Vascular Specialist International
pISSN 2288-7970 • eISSN 2288-7989
(2025) 41:10

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Original Article

Effects of Pre-emptive Aortic Side Branch Embolization on Early-Stage Type II Endoleaks and Sac Changes After Endovascular Aneurysm Repair for Abdominal Aortic Aneurysm

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Purpose: This study evaluated the effect of pre-emptive embolization of the aortic side branches on the short-term incidence of type II endoleaks (T2ELs) and aneurysm sac changes following endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms (AAAs).

Materials and Methods: Data of 157 patients with degenerative large AAAs (≥50 mm for males and ≥45 mm for females) treated between January 2019 and October 2024 were retrospectively analyzed. The patients were categorized into the pre-emptive embolization (n=30, 19.1%) and non-embolization (n=127) groups. Embolization was considered for patients with high-risk factors for T2EL, specifically an inferior mesenteric artery (IMA) diameter ≥3 mm and a large lumbar artery diameter. Outcomes were assessed 1 month and 1 year postoperatively. The primary outcome was T2EL incidence, and the secondary outcomes were changes in aneurysm size and reintervention rates.

Results: At 1 month postoperatively, T2EL incidence was significantly lower in the embolization group (3.3%, 1/30) than in the non-embolization group (22.8%, 29/127) (P=0.015). At 1 year postoperatively, the T2EL incidence was 8.0% (2/25) in the embolization group and 23.0% (23/100) in the non-embolization group, although the difference was not statistically significant (P=0.094). Sac expansion was absent in the embolization group, whereas 4.0% of patients in the non-embolization group experienced expansion (P=0.583). Subgroup analysis for 37 patients with patent IMA ≥3 mm showed a significantly lower T2EL incidence at 1 month (5.6% vs. 52.6%, P=0.002) and 1 year (12.5% vs. 52.9%, P=0.014) postoperatively in the embolization group. Additionally, sac shrinkage was higher in the embolization group than in the non-embolization group (56.2% vs. 23.5%, P=0.055). Multivariable analysis confirmed pre-emptive embolization as an independent protective factor for T2EL at 1 year postoperatively (odds ratio 0.071, 95% confidence interval, 0.008-0.663; P=0.020).

Conclusion: Pre-emptive embolization reduced the incidence of early T2EL after EVAR and showed stable outcomes at the 1-year follow-up, particularly in patients with large patent IMA diameters.

Key Words: Abdominal aortic aneurysm, Endovascular aneurysm repair, Embolization, Endoleak, Preoperative care

Received February 5, 2025
Revised April 12, 2025
Accepted April 15, 2025
Published on May 15, 2025

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Cite this article; Vasc Specialist Int 2025. https://doi.org/10.5758/vsi.250010

INTRODUCTION

During open surgical repair for abdominal aortic aneurysms (AAAs), the inferior mesenteric artery (IMA) and lumbar arteries (LAs) are typically ligated to control bleeding, thereby eliminating the risk of type II endoleak (T2EL), a common complication associated with endovascular aneurysm repair (EVAR). By contrast, standard EVAR leaves the aortic side branches, including the IMA and LAs, which can contribute to T2EL development.

Although T2ELs often resolve spontaneously, they persist in 15% to 30% of cases [1,2]. The long-term consequences of persistent T2EL remain controversial, but they have been associated with sac expansion, secondary type I endoleak (T1EL) owing to seal zone failure, reintervention, and in rare cases, aneurysm rupture or death [2-6]. The ENGAGE registry, a large-scale multicenter study of EVAR outcomes, revealed a 15.6% incidence of T2EL over 5 years; 11% of these cases progressed to late T1EL [1]. Similarly, a Japanese registry identified persistent T2EL as a contributing factor in 30.7% of the T1EL cases [7]. A nationwide study involving more than 17,000 Japanese patients demonstrated significantly higher AAA-related mortality and rupture rates in patients with persistent T2EL, underscoring its potential severity [2].

Hence, pre-emptive embolization of the aortic side branches has been proposed to mitigate the risk of persistent T2EL and its complications. Several studies have indicated that pre-emptive embolization reduces T2EL incidence and the need for reintervention [3,4,8]. However, other studies did not show the advantages of pre-emptive embolization, including aneurysmal sac shrinkage or reintervention rates [9,10]. Additionally, evidence is limited owing to the lack of randomized controlled trials (RCTs) [4], and information supporting pre-emptive embolization remains sparse. Thus, this study evaluated the short-term incidence of T2EL and clinical outcomes in patients undergoing EVAR for AAAs, comparing those who underwent pre-emptive embolization of the aortic side branches with those who did not.

MATERIALS AND METHODS

1) Study design and data collection

This retrospective study was approved by the Institutional Review Board (IRB) of Kyungpook National University Chilgok Hospital (IRB No. 2024-10-031), with a waiver of the requirement for informed consent. Between January 2019 and October 2024, 236 patients with aortoiliac aneurysms underwent EVAR. Patients were excluded if they presented with isolated iliac artery aneurysms (n=22), iliac artery aneurysms combined with a small AAA of <50 mm in males and <45 mm in females (n=15), or a ruptured AAA requiring urgent EVAR (n=12). Cases involving infected AAA (n=5), aneurysms associated with vasculitis (e.g., Behçet's disease, Takayasu's arteritis) (n=2), suprarenal AAA (n=3), penetrating aortic ulcer-induced saccular aneurysms (n=13), and no follow-up images at 1 month (n=7) were also excluded. After these exclusions, 157 patients with degenerative fusiform AAAs of ≥50 mm in males and ≥45 mm in females were included in the analysis.

At our institution, pre-emptive embolization was introduced in 2019 and initially limited to the IMA. In 2020, the indications were expanded to include LAs. During the study period, pre-emptive embolization and EVAR were performed at two tertiary centers. At one center, embolization was conducted by an interventional radiologist, typically 1 week prior to EVAR, which was performed by a vascular surgeon. At the other center, embolization and EVAR were performed concurrently by a vascular surgeon.

Medical records and preoperative and postoperative imaging data were comprehensively reviewed to obtain patient characteristics, imaging findings, procedural details, and follow-up outcomes. The demographic data included age, sex, and comorbidities. Preoperative anatomic characteristics included aneurysm diameter, IMA patency and diameter, and the number of patent LAs originating from the infrarenal aorta and aneurysmal sac. Postoperative data included the presence and type of endoleaks and the aneurysm sac diameter during follow-up. The procedural details recorded were technical success of embolization, materials used for embolization, whether IMA and LA embolization were performed, number of embolized LAs, and total operation and fluoroscopic times.

2) Indications for pre-emptive embolization and follow-up protocol

In early 2019, indications for IMA embolization were selectively applied to cases in which the patent IMA had a diameter of ≥3.0 mm, based on prior studies identifying anatomic risk factors for T2EL [11]. Since 2020, the indications were expanded to include LAs with diameters >2.5 mm that originated from the aneurysmal sac, for which embolization was considered [12]. However, the decision to perform pre-emptive embolization was ultimately guided by the surgeon's clinical judgment, considering patient age, comorbidities, aneurysm diameter, and lengths of the proximal and distal landing zones. Pre-emptive embolization was more actively pursued in patients requiring long-term follow-up to reduce the risk of late T2EL-related complications.

The typical postoperative follow-up protocol included duplex ultrasonography for detecting clinically significant endoleaks (e.g., T1EL or type III endoleak [T3EL]) before discharge; computed tomography angiography (CTA) with a delayed phase at 1 month; clinical evaluation at 6 months; CTA at 1 year; and annual CTA thereafter. Among patients with renal insufficiency, duplex ultrasonography for detecting endoleaks, combined with non-contrast CT for determining the sac diameter, served as an alternative to CTA. At each follow-up visit, physical examinations were conducted to assess the abdominal and femoral pulsations. If T1EL or T3EL were detected or if T2EL-induced sac expansion exceeding 5 mm occurred, reintervention was considered and discussed based on the patient's comorbidities and overall condition.

3) Study outcomes and definitions

This study evaluated the presence of T2EL and changes in aneurysm sac diameter after EVAR, with or without pre-emptive embolization. The primary outcome was the presence of T2EL at the 1-month and 1-year follow-ups. The secondary outcomes included changes in sac diameter at 1 year post-EVAR and any reintervention during follow-up. In addition, the causes and types of reinterventions were assessed. Changes in the sac diameter were categorized as shrinkage, stability, or expansion. Sac shrinkage was defined as a reduction in aortic sac diameter of ≥5 mm compared with the preoperative measurement, whereas sac expansion was defined as an increase of ≥5 mm [13]. A stable sac was defined as any change between these two thresholds.

Both approaches to pre-emptive embolization, conducted before or simultaneously with EVAR, were classified into the pre-emptive embolization group. Technical failure was defined as the inability to achieve guidewire or catheter passage for the planned embolization of the aortic side branches. For patients who underwent embolization prior to EVAR, the total operative time and fluoroscopic time were calculated by summing the duration of the embolization procedure and the subsequent EVAR procedure. The total operative time was defined as the period from entering the operating room or angiography suite to leaving it, including the anesthesia time.

4) Statistical methods

For comparisons between the embolization and non-embolization groups, Student t-test or Mann–Whitney U-test was used for continuous variables, depending on data normality. Data normality was assessed using the Shapiro–Wilk test. Preoperative age, AAA and IMA diameters, and the number of patent LAs from the infrarenal aorta and aneurysm sac were analyzed using the Mann–Whitney U-test. Total operative time and fluoroscopic time were compared using the Student t-test. The chi-squared test (for adequately sized samples) or Fisher exact test (for smaller samples) was used for categorical variables. Normally distributed continuous variables are presented as means±standard deviations, whereas those with a skewed distribution are expressed as medians with interquartile ranges (IQRs). Complete-case analysis was performed on 1-year outcomes, including only patients with available imaging data at 1 year. No imputation was applied to any missing data. The completion rates for 1-year follow-up imaging were similar between the embolization and non-embolization groups (83.3% vs. 78.7%, P=0.574), reducing the likelihood of differential bias.

Multivariable analysis was performed to assess factors associated with T2EL and sac shrinkage at 1 year in patients with an IMA diameter of ≥3 mm, using binary logistic regression. The variables included in the multivariable model were those with a P≤0.2 in the univariable analysis and those considered clinically relevant. Kaplan–Meier analysis with the log-rank test was conducted to analyze T2EL-related reintervention-free survival according to pre-emptive embolization. All statistical analyses were performed using IBM SPSS Statistics ver. 25.0 (IBM Co.), and significance was set at P<0.05.

RESULTS

1. Baseline characteristics

In total, 157 patients who underwent EVAR for degenerative fusiform AAA during the study period were included in the analysis. Among them, 30 (19.1%) underwent pre-emptive aortic side-branch embolization involving the IMA and/or LAs. Isolated IMA embolization was performed in 9 patients, isolated LA embolization in 10 patients, and both procedures in 11 patients. These 30 patients were classified into the embolization group, and the remaining 127 patients who underwent EVAR without aortic side-branch embolization comprised the non-embolization group (Fig. 1).

Patient characteristics and anatomical details of the AAAs are summarized in Tables 1 and 2, respectively. No significant baseline differences in clinical characteristics were observed between the groups, except for median age, which was lower in the embolization group (median: 73.5 years [IQR 67.0-78.0]) than in the non-embolization group (median: 78.0 years [IQR 71.0-83.0], P=0.004). Regarding anatomical details, the embolization group had a smaller

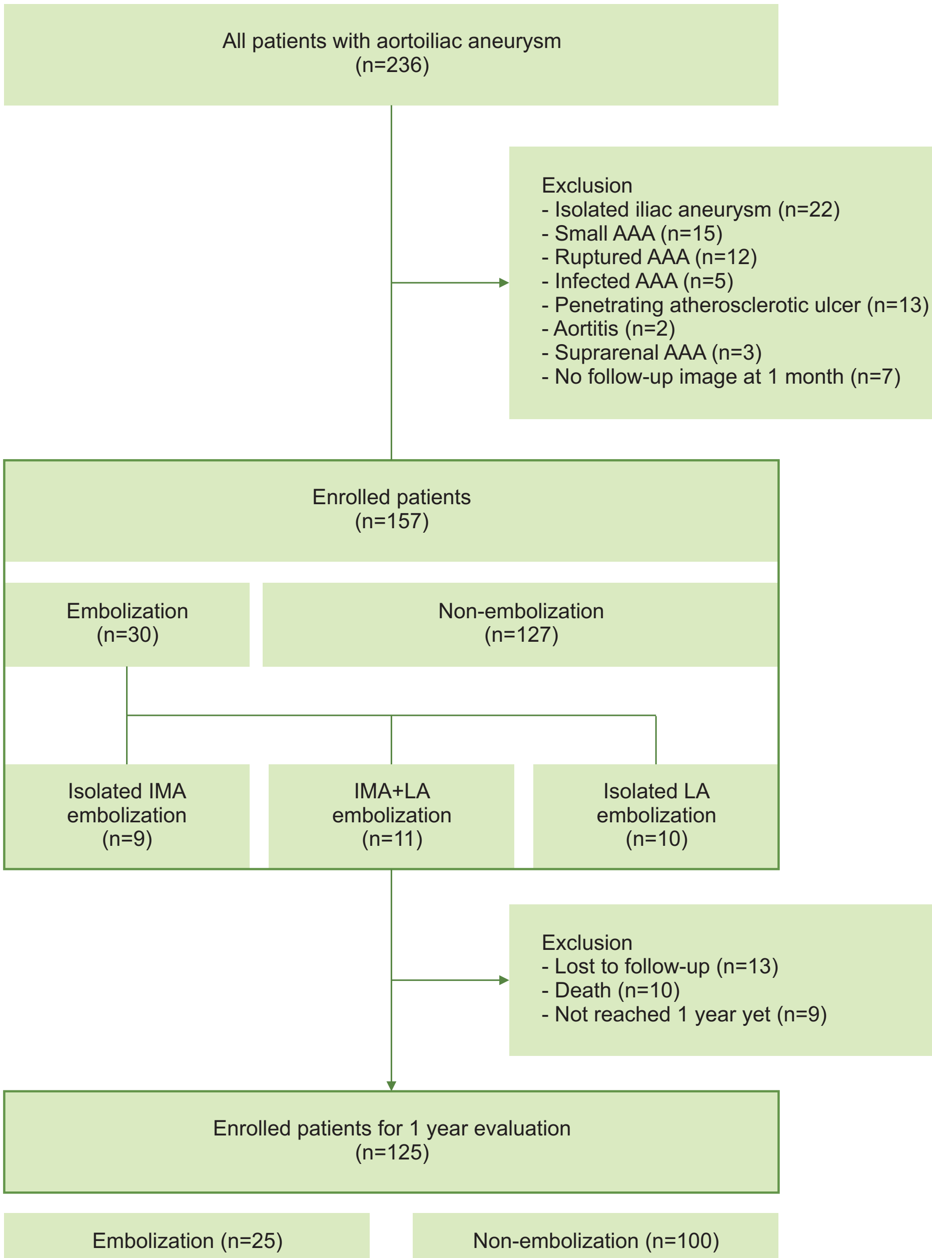

Fig. 1. Patient enrollment flowchart. AAA, abdominal aortic aneurysm; IMA, inferior mesenteric artery; LA, lumbar artery.

baseline maximum AAA diameter than the non-embolization group (median: 53.5 mm [IQR 52.0–56.1] vs. 57.9 mm [IQR 54.0–63.3], P=0.001). In the entire cohort, 70.1% (110/157) of patients had a patent IMA, and 23.6% (37/157) had an IMA diameter ≥3 mm.

Of 110 patients with patent IMA, 20 underwent embolization. The median diameter of the patent IMA was significantly larger in the IMA embolization group (3.17 mm [IQR 3.03-3.25]) than in the IMA non-embolization group (2.48 mm [IQR 2.10-2.87], P<0.001) (Supplementary Table 1).

Concerning LA embolization, 21 patients (13.4%, 21/157) underwent LA embolization. The median numbers of patent LAs originating from the infrarenal aorta and aneurysmal sac were 5.00 (IQR 4.00-6.50) and 3.00 (IQR 3.00-4.00), respectively, in the LA embolization group, and 5.00 (IQR 3.00-6.00) and 3.00 (IQR 2.00-4.00), respectively, in the LA non-embolization group. These differences were not statistically significant (P=0.121 for LAs in the infrarenal aorta and P=0.102 for LAs in the aneurysmal sac) (Supplementary Table 1).

2) Procedural data

The operative variables are summarized in Supplementary Table 2. Six technical failures resulted in an overall technical success rate of 86.7% for the embolization proce-

Table 1. Baseline clinical characteristics

	Embolization (n=30)	Non-embolization (n=127)	P-value
Sex, male	25 (83.3)	104 (81.9)	0.853
Age, median (y)	73.5 (67.0-78.0)	78.0 (71.0-83.0)	0.004
Hypertension	22 (73.3)	82 (64.6)	0.361
Diabetes mellitus	8 (26.7)	28 (22.0)	0.588
Coronary artery disease	11 (36.7)	39 (30.7)	0.529
Congestive heart failure	2 (6.7)	10 (7.9)	0.823
Arrhythmia	7 (23.3)	20 (15.7)	0.322
Cerebrovascular disease	4 (13.3)	27 (21.3)	0.327
Current smoker	5 (16.7)	28 (22.0)	0.515
Chronic obstructive lung disease[a]	15 (50.0)	57 (44.9)	0.613
Renal insufficiency	6 (20.0)	28 (22.0)	0.807
Dialysis	3 (10.0)	3 (2.4)	0.084
Dyslipidemia	18 (60.0)	66 (52.0)	0.428
Chronic liver disease	7 (23.3)	19 (15.0)	0.267
History of malignancy	8 (26.7)	39 (30.7)	0.664

Values are presented as number (%) or median (IQR).
IQR, interquartile range.
[a]Chronic obstructive lung disease included all cases classified as mild obstructive disease or higher, based on pulmonary function tests.

Table 2. Preoperative anatomical details regarding abdominal aortic aneurysm

	Embolization (n=30)	Non-embolization (n=127)	P-value
AAA diameter, median (mm)	53.5 (52.0-56.1)	57.9 (54.0-63.3)	0.001
Patent IMA	27 (90.0)	83 (65.4)	0.008
Patent IMA diameter, median (mm)[a]	3.11 (2.56-3.23)	2.50 (2.12-2.87)	0.003
IMA diameter ≥3.0 mm[a]	18 (66.7)	19 (22.9)	<0.001
Patent LA number, infrarenal aorta, median	5.0 (3.75-6.25)	5.0 (3.00-6.00)	0.065
Patent LA number, aneurysmal sac, median	3.0 (2.0-4.0)	3.0 (2.0-4.0)	0.063
Patent LA number ≥2.5 mm, aneurysmal sac, median	2.0 (1.0-2.0)	1.0 (0.0-1.0)	<0.001
Preoperative occlusion of IIAs			0.631
Unilateral	2 (6.7)	9 (7.1)	
Bilateral	0 (0.0)	3 (2.4)	
Postoperative occlusion of IIAs[b]			0.090
Unilateral	4 (13.3)	39 (30.7)	
Bilateral	2 (6.7)	7 (5.5)	

Values are presented as number (%) or median (IQR).
IQR, interquartile range; IMA, inferior mesenteric artery; LA, lumbar artery, IIAs, internal iliac arteries.
[a]Data from 110 patients with patent IMA were analyzed.
[b]Postoperative occlusion of the IIAs refers to the final patency status of the IIA after endovascular aneurysm repair, including cases in which embolization or occlusion occurred because of stent graft coverage.

dures. In four patients, wire selection for one LA failed after successful IMA embolization. In two additional patients, technical failures occurred (during IMA and LA embolization); both patients were classified into the non-embolization group.

For IMA embolization, metallic coils were used in 18 patients, whereas a vascular plug was utilized in the remaining two patients. All LA embolization procedures were performed using metallic coils. Additional glue embolization was performed in four patients—one following IMA embolization and three following LA embolization.

Total operative time was significantly longer in the embolization group than in the non-embolization group (241.7±78.84 vs. 209.4±71.00 minutes; P=0.013). Similarly, total fluoroscopic time was significantly longer in the embolization group than in the non-embolization group (39.3±15.5 vs. 27.4±16.6 minutes; P<0.001). No in-hospital mortalities were observed in either group. Among the patients who underwent embolization, no complications, such as the need for temporary dialysis, bowel ischemia, non-target embolization, or vascular injury were noted.

3) T2EL at 1 month and 1 year post-EVAR

The incidences of T2EL 1 month and 1 year post-EVAR are summarized in Table 3. The incidence of T2EL 1 month post-EVAR was significantly lower in the embolization group (3.3%, 1/30) than in the non-embolization group (22.8%, 29/127; P=0.015). In the non-embolization group, T2ELs were associated with isolated IMA in eight patients, with isolated LAs in 18 patients, and with both IMA- and LA-related T2EL in three patients. In the embolization group, a single patient exhibited an LA-related T2EL.

At 1 year post-EVAR, imaging data were available for 125 patients. The incidence of T2EL was 8.0% (2/25) in the embolization group and 23.0% (23/100) in the non-embolization group, although the difference was not statistically significant (P=0.094). In the embolization group, both T2ELs were associated with the LAs. In the non-embolization group, five patients exhibited isolated IMA-associated T2EL, 15 patients showed isolated LA-associated T2EL, and three patients displayed both IMA- and LA-related T2EL.

Among the 30 patients with T2EL identified 1 month post-EVAR, 1-year imaging data were available for 23. Of these, 17 patients (74%) had persistent T2EL, whereas six patients exhibited interval resolution of T2EL (isolated IMA-related T2EL in one patient and isolated LA-associated T2EL in five patients; both IMA- and LA-related T2ELs persisted in all three patients).

4) Sac changes at 1 year post-EVAR and T2EL-related reinterventions during follow-up

The aortic sac changes 1 year after EVAR in 125 patients with available images are summarized in Table 3. In the non-embolization group (n=100), sac shrinkage was observed in 47 patients, whereas the sac was stable in 49 patients. Sac expansion ≥5 mm was identified in four patients (4.0%); all cases were associated with T2EL, and no T1EL or T3EL was present on the 1-month and 1-year imaging scans. Among these four patients, two had the source of T2EL as both the IMA and LAs, whereas the remaining two exhibited isolated LA-related T2EL. The same endoleak sources were identified in these four patients 1 month post-EVAR. Additionally, the non-embolization group included 29 patients with T2EL at 1 month, 23 of whom had available imaging data at 1 year. Among these 23 patients, 20 had isolated IMA- or LA-associated endoleaks, with sac expansion observed in two patients (10.0%) at 1 year. By contrast, three patients had combined IMA and LA endoleaks, and two of them (67%) showed sac expansion at 1 year (P=0.026).

Table 3. T2ELs at 1 month and 1 year post-EVAR, and sac changes 1 year post-EVAR in the entire cohort

	Embolization (n=30)	Non-embolization (n=127)	P-value
1-month T2EL			
Incidence	3.3% (1/30)	22.8% (29/127)	0.015
Isolated IMA-associated	0	8	
Isolated LA-associated	1	18	
Combined IMA and LA-associated	0	3	
1-year T2EL[a]			
Incidence	8.0% (2/25)	23.0% (23/100)	0.094
Isolated IMA-associated	0	5	
Isolated LA-associated	2	15	
Combined IMA and LA-associated	0	3	
1-year sac change[a]			
Expansion	0	4.0% (4/100)	0.583
Isolated LA-related T2EL	0	2	
Both IMA and LA-related T2EL	0	2	
Shrinkage	52% (13/25)	47% (47/100)	0.654
Stable sac	48% (12/25)	49% (49/100)	0.929

T2EL, type II endoleak; EVAR, endovascular aneurysm repair; IMA, inferior mesenteric artery; LA, lumbar artery.
[a]Data for 125 patents with available 1-year imaging data post-EVAR were analyzed.

In the embolization group (n=25), 52% (13/25) of the patients exhibited sac shrinkage, whereas the remaining 12 patients (48%) demonstrated a stable sac. Sac expansion was not observed in the embolization group at 1-year follow-up.

The median follow-up duration was 24.4 months (IQR 11.8-36.4) [embolization group: 32.1 months (IQR 13.5-46.0), non-embolization group: 24.3 months (IQR 11.3-35.7)]. During the follow-up period, 25 reinterventions were performed in 20 patients. In the embolization group, six reinterventions were performed in six patients. Of these, three were endoleak-related (type IB, 1; type II, 1; and type V, 1), two were because of limb occlusion, and one was because of puncture site occlusion. In the non-embolization group, 19 reinterventions were performed in 14 patients. Among them, 14 were endoleak-related (type IA, 3; type IB, 1; type IC, 4; type II, 4; and type III, 2), four were because of limb occlusion or stenosis, and one was because of puncture site occlusion.

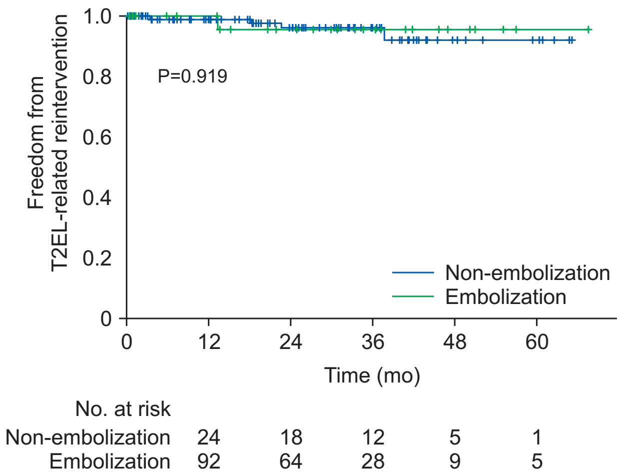

Fig. 2. Kaplan–Meier analysis of freedom from type II endoleaks (T2ELs)-related reintervention stratified by pre-emptive embolization status.

During follow-up, T2EL-related reinterventions were performed in four patients in the non-embolization group and one patient in the embolization group. The rate of freedom from T2EL-related reintervention did not differ significantly between the embolization and non-embolization groups (P=0.919) (Fig. 2).

5) Subgroup analysis for patients with patent IMA >3 mm in diameter

Among the 37 patients with patent IMA diameter ≥3 mm, 18 received pre-emptive embolization, and 19 did not. The outcomes are summarized in Table 4. The incidence of T2EL at the 1-month and 1-year follow-up was 5.6% and 12.5%, respectively, in the embolization group and 52.6% and 52.9%, respectively, in the non-embolization group (P=0.002 for 1 month T2EL and P=0.014 for 1 year T2EL). At the 1-year follow-up, the aneurysm sac shrinkage rates were 56.2% and 23.5% in the embolization and non-embolization groups, respectively (P=0.055). Multivariable analysis revealed pre-emptive embolization as an independent

Table 4. T2ELs at 1 month and 1 year, and sac changes 1 year post-EVAR in the cohort with patent IMA ≥3 mm

	Embolization (n=18)	Non-embolization (n=19)	P-value
1-month T2EL			
Incidence	5.6% (1/18)	52.6% (10/19)	0.002
Isolated IMA-associated	0	3	
Isolated LA-associated	1	4	
Combined IMA and LA-associated	0	3	
1-year T2EL[a]			
Incidence	12.5% (2/16)	52.9% (9/17)	0.014
Isolated IMA-associated	0	2	
Isolated LA-associated	2	4	
Combined IMA and LA-associated	0	3	
1-year sac change[a]			
Expansion	0.0%	4.0% (2/17)	0.485
Isolated LA-related T2EL	0	0	
Both IMA and LA-related T2EL	0	2	
Shrinkage	56.2% (9/16)	23.5% (4/17)	0.055
Stable sac	43.8% (7/16)	64.7% (11/17)	0.227

T2EL, type II endoleak; EVAR, endovascular aneurysm repair; IMA, inferior mesenteric artery; LA, lumbar artery.
[a]Data for 33 patents with available 1-year imaging data post-EVAR were analyzed.

Table 5. Univariable and multivariable analyses of risk factors for T2EL in patients with an IMA diameter of ≥3 mm at the 1-year follow-up

	Univariable analysis		Multivariable analysis	
	OR (95% CI)	P-value	aOR (95% CI)	P-value
Sex, female	2.10 (0.12-37.12)	0.613		
Age	1.03 (0.94-1.13)	0.492		
Pre-emptive embolization	0.13 (0.02-0.74)	0.022	0.071 (0.008-0.663)	0.020
Preoperative sac diameter	1.05 (0.97-1.13)	0.223		
IMA diameter	3.03 (0.49-18.61)	0.231		
Hypertension	1.69 (0.28-10.17)	0.568		
Diabetes mellitus	1.46 (0.34-6.35)	0.615		
Coronary artery disease	2.22 (0.49-10.09)	0.301		
Cerebrovascular disease	3.62 (0.64-20.41)	0.145		
Renal insufficiency	1.41 (0.20-9.96)	0.732		
Dialysis	4.67 (0.37-58.29)	0.232		
Dyslipidemia	1.44 (1.34-6.16)	0.623		

T2EL, type II endoleak; IMA, inferior mesenteric artery; LA, lumbar artery; OR, odds ratio; CI, confidence interval.

protective factor for T2EL at 1 year post-EVAR (odds ratio 0.071, 95% confidence interval, 0.008-0.663; P=0.020); however, no independently associated factors associated with sac shrinkage were identified (Table 5, Supplementary Table 3).

DISCUSSION

This study evaluated the impact of pre-emptive embolization of the aortic side branches, including the IMA and LAs, on early T2EL incidence and aneurysm sac changes following EVAR. Pre-emptive embolization significantly reduced the early T2EL incidence, with a lower rate at 1 month post-EVAR in the embolization group than in the non-embolization group (3.3% vs. 22.8%). Although the 1-year T2EL incidence remained low in the embolization group (8.0% vs. 23.0%), the difference was not statistically significant. In patients with an IMA diameter ≥ 3 mm, pre-emptive embolization significantly reduced T2EL incidence at both 1 month (5.6% vs. 52.6%, P=0.002) and 1 year post-EVAR (12.5% vs. 52.9%, P=0.014). Moreover, the multivariable analysis identified pre-emptive embolization as an independent protective factor against T2EL 1 year post-EVAR.

Despite the reduction in T2EL incidence, pre-emptive embolization did not translate into a significantly higher aneurysm sac regression rate at 1 year. Sac expansion occurred exclusively in the non-embolization group, where T2EL were the sole cause of expansion; however, the overall sac regression rates between the two groups did not differ significantly. Interestingly, in the subgroup of patients with large IMA diameters, sac regression was marginally higher in the embolization group than in the non-embolization group (56.2% vs. 23.5%, P=0.055). Thus, although pre-emptive embolization reduces early T2EL, its impact on sac regression remains uncertain, warranting further investigation with a longer follow-up period.

T2ELs observed intraoperatively often resolve spontaneously; however, persistent T2EL occurs in some cases, with reported persistence rates ranging from 15% to 30% [1,2]. Our findings are consistent with these reports, showing a 1-month T2EL incidence of 22.8% and 1-year incidence of 23.0% in the non-embolization group. Current guidelines recommend annual imaging surveillance if a T2EL is detected 1 month post-EVAR [14,15], with the Society for Vascular Surgery also suggesting an additional 6-month follow-up [14]. However, the persistence or resolution of T2EL at 1 month remains unclear. A retrospective study showed that 41% of T2EL cases identified at 1 month were resolved by 1-year follow-up [16]. Additionally, another study has reported that only 35 of 90 (39%) T2EL cases persisted beyond 6 months during a mean follow-up of 21.7±16 months [17]. This study showed a higher persistence rate of T2EL at 1 month compared to previous studies, with 74% of cases persisting for 1 year. The origins of T2EL at 1 year were similar to those at 1 month, suggesting that early detected T2ELs often persisted. Considering this high persistence rate, careful monitoring is essential when T2EL are present 1 month post-EVAR.

The effect of persistent T2EL remains controversial and requires long-term follow-up. Historically, T2EL have been considered benign owing to their low association with aneurysm rupture and mortality, with outcomes similar to those of patients without T2EL [18]. However, a recent nationwide propensity-matched analysis from Japan has reported a 29% incidence of persistent T2EL, with a 2% risk of AAA-related mortality and rupture at 10 years, which was significantly higher than that in patients without persistent T2EL [2].

Several studies have investigated the use of pre-emptive embolization to mitigate the risk of persistent T2EL. Recent prospective single-arm trials have reported early aneurysmal sac shrinkage and a reduced need for T2EL-related reinterventions 1 year post-EVAR [19,20]. A retrospective study has also indicated that sac growth >5 mm was more common in patients with patent IMA and LAs, especially L4 LA; such growth occurred in 18% of these patients compared to 5.3% of those with occluded IMA and L4 LA [21]. Furthermore, pre-emptive IMA embolization was identified as a protective factor against secondary interventions in a multivariable analysis [21]. Although our study has a relatively small sample size, the findings indicate that patients with combined IMA and LA endoleaks (67% sac expansion rate at 1 year) may be at a higher risk of sac expansion than those with isolated endoleaks (10% sac expansion rate).

By contrast, other studies have failed to demonstrate the benefits of pre-emptive embolization. Väärämäki et al. [10] compared routine IMA embolization before EVAR with a non-embolization strategy and determined no significant clinical benefits. The rates of T2EL-related reintervention, sac enlargement, rupture, and open conversion were similar between the two groups. Similarly, Petit et al. [9] have reported no significant benefit of pre-emptive IMA embolization in the evaluation of standard and fenestrated EVAR outcomes. Their analysis showed no differences in sac size reduction, overall survival, or reintervention rates at 24 months among patients with embolized IMAs, non-embolized patent IMAs, and chronically occluded IMAs.

Only one RCT has evaluated pre-emptive selective embolization, apart from studies on nonselective sac embolization [4]. This study defined the high-risk group of T2EL as AAAs with a patent IMA along the IMA diameter >3 mm, LAs >2 mm, and aortoiliac aneurysms. Five-year comparative outcomes of pre-emptive IMA embolization in this high-risk group showed T2EL incidences of 28% and 55% in the embolization and standard EVAR groups, respectively. Additionally, the mean sac diameter changes were −4.5 mm in the embolization group and −0.42 mm in the standard EVAR group [4], both significantly more favorable in the embolization group, with outcomes similar to those in the low-risk group.

Considering these findings, routine pre-emptive embolization does not provide a clear clinical benefit. However, selective embolization may be a more valuable approach in high-risk patients. Recent guidelines also recommend against routine pre-emptive embolization of the IMA and LAs, focusing instead on identifying the patient or aneurysm characteristics that may benefit from this intervention [15]. Our study's short-term outcomes align with those of previous studies, demonstrating a lower T2EL incidence on follow-up imaging and no sac expansion at 1 year. Although sac regression after pre-emptive embolization for large IMA diameters showed favorable outcomes compared with the non-embolization group, the overall embolization group did not exhibit higher regression rates. However, the embolization group of our study primarily comprised high-risk patients (e.g., those with IMA ≥3 mm or large LAs), and their outcomes were comparable to those of the lower-risk non-embolization group. Thus, selective pre-emptive embolization may help mitigate the risk of T2EL and related complications in anatomically high-risk patients. Long-term follow-up and careful patient selection for pre-emptive embolization are required in future studies.

Pre-emptive embolization has also been criticized for overtreatment and cost-effectiveness concerns because the risk of T2EL-induced rupture and aneurysm-related mortality is minimal. Additionally, pre-emptive embolization may result in unnecessary morbidity owing to radiation exposure, contrast use, and prolonged operative times. In our study, although pre-emptive embolization increased procedure and fluoroscopy times, no procedure-related complications were observed. These drawbacks should be considered; however, in high-risk patients with large patent IMAs, the potential to reduce early T2EL and secondary interventions may justify their selective use. Additionally, if pre-emptive embolization reduces early endoleaks and secondary reinterventions, it may mitigate initial concerns regarding radiation exposure and cost-effectiveness, as reduced endoleaks would decrease the need for frequent CTA follow-ups and, in turn, lower radiation exposure during reinterventions [22].

This study has some limitations. First, this was a retrospective observational study conducted at two centers, introducing the potential for selection bias and variability in procedural techniques. Procedural heterogeneity due to different operators and center-specific protocols may have influenced the comparability of the outcomes. Second, the sample size, particularly in the embolization group, was relatively small, limiting the statistical power to detect differences in certain outcomes, such as sac changes and reintervention rates. Third, the follow-up duration was relatively short, preventing a comprehensive assessment of long-term outcomes (e.g., late rupture, aneurysm-related mortality, and sac shrinkage durability). Fourth, the decision to perform pre-emptive embolization was based on the operator's discretion, potentially permitting variability in patient selection and procedural outcomes. However, in most cases, pre-emptive embolization was performed for IMAs with a diameter of ≥3 mm and for large LAs, adhering to established anatomic criteria. Additionally, although the IMA diameter was routinely measured, the diameters of all LAs were not systematically recorded.

Future studies, particularly RCTs with larger sample sizes and longer follow-up durations [23], are needed to confirm the efficacy and safety of pre-emptive embolization for reducing the risk of T2EL and its associated complications.

CONCLUSION

Pre-emptive embolization reduced the incidence of early T2EL after EVAR and demonstrated stable outcomes at the 1-year follow-up, particularly in patients with large patent IMA diameters. However, further studies, including large-scale trials with long-term follow-ups, are needed to confirm these findings and refine the treatment protocols.

FUNDING

None.

SUPPLEMENTARY MATERIALS

Supplementary data can be found via https://doi.org/10.5758/vsi.250010

CONFLICTS OF INTEREST

Hyung-Kee Kim has served as the Editor-in-Chief of VSI since 2023. Deokbi Hwang has been an Editorial Board Member of VSI since 2025.

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