

Comparison of Computed Tomography Angiography (CTA) Findings in Post-Endovascular Aortic Aneurysm Repair CTA between Persistent and Transient Type II Endoleak

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ABSTRACT

Objective: To compare first post-endovascular aortic aneurysm repair (EVAR) computed tomography angiography (CTA) imaging characteristics between transient and persistent type II endoleaks.

Methods: This retrospective study enrolled patients who underwent EVAR and were diagnosed with type II endoleak from first post-operative CTA during January 2005 to October 2017 at the Department of Radiology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand. Aneurysmal sac size, aneurysmal sac growth, and endoleak were recorded among patients whose endoleak disappeared within 6 months (transient group), and among patients whose endoleak persisted for more than 6 months (persistent group).

Results: Eighty-eight patients with a mean age of 75.3 ± 7.3 years were included. Of those, 12 and 76 patients were in the transient group and persistent group, respectively. There were 71 males and 17 females. Univariate analysis showed number of feeding arteries (odds ratio [OR]: 9.9, $p=0.012$) and presence of inferior mesenteric artery (IMA) as an endoleak source (OR: 4.3, $p=0.026$) to be found more frequently in the persistent group than in the transient group; however, neither factor survived multivariate analysis. No significant difference between two groups was seen for endoleak diameter, endoleak complexity, or aneurysmal sac enlargement.

Conclusion: The number of feeder arteries and presence of IMA as an endoleak source on first postoperative CTA to be more likely found in patients with persistent type II endoleak. Further prospective study in a larger study population is necessary to identify any existing statistically significant differences and/or associations.

Keywords: Comparison; computed tomography angiography; CTA; post-endovascular aortic aneurysm repair CTA; persistent and transient type II endoleak (Siriraj Med J 2020; 72: 67-73)

INTRODUCTION

Endovascular aortic aneurysm repair (EVAR) is now generally accepted worldwide as a treatment procedure for abdominal aortic aneurysm because it has a lower rate

of perioperative mortality than open surgical repair.^{1,2} However, despite EVAR being shown to have superior perioperative survival advantage, its most well-known complication – endoleak, which is defined as continuous

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Received 16 August 2019 Revised 30 September 2019 Accepted 30 October 2019

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<http://dx.doi.org/10.33192/Smj.2020.09>

perfusion within the aneurysm sac – can cause aneurysmal sac expansion that can lead to compromised long-term survival outcome.

Type II endoleaks originate from retrograde flow from collateral arteries to the aneurysmal sac. Typical aortic side branches that cause type II endoleaks are the inferior mesenteric artery (IMA), the lumbar arteries (LAs), the median sacral artery, and the accessory renal arteries. Early-onset type II endoleak is defined as an endoleak found within 90 days after EVAR, while late-onset type II endoleak is detected after 90 days. Early-onset type II endoleak can be classified as transient (one that spontaneously resolves within 6 months) or persistent (endoleak that lasts longer than 6 months after EVAR) type II endoleak.

Persistent type II endoleaks have been reported as a cause of late adverse outcome including aneurysmal sac diameter enlargement, the need for switching to open repair/ reintervention and rupture.³ Researchers have investigated for factors that predict persistent type II endoleaks. In addition to demographic and patient characteristics that have been discussed and reported in previous studies as predictors of persistent type II endoleak⁴, several other studies examined preprocedural imaging findings to identify factors that predict this particular variety of endoleak.⁵⁻⁹

The objective of this study was to identify the relation of computed tomography angiography (CTA) findings in first post-EVAR CTA between transient and persistent type II endoleaks.

MATERIALS AND METHODS

Patients

This study was approved by the Siriraj Institutional Review Board (SIRB) of the Faculty of Medicine Siriraj Hospital, Mahidol University (Si 051/2018). The study retrospectively enrolled patients who were diagnosed with type II endoleak from first post-operative CTA during January 2005 to October 2017 at the Department of Radiology, Faculty of Medicine Siriraj Hospital, Mahidol University. The first post-operative CTA should be performed within 90 days after EVAR to evaluate for early-onset type II endoleak, and the next post-operative CTA should be performed at least 6 months after EVAR according to the criteria for persistent type II endoleak. A search of our center's database and radiology reporting system revealed 88 patients (mean age 75.3 ± 7.3 years, 71 males and 17 females) that met the inclusion criteria. Patients with other types of endoleak or who were lost to follow-up were excluded.

Imaging study

Two sessions of CTA were interpreted. The first postoperative CTA within the first 90 days after EVAR was used for measurement of CTA findings, and the follow-up CTA was used to assess the outcome. CT scans were obtained using helical CT scanners (1. Somatom Definition dual source CT; Siemens, Forchheim, Germany or 2. Discovery CT 750HD; GE Healthcare, Milwaukee, USA or 3. Revolution CT, GE Healthcare, Milwaukee, USA). The CTA protocol consists of unenhanced CT scan, contrast-enhanced CTA phase, and delayed phase. Contrast-enhanced CTA phase was used by bolus-tracking technique with a threshold of 150 Hounsfield units (HUs) at abdominal aorta proximal to the endovascular stent graft. Delayed phase was performed 2 minutes after bolus trigger.

Imaging interpretation

Evaluation and measurement of CTA findings were performed by two radiologists who separately interpreted the image findings on our center's Picture Archive Communication System (PACS). The following CTA findings were analyzed from the first postoperative CTA: size of the aneurysmal sac, name of feeder artery and total number of arteries feeding the endoleak, endoleak complexity, diameter of endoleak, attenuation of the endoleak, and attenuation of aorta.

Aneurysmal sac diameter was measured using electronic caliper to the nearest millimeter. Measurement of the orthogonal sac diameter using double oblique short axis was performed (Fig 1). The number of feeder arteries included all visible arterial feeders. A common vessel of lumbar arteries, and a common vessel of the fourth lumbar artery and the median sacral artery were counted as one feeding artery. Endoleak complexity was classified into simple type and complex type. A simple type endoleak was defined as an endoleak that is fed by a single artery, while a complex type endoleak was defined as being fed by two or more arteries. Measurement of endoleak diameter was performed in axial plane of delayed phase CT images. The maximal diameter was measured and recorded (Fig 2). Attenuation of endoleak and aorta was measured using the greatest possible circular region of interest (ROI) on both CTA and delayed phases in Hounsfield units. Relative attenuation of endoleak was calculated using the following formula: (endoleak cavity attenuation on CTA and delayed phases – endoleak cavity attenuation on unenhanced images) / (attenuation of stent graft lumen on CTA and delayed phases – attenuation of stent graft lumen on unenhanced images). The outcome

(transient or persistent type II endoleak and aneurysmal sac diameter enlargement) was recorded based on the findings from CTA study performed 6 months after EVAR.

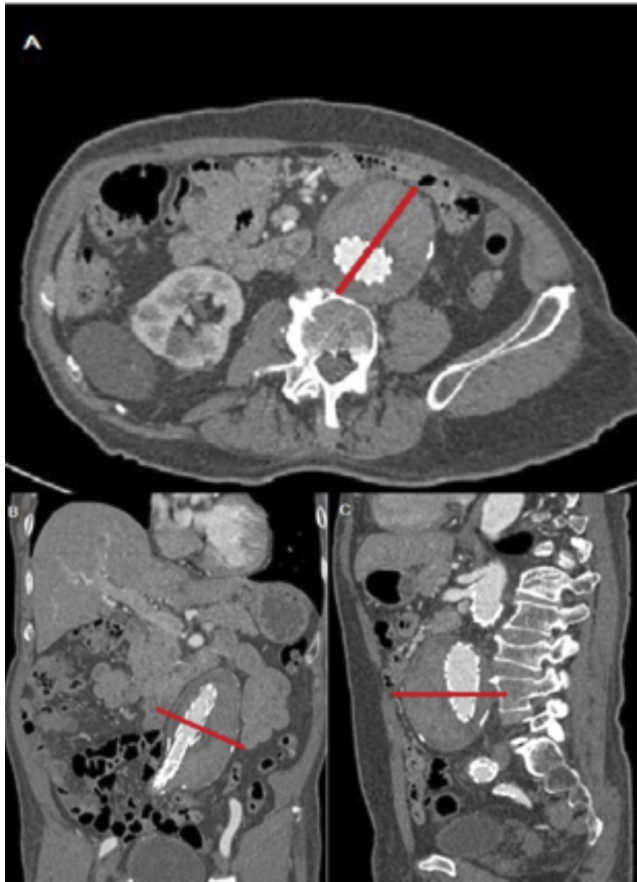


Fig 1. Post-EVAR CTA images show the method used to measure the aneurysmal sac. (A) Double oblique axial view used to measure the orthogonal diameter of the aneurysm. (B) and (C) Coronal and sagittal reformatted images, respectively.



Fig 2. Post-EVAR CTA, delayed phase, axial plane image demonstrates measurement of endoleak diameter.

Statistical analysis

All data analyses were performed using SPSS Statistics software (SPSS, Inc., Chicago, IL, USA). Values are shown as mean and/or standard deviation or number and percentage, as appropriate. Univariate analysis was performed using chi-square test, *t*-test or Mann-Whitney U test, and multivariate analysis was done using logistic regression analysis to identify factors independently associated with persistent type II endoleak. Interobserver agreement of aneurysmal sac diameter measurement, number of feeder arteries, endoleak diameter measurement, and attenuation measurement was calculated using intraclass correlation coefficient (ICC, *r*). The *r* values were classified, as follows: 1.0, perfect agreement; 0.81-0.99, almost perfect agreement; 0.61-0.80, substantial agreement; 0.41-0.60, moderate agreement; 0.21-0.40, fair agreement; and, less than 0.2, slight agreement. Interobserver agreement of endoleak complexity, presence of each feeder, and aneurysmal sac enlargement were calculated using kappa statistic. The kappa values (*k*) were interpreted, as follows: 0.81-1.00, very good agreement; 0.61-0.80, good agreement; 0.41-0.60, moderate agreement; 0.21-0.40, fair agreement; and, less than 0.20, poor agreement. A *p*-value < 0.05 was considered statistically significant.

RESULTS

Patients

Eighty-eight patients were included. There was no significant difference in age between the transient and persistent groups (73.92 ± 7.15 years vs. 75.57 ± 7.30 years, respectively). Of the 88 early-onset type II endoleak patients, 12 patients had spontaneous resolution (transient group), and 76 patients still had type II endoleak on the 6-month follow-up CTA (persistent group). There was no significant difference in gender distribution between groups ($p=0.236$). Patient demographic data are shown in Table 1.

Endoleak characteristics

Initial mean aneurysmal sac diameter in the transient group and persistent group was 57.0 ± 11.57 mm and 58.92 ± 14.34 mm, respectively ($p=0.66$). In terms of endoleak complexity, simple endoleak was found in 7 transient type II endoleak patients, and in 24 persistent type II endoleak patients. Complex endoleak was observed in 5 transient type II endoleak patients, and in 52 persistent type II endoleak patients ($p=0.103$).

The number of arterial feeders of endoleak was separated into two groups: 2 or less arterial feeders, and more than 2 feeders. The transient type II endoleak

TABLE 1. Patient characteristics.

	Type II endoleak during follow-up		P-value
	Transient (n=12)	Persistent (n=76)	
Mean age (years)	73.92±7.15	75.57±7.30	0.468
Gender			
Male	8 (66.7%)	63 (82.9%)	0.236
Female	4 (33.3%)	13 (17.1%)	

A *p*-value<0.05 indicates statistical significance

group had a significantly greater proportion of 2 or less arterial feeders than the persistent group (91.7% vs. 52.6%, respectively; odds ratio [OR]: 9.9, 95% confidence interval [CI]: 1.217-80.526; *p*=0.012).

Regarding endoleak sources (different arterial feeders), presence of inferior mesenteric artery (IMA) was found more frequently in the persistent group (68.4%) than in the transient group (33.3%) (OR: 4.3, 95% CI: 1.18-15.8; *p*=0.026). However, there was no significant difference between two groups for presence of lumbar artery (LA), median sacral artery, or accessory renal artery as an arterial feeder of type II endoleak.

Endoleak diameter showed no significant difference between groups. Median diameter of type II endoleak in the transient group and persistent group was 15 mm and 13 mm, respectively (*p*=0.87). The mean attenuation of the endoleak cavity was not significantly different between the transient and persistent groups in unenhanced (43.0 vs. 43.8 HUs), arterial (281.6 vs. 273.7 HUs), and delayed (140.1 vs. 128.9) phases. The relative attenuation of endoleak in both the arterial and delayed phases was also not significantly different between two groups. The data are shown in Table 2. Regarding aneurysmal sac enlargement, the persistent type II endoleak group had 6 patients (7.9%) with enlarged aneurysmal sac, while the transient type II endoleak group had no patients (0%) with enlarged aneurysmal sac (*p*=0.591).

Multivariate analysis

Univariate analysis revealed statistically significant differences in three variables, including endoleak complexity, number of arterial feeders, and presence of inferior mesenteric mesentery feeder. However, when entered into multivariate analysis, none of these factors was found to be an independent predictor of persistent type II endoleak. The odd ratio for endoleak complexity, number of arterial feeders, and presence of inferior mesenteric feeder was 1.302 (95% CI: 0.318-5.333), 5.532

(95% CI: 0.487-62.797), and 2.285 (95% CI: 0.557-9.365), respectively (Table 3).

Interobserver agreement

Intraclass correlation coefficient (ICC) revealed almost perfect agreement for aneurysmal sac diameter measurement (*r*=0.961) and endoleak diameter measurement (*r*=0.922). There was substantial agreement in the counting of the number of arterial feeders (*r*=0.635). Attenuation measurement was moderate to substantial agreement (*r*=0.413-0.781). Cohen's kappa was used to calculate the kappa value. Presence of type II endoleak on follow-up study, endoleak complexity, and identification of arterial feeders showed good agreement (*k*=0.779-0.855). Aneurysmal sac enlargement evaluation showed moderate agreement (*k*=0.482).

DISCUSSION

A few studies^{10,11} investigated type II endoleak and found no significant association relative to age, gender, or initial postoperative aneurysmal sac diameter in both transient and persistent type II endoleak – all of which is similar to our study.

Type II endoleaks can be classified into two subtypes, simple and complex subtypes based on number of inflow/outflow vessels. The authors compared simple and complex type II endoleak and found complex type II endoleak to be observed more frequently in the persistent group (68.4%) than in the transient group (41.7%). However, the difference between two groups was not statistically significant. This result is not directly comparable to that reported by Muller-Wille R, *et al.*⁸ because they used a different method of outcome measurement. Muller-Wille R, *et al.*⁸ classified simple and complex type II endoleak into four subtypes, and they found complex IMA-LA subtype to be significantly associated with aneurysmal sac enlargement (OR: 20.8, 95% CI: 4.9-88.9; *p*<0.001).

TABLE 2. Endoleak CTA characteristics.

	Type II endoleak during follow-up		Odds ratio (95% CI)	P-value
	Transient (n=12)	Persistent (n=76)		
Aneurysmal sac diameter (mm), mean±SD	57.0±11.57	58.92±14.34		0.66
Endoleak complexity, n (%)				
Simple	7 (58.3%)	24 (31.6%)	3.03(0.87-10.5)	0.10
Complex	5 (41.7%)	52 (68.4%)		
Number of feeders, n (%)			9.9 (1.217-80.526)	0.012
≤2	11 (91.7%)	40 (52.6%)		
>2	1(8.3%)	36 (47.4%)		
Endoleak source, n (%)				
IMA	4 (33.3%)	52 (68.4%)	4.3 (1.18-15.8)	0.026
LAs	10 (83.3%)	60 (78.9%)	0.75 (0.15-3.77)	1
Median sacral artery	0 (0.0%)	2 (2.6%)	0.86 (0.79-0.94)	1
Accessory renal artery	0 (0.0%)	2 (2.6%)	0.86 (0.79-0.94)	1
Endoleak diameter (mm), median (range)	15 (6, 23)	13 (3, 62)		0.87
Attenuation (HU±SD)				
Unenhanced	43±9.4	43.86±13.77		0.835
Arterial phase	281.58±100	273.7±104.2		0.807
Delayed phase	140.1±27.14	128.9±27.47		0.257
Relative attenuation CTA	0.638±0.278	0.693±0.394		0.64
Relative attenuation delay	1.058±0.601	0.914±0.307		0.248
Sac enlargement, n (%)	0 (0.0%)	6 (7.9%)	0.854 (0.78-0.934)	0.591

A *p*-value<0.05 indicates statistical significance

Abbreviations: CTA, computed tomography angiography; CI, confidence interval; SD, standard deviation; IMA, inferior mesenteric artery; LAs, lumbar arteries; HU, Hounsfield unit

TABLE 3. Multivariate analysis.

	Odd ratios (95%CI)	P-value
Complexity	1.302 (0.318-5.333)	0.714
Number of feeders	5.532 (0.487-62.797)	0.168
IMA feeder	2.285 (0.557-9.365)	0.251

A *p*-value >0.05 indicates statistical significance

Abbreviations: CI, confidence interval; IMA, inferior mesenteric artery

TABLE 4. Interobserver agreement.

	Intraclass correlation coefficient (ICC, r)
Sac diameter	0.961
Number of feeders	0.635
Endoleak diameter	0.922
Attenuation measurement	0.781
	Kappa value (k)
Presence of type II endoleak	0.782
Endoleak complexity	0.779
Presence of each feeder	0.855
Aneurysmal sac enlargement	0.482

Number of feeder arteries showed a significantly greater proportion of 2 or less arterial feeders in the transient group (91.7%) than in the persistent group (52.6%); $p = 0.012$. The authors found this result to be similar to that from a study by Maeda, *et al.*¹⁰ Maeda, *et al.*¹⁰ found multiple vessels responsible for type II endoleak to be a significant factor for prediction of persistent type II endoleak. In the present study, that relationship did not survive multivariate analysis. This difference in results may be due to the small size of our transient group that influenced a widely ranging 95% confidence interval.

Regarding sources of type II endoleak, we found a significantly greater proportion of inferior mesenteric artery feeder in the persistent group (68.4%) than in the transient group (33.3%). According to a study by Muller-Wille R, *et al.*⁸ (despite this result being not directly comparable due to differences in outcome measurement between studies), presence of inferior mesenteric artery as a feeder artery was shown to be more frequent in the aneurysm enlargement group than in the non-enlargement group.

In contrast to Timaran, *et al.*¹¹, Keedy, *et al.*¹², and Dudeck, *et al.*¹³, the endoleak diameter measured on axial CT in our study was not significantly different between the groups. Timaran, *et al.*¹¹ found maximum endoleak cavity diameter more than 15 mm to be associated with increased risk of increase aneurysmal sac diameter (relative risk [RR]: 11.1, 95% CI: 1.4-85.8; $p=0.02$). Keedy, *et al.*¹² reported transverse diameter of the endoleak cavity in the intervention group to be significantly greater than in the nonintervention group (1.85 ± 1.01 vs. 1.13 ± 0.83 cm, respectively; $p=0.007$). Dudeck, *et al.*¹³ also identified

diameter and area of endoleak nidus in the reintervention group to be significant greater than in the surveillance group. However, our endoleak diameter study result was similar to reports from Mursalin, *et al.*⁷ and Muller-Wille R, *et al.*⁸, both of which reported that simple evaluation of diameter and area of endoleak cavity was not of predictive value.

Attenuation measurement of endoleak from our study showed no significant difference between that transient and persistent groups for both absolute measurement and relative attenuation. The results of absolute attenuation measurement corresponded to a report from Muller-Wille R, *et al.*⁸ that reported no significant difference between the no aneurysm enlargement group and the aneurysm enlargement group.

Presence of aneurysmal sac enlargement in the present study showed no significant difference between the transient and persistent groups, and this corresponds with the findings of Kaley Pippin, *et al.*¹⁴ However, both our study and that study had a small sized study population.

Limitations

This study has three major limitations. First, the retrospective design of this study made it difficult to control the CT protocol. Second, the small number of patients in the transient type II endoleak group may have adversely affected the ability of our statistical analysis to detect significant differences between groups. Third, our results cannot be applied in late-onset type II endoleak because we included only patients with early-onset type II endoleak.

CONCLUSION

The results of this study revealed endoleak complexity, the number of feeder arteries and presence of IMA as an endoleak source on first postoperative CTA to be more likely found in patients with persistent type II endoleak. Further prospective study in a larger study population is needed to identify any existing statistically significant differences and/or associations.

ACKNOWLEDGMENTS

The authors gratefully acknowledge Miss Julaporn Pooliam of the Division of Clinical Epidemiology, Department of Research, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand for assistance with statistical analysis.

Conflict of interest declaration: Both authors declare no personal or professional conflicts of interest, and no financial support from the companies that produce and/or distribute the drugs, devices, or materials described in this report.

Funding disclosure: This was an unfunded study.

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