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CLINICAL RESEARCH

How much does elective cardioversion increase the risk of ischaemic stroke compared to the baseline risk in atrial fibrillation? A nationwide study

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Aims

Patients with atrial fibrillation (AF) undergoing cardioversion (CV) are exposed to increased risk of ischaemic stroke (IS), but the exact magnitude is unknown. We compared IS rates during the post-CV period with the long-term risk in AF patients using guideline-recommended anticoagulation therapy.

Methods and results

This nationwide register-based study included all AF patients undergoing first-ever elective CV between 2012 and 2018 in Finland. Breakpoint analysis identified a cut-off point in the IS rate at 2 weeks after CV. Follow-up was split into two intervals: the immediate 2-week post-CV period and the subsequent period up to 360 days. Stroke rates were calculated, and incidence rate ratios were estimated with Poisson regression. Interactions between the two follow-up periods and conventional IS risk factors as well as anticoagulation treatment were assessed. A total of 9625 patients were identified (mean age 67.7 ± 9.9 years, 61.2% men, mean CHA_2DS_2-VA score 2.2 ± 1.4). Warfarin was used in 6245 (64.9%) and non-vitamin K oral anticoagulants in 3380 (35.1%) patients. Overall, 92 (1.0%) patients experienced IS during the year after CV. Breakpoint analysis and survival plot displayed a higher incidence of IS within the first 2 weeks after CV, stabilizing thereafter to a consistent level. The adjusted IS rate during the first 2 weeks was 7.5-fold (95% confidence interval: 4.8–11.8) compared to the subsequent IS rate. This excess risk was independent of the anticoagulation type or conventional stroke risk factors.

Conclusion

The rate of IS was roughly seven times higher during the first 2 weeks after elective CV compared to the subsequent 360 days.

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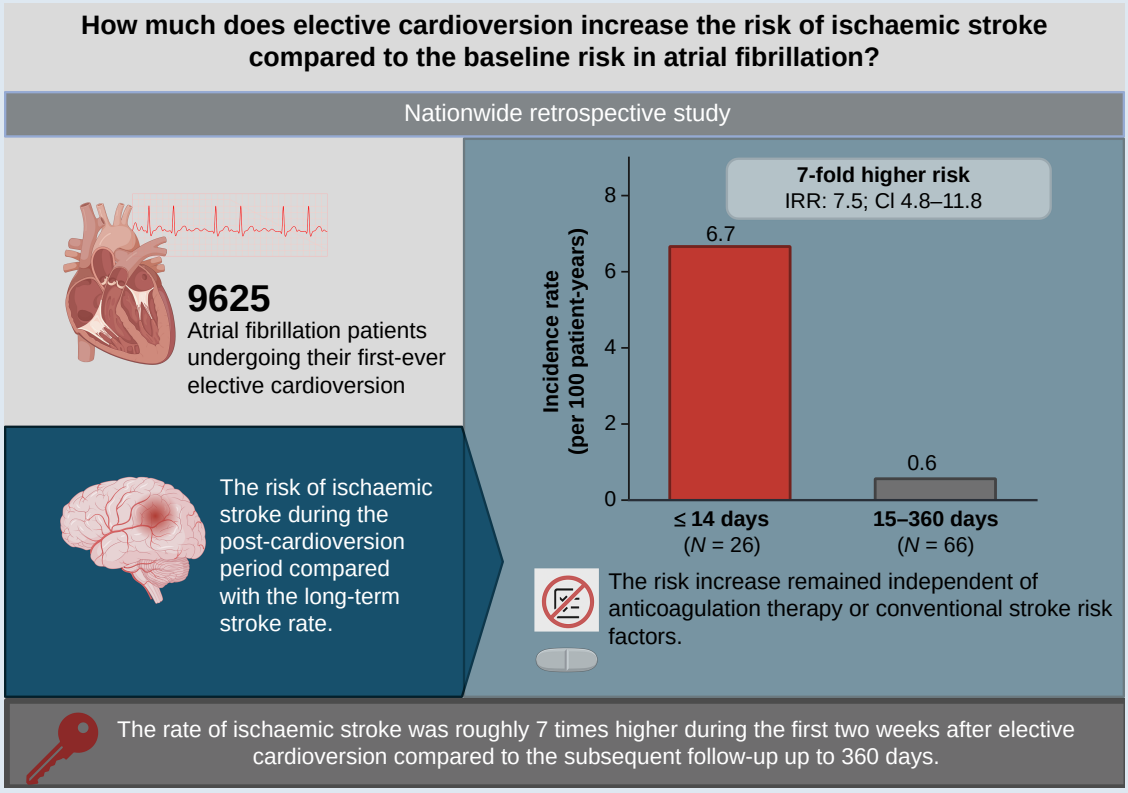
Graphical Abstract



Keywords Atrial fibrillation • Elective cardioversion • Ischaemic stroke

What's new?

- In this nationwide study including 9625 patients with atrial fibrillation (AF) undergoing their first elective cardioversion (CV) on guideline-recommended anticoagulation, the risk of ischaemic stroke was approximately seven times higher during the first 2 weeks after elective CV compared with the subsequent long-term follow-up.
- The heightened stroke risk in the early post-CV period occurred regardless of the anticoagulant type or traditional risk factors, suggesting it represents a universal phenomenon.
- This is the first study to quantify the magnitude of the increased stroke risk during the early post-CV period in relation to baseline AF risk.
- These findings underline the early post-CV period as a vulnerable window and support the need to optimize peri-procedural anticoagulation strategies and patient adherence during this time.

Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia affecting up to 5% of the adult population[1] with 4.48 million incident cases worldwide.[2] Prevention of stroke remains the cornerstone for management of AF. The AF-related risk of stroke with oral anticoagulation (OAC) is 0.1–0.2%/month in the long-term treatment.[3,4] Meanwhile, the risk of ischaemic stroke (IS) after elective cardioversion (CV) performed during therapeutic OAC has ranged from 0.3 to 0.9% in previous studies.[5–8] Therefore, patients undergoing CV are exposed to a higher risk for IS during the post-CV period compared to patients without CV. Most of the thromboembolic complications (TECs) occur within 7–10 days after elective CV.[8–11] Hence, current guidelines recommend anticoagulation up to 4 weeks after CV to reduce the risk of TEC also in patients with low thromboembolic risk without indication for long-term OAC therapy.[12,13]

The exact magnitude of IS risk related to elective CV with preceding appropriate OAC therapy is yet unknown. This study aimed to fill that gap by comparing the risk of IS following elective CV with the long-term stroke rate in AF patients using guideline-recommended OAC therapy. Additionally, we sought to identify potential factors that may increase the IS risk following CV.

Methods

Study design

This substudy is part of a larger nationwide retrospective cohort study, Finnish AntiCoagulation in Atrial Fibrillation (FinACAF), including all patients with AF diagnosis ($n = 411\,387$) in Finland from 2004 to 2018.[14] All national health care registers available were used to collect patient data, including hospitalizations and outpatient specialist visits (HILMO), primary health care visits (AvoHILMO), drug prescriptions from the National Reimbursement Register maintained by the Social Insurance Institution (KELA), and the National Causes of Death Register. The inclusion criteria were International Classification of Diseases 10th revision (ICD-10) diagnosis code I48, including both AF and atrial flutter, in any of the registers.

For the current study, we included a cohort of patients undergoing their first-ever elective CV and using OAC therapy at least 3 weeks prior to elective CV. The inclusion criteria were new-onset AF, initiation of OAC therapy, and first-ever elective CV earliest 21 days from the AF diagnosis between 2012 and 2018. Our study begins in 2012 to ensure the identification of AF patients diagnosed outside hospital care registers, given the primary health care register was introduced in Finland in 2011. The cohort selection process is summarized in *Figure 1*.

The hospital diagnoses were registered according to the ICD-10 and primary health care diagnoses according to the International Classification of Primary Care (ICPC-2). Oral anticoagulation (OAC) treatment was defined as any prescribed non-vitamin K oral anticoagulant (NOAC) or warfarin regimen between the time of cohort entry and index elective CV. The reduced drug dose was defined according to the manufacturer's recommended reduced-dose regimens for each NOAC as follows: one pill per day for rivaroxaban 15 mg and edoxaban 30 mg and two pills per day for apixaban 2.5 mg and dabigatran 110 mg. The CHA_2DS_2-VA [congestive heart failure, hypertension, age 65–74, diabetes, vascular disease give one point each; age >75, previous stroke/transient ischaemic attack (TIA) give 2 points] was used to stratify the risk of stroke in AF patients. The procedure code identified for CV was TFP20, according to the Nordic Classification of Surgical Procedures. All elective CVs were performed electrically. Elective CV was performed only after confirming, in strict accordance with clinical protocols, that warfarin-treated patients have maintained international normalized ratio (INR) values within the therapeutic range (2.0–3.0) for at least three consecutive weeks before the procedure, ensuring near-complete therapeutic control during the pre-procedural period.

Endpoints

First, we identified the cut-off point between the time period of the increased stroke rate post-CV and the stabilized baseline level by using breakpoint analysis. Hence, the follow-up time was split into two intervals, the 2-week post-CV period and the subsequent period up to 360 days. Stroke rates were calculated separately for these periods, and Poisson regression was used to estimate unadjusted and adjusted incidence rate ratios (IRRs). Moreover, interaction between the two follow-up periods and conventional IS risk factors as well as OAC treatment was assessed. An IS was defined as ICD-10 code I63, I64, or I69.3-I69.8. The outcome analysis included both new and recurrent IS events. A recurrent stroke was defined as an endpoint event if the new or recurrent stroke was the primary diagnosis during hospitalization in a patient with a history of IS. The time to complication was calculated by subtracting the date of the IS from the date of the first elective CV. The follow-up was terminated if a patient underwent a recurrent CV, experienced an IS, died, or until 360 days from the index elective CV, or until the end of the study period 31 December 2018, which ever came first. All definitions for baseline comorbidities and outcome events used for the analyses are listed in Supplementary material online, *Table S1*.

Falsification endpoint

We performed a similar visual assessment of the survival curve and a breakpoint analysis for gastrointestinal bleeding to assess whether this endpoint, which lacks a plausible biological reason to increase after elective CV, behaves similarly to IS.

Statistical analysis

Normally distributed continuous data were reported as mean [± standard deviation (SD)] and skewed continuous data as median [interquartile range (IQR)]. A survival plot was used to illustrate the trend of survival outcome over the study period. The risk of IS was estimated by calculating incidence rates and IRRs with Poisson regression and results reported with the 95% confidence interval (CI). Adjusted IRRs were calculated in a model, which included age (three categories: under 65, from 65 to 74, and 75 or more), sex, comorbidities (hypertension, heart failure, diabetes, prior IS or TIA, vascular disease, chronic kidney disease, and alcohol use disorder), and OAC type (NOAC or warfarin). Thereafter, we fitted the adjusted model with an interaction term between the split follow-up time (the 2-week post-CV period vs. the subsequent period) and the other covariates separately to assess whether their impact on IS risk differs during the post-CV period compared to the subsequent time period. The risk factors selected for analysis as predictors were based on prior knowledge about their relationship with the outcomes.[8] Statistical analyses were performed with the IBM SPSS Statistics software, version 28.0 (IBM Corp.), and R, version 4.0.5 (R Core Team; https://www.R-project.org). Breakpoint analysis was conducted using the *segmented* R package.

Ethical considerations

The study protocol was approved by the Ethics Committee of the Medical Faculty of Helsinki University, Helsinki, Finland (15/2017) and granted research permission from the Helsinki University Hospital (HUS/46/2018 and updated as HUS/60/2019). Respective permissions were obtained from KELA (138/522/2018), Finnish Institute for Health and Welfare (THL) (THL/2101/5.05.00/2018), Population Register Center (VRK/1291/2019-3), Statistics Finland (TK-53-1713-18/u1281), the and Tax Register (VH/874/07.01.03/2019). Informed consent was not required due to the register-based nature of the study. All patient data were pseudonymized, ensuring full data protection of the patients according to the European General Data Protection Regulation (EGDPR).

Results

We identified 9625 patients [61.2% men; mean age 67.7 (SD ± 9.9) years; mean CHA_2DS_2-VA score 2.2 (SD ± 1.41)] with new-onset AF and undergoing their first elective CV. The baseline characteristics and the risk of IS defined by CHA_2DS_2-VA score are presented in *Table 1*. Warfarin was used in 6245 (64.9%) and NOAC in 3380 (35.1%) patients. Among all patients treated with a NOAC, 237 (7.0%) received a reduced dose.

Outcomes

Overall, 92 (1.0%) patients suffered an IS during the year after elective CV. The breakpoint was estimated at 14.6 (±3.6) days after elective CV. Also in the survival plot, a higher incidence of IS within the first 2 weeks after CV is displayed, stabilizing thereafter to a consistent level (*Figures 2* and *3*). Of these ISs, 78 (84.8%) were first-ever events, whereas 14 (15.2%) were recurrent. In warfarin-treated patients who experienced IS, the mean INR value at the time of the event was 2.3 (±0.9). Four (4.3%) of the patients who experienced an IS were receiving a reduced NOAC dose. Twenty-seven (0.3%) deaths occurred during the 360-day follow-up, out of which two were during the first 2-week period.

During the initial 14 days following elective CV, the incidence rate of IS was 6.7 per 100 patient-years ($n = 26$), as compared with 0.9 per 100 patient-years ($n = 66$) during the subsequent period. In the unadjusted regression analyses, the risk of stroke within the first 2 weeks after elective CV was roughly seven times higher (IRR 7.4; CI 4.7–11.6) compared to the subsequent stroke risk (*Table 2*). After adjusting for the conventional risk factors, the risk of stroke was similar (adjusted IRR 7.5; CI 4.8–11.8).

The significant independent predictors of IS within 1 year of elective CV were heart failure (IRR 1.72; 95% CI 1.03–2.87) and previous IS/TIA (IRR 2.53; 95% CI 1.42–4.51) (*Table 3*).

We observed no significant interaction between any of the conventional IS risk factors or OAC treatment and the two follow-up time periods, indicating that their association with the IS rate did not differ during the post-CV period compared to the subsequent time (see Supplementary material online, *Table S2*).

There were 63 (0.7%) gastrointestinal bleeding events during the 1-year follow-up after elective CV. The survival plot curve for gastrointestinal bleeding was consistent and displayed no noticeable changes in bleeding incidence over time (see Supplementary material online, *Figure S1*). Furthermore, no meaningful breakpoint was identified for gastrointestinal bleeding, unlike for IS. The breakpoint analysis calculated a breakpoint at 341 days, but this is unlikely to be clinically related to elective CV.

Discussion

We aimed to assess the magnitude of stroke risk related to elective CV compared to the long-term stroke risk associated with AF. The main finding of our study is that the increase in stroke risk is confined to the first 2

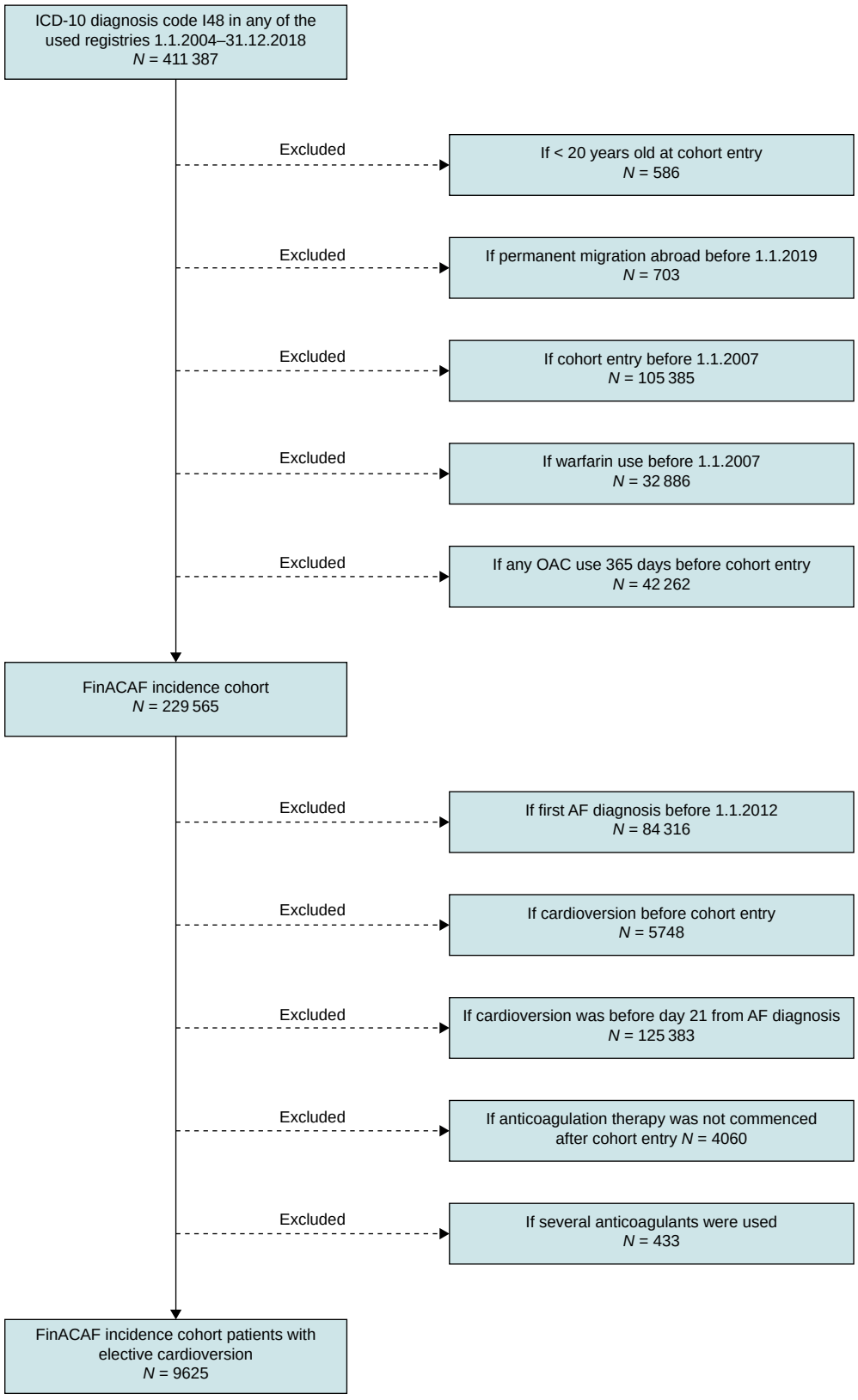


Figure 1 Flow chart of the patient selection process. Created with Biorender.com

Table 1 Baseline characteristics of the cohort undergoing elective CV

Variables	All patients $n = 9625$	Ischaemic stroke ≤ 14 days $n = 26$	Ischaemic stroke 15–360 days $n = 66$	*P*-value
Age (median, IQR)	68.4 years (62–75)	72.3 years (63–82)	69.9 years (63–76)	0.140
Female sex	3733 (38.8%)	13 (50.0%)	30 (45.5%)	0.694
CHA_2DS_2-VA score (mean ± SD)	2.24 (±1.41)	2.23 (±1.37)	2.73 (±1.64)	0.540
HAS-BLED score (mean ± SD)	1.84 (±1.0)	1.81 (±1.2)	2.14 (±1.0)	0.468
Comorbidity				
Congestive heart failure	1192 (12.4%)	4 (15.4%)	15 (22.7%)	0.433
Hypertension	6870 (71.4%)	15 (57.7%)	45 (68.2%)	0.342
Diabetes mellitus	2012 (20.9%)	3 (11.5%)	17 (25.8%)	0.137
Hyperlipidaemia	4472 (46.5%)	11 (42.3%)	36 (54.5%)	0.290
Previous stroke or TIA	670 (7.0%)	3 (11.5%)	11 (16.7%)	0.537
Vascular disease	1721 (17.9%)	2 (7.7%)	16 (24.2%)	0.072
Chronic kidney disease	190 (2.0%)	0 (0%)	2 (3.0%)	0.369
Abnormal liver function	26 (0.3%)	0 (0%)	0 (0%)	
Coronary heart disease	1336 (13.9%)	1 (3.8%)	15 (22.7%)	0.031
Previous myocardial infarction	432 (4.5%)	0 (0%)	4 (6.1%)	0.199
Previous pulmonary embolism	67 (0.7%)	0 (0%)	0 (0%)	
Previous venous thromboembolism	486 (5.0%)	0 (0%)	2 (3.0%)	0.369
Alcohol overuse	337 (3.5%)	1 (3.8%)	5 (7.6%)	0.514
Thyrotoxicosis	106 (1.1%)	0 (0%)	0 (0%)	
Cancer	1425 (14.8%)	6 (23.1%)	19 (28.8%)	0.579
Dementia	41 (0.4%)	0 (0%)	1 (1.5%)	0.528
Mental health disorder	1146 (11.9%)	4 (15.4%)	15 (22.7%)	0.433

CHA_2DS_2-VA, Congestive heart failure, Hypertension, Age 65–74, Diabetes, Vascular disease give one point each; age >75, previous stroke/TIA give 2 points; HAS-BLED, Hypertension, Abnormal renal and liver function, Stroke, Bleeding, Elderly, Drugs or alcohol (labile INR not calculated); CV, cardioversion; IQR, interquartile range; SD, standard deviation; TIA, transient ischaemic attack .

weeks after CV. During this period, the risk is approximately seven-fold higher compared with long-term stable stroke risk of the same patients, and the conventional stroke risk factors do not modify the relative risk increase. While the absolute early post-CV IS risk of 0.4% may appear low, our study provides a crucial perspective by demonstrating how substantial this seven-fold increased risk is in relation to the baseline AF risk. This risk increase has not been quantified previously.

According to previous studies, up to 90–98% of thromboembolic (TE) events occur within 10 days of elective CV.[9,10,15] This is in line with our study, where we observed a higher stroke risk of <14 days post-CV. The rate of IS was roughly seven times higher during the first 2 weeks after elective CV compared to the subsequent follow-up time in AF patients using guideline-recommended OAC treatment. Several factors contribute to this transient elevated stroke risk after elective CV, including destabilizing of pre-existing thrombi, transient atrial stunning after CV, or changes in atrial systolic function and size that may trigger thrombus formation.[16] Therefore, the initial period after elective CV remains a vulnerable window, despite a guideline-recommended anticoagulation therapy, which underscores the importance of re-evaluation of how stroke prevention strategies are implemented immediately following elective CV. For example, more aggressive anticoagulation may be considered in the early post-CV period to mitigate the elevated stroke risk, particularly for patients with high baseline thromboembolic risk.

Previous studies have suggested that maintaining a higher intensity of peri-procedural anticoagulation can significantly reduce the risk of stroke. For instance, several studies have shown that patients with a high INR (≥2.5) had a lower risk of TEC after elective CV than those with a lower INR.[8,17,18] Additionally, the EMANATE trial reported no TECs in patients receiving a loading dose of apixaban pre-CV.[19] Notably, a short-term increase in anticoagulation intensity does not appear to significantly increase the risk of bleeding, as trials investigating anticoagulation for AF[2] and pulmonary embolism with higher doses of NOACs have shown similar bleeding rates of 0.1–0.3% per month.[20] These findings suggest that intensified anticoagulation during the post-CV period could offer a favourable balance between efficacy and safety. However, confirming this hypothesis through a randomized trial would be challenging due to the large sample size required.

One of the strengths of our study is its longitudinal, patient-specific approach, which highlights that the relative increase in stroke risk after elective CV is independent of conventional risk factors or anticoagulant choices. Instead, the increase in stroke risk during the early days post-CV seems to be a universal phenomenon, regardless of the baseline stroke risk or anticoagulant regimen. In other words, the risk of IS during the first 2 weeks remains approximately seven times higher than in the subsequent period, and importantly, this risk ratio is not reduced by using NOACs instead of warfarin. Our previous study, which compared TECs 30 days post-CV in AF patients undergoing their first elective CV, found no significant difference between NOAC and warfarin groups, which supports our current results that the choice of OAC does not influence stroke risk.[8] These findings emphasize the need for individualized risk stratification and more tailored approaches in managing AF patients undergoing CV.

In our study, female sex was not associated with an increased risk of IS during the 1-year follow-up period. This finding is consistent with

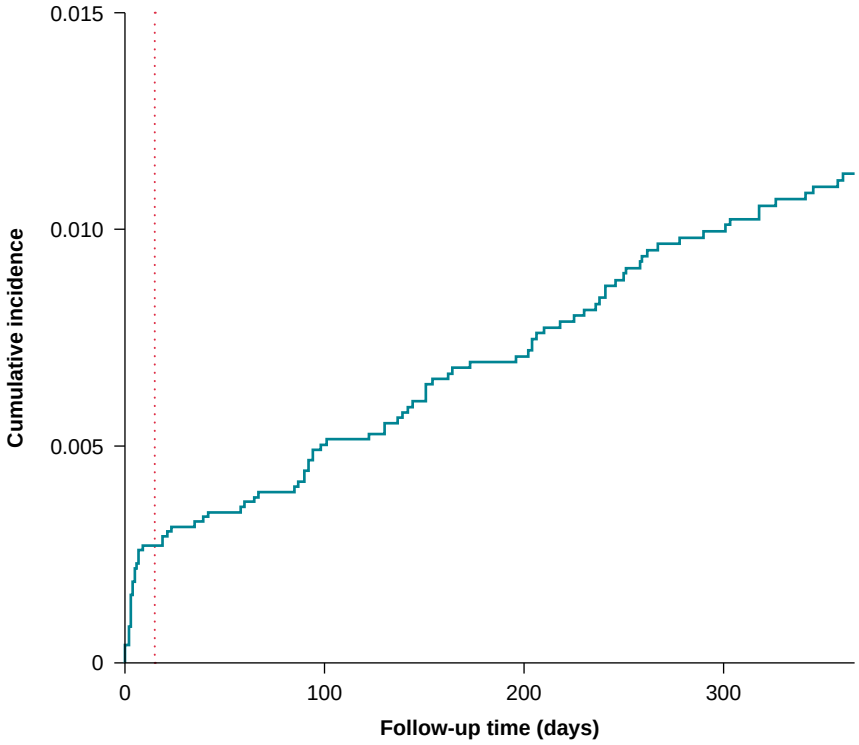


Figure 2 Cumulative risk of IS after elective CV with the 2-week cut-off point (broken line).

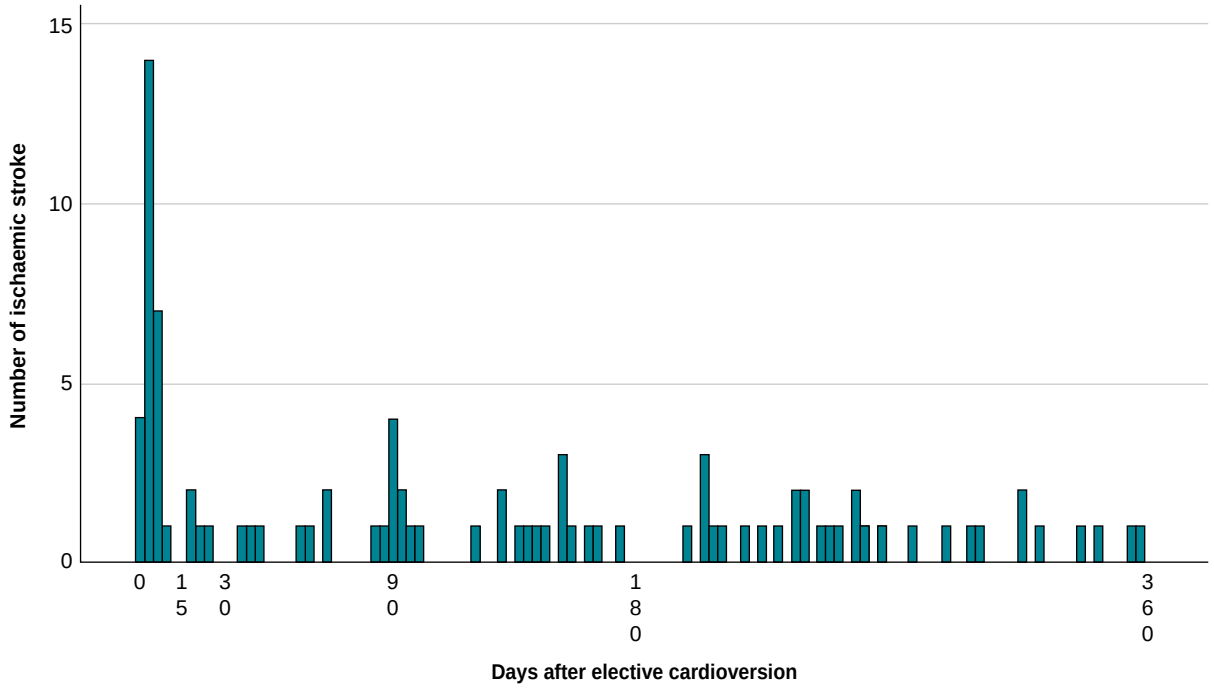


Figure 3 The time distribution of IS during 360 days after elective CV.

recent Finnish and international data suggesting that the previously reported excess stroke risk among women with AF has attenuated in contemporary populations. Teppo *et al.*[21] recently demonstrated that the association between female sex and IS rate has decreased and become non-significant among Finnish AF patients. Similarly, Yoshimura *et al.*[22] observed no sex differences in TE risk among lower-

Table 2 Incidence rates of IS according to the time after elective CV (2012–18)

Days after elective CV	P-years (100 years)	Events, *n*	Incidence (per 100 P-years)	Unadjusted IRR (95% CI)	Adjusted IRR[a] (95% CI)
≤14 days	3.9	26	6.7 (95% CI 4.4–9.8)	7.4 (4.7–11.6)	7.5 (4.8–11.8)
15–360 days	73.1	66	0.9 (95% CI 0.7–1.1)	1.0 (reference)	1.0 (reference)

CI, confidence interval; CV, cardioversion; IRR, incidence rate ratio; IS, ischaemic stroke; P-year, patient-year.
[a]Adjusted for anticoagulation treatment, sex, age, hypertension, heart failure, diabetes, previous IS/TIA, vascular disease, chronic kidney disease, and alcohol use disorder.

Table 3 Variables included in the Poisson regression model for predictors of IS

Factor	IRR	95% CI
NOAC vs. warfarin	0.81	0.51–1.29
Female sex	1.41	0.92–2.18
Age 65–75	0.92	0.55–1.54
Age 75 or more	1.36	0.78–2.37
Hypertension	0.63	0.40–1.01
Heart failure	1.72	1.03–2.87
Diabetes	1.17	0.69–1.96
Previous IS/TIA	2.53	1.42–4.51
Vascular disease	1.03	0.61–1.75
Abnormal renal function	1.03	0.25–4.27
Alcohol abuse	2.28	0.98–5.29

CI, confidence interval; IRR, incidence rate ratio; IS, ischaemic stroke; NOAC, non-vitamin K oral anticoagulant; TIA, transient ischaemic attack.

risk patients and questioned the role of female sex as a separate component of the CHA_2DS_2-VASc score. Our results therefore support the growing consensus that female sex alone should not be considered an independent factor of increased stroke risk after elective CV when guideline-recommended anticoagulation is used.

Rhythm conversion may give patients a false impression of treatment success, leading to interruptions in anticoagulation during this most vulnerable high-risk period. Earlier studies have shown that compliance problems and subtherapeutic OAC intensity after CV are relatively common phenomena.[10,23] Therefore, it is crucial for clinicians to emphasize the importance of continuing anticoagulation for stroke prevention, particularly in the early post-CV phase. In our cohort, however, the mean INR among warfarin-treated patients who experienced an IS was within the therapeutic range, and the proportion of patients receiving a reduced NOAC dose was not higher among those with stroke compared to the overall NOAC-treated population. These findings suggest that, although suboptimal adherence or inappropriate dosing cannot be completely excluded, insufficient anticoagulation intensity was unlikely to explain the excess stroke risk observed after CV. Taken together, the elevated stroke risk in this early period should be taken into consideration when planning rhythm control strategies for especially high-risk patients and determining how many CVs are performed.

In our analysis, we included gastrointestinal bleeding as a falsification endpoint to assess whether an unrelated endpoint would exhibit a similar pattern to IS. The analysis revealed no meaningful breakpoint for gastrointestinal bleeding, unlike for IS. This finding supports the validity of our analysis and suggests that the observed plausible temporal association between elective CV and increased IS risk may represent causality.

This study also has some limitations. Given the nature of retrospective design, this study is subject to inaccurate data and potential misclassification of diagnoses and procedures. Additionally, given that ICD-10, ICPC-2, and TFP20 codes were used to identify diagnoses and procedures, some variables may lack clinical accuracy due to human data entry errors. However, the Finnish register for hospitalizations of IS as an endpoint has a long history of high quality and is well validated.[24] Moreover, information on pre-CV transoesophageal echocardiography were not available in our registries, and therefore, its potential association with IS could not be evaluated. Also, information on stroke severity (i.e. whether the stroke was disabling or non-disabling) and stroke-related causes of death was not available, and thus, differentiation between fatal and non-fatal or disabling and non-disabling strokes was not possible. Furthermore, patient adherence to NOAC therapy was assessed based on purchase records, which may not fully reflect actual usage, as patients might not have taken the prescribed medication as intended. Nevertheless, this study represents a real-life setting and reflects the usual behaviour and care of patients undergoing elective CV. Finally, our study focused solely on IS, without evaluating other TECs. However, it is assumed that other TECs follow a similar pattern.

In conclusion, our study found a seven times higher risk of IS during the first 2 weeks following elective CV compared to the baseline risk in AF patients. This risk increase remained independent of anticoagulation therapy or traditional stroke risk factors. While guideline-recommended peri-procedural anticoagulation therapy is essential, these findings suggest a need for additional strategies for stroke prevention, for instance, higher intensity anticoagulation treatment, during this high-risk period.

Supplementary material

Supplementary material is available at *Europace* online.

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Data availability

The data underlying this article cannot be shared publicly due to its sensitive nature and in accordance with the agreements made with the Finnish registries. Requests to access the dataset from qualified researchers trained in human subject confidentiality protocols may be sent to the Finnish national register holders (KELA, Finnish Institute for Health and Welfare, Population Register Centre, and Tax Register).

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