Inferior Mesenteric Artery Associated Type II Endoleaks: Are They Predictable?

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Abstract

Purpose This study aims to evaluate the relationship between inferior mesenteric artery (IMA) diameter and risk of type II endoleak.

Subjects and Methods A retrospective study design to review all EVARs performed over a 4-year period at a tertiary care center. Out of the total cohort of 400 patients who underwent EVAR, 41 patients (10.3%) developed type II endoleak. The mean IMA ostial diameter for patients with type II endoleak secondary to IMA contributories was 4mm, while the mean IMA diameter for patients with lumbar arteries contributing to the type II endoleak was 3.7mm.

Results Statistical analysis using a paired t-test did not show a statistically significant difference in the IMA ostial diameter between the two groups.

Conclusion There is no significant correlation between preprocedural IMA ostium diameter and type II endoleak development and hence, preprocedural IMA embolization is not an appropriate prophylactic management strategy.

Keywords
- abdominal aortic
- endoleak
- vascular interventional

Introduction

Endovascular aortic aneurysm repair (EVAR) has proven to be viable and less-invasive approach to open surgical repair for both thoracic and abdominal aortic aneurysm treatment. The most common complication association with the endovascular approach is the occurrence of endoleaks. Endoleaks refer to persistent blood flow into the aneurysmal sac despite graft placement and are categorized according to etiology. The most common of these are type II endoleaks that occur due to retrograde blood flow through branch vessels that continue to fill the aneurysmal sac. Type II endoleak is associated with increasing incidence of adverse events such as reintervention, conversion to open repair, and aneurysm rupture. Persistent flow in the inferior mesenteric artery (IMA) is estimated to be responsible for 45 to 85% of retrograde flow within the aneurysmal sac.[1,2] Accordingly, it has been postulated that pre-EVAR IMA embolization reduces the incidence of type II endoleaks.[3–6] However, the cohort of patients and their respective risk factors for whom this intervention will yield maximum benefit is yet to be clearly defined. One of the factors proposed to increase the risk of type II endoleak development is increasing IMA ostium size. As such, this study aims to evaluate whether IMA ostium size is associated with the risk of developing type II endoleak in patients undergoing EVAR for infrarenal abdominal aortic aneurysms at our tertiary center.

Subjects and Methods

This study utilized a retrospective approach to evaluate all EVARs performed over a 4-year period (2014–2018) in a

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Table 1 Results detailing number of patients in each category, mean ostial diameter, and post-EVAR interventions

	Group A IMA + LA	Group B LA	Group C No feeding vessel
Number of patients ($n = 36$)	14 (3—IMA, 11—IMA + LA)	18	4
Mean IMA ostial diameter (mm)	4.0	3.7	2.7
Post-EVAR intervention	**3**—IMA embolization **1**—Open aneurysm repair	**2**—LA embolization	

Abbreviations: EVAR, endovascular aortic aneurysm repair; IMA, inferior mesenteric artery; LA, lumbar artery.

single tertiary center. Information for analysis was gathered using Radiology Information System/Primary and Acute Care System and integrated clinical environment (ICE) clinical systems. The computed tomographic (CT) images pre- and postprocedure have been reviewed by vascular interventional radiologists at our institution. In this study, these scans were further reviewed by vascular interventional radiologists to document the confirmation of type II endoleaks, sac size, patency, and diameter of relevant vessels. All patients ($n = 400$) had a planning CT scan preprocedure followed by regularly scheduled clinic appointments and follow-up scans postprocedure (**Appendix 1**). For the purposes of this study, the number of patients going on to develop type II endoleak were recorded and categorized according to whether the IMA was patent or occluded on preprocedural scan. The patients with a patent IMA on preprocedural scan were further subdivided according to which arteries were contributing to the type II endoleak: group A consisted of patients identified as having IMA or IMA and lumbar arteries (LAs) contributing to the endoleak; group B consisted of patients with only LAs contributing to the endoleak, while group C consisted of patients with no identifiable feeding vessels on scans. In terms of statistical analysis, the significance of the difference between IMA ostial size in groups A and B was quantified using a *t*-test. A *p*-value of ≤ 0.05 was considered to indicate a statistically significant difference.

Results

A total of 400 patients underwent elective EVAR procedures at our tertiary center between 2014 and 2018. Out of 400, a total number of patients with a patent IMA were 355 patients and 45 patients had an occluded IMA or there was lack of opacification of IMA. Out of the total cohort, 41 patients (10.3%) went on to develop type II endoleaks. For those that subsequently developed type II endoleaks, 36 patients (87.8%) were found to have patent IMAs on preprocedural scans, while 5 patients (12.2%) had occluded IMAs before procedure. All the patients that developed a type II endoleak with a sac increases had a patent IMAs ($n = 36$). They were categorized according to feeding vessels contributing to the significant endoleak. Group A (IMA ± LA feeding vessels) consisted of 14 patients (38.8%) with a mean IMA ostial diameter of 4 mm. Three of the group A patients were identified to have only the IMA contributing to the endoleak, while 11 patients had both IMA and LA feeding vessels contributing to the endoleak. A total of three group A patients required post-EVAR intervention with IMA embolization and one patient required open surgical aneurysm repair. Group B (LA contributing vessels only) consisted of 18 patients (50%) with a mean IMA ostial diameter of 3.7mm. Two of these patients subsequently required LA embolization post-EVAR. Group C (no feeding vessels identified) consisted of four patients (12.2%) and had a mean IMA ostial diameter of 2.7 mm (►**Table 1**).

Statistical analysis using a *t*-test to compare the significance of the difference between the mean IMA ostial diameter in group A (4mm) and group B (3.7mm) gave a *p*-value of 0.6.

Discussion

Our study shows that the incidence of type II endoleak resulting in sac increase in patients undergoing EVAR is ~10% that is comparable to the incidence of 10 to 20% reported in the literature.[7–9] Most of these patients (87.8%) were found to have patent IMAs on preprocedural scans and were included in further data analysis. Patients who developed type II endoleak with an occluded IMAs on preprocedural scans without a sac increase were not included in further data analysis. On the whole, our sample size for analysis is small.

From our data, it appears that LAs were more commonly associated with type II endoleak; a cumulative of 29 patients (80.5%) in group A and group B was found to have LAs contributing to endoleak formation. Several studies have concluded that the number and diameter of LAs are independent risk factors with regard to developing type II endoleak. However, the success of preoperative LA embolization has been variable and is possibly hampered by the fact that LA embolization has proven to be a more technically difficult procedure owing to smaller diameters, increased tortuosity, and number of LAs involved.[10–12]

Patients with type II endoleaks secondary to IMA contributors (group A) demonstrated a significantly higher reintervention rate post-EVAR: 28.6 versus a 9% reintervention rate for group B. Three of the group A patients required post-EVAR IMA embolization, while one patient required an open surgical aneurysm repair. There is no consensus currently on which clinical features would be an indication for reintervention in cases of type II endoleak; NICE guidelines in the UK currently recommend intervention only in

cases of persistent type II endoleak with aneurysm sac enlargement.[13]

Current evidence regarding the association between IMA ostial diameter and risk of significant type 2 endoleak is contradictory. Fukuda et al[14] conducted a retrospective study of 120 patients undergoing EVAR and concluded that both persistent and transient type II endoleaks had a proximal IMA diameter of more than 2.5mm in comparison to those without type II endoleak. A further study by Otsu et al[15] suggested that an IMA ostial diameter of more than 2.6mm was an independent risk factor for developing type 2 endoleak. Conversely, Güntner et al[16] performed a retrospective review of 322 patients undergoing EVAR and concluded that there was no significant correlation between preprocedural IMA ostium diameter and type II endoleak development. Similarly, Zhou et al[17] performed a retrospective study of 183 patients who underwent EVAR and did not find any significant correlation between the occurrence of type II endoleaks and IMA diameter. Our study findings are in line with Güntner et al[16] and Zhou et al[17]; there is no significant correlation between preprocedural IMA ostium diameter and type II endoleak development.

Conclusion

Our study demonstrates a higher incidence of type II endoleaks in patients with patent IMA and LAs pre-EVAR. However, in our cohort, the diameter of the IMA does not influence the development of IMA type II endoleaks (p value of 0.6). Unfortunately, the numbers evaluated in the study are small. Based on our finding, there is no clear benefit of preprocedural embolization of the IMA prior to EVAR. Patent LAs are a potential additional risk factor for type II endoleaks. Further evaluation of the relationship between LA diameter and the risk of developing type II endoleaks is required to quantify the potential success of preprocedural LA embolization.

Ethics Approval and Consent to Participate

Informed consent was obtained for the procedure. Institutional board approval was not required for this publication.

Consent for Publication

Patient's informed consent for the use of images and anonymous publication was obtained.

Competing Interests

The authors declare that they have no competing interests. This study was not supported by any funding.

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Appendix 1

Conventional EVAR follow-up regime

Intervals post discharge

Two weeks: Nurse-led clinic, 1st postoperative duplex scan, X-ray of abdomen and iliacs anteroposterior and lateral. Wound check.
Three months: Nurse-led clinic duplex scan.
Six months: Nurse-led clinic duplex scan.
One year: Nurse-led clinic duplex scan, X-ray abdominal and iliacs anteroposterior and lateral.
One to five years: Nurse-led clinic six monthly with duplex scan, yearly X-ray of abdomen and iliacs anteroposterior and lateral.
Five years onwards: Nurse-led clinic yearly with duplex scan and X-ray of abdomen and iliacs anteroposterior and lateral.

