Complications Post-Endovascular Abdominal Aortic Aneurysm Repair in Patients with Diabetes Mellitus: A Systematic Review and Meta-Analysis

Eman Ashraf Adly Aly Otify,[1,2] I. Sanders,[2,3] J. Borucki,[4] K. Dhatariya,[2,5] and P. Stather,[2,6] London, UK

Background: People with diabetes mellitus (DM) have higher long-term mortality following abdominal aortic aneurysm (AAA) repair than those without DM. However, whether this adverse outcome is directly related to their aneurysm is unclear. The aim of the study was to determine the rates of complications in people with and without DM post-endovascular AAA repair. Primary outcome data include AAA sac enlargement, reinterventions, endoleaks, postoperative AAA rupture, and conversion to open surgical repair.

Methods: PubMed, Embase, and Cochrane databases were searched for primary research studies between 2005 and 2025 according to Preferred Reporting Items for Systematic Review and Meta-analyses guidelines. Those undergoing AAA repair via endovascular aneurysm repair were included.

Results: Forty-four studies were identified totaling 90,347 people in the control group, and 23,988 in the DM group. Those with DM had a lower rate of reintervention compared to controls (9.51% v 11.92%; odds ratio [OR] 0.82, 95% confidence interval [CI] [0.76−0.88; $P < 0.001$). However, there was no significant difference in the rate of overall, type I or type II endoleaks ($P = 0.22$, $P = 0.237$, $P = 0.667$, respectively).People with DM were also less likely to have sac enlargement post-AAA repair (9.05% DM v 10.38% controls; OR 0.86 [0.78−0.96]; $P = 0.005$). Those with DM were more likely to have sac shrinkage (42.89% DM v 41.26% controls; OR 0.92, 95% CI [0.89−0.95]; $P < 0.001$). In addition, people with DM had a reduced rate of conversion to open surgery (2.11% DM v 3.12% control; OR 0.80, CI [0.66−0.97]; $P = 0.023$).

Conclusion: Reinterventions, sac enlargement post-AAA repair, and conversion to open surgical repair were associated with a significant reduction in people with DM; however, the cause for these differences remains unclear.

Conflict of interest: The authors declare no competing interests.
Funding: None.

[1]*St Georges University Hospital NHS Trust, London, UK.*

[2]*Norwich Medical School, University of East Anglia, Norwich, UK.*

[3]*Barts Health NHS Trust, London, UK.*

[4]*Department of General Surgery, Norfolk and Norwich University Hospital NHS Trust, Norwich, UK.*

[5]*Department of Endocrinology, Norfolk and Norwich University Hospital NHS Trust, Norwich, UK.*

[6]*Department of Vascular Surgery, Norfolk and Norwich University Hospital NHS Trust, Norwich, UK.*

Correspondence to: E. Otify, St George's University Hospitals NHS Trust, Blackshaw Rd, London SW17 0QT, UK; Norwich Medical School, University of East Anglia, Norwich, UK.; E-mails: eman.otify@nhs.net or eman.otify@stgeorges.nhs.uk

Ann Vasc Surg 2026; 130: 518–531
https://doi.org/10.1016/j.avsg.2026.04.047

Manuscript received: February 16, 2026; manuscript accepted: April 4, 2026; published online: 12 May 2026

INTRODUCTION

There is a well-established inverse association between diabetes mellitus (DM) and abdominal aortic aneurysm (AAA) growth.[1] In particular, fasting glucose levels have been shown to inversely correlate with aortic diameter, suggesting a relationship between glucose metabolism and AAA formation.[2,3] However, serum glucose levels alone do not appear to be associated with aortic expansion.[4] Glycated hemoglobin, a marker of long-term glycemic control, has also demonstrated an inverse association with AAA growth rate in people with and without DM, further supporting a mechanistic relationship between chronic hyperglycemia and AAA progression.[4] These observations contrast with the well-established positive association between DM and other vascular diseases and their complications[5–7] giving rise to the so-called diabetes paradox in AAA disease.

With advances in device technology and technique, endovascular aneurysm repair (EVAR) has increasingly replaced open surgical repair (OSR) for AAA management.[8] Although EVAR is associated with lower perioperative mortality, it carries higher long-term risks of all-cause mortality, reintervention, and secondary rupture rates compared to OSR.[9] Individuals with DM are known to have higher rates of complications following OSR, such as myocardial infarction, pneumonia, superficial surgical site infection, or pancreatitis.[10,11] In contrast, complications following EVAR differ in nature, with issues such as endoleaks, endograft migration, and reintervention rates in approximately 16% to 30%.[12] A previous meta-analysis demonstrated that DM is not a risk factor for type II endoleak;[13] however, there remains little evidence evaluating the broader impact of DM on post-EVAR complications.

The aim of this systematic review and meta-analysis was to evaluate the effect of DM on complications following EVAR.

METHODS

Eligibility Criteria

For inclusion in this study the following inclusion and exclusion criteria were required.

Inclusion criteria

- Adults with DM aged ≥18 years old
- Patients with DM who underwent AAA repair using EVAR
- Publications translated into English language
- Report on at least 1 complication from reinterventions, sac enlargement, endoleak, postoperative AAA rupture, or conversion to OSR.

Exclusion criteria

- Case reports due to a lack of comparative data
- Laboratory-based studies
- Conference abstracts
- Studies investigating thoracic aortic aneurysms
- Systematic reviews

Literature Search

Two independent reviewers (E.O. and I.S.) performed a systematic search of the following 6 databases, PubMed, Medline, Embase, Cochrane Library, The Web of Science, and Google Scholar, for articles published between 2005 and 2025 using the search terms using the Medical Subject Headings ''MeSH'' terms *Reintervention* AND *''Abdominal Aortic Aneurysm''* AND *''Diabetes Mellitus''*, as well as *''Endoleak''* AND *''Abdominal Aortic Aneurysm''* AND *''Diabetes Mellitus''*. Researchers were contacted about published or unpublished trials identified through conferences or social media, to ensure all relevant studies were included. Ongoing trials were identified on www.clinicaltrials.gov. No contact was required to be made with authors. Zero publications required translation to be used and therefore all literature were in English language.

Study Identification and Selection

A systematic search of the databases was undertaken by 2 reviewers (E.O. and I.S.) with duplicates removed, before screening the title and abstract of identified studies. Any discrepancies were discussed with the senior author. The remaining full-text articles were retrieved and independently reviewed for eligibility.

Data Extraction and Quality Assessment

Data extraction was undertaken by 2 reviewers using a standardized template. Any discrepancies were discussed. Descriptive, methodological, and outcome data were input into a standard form. The reviewers extracted the following from each study: publication year, number of people with DM and controls, mean length of follow-up, and outcomes reported from each of the studies to include endoleaks, reintervention, sac expansion, sac shrinkage, and conversion to open repair. The Newcastle—Ottawa scale was used to ensure the quality of nonrandomized studies with a score ≥7 being rated as good.[14]

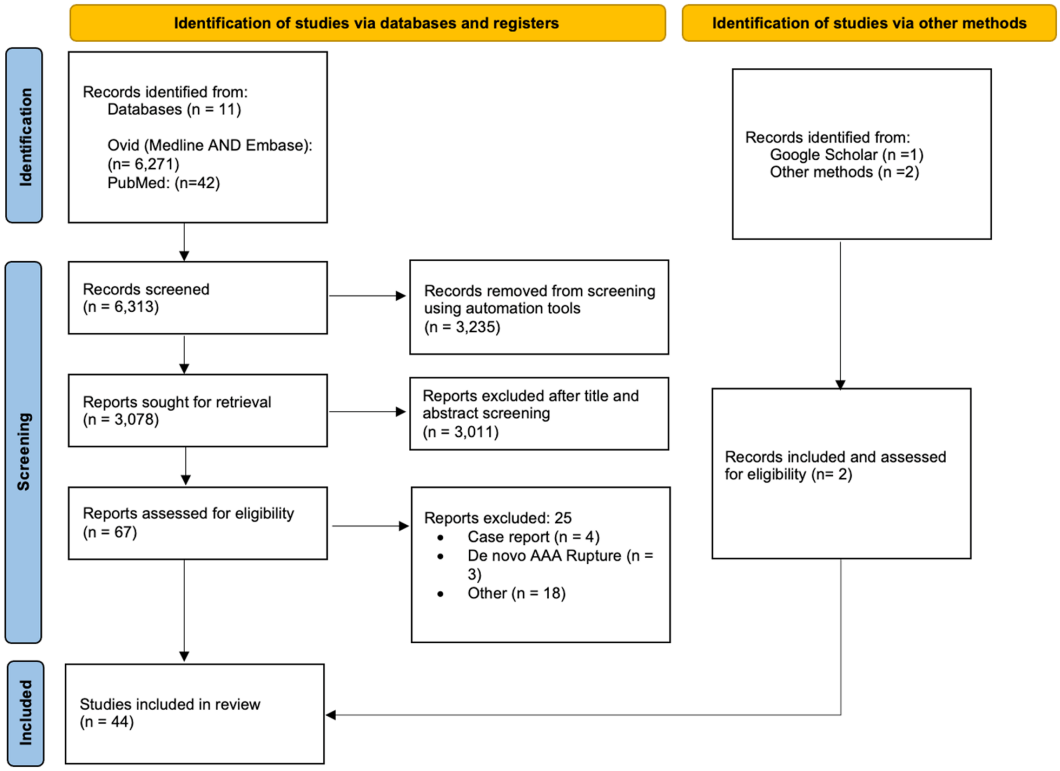

Fig. 1. PRISMA flow diagram. PRISMA, Preferred Reporting Items for Systematic Review and Meta-analyses.

Data Synthesis and Analysis

Meta-analysis was conducted of the adjusted risk estimates (where available) with use of the inverse variance method in Stata MP 17. Odds ratio (OR) was used to measure effect of each outcome. Statistical heterogeneity was assessed, with forest plots with an $I^2 \leq 50\%$ analyzed using a fixed effects model (except AAA sac expansion where random effect model was used as each of the studies included had a different definition for AAA sac growth). For studies that published early and late results these were combined to determine an overall rate of each outcome. Sensitivity analyses were performed for all significant outcomes.

A final version of this protocol has been registered on PROSPERO in January 2023 before conducting the systematic review and meta-analysis (website: www.crd.york.ac.uk/prospero/and ID: CRD420 23379545). The review was conducted according to the Preferred Reporting Items for Systematic Review and Meta-analyses guidelines.[15]

RESULTS

The literature search identified 6,313 articles, of which 3,235 were duplicates. 3,078 articles were potentially suitable for inclusion in the review; 44 of these studies were eligible for inclusion (Fig. 1). The characteristics of the included studies are outlined in Table I.

Reintervention

Meta-analysis of 12 studies including a total of 64,327 in the control group and 10,158 in the DM group identified significantly fewer reinterventions in those with DM (9.65% v 11.97%; OR 0.83, 95% confidence interval [CI] [0.77−0.89]; $P < 0.00001$), (Fig. 2).

Endoleaks

There was no significant difference in all endoleaks DM (28.33% v 31.97% controls; OR 0.91, 95% CI [0.53−1.58]; $P = 0.22$). Seven studies reported on rates of type I endoleak and showed no significant difference DM (6.29% v 8.11%; OR 0.90, 95% CI [0.77−1.07]; $P = 0.237$) (Fig. 3). Seventeen studies reported on rates of type II endoleak and showed no significant difference DM (24.60% v 25.92% control; OR 0.98, 95% CI [0.92−1.06]; $P = 0.667$) (Fig. 4). Four studies reported on the rate of type III endoleak, identifying a lower rate of type III endoleak in those with DM (2.16% v 4.02%; OR 0.68, 95% CI [0.47−0.97]; $P = 0.03$) (Fig. 5).

Table I. Studies included in the meta-analysis

Study	Study design, country	No. of DM	No. of control	Inclusion criteria
Png, 2017	Retrospective cohort, United States	183	810	Sac enlargement, rupture, reintervention and type I, II, III and total Endoleak, conversion to open surgical repair
Taimour S., 2019	Prospective cohort, Sweden	748	2630	Reintervention
Leurs L.J., 2005	Retrospective cohort, Netherlands	731	5,286	Reintervention, type I, II, III endoleak, conversion to open surgical repair
Hall M.R., 2017	Retrospective cohort, United States	29	50	Endoleak
Diehm, 2007	Prospective cohort, The Netherlands	810	5,573	AAA sac enlargement, reintervention, type I endoleak, conversion to open surgical repair
Takahara M., 2022	Prospective cohort, Japan	226	703	Aortic sac enlargement, reintervention, total endoleak
Praca, 2018	Retrospective cohort, Belgium	57	191	Aortic sac enlargement, type II and total endoleak
Seike Y., 2022	Prospective cohort, Japan	2526	14,573	Type II endoleak
Morisaki K., Matsubara Y., 2022	Retrospective cohort, Japan	60	208	Sac shrinkage
Tseng HW,2022	Retrospective cohort, Taiwan	36	155	Sac enlargement and shrinkage
Candell L, 2014	Retrospective cohort, United States	441	1,290	Postoperative (EVAR) AAA rupture
Hoshina K., 2019	Retrospective survey, Japan	4,611	33,397	Sac enlargement and reintervention
Lee JS, 2022	Retrospective cohort, South Korea	60	294	Aneurysm sac enlargement
Koole D., 2011	Prospective cohort, The Netherlands	767	5,570	AAA sac enlargement and shrinkage after EVAR
Buijs RV, 2016	Retrospective case-control, The Netherlands	10	50	Type I endoleak
Andersen R.M., 2018	Retrospective cohort, Denmark	57	364	Endoleak after EVAR
Eden CL, 2020	Retrospective cohort, United States	54	335	Reintervention, type II endoleak
Morisaki K., Yamaoka T, 2017	Retrospective cohort, Japan	29	182	Sac enlargement and type II endoleak
Tan TW, 2016	Retrospective cohort, United States	466	1,936	Type I endoleak
Vaaramaki S, 2021	Prospective cohort, Finland	30	189	Sac shrinkage, endoleak
Walker J., 2014	Prospective cohort, United States	442	1,294	Type II endoleak
Suarez G. LA, 2023	Retrospective cohort, Spain	33	107	Endoleak after EVAR
Wang Y., 2022	Retrospective cohort, China	51	236	Type II endoleak
Major M., 2022	Retrospective cohort, United States	54	335	Type I endoleak
Fujimura *N*, 2016	Retrospective cohort, Japan	125	707	Type II endoleak
Shingaki M., 2018	Retrospective cohort, Japan	25	148	Sac shrinkage
Kray J., 2015	Retrospective cohort, United States	46	145	Type II endoleak
Suckow B.D., 2022	Retrospective cohort, United States	4,492	11,445	conversion to open surgical repair
Boniakowski A.E, 2016	Retrospective cohort, United States	11	45	Type II endoleak
Sakaki M., 2020	Retrospective cohort, Japan	96	454	Type II endoleak

(*Continued*)

Table I. Continued

Study	Study design, country	No. of DM	No. of control	Inclusion criteria
Gibello L, 2020	Prospective cohort, Italy	139	509	Sac enlargement
Conrad M.F., 2009	Retrospective cohort, United States	112	720	Reintervention
Le T.B., 2018	Retrospective cohort, South Korea	27	54	Endoleak post-EVAR
Ichihashi S., 2019	Retrospective cohort, Japan	71	362	Type III endoleak
Menges A.-L., 2022	Retrospective cohort, Switzerland	5	30	Reintervention
Tara A. R. 2025	Retrospective cohort, Netherlands	94	412	Reintervention, sac expansion and shrinkage, and endoleak
Marcos F. 2024	Retrospective cohort, Spain	541	1,082	Sac enlargement and shrinkage, reintervention, types of endoleak
Rasiova 2022	Retrospective cohort, Slovakia	42	136	Type II endoleak
Farres et al. 2024	Retrospective cohort, United States	22	65	Type II endoleak
Chen et al. 2024	Retrospective cohort, Taiwan	2,043	13,349	Reintervention
Ikea et al. 2022	Retrospective cohort, Japan	74	479	Sac shrinkage
Kano et all	Retrospective cohort, Japan	30	113	Sac shrinkage
Rastogi V. et al. 2024	Retrospective cohort, United States	3,453	13,512	Sac enlargement and shrinkage
Pratama M Y. et al. 2025	Retrospective cohort, United States	23	123	Type II endoleak

Sac Change

Meta-analysis of 13 studies including a total of 62,390 in the control group and 11,006 in the DM group were assessed for AAA sac enlargement. Those with DM were less likely to have sac enlargement post-AAA repair DM (9.05% v 10.36%; OR 0.85, 95% CI [0.75−0.96]; $P = 0.01$), (Fig. 6). Those with DM were more likely to have sac shrinkage DM (42.81% v 41.28%; OR 0.92, 95% CI [0.89−0.95]; $P < 0.00001$) across 10 studies, (Fig. 7).

Rupture and Conversion

There was no significant difference in the rate of AAA rupture post-EVAR DM (0.65% v 0.56%; OR 1.11, 95% CI [0.82−1.51]; $P = 0.505$) across 7 studies, (Fig. 8).

Conversion from EVAR to OSR was reported in 4 studies which was significantly less common in those with DM (2.11% v 3.12%; OR 0.80, CI [0.66−0.97]: $P = 0.023$), (Fig. 9).

Sensitivity Analysis

Sensitivity analysis was undertaken for studies of >500 participants. The findings from the subgroup sensitivity analysis remained consistent with those of the overall pooled analysis, demonstrating lower complication rates in the diabetes group for reintervention, AAA sac expansion, and AAA sac shrinkage ($P < 0.00001$, $P = 0.01$ and $P < 0.00001$ respectively) (Table II).

The findings from the subgroup sensitivity analysis remained consistent with those of the overall pooled analysis, demonstrating lower complication rates in the diabetes group.

Sensitivity analysis was also undertaken utilizing only studies that score as 'good' on the Newcastle-Ottawa Scale. The results for reintervention, sac expansion, and open surgical conversion remained significant ($P < 0.00001$, $P = 0.01$ and $P < 0.00001$ respectively). There are results of type I or type II endoleak, AAA rupture post-EVAR, and AAA sac shrinkage remained nonsignificant (Table III).

Publication Bias

Funnel plots were reviewed for reintervention (Fig. 10), sac expansion (Fig. 11), sac shrinkage (Fig. 12), and type II endoleak (Fig. 13). As there were ≥10 studies included in each of those domains, Egger test was undertaken for reintervention, sac expansion, type II endoleak, and sac shrinkage. These showed no evidence of publication bias, P values were 0.375, 0.775, 0.254, and 0.836, respectively (Table IV).

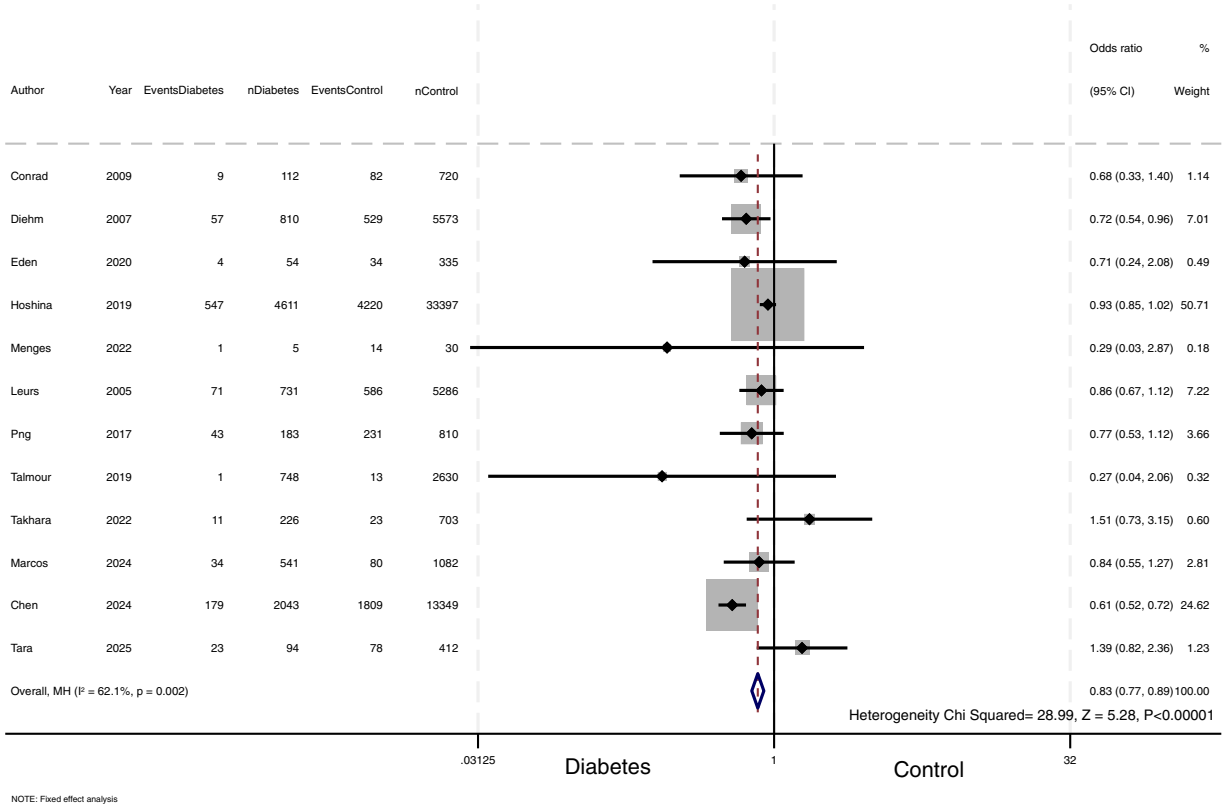

Fig. 2. Forest plot of reintervention rates post-EVAR in those with DM and controls. *P*-values were calculated using the MH (Mantel-Haenszel) chi-square test.

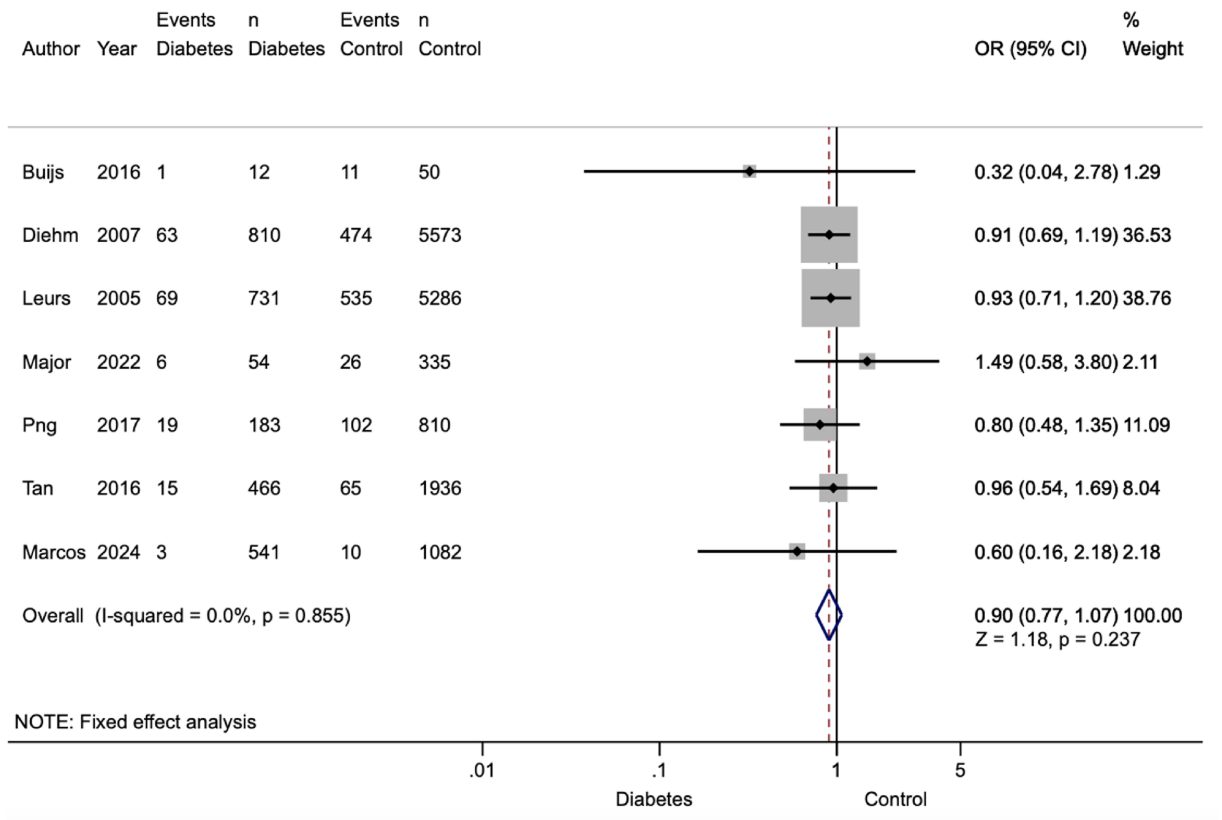

Fig. 3. Forest plot of type I endoleak post-EVAR in those with DM and controls.

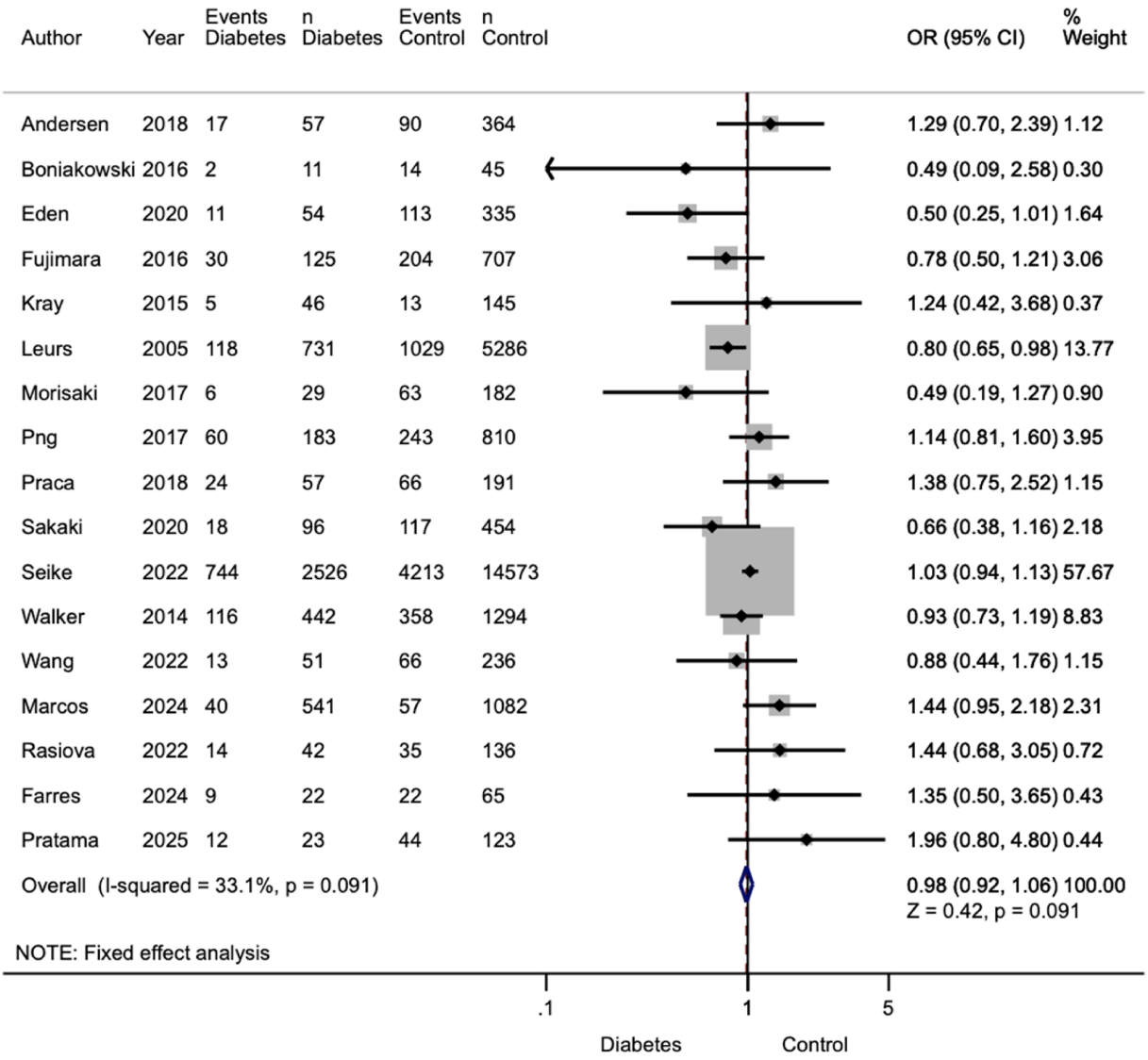

Fig. 4. Forest plot of type II endoleak post-EVAR in those with DM and controls.

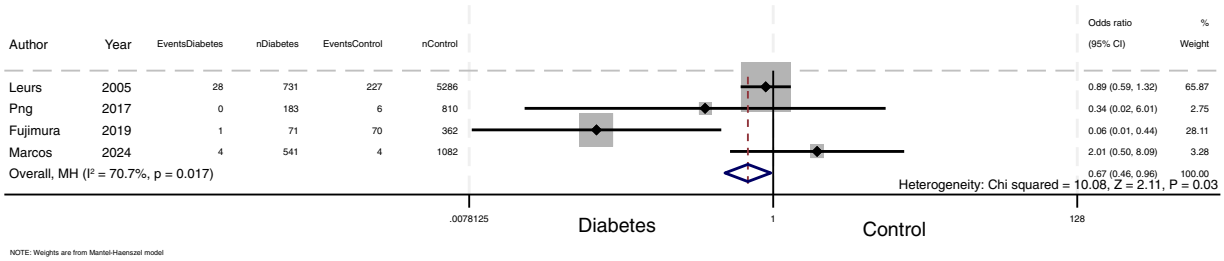

Fig. 5. Forest plot of type III post-EVAR in those with DM and controls. MH, Mantel-Haenszel.

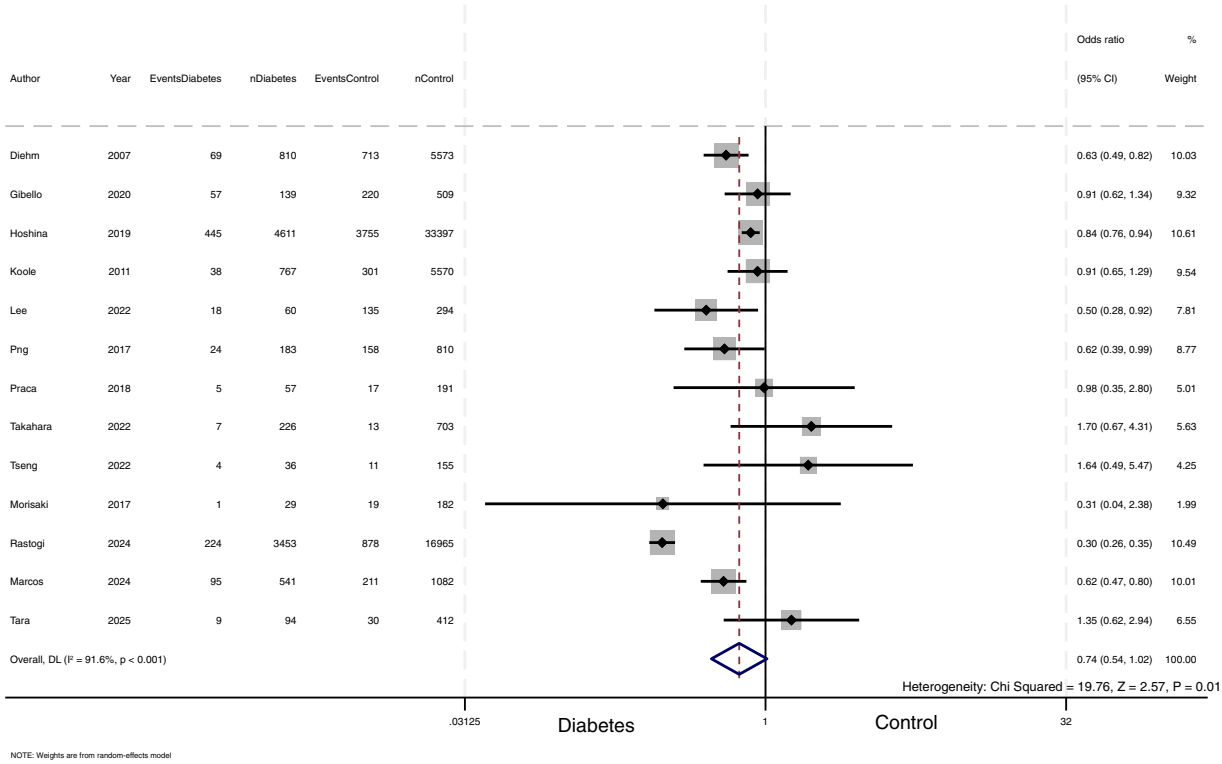

Fig. 6. Forest plot of AAA sac expansion in those with DM and controls. Heterogeneity was assessed using the DL (DerSimonian-Laird) method.

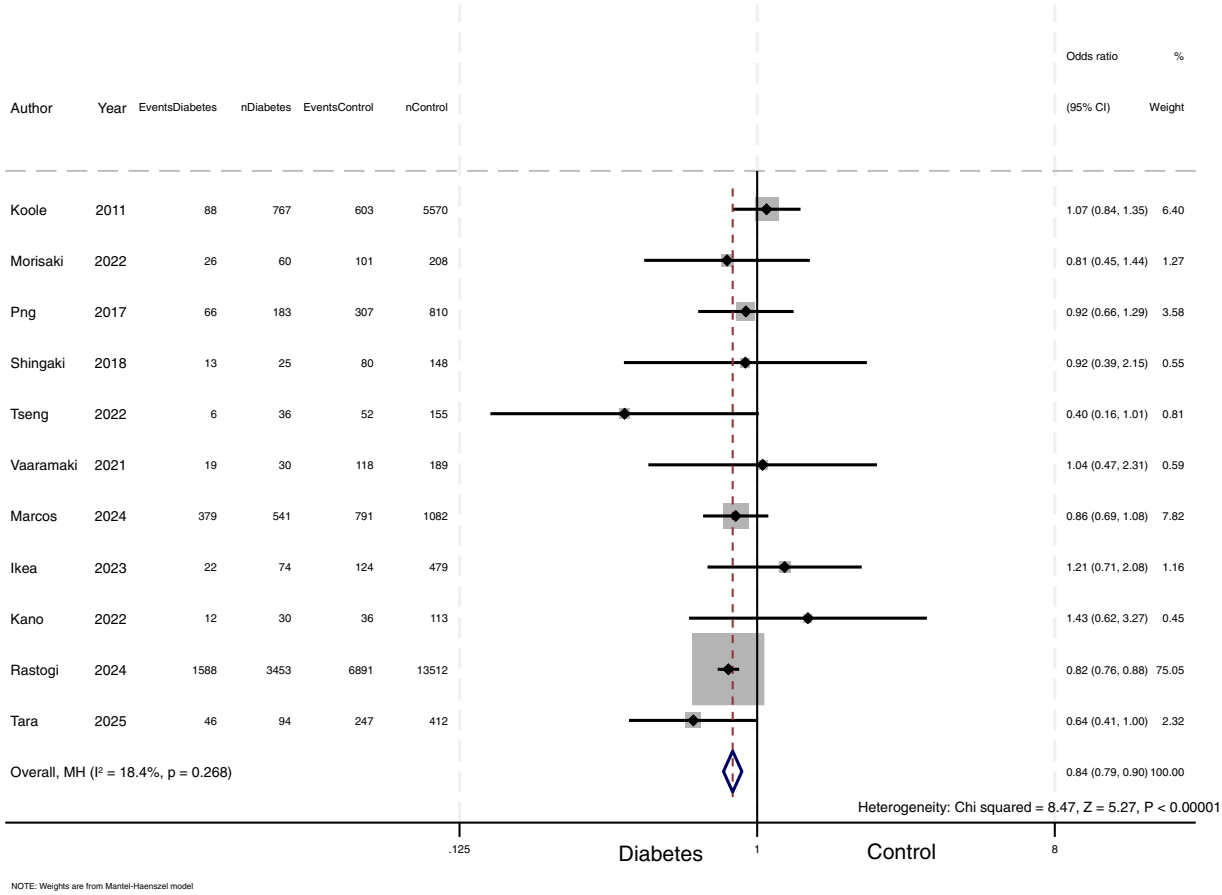

Fig. 7. Forest plot AAA sac shrinkage in those with DM and controls. MH, Mantel-Haenszel.

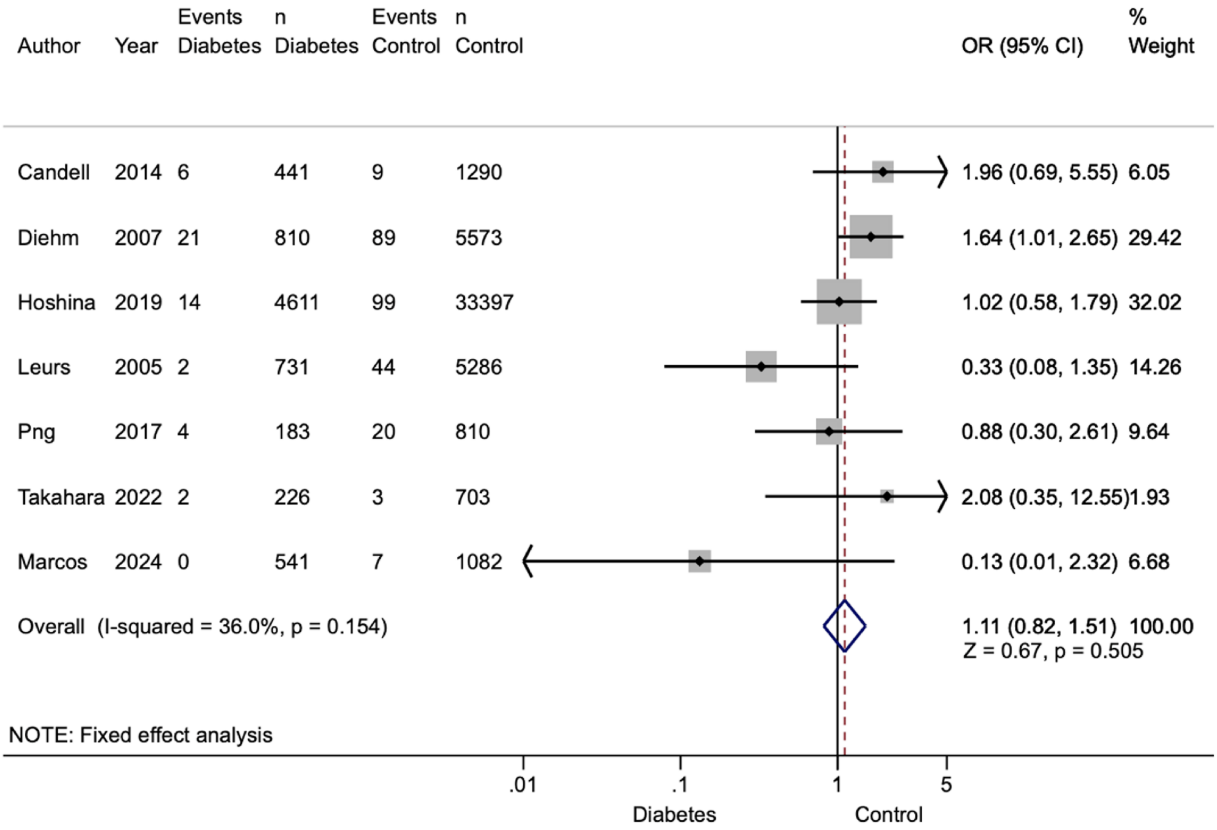

Fig. 8. Forest plot AAA rupture post-EVAR in those with DM and controls.

Meta-Regression for Follow-Up

Length of follow-up (months) across the studies is not a significant modifier of sac expansion *P* value = 0.522 (Fig. 14) and reintervention *P* value = 0.697 (Fig. 15)

DISCUSSION

This systematic review and meta-analysis demonstrates that DM is associated with significantly lower rates of reintervention, sac enlargement and shrinkage, type III endoleak, and conversion to OSR following EVAR, without significant differences in overall endoleak rates, type 1 or 2 endoleaks, or post-EVAR rupture.

A key finding of this analysis is the reduction in sac expansion among patients with diabetes, which likely represents the central mechanism underpinning the observed reduction in reintervention. One of the earliest studies to highlight the diabetes paradox in AAA patients was an observational case-control study[16] utilizing the UK national AAA screening program. This highlighted a significant reduction in AAA growth rates by 56% in patients using medications to control diabetes. Sac enlargement following EVAR is a major driver of secondary intervention, often reflecting ongoing aneurysm pressurization or disease progression. The observed reduction in sac expansion is therefore consistent with prior evidence demonstrating slower AAA growth in patients with diabetes in the untreated setting.[1] This suggests that the biological processes influencing aneurysm progression may persist even after endovascular exclusion.

People with diabetes are more likely to undergo EVAR as opposed to OSR, largely due to microvascular and macrovascular complications associated with diabetes, which increases operative risk.[17] Although EVAR offers lower early mortality, it is associated with higher rates of aneurysm-related complications due to progress of aortic degeneration and dilatation.[18] Within this context, the apparent protective effect of diabetes on sac behavior and downstream complications is particularly notable.

Reintervention remains a key limitation of EVAR. Although 1 study included (Diehm et al.)[19]

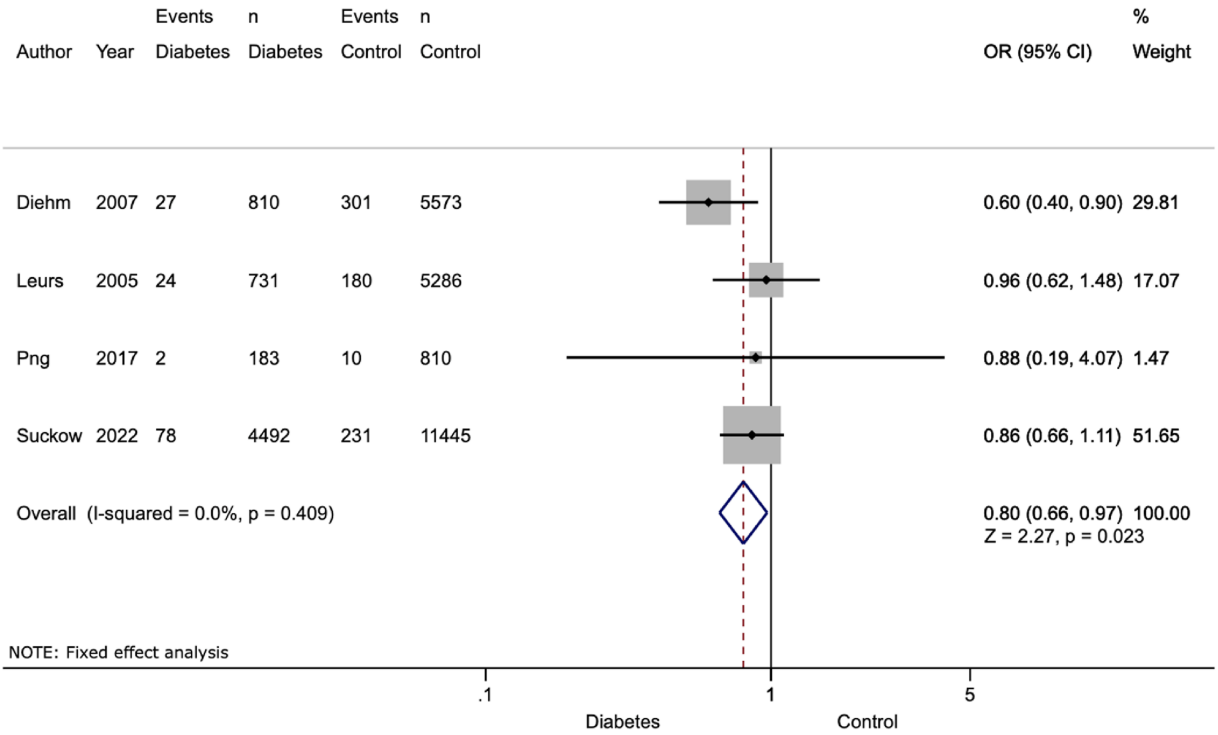

Fig. 9. Forest plot conversion to open surgical repair in those with DM and controls.

Table II. Sensitivity analysis of reinterventions and AAA sac expansion of sample size >500 participants

Outcome	No. Studies	DM (event)	Total DM	Control (event)	Total control	Total participants	OR, 95% CI	I^2	*P* value
Reinterventions	10	975	10.099	7,651	63,962	74,061	0.85 [0.80—0.90]	68%	<0.00001
AAA sac expansion	9	968	10,824	6,279	61,568	72,392	0.86 [0.76—0.98]	45%	0.02
AAA sac shrinkage	6	2,112	5,112	8,963	21,865	26,977	0.92 [0.89—0.95]	26%	<0.00001

showed a significant reduction in reintervention rates individually, pooled analysis identified a consistent and statistically significant reduction in patients with DM, which persisted across sensitivity analyses. Given that endoleaks and sac expansion are the principal drivers of reintervention, further analysis of these outcomes provides insight into potential causal pathways. One of the main causes for reinterventions is endoleak. Six studies reporting type I endoleak showed a no significant association with DM, consistent with meta-analysis. Similarly, 14 studies reported on type II endoleak of which only the study by Leurs et al.[18] showed a significant reduction of type II endoleak in those with diabetes. These findings are consistent with the meta-analysis by Guo et al.[13] Three studies reported type III endoleak, with the study by Fujimura et al.[20] suggesting a significant reduction in type III endoleak in those with DM, which was confirmed on meta-analysis. This may reflect reduced aneurysm sac dynamics and biomechanical stress on the endograft, although the precise mechanism remains unclear.

Previous work has highlighted a reduction in untreated AAA growth rates in people with DM[1] and the current study suggests that this effect persists following EVAR. Sac expansion which could be considered a surrogate for AAA growth was lower in those with DM post-EVAR in 4 of the 10 studies included, and remained significant on meta-analysis and sensitivity analyses.[17] Leurs

Table III. Newcastle–Ottawa Scale quality assessment sensitivity analysis

Outcome	No. Studies	DM (event)	Total DM	Control (event)	Total control	Total participants	OR, 95% CI	I^2	*P* value
Reinterventions	10	429	5,493	3,445	30,595	36,088	0.75 [0.68–0.82]	51%	<0.00001
AAA sac expansion	10	529	6,299	2,560	28,544	15,538	0.87 [0.74–1.02]	41%	0.08
Type I endoleak	6	175	2,785	1,212	15,022	17,807	0.92 [0.79–1.07]	0%	0.28
Type II endoleak	10	1,129	4,229	6,296	23,651	27,880	0.98 [0.91–1.06]	6%	0.68
Post-EVAR AAA rupture	6	35	2,932	172	14,744	17,676	1.15 [0.80–1.66]	44%	0.45
Conversion	3	53	1,724	491	11,669	13,393	0.74 [0.55–0.99]	19%	0.04
Sac shrinkage	7	2,199	5,093	9,037	21,723	26,816	0.92 [0.89–0.95]	0%	<0.00001

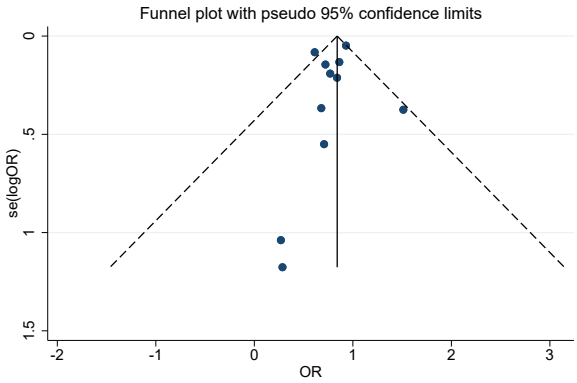

Fig. 10. Funnel plot of AAA reintervention.

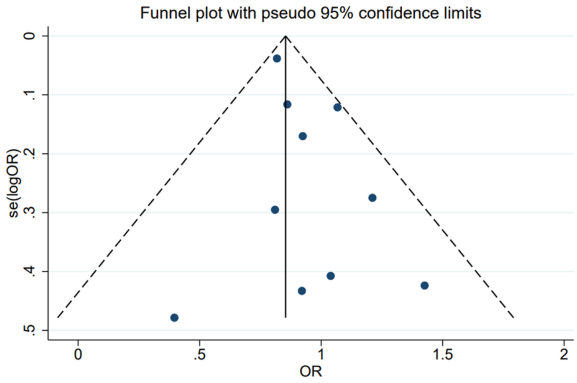

Fig. 12. Funnel plot of AAA sac shrinkage.

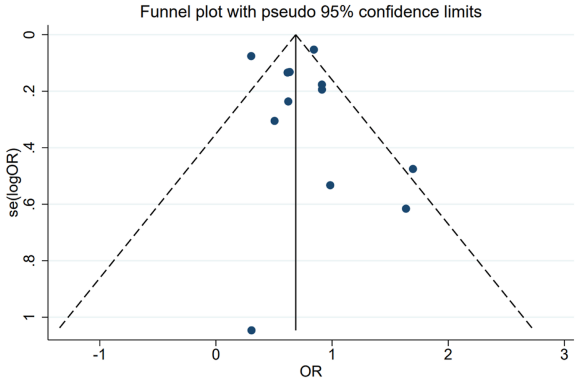

Fig. 11. Funnel plot of AAA sac expansion.

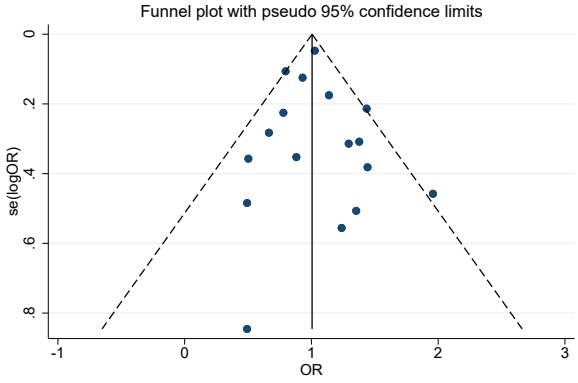

Fig. 13. Funnel plot of type II endoleak.

et al.[18] reported that those with type 2 DM who were insulin-controlled had a significantly lower rate of endoleaks, which resulted in fewer secondary interventions compared to those taking oral hypoglycemic agents or people without diabetes. One explanation for this could be because people with insulin-treated DM receive more intensive monitoring.[21] Chronic hyperglycemia promotes the formation of advanced glycation end-products, resulting in increased collagen cross-linking and a

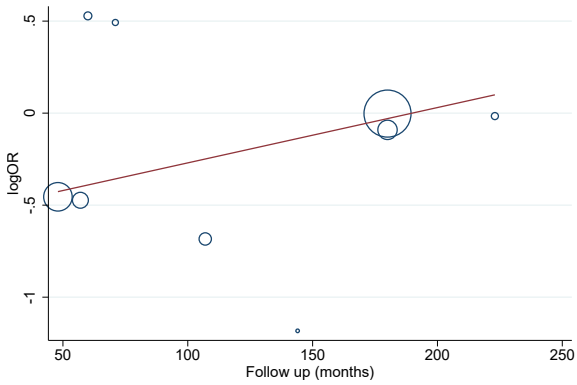

Fig. 14. Bubble plot sac expansion meta-regression.

Table IV. Diabetes and AAA: Results from Eggers test

Meta-analysis	Result	*P* value
Reintervention	no small-study effects	0.375
Sac expansion	no small-study effects	0.775
Sac shrinkage	no small-study effects	0.254
Type II endoleak	no small-study effects	0.836

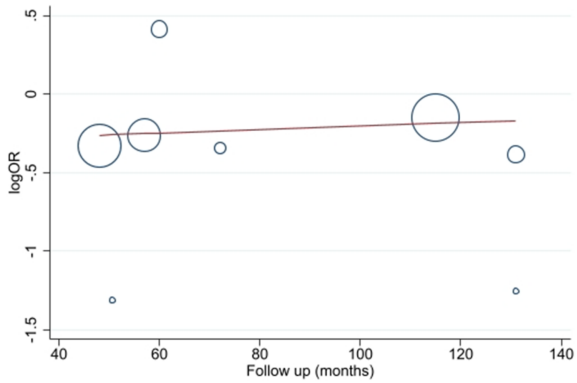

Fig. 15. Bubble plot reintervention meta-regression.

stiffer extracellular matrix, which may reduce susceptibility to aneurysm expansion. In addition, diabetes-associated microvascular disease involving the vasa vasorum may limit neovascularization and the infiltration of inflammatory cells into the aortic wall, thereby attenuating aneurysm progression.[22]

Treatment-related factors may also contribute. Leurs et al.[18] reported that insulin-treated patients had lower rates of endoleak and reintervention compared to those receiving oral hypoglycemic agents or without diabetes. This may reflect more intensive monitoring and follow-up in insulin-treated patients,[21] although differences in underlying disease severity and treatment selection cannot be excluded. Unfortunately, subgroup analysis based on diabetes type or treatment was not possible across the included studies.

No significant difference was observed in post-EVAR rupture, despite trends toward lower rates in patients without diabetes. This may reflect limited statistical power, given the relatively low event rates. Similarly, although individual studies such as Diehm et al.[19] did not consistently demonstrate reduced conversion rates, pooled analysis identified a significant reduction in conversion to open repair among patients with diabetes.

Clinical decision-making may also influence these findings. Reintervention following EVAR is often nuanced, particularly for type 2 endoleaks. Type II endoleaks are often managed conservatively, with most clinicians not undertaking intervention unless the sac expands by at least 10 mm.[23–25] In addition, conversion to OSR is a very complex procedure with a significant mortality.[26] There may therefore be an underlying selection bias in some of these results as those with diabetes may be less likely to have their EVAR explanted due to their comorbidities. It is also unknown as to whether people with diabetes were more aggressively treated with EVAR, resulting in some grafts potentially being used outside of the manufacturers' instructions for use, which would increase the risk of endoleaks, and post-EVAR rupture.[27] Interestingly, although post-EVAR rupture was more common in those without DM, this was not significant, and the rate of type 1 endoleaks was lower in those with DM, although this was also not significant. What remains unknown is the proportion of patients diagnosed with a type I or III endoleak who underwent intervention, and whether this varies between groups, which would affect post-EVAR rupture rates.

This study has several important limitations. There was significant variability in outcome reporting, and not all studies provided comparative analyses. This is further highlighted by the lack of available data on iliac complications, with no reports on limb occlusion rates identified. Furthermore, most of the studies included did not differentiate between types of DM or treatment. Given the distinct pathophysiological characteristics between type 1 and type 2 DM and the potential benefits of some glucose-lowering agents, it is possible that relationships with AAA differ. The presence or absence of diabetes related comorbidities were also not often reported, thus increasing the risk of selection bias,

and potentially excluding those with more significant disease burden from inclusion into the studies.

The majority of the studies included are nonrandomized retrospective observational cohort studies. Although this enables inclusion of a large number of participants and is highly efficient for examining outcomes, they are inherently subject to confounding and bias. The results of this meta-analysis should therefore be interpreted with caution as associations that require further validation, as the overall quality of the evidence is low. Baseline differences in comorbidities between groups were not consistently adjusted for, and studies differed in the duration of the follow-up which may have influenced outcomes. The Newcastle−Ottawa Scale Quality Assessment sensitivity analysis was used to try to explore this and account for such bias; however, these findings should still be interpreted as associative rather than causal.

CONCLUSION

This systematic review and meta-analysis of available data has shown that there is an association between people with diabetes having lower AAA sac expansion and shrinkage, reintervention rates, and conversion rates to OSR post-EVAR compared to those without diabetes. These conclusions are limited by the lack of high quality randomized publications, and further work is necessary to establish why these differences occur.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

E. Otify: Writing − review & editing, Writing − original draft, Software, Resources, Project administration, Methodology, Formal analysis, Data curation, Conceptualization. **I. Saunders:** Investigation, Data curation. **J. Borucki:** Software, Investigation, Formal analysis, Data curation. **K. Dhatariya:** Visualization, Validation, Supervision, Conceptualization. **P. Stather:** Writing − review & editing, Visualization, Validation, Supervision.

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