

Title	Impact of the Patency of Inferior Mesenteric Artery on 7-Year Outcomes After Endovascular Aneurysm Repair
Author(s)	Ide, Toru; Shimamura, Kazuo; Kuratani, Toru et al.
Citation	Journal of Endovascular Therapy. 2022, p. none
Version Type	AM
URL	https://hdl.handle.net/11094/89662
rights	
Note	

Osaka University Knowledge Archive : OUKA

<https://ir.library.osaka-u.ac.jp/>

Osaka University

1 **Impact of the patency of the inferior mesenteric artery on 7-year outcomes after**
2 **endovascular aneurysm repair**

3

4 **ABSTRACT**

5 **Purpose:** The impact of the preoperative patent inferior mesenteric artery (IMA) on late
6 outcomes following endovascular aneurysm repair (EVAR) remains unclear. This study
7 aimed to investigate the specific influence of IMA patency on 7-year outcomes after
8 EVAR.

9 **Materials and Methods:** In this retrospective cohort study, 556 EVARs performed for
10 true abdominal aortic aneurysm cases between January 2006 and December 2019 at our
11 institution were reviewed. EVARs performed using a commercially available device
12 with no type-I or type-III endoleak (EL) during follow-up and with follow-up ≥ 12
13 months were included. A total of 336 patients were enrolled in this study. The cohort
14 was divided into the patent IMA group and occluded IMA group according to
15 preoperative IMA status. The late outcomes, including aneurysm sac enlargement,

1 reintervention, and mortality rates were compared between both groups using
2 propensity-score matched data.

3 **Results:** After propensity-score-matching, 86 patients were included in each group. The
4 median follow-up period was 56 months (interquartile range: 32-94 months). The
5 incidence of type-II EL at discharge was 50% in the patent IMA group and 19% in the
6 occluded IMA group ($p<0.001$). The type-II EL from the IMA and the lumbar arteries
7 was significantly higher in the patent IMA group than in the occluded IMA group
8 ($p<0.001$ and $p=0.002$). The rate of freedom from aneurysm-sac enlargement with type-
9 II EL was significantly higher in the occluded IMA group than in the patent IMA group
10 (94% vs. 69% at 7 years; $p<0.001$). The rate of freedom from reintervention was
11 significantly higher in the occluded IMA group than in the patent IMA group (90% vs.
12 74% at 7 years; $p=0.007$). Abdominal aortic aneurysm-related death and all-cause
13 mortality did not significantly differ between groups ($p=0.32$ and $p=0.34$).

14 **Conclusions:** IMA patency could affect late reintervention and aneurysm sac
15 enlargement but did not have a significant impact on mortality. Preoperative assessment

1 and embolization of the IMA might be an important factor for improvement in late
2 EVAR outcomes.

3

4 **Keywords:** Abdominal aortic aneurysm; Endovascular aneurysm repair; Endoleak;

5 Inferior mesenteric artery

6

7

8

9

10

11

12

13

14

1 INTRODUCTION

2 Although lower rates of early mortality and morbidity were reported for
3 endovascular aneurysm repair (EVAR) in previous studies,¹⁻⁴ EVAR failed to show
4 long-term benefits in randomized controlled trials (RCTs).⁴⁻⁷ Even a higher
5 reintervention rate was reported in three of those trials on EVAR.⁴⁻⁶

6 Following EVAR, there is a high chance of residual blood flow, known as
7 endoleak (EL), which contributes to the worsening of late outcomes. A type-II EL is
8 characterized by retrograde blood flow from small branches originating from aneurysms,
9 such as those of the inferior mesenteric artery (IMA) and lumbar arteries (LAs); it is the
10 most common EL that is associated with a higher incidence of aneurysm sac enlargement
11 and reintervention than in patients without an EL.^{8,9}

12 In the previous risk analysis, IMA had the highest hazard ratio for sac
13 enlargement after EVAR.¹⁰ A retrospective study has also revealed the association
14 between patent IMA and the incidence of reintervention.¹¹ Although the number of patent
15 LAs has also been reported to be associated with the incidence of type-II EL and sac

1 enlargement,¹² neither study adjusted the number of patent LAs when comparing results
2 between patients with preoperative patent and occluded IMA. In addition, one RCT
3 reporting the efficacy of embolizing patent IMA¹³ did not show the distribution of patent
4 LAs in their cohort. Moreover, the mean follow-up periods in the previous studies were
5 approximately 2–3 years, with little detail on the longer-term results. Therefore, this study
6 aimed to assess the specific influence of a patent IMA on the 7-year results following
7 EVAR.

8

9 **MATERIALS AND METHODS**

10 *Study design*

11 A retrospective cohort study was conducted on 556 consecutive patients
12 with a true infrarenal abdominal aortic aneurysm (AAA) treated by EVAR at our
13 hospital between January 2006 and December 2019. EVAR performed using a
14 commercially available stent graft was performed in 446 patients. Fifty-two patients
15 with a type-I EL or a type-III EL during follow-up and 58 patients with <12-month

1 follow-up after EVAR were excluded. Finally, a total of 336 patients were included in
2 this study.

3 Patients were divided into the patent IMA group and occluded IMA group
4 according to the preoperative IMA status based on contrast computed tomography (CT)
5 examinations conducted preoperatively. The patients' background characteristics of
6 both groups were propensity-score-matched, and outcome variables were compared
7 between the two groups (Supplementary Figure 1).

8 This study was approved by the Institutional Review Board, and informed
9 consent was obtained from all patients. The collected data are listed in Supplementary
10 Table 1.

11 *Surgical indications*

12 Preoperatively, all patients underwent contrast multidetector CT (MDCT) for
13 the assessment of AAA diameter and morphology. Treatment for AAA was indicated in
14 the following cases: maximum diameter ≥ 50 mm, rapid expansion, and saccular
15 morphology and expansion during follow-up. In patients with an intermediate-high

1 surgical risk, EVAR was adopted when the anatomical assessment met the EVAR
2 criteria for each commercially available device.

3 *CT measurement*

4 All MDCT images were reconstructed using three-dimensional image
5 reconstruction software (AquariusNET; TeraRecon Inc., San Mateo, CA, USA) in
6 conjunction with thin-slice (<1 mm) MDCT images. Both a radiologist and a
7 cardiovascular surgeon, in consensus, obtained and retrospectively analyzed the EL and
8 anatomical data, including the aneurysm sac diameter, proximal neck length, angulation,
9 IMA patency, and number of patent LAs in each patient using the arterial and delayed
10 phases of contrast MDCT in a blinded manner. The definition of anatomical
11 measurements has been previously elucidated.¹⁰ The IMA was deemed patent upon
12 fulfilling the following criteria: (1) showing contrast agent within the lumen on contrast
13 MDCT and (2) not coil-embolized prior to EVAR. When the IMA did not meet one of
14 these two criteria, it was judged to be occluded.

15 *Follow-up protocol*

1 Postoperative contrast MDCT was performed at discharge. Follow-up was
2 performed at 6 months, 1 year after EVAR, and yearly thereafter using MDCT. If the
3 follow-up MDCT revealed sac enlargement, contrast MDCT was immediately
4 considered to clarify the cause. Subsequently, reintervention was considered, taking the
5 cause of sac enlargement as well as the patient's age and comorbidities into account.

6 *Endpoints and definitions*

7 The primary endpoint was the occurrence of aneurysm-sac enlargement. The
8 secondary endpoints were all-cause mortality, AAA-related death, AAA rupture,
9 reintervention, and type-II EL. Aneurysm sac enlargement was defined as growth of
10 aneurysm sac diameter ≥ 5 mm from CT at discharge.

11 *Statistical analysis*

12 Continuous data are expressed as mean \pm standard deviation (SD), whereas
13 categorical data are presented as number and percentage. Categorical and continuous
14 data were compared between the study groups using the χ^2 and Wilcoxon tests,
15 respectively.

1 Propensity scores were estimated by accounting all risk factors that were
2 significantly associated with either the patent IMA group or occluded IMA group on
3 logistic regression analysis. Patients in the patent and occluded IMA groups were
4 subsequently paired at a 1:1 ratio according to the propensity scores using exact
5 matching, with a standard caliper size of $0.05 \times \log [\text{SD of propensity scores}]$.
6 Standardized differences were estimated before and after matching to evaluate the
7 balance of covariates; small absolute values (<0.05) indicate balance between the two
8 groups. The Kaplan–Meier survival curve with log-rank test was used to estimate the
9 time-to-event rates between both groups.

10 All p -values <0.05 (two-sided) were considered statistically significant. All
11 statistical analyses were performed using JMP Pro statistical software version 14.3.0
12 (SAS Institute Inc., Cary, NC, USA).

13 **RESULTS**

14 *Patient characteristics before propensity-score-matching*

1 The study cohort comprised 336 patients prior to propensity-score-
2 matching. The median follow-up period was 65 months (interquartile range: 36-105
3 months). Preoperative MDCT revealed 254 (76%) cases of IMA with contrast agent in
4 the lumen. Among these, 26 patients underwent IMA embolization before or during
5 EVAR. Therefore, 228 patients were included in the patent IMA group, whereas 108
6 patients were included in the occluded IMA group.

7 Patient characteristics and stent grafts used in both groups are summarized
8 in Table 1. The occluded IMA group had a significantly larger aneurysm diameter than
9 the patent IMA group had ($p<0.001$). Furthermore, the distribution of the number of
10 patent LAs was significantly different between both groups ($p=0.025$) (Figure 1a). The
11 mean diameter of the embolized IMA in the occluded IMA group was 3.6 ± 1.0 mm,
12 which was significantly larger than that in the patent IMA group without embolization
13 (3.2 ± 0.9 mm) ($p=0.032$).

14

15 *Results before propensity-score-matching*

1 The incidence of type-II EL at discharge was 49% (112 patients) in the patent
2 IMA group and 17% (18 patients) in the occluded IMA group ($p<0.001$). After
3 discharge, additional onset of type-II EL occurred in 29 out of 116 patients in the patent
4 IMA group and 7 out of 90 in the occluded IMA group. Overall, type-II EL was
5 observed in 141 out of 228 patients in the patent IMA group and 25 out of 108 in the
6 occluded IMA group during follow-up (Supplementary Table 2).

7 Sac enlargement occurred in 68 of 141 patients and 4 of 25 patients with type-II
8 EL in the patent IMA and the occluded IMA groups (Supplementary Table 2). Only
9 1/26 patients with an IMA with contrast agent in the lumen preoperatively and who
10 underwent IMA embolization prior to EVAR reported aneurysm sac enlargement
11 (Supplementary Table 3). This patient developed sac enlargement at 55 months after
12 EVAR. Supplementary Figure 2 shows the number of patients who developed sac
13 enlargement at 5 years after EVAR in relation to the number of patent LAs in the patent
14 and occluded IMA groups. Though one of three patients with 6 or 7 patent LAs in the
15 occluded IMA group developed sac enlargement at 5 years after EVAR, patients with
16 0–5 patent LAs in the occluded IMA group had no sac enlargement at 5 years after

1 EVAR.

2 The rates of freedom from aneurysm sac enlargement at 3, 5, and 7 years were
3 87%, 79%, and 75% in the patent IMA group and 100%, 98%, and 95% in the occluded
4 IMA group, respectively ($p<0.001$; Figure 2a).

5 Reintervention procedures are summarized in Supplementary Table 2. Of 228
6 patients in the patent IMA group, 38 underwent 56 reintervention procedures including
7 14 cases of IMA embolization, 30 of LA embolization, 3 cases of embolization of IMA
8 and LA, 5 of sac embolization, and 4 cases of open repair. In the occluded IMA group,
9 5 of 108 patients underwent 5 reintervention procedures including 4 cases of LA
10 occlusion and 1 case of sac embolization. A total of 27 patients did not receive
11 reintervention because of poor general condition or concomitant disease.

12 The rates of freedom from reintervention at 3, 5, and 7 years were 90%, 82%, and
13 80% in the patent IMA group and 99%, 95%, and 93% in the occluded IMA group,
14 respectively ($p=0.004$; Figure 3a).

15 AAA-related death (rupture) occurred in three patients with type-II EL in the

1 patent IMA group at 69, 137, and 145 months after EVAR (Supplementary Table 2).

2 The rate of freedom from AAA rupture and AAA-related death at 7 years was
3 99% in the patent IMA group and 100% in the occluded IMA group ($p=0.29$).

4 Overall, death occurred in 61 out of 228 patients in the patent IMA group and 15
5 out of 108 patients in the occluded IMA group. The major cause of death was cancer in
6 both groups (41% in the patent IMA group and 47% in the occluded IMA group;
7 Supplementary Table 2).

8 The rates of freedom from all-cause mortality at 3, 5, and 7 years were 91%, 82%,
9 and 76% in the patent IMA group and 95%, 86%, and 81% in the occluded IMA group,
10 respectively ($p=0.17$; Figure 4a).

11

12 **Patient characteristics after propensity-score-matching**

13 Individual propensity scores were calculated through logistic regression modeling
14 based on aneurysm diameter and distribution of the number of patent LAs, which were
15 the two covariates identified to be significantly associated with either the patent IMA

1 group or occluded IMA group. Patients in the patent and occluded IMA groups were
2 subsequently paired at a 1:1 ratio according to the propensity scores using exact
3 matching.

4 After propensity-score-matching, 86 patients in each group were matched
5 for the analysis. Patient characteristics showed no significant difference between the
6 two groups (Figure 1b, Table 2). The mean IMA diameter after matching in the patent
7 IMA group was 3.2 ± 0.9 mm. The median follow-up period was 56 months (interquartile
8 range: 32-94 months).

9 Supplementary Table 4 summarizes late events following EVAR in both
10 groups after propensity score matching.

11 **Matched comparison of the incidence of type-II EL at discharge**

12 After propensity score matching, the incidence of overall type-II EL at discharge
13 was 50% (43 patients) in the patent IMA group and 19% (16 patients) in the occluded
14 IMA group ($p < 0.001$, Figure 5a). Incidence of type-II EL from IMA at discharge was
15 29% (25 patients) in the patent IMA group and 1% (1 patients) in the occluded IMA

1 group ($p<0.001$; Figure 5b). One patient with no contrast within IMA and judged
2 occluded developed type-II EL from IMA. Incidence of type-II EL from LA at
3 discharge was 40% (34 patients) in the patent IMA group and 19% (16 patients) in the
4 occluded IMA group ($p=0.002$) (Figure 5c).

5 **Matched comparison of the incidence of aneurysm-sac enlargement**

6 The rates of freedom from aneurysm-sac enlargement at 3, 5, and 7 years were
7 88%, 81%, and 69% in the patent IMA group and 100%, 97%, and 94% in the occluded
8 IMA group, respectively ($p<0.001$; Figure 2b).

9 **Matched comparison of the incidence of reintervention**

10 The rates of freedom from reintervention at 3, 5, and 7 years were 86%, 79%,
11 and 74% in the patent IMA group and 99%, 94%, and 90% in the occluded IMA group,
12 respectively ($p=0.007$; Figure 3b).

13 **Matched comparison of the incidence of AAA rupture and AAA-related death**

14 No AAA rupture and AAA-related death were observed within 7 years in both groups
15 after propensity score matching, although one AAA-related death (rupture) was reported

1 in the patent IMA group, 12 years after EVAR. AAA rupture and AAA-related death
2 did not significantly differ between the groups ($p=0.32$).

3

4 **Matched comparison of all-cause mortality**

5 The rates of freedom from all-cause mortality at 3, 5, and 7 years were 89%, 77%,
6 and 71% in the patent IMA group and 94%, 82%, and 77% in the occluded IMA group,
7 respectively ($p=0.34$; Figure 4b).

8

9 **DISCUSSION**

10 The impact of preoperative IMA patency on the late outcomes remains unclear.
11 We performed an evaluation of the 7-year outcomes based on a propensity-score
12 matched comparison between patients with and without preoperative patent IMA. Our
13 results revealed that a patent IMA contributed to late adverse outcomes following
14 EVAR. Events such as aneurysm-sac enlargement and reinterventions were significantly
15 increased in patients with patent IMA in our cohort.

1 IMA has been reported to be related to 85% of type-II EL,¹⁴ with IMA patency
2 being a risk factor for type-II EL from the IMA. Previous studies have revealed the
3 association between the IMA and the occurrence of type-II EL.¹⁵⁻¹⁷ As expected, the
4 occurrence of type-II EL and type-II EL from IMA was lower in patients with occluded
5 IMA than that of patients with patent IMA. Furthermore, our present study showed that
6 the occurrence of type-II EL from LAs was lower in patients with occluded IMA than
7 that of patients with patent IMA.

8 The association between the IMA and the incidence of aneurysm sac
9 enlargement and reintervention for type-II EL has gradually become clear. Previously, a
10 retrospective risk analysis of 320 patients showed that the IMA played a particular
11 significant role in aneurysm-sac enlargement with type-II EL following EVAR.¹⁰ The
12 hazard ratio of IMA patency for sac enlargement was about 18, which was higher than
13 that of other factors associated with aneurysm-sac enlargement, such as the number of
14 patent LAs and chronic kidney disease \geq stage 4. A retrospective risk analysis of 490
15 patients showed the significant association between patent IMA and reintervention.¹¹
16 However, these studies were limited by their retrospective design, and anatomical

1 factors such as the number of patent LAs and aneurysm sac diameter were not adjusted
2 for the evaluation of the impact of the IMA. Actually, the aneurysm diameter and the
3 distribution of the number of patent LAs were significantly different between the patent
4 and occluded IMA groups in our study before propensity-score matching. Two of the
5 major features of our study are that anatomical factors were adjusted in our study when
6 comparing the results between patients with and without preoperative patent IMA and
7 that the time-to-event rate was prospectively evaluated in our study using the Kaplan–
8 Meier curve. To our knowledge, no previous study has prospectively compared late
9 outcomes between patients with preoperative patent IMA and those with preoperative
10 occluded IMA using the Kaplan–Meier curve.

11 It is technically much easier to access the IMA from the aortic lumen before
12 EVAR than through a circuitous route via the superior mesenteric artery collaterals after
13 EVAR.¹¹ Moreover, once type-II EL has been established, the efficacy of reintervention
14 becomes limited.¹⁸ Several studies reported that IMA embolization significantly
15 reduced the occurrence of type-II EL.¹⁹⁻²² An RCT showed the efficacy of IMA
16 embolization in preventing type-II EL and aneurysm-sac enlargement.¹³ However, the

1 mean follow-up period in that trial is 22 months, and the result of a longer follow-up is
2 awaited. Although our study did not directly reveal the efficacy of IMA embolization, it
3 seemed to be useful for predicting the efficacy of IMA embolization by showing the
4 impact of IMA patency on late outcomes. Moreover, in our study, the incidence of
5 aneurysm-sac enlargement and reintervention in the occluded IMA group with IMA
6 embolization before EVAR seemed to be similar with the incidence in the occluded
7 IMA group without IMA embolization.

8 Several studies reported that embolization of both the IMA and LAs reduced the
9 incidence of type-II EL than IMA embolization alone.²³⁻²⁷ Nevertheless, the LAs were
10 often so small and tortuous that the success rate of LA embolization was relatively
11 low.^{23,24,26} In our study, aneurysm-sac enlargement in the occluded IMA group was low.
12 Similarly, a previous study reported that when the IMA was occluded before EVAR, the
13 occurrence of type-II EL from the LAs would not lead to aneurysm-sac enlargement.¹³
14 Mixed type-II EL from the IMA and LA had also been reported to be a predictive factor
15 for aneurysm-sac enlargement.²⁸ Considering these results, when the IMA was occluded
16 and mixed type-II EL seemed to be avoidable, embolization of LAs before the EVAR

1 procedure may not be necessary for the prevention of sac enlargement.

2 In our study cohort, aortic rupture occurred in 3 of 336 patients (0.9%), and no
3 significant difference in mortality was observed between the patent and occluded IMA
4 groups. Similarly, a multicenter retrospective cohort study reported no difference in the
5 overall survival between patients with and without type-II EL.²⁹ This could be because
6 of the study design excluding patients with type-I EL or type-III EL during follow-up.
7 Per previous reports, type-II EL was associated with an increased incidence of late type-
8 I EL,^{30,31} and a retrospective analysis of 38,008 patients in the Japan Committee for
9 Stentgraft Management registry showed a significant association between type-II EL
10 and aortic event.³² However, it is difficult to distinguish primary type-I or III EL and
11 type-I or III EL as a result of type-II EL. Type-I EL and type-III EL occurring as a
12 result of type-II EL may be excluded from our study, which might have underestimated
13 the impact of the IMA on aortic rupture. Furthermore, aggressive reintervention,
14 including open repair, may result in the low incidence of rupture. Further examination
15 assessing the true impact of IMA on aortic rupture is required.

16

1 **Limitations**

2 This study had some limitations. First, this study had an observational single-
3 center study design. There might be a selection bias because 58 patients were excluded
4 owing to a lack of CT follow-up beyond 12 months. Second, although large IMAs
5 tended to be selected for pre-embolization, the detailed patient selection criteria for
6 embolization of patent IMAs before EVAR have not been established in our institution.
7 Third, reintervention and type of reintervention have not been standardized in our
8 institution, since both depended on the outpatient doctor and treating physician. Thus,
9 there might have been a selection bias in relation to identification of patients who were
10 meant to receive reinterventions and the selection of the appropriate type of
11 reintervention. Forth, only 26 patients underwent IMA embolization for preoperative
12 patent IMA in our study, which is not enough to determine the usefulness of IMA
13 embolization or the specific influence of the remaining LAs on sac enlargement after
14 EVAR. Finally, long-term outcomes could not be completely assessed, as the records
15 were unavailable when they did not present to this institution at that time.

16

1 **Conclusion**

2 In this study, IMA patency seemed to significantly affect late reintervention and
3 aneurysm sac enlargement with type-II EL, but did not have a significant impact on
4 aneurysm-related death. Preoperative assessment and embolization of the IMA might be
5 an important factor for improvement in late EVAR outcomes.

6 **References**

- 7 1. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG, EVAR trial
8 participants. Comparison of endovascular aneurysm repair with open repair in patients
9 with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results:
10 randomised controlled trial. *Lancet* 2004; 364(9437): 843-848. doi:[10.1016/S0140-](https://doi.org/10.1016/S0140-6736(04)16979-1)
11 [6736\(04\)16979-1](https://doi.org/10.1016/S0140-6736(04)16979-1).
- 12 2. Blankensteijn JD, de Jong SE, Prinszen M, van der Ham AC, Buth J, van
13 Sterkenburg SM, et al. Two-year outcomes after conventional or endovascular repair of
14 abdominal aortic aneurysms. *N Engl J Med* 2005; 352(23): 2398-2405.
15 doi:[10.1056/NEJMoa051255](https://doi.org/10.1056/NEJMoa051255).

- 1 3. Lederle FA, Freischlag JA, Kyriakides TC, Padberg FT, Matsumura JS, Kohler
2 TR, et al. Outcomes following endovascular vs open repair of abdominal aortic
3 aneurysm: a randomized trial. *JAMA* 2009; 302(14): 1535-1542.
4 doi:[10.1001/jama.2009.1426](https://doi.org/10.1001/jama.2009.1426).
- 5 4. Becquemin JP, Pillet JC, Lescalie F, Sapoval M, Goueffic Y, Lermusiaux P, et
6 al. A randomized controlled trial of endovascular aneurysm repair versus open surgery
7 for abdominal aortic aneurysms in low- to moderate-risk patients. *J Vasc Surg* 2011;
8 53(5): 1167-1173.e1. doi:[10.1016/j.jvs.2010.10.124](https://doi.org/10.1016/j.jvs.2010.10.124).
- 9 5. de Bruin JL, Karthikesalingam A, Holt PJ, Prinssen M, Thompson MM,
10 Blankensteijn JD, et al. Predicting reinterventions after open and endovascular
11 aneurysm repair using the St George's Vascular Institute score. *J Vasc Surg* 2016; 63(6):
12 1428-1433.e1. doi:[10.1016/j.jvs.2015.12.028](https://doi.org/10.1016/j.jvs.2015.12.028).
- 13 6. Patel R, Powell JT, Sweeting MJ, Epstein DM, Barrett JK, Greenhalgh RM.
14 The UK EndoVascular aneurysm repair (EVAR) randomised controlled trials: long-term
15 follow-up and cost-effectiveness analysis. *Health Technol Assess* 2018; 22(5): 1-132.

- 1 doi:[10.3310/hta22050](https://doi.org/10.3310/hta22050).
- 2 7. Lederle FA, Kyriakides TC, Stroupe KT, Freischlag JA, Padberg FT,
3 Matsumura JS, et al. Open versus Endovascular Repair of abdominal aortic aneurysm. *N*
4 *Engl J Med* 2019; 380(22): 2126-2135. doi:[10.1056/NEJMoa1715955](https://doi.org/10.1056/NEJMoa1715955).
- 5 8. van Marrewijk CJ, Fransen G, Laheij RJ, Harris PL, Buth J, EUROSTAR
6 Collaborators. Is a type II endoleak after EVAR a harbinger of risk? Causes and
7 outcome of open conversion and aneurysm rupture during follow-up. *Eur J Vasc*
8 *Endovasc Surg* 2004; 27(2): 128-137. doi:[10.1016/j.ejvs.2003.10.016](https://doi.org/10.1016/j.ejvs.2003.10.016).
- 9 9. Jones JE, Atkins MD, Brewster DC, Chung TK, Kwolek CJ, LaMuraglia GM,
10 et al. Persistent type 2 endoleak after endovascular repair of abdominal aortic aneurysm
11 is associated with adverse late outcomes. *J Vasc Surg* 2007; 46(1): 1-8.
12 doi:[10.1016/j.jvs.2007.02.073](https://doi.org/10.1016/j.jvs.2007.02.073).
- 13 10. Ide T, Masada K, Kuratani T, et al. Risk analysis of aneurysm sac enlargement
14 caused by type II endoleak after endovascular aortic repair. *Ann Vasc Surg* 2021;S0890;
15 5096(21): 00555-0. doi:[10.1016/j.avsg.2021.06.013](https://doi.org/10.1016/j.avsg.2021.06.013).

- 1 11. Chew DK, Dong S, Schroeder AC, Hsu HW, Franko J. The role of the inferior
2 mesenteric artery in predicting secondary intervention for type II endoleak following
3 endovascular aneurysm repair. *J Vasc Surg* 2019; 70(5): 1463-1468.
4 doi:[10.1016/j.jvs.2019.01.090](https://doi.org/10.1016/j.jvs.2019.01.090).
- 5 12. Seike Y, Matsuda H, Fukuda T, Inoue Y, Omura A, Uehara K, et al. The
6 influence of 4 or more patent lumbar arteries on persistent Type II endoleak and sac
7 expansion after endovascular aneurysm repair. *Ann Vasc Surg* 2018; 50: 195-201.
8 doi:[10.1016/j.avsg.2017.12.014](https://doi.org/10.1016/j.avsg.2017.12.014).
- 9 13. Samura M, Morikage N, Otsuka R, Mizoguchi T, Takeuchi Y, Nagase T, et al.
10 Endovascular aneurysm repair With inferior mesenteric artery embolization for
11 preventing Type II endoleak: A prospective randomized controlled trial. *Ann Surg* 2020;
12 271(2): 238-244. doi:[10.1097/SLA.0000000000003299](https://doi.org/10.1097/SLA.0000000000003299).
- 13 14. Velazquez OC, Baum RA, Carpenter JP, Golden MA, Cohn M, Pyeron A, et al.
14 Relationship between preoperative patency of the inferior mesenteric artery and
15 subsequent occurrence of type II endoleak in patients undergoing endovascular repair of

- 1 abdominal aortic aneurysms. *J Vasc Surg* 2000; 32(4): 777-788.
2 doi:[10.1067/mva.2000.108632](https://doi.org/10.1067/mva.2000.108632).
- 3 15. Ward TJ, Cohen S, Patel RS, Kim E, Fischman AM, Nowakowski FS, et al.
4 Anatomic risk factors for type-2 endoleak following EVAR: a retrospective review of
5 preoperative CT angiography in 326 patients. *Cardiovasc Intervent Radiol* 2014; 37(2):
6 324-328. doi:[10.1007/s00270-013-0646-7](https://doi.org/10.1007/s00270-013-0646-7).
- 7 16. Samura M, Morikage N, Mizoguchi T, Takeuchi Y, Ueda K, Harada T, et al.
8 Identification of anatomical risk factors for Type II endoleak to guide selective inferior
9 mesenteric artery embolization. *Ann Vasc Surg* 2018; 48: 166-173.
10 doi:[10.1016/j.avsg.2017.10.016](https://doi.org/10.1016/j.avsg.2017.10.016).
- 11 17. Kray J, Kirk S, Franko J, Chew DK. Role of type II endoleak in sac regression
12 after endovascular repair of infrarenal abdominal aortic aneurysms. *J Vasc Surg* 2015;
13 61(4): 869-874. doi:[10.1016/j.jvs.2014.11.003](https://doi.org/10.1016/j.jvs.2014.11.003).
- 14 18. Ultee KHJ, Büttner S, Huurman R, Bastos Gonçalves F, Hoeks SE, Bramer
15 WM, et al. Editor's choice - systematic review and meta-analysis of the outcome of

- 1 treatment for Type II endoleak following endovascular aneurysm repair. *Eur J Vasc*
2 *Endovasc Surg* 2018; 56(6): 794-807. doi:[10.1016/j.ejvs.2018.06.009](https://doi.org/10.1016/j.ejvs.2018.06.009).
- 3 19. Axelrod DJ, Lookstein RA, Guller J, Nowakowski FS, Ellozy S, Carroccio A,
4 et al. Inferior mesenteric artery embolization before endovascular aneurysm repair:
5 technique and initial results. *J Vasc Interv Radiol* 2004; 15(11): 1263-1267.
6 doi:[10.1097/01.RVI.0000141342.42484.90](https://doi.org/10.1097/01.RVI.0000141342.42484.90).
- 7 20. Nevala T, Biancari F, Manninen H, Matsi P, Mäkinen K, Ylönen K, et al.
8 Inferior mesenteric artery embolization before endovascular repair of an abdominal
9 aortic aneurysm: effect on type II endoleak and aneurysm shrinkage. *J Vasc Interv*
10 *Radiol* 2010; 21(2): 181-185. doi:[10.1016/j.jvir.2009.10.014](https://doi.org/10.1016/j.jvir.2009.10.014).
- 11 21. Ward TJ, Cohen S, Fischman AM, Kim E, Nowakowski FS, Ellozy SH, et al.
12 Preoperative inferior mesenteric artery embolization before endovascular aneurysm
13 repair: decreased incidence of type II endoleak and aneurysm sac enlargement with 24-
14 month follow-up. *J Vasc Interv Radiol* 2013; 24(1): 49-55.
15 doi:[10.1016/j.jvir.2012.09.022](https://doi.org/10.1016/j.jvir.2012.09.022).

- 1 22. Müller-Wille R, Uller W, Gössmann H, Heiss P, Wiggermann P, Dollinger M,
2 et al. Inferior mesenteric artery embolization before endovascular aortic aneurysm
3 repair using amplatzer vascular plug type 4. *Cardiovasc Intervent Radiol* 2014; 37(4):
4 928-934. doi:[10.1007/s00270-013-0762-4](https://doi.org/10.1007/s00270-013-0762-4).
- 5 23. Parry DJ, Kessel DO, Robertson I, Denton L, Patel JV, Berridge DC, et al. Type
6 II endoleaks: predictable, preventable, and sometimes treatable? *J Vasc Surg* 2002;
7 36(1): 105-110. doi:[10.1067/mva.2002.125023](https://doi.org/10.1067/mva.2002.125023).
- 8 24. Bonvini R, Alerci M, Antonucci F, Tutta P, Wytttenbach R, Bogen M, et al.
9 Preoperative embolization of collateral side branches: a valid means to reduce type II
10 endoleaks after endovascular AAA repair. *J Endovasc Ther* 2003; 10(2): 227-232.
11 doi:[10.1177/152660280301000210](https://doi.org/10.1177/152660280301000210).
- 12 25. Alerci M, Giamboni A, Wytttenbach R, Porretta AP, Antonucci F, Bogen M, et
13 al. Endovascular abdominal aneurysm repair and impact of systematic preoperative
14 embolization of collateral arteries: endoleak analysis and long-term follow-up. *J*
15 *Endovasc Ther.* 2013; 20: 663-671.

- 1 26. Aoki A, Maruta K, Hosaka N, Omoto T, Masuda T, Gokan T. Evaluation and
2 coil embolization of the aortic side branches for prevention of Type II endoleak after
3 endovascular repair of abdominal aortic aneurysm. *Ann Vasc Dis* 2017; 10(4): 351-358.
4 doi:[10.3400/avd.oa.17-00088](https://doi.org/10.3400/avd.oa.17-00088).
- 5 27. Li Q, Hou P. Sac embolization and side branch embolization for preventing
6 Type II endoleaks After endovascular aneurysm repair: A meta-analysis. *J Endovasc*
7 *Ther* 2020; 27(1): 109-116. doi:[10.1177/1526602819878411](https://doi.org/10.1177/1526602819878411).
- 8 28. Müller-Wille R, Schötz S, Zeman F, Uller W, Güntner O, Pfister K, et al. CT
9 features of early type II endoleaks after endovascular repair of abdominal aortic
10 aneurysms help predict aneurysm sac enlargement. *Radiology* 2015; 274(3): 906-916.
11 doi:[10.1148/radiol.14140284](https://doi.org/10.1148/radiol.14140284).
- 12 29. Mulay S, Geraedts ACM, Koelemay MJW, Balm R, ODYSSEUS study group.
13 Type 2 endoleak with or without intervention and survival after endovascular aneurysm
14 repair. *Eur J Vasc Endovasc Surg* 2021; 61(5): 779-786. doi:[10.1016/j.ejvs.2021.01.017](https://doi.org/10.1016/j.ejvs.2021.01.017).

- 1 30. Dijkstra ML, Zeebregts CJ, Verhagen HJM, Teijink JAW, Power AH, Bockler
2 D, et al. Incidence, natural course, and outcome of type II endoleaks in infrarenal
3 endovascular aneurysm repair based on the ENGAGE registry data. *J Vasc Surg* 2020;
4 71(3): 780-789. doi:[10.1016/j.jvs.2019.04.486](https://doi.org/10.1016/j.jvs.2019.04.486).
- 5 31. Eden CL, Long GW, Major M, Studzinski D, Brown OW. Type II endoleak
6 with an enlarging aortic sac after endovascular aneurysm repair predisposes to the
7 development of a type IA endoleak. *J Vasc Surg* 2020; 72(4): 1354-1359.
8 doi:[10.1016/j.jvs.2020.01.038](https://doi.org/10.1016/j.jvs.2020.01.038).
- 9 32. Hoshina K, Ishimaru S, Sasabuchi Y, Yasunaga H, Komori K, Japan Committee
10 for Stentgraft Management (JACSM)*. Outcomes of endovascular repair for abdominal
11 aortic aneurysms: A nationwide survey in Japan. *Ann Surg* 2019; 269(3): 564-573.
12 doi:[10.1097/SLA.0000000000002508](https://doi.org/10.1097/SLA.0000000000002508).
- 13
14
15

1 **Legends**

2 Figure 1. Comparison of the distribution of the number of patent lumbar arteries between
3 the patent inferior mesenteric artery (IMA) group and occluded IMA group before and
4 after propensity-score-matching.

5

6 Figure 2. Freedom rate from aneurysm-sac enlargement (≥ 5 mm) compared between the
7 patent inferior mesenteric artery (IMA) group and occluded IMA group before and after
8 propensity-score-matching.

9

10 Figure 3. Freedom rate from reintervention compared between the patent inferior
11 mesenteric artery (IMA) group and occluded IMA group before and after propensity-
12 score-matching.

13

14 Figure 4. Freedom rate from all-cause mortality compared between the patent inferior

1 mesenteric artery (IMA)group and occluded IMA group before and after propensity-
2 score-matching.

3

4 Figure 5. Incidence of overall type-II endoleak (EL), type-II EL from inferior
5 mesenteric artery (IMA), and type-II EL from lumbar artery (LA) compared between
6 the patent inferior mesenteric artery (IMA)group and occluded IMA group after
7 propensity-score-matching.

8

9 Supplementary Figure 1. Study design

10

11 Supplementary Figure 2. Bar charts showing the number of patients with sac
12 enlargement at 5 years after EVAR in relation to the number of patent lumbar arteries in
13 the patent and occluded IMA groups before propensity-score-matching.