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# A systematic review of anatomic predictors of abdominal aortic aneurysm remodeling after endovascular repair

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## ABSTRACT

**Objective:** The long-term outcomes after endovascular abdominal aneurysm repair (EVAR) of abdominal aortic aneurysms (AAAs) have been inferior to those after open surgical repair with regard to reinterventions and late mortality. AAA sac remodeling after EVAR has been associated with endoleaks, reinterventions, and mortality. Therefore, knowledge of the predictors of AAA sac remodeling could indirectly give insight into the long-term EVAR outcomes. In the present review, we aimed to provide an overview of the evidence for anatomic predictors of positive and negative AAA sac remodeling after EVAR.

**Methods:** A systematic literature review and analysis were conducted in accordance with the PRISMA (preferred reporting items for systematic reviews and meta-analyses) and Cochrane guidelines. The PubMed and Scopus databases were searched using terms of AAA sac growth, shrinkage, and remodeling. Eligible studies were identified, and only those studies that had included currently used endografts were included.

**Results:** A total of 19 studies that had reported on a total of 27 anatomic parameters of the aortoiliac anatomy were included. Only 4 parameters had been investigated by more than five studies, 7 parameters were investigated by three to five studies, 7 parameters were investigated by two studies, and 9 parameters were investigated by one study. For the presence of neck thrombus, three of four studies had reported similar results, indicating that the presence of neck thrombus might predict for less AAA sac shrinkage. AAA thrombus, the total AAA volume, the flow-lumen volume, aortic calcification, and the number of hostile neck parameters were only investigated by two to three studies. However, these parameters seemed promising for the prediction of sac remodeling. For hostile neck anatomy, neck length, infrarenal neck angulation, and patency of the inferior mesenteric artery, no significant association with any category of AAA sac remodeling was found.

**Conclusions:** The present review demonstrates neck thrombus, AAA thrombus, number of hostile neck parameters, total AAA volume, AAA flow-lumen volume, and aortic calcification as important anatomic features that are likely to play a role in AAA remodeling after endovascular repair and should be further explored using advanced imaging techniques. We also found that strong, consistent evidence regarding the anatomic predictors of AAA sac remodeling after EVAR is lacking. Therefore, further research with large patient groups for a broad range of predictors of AAA sac change after EVAR is needed to complement the current gap in the evidence. (J Vasc Surg 2022;75:1777-85.)

**Keywords:** Abdominal aortic aneurysm; Aneurysm remodeling; Endovascular aneurysm repair; Predictors

The goal of endovascular abdominal aneurysm repair (EVAR) is to prevent aneurysm growth and the mortality caused by aneurysm rupture. When comparing EVAR with open surgical repair, EVAR has shown an early benefit with regard to morbidity and mortality, but the long-term reintervention rate, rupture risk, and mortality rates are increased.[1,2] The reasons for the increased mortality after EVAR are still poorly understood, because most patients have died of cardiac and pulmonary disease.[3] The treatment success of EVAR includes the prevention of aneurysm rupture, death from aneurysm rupture, and aneurysm-related death that can result from primary or secondary treatment.[4]

Abdominal aortic aneurysm (AAA) sac expansion is thought to represent treatment failure, because growth reflects an increased pressure within the AAA, indicating that the patient is still at risk of rupture-related death, despite the intervention.[5] The reporting standards of the Society for Vascular Surgery have defined significant sac size change as a diameter change of ≥5 mm or a volume change of ≥5% owing to the intra- and interrater variability of these measurements.[4]

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A recent study showed that 8.5% of 1060 EVAR patients experienced significant sac expansion at some point during follow-up.[6]

A stable sac diameter has traditionally been associated with treatment success. O'Donnell et al,[7] however, reported that, not only sac expansion, but also any failure of the sac to regress is associated with higher long-term mortality, independent of reinterventions or the presence of endoleaks. This was recently confirmed in a meta-analysis, which also showed that AAA shrinkage at 1 year after EVAR correlated with a significantly lower rate of reinterventions, late complications, and rupture.[8] This has raised the question of whether a stable AAA diameter after EVAR can still be considered as success.[7]

The absolute mean sac shrinkage was found to increase over the years to up to −14.9 mm at >10 years, with most of the shrinkage occurring within the first 2 years after EVAR.[6] Approximately 80% of the patients had experienced sac shrinkage of >5 mm at 4 years after treatment.[6] Sac shrinkage after EVAR generally indicates successful exclusion of the AAA from the circulation and predicts a low risk of failure during the first 5 postoperative years.[9,10] Houbballah et al[10] reported that patients with AAA sac shrinkage >75% had significantly lower rates of endoleak types I and II (2.2% vs 15.4%; $P < .001$; and 3.3% vs 29.4%; $P < .001$) and reinterventions (3.3% vs 13.3%; $P < .05$) compared with patients without shrinkage. Furthermore, none of the 92 patients with shrinkage had experienced aneurysm rupture after a mean follow-up of 26 ± 21 months.[10]

AAA sac remodeling after EVAR is an important factor that has been associated with the development of new endoleaks, the requirement for reintervention, and long-term mortality.[7] Sac remodeling has even been proposed as a more sensitive assessor of AAA exclusion than the absence of endoleaks.[6] Because sac remodeling has been correlated with long-term EVAR outcomes, finding the predictors of AAA sac remodeling is important. Many separate studies have already analyzed a broad range of parameters for their relationship to AAA sac remodeling. However, the current evidence as a whole has not yet been analyzed. Because the spectrum of these parameters is quite large, the synthesis of the total evidence must be performed by category. In the present study, we aimed to systematically review all the available evidence on anatomic predictors of AAA sac remodeling after EVAR.

## METHODS

A systematic review was conducted of the results obtained for factors that influence post-EVAR AAA sac remodeling. The approach of the present systematic review was based on the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statements, Cochrane guidelines, and the recently reported stepwise approach for systematic reviews on endovascular interventions of Antoniou et al.[11-13] Before the full-text screening, a detailed protocol of our systematic review was registered in the PROSPERO (Prospective Register of Systematic Reviews; identification no. CRD42020201422).

**Databases and search terms.** The search interfaces Scopus (Elsevier BV, Amsterdam, The Netherlands) and PubMed (National Center for Biotechnology Information, Bethesda Md) were used to access MEDLINE, EMBASE, PubMed Central, and National Center for Biotechnology Information bookshelf archives. The free-text search terms "AAA," "abdominal aortic aneurysm," "EVAR," "sac regression," "sac reduction," "sac expansion," "sac enlargement," "sac change," "sac behavior," "sac shrinkage," "sac growth," and "sac remodeling" were used in combination with the Boolean operators "AND" and "OR."

**Study selection.** The studies found from all the database searches were compiled using Mendeley software (Elsevier B.V., Amsterdam, The Netherlands), and all duplicates were removed using Microsoft Excel, version 2011 (Microsoft Corp, Redmond, Wash) with the Ablebits add-in (4Bits Ltd, Gomel, Belarus). No search was performed to retrieve any unpublished data or abstracts. The title and abstracts of all the studies were reviewed for the full-text assessment by two of us (E.G.J. and R.V.R.) to make a decision for full-text inclusion. Next, a full-text assessment was performed by the same two investigators (E.G.J. and R.V.R.). Any disagreements were resolved by screening by a third investigator (M.R.).

Studies that had reported evidence of the factors that did or did not have predictive value for AAA sac remodeling after EVAR were included. The studies were required to have reported original patient data and to have included ≥10 patients with an infrarenal, unruptured AAA who had undergone EVAR. Only studies of devices that are currently on the market were included. These devices included the Excluder (W.L. Gore & Associates, Flagstaff, Ariz), Endurant (Medtronic, Minneapolis, Minn), Zenith (Cook Medical, Bloomington, Ind), Incraft (CardinalHealth, Dublin, Ireland), AFX (Endologix, Irvine, Calif), Anaconda (Terumo Aortic, Inchinnan, UK), and Aorfix (Lombard, Oxfordshire, UK) endografts. Many studies had described a mixed use of devices; therefore, ≥90% of the included devices had to be currently on the market. Finally, the reports had to have been written in the English or Dutch language.

Studies were excluded if they had included <10 patients or had included patients with ruptured, juxtarenal, or suprarenal AAAs, thoracoabdominal aneurysms, or thoracic aneurysms. In addition, studies reporting treatment with endografts that are not currently used in practice, fenestrated or branched endografts, iliac branched devices, endovascular aneurysm sealing, or chimney procedures were excluded. Also, studies were excluded that

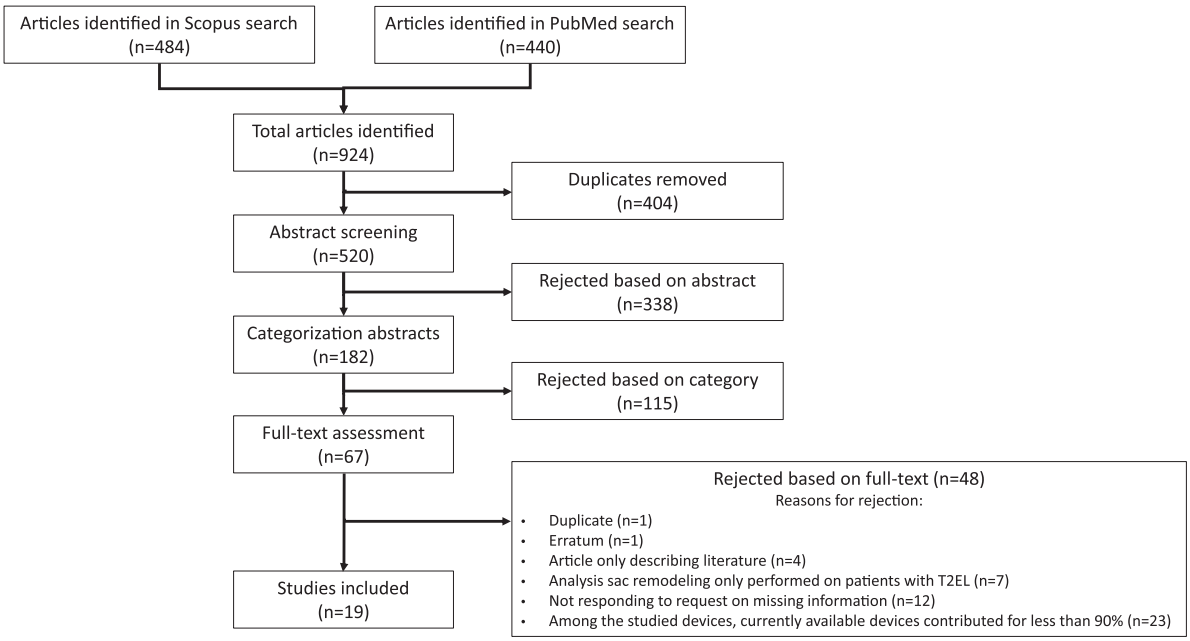


**Fig.** Flow chart illustrating study selection.

had included only patients with type II endoleaks, because of the high risk of bias.

**Assessment of study quality and risk of bias.** The risk of bias and study quality of the included studies was examined using the QUIPS (Quality In Prognostic Studies) tool by one of us (R.V.R.).[14] This assessment considers bias in six domains, including study participation, study attrition, prognostic factor measurement, outcome measurement, confounding factor assessment, and statistical analysis and reporting.[14] Low risk in a domain indicates that it is unlikely that this domain caused bias, a moderate risk indicates that this domain might induce bias, and a high risk indicates that this domain is very likely to cause bias.[14] Furthermore, the risk of bias across studies was assessed.

**Data extraction and processing.** Data were extracted from the included studies by two of us (R.V.R. and E.G.J.). The anatomic parameters and their correlation with AAA remodeling, as reported in the included studies, were compiled into tabular format. The parameters were grouped by common themes, such as AAA neck anatomy, preoperative AAA size, aneurysm thrombus, aortic calcification score, patency of aortic side branches, and iliac anatomy. Relevant findings of the anatomic predictors have been described in more detail. Parameters that were investigated in the reported studies but that did not show any effect on AAA sac remodeling have also been presented to achieve a complete overview of the evidence. Hostile neck anatomy (HNA) was defined as the presence of one or more of the following parameters: neck diameter >28 mm, neck length <15 mm, neck angulation >60°, presence of neck thrombus, and/or the presence of a reverse tapered neck.[15]

## RESULTS

**Study selection.** The initial search yielded 520 distinct reports, of which 338 were removed after the title and abstract review (Fig). The remaining studies were categorized, and those reporting on nonanatomic predictors were rejected. A total of 67 studies of anatomic predictors were subjected to the full-text evaluation, with 19 studies included in the final dataset. A detailed overview of the included studies is presented in Table I, which also illustrates the variety in the definition and measurement methods of AAA sac change used by the studies.

The estimated risk of bias for every included study is presented in Table II. Study participation, assessment of confounding factors, and statistical analysis and reporting were found to be more likely to attribute to bias than were the other domains. Most studies were found to have a moderate risk of bias in the study participation domain owing to the low number of included patients, the use of only one type of endograft, or the exclusion of patient subgroups. Most studies also had a moderate risk of bias in the confounding factor domain because the reporting and/or assessment of potential confounding factors were deemed to be insufficient. The statistical analysis had resulted in a moderate risk of bias in some studies because they had only reported the factors with a significant association with AAA sac remodeling, and it was unclear whether other variables had also been studied.

**Table I.** Studies included in current review

| Investigator | Country or region | Inclusion period | Patients, No. | EGs used, % | Timeframe used to determine sac change (T1 vs T2) | Definition of AAA sac growth and/or shrinkage, mm |
|---|---|---|---|---|---|---|
| Nishibe et al,[16] 2020 | Japan | October 2013 to May 2017 | 155 | 100 | Preoperative vs 1 and 2 years | ≥5 |
| Hori et al,[17] 2019 | Japan | August 2008 to December 2014 | 135 | 93.3 | Preoperative vs 3 years | ≥5 |
| Jeong et al,[18] 2019 | Korea | January 2008 to December 2015 | 168 | 93.5 | Preoperative vs last follow-up CTA (median, 44 months) | ≥5 |
| Muhs et al,[19] 2018 | Multicenter, 43 sites in USA and Europe | June 2012 to September 2014 | 99 | 90.9 | Preoperative vs 1 and 2 years | ≥5 |
| Lee et al,[20] 2018 | Korea | January 2012 to December 2017 | 38 | 100 | Preoperative vs last follow-up CTA (mean, 11.3 ± 13.5 months) | NR |
| Marie et al,[21] 2018 | France | February 2010 to March 2013 | 33 | 97.0 | Preoperative vs 6 months | AAA volume as continuous parameter and categorized as mild in first tercile (<29 mL), mid-to-moderate in second tercile (29-56 mL), and pronounced in third tercile (>56 mL) |
| Malas et al,[22] 2017 | USA | April 28, 2006 to September 30, 2011 | 218 | 100 | Preoperative vs 5 years | ≥5 |
| Giménez-Gaibar et al,[23] 2017 | Spain | 2006 to 2013 | 127 | 98.4 | Preoperative vs 1, 6, 12, and 24 months | ≥5 |
| McGillicuddy et al,[24] 2017 | USA | 2006 to 2011 | 218 | 100 | Preoperative vs 1 and 4 years | ≥5 |
| De Haro et al,[25] 2016 | Spain | NR | 192 | 100 | 6 vs 18 months after EVAR | Annual sac expansion rate, 5.7% |
| Kim et al,[26] 2016 | Korea | April 2005 to July 2013 | 97 | 100 | Preoperative vs mean follow-up: 45.8 ± 23.3 months | ≥5 |
| Hiraoka et al,[27] 2015 | Japan | January 2006 to May 2011 | 151 | 92.6 | Preoperative vs not described | ≥5 |
| Nakai et al,[28] 2015 | Japan | February 2008 to February 2014 | 143 | 92.3 | Preoperative vs mean follow-up: 12 months | ≥5 |
| Iwakoshi et al,[29] 2014 | Japan | July 1999 to August 2011 | 127 | 100 | First postoperative CTA vs median follow-up: 43 months | ≥5 |
| Welborn et al,[30] 2014 | USA | June 2011 to February 2013 | 108 | 100 | CTA within 3 months after EVAR, or, if unavailable, preoperative vs mean follow-up: 5 ± 2 months | ≥5 |
| Bastos Gonçalves et al,[31] 2012 | The Netherlands | January 2000 to December 2007 | 144 | 100 | 30 days postoperatively vs mean follow-up: 5 years | ≥5 |
| Greenberg et al,[32] 2008 | USA | 2000-2003 | 739 | 100 | Not clearly defined | ≥5 |
| Fairman et al,[33] 2006 | USA | 2000-2001 | 351 | 100 | Predischarge CTA vs 1, 6, 12, and 24 months | ≥5 |
| Love et al,[34] 2005 | UK | December 1998 to February 2002 | 57 | 95.9 | Preoperative vs 6 months | Mean sac shrinkage between groups of calcification grades |

*AAA*, Abdominal aortic aneurysm; *CTA*, computed tomography angiography; *EGs*, endografts used in current clinical practice; *EVAR*, endovascular aneurysm repair; *NR*, not reported; *T1*, first evaluation; *T2*, second evaluation.

**Table II.** Bias and study quality assessment of included studies using QUIPS tool

| Investigator | Study participation | Study attrition | Prognostic factor measurement | Outcome measurement | Study confounding | Statistical analysis and reporting |
|---|---|---|---|---|---|---|
| Nishibe,[16] 2020 | Low | Moderate | Low | Low | Low | Low |
| Hori,[17] 2019 | Low | Low | Low | Low | Moderate | Moderate |
| Jeong,[18] 2019 | Moderate | Low | Low | Low | Moderate | Low |
| Muhs,[19] 2018 | Low | Low | Low | Low | Moderate | Low |
| Lee,[20] 2018 | Moderate | Low | Low | Low | Moderate | Low |
| Marie,[21] 2018 | Moderate | Low | Moderate | Moderate | Moderate | Moderate |
| Malas,[22] 2017 | Moderate | Moderate | Low | Low | Moderate | Moderate |
| Giménez-Gaibar,[23] 2017 | Low | Low | Low | Low | Moderate | Moderate |
| McGillicuddy,[24] 2017 | Low | Low | Low | Low | Low | Low |
| De Haro,[25] 2016 | Moderate | Low | Low | Low | Low | Low |
| Kim,[26] 2016 | Moderate | Low | Low | Low | Moderate | Low |
| Hiraoka,[27] 2015 | Moderate | Low | Low | Low | Moderate | Moderate |
| Nakai,[28] 2015 | Low | Low | Low | Low | Low | Low |
| Iwakoshi,[29] 2014 | Moderate | Low | Low | Low | Moderate | Moderate |
| Welborn,[30] 2014 | Moderate | Low | Low | Low | Moderate | Moderate |
| Bastos Gonçalves,[31] 2012 | Moderate | Low | Low | Low | Moderate | Moderate |
| Greenberg,[32] 2008 | Moderate | Low | Low | Low | Moderate | Low |
| Fairman,[33] 2006 | Moderate | Low | Low | Low | Low | Low |
| Love,[34] 2005 | Low | Low | Low | Low | Moderate | Moderate |

**Anatomic predictors.** The main findings from each study on the correlation of the anatomic parameters with AAA sac growth and shrinkage are presented in Table III.[16-34] A total of 27 anatomic parameters were investigated in the 19 studies. Of the 27 parameters, 4 were investigated by more than five studies, 7 were investigated by three to five studies, 7 were investigated by two studies, and 9 were investigated by just one study.

Preoperative neck thrombus was reported to predict for less AAA sac shrinkage after EVAR.[19,24,26,33] The presence of preoperative neck thrombus was found to be predictive of less AAA shrinkage at 6, 12, and 24 months after EVAR in two independent studies ($P < .05$).[19,33] Also, the absence of neck thrombus was reportedly related to faster shrinkage of the AAA maximum diameter ($P < .01$).[33]

For the grade of neck thrombus, mixed results were found. When scored as 1 (mild) to 3 (severe), a higher thrombus grade was significantly related to greater sac enlargement at 4 years after EVAR (odds ratio, 0.11; 95% confidence interval [CI], 0.02-0.76; $P = .02$), which had not been present at 1 year after EVAR.[24] Also, when the neck thrombus grade was expressed as the absolute degrees of the neck thrombus circumference in a different population, a high degree was significantly more present in the patients without AAA shrinkage ($P = .003$).[19] However, when scored from 0 to 3 in another study, no significant association between the grade of AAA neck thrombus and AAA sac growth was found after a mean follow up of 45.8 ± 23.3 months.[26] Thrombus thickness in the neck was not found to be significantly associated with AAA sac remodeling.[19]

Although they were only investigated by two to three studies, the number of hostile neck parameters, total AAA volume, AAA flow-lumen volume, AAA thrombus, and aortic calcification seemed promising for predicting AAA remodeling. For the hostile neck, patients with fewer hostile neck parameters had a greater chance of late AAA shrinkage ($P = .05$).[19] Patients with two features of a hostile neck had a significantly lower rate of freedom from sac growth compared with patients with one feature and non-HNA ($P < .001$).[29] For the total AAA volume and flow-lumen volume, both studies had described significant, but contradicting, associations with AAA sac remodeling.[21,27] A larger preoperative total AAA volume was reported as a predictor of AAA sac expansion (hazard ratio [HR], 1.04; 95% CI, 1.01-1.07; $P = .012$), but it was also reported to correlate with more AAA volume shrinkage at 6 months after EVAR ($r = 0.47$; $P = .006$).[21,27] The AAA flow-lumen volume

**Table III.** Anatomic parameters and their correlation to AAA sac remodeling in reported studies

| Investigator | EGs, % | Neck length | Neck diameter | Neck calcification | Neck thrombus | Suprarenal neck angulation | Infrarenal neck angulation | Neck tortuosity | Neck shape | Hostile neck yes/no | Hostile neck parameters, No. | Preoperative maximum AAA diameter | Infrarenal aorta length |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Nishibe,[16] 2020 | 100 | X | X | – | – | – | – | –- | – | – | – | ↓ | – |
| Hori,[17] 2019 | 93.3 | X | – | – | – | – | – | – | – | – | – | ↑↑ | ↑↑ |
| Jeong,[18] 2019 | 93.5 | X | X | – | – | – | – | – | – | – | – | X | – |
| Muhs,[19] 2018 | 90.9 | X | ↓ | X | ↓ | X | X | X | – | X | ↓ | X | – |
| Lee,[20] 2018 | 100 | – | – | – | – | – | – | – | – | X | – | X | – |
| Marie,[21] 2018 | 97.0 | – | – | – | – | – | – | – | – | – | – | X | – |
| Malas,[22] 2017 | 100 | – | – | – | – | – | X | – | – | – | – | – | – |
| Giménez-Gaibar,[23] 2017 | 98.4 | – | – | – | – | – | – | – | – | X | – | – | – |
| McGillicuddy,[24] 2017 | 100 | X | ↑, 1 year; X, 4 years | – | ↑↑ | – | X | – | – | – | – | X | – |
| De Haro,[25] 2016 | 100 | ↓↓ | – | – | – | – | – | – | – | – | – | – | – |
| Kim,[26] 2016 | 100 | X | ↑↑ | ↑↑ | X | X | X | – | X | – | – | X | – |
| Hiraoka,[27] 2015 | 92.6 | X | X | – | – | X | X | – | – | – | – | X | – |
| Nakai,[28] 2015 | 92.3 | X | X | – | – | ↑↑ | ↑↑ | – | – | – | – | ↑↑ | – |
| Iwakoshi,[29] 2014 | 100 | X | – | – | – | ↑↑ | X | – | X,↑↑ A | X | ↑↑ | ↑↑ | – |
| Welborn,[30] 2014 | 100 | ↑ | X | – | – | – | X | – | – | – | – | X | – |
| Bastos Gonçalves,[31] 2012 | 100 | – | ↑↑ | – | – | – | X | – | – | – | – | – | – |
| Greenberg,[32] 2008 | 100 | – | – | – | – | – | – | – | – | – | – | ↑↑ | – |
| Fairman,[33] 2006 | 100 | X | – | – | ↓ | – | – | – | X | – | – | ↑ | – |
| Love,[34] 2005 | 95.9 | – | – | – | – | – | – | – | – | – | – | – | – |

Empty cell, parameter not researched in the study; ↑, Significant positive association with abdominal aortic aneurysm sac shrinkage (larger value results in more shrinkage); ↓, significant negative association with abdominal aortic aneurysm sac shrinkage (smaller value of results in more shrinkage); ↑↑, significant positive association with abdominal aortic aneurysm sac enlargement (larger value results in more enlargement); ↓↓, significant negative association with abdominal aortic aneurysm sac enlargement (smaller value results in more enlargement); *AAA*, abdominal aortic aneurysm; *AS*, angulated and short neck; *CIA*, common iliac artery; *EGs*, endografts used in current clinical practice; *HU*, Hounsfield units; *IMA*, inferior mesenteric artery; ***X***, parameter researched but no significant relationship found with either abdominal aortic aneurysm sac enlargement or shrinkage.

was significantly larger in the patients with sac expansion ($P = .003$).[27] However, a larger preoperative flow-lumen volume was also described as a significant predictor of more AAA volume regression after EVAR ($R^2 = 0.42$; $P < .001$).[21] For AAA thrombus, a lower volume percentage was reported as a significant predictor of AAA expansion (HR, 0.90; 95% CI, 0.84-0.96; $P = .003$).[27] Similarly, a larger preoperative absolute thrombus volume was significantly associated with more AAA volume shrinkage at 6 months after EVAR ($r = +0.41$; $P = .03$).[21] In addition, a higher mean Hounsfield unit value of the AAA thrombus was significantly associated with less sac shrinkage (HR, 0.97; 95% CI, 0.95-0.99; $P = .013$).[18] For aortic calcification, a higher calcification grade was shown to be related to less AAA shrinkage in two independent studies using various timepoints (6 months, $P = .01$; 1 year, $P = .05$; 2 years, $P = .05$; 4 years, $P = .02$).[24,34]

Other relevant findings from the present study included that most of the reporting studies had found no significant association with any category of AAA sac remodeling for neck length, infrarenal neck angulation, the presence of HNA, and patency of the inferior mesenteric artery (IMA).

## DISCUSSION

The results of the present systematic review showed that neck thrombus might play a vital role in AAA sac shrinkage. Furthermore, AAA thrombus, total AAA volume, AAA flow-lumen volume, aortic calcification, and the number of hostile neck parameters seemed promising for predicting AAA remodeling, although these were only investigated by a few studies. In addition, the presence of HNA, infrarenal neck angulation, neck length, and patency of the IMA were not predictive of AAA sac remodeling after EVAR. It is surprising that the patency of the IMA was not related to AAA sac remodeling, since the IMA is a common origin of persistent type II endoleaks, which has repeatedly been shown to correlate with AAA sac remodeling.[7,26,35,36]

In general, strong consistent evidence on the anatomic predictors of AAA sac remodeling after EVAR is missing, which could have resulted from several factors. First, many studies only included a small range of anatomic parameters, which was problematic because the parameters of various domains could have been interacting. By only analyzing a small selection of anatomic parameters, the possible interaction or confounding factors can be missed, the risk of bias increases, and the conclusions

**Table III.** Continued.

| AAA sac length | Total AAA volume | AAA flow-lumen volume | AAA thrombus volume | Mean HU of AAA thrombus | Aortic calcification | Patency of IMA | Diameter of IMA | Thrombus at IMA level | Other aortic side branches, No. | Maximum diameter of CIA | Unfavorable iliac anatomy | Iliac artery length | Iliac artery angle | Iliac calcification |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | **X** | – | – | **X** | – | – | – | – | – |
| **X** | – | – | – | ↓ | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | **X** | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | – | ↑↑ | – | – | – |
| – | ↑ | ↑ | ↑ | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | ↓ | – | – | – | – | ↑, 1 year; **X**, 4 years | – | **X** | **X** | **X** |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | **X** | **X** | **X** | ↑↑ | – | – | – | – | – |
| – | ↑↑ | ↑↑ | ↓↓ | – | – | **X** | – | – | **X** | **X** | – | **X** | – | – |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| – | – | – | – | – | – | **X** | – | – | – | – | – | – | – | – |
| – | – | – | – | – | ↓ | – | – | – | – | – | – | – | – | – |

drawn might not be valid. It is also important to combine the assessment of anatomic predictors of AAA sac remodeling after EVAR with other types of predictors because confounding factors are not bound by categories. In the study selection for the present review, 115 studies were rejected because the described predictors were not of the anatomic category. Thus, many more potentially important categories exist of predictors of AAA sac remodeling. Hori et al[17] reported that the postoperative pulse wave velocity was significantly and independently associated with AAA sac growth. Also, Welborn et al[30] found that AAA sac shrinkage was significantly associated with a larger graft-to-aortic apposition surface area.[17] In a recent editorial, Mitsouras and Leach[37] also highlighted that the combined assessment of possible predictive parameters is a promising and important tool. In addition to knowledge of the predictors of AAA sac remodeling, it is also likely to improve our understanding of the underlying disease process of AAA progression.

Two other reasons for the observed lack of evidence were that most parameters were investigated by just a few studies and that the results of the studies were inconsistent. For the 23 parameters researched by five or fewer studies, the small amount of research provided too little evidence to be implemented into clinical decision making. The results of the studies were inconsistent; thus, generating strong evidence and its implementation in clinical care would be more difficult. Therefore, studies with large sample sizes should be conducted of a broad range of predictors for AAA sac change after EVAR with extensive statistical analyses. By assessing the predictive value of many parameters on the same patient group, the possible intersecting or confounding factors can be identified. This would result in more solid conclusions regarding the predictive variables of AAA sac remodeling, including a list of factors that do and do not significantly affect AAA sac remodeling after EVAR.

The last factor that has complicated the generation of evidence has been the inconsistent use of the definition of AAA sac growth and shrinkage across the studies. Every included study used a different timeframe to assess the AAA sac size change after EVAR. Also, the AAA sac size change was defined using five different descriptions. This complicated the comparison of the studies. Therefore, we decided not to proceed with a meta-analysis of this dataset. The inconsistency in the definitions might have been because the Society for Vascular Surgery and European Society for Vascular Surgery guidelines do not provide information on the appropriate timeframe for AAA sac size change

assessment after EVAR.[4,38] Therefore, every study has created and executed its own method. Because of the heterogeneity of the included studies, research should be performed using the most optimal method and timeframe to assess AAA sac remodeling. Thus, best practice guidelines can be created from this evidence.

The present review was limited to the available data and was susceptible to publication bias. An attempt was made to minimize this effect by also describing the parameters of the included studies that did not show a significant association with AAA sac remodeling.

To augment the clinical applicability of the present review, studies were included based on currently available endografts rather than the year of publication or the period of data inclusion. One disadvantage of this method was that 12 studies had to be excluded because the investigators had not reported information on the endografts used in their report and did not respond to a request for this information. Furthermore, our results might have been influenced by the choice of endograft, their specific instructions for use, and the variability in procedural practices. It was beyond the scope of the present review; however, the influence of the choice of endografts and additional maneuvers regarding AAA remodeling should be investigated.

Evidence on HNA was limited to the four studies that had investigated HNA explicitly, although the hostile neck is a composite of different unfavorable infrarenal neck parameters. Evidence of the neck length, diameter, thrombus, shape, and angle could not be combined to provide evidence for HNA, because it was not reported whether patients with an absence of the studied HNA parameter (eg, neck thrombus) had had other HNA parameters (eg, flared neck). Kim et al[26] studied all HNA parameters separately but failed to include HNA as a composite, preventing the opportunity for contributing to the evidence on HNA. This emphasizes the need for reporting specifically on HNA, even if all separate HNA parameters were reported.

Future research should further explore the promising anatomic features addressed in our report using advanced imaging techniques and postprocessing software. With these techniques, the anatomic features could be studied in more depth, providing more detailed information, such as the distribution of the thrombus along the length of the AAA instead of just the size of the AAA thrombus volume at a certain level. Such detailed information of the promising anatomic parameters would provide more insight into the strong predictors of AAA remodeling after EVAR and its underlying process.

A better understanding of the factors that play a role in AAA sac size change after EVAR could aid in optimizing treatment and aid the creation of a patient-specific risk-stratified follow-up program after EVAR. If parameters were found that had a predictive value for AAA sac size change, patients could be stratified by their individual risk. Follow-up algorithms could then become more patient-specific in accordance with the anticipated risks. Adjusting follow-up imaging to the individual patient is thought to have multiple advantages. In the high-risk group, AAA-related deaths could be prevented because complications will be identified in an early stage through extensive monitoring. In contrast, patients in the low-risk group will undergo less unnecessary and harmful radiation from computed tomography angiography and nephrotoxic contrast and will require fewer hospital visits, which will also save costs.

## CONCLUSIONS

In the present systematic review, we identified the presence of neck thrombus as a predictor of less AAA sac shrinkage after EVAR. Other anatomic features that are likely to play a role in the underlying process of AAA remodeling included AAA thrombus, total AAA volume, flow-lumen volume, aortic calcification, and the number of hostile neck parameters, although they were only investigated by a few studies. The presence of HNA, infrarenal neck angulation, neck length, and patency of the IMA were found to be non-predictive of AAA sac remodeling after EVAR. In general, the present review revealed a current lack of strong evidence on anatomic predictors of AAA sac remodeling, which complicated overall comparison. To complement the current evidence, further research with large patient groups on a broad range of predictors of AAA sac change after EVAR is needed. In addition, a detailed assessment of the promising anatomic predictors with advanced imaging techniques and postprocessing software is required.

## AUTHOR CONTRIBUTIONS

Conception and design: RR, EG, MR
Analysis and interpretation: RR, EG, CZ, MR
Data collection: RR, EG
Writing the article: RR
Critical revision of the article: EG, CZ, MR
Final approval of the article: RR, EG, CZ, MR
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