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

ABSTRACT

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

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

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

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

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

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

Keywords: Abdominal aortic aneurysm; Endovascular aneurysm repair; Endoleak; Inferior mesenteric artery

INTRODUCTION

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

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

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

enlargement,[12] neither study adjusted the number of patent LAs when comparing results between patients with preoperative patent and occluded IMA. In addition, one RCT reporting the efficacy of embolizing patent IMA[13] did not show the distribution of patent LAs in their cohort. Moreover, the mean follow-up periods in the previous studies were approximately 2–3 years, with little detail on the longer-term results. Therefore, this study aimed to assess the specific influence of a patent IMA on the 7-year results following EVAR.

MATERIALS AND METHODS

Study design

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

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

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

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

Surgical indications

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

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

CT measurement

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

Follow-up protocol

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

Endpoints and definitions

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

Statistical analysis

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

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

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

RESULTS

Patient characteristics before propensity-score-matching

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

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

Results before propensity-score-matching

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

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

EVAR.

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

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

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

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

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

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

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

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

Patient characteristics after propensity-score-matching

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

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

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

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

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

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

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

Matched comparison of the incidence of aneurysm-sac enlargement

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

Matched comparison of the incidence of reintervention

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

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

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

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

Matched comparison of all-cause mortality

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

DISCUSSION

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

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

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

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

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

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

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

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

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

Limitations

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

Conclusion

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

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Legends

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

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

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

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

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

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

Supplementary Figure 1. Study design

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