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REVIEW

Editor's Choice – Systematic Review and Meta-Analysis of the Outcome of Treatment for Type II Endoleak Following Endovascular Aneurysm Repair

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WHAT THIS PAPER ADDS

The efficacy and need for secondary intervention for type II endoleaks following endovascular abdominal aortic aneurysm repair remain controversial. This systematic review and meta-analysis shows that despite routine performance, there is little evidence supporting the efficacy of secondary intervention for type II endoleaks after EVAR. Although the currently available techniques are reported to be generally safe, the clinical course after intervention may not be different from conservative treatment.

Objectives: The efficacy and need for secondary interventions for type II endoleaks following endovascular abdominal aortic aneurysm repair (EVAR) remain controversial. This systematic review aimed at investigating the clinical outcomes of different type II endoleak treatments in patients with a persistent type II endoleak after EVAR.
Data sources: Embase, Medline via Ovid, Web of Science Core Collection, the Cochrane CENTRAL, and Google Scholar.
Review methods: This systematic review was performed in accordance with the PRISMA Statement. Outcomes of interest were technical and clinical success, change in sac diameter, complications, need for additional interventions, abdominal aortic aneurysm (AAA) rupture, and (AAA related) mortality. Meta-analyses were performed with random effects models.
Results: A total of 59 studies were included, with a cumulative cohort of 1073 patients with persistent type II endoleak. Peri-operative complications following treatment of type II endoleaks occurred in 3.8% of patients (95% CI 2.7–5.2%), and AAA related mortality was 1.8% (95% CI 1.1–2.7%). Overall technical success was 87.9% (95% CI 83.1–92.1%), while clinical success was 68.4% (95% CI 61.2–75.1%). Among studies detailing sac dynamics, decrease or stable sac, with or without resolution, was achieved in 78.4% (95% CI 70.2–85.6%). Changes in sac diameter following type II endoleak treatment were documented in 157 patients to at least 24 months. Within this group an actual decrease in sac diameter was reported in only 27 of 40 patients.
Conclusion: There is little evidence supporting the efficacy of secondary intervention for type II endoleaks after EVAR. Although generally safe, the lack of evidence supporting the efficacy of type II endoleak treatment leads to difficulty in assessing its merits.

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INTRODUCTION

The beneficial peri-operative outcomes of endovascular repair (EVAR) for abdominal aortic aneurysm (AAA)[1–4] have resulted in a rapid increase in its use since its introduction in 1991.[5,6] Currently in many countries, over 80% of infrarenal AAA repairs are performed endovascularly.[7–9] Despite the early advantages of the technique, EVAR has more delayed AAA related complications.[10,11] Among these, there is the potential

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for endoleaks, in which the aneurysm is not completely excluded from the circulation because of persistent blood flow within the aneurysm sac. Of the various types of endoleaks, general consensus exists on the relevance of type I and III, which are considered life threatening conditions that require secondary intervention. The significance of type II endoleaks, however, is not so clear. These are the most common type of endoleaks, with a reported occurrence rate between 8% and 44%.[12,13] Although the pathophysiology by which branch vessels provide arterial perfusion of the aneurysm sac is well understood, the clinical significance and thus the necessity for their treatment continues to be a topic of debate.

Over the years it has become clearer that the risk of aneurysm rupture in the presence of an isolated type II endoleak is exceptionally low.[14] This has led to the general practice that only type II endoleaks in the presence of aneurysm growth are a potential target for treatment.[14–16] Aside from conservative management, there are a variety of treatment options that may be considered when a type II endoleak is diagnosed, all with their inherent flaws and advantages. First, the endoleak can be approached through a transarterial catheter, which enables embolisation using coils, thrombin, or glue. As an alternative, the aneurysm sac may be approached directly through a translumbar or transcaval route. Furthermore, the collateral vessels giving rise to the endoleak can be ligated either through an open transabdominal or laparoscopic procedure. Employing any of these techniques remains controversial, because of the unclear benefit on the one hand, and the risk of doing harm on the other. Furthermore, the true success rate of this treatment remains mostly unknown as usually only the immediate technical success of the procedure is reported, the definition of success varies per study, and the effect of type II endoleak treatment on sac size is usually not reported.

This systematic review investigated the peri-operative and mid- to long-term clinical outcomes of different type II endoleak treatments in patients with a persistent type II endoleak after EVAR. In particular, the review aimed at analysing aneurysm sac dynamics after endoleak motivated interventions, as well as potential mortality and morbidity caused by such interventions.

METHODS

Literature search

A systematic review was performed in accordance with the PRISMA Statement (http://www.prisma-statement.org).[17] Embase, Medline via Ovid, Web of Science Core Collection, the Cochrane CENTRAL, and Google Scholar, were all comprehensively reviewed with the help of a medical librarian using the search terms provided in the supplementary documentation (Table S1). The last search was conducted on the June 26, 2017.

Study selection

The titles and abstracts of the retrieved articles were independently reviewed by two reviewers. Disagreements were discussed with a third reviewer. All studies reporting on the outcome of interventions for type II endoleaks with a minimum follow up of 6 months were initially included. Exclusion criteria were non-abdominal aneurysms, publication date before 2000, and non-English language. Additionally, studies were excluded if they did not distinguish type II endoleak outcomes from endoleaks from other sources. Case reports demonstrating the technical feasibility of secondary re-intervention were also excluded, because of the possibility of publication bias. The initial goal was to examine studies with a minimum follow up of 12 months, and report on changes in sac diameter following type II endoleak intervention. However, too few studies met these requirements, so it was decided to expand the inclusion criteria. Instead, sub-analyses were performed in accordance with follow up time and sac dynamics.

Data extraction

Two reviewers (KU and SB) working independently determined eligibility, and extracted descriptive, methodological, and outcome data from each eligible study, using a standard form. The reviewers extracted the following from each study: publication year, method of type II endoleak treatment, criteria for intervention, median or mean follow up, technical success rate, clinical success rate, change in sac diameter, complications, need for additional interventions, AAA rupture, and AAA related mortality. After data extraction, values were evaluated by a third researcher (RH). Technical success was defined as resolution of the previously identified backflow on post-operative imaging. The definition of clinical success varied between studies, and included lack of sac enlargement, sac diameter decrease, absence of flow on follow up imaging, and resolution or lack of flow in the aneurysm sac on follow up imaging. The definition was therefore individually detailed.

Quality assessment

To compare the quality of the included studies, the Newcastle-Ottowa Quality Assessment Scale was used (KU and SB). Studies with a score below 6 were excluded, because of insufficient quality. All studies met this quality standard, therefore further scoring was omitted (Table S2).

Outcome assessment

To account for the potential inter-study heterogeneity, random effects modelling was used to estimate the proportion of technical success, clinical success, as well as adverse outcomes. Statistical heterogeneity was assessed using the I^2 statistic, which quantifies the proportion of total variance observed between the studies attributable to differences between the studies rather than chance. Pooled results are presented as percentages with 95% CI. Analyses were performed using MedCalc Version 18 (MedCalc Software, Ostend, Belgium).

RESULTS

The electronic search yielded 1876 potentially relevant research papers. After applying the exclusion criteria, 59

studies were considered suitable for inclusion (Table 1, Fig. 1). This group of studies included 54 retrospective studies, four prospectively conducted studies, and one multicentre study. A total of 1073 patients with persistent type II endoleak who underwent one or more interventions for type II endoleak were included in this review.

The time to treatment varied from 2.5 to 52.0 months after the initial procedure. In the majority of cases (73.8%; 792/1073), sac expansion was the indication for attempting to resolve the type II endoleak. Secondary intervention for the type II endoleak was performed at the surgeon's discretion in 4.4% (47/1073) of patients, persistent

Table 1. Study characteristics.

Study	Year	Method of treatment	No. of patients	Criteria for treatment	Follow up after treatment, months	Total follow up, months	Median time to treatment, months
Arko[39]	2003	Transarterial embolisation	6	Persistence and high velocities	13.6	30.2	16.6
Aziz[36]	2012	Translumbar/transarterial embolisation	42	Sac expansion	22.6	48.4	25.8
Baum[40]	2002	Transarterial embolisation	20	Type II endoleak	13.1	N.S.	N.S.
Baum[40]	2002	Translumbar embolisation	13	Type II endoleak	8.4	N.S.	N.S.
Beeman[41]	2010	Translumbar/transarterial embolisation/open ligation	11	Sac expansion	N.S.	65.0	N.S.
Buckenham[42]	2009	Transarterial embolisation	4	Sac expansion	10.3	N.S.	26.4
Carrafiello[43]	2016	Translumbar embolisation	8	Sac expansion	37.0	N.S.	N.S.
Chikazawa[44]	2014	Open ligation	7	Sac expansion	6.6	N.S.	43.0
Cho[45]	2004	Transarterial embolisation	2	Sac expansion	N.S.	48.0	N.S.
Cieri[21]	2014	Various embolisation techniques	52	Sac expansion	N.S.	45.0	N.S.
Coppi[46]	2014	Transarterial technique	17	Sac expansion	21.5	N.S.	50.0
Dobes[47]	2016	Open ligation	10	Sac expansion	12	N.S.	N.S.
El Batti[48]	2013	Transarterial embolisation	31	N.S.	N.S.	36.7	N.S.
Faries[49]	2003	Transarterial embolisation	16	Sac expansion	24.5	N.S.	12.3
Ford[50]	2017	Transarterial/CT/ fluoroscopic guided embolisation	14	Sac expansion	13.2	N.S.	13.3
Fujimura[51]	2015	Transarterial embolisation	11	Sac expansion	16.0	N.S.	22.4
Funaki[52]	2012	Transarterial embolisation	16	Sac expansion	27.5	N.S.	48.0
Gallagher[53]	2012	Transarterial embolisation	11	Sac expansion	42.0	N.S.	N.S.
Gallagher[53]	2012	Translumbar embolisation	17	Sac expansion	42.0	N.S.	N.S.
Gallagher[53]	2012	Laparoscopic clipping	1	Sac expansion	42.0	N.S.	N.S.
Gandini[54]	2014	Nonselective transcaval embolisation	9	Sac expansion	25.9	N.S.	12.3
Gandini[54]	2014	Selective transcaval embolisation	20	Sac expansion	24.1	N.S.	12.3
Giles[55]	2015	Transcaval embolisation	26	Sac expansion	16.5	N.S.	N.S.
Goerich[56]	2000	Transarterial embolisation	11	Type II endoleak	24.5	N.S.	N.S.
Haq[57]	2017	Transarterial embolisation	17	Surgeon's discretion	N.S.	37.32	N.S.
Haq[57]	2017	Translumbar embolisation	11	Surgeon's discretion	N.S.	37.32	N.S.
Haulon[58]	2001	Superselective catheterisation	18	Type II endoleak	13.3	N.S.	N.S.
Higashiura[59]	2007	Transarterial embolisation	11	N.S.	N.S.	24.0	N.S.
Hongo[60]	2014	Transarterial embolisation	20	Sac expansion	18.5	N.S.	N.S.
Ishibashi[61]	2014	Transarterial embolisation	3	Sac expansion	N.S.	27.5	N.S.
Jones[31]	2007	Translumbar/ transarterial/open ligation	16	Sac expansion	N.S.	43.1 N.S.	N.S.
Kasirajan[62]	2003	Superselective catheterisation	8	Sac expansion	9.0	N.S.	N.S.
Kim[63]	2016	Transarterial embolisation	16	various (majority sac growth)	N.S.	N.S.	27.3
Kumar[64]	2017	Various embolisation techniques	16	Persistence and sac expansion	N.S.	22.8	N.S.
Liewald[65]	2001	Transarterial embolisation	14	type II endoleak	N.S.	18.0	N.S.

Table 1-*continued*

Study	Year	Method of treatment	No. of patients	Criteria for treatment	Follow up after treatment, months	Total follow up, months	Median time to treatment, months
Maitrias[66]	2016	Open ligation	21	Sac expansion	24.0	N.S.	24.0
Maitrias[67]	2016	Open ligation	10	Sac expansion	12.0	N.S.	12.0
Mansueto[68]	2007	Transcatheter transcaval embolisation	12	Persistence	12.0	N.S.	47.0
Marcelin[69]	2017	Transarterial embolisation/Direct puncture embolisation	28	Persistence and sac expansion >5 mm	N.S.	N.S.	26
Martin[70]	2001	Transarterial embolisation	4	Persistence	7.1	N.S.	18.1
Moulakakis[71]	2017	Transarterial/translumbar embolisation	10	Sac expansion	31.2	N.S.	32.3
Moulakakis[71]	2017	Open ligation	19	Sac expansion	20.1	N.S.	37.2
Müller-Wille[72]	2013	Transarterial embolisation	11	Sac expansion	26.0	N.S.	20.5
Nevala[73]	2010	Transarterial embolisation	10	Sac expansion	N.S.	54.0	N.S.
Nevala[73]	2010	Translumbar embolisation	3	Sac expansion	N.S.	54.0	N.S.
Nevala[73]	2010	Transabdominal embolisation	1	Sac expansion	N.S.	54.0	N.S.
Parry[74]	2002	Transarterial embolisation	6	Persistence without sac shrinkage	N.S.	24.0	N.S.
Piffaretti[75]	2017	Laparoscopic ligation	11	Persistent leak or sac expansion	46	N.S.	N.S.
Piffaretti[75]	2017	Transarterial embolisation	10	Persistent leak or sac expansion	46	N.S.	N.S.
Quinones[76]	2014	Perigraft arterial embolisation	1	Sac expansion	23.1	N.S.	40.1
Rayt[77]	2009	Various embolisation techniques	1	Sac expansion	N.S.	N.S.	36.0
Rial[78]	2004	Translumbar embolisation	3	>6 months	6.0	N.S.	N.S.
Ribe[79]	2017	Transarterial embolisation	18	Sac expansion	19	N.S.	N.S.
Richardson[80]	2003	Transarterial embolisation or laparoscopic ligation	4	Sac expansion	16.0	N.S.	2.5
Sarac[18]	2012	Various embolisation techniques	95	Sac expansion	>24.0	N.S.	26.1
Scali[81]	2013	Transcaval embolisation	6	>6 months + sac expansion	8.1	N.S.	52.0
Silverberg[23]	2006	Transfemoral/translumbar embolisation	19	Surgeon's discretion	N.S.	N.S.	19.9
Stavropoulos[82]	2009	Translumbar embolisation	62	Sac expansion	20.2	N.S.	N.S.
Stavropoulos[82]	2009	Modified transarterial embolisation	23	Sac expansion	17.3	N.S.	N.S.
Steinmetz[83]	2004	Translumbar/transarterial embolisation	5	Sac expansion	N.S.	N.S.	18.2
Tolia[24]	2005	Transarterial embolisation	2	Sac expansion	36.0	N.S.	N.S.
Tutein[84]	2001	Translumbar/transarterial embolisation	4	N.S.	N.S.	N.S.	N.S.
Van Bindsbergen[85]	2010	Translumbar embolisation	5	Sac expansion	7.0	N.S.	N.S.
Van Lammeren[86]	2010	Transarterial embolisation	8	Sac expansion >5 mm	N.S.	81.0	29.0
Voute[87]	2013	Laparoscopic fenestration	4	Sac expansion	45.6	N.S.	19.8
Walker[34]	2015	Various embolisation techniques	82	Sac expansion	N.S.	32.2	9.0
Wojtaszek[88]	2017	Transarterial embolisation	22	Persistence and sac expansion	17	60	43
Yamada[89]	2015	Open ligation	5	Sac expansion	16.2	N.S.	17.6
Yang[90]	2017	Transarterial or direct puncture embolisation	23	Sac expansion	21.8	N.S	17.7
Total			1073		6.0—46.0	18.0—81.0	2.5—52.0

N.S. = not stated.

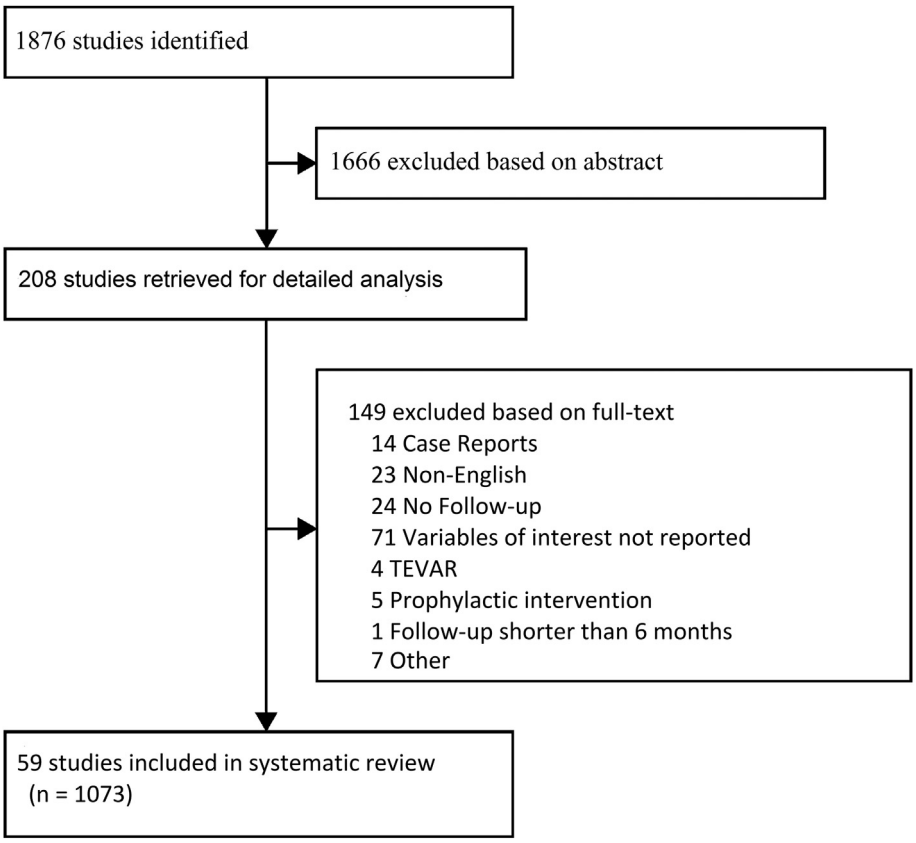

Figure 1. Study flowchart.

endoleak with or without sac expansion was the indication for 18.6% (200/1073), and 4.3% (46/1073) of the studies did not state the exact indication for the procedure.

Technical and clinical success

Although the exact definition of technical success was often not stated, the most commonly employed definition was no evidence of flow entering the aneurysm sac at the end of the procedure. Among the 50 studies detailing technical success, 87.9% (95% CI 83.1—92.1%; $I^2 = 72.2\%$) of procedures were deemed technically successful (Table 2). Stratification by type of procedure, where possible, demonstrated high technical success rates for all techniques. For trans-arterial embolisation procedures, technical success was 84.0% (77.2—89.8%; $I^2 = 56.2\%$), while for translumbar, and transcaval embolisation, this was 98.7% (95% CI 95.6—100%; $I^2 = 0.0\%$) and 93.3% (95% CI 85.1—98.4; $I^2 = 27.1\%$), respectively. Branch vessel interruption, either through a laparoscopic or open procedure, also proved to be very successful with a technical success rate of 98.1% (95% CI 94.3—100%; $I^2 = 0.0\%$).

Follow up after the treatment of the type II endoleak ranged between 6.0 and 46.0 months. The definition of clinical success was not homogenous, with approximately half of the studies employing a definition of resolution of the endoleak, while the other half used sac diameter stabilisation or decrease on latest follow up as the determinant of clinical success. The overall clinical success rate was 68.4% (95% CI 61.2—75.1%; $I^2 = 83.0\%$). Clinical success solely defined as resolution on follow up imaging was confirmed in 67.5% (95% CI 57.0—77.2) of patients. Because different definitions were employed for clinical success, it is worthwhile looking at changes in sac diameter separately. Among 40 studies detailing sac dynamics, decrease or stable sac, with or without resolution, was achieved in 78.4% (95% CI 70.2—85.6%; $I^2 = 83.4\%$).

Long-term follow up and strict outcome measures

The total number of patients followed for at least 12 months, who underwent the secondary procedure for sac growth, and for whom sac dynamics were reported as an outcome was 373 across 18 studies (Fig. 2). Median follow up across the studies in this sub-cohort was 21.5 months (12.0—45.6 months). The technical success rate after secondary intervention was 88.8% (95% CI 81.5—94.4%; $I^2 = 71.3\%$). An absence of a subsequent increase in sac diameter was reported in 83.3% of patients (95% CI 73.0—91.5%; $I^2 = 81.3\%$).

Twenty-four months after the secondary intervention, patients treated for sac growth numbered 241 in 11 studies. In 157 of these patients details regarding sac dynamics were reported. Within this group, 83.7% (95% CI 55.6—99.0%; $I^2 = 91.2\%$) had a stable or decrease in sac diameter. An actual decrease in sac size was reported in 27 of 40 patients

Table 2. Effectiveness of surgical intervention for type II endoleak.

Study	Year	Method of treatment	No. of patients	Technical success	Sac diam. decreased	Sac diam. stable	Sac growth	Clinical success	Definition clinical success
Arko[39]	2003	Transarterial embolisation	6	6	N.S.	N.S.	N.S.	5	Flow velocity and resolution
Aziz[36]	2012	Translumbar/transarterial embolisation	42	N.S.	N.S.	N.S.	N.S.	11	Resolution
Baum[40]	2002	Transarterial embolisation	20	17	N.S.	N.S.	N.S.	4	Resolution
Baum[40]	2002	Translumbar embolisation	13	13	N.S.	N.S.	N.S.	12	Resolution
Beeman[41]	2010	Translumbar/transarterial embolisation/open ligation	11	N.S.	5	N.S.	6	5	Resolution
Buckenham[42]	2009	Transarterial embolisation	4	4	1	3	0	1	Resolution
Carrafiello[43]	2016	Translumbar embolisation	8	8	3	5	0	8	Resolution or stability of sac
Chikazawa[44]	2014	Open ligation	7	7	N.S.	N.S.	0	7	Lack of sac enlargement
Cho[45]	2004	Transarterial embolisation	2	2	0	0	2	0	Lack of sac enlargement
Cieri[21]	2014	Various embolisation techniques	52	N.S.	N.S.	N.S.	22	30	Lack of sac enlargement
Coppi[46]	2014	Transarterial technique	17	16	5	3	3	8	Lack of sac enlargement and resolution
Dobes[47]	2016	Open ligation	10	10	N.S.	N.S.	N.S.	10	Resolution
El Batti[48]	2013	Transarterial embolisation	31	N.S.	N.S.	N.S.	24	7	Lack of sac enlargement
Faries[49]	2003	Transarterial embolisation	16	14	5	N.S.	N.S.	N.S.	N.S.
Ford[50]	2017	Transarterial/CT/ fluoroscopic-guided embolisation	14	14	8	1	5	7	Resolution
Fujimura[51]	2015	Transarterial embolisation	11	10	4	2	5	4	Resolution
Funaki[52]	2012	Transarterial embolisation	16	14	N.S.	N.S.	0	16	Lack of sac enlargement
Gallagher[53]	2012	Transarterial embolisation	11	N.S.	N.S.	N.S.	N.S.	8	Resolution
Gallagher[53]	2012	Translumbar embolisation	17	N.S.	N.S.	N.S.	N.S.	3	Resolution
Gallagher[53]	2012	Laparoscopic clipping	1	N.S.	N.S.	N.S.	N.S.	0	Resolution
Gandini[54]	2014	Nonselective transcaval embolisation	9	9	N.S.	N.S.	N.S.	5	Resolution
Gandini[54]	2014	Selective transcaval embolisation	20	20	N.S.	N.S.	N.S.	20	Resolution
Giles[55]	2015	Transcaval embolisation	26	22	N.S.	N.S.	8	18	Lack of sac enlargement
Goerich[56]	2000	Transarterial embolisation	11	11	N.S.	N.S.	N.S.	11	Resolution
Haq[57]	2017	Transarterial embolisation	17	N.S.	2	5	10	2	Resolution
Haq[57]	2017	Translumbar embolisation	11	N.S.	0	3	8	3	Resolution
Haulon[58]	2001	Superselective catheterisation	18	17	13	5	0	16	Resolution
Higashiura[59]	2007	Transarterial embolisation	11	N.S.	N.S.	N.S.	N.S.	7	Resolution
Hongo[60]	2014	Transarterial embolisation	20	18	2	14	2	16	Sac expansion <5 mm
Ishibashi[61]	2014	Transarterial embolisation	3	3	N.S.	N.S.	0	3	Lack of sac enlargement
Jones[31]	2007	Translumbar/ transarterial/open ligation	16	N.S.	N.S.	N.S.	N.S.	9	Resolution
Kasirajan[62]	2003	Superselective catheterisation	8	8	6	1	1	6	Sac diameter decrease
Kim[63]	2016	Transarterial embolisation	16	9	N.S.	N.S.	N.S.	5	Lack of sac enlargement

Continued

Table 2-*continued*

Study	Year	Method of treatment	No. of patients	Technical success	Sac diam. decreased	Sac diam. stable	Sac growth	Clinical success	Definition clinical success
Kumar[64]	2017	Various embolisation techniques	16	15	6	2	0	6	Sac diameter decrease
Liewald[65]	2001	Transarterial embolisation	14	8	N.S.	N.S.	4	5	Resolution
Maitrias[66]	2016	Open ligation	21	21	21	0	0	21	Resolution
Maitrias[67]	2016	Open ligation	10	10	10	0	0	10	Resolution
Mansueto[68]	2007	Transcatheter transcaval embolisation	12	11	10	N.S.	N.S.	10	Sac diameter decrease
Marcelin[69]	2017	Transarterial embolisation/direct Puncture embolisation	28	6	5	22	1	27	Lack of sac enlargement
Martin[70]	2001	Transarterial embolisation	4	3	4	0	0	4	Sac diameter decrease
Moulakakis[71]	2017	Transarterial/translumbar Embolisation	10	8	N.S.	N.S.	N.S.	7	Resolution
Moulakakis[71]	2017	Open ligation	19	19	N.S.	N.S.	N.S.	17	Resolution
Müller-Wille[72]	2013	Transarterial embolisation	11	6	3	5	3	8	Lack of sac enlargement
Nevala[73]	2010	Transarterial embolisation	10	4	N.S.	N.S.	N.S.	6	Resolution of leak without enlargement
Nevala[73]	2010	Translumbar embolisation	3	3	N.S.	N.S.	N.S.	2	Resolution of leak without enlargement
Nevala[73]	2010	Transabdominal embolisation	1	1	N.S.	N.S.	N.S.	0	Resolution of leak without enlargement
Parry[74]	2002	Transarterial embolisation	6	3	3	0	0	3	Lack of sac enlargement
Piffaretti[75]	2017	Laparoscopic ligation	11	11	N.S.	N.S.	N.S.	9	Resolution
Piffaretti[75]	2017	Transarterial embolisation	10	10	N.S.	N.S.	N.S.	7	Resolution
Quinones[76]	2014	Perigraft arterial embolisation	1	1	0	1	0	N.S.	N.S.
Rayt[77]	2009	Various embolisation techniques	1	0	0	0	1	0	Lack of sac enlargement
Rial[78]	2004	Translumbar embolisation	3	3	N.S.	N.S.	N.S.	3	Resolution
Ribe[79]	2017	Transarterial embolisation	18	18	N.S.	N.S.	N.S.	15	resolution
Richardson[80]	2003	Transart. embolisation or laparoscopic ligation	4	3	4	0	0	4	Lack of sac enlargement
Sarac[18]	2012	Various embolisation techniques	95	72	N.S.	N.S.	53	42	Resolution
Scali[81]	2013	Transcaval embolisation	6	6	N.S.	1	1	4	Resolution
Silverberg[23]	2006	Transfemoral/translumbar embolisation	19	16	N.S.	N.S.	4	11	Resolution
Stavropoulos[82]	2009	Translumbar embolisation	62	62	N.S.	N.S.	10	45	Lack of sac enlargement
Stavropoulos[82]	2009	Modified transarterial embolisation	23	22	N.S.	N.S.	3	18	Lack of sac enlargement
Steinmetz[83]	2004	Translumbar/transarterial embolisation	5	5	N.S.	N.S.	N.S.	5	Resolution
Tolia[24]	2005	Transarterial embolisation	2	1	N.S.	N.S.	0	2	Resolution and sac enlargement
Tutein[84]	2001	Translumbar/transarterial embolisation	4	4	N.S.	N.S.	N.S.	4	Resolution
Van Bindsbergen[85]	2010	Translumbar embolisation	5	5	N.S.	N.S.	N.S.	5	Resolution
Van Lammeren[86]	2010	Transarterial embolisation	8	7	N.S.	N.S.	1	7	Lack of sac enlargement
Voute[87]	2013	Laparoscopic fenestration	4	4	N.S.	N.S.	2	2	Lack of sac enlargement
Walker[34]	2015	Various embolisation techniques	82	N.S.	N.S.	N.S.	37	32	Lack of sac enlargement
Wojtaszek[88]	2017	Transarterial embolisation	22	17	N.S.	N.S.	N.S.	17	Resolution

Table 2-*continued*

Study	Year	Method of treatment	No. of patients	Technical success	Sac diam. decreased	Sac diam. stable	Sac growth	Clinical success	Definition clinical success
Yamada[89]	2015	Open ligation	5	5	5	0	0	5	Resolution and sac diameter decrease
Yang[90]	2017	Transarterial or direct puncture embolisation	23	15	N.S.	N.S.	2	16	Resolution
Total			1073	87.9 (83.1—92.1)				68.4 (61.2—75.1)	

N.S. = not stated.

(62.3%; 95% CI 8.4—99.8%; $I^2 = 93.6\%$). Because of small numbers, analysis at 36 month follow up was not performed.

Adverse outcomes of secondary intervention

With regard to adverse clinical outcomes (Table 3), the incidence following repair was generally low. The pooled estimate of AAA related mortality following treatment of type II endoleaks of 53 studies was 1.8% (95% CI 1.1—2.7%; $I^2 = 0.0\%$). These mortality cases were distributed as follows: three after transarterial coil embolisation, two after open ligation, and one after unspecified embolisation. Perioperative complications occurred in 3.8% (95% CI 2.7—5.2%; $I^2 = 2.4\%$) of patients, and included cardiac complications, colonic ischaemia, contrast nephropathy, and displacement of a coil between the vena cava and the aorta during an embolisation procedure. The overall re-intervention rate was 14.7% (95% CI 11.2—18.5%; $I^2 = 56.9\%$), and conversion to open repair was performed in 4.3% of patients (95% CI 2.7—6.2%; $I^2 = 40.6\%$). Secondary rupture was reported in 1.8% (95% CI 1.1—2.8%; $I^2 = 0.0\%$) of patients after treatment for type II endoleak. Among the studies describing patients with follow up greater than 12 months, the secondary intervention and conversion rate were, respectively, 14.6% (95% CI 10.5—19.3%; $I^2 = 58.2\%$) and 3.6% (95% CI 2.1—5.5%; $I^2 = 20.6\%$). Rupture occurred in 1.5% of patients (95% CI 0.7—2.5%; $I^2 = 0.0\%$). A total of six aneurysm related deaths occurred, of which four were attributed to late secondary rupture. One death occurred because of colonic ischaemia after an open ligation procedure. The final patient died during explantation of the graft, which was performed because of an aorto-duodenal fistula.

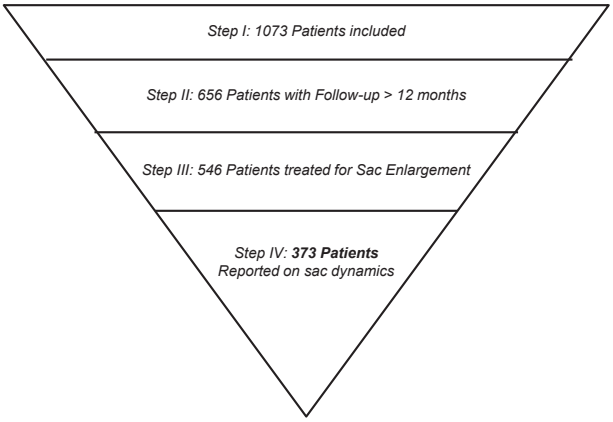

Figure 2. Total number of patients followed for at least 12 months, who underwent the secondary procedure for sac growth, and for whom sac dynamics were reported as an outcome across 18 studies.

DISCUSSION

The present study has demonstrated that although the technical success rate of type II endoleak treatment is high, ranging from 84% to 100% depending on the technique, one third failed to either completely resolve or show signs of sac diameter stabilisation or decrease at the latest follow up. In addition, the rate of rupture after intervention for type II endoleak was less than 2% in studies reporting at least 12 months of follow up, which does not appear to be different from EVAR studies in general.[1]

The relevance of type II endoleaks has been a matter of debate for many years and was thoroughly investigated in a recent systematic review by Sidloff et al.[14] However, this review did not specifically evaluate the outcome of treatment, as no distinction was made for the definition of successful treatment.[14] Moreover, only a very limited number of patients undergoing intervention were included in this study, particularly considering that no criteria were set for minimum follow up time. Although the clinical success rate in this review indicates reasonable outcomes, it has been shown before that the efficacy of type II endoleak treatment in terms of the prevention of sac growth deteriorates over time, with a rapid decrease one year post embolisation.[18] This supports the complexity and multifactorial aetiology of type II endoleaks as described by Solis et al., and indicates that it is likely there are multiple sources of in- and outflow of the aneurysmal sac.[19] Additionally, when reviewing the included articles, it became apparent that multiple definitions of clinical success were in use. While the current consensus on the purpose of treating type II endoleaks is eliminating persistent aneurysm sac growth, thereby diminishing the risk of secondary rupture, studies often consider only lack of radiological evidence of arterial backflow into the aneurysm sac as the primary endpoint.[20,21] However, there is evidence that not all patent (nor persistent) type II endoleaks are associated with sac growth.[22—25] Moreover, multiple studies, including a recent meta-analysis, demonstrated that with both contrast CT, as well as contrast enhanced ultrasound, a substantial number of type II endoleaks may still be missed on follow up imaging.[25—30] Also, some methods of treatment involving coils

Table 3. Adverse outcomes after Type 2 endoleak treatment.

Study	Year	Method of treatment	No. of patients	Serious complications	Re-re-intervention	Conversion	Rupture	AAA related mortality
Arko[39]	2003	Transarterial embolisation	6	0	1	0	0	0
Aziz[36]	2012	Translumbar/transarterial embolisation	42	1	16	0	0	0
Baum[40]	2002	Transarterial embolisation	20	0	9	0	0	0
Baum[40]	2002	Translumbar embolisation	13	0	0	0	0	0
Beeman[41]	2010	Translumbar/transarterial embolisation/open ligation	11	0	1	0	0	0
Buckenham[42]	2009	Transarterial embolisation	4	0	3	0	0	0
Carrafiello[43]	2016	Translumbar embolisation	8	0	0	0	0	0
Chikazawa[44]	2014	Open ligation	7	0	0	0	0	0
Cho[45]	2004	Transarterial embolisation	2	0	0	1	0	0
Cieri[21]	2014	Various embolisation techniques	52	0	15	21	0	0
Coppi[46]	2014	Transarterial technique	17	1	2	1	0	0
Dobes[47]	2016	Open ligation	10	4	0	0	0	0
El Batti[48]	2013	Transarterial embolisation	31	N.S.	N.S.	N.S.	N.S.	N.S.
Faries[49]	2003	Transarterial embolisation	16	N.S.	2	N.S.	N.S.	N.S.
Ford[50]	2017	Transarterial/CT/ fluoroscopic-guided embolisation	14	1	4	1	0	0
Fujimura[51]	2015	Transarterial embolisation	11	0	1	0	4	2
Funaki[52]	2012	Transarterial embolisation	16	0	0	0	0	0
Gallagher[53]	2012	Transarterial embolisation	11	N.S.	2	0	0	0
Gallagher[53]	2012	Translumbar embolisation	17	N.S.	4	2	0	0
Gallagher[53]	2012	Laparoscopic clipping	1	N.S.	0	1	0	0
Gandini[54]	2014	Nonselective transcaval embolisation	9	0	4	0	1	0
Gandini[54]	2014	Selective transcaval embolisation	20	0	0	0	0	0
Giles[55]	2015	Transcaval embolisation	26	0	5	0	0	0
Goerich[56]	2000	Transarterial embolisation	11	0	0	0	0	0
Haq[57]	2017	Transarterial embolisation	18	0	0	0	0	0
Haq[57]	2017	Translumbar embolisation	17	0	4	0	0	0
Haulon[58]	2001	Superselective catheterisation	11	0	2	0	0	0
Higashiura[59]	2007	Transarterial embolisation	11	0	0	0	0	0
Hongo[60]	2014	Transarterial embolisation	20	0	1	1	0	0
Ishibashi[61]	2014	Transarterial embolisation	3	N.S.	1	0	0	N.S.
Jones[31]	2007	Translumbar/ transarterial/open ligation	16	N.S.	N.S.	2	1	N.S.
Kasirajan[62]	2003	Superselective catheterisation	8	1	1	0	0	0
Kim[63]	2016	Transarterial embolisation	16	0	6	0	0	0
Kumar[64]	2017	Various embolisation techniques	16	0	1	0	1	1
Liewald[65]	2001	Transarterial embolisation	14	0	N.S.	0	0	0
Maitrias[66]	2016	Open ligation	10	2	0	0	0	0
Maitrias[67]	2016	Open ligation	21	6	0	1	0	1
Mansueto[68]	2007	Transcatheter transcaval embolisation	12	0	1	0	0	0
Marcelin[69]	2017	Transarterial embolisation/Direct puncture embolisation	28	0	6	0	0	0
Martin[70]	2001	Transarterial embolisation	4	0	0	0	0	0
Moulakakis[71]	2017	Transarterial/translumbar embolisation	10	0	3	0	0	0
Moulakakis[71]	2017	Open ligation	19	3	0	1	0	1
Müller-Wille[72]	2013	Transarterial embolisation	11	0	0	0	0	0

Table 3-*continued*

Study	Year	Method of treatment	No. of patients	Serious complications	Re-re-intervention	Conversion	Rupture	AAA related mortality
Nevala[73]	2010	Transarterial embolisation	10	0	3	0	0	0
Nevala[73]	2010	Translumbar embolisation	3	0	1	0	0	0
Nevala[73]	2010	Transabdominal embolisation	1	0	0	0	0	0
Parry[74]	2002	Transarterial embolisation	6	0	1	0	0	0
Piffaretti[75]	2017	Laparoscopic ligation	11	0	2	1	0	0
Piffaretti[75]	2017	Transarterial embolisation	10	0	3	2	0	0
Quinones[76]	2014	Perigraft arterial embolisation	1	0	0	0	0	0
Rayt[77]	2009	Various embolisation techniques	1	0	1	0	0	0
Rial[78]	2004	Translumbar embolisation	3	0	N.S.	N.S.	N.S.	0
Ribe[79]	2017	Transarterial embolisation	18	0	1	0	0	0
Richardson[80]	2003	Transart. embolisation or laparoscopic ligation	4	0	1	0	0	0
Sarac[18]	2012	Various embolisation techniques	95	8	19	8	0	0
Scali[81]	2013	Transcaval embolisation	6	0	1	0	0	0
Silverberg[23]	2006	Transfemoral/translumbar embolisation	19	N.S.	0	0	0	N.S.
Stavropoulos[82]	2009	Translumbar embolisation	62	2	10	0	0	0
Stavropoulos[82]	2009	Modified transarterial embolisation	23	0	3	0	0	0
Steinmetz[83]	2004	Translumbar/transarterial embolisation	5	0	0	0	0	0
Tolia[24]	2005	Transarterial embolisation	2	0	0	0	0	0
Tutein[84]	2001	Translumbar/transarterial embolisation	4	0	0	0	0	0
Van Bindsbergen[85]	2010	Translumbar embolisation	5	0	0	0	0	0
Van Lammeren[86]	2010	Transarterial embolisation	8	0	0	0	1	1
Voute[87]	2013	Laparoscopic fenestration	4	0	2	2	0	0
Walker[34]	2015	Various embolisation techniques	82	N.S.	N.S.	N.S.	N.S.	N.S.
Wojtaszek[88]	2017	Transarterial embolisation	22	0	3	0	0	0
Yamada[89]	2015	Open ligation	5	0	0	0	0	0
Yang[90]	2017	Transarterial or direct puncture embolisation	23	1	8	0	0	0
Total			1073	3.8 (2.7—5.2)	14.7 (11.2—18.5)	4.3 (2.7—6.2)	1.8 (1.1—2.8)	1.8 (1.1—2.7)

N.S. = not stated.

or contrast impregnated bioglue render subsequent observation of endoleaks exceedingly difficult. Furthermore, reappearance of other collaterals on follow up studies are frequently reported.[18,25] The use of short-term type II endoleak resolution as the primary measurement of success may therefore be unreliable and should be avoided. When looking at studies with appropriate patient selection and follow up time, in addition to using sac dynamics as the endpoint for clinical success instead of resolution, the evidence is particularly scarce. In the present review, only three studies reported on decreases in sac diameter in patients experiencing initial sac growth for at least 24 months, with a combined sample of only 40 patients.

With a cumulative peri-operative complication rate of 4%, procedural morbidity of type II endoleaks is low regardless of modality. However, one must acknowledge that these studies were mostly conducted in experienced centres and it is likely that publication bias affects reported outcomes. As a result, real world complication rates may be underestimated. Moreover, because as many as 15% of patients undergo a second or even a third procedure, these figures can amount to a significant risk during the course of treatment.[18]

Type II endoleak has been associated with an increased risk of rupture and AAA related mortality.[31,32] As prevention of type II endoleaks is not possible in most cases, the most important question is whether these interventions reduce the chances of rupture and AAA related death. From the accumulated data, the risk of secondary rupture was low, with only 2% experiencing rupture during a minimum of 12

months' follow up after treatment of their type II endoleak. This is similar to the widely reported rupture rate after EVAR in general and compares well with rupture rates in conservatively treated patients with type II endoleak.[14,33] However, longer follow up data after re-interventions are needed to draw more reliable conclusions on the total incidence of secondary rupture. In addition, it should be noted that a retrospective comparison is difficult, particularly when comparing with conservative treatment strategies, because of confounding by indication in retrospective studies. Nevertheless, Walker et al. found that all cause and aneurysm related mortality were unaffected by type II endoleaks in a multicentre EVAR registry.[34] Moreover, even in the presence of sac growth, no differences were observed in aneurysm related outcomes between patients who underwent a re-intervention and those who were simply observed.

Current ESVS guidelines advise a secondary surgical intervention when sac enlargement of more than 10 mm is detected.[15] In the studies reviewed, it was noted that many authors use sac enlargement of five mm or less, or even no growth as an indication for intervention. The lack of apparent clinical benefit of these interventions underlines the need for careful patient selection. Because of the relatively liberal use of current guidelines, no comment can be made on the possible advantages of intervention in the cohort solely consisting of patients with 10 mm sac growth, as recommended.[15] In addition, studies have demonstrated that spontaneous sealing of type II endoleaks occurs frequently, even after longer periods of time.[25,35] Because of this natural behavior of type II endoleaks, it remains difficult to assess the merits of secondary intervention, as it is likely that not all clinical successes should be attributed to the secondary procedure. Also, it remains difficult to assess which type II endoleaks are prone to seal in the retrospective studies in the present cohort. In light of this, it is desirable that prospective observational studies be conducted with the specific goal of following the natural course of type II endoleaks.

In addition to a lack of prospectively gathered data, it is likely that publication bias occurred with regards to technical success of secondary intervention techniques in the current literature. The aim of a significant number of included studies was to demonstrate the usefulness of a secondary intervention. It stands to reason that these studies have a smaller chance of being published by the authors, if the demonstrated tool or technique is unsuccessful or has serious problems or shortcomings. The results of this study therefore are likely to show an overestimation of technical and clinical success. This, in combination with the scarcity of data on type II endoleak treatment despite routine use of EVAR and secondary interventions,[5] underlines the paucity of evidence supporting treatment. Yet it should also be noted that the wide inclusion of studies employing various indications for secondary interventions means that the instances when secondary interventions may actually have been warranted because of the imminent risk of secondary rupture could not be distilled from the present data. Finally, a lack of separate reporting of unsuccessful procedures means that the specific outcome after failed secondary intervention for type II endoleak unfortunately could not be investigated using these data.

Clinicians should be aware that type II endoleak in combination with sac growth may actually be the result of an unexpected underlying type I or III endoleak as described by Aziz et al. and more recently by Madigan et al.[36,37] In this context, type II endoleaks may be seen as sentinel endoleaks and may warrant thorough investigation of the efficacy of the graft integrity and the proximal and distal seal. Direct endoleaks may even be intermittent, as demonstrated before, which complicates diagnosis even further.[38]

This systematic review has several limitations that restrict its conclusions. First, it was not always possible to differentiate between patients who had already undergone a prior secondary intervention, before the studied intervention and it was not always possible to identify patients with multiple type II endoleaks. Because these two patient groups are more likely to represent relatively complex cases, this might negatively affect treatment outcome. Also, insufficiently clear data on the recurrence of an endoleak after initial technical success precluded assessment of recurrence in those achieving post-operative resolution. Finally, the limited amount of evidence on type II endoleak treatment meant that it was not possible to perform a fair comparison of the different treatment techniques.

In conclusion, although performed frequently, there is little evidence supporting the efficacy of secondary intervention for type II endoleaks after EVAR, even in patients with growing aneurysms. The currently available techniques are reported to be generally safe, causing only limited post-operative morbidity and mortality. Yet, the clinical course after intervention may not be different from conservative treatment. Despite their safety, there are virtually no prospective studies comparing intervention with observation, and only three retrospective studies with a combined volume of only 40 patients reported on decreases in sac diameter with sufficient follow up in a group of patients experiencing initial sac growth. Among these, an actual decrease in sac diameter was only observed in 27 patients. The lack of data supporting its efficacy, together with the multifactorial cause of sac growth after EVAR, leads to difficulty in assessing the merits of secondary intervention for type II endoleak with regards to reducing the risk of aneurysm rupture.

CONFLICT OF INTEREST

None.

FUNDING

None.

APPENDIX A. SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.ejvs.2018.06.009.

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