the EXCLUDER Stent-Graft System.

Variables	All (N = 167)	Patent IMA (n = 96)	Occluded IMA (n = 71)	p Value
Overall	75 (75/167; 44.9)	50 (50/96; 52.1)	25 (25/71; 35.2)	0.041*
Early	45 (45/167; 26.9)	31 (31/96; 32.3)	14 (14/71; 19.7)	0.080
Persistent	27 (27/167; 16.2)	22 (22/96; 22.9)	5 (5/71; 7.0)	0.006*
De-novo	30 (30/167; 18.0)	19 (19/96; 19.8)	11 (11/71; 15.5)	0.544
Late EL-II	57 (57/167; 34.1)	41 (41/96; 42.7)	16 (16/71; 22.5)	0.008*

EL-II: type II endoleak; IMA: inferior mesenteric artery

Data are expressed as raw numbers followed by proportions and percentages in parentheses.

*p < 0.05.

and antiplatelet therapy (41.7% [40/96] vs. 25.3% [18/71]; p = 0.033) compared with the occluded IMA group. Anatomical factors were not significantly different between the groups.

EL-II incidence and origin of late EL-II

Table 2 summarizes EL-II incidence in patients with patent and occluded IMA. The overall incidence of EL-II was 44.9% (75/167; 95% CI, 37.2-52.8). The incidence of persistent EL-II was significantly higher in patients with a patent IMA at 22.9% (22/96; 95% CI, 14.7-31.2) compared with 7.0% (5/71; 95% CI, 2.3-15.7) in patients with an occluded IMA (p = 0.006). The incidence of late EL-II was higher in the patent IMA group at 42.7% (41/96; 95% CI, 33.3-52.7), compared with 22.5% (16/71; 95% CI, 13.5-34.0) in the occluded IMA group (p = 0.008). Among 57 patients with late EL-II, EL-II origin was isolated IMA in 7 (7/57; 12.3%), the isolated LAs in 40 (40/57; 70.2%), and both IMA and LAs in 10 (10/57; 17.5%) (**Table 3**).

Aneurysm sac enlargement

Aneurysm sac enlargement was observed in 50 (50/167; 29.9%) of 167 patients, including 34 (34/96; 35.4%) of the patients with patent IMA, and 16 (16/71; 22.5%) of the patients with occluded IMA during the follow-up period. Freedom from aneurysm sac enlargement at 1, 3, and 5 years

Table 3. Details on Late EL-II Origin and Its Outcomes.

Variables	All (N = 57)	IMA (n = 7)	LAs (n = 40)	Both IMA and LAs (n = 10)
Aneurysm sac enlargement	33 (33/57; 57.9)	4 (4/7; 57.1)	20 (20/40; 50.0)	9 (9/10; 90.0)
TAE for EL-II	23 (23/57; 40.4)	3 (3/7; 42.9)	11 (11/40; 27.5)	9 (9/10; 90.0)
TAE success	11 (11/23; 47.8)	2 (2/3; 66.7)	4 (4/11; 36.4)	5 (5/9; 55.6)
TAE failure	12 (12/23; 52.2)	1 (1/3; 33.3)	7 (7/11; 63.6)	4 (4/9; 44.4)

EL-II: type II endoleak; IMA: inferior mesenteric artery; LA: lumbar artery

Data are expressed as raw numbers followed by proportions and percentages in parentheses.

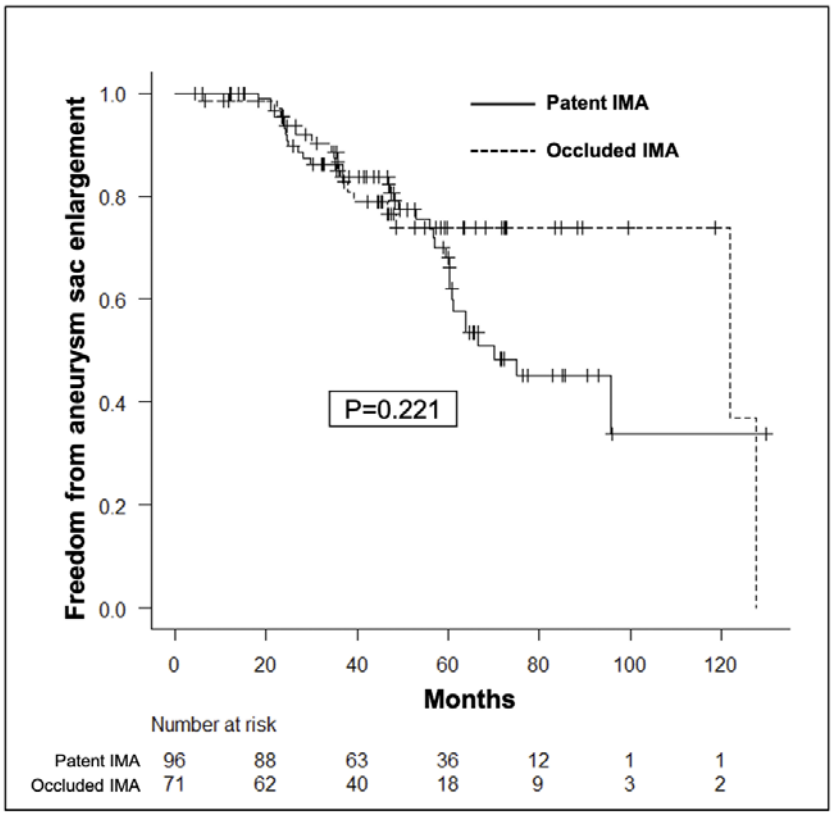

Figure 2. Kaplan–Meier analysis of freedom from aneurysm sac enlargement in patients with patent and occluded inferior mesenteric artery.
IMA: inferior mesenteric artery

was 100%, 85.0%, and 68.1% in the patients with patent IMA, and 98.9%, 86.7%, and 73.9% in the patients with occluded IMA, respectively; there was no significant difference between the 2 groups (p = 0.221; **Fig. 2**). Freedom from aneurysm sac enlargement at 1, 3, 5 years was 100%, 75.0%, 33.3% in patients with persistent EL-II, and 99.3%, 87.8%, and 79.5% in the patients without persistent EL-II (p < 0.001) (**Fig. 3A**). Similarly, freedom from aneurysm sac enlargement at 1, 3, and 5 years was 100%, 76.9%, 43.5% in the patients with late EL-II, and 99.1%, 90.6%, and 87.8% in the patients without late EL-II (p < 0.001) (**Fig. 3 B**). Among the 57 patients with late EL-II, aneurysm sac enlargement was observed in 33 patients (33/57; 57.9%), including 4 patients (4/7; 57.1%) with IMA-related EL-II, 20 patients (20/40; 50.0%) with LAs-related EL-II, and 9 patients (9/10; 90.0%) with both IMA and LAs-related late EL-II (**Table 3**).

TAE outcome for late EL-II

Among the 57 patients with late EL-II, 23 (23/57, 40.4%) underwent TAE for late EL-II; this included 3 patients (3/7; 42.9%) with IMA-related EL-II, 11 patients (11/40; 27.5%) with LAs-related EL-II, and 9 (9/10; 90.0%) with both IMA and LAs-related EL-II (**Table 3**). In 23 patients who underwent TAE, EL-II persisted in 10 (10/23; 43.8%) after the procedures. TAE success was achieved in 11 patients (11/23; 47.8%), whereas it was not achieved in 12 patients (12/23; 52.2%). Subsequently, 6 (6/12; 50.0%) patients who failed TAE underwent open conversion.

Risk factors for late EL-II after EVAR

Table 4 presents the risk factor analysis for late-EL-II. Univariate logistic regression analysis revealed that patent IMA (OR, 3.43; 95% CI, 1.43-8.21; p < 0.001), and an in-

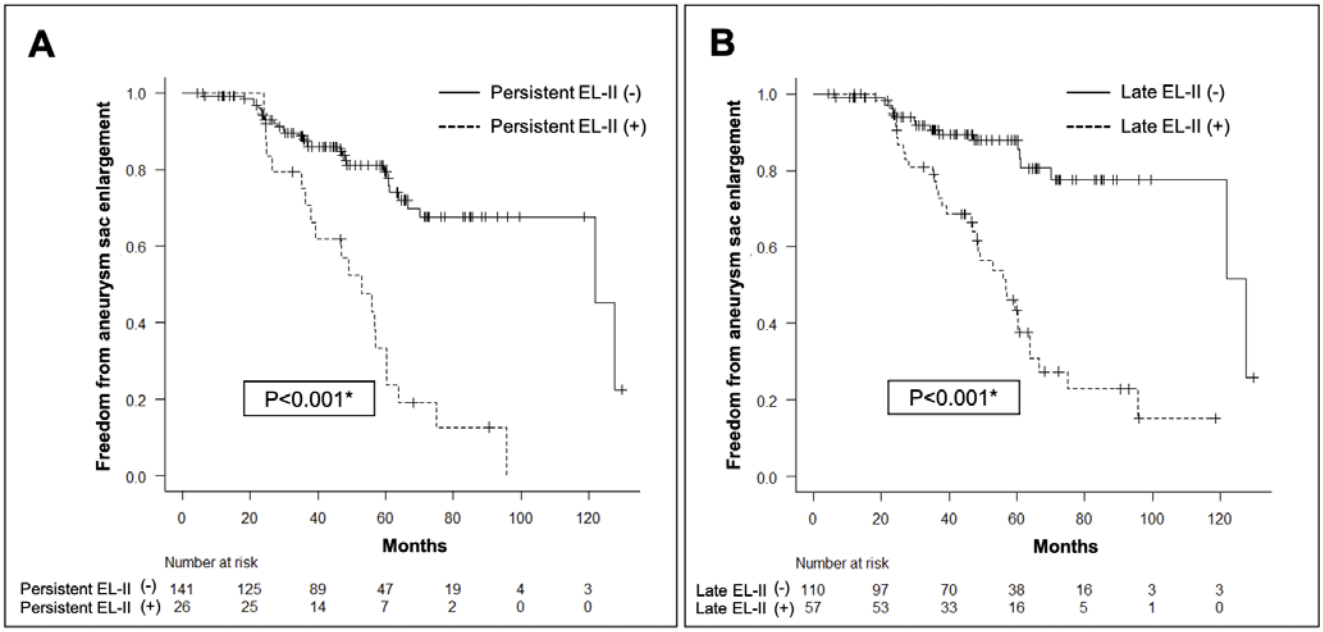

Figure 3. A: Kaplan–Meier analysis of freedom from aneurysm sac enlargement in patients with and without persistent type II endoleak. B: Kaplan–Meier analysis of freedom from aneurysm sac enlargement in patients with and without late type II endoleak.
EL-II: type II endoleak
* p-values less than 0.05.

Table 4. Univariate Comparison of Factors Associated with Late EL-II.

Variables	OR (95% CI)	p-Value
Age	0.93 (0.88-0.99)	0.034*
Sex	2.22 (0.82-6.01)	0.116
Hypertension	1.94 (0.77-4.93)	0.163
Hyperlipidemia	1.51 (0.63-3.63)	0.355
Diabetes mellitus	0.70 (0.21-2.33)	0.561
COPD	1.58 (0.41-6.08)	0.502
Coronary artery disease	0.69 (0.29-1.68)	0.415
Cerebrovascular disease	0.37 (0.04-3.15)	0.363
Chronic kidney disease	0.49 (0.20-1.21)	0.124
Current smoker	1.22 (0.46-3.26)	0.693
Anticoagulant therapy	0.70 (0.22-2.26)	0.551
Antiplatelet therapy	2.10 (0.75-5.87)	0.159
Aneurysm sac diameter	1.01 (0.96-1.06)	0.753
Aneurysm shape	1.92 (0.60-6.11)	0.270
Proximal neck diameter	1.10 (0.96-1.27)	0.169
Patent IMA	3.43 (1.43-8.21)	<0.001*
Number of patent LAs	2.14 (1.48-3.08)	<0.001*
Maximum LA diameter	1.15 (0.64-2.05)	0.640
Iliac aneurysm	2.36 (0.49-11.5)	0.070
IIA embolization	0.61 (0.21-1.77)	0.359

COPD: chronic obstructive pulmonary disease; EL-II: type-II endoleak; IMA: inferior mesenteric artery; LA: lumbar artery; IIA: internal iliac artery
The OR and 95% CI were calculated using univariate logistic regression analysis.
*Statistical significance was set at $p < 0.05$.

crease in the number of patent LAs (OR, 2.14; 95% CI, 1.48-3.08; $p < 0.001$) were risk factors for late EL-II. Older age (OR, 0.93; 95% CI, 0.88-0.99; $p = 0.034$) was protective factor for late EL-II.

Discussion

This study aimed to investigate the incidence of EL-II and aneurysm sac enlargement after EVAR using a single device (EXCLUDER) in patients with AAA. Although patent IMA was a risk factor for EL-II, it was not associated with aneurysm sac enlargement. Late EL-II was associated with aneurysm sac enlargement, and successful rates of TAE for EL-II were not high. Patent IMAs and increasing number of patent LAs before EVAR was identified as a risk factor for late EL-II.

The differences in EL-II incidence between the types of stent grafts have been discussed. Some researchers have reported a significant correlation between the stent graft type and EL-II incidence [14, 15, 17]. EL-II incidence ranges between 22 and 28% and 41 to 46% after EVAR with ENDURANT and EXCLUDER, respectively [14, 15]. In this study, the overall incidence of EL-II was 44.9%, with an incidence of late EL-II of 34.1% after EVAR using the EXCLUDER system. In a previous ENDURANT study involving 103 patients with occluded IMA, the overall incidence of EL-II was 9.7%, with an incidence of late EL-II of 7.8% [16]. In this study, the overall incidence of EL-II was 35.2%, with an incidence of late EL-II of 22.5% in patients with occluded IMA after EVAR using EXCLUDER. The higher incidence rate of EL-II using the EXCLUDER device can be attributed to the fact that the EXCLUDER, except

for the recent GORE EXCLUDER Conformable AAA Endoprosthesis system (W.L. Gore & Associates), has a short main body. The short main body of the EXCLUDER cannot seal the aortic side branches in patients with AAA and long proximal necks [14]. Another reason could be that the EXCLUDER system has low fabric porosity, which reduces pressure in the aneurysm sac after EVAR, facilitating backflow via the aortic side branches [18, 19]. Thus, the EXCLUDER is associated with a higher incidence of EL-II, requiring more caution in EL-II management than other EVAR devices.

Patent IMA is considered a risk factor for EL-II after EVAR [20, 21]. Numerous studies emphasize the effectiveness of pre-emptive IMA embolization to prevent EL-II and subsequent aneurysm sac enlargement [8-11]. The Japanese guideline for aortic aneurysm have suggested that IMA embolization should be considered at the time of EVAR (class of recommendation; IIa) [22]. Consistent with previous studies, our research confirmed that patent IMA is associated with higher rates of persistent and late EL-II. However, in this study, there was no significant difference in aneurysm sac enlargement between the patent IMA and the occluded IMA group after EVAR. This discrepancy may be due to confounding factors other than IMA-related EL-II contributing to sac enlargement. Recent studies have shown that the pre-emptive IMA embolization did not prevent the aneurysm sac enlargement or reintervention, suggesting that many cases of EL-II leading to sac enlargement and reintervention are attributable to LAs rather than the IMA-related EL-II [23, 24]. Nonetheless, our study observed that aneurysm sac enlargement occurred in 57.1% (4/7) of late EL-II cases involving IMA-related EL-II, and 90.0% (9/10) of cases involving both the IMA and LAs-related EL-II, and in some cases, required reintervention or, open conversion. Our study indicates that IMA-related EL-II results in aneurysm sac enlargement after EVAR and should not be disregarded. Therefore, these findings do not provide sufficient grounds to negate the strategy of pre-emptive IMA embolization before EVAR.

The current guidelines recommend reintervention for EL-II if the aneurysm enlarges by >5 mm or >10 mm during the follow-up period [4, 5]. TAE is the first-line treatment of EL-II after EVAR. However, 33.7%-60% of aneurysms continue to enlarge after TAE [6, 7, 25]. In this study, 52.2% (12/23) of patients who underwent TAE for EL-II experienced aneurysm sac enlargement, consistent with previous studies. Systematic reviews and meta-analyses have shown little conclusive evidence on the efficacy of TAE in EL-II treatment [26]. Some researchers suggest pre-emptive embolization of aortic branches before EVAR to prevent EL-II and aneurysm enlargement and reduce the need for reintervention [27-30]. This study identified a patent IMA and an increase in the number of patent LAs before EVAR as risk factors for late EL-II in AAA patients. Consequently, the study recommends pre-emptive embolization of LAs in addition to IMA to prevent EL-II and contribute to aneurysm sac stabilization after EVAR with the EXCLUDER.

This study has several limitations. First, it was conducted using a single EVAR device, the EXCLUDER, and compared the incidence of EL-II with historical control data from the ENDURANT device. To accurately assess differences in EL-II incidence between devices, direct comparisons within cohorts with similar patient backgrounds are necessary. The second limitation was its retrospective design. Selective bias exists regarding patients undergoing preemptive IMA embolization or secondary intervention for EL-II after EVAR, which can affect the study outcomes. Additionally, the significantly high rates of current smoking, antiplatelet therapy, and anticoagulation therapy among patients with a patent IMA may contribute to the incidence of EL-II. However, these factors were not identified as risk factors for EL-II in this study. Finally, not all patients underwent regular CTA or ultrasound examinations to evaluate blood flow within the aneurysm during the follow-up period, possibly leading to an underestimation of endoleak. For example, patients without early EL-II who did not undergo CECT might have developed late EL-II. Therefore, further prospective and direct comparative studies are needed to address these issues.

In conclusion, EL-II incidence after EVAR using the EXCLUDER for AAA with IMA was high. Patent IMA before EVAR is a significant risk factor for late EL-II. However, it is not associated with aneurysm sac enlargement after EVAR using the EXCLUDER device. Late EL-II, regardless of the origin of the aortic side branches, was associated with aneurysm sac enlargement; success rates of TAE were not high in preventing aneurysm sac enlargement. Pre-emptive embolization of LAs in addition to IMA may prevent late EL-II and aneurysm sac enlargement after EVAR using the EXCLUDER.

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