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REVIEW

Safety of discontinuing oral anticoagulation after atrial fibrillation ablation: an updated systematic review and meta-analysis

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Abstract

Oral anticoagulant (OAC) therapy after atrial fibrillation (AF) ablation is recommended according to thromboembolic risk rather than procedural success alone. However, the clinical balance between residual thromboembolic risk and bleeding risk after OAC discontinuation remains uncertain because randomized controlled trials (RCTs) and observational studies have reported variable estimates. We conducted an updated systematic review and meta-analysis comparing OAC discontinuation with OAC continuation after AF ablation. Online databases were searched from inception to May 2026 for RCTs and observational studies reporting thromboembolic events, major bleeding, or all-cause mortality after post-ablation OAC discontinuation *versus* continuation. Random-effects Mantel-Haenszel models were used, and estimates were reported as risk ratios (RRs) with corresponding 95% confidence intervals (CIs). Thirty-two studies comprising 271,808 patients were included, with 88,513 patients discontinuing OAC after ablation. OAC discontinuation, compared with continuation, was not associated with a statistically significant difference in thromboembolic events (RR=0.93; 95%CI=0.72 to 1.20; P=0.58; I^2=53%). In contrast, OAC discontinuation significantly reduced major bleeding (RR=0.37; 95%CI=0.27 to 0.52; P<0.00001; I^2=60%). No significant effect was observed for all-cause mortality (RR=0.87; 95%CI=0.70 to 1.08; P=0.22; I^2=0%). In this updated systematic review and meta-analysis, OAC discontinuation after AF ablation was associated with lower major bleeding without a statistically significant difference in thromboembolic events or all-cause mortality. These findings support individualized, risk-based anticoagulation decisions among selected post-ablation patients rather than routine discontinuation. However, the very low certainty of evidence for thromboembolic outcomes, coupled with a CI that does not exclude potential harm (RR up to 1.20), underscores that these findings should not be interpreted as evidence of safety.

Key words: atrial fibrillation; catheter ablation; oral anticoagulation; thromboembolism; major bleeding.

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Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and its prevalence continues to increase worldwide.[1,2] In addition to symptoms and impaired quality of life, AF is associated with thromboembolic events, heart failure (HF), and mortality.[1,3] Catheter ablation has become an established rhythm-control strategy for symptomatic AF and has been shown to improve rhythm outcomes and quality of life compared with medical therapy.[4-7] However, the effect of catheter ablation on long-term thromboembolic risk remains less certain.[5,8] This uncertainty is clinically relevant because

atrial substrate and left atrial dysfunction may persist despite rhythm control, particularly in patients with cardiometabolic risk factors and structural atrial remodeling.[8,9]

Oral anticoagulation (OAC) remains the main treatment for prevention of thromboembolic events in patients with AF.[10,11] Current guidelines recommend that long-term OAC decisions after AF ablation should be based on thromboembolic risk profile rather than apparent procedural success alone.[10,11] The CHA_2DS_2-VASc score is widely used to estimate stroke risk, while bleeding risk assessment is used to identify modifiable bleeding risk factors and guide follow-up intensity.[12,13] Although direct oral anticoagulants (DOACs) have improved safety compared with warfarin, continued anticoagulation still exposes patients to bleeding events.[14] Therefore, the decision to continue or discontinue OAC after AF ablation requires balancing residual thromboembolic risk against the bleeding risk of long-term therapy.

Several observational studies have evaluated OAC discontinuation following AF ablation, but differences in patient selection, stroke-risk profile, timing of OAC discontinuation, follow-up duration, and rhythm monitoring have limited interpretation. Randomized evidence from ALONE-AF, and OCEAN-AF, has helped reduce some of this uncertainty although these trials differed in design, comparator treatment, baseline risk, and outcome ascertainment.[15,16] In this systematic review and meta-analysis, we sought to pool data from randomized and observational studies comparing OAC discontinuation with continuation after AF ablation to estimate associations with thromboembolic events, major bleeding, and all-cause mortality.

Materials and Methods

This review was conducted following Cochrane Collaboration guidance, and the findings were reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.[17,18]

Data sources and search

Literature searches were performed in PubMed/Medical Literature Analysis and Retrieval System Online (MEDLINE) and the Cochrane Central Register of Controlled Trials (CENTRAL) from database inception to May 2026. Independent searches were performed by 2 authors (A.A.A.C. and A.A.C.) using the following search terms: atrial fibrillation, ablation, catheter ablation, pulmonary vein isolation, oral anticoagulation, anticoagulant discontinuation, anticoagulant continuation, warfarin, direct oral anticoagulant, thromboembolism, stroke, and bleeding. Abstracts and presentations from major cardiovascular conferences and ClinicalTrials.gov were also searched. Titles and abstracts were screened, and full texts and reference lists were reviewed when eligibility or outcome data could not be determined from the abstract. Details of the search strategy are provided in Supplementary Table 1.

Study selection

Two independent reviewers (A.A.A.C. and A.A.C.) screened the search results and included studies if they met the following criteria: 1) adult patients undergoing catheter ablation for atrial fibrillation (AF); 2) an intervention arm comprising OAC discontinuation after ablation; 3) a comparison arm comprising OAC continuation after ablation; and 4) at least 1 outcome of interest was reported. Randomized controlled trials (RCTs), prospective observational studies, and retrospective observational studies were eligible. Reviews, editorials, case reports, case series without a comparison group, duplicate cohorts, and studies without extractable outcome data were excluded. There was no restriction on sample size or follow-up duration. Disagreements were resolved by a third reviewer (M.T.A.).

Quality assessment and data extraction

Two investigators (A.A.A.C. and A.A.C.) independently abstracted data from studies meeting inclusion criteria, and any disagreements were resolved by a third reviewer (M.T.A.). A structured data collection form was used to abstract the baseline characteristics of the study populations and outcomes of interest. For study characteristics, baseline group sizes were extracted according to the full OAC discontinuation and OAC continuation cohorts. For outcome analyses, event counts and denominators were extracted from the population used for each specific outcome. When studies reported propensity-matched or adjusted analytic cohorts for outcome estimation, those analytic cohorts were used for the corresponding outcome. Therefore, outcome-specific denominators in the forest plots may differ from the total baseline population. In addition, we extracted data on outcomes in the following predefined subgroups: study design, geographic region, OAC type, aspirin use, adjusted analytic cohorts, and risk of bias. The quality of RCTs was assessed using the revised Cochrane Risk of Bias tool for randomized trials (RoB 2).[19] The quality of observational studies was assessed using the Risk Of Bias In Non-randomized Studies of Interventions (ROBINS-I) tool.[20] Certainty of evidence was assessed using the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) approach.[21]

Outcome measures

The primary outcome was thromboembolic events. This included ischemic stroke, transient ischemic attack, systemic embolism, or a composite thromboembolic endpoint as reported in the individual studies. The secondary outcomes included major bleeding and all-cause mortality. Major bleeding was defined according to the definition used in each included study (Supplementary Table 2).

Statistical analysis

The Mantel-Haenszel method for dichotomous data was used to calculate pooled risk ratios (RRs) with corresponding 95%

confidence intervals (CIs). Random-effects models were used for all outcomes because clinical and methodological heterogeneity was expected across study designs, populations, anticoagulation strategies, timing of OAC discontinuation, rhythm monitoring protocols, and outcome definitions.[22] Between-study variance was estimated using the DerSimonian-Laird method, and CIs were calculated using the Wald-type method. Statistical significance was defined as a 2-sided P value <0.05. Statistical heterogeneity was assessed using Cochran Q test and quantified using the I^2 statistic.[23] Leave-one-out sensitivity analyses were performed by sequentially excluding one study at a time to assess whether pooled estimates were driven by any single study. Subgroup analyses were performed according to study design, adjusted analytic cohorts, geographic region, OAC type, aspirin use, and risk of bias when sufficient studies were available.

Univariable meta-regression was performed for outcomes with residual between-study heterogeneity and sufficient numbers of studies. Candidate study-level moderators included study design, total sample size, male proportion, paroxysmal AF proportion, and follow-up duration. Meta-regression was considered exploratory because analyses were based on aggregate study-level data. Small-study effects were assessed by visual inspection of funnel plots and Egger regression test when at least 10 studies were available for an outcome.[24] Analyses were performed using Review Manager Web and R version 4.5.3.

Results

Study characteristics

The literature search and study selection process are summarized in the PRISMA flow diagram Figure 1. A total of 32 studies comprising 271,808 patients were included in this meta-analysis. Of these, 3 were RCTs, 10 were prospective observational studies, and 19 were retrospective observational studies. Overall, 88,513 patients discontinued OAC after ablation, whereas 183,295 patients continued OAC. The baseline characteristics of the included studies are shown in Table 1. The studies were published between 2006 and 2025, and follow-up ranged from 1 to 5.1 years. Warfarin was used in 15 studies, DOACs were used in 4 studies, and both warfarin and DOACs were used in 13 studies.

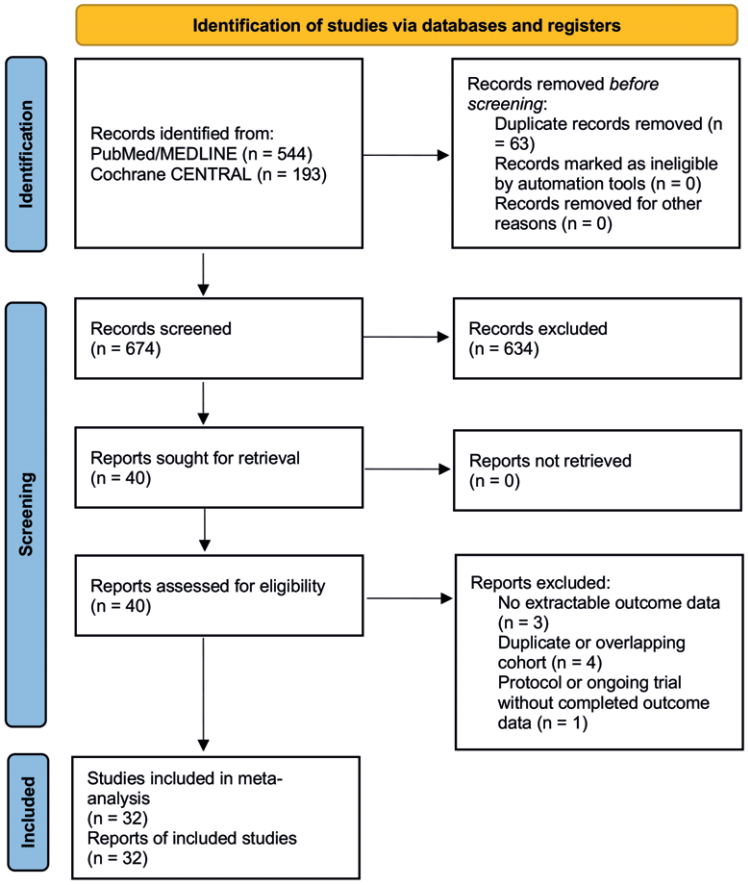

Figure 1. Flowchart of study selection in the meta-analysis.

CENTRAL, Cochrane Central Register of Controlled Trials; MEDLINE, Medical Literature Analysis and Retrieval System Online; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

Table 1. Baseline characteristics of included studies.

Study	Year	Design	OAC discontinuation, n	OAC continuation, n	Total, n	Age, y†	Male sex, n (%)	Hypertension, n (%)	Diabetes mellitus, n (%)	Heart failure, n (%)	CAD/IHD/vascular disease, n (%)	Prior stroke/TIA/TE, n (%)	CHA_2DS_2-VASc score/category †	$CHADS_2$ score/category †	AF type, n (%)	OAC type	OAC discontinuation timing after ablation, mo*	Ablation energy source	Follow-up, y†
Oral 2006	2006	Prospective observational	398	357	755	Overall by stroke-risk strata: 51 ± 9 / 58 ± 10	577 (76%) overall	NR	NR	NR	NR	NR	NR	0 = 53%, ≥1 = 47% / 0 = 37%, ≥1 = 63%	PAF: 490 (64%) overall	Warfarin	3	RFA	2.1
Nademanee 2008	2008	Prospective observational	434	201	635	NR	423 (66%) overall	NR	NR	NR	NR	NR	NR	NR	PAF: 254 (40%) overall	Warfarin	3	RFA	2.3
Bunch 2009	2009	Prospective observational	123	507	630	59.8 ± 10.7 / 66.0 ± 10.1	81 (65%) / 294 (58%)	NR	NR	NR	NR	NR	NR	≤1 = 123 (100%) / ≥3 = 456 (71%)	PAF: 96 (78%) / 229 (45%)	Warfarin	3	RFA	1
Themistoclakis 2010	2010	Retrospective observational	2,692	663	3,355	NR	2,116 (79%) / 463 (70%)	NR	NR	NR	NR	NR	NR	0 = 1,622 (60%), 1 = 723 (27%), ≥2 = 347 (13%) / 0 = 155 (23%), 1 = 261 (39%), ≥2 = 247 (37%)	PAF: 1,682 (62%) / 340 (51%)	Warfarin	3 to 6	NR	2
Guiot 2011	2011	Retrospective observational	561	455	1,016	Overall: 70 ± 4	728 (71%) overall	NR	NR	NR	NR	NR	NR	NR	PAF: 613 (60%) overall	Warfarin	3	RFA	2.8
Hunter 2011	2011	Prospective observational	809	464	1,273	NR	942 (74%) overall	NR	NR	NR	NR	NR	NR	0.7 ± 0.9 (71%) / 0.9 ± 0.9 (69%)	PAF: 713 (56%) overall	Warfarin	3	RFA or CBA	3
Hussein 2011	2011	Prospective observational	449	382	831	NR	644 (77%) overall	NR	NR	NR	NR	NR	NR	NR	PAF: 482 (58%) overall	Warfarin	12	RFA	3.6
Saad 2011	2011	Retrospective observational	298	29	327	Overall: 63 ± 13	258 (79%) overall	NR	NR	NR	NR	NR	NR	<2 = 103 (31%); 2 = 148 (45%); 3 = 76 (23%)	PAF: 195 (60%) overall	Warfarin	3	RFA	3.1
Yagishita 2011	2011	Retrospective observational	432	91	523	NR	427 (81%) overall	NR	NR	NR	NR	NR	NR	0 = 49%, 1 = 36%, ≥2 = 15% / 0 = 46%, 1 = 36%, ≥2 = 18%	PAF: 100%	Warfarin	3	RFA	3.6
Winkle 2013	2013	Prospective observational	60	48	108	Overall: 66.2 ± 9.0	68 (62%) overall	Overall: 62.9%	Overall: 14.5%	NR	Overall CAD: 26.8%	100% prior CVA/TIA cohort	4.1 ± 1.4	3.0 ± 0.9	PAF: 40 (37%) overall	Warfarin/DOAC	3	RFA	2.8
Karasoy 2014	2014	Retrospective observational	1,425	2,625	4,05	NR	2,976 (73%) overall	NR	NR	NR	NR	NR	NR	NR	NR	Warfarin	3	RFA	3.3
Riley 2014	2014	Retrospective observational	1,031	959	1,99	NR	808 (78%) / 727 (76%)	NR	NR	NR	NR	NR	NR	0 = 53%, 1 = 37%, ≥2 = 10% / 0 = 34%, 1 = 48%, ≥2 = 18%	PAF: 764 (74%) / 543 (57%)	Warfarin	1	NR	4
Uhm 2014	2014	Retrospective observational	296	312	608	NR	228 (77%) / 239 (76%)	NR	NR	NR	NR	NR	1.45 ± 1.34 / 1.55 ± 1.36	0.91 ± 0.98 / 1.04 ± 1.04	PAF: 222 (75%) / 234 (75%)	Warfarin	3	RFA	1.5
Gallo 2016	2016	Retrospective observational	500	500	1	NR	361 (72%) / 323 (64.6%)	NR	NR	NR	NR	NR	1.9 ± 0.9 / 2.4 ± 1	NR	PAF: 276 (55%) / 232 (46%)	Warfarin	3	NR	5
Själander 2016	2016	Retrospective observational	360	815	1,175	NR	NR	NR	NR	NR	NR	NR	1.5 ± 1.4	NR	NR	Warfarin	1	NR	2.6
Kochhäuser 2017	2017	Retrospective observational	276	122	398	NR	300 (75%) overall	NR	NR	NR	NR	NR	NR	0 = 53%; 1 = 40%; ≥2 = 7% / 0 = 17%; 1 = 36%; ≥2 = 47%	PsAF: 119 (29.9%)	Warfarin/DOAC	3	RFA	1.5
Liang 2018	2018	Retrospective observational	174	226	400	NR	151 (86%) / 177 (78%)	NR	NR	NR	NR	NR	0 or 1 = 77%, ≥2 = 22% / 0 or 1 = 46%, ≥2 = 53%	NR	PsAF: 109 (48%) / 91 (52%)	Warfarin/DOAC	NR	NR	3.6
Arai 2019	2019	Retrospective observational	230	282	512	61.4 ± 10.7 / 65.1 ± 9.9	182 (79%) / 207 (73%)	126 (55%) / 178 (63%)	37 (16%) / 72 (26%)	19 (8%) / 48 (17%)	20 (9%) / 36 (13%)	15 (7%) / 48 (17%)	≥3 = 57 (29%) / 140 (71%)	1.03 ± 0.98 / 1.56 ± 1.18	PAF: 144 (63%) / 134 (48%)	Warfarin/DOAC	3	RFA or CBA	2.3
Okumura 2019	2019	Retrospective observational	1,836	1,615	3,451	NR	1,407 (76%) / 1,152 (71%)	NR	NR	NR	NR	NR	≤2 = 1,420 (78%), ≥3 = 416 (22%) / ≤2 = 866 (53%), ≥3 = 749 (47%)	≤2 = 1,737 (95%), ≥3 = 99 (5%) / ≤2 = 1,338 (83%), ≥3 = 277 (17%)	PAF: 1,231 (67%) / 1,926 (57%)	Warfarin/DOAC	3	RFA or CBA	1.6
Hermida 2020	2020	Prospective observational	75	375	450	NR	59 (79%) / 290 (77%)	NR	NR	NR	NR	NR	0.7 ± 1.0 / 1.8 ± 1.3	NR	PAF: 242 (54%) overall	Warfarin/DOAC	3	CBA	2.5
Lauritzen 2020	2020	Retrospective observational	150	286	436	NR	112 (74%) / 220 (76%)	NR	NR	NR	NR	NR	≤2 = 86 (57%), ≥3 = 64 (42%) / ≤2 = 123 (46%), ≥3 = 141 (53%)	≤2 = 134 (89%), ≥3 = 16 (10%) / ≤2 = 229 (80%), ≥3 = 57 (20%)	PAF: 114 (76%) / 163 (57%)	Warfarin	3 and 12	RFA or CBA	5.1
Yang 2020	2020	Prospective observational	3,149	1,363	4,512	NR	2,040 (64%) / 824 (60%)	NR	NR	NR	NR	NR	2.3 ± 1.3 / 2.7 ± 1.4	NR	PsAF: 940 (29%) / 453 (33%)	Warfarin/DOAC	3	RFA	2
Yu 2020	2020	Retrospective observational	989	502	1,491	Overall: 59.6 ± 12.1	615 (62%) / 303 (60%)	NR	NR	NR	NR	NR	1.5 ± 1.4 / 2.4 ± 1.7	NR	PAF: 100%	Warfarin/DOAC	3	RFA or CBA	2.3
Inoue 2021	2021	Prospective observational	479	496	975	NR	372 (77%) / 338 (68%)	NR	NR	NR	NR	NR	≤2 = 391 (81%) / 280 (56%); ≥3 = 88 (18%) / 216 (44%)	<3 = 474 (95%) / 476 (99%); ≥4 = 3 (0.6%) / 22 (4.6%)	PAF: 324 (67%) / 279 (56%)	Warfarin/DOAC	NR	NR	2.3
Fei 2024	2024	Retrospective observational	1,055	1,062	2,117	68.60 ± 9.35 / 69.29 ± 9.05	594 (56%) / 571 (53%)	768 (72.8%) / 763 (71.8%)	264 (25.0%) / 281 (26.5%)	239 (22.7%) / 268 (25.2%)	430 (40.8%) / 433 (40.8%)	281 (26.6%) / 279 (26.3%)	NR	3.41 ± 1.12 / 3.47 ± 1.12	PsAF: 446 (42%) / 465 (43%)	Warfarin/DOAC	3	NR	3.1
Kanaoka 2024	2024	Retrospective observational	66,736	164,638	231,374	NR	161,440 (70%) overall	NR	NR	NR	NR	NR	NR	≤1 = 56,038 / 105,132; 2 = 8,577 / 41,400; ≥3 = 2,121 / 18,106	PAF: 42,093 (63%) / 83,016 (50%)	Warfarin/DOAC	6	RFA or CBA	3
Schrickel 2024 (ODIn-AF)	2024	RCT	101	99	200	NR	56 (56%) / 56 (57%)	NR	NR	NR	NR	NR	≤2 = 58 (59%), ≥3 = 43 (41%) / ≤2 = 51 (51%), ≥3 = 50 (49%)	NR	PAF: 68 (67%) / 65 (66%)	DOAC	6	RFA or CBA	1
ALONE-AF 2025	2025	RCT	417	423	840	Overall: 64 ± 8	321 (77%) / 310 (73%)	NR	NR	NR	NR	NR	2 ± 1.9 / 2 ± 1.49	NR	PAF: 276 (66.2%) / 292 (69%)	DOAC	NR	RFA or CBA	2

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Table 1. Continued from previous page.

Study	Year	Design	OAC discontinuation ,n	OAC continuation, n	Total, n	Age, y†	Male sex, n (%)	Hypertension, n (%)	Diabetes mellitus, n (%)	Heart failure, n (%)	CAD/IHD/vascular disease, n (%)	Prior stroke/TIA/TE, n (%)	CHA_2DS_2-VASc score/category †	$CHADS_2$ score/category †	AF type, n (%)	OAC type	OAC discontinuation timing after ablation, mo*	Ablation energy source	Follow-up, y†
Iwawaki 2025	2025	Retrospective observational	899	922	1,821	60.5 ± 12.1 / 66.6 ± 10.4	696 (77%) / 643 (69%)	356 (39.6%) / 541 (58.7%)	89 (9.9%) / 189 (20.5%)	63 (7.0%) / 228 (24.7%)	IHD 51 (5.7%) / 85 (9.2%); vascular 59 (6.6%) / 96 (10.4%)	Ischemic stroke: 23 (2.6%) / 97 (10.5%)	1.43 ± 1.32 / 2.56 ± 1.57	0.71 ± 0.84 / 1.51 ± 1.12	PAF: 644 (71%) / 555 (60%)	Warfarin/DOAC	12	RFA or CBA	5
Nakano 2025	2025	Retrospective observational	222	205	427	61.9 ± 9.2 / 66.4 ± 8.5	179 (81%) / 147 (72%)	103 (46%) / 137 (67%)	17 (8%) / 36 (18%)	28 (13%) / 29 (14%)	9 (4%) / 25 (12%)	6 (3%) / 3 (2%)	≥2 = 95 (43%) / 149 (73%)	≤2 = 222 (100%) / 205 (100%)	PSAF: 54 (24%) / 47 (23%)	Warfarin/DOAC	3	NR	3.2
OCEAN-AF 2025	2025	RCT	643	641	1,284	NR	NR	NR	NR	NR	NR	NR	2.2 ± 1.1 / 2.2 ± 1.1	NR	PAF: 421 (65.5%) / 431 (67.2%)	DOAC	NR	NR	3
Oda 2025	2025	Prospective observational	1,214	1,63	2,844	NR	2,016 (71%) overall	NR	NR	NR	NR	NR	≥1 = 1856 (65%); ≥1 = 1016 (36%)	NR	PAF : 1,821 (64%) overall	DOAC	3	NR	1

Values are presented as OAC discontinuation/OAC continuation when arm-specific data were available. Overall cohort values are shown only when arm-specific values were not available.

†Continuous variables are presented as mean ± SD or median [IQR], according to the source study. Follow-up is reported in years. OAC discontinuation timing is reported in months after ablation.

*In studies reporting more than one discontinuation time point or a range, OAC was discontinued between the stated time points according to rhythm status and/or stroke-risk criteria in the original study.

AF, atrial fibrillation; CAD, coronary artery disease; CBA, cryoballoon ablation; DOAC, direct oral anticoagulant; IHD, ischemic heart disease; NR, not reported; OAC, oral anticoagulant; PAF, paroxysmal atrial fibrillation; PsAF, persistent atrial fibrillation; RCT, randomized controlled trial; RFA, radiofrequency ablation; TE, thromboembolism; TIA, transient ischemic attack.

Thromboembolic events

Thirty-two studies reported thromboembolic events. A total of 789 thromboembolic events occurred among 77,782 patients in the OAC discontinuation group, whereas 709 events occurred among 74,639 patients in the OAC continuation group. OAC discontinuation compared with OAC continuation was not associated with a significant difference in thromboembolic events (RR, 0.93; 95% CI, 0.72 to 1.20; P=0.58; I^2=53%) (Figure 2).

Subgroup analyses according to prospective *versus* retrospective design, RCTs *versus* adjusted cohort studies, geographic region, and aspirin use showed no statistically significant interaction for thromboembolic events (P for subgroup difference = 0.35, 0.50, 0.37, and 0.68) (Supplementary Figures 1-4). No significant subgroup differences were identified across these analyses. When the analysis was restricted to studies at low or moderate risk of bias, OAC discontinuation was not associated with a significant difference in thromboembolic events (RR, 1.34; 95% CI, 0.99 to 1.81; P=0.06; I^2=59%) (Supplementary Figure 5).

Major bleeding

Twenty-two studies reported major bleeding. A total of 493 major bleeding events occurred among 74,815 patients in the OAC discontinuation group, whereas 871 events occurred among 71,622 patients in the OAC continuation group. OAC discontinuation compared with OAC continuation significantly reduced major bleeding (RR, 0.37; 95% CI, 0.27 to 0.52; P<0.00001; I^2=60%) (Figure 3).

Subgroup analyses according to OAC type, RCTs *versus* adjusted cohort studies, aspirin use, geographic region, and prospective *versus* retrospective design showed no statistically significant interaction for major bleeding (P for subgroup difference = 0.08, 0.36, 0.74, 0.08, and 0.59) (Supplementary Figures 6-10). No significant subgroup differences were identified across these analyses. When the analysis was restricted to studies at low or moderate risk of bias, OAC discontinuation remained associated with a significant reduction in major bleeding (RR, 0.52; 95% CI, 0.38 to 0.70; P<0.0001; I^2=50%) (Supplementary Figure 11).

All-cause mortality

Fifteen studies reported all-cause mortality. A total of 130 deaths occurred among 8,349 patients in the OAC discontinuation group, whereas 188 deaths occurred among 9,104 patients in the OAC continuation group. OAC discontinuation compared with OAC continuation was not associated with a significant difference in all-cause mortality (RR, 0.87; 95% CI, 0.70 to 1.08; P=0.22; I^2=0%) (Figure 4).

Subgroup analyses according to RCTs *versus* adjusted cohort studies, geographic region, and prospective *versus* retrospective design showed no statistically significant interaction for all-cause mortality (P for subgroup difference = 0.65, 0.36, and 0.64) (Supplementary Figures 12-14). No significant subgroup differences were identified across these analyses. When the analysis was restricted to studies at low or moderate risk of bias, OAC discontinuation was not associated with a significant difference in all-cause mortality (RR, 0.92; 95% CI, 0.73 to 1.16; P=0.48; I^2=0%) (Supplementary Figure 15).

Leave-one-out sensitivity analyses

Leave-one-out sensitivity analyses showed that the pooled estimates were not driven by any single study. For thromboembolic events, sequential exclusion of individual studies did not change the direction of the pooled estimate, and the association remained statistically non-significant across all sensitivity iterations (Supplementary Figure 16). Heterogeneity remained moderate after individual study exclusions, indicating that no single study fully accounted for the observed between-study variability.

For major bleeding, the pooled association remained statistically significant in favor of OAC discontinuation after each single-study omission (Supplementary Figure 17). Heterogeneity persisted across the sensitivity analyses, although its magnitude varied modestly after individual study exclusions. Overall, these findings support the stability of the primary pooled estimates while suggesting that residual heterogeneity was not attributable to any single study.

Meta-regression analyses

Univariable meta-regression was performed for thromboembolic events and major bleeding to explore potential study-level sources of residual between-study heterogeneity. For thromboembolic events, longer follow-up duration was associated with larger study-level effect estimates (β=0.3186; 95% CI, 0.0759-0.5612; P=0.010), with inclusion of follow-up duration explaining 31.6% of the estimated between-study heterogeneity (pseudo-R^2 = 31.6%) (Supplementary Figure 18). Design category, total sample size, male proportion, and paroxysmal AF proportion were not significantly associated with thromboembolic effect estimates (Supplementary Figures 19-22). For major bleeding, none of the evaluated moderators were significantly associated with the pooled effect estimates (Supplementary Table 3; Supplementary Figures 23-27).

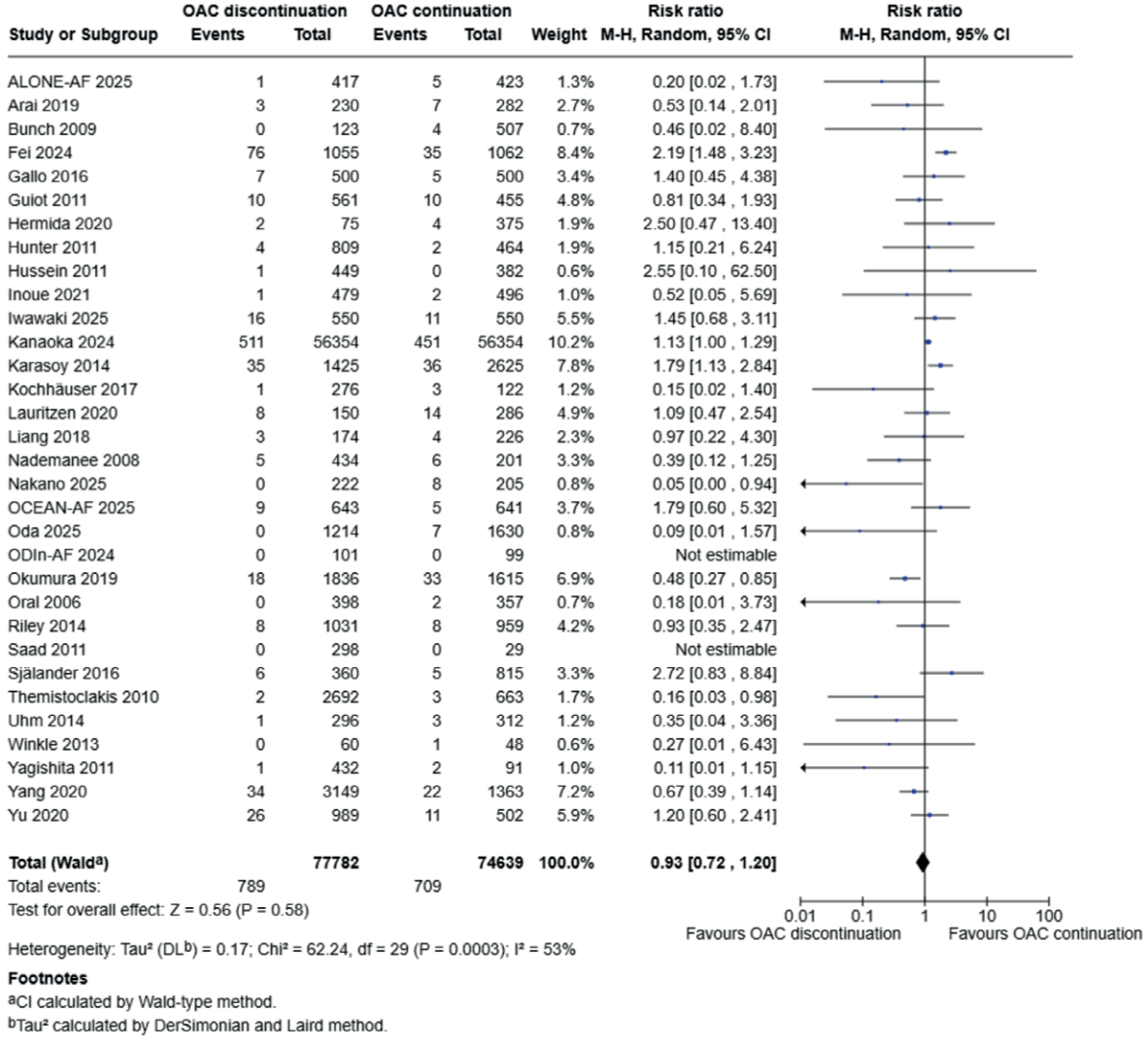
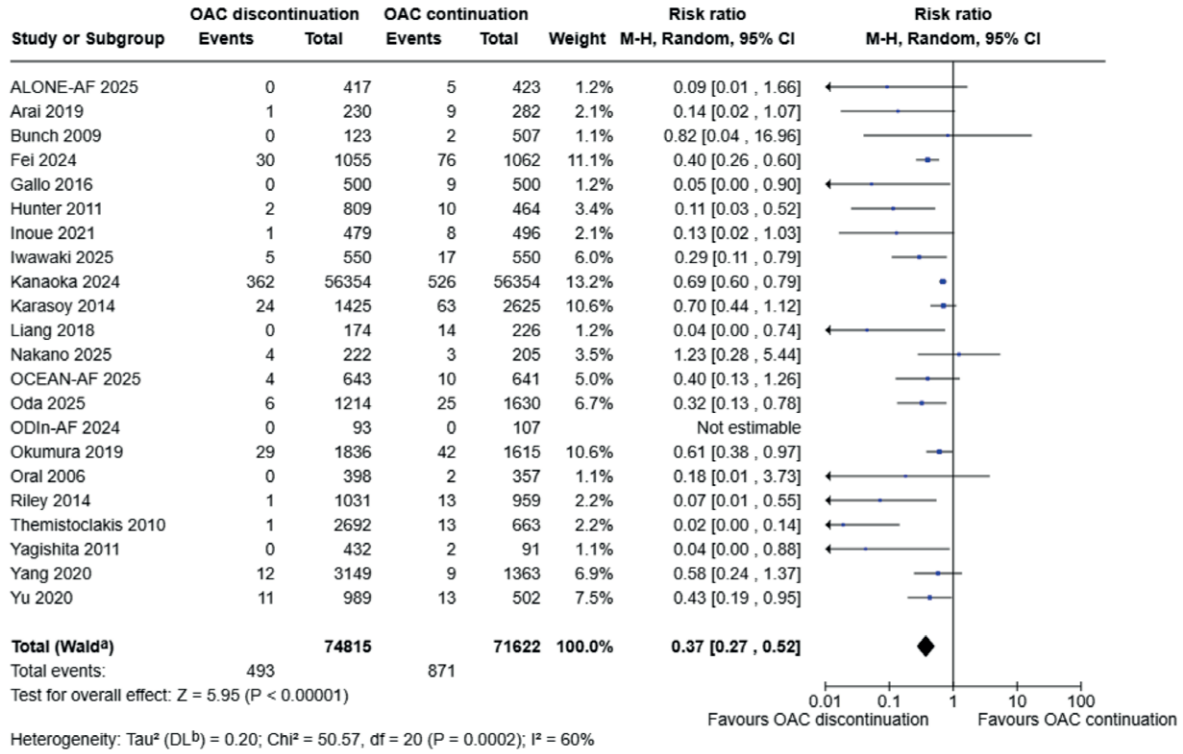


Figure 2. Forest plot for thromboembolic events with oral anticoagulation (OAC) discontinuation versus OAC continuation after atrial fibrillation ablation. CI, confidence interval; M-H, Mantel-Haenszel; OAC, oral anticoagulation; RR, risk ratio.

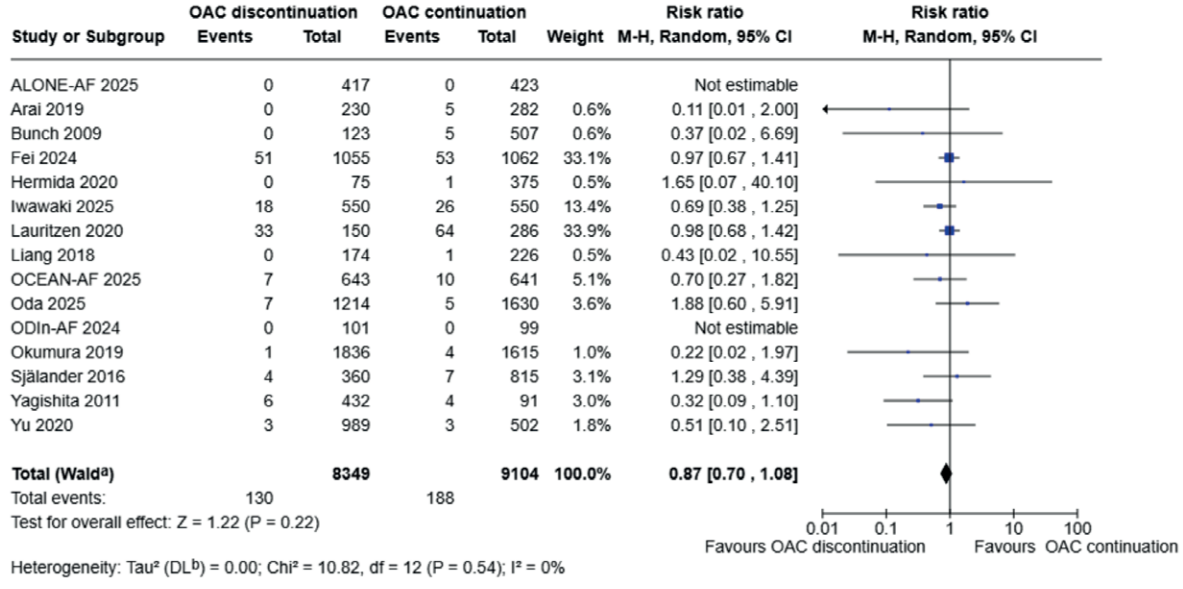


Footnotes

[a]CI calculated by Wald-type method.

[b]Tau² calculated by DerSimonian and Laird method.

Figure 3. Forest plot for major bleeding with oral anticoagulation (OAC) discontinuation versus OAC continuation after atrial fibrillation ablation. CI, confidence interval; M-H, Mantel-Haenszel; OAC, oral anticoagulation; RR, risk ratio.



Footnotes

[a]CI calculated by Wald-type method.

[b]Tau² calculated by DerSimonian and Laird method.

Figure 4. Forest plot for all-cause mortality with oral anticoagulation (OAC) discontinuation versus OAC continuation after atrial fibrillation ablation. CI, confidence interval; M-H, Mantel-Haenszel; OAC, oral anticoagulation; RR, risk ratio.

Quality assessment

The 3 randomized controlled trials, ALONE-AF 2025, OCEAN-AF 2025, and ODIn-AF 2024, were assessed with the RoB 2.[15,16,25] All 3 randomized controlled trials were rated as low risk of bias overall, with low risk across the domains of randomization, deviations from intended interventions, missing outcome data, outcome measurement, and selection of the reported result (Supplementary Figure 28).

The 29 observational studies were assessed with the ROBINS-I tool. Overall, 9 studies were judged to have moderate risk of bias, 10 had serious risk of bias, and 10 had critical risk of bias. Moderate risk of bias was assigned to Fei 2024, Hermida 2020, Iwawaki 2025, Kanaoka 2024, Karasoy 2014, Lauritzen 2020, Själander 2016, Uhm 2014, and Yang 2020.[26-34] Serious risk of bias was assigned to Arai 2019, Gallo 2016, Kochhäuser 2017, Liang 2018, Nakano 2025, Oda 2025, Okumura 2019, Riley 2014, Saad 2011, and Themistoclakis 2010.[35-44] Critical risk of bias was assigned to Bunch 2009, Guiot 2011, Hunter 2011, Hussein 2011, Inoue 2021, Nademanee 2008, Oral 2006, Winkle 2013, Yagishita 2011, and Yu 2020.[45-54] The principal limitations were imbalance in baseline thromboembolic risk, confounding by indication, and non-random selection of patients for OAC continuation or discontinuation (Supplementary Figure 29).

Egger regression suggested funnel plot asymmetry for thromboembolic events and major bleeding, but not for all-cause mortality. The Egger test was significant for thromboembolic events (32 studies; P=0.016) and major bleeding (22 studies; P<0.001), whereas no significant asymmetry was detected for all-cause mortality (15 studies; P=0.117) (Supplementary Figures 30-32).

Certainty of evidence

Using the GRADE approach, the certainty of evidence was rated as very low for thromboembolic events, low for major bleeding, and very low for all-cause mortality (Supplementary Table 4).

For thromboembolic events, certainty was rated as very low. The rating was limited by the predominance of observational evidence, important risk of bias from confounding and patient selection, inconsistency across studies, imprecision around the pooled estimate, and evidence of small-study effects (Supplementary Table 4).

For major bleeding, certainty was rated as low. The rating was limited by risk of bias and indirectness related to variation in bleeding definitions across studies. Certainty was not further downgraded for imprecision because the pooled estimate was precise and the confidence interval did not cross the null (Supplementary Table 4).

For all-cause mortality, certainty was rated as very low. The rating was limited by very serious risk of bias, imprecision, and concern for selective outcome reporting. Although statistical heterogeneity was low, mortality was not consistently prespecified or reported across studies, which limited confidence in the estimate (Supplementary Table 4).

Overall, certainty was limited mainly by nonrandomized study design, confounding by indication, imbalance in baseline thromboembolic risk, heterogeneity in outcome definitions, small-study effects for thromboembolic events and major bleeding, and incomplete consistency of mortality reporting.

Discussion

We report several important findings in this systematic review and meta-analysis. OAC discontinuation after AF ablation was not associated with a statistically significant difference in thromboembolic events compared with OAC continuation. In contrast, OAC discontinuation was associated with a significant reduction in major bleeding. All-cause mortality did not differ significantly between strategies. The overall clinical signal was therefore driven by a reduction in major bleeding, without a measurable difference in thromboembolic or mortality outcomes.

Previous meta-analyses have also shown that OAC discontinuation after AF ablation was not associated with a statistically significant difference in thromboembolic events in selected post-ablation patients.[55-58] Additionally, these analyses reported lower bleeding or intracranial hemorrhage events after OAC discontinuation, although bleeding definitions and included study designs varied across reports.[55-58] However, these analyses were limited by observational study designs, selected patient populations, differences in baseline thromboembolic risk, variable rhythm surveillance, and limited randomized data. The present meta-analysis incorporates recent RCTs and observational studies and shows that OAC discontinuation was associated with significantly lower major bleeding, while no statistically significant difference was observed for thromboembolic events or all-cause mortality. Therefore, these findings support individualized, risk-based decision-making rather than routine anticoagulation discontinuation after AF ablation.

The randomized evidence remains clinically informative but not definitive. In ALONE-AF, OAC discontinuation after apparently successful ablation reduced the composite of stroke, systemic embolism, and major bleeding, with the overall signal largely influenced by fewer major bleeding events.[15] In OCEAN-AF, continued rivaroxaban was not superior to aspirin for prevention of stroke, systemic embolism, or covert cerebral embolic lesions after successful ablation in patients with stroke risk factors.[16] However, the use of an aspirin comparator arm, rather than a strategy of sustained OAC continuation, limits the generalizability of these findings to the broader clinical question of OAC discontinuation, because aspirin is not indicated as a primary stroke prophylaxis strategy in contemporary AF management. ODIn-AF evaluated dabigatran after pulmonary vein isolation using silent cerebral embolic lesions and clinically relevant cardioembolic events as outcomes.[25] These trials move the field beyond observational inference, but their selected populations and low event rates limit the ability to define a universal post-ablation anticoagulation strategy.

Observational evidence further supports the need for risk based selection. In a large Japanese cohort, the balance between thromboembolic protection and bleeding risk differed across

baseline stroke-risk categories.[29] In another recent cohort, OAC discontinuation was associated with higher thromboembolic risk among patients with asymptomatic AF, reduced cardiac function, and larger left atrial diameter, while bleeding risk was lower after discontinuation in patients with higher bleeding risk.[28] Earlier registry studies also suggested that discontinuation may be less appropriate in higher-risk patients, whereas carefully selected and monitored patients appeared to have low absolute event rates.[30,32,59] These data are consistent with our findings and reinforce that ablation success alone is insufficient to determine long-term anticoagulation strategy.

The reduction in major bleeding was the most consistent clinical signal in this analysis. This finding is clinically plausible because long-term anticoagulation reduces thromboembolic risk but does not eliminate bleeding risk, even with contemporary DOAC therapy.[14] Stroke and bleeding risk scores remain relevant because they capture clinical risk factors that persist after rhythm control, including age, vascular disease, prior thromboembolism, renal dysfunction, and bleeding vulnerability.[12,13] The clinical implication is therefore not that OAC discontinuation is broadly safe, but that the bleeding benefit may be meaningful in patients with durable rhythm control, low residual thromboembolic risk, and elevated bleeding vulnerability.

Current guidelines remain appropriately conservative. The 2023 American College of Cardiology/American Heart Association/American College of Chest Physicians/Heart Rhythm Society (ACC/AHA/ACCP/HRS) guideline and the 2024 European Society of Cardiology (ESC) guideline recommend OAC during the early post-ablation period and advise that long-term anticoagulation should be guided by thromboembolic risk rather than the perceived success of ablation alone.[10,11] The 2024 European Heart Rhythm Association/Heart Rhythm Society/Asia Pacific Heart Rhythm Society/Latin American Heart Rhythm Society (EHRA/HRS/APHRS/LAHRS) expert consensus statement similarly emphasizes structured follow-up and individualized assessment after catheter or surgical ablation.[4] Our findings are aligned with this framework. They support individualized decision-making, but they do not justify stopping anticoagulation solely because ablation was performed or because symptoms improved.

This individualized approach is particularly important in patients with competing risks. Alternative stroke prevention strategies, including left atrial appendage closure (LAAC), are increasingly studied in populations in whom long-term anticoagulation presents significant clinical challenges. Specifically, LAAC has demonstrated safety and efficacy as an alternative for stroke prevention in patients with concurrent malignancies.[60] Furthermore, antithrombotic decisions must be integrated with the comprehensive management of coexisting cardiovascular conditions. Frailty has been associated with adverse in-hospital outcomes among patients with heart failure with reduced ejection fraction (HFrEF),[61] whereas sodium-glucose cotransporter 2 inhibitors have been associated with improved health status in patients with heart failure.[62] Although these specific findings originate from heart failure cohorts rather than post-ablation populations, they establish the critical importance of assessing overall clinical vulnerability. Therefore, post-ablation care must extend beyond rhythm monitoring to encompass holistic risk optimization according to the latest global implementation guidelines for highly prevalent comorbidities, specifically heart failure,[63] and obesity.[64] This comprehensive framework is essential because patients most likely to benefit from reduced bleeding exposure often possess concurrent metabolic and structural characteristics that elevate their residual thromboembolic risk.

The heterogeneity in rhythm monitoring intensity across the included studies carries substantial clinical implications. Surveillance strategies relying on intermittent electrocardiography inevitably fail to detect asymptomatic recurrences when compared with continuous monitoring modalities, thereby potentially compromising the reliability of a 'successful ablation' designation. It is imperative for clinicians to recognize that the decision to discontinue OAC therapy is only as robust as the monitoring strategy utilized to confirm durable sinus rhythm.[16] Furthermore, AF burden is increasingly understood as a graded clinical construct rather than a binary state, and the currently pooled data are insufficient to define the specific burden of asymptomatic recurrence at which anticoagulation must be resumed.[65] Until threshold studies definitively address this gap, clinicians are advised to adopt a conservative, risk-based approach, in which any detected recurrence prompts a rigorous clinical reassessment of OAC continuation. In addition, atrial cardiomyopathy, left atrial dysfunction, endothelial dysfunction, and systemic vascular risk may persist after apparent rhythm control.[66,67] These mechanisms provide a robust biological rationale for guideline recommendations that long-term anticoagulation decisions must remain risk-based after ablation.

Sensitivity and exploratory analyses supported a cautious interpretation of the pooled findings. Leave-one-out analyses showed that the thromboembolic estimate remained statistically non-significant and the major bleeding estimate remained statistically significant after sequential omission of individual studies, suggesting that the primary findings were not driven by a single study. In univariable meta-regression, longer follow-up was the only moderator associated with thromboembolic events, which may reflect greater event accrual with longer observation rather than a direct treatment effect.[68] No evaluated moderator significantly explained variability in major bleeding. Egger regression suggested possible small-study effects for thromboembolic events and major bleeding.[24] These findings should not be interpreted as definitive evidence of publication bias because funnel plot asymmetry may also reflect clinical heterogeneity, differences in outcome definitions, follow-up duration, or study-size related differences in patient selection.[23]

The ongoing trial landscape reflects these unresolved questions. DESTINATION (NCT06615596) is evaluating whether anticoagulation can be discontinued after successful catheter ablation in patients without recurrence during the post-ablation period.[69] DIAMOND-AF (NCT06871228) is testing a smartwatch-guided anticoagulation discontinuation strategy after successful ablation.[70] POCKET-OAC (NCT06216769) is evaluating an intermittent, pill-in-the-pocket anticoagulation strategy after AF ablation.[71] These trials may help determine whether future

management should move from a binary continuation *versus* discontinuation strategy toward monitoring-guided or phenotype-guided anticoagulation.

Strengths and limitations

This study has several strengths. It provides an updated synthesis of randomized and observational evidence evaluating OAC discontinuation after AF ablation, incorporates recent DOAC therapy studies, and evaluates clinically relevant thromboembolic, bleeding, and mortality outcomes. We also assessed RoB using design-specific tools, evaluated certainty of evidence, and performed leave-one-out sensitivity analyses.[19-21] These sensitivity analyses showed that the thromboembolic findings remained statistically non-significant and the major bleeding findings remained statistically significant after sequential omission of individual studies, supporting the stability of the main pooled estimates.

Several limitations should be considered. First, most included studies were observational and therefore vulnerable to confounding by indication, patient selection, and residual unmeasured confounding. Patients selected for OAC discontinuation may have differed from those maintained on OAC in ways not fully captured by adjustment methods. Second, definitions of successful ablation, timing of OAC discontinuation, rhythm monitoring intensity, and outcome definitions varied across studies. Third, event rates were low, particularly for ischemic stroke, systemic embolism, and mortality, limiting precision. Fourth, the certainty of evidence for thromboembolic and mortality outcomes is limited by broad confidence intervals that encompass both clinically relevant benefit and potential harm. Critically, this lack of statistical significance must not be conflated with clinical equivalence or safety. The upper boundary of the CI allows for up to a 20% relative increase in thromboembolic risk (RR 1.20), translating to a potentially significant absolute risk increase in higher-risk cohorts, particularly individuals with a CHA_2DS_2-VASc score of 4 or greater. Extrapolating these pooled estimates to patient-level care demands rigorous appraisal of baseline thromboembolic risk and explicit communication of residual uncertainty during shared decision-making paradigms. Fifth, although this meta-analysis yields robust aggregate estimates, it inherently obscures the patient-level granularity requisite for nuanced clinical decision-making. Such factors include the qualitative heterogeneity of CHA_2DS_2-VASc risk parameters; for instance, a score of 2 driven by advanced age and hypertension poses a distinct clinical profile compared to a score of 2 driven by prior vascular disease and diabetes. Furthermore, the varying intensity of post-ablation rhythm surveillance, ranging from intermittent electrocardiography to continuous implantable loop recorders, and the unquantified burden of asymptomatic AF recurrence limit generalizability. Consequently, these pooled estimates do not establish a universal threshold for OAC discontinuation, necessitating the integration of individual clinical variables to optimize patient-tailored therapy. Finally, residual heterogeneity persisted in sensitivity analyses, indicating that variability was more likely related to differences in study design, patient selection, baseline risk, rhythm surveillance, and endpoint definitions than to a single influential study.

Conclusions

In this systematic review and meta-analysis, OAC discontinuation following AF ablation was associated with a significant reduction in major bleeding, without statistically significant differences in thromboembolic events or all-cause mortality compared with OAC continuation. These findings endorse an individualized, risk-stratified approach to patient management, advising against the routine cessation of anticoagulation post-ablation.

However, several important caveats must be emphasized when translating these findings to clinical practice. First, the absence of statistical significance for thromboembolic events should not be misinterpreted as definitive evidence of safety, because the confidence interval includes up to a 20% relative increase in risk, which may be clinically meaningful for higher-risk patients. Second, aggregate meta-analytic data necessarily obscure individual-level granularity, including the intensity and duration of rhythm monitoring, the qualitative nature of CHA_2DS_2-VASc risk factors, and the burden of post-ablation AF recurrence, all of which are critical to informed decision-making. Third, the very low certainty of evidence for thromboembolic outcomes according to GRADE criteria limits confidence in the pooled estimates and underscores the need for cautious, personalized application of these data.

Clinicians should approach post-ablation OAC decisions through a framework of shared decision-making that explicitly discusses the uncertainty in the evidence, the trade-off between thromboembolic and bleeding risks, the importance of structured rhythm monitoring, and the individual values and preferences of the patient. Importantly, the decision to discontinue OAC is not permanent and should be revisited dynamically in response to ongoing rhythm monitoring and changes in the patient's clinical status. Patients at high thromboembolic risk, such as those with a CHA_2DS_2-VASc score of 4 or greater, prior stroke, or structural heart disease, should generally continue OAC, whereas discontinuation may be reasonable in selected low-risk patients with confirmed durable sinus rhythm and elevated bleeding vulnerability.

Adequately powered RCTs with standardized rhythm surveillance, prespecified risk stratification, and rigorous adjudication of both thromboembolic and bleeding endpoints are required to definitively identify the optimal candidates for safe OAC discontinuation.

Authors' contributions

All the authors read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest

The authors declare no potential conflicts of interest.

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Ethics approval and consent to participate

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Online supplementary material: