High-power short-duration setting prevents changes of periprocedural thrombotic markers and the onset of silent stroke in patients with atrial fibrillation

Masashi Kamioka, MD, Tomonori Watanabe, MD, Hiroaki Watanabe, MD, Takafumi Okuyama, MD, Ayako Yokota, MD, Takahiro Komori, MD, Tomoyuki Kabutoya, MD, Yasushi Imai, MD, Kazuomi Kario, MD, PhD

From the Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical University School of Medicine, Shimotsuke, Japan.

BACKGROUND It remains unclear whether the newly adopted high-power, short-duration (HP-SD) setting in ablation for atrial fibrillation (AF) impacts periprocedural thrombotic markers or silent stroke (SS) onset.

OBJECTIVE The aim of the present study was to investigate the clinical impact of HP-SD setting ablation on changes in periprocedural thrombotic markers and the onset of SS.

METHODS We enrolled 101 AF patients: the HP-SD group (n = 67) using 50 W and the conventional ablation group (n = 34) using 30 to 40 W. D-dimer, thrombin-antithrombin complex (TAT), and total plasminogen activator inhibitor-1 (tPAI-1) were analyzed the day before, immediately after, and 1 day after the procedure. Magnetic resonance imaging was performed within 48 hours after the procedure.

RESULTS Left atrial dwelling time was significantly shorter in the HP-SD group ($P < .05$). In the conventional ablation group, the D-dimer and tPAI-1 levels continued to increase until 1 day postprocedure, while the TAT peaked immediately after the ablation. On the other hand, the range of the variation of these thrombotic markers in the HP-SD group was smaller. SS occurred more frequently in the conventional ablation group than in the HP-SD group (26% vs 5%, $P < .05$). In the logistic regression analysis, the HP-SD setting and TAT difference (postprocedure – preprocedure) were independent predictors for SS (odds ratios 0.141 and 5.838, respectively; $P < .05$).

CONCLUSIONS The HP-SD setting led to a shorter left atrial dwelling time and reduced change in thrombotic markers, resulting in lower prevalence of SS.

KEYWORDS Atrial fibrillation; Catheter ablation; Pulmonary vein isolation; Silent stroke; thrombosis

(Heart Rhythm O^2 2024;5:917–924) © 2024 Heart Rhythm Society. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Introduction

Radiofrequency catheter ablation has been established as a first-line therapy for patients with atrial fibrillation (AF).[1] However, a certain number of patients experience recurrence of atrial tachyarrhythmia after ablation, most of which is due to pulmonary vein (PV) reconduction.[2–5] Therefore, high-power, short-duration (HP-SD) ablation has been introduced as an ablation method for more efficient creation of transmural lesions and more durable pulmonary vein isolation (PVI).[6] HP-SD ablation has been reported to have 2 advantages. One is that it can create a shallow and wide ablation lesion that is deep enough to create a transmural lesion in the atrial muscle; the other is that it can reduce the risk of thermal injury to surrounding organs due to its short-term duration.[7] In fact, prolonged procedure time is a well-known risk factor for perioperative thromboembolism, and HP-SD ablation has been reported to reduce the procedure time as well as left atrial (LA) dwelling time compared with conventional ablation methods.[8] However, data are scarce regarding the HP-SD ablation setting's impact on the occurrence of perioperative thromboembolic events compared with conventional ablation methods. In the present study, we investigated the hypothesis that HP-SD ablation might reduce the risk of perioperative thromboembolism. To assess the validity of this hypothesis, we examined alterations in perioperative thrombotic markers and the incidence of postoperative silent strokes (SSs).

Methods

Study subjects

This was a single-center prospective observational study of 101 patients with symptomatic AF who were referred for first-time ablation to Jichi Medical University hospital from

Address reprint requests and correspondence: Dr Masashi Kamioka, Division of Cardiovascular Medicine, Jichi Medical University, 3311-1 Yakushiji, Shimotsuke-city, Tochigi 329-0498, Japan. E-mail address: kmasashi@jichi.ac.jp.

2666-5018/© 2024 Heart Rhythm Society. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

https://doi.org/10.1016/j.hroo.2024.09.019

KEY FINDINGS

- The ablation procedure using the high-power, short-duration (HP-SD) setting was associated with shorter left atrial dwelling time and radiofrequency application time, and with a better ablation outcome, compared with conventional ablation.
- The incidence of silent stroke was significantly higher in the conventional group than in the HP-SD group.
- Significant periprocedural changes of thrombotic markers such as change in thrombin-antithrombin complex from immediately after the ablation to pre-procedure >3.3, the HP-SD setting, and the left atrial dwelling time were proven to be independent markers of silent stroke occurrence.

December 2020 to 2022 (Figure 1). Patients who underwent cryoballoon ablation or had contraindications for cardiac magnetic resonance imaging (MRI) were excluded from the study. Only patients who consented to undergo head MRI imaging were included as participants in this study. The study subjects were assigned to 2 different ablation methods based on the physician's discretion: the HP-SD group (n = 67), comprising patients who underwent AF ablation with the HP-SD setting, and the conventional group (n = 34), on whom the conventional settings were used. All patients included in this study were symptomatic and refractory to at least 1 antiarrhythmic drug before PVI. Structural heart disease was diagnosed by echocardiography and coronary angiography or by coronary artery imaging with computed tomography. All patients provided written informed consent prior to the procedure. The present study was approved by the local ethics committee of the Jichi Medical University, Tochigi, Japan (21-067). The data that support the findings of this study are available from the corresponding author upon reasonable request. The investigation conforms with the principles outlined in the Declaration of Helsinki, and reporting of the study conforms to STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) and the broader EQUATOR (Enhancing the Quality and Transparency of Health Research) guidelines.

Blood sampling and the analysis

Levels of D-dimer (DD), thrombin-antithrombin complex (TAT), and total plasminogen activator inihibitor-1 (tPAI-1) were taken and measured the day before, immediately after, and 1 day after the procedure.

Electrophysiological study and ablation procedure

Details of the ablation procedure have been described.[2,9] Briefly, antiarrhythmic drugs were withheld for at least 5 half-lives and the left atria were checked for pre-existing thrombi by transesophageal echocardiography or computed tomography. Ablation was performed under mild sedation with intravenous dexmedetomidine and fentanyl. Twelve-lead surface electrocardiograms and intracardiac electrocardiograms were recorded simultaneously by a digital multichannel system (RMC-5000; Nihon-Kohden), filtered at 30 to 400 Hz for bipolar and 0.05 to 400 Hz for unipolar electrograms. Following transseptal puncture, high density 3-dimensional electroanatomic mapping of the LA (>1000 points) was performed using a CARTO 3 mapping system (Biosense Webster). Circumferential PVI was performed in the power-controlled mode using a contact force–sensing radiofrequency catheter (THERMOCOOL ST-SF; Biosense Webster). In the HP-SD group, radiofrequency energy was delivered at 50 W with a target ablation index of 400 along the posterior wall and 500 in the anterior wall and with a temperature limit of 45 °C, with interlesion distance within 6 mm and with a contact force of 10 to 40 g, according to the modified CLOSE protocol optimized for Asians.[10] In the conventional group, ablation was performed using 40 W in the anterior wall and 30 W in the posterior wall. Other settings were the same as in the HP-SD group. All patients were administered 1 of 4 commercially available direct oral anticoagulants at least 1 month prior to the ablation, with a single preoperative discontinuation of the anticoagulant scheduled immediately before ablation (Figure 2). Specifically, dabigatran or apixaban was interrupted only in the morning or afternoon depending on the time of procedure. Ribaroxaban or edoxaban was interrupted in the morning and was taken 2 hours after the procedure. During the procedure, unfractionated heparin was administered to maintain an activated clotting time (ACT) of over 300 seconds. After successful PVI, intravenous isoproterenol was administered to provoke the recovered PV conduction. If PV was still isolated and non-PV firing was not detected, 40 mg adenosine was administered to check for dormant conduction and the provocation of non-PV foci firing. If no AF induction was confirmed by coronary sinus burst pacing of up to 200 ms, the procedure was deemed completed. In the case of AF induction, additional procedures for substrate modification, such as linear ablation, complex fractionated atrial electrogram ablation, or modification of a low-voltage area (a bipolar voltage <0.5 mV) and/or dense scar (the absence of local voltage or a bipolar voltage <0.1 mV without capture) were performed based on the discretion of the operators.

MRI analysis

MRI was performed within 48 hours after the procedure on a 3.0T scanner (Skyra; Siemens Healthineers). The following MRI sequences were used: T1-weighted spin echo sequence to rule out acute cerebral hemorrhage, diffusion-weighted imaging and apparent diffusion coefficient mapping to assess acute cerebral infarction, and a fluid-attenuated inversion recovery sequence to differentiate acute from chronic ischemic brain lesions. An acute SS after the ablation was defined as asymptomatic, with hyperintensity regions detected on diffusion-weighted imaging and no pathological changes

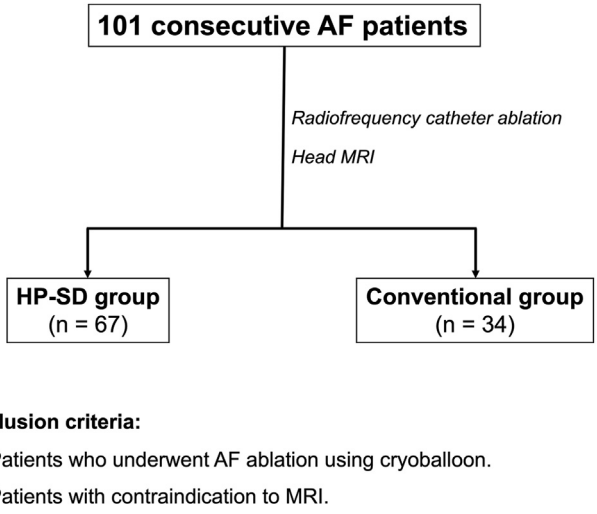

Figure 1 Patient flow of the study. AF = atrial fibrillation; HP-SD = high power, short duration; MRI = magnetic resonance imaging.

observed on fluid-attenuated inversion recovery. All the MRI analyses were performed by an independent radiologist blinded to the assigned patient group.

Statistical analysis

Data comparison between the 2 groups was performed using a 2-tailed paired Student's *t* test, and the results are presented as mean ± SD. A chi-square test was used to compare dichotomous data, and the results are presented as number and percentage. Receiver-operating characteristic (ROC) analysis was performed to calculate the sensitivity, specificity, area under the ROC curve, and the optimal cutoff with a 95% confidence interval. Binominal logistic regression analysis was performed to elucidate the predictors of SS. All analyses were performed with SPSS for Windows, version 26.0 (IBM). All statistical tests were 2-sided. A P value of $<.05$ was considered statistically significant.

Results

Patient characteristics

The baseline characteristics of the conventional and the HP-SD patients are compared in Table 1. There was no significant difference in the mean age, male sex, body mass index, AF class, and presence of comorbidities such as hypertension, diabetes mellitus, dyslipidemia, and prior history of cerebral infarction and heart failure. In addition, no statistical difference was detected regarding echocardiographic data (left ventricular ejection fraction and LA volume index) and blood analysis (N-terminal pro–B-type natriuretic

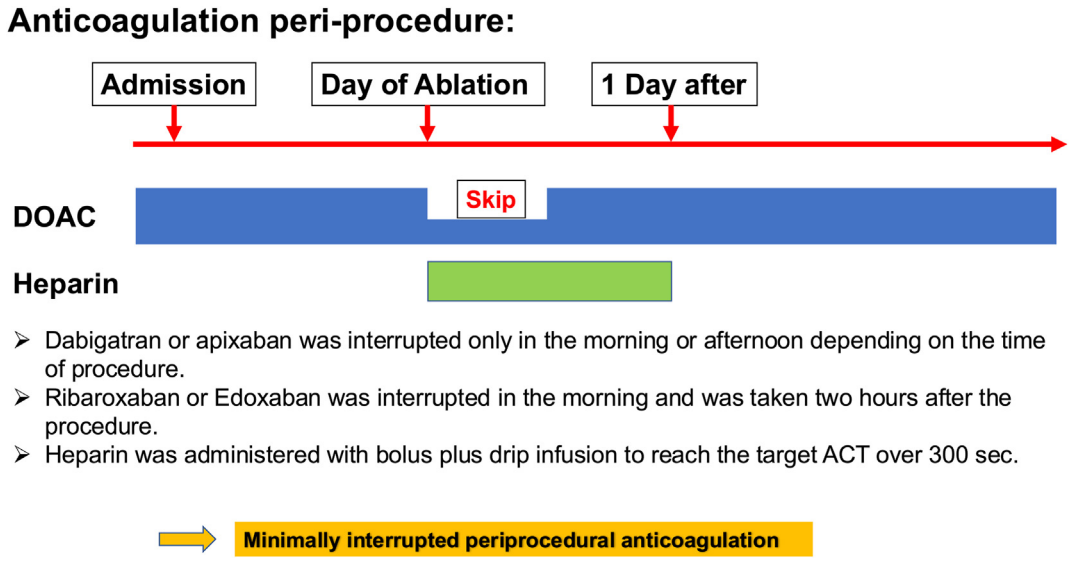

Figure 2 Anticoagulation protocol of the present study. ACT = activated clotting time; DOAC = direct oral anticoagulant.

Table 1 Patient characteristics of the 2 groups.

	Conventional group (n = 34)	HP-SD group (n = 67)	*P* value
Age, y	67 ± 7	64 ± 10	.071
Male	23 (68)	44 (66)	.845
BMI, kg/m^2	24.2 ± 2.9	25.0 ± 3.9	.277
Paroxysmal AF	21 (62)	42 (63)	.929
HTN	20 (59)	44 (66)	.505
DM	9 (26)	14 (21)	.533
DLp	15 (44)	35 (52)	.447
Prior cerebral infarction	2 (6)	3 (4)	.533
CHF	4 (12)	9 (13)	.533
NT-proBNP, pg/mL	515 ± 783	572 ± 475	.700
eGFR, mL/min/1.73 m^2	67 ± 17	64 ± 15	.363
LAVI, mL/m^2	39 ± 18	37 ± 21	.663
LVEF, %	65 ± 7	62 ± 10	.106
CHA_2DS_2-VASc score	2.0 ± 1.3	2.1 ± 1.6	.781

Values mean ± SD or n (%).

AF = atrial fibrillation; BMI = body mass index; CHF = congestive heart failure; CHA_2DS_2-VASc = congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, prior stroke or transient ischemic attack or thromboembolism, vascular disease, age 65–74 years, sex category; DLp = dyslipidemia; DM = diabetes mellitus; eGFR = estimated glomerular filtration rate; HTN = hypertension; HP-SD = high power, short duration; LAVI = left atrial volume index; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro–B-type natriuretic peptide.

peptide and estimated glomerular filtration rate) between the 2 groups.

Ablation outcome and periprocedural parameters

Kaplan-Meier curve analysis revealed that the recurrence rate in the HP-SD group was significantly lower than that in the conventional treatment group as shown in Figure 3. The clinical parameters regarding the ablation procedure are summarized in Table 2 and Supplemental Table 1. In the HP-SD group, the LA dwelling time and RF application time were significantly shorter than those in the conventional group (59 ± 24 minutes vs 70 ± 26 minutes, $P = .046$; and 12 ± 6 minutes vs 20 ± 10 minutes, $P < .001$, respectively).

Table 2 Periprocedural parameters of the 2 groups.

	Conventional group (n = 34)	HP-SD group (n = 67)	*P* value
Procedure time, min	178 ± 56	149 ± 56	.009
LA dwelling time, min	70 ± 26	59 ± 24	.046
RF application time, min	20 ± 10	12 ± 6	<.001
First-pass PVI			
RPV	22 (65)	59 (88)	.005
LPV	23 (68)	61 (91)	.003
Ablation procedure			
PVI only	26 (76)	49 (73)	.720
CTI	5 (15)	11 (16)	.826
Roof line	3 (9)	3 (6)	.598
Posterior wall isolation	2 (6)	6 (9)	.593
SVCI	3 (9)	4 (7)	.813
Substrate modification	1 (3)	2 (3)	.990
Cardioversion	3 (9)	7 (10)	.799
ACT, s	284 ± 19	283 ± 12	.719
Complication	0 (0)	2 (3)	.214

ACT = activated clotting time; CTI = cavotricuspid isthmus; HP-SD = high power, short duration; LA = left atrial; LPV = left pulmonary vein; PVI = pulmonary vein isolation; RF = radiofrequency; RPV = right pulmonary vein; SVCI = superior vena cava isolation.

The mean duration of ablation per lesion was significantly shorter in the HP-SD group than in the conventional group, but there were no significant differences in contact force, ablation index, Δimpedance drop, or interlesion distance. First-pass isolation of bilateral PV was more frequently achieved in the HP-SD group than the conventional group (right PV: 88% vs 65%, $P = .005$; left PV: 91% vs 68%, $P = .03$). As for the ablation methods, only PVI was performed in over 70% of study patients, and there was no significant difference in the requirement for additional ablations between the 2 groups. During the procedure, cardioversion was performed in about 10% of patients in each group. The mean ACT level was kept as the same. With regard to the complications, 1 patient showed gastric hypomotility and another showed a groin hematoma not requiring blood transfusion.

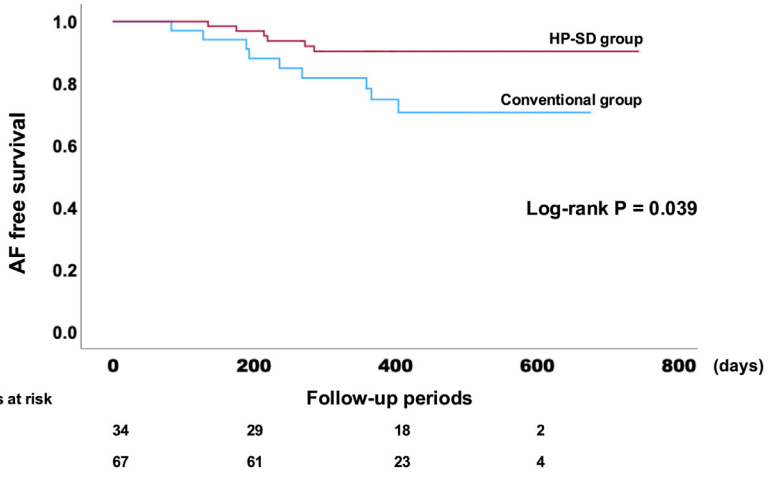

Figure 3 Kaplan-Meier analysis for the ablation outcome between the conventional and high-power, short-duration (HP-SD) groups. AF = atrial fibrillation.

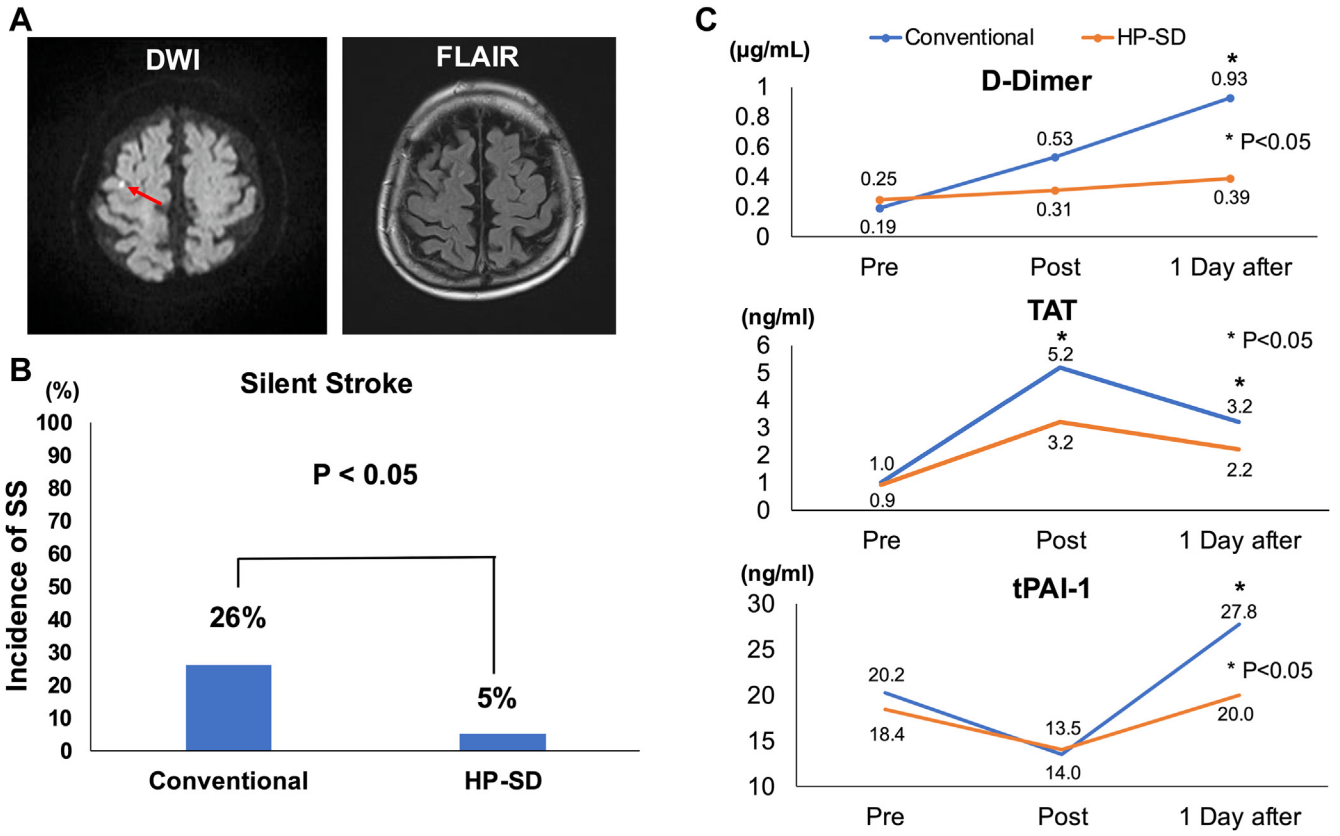

Figure 4 A: Representative head magnetic resonance imaging (MRI) within 48 hours after ablation. A diffusion-weighted echo-planar imaging (DWI) sequence with 1 lesion (white arrow) (right) and a corresponding fluid-attenuated inversion recovery (FLAIR) sequence without a lesion (left). B: The incidence of silent stroke (SS) in the high-power, short-duration (HP-SD) and conventional groups. C: Time course change of the thrombotic markers 1 day before, just after, and 1 day after the ablation procedure. TAT = thrombin-antithrombin complex; tPAI-1, total plasminogen activator inihibitor-1.

Incidence of SS during the ablation procedure

SS was detected in a small portion of patients by the MRI performed within 48 hours after the ablation procedure (Figure 4A). The incidence of SS was significantly higher in the conventional patients than the HP-SD patients (26% vs 5%, $P = .010$), as shown in Figure 4B.

Periprocedural change of thrombotic markers

Differences among thrombotic marker levels measured the day before, immediately after, and the day after ablation were examined and compared between groups between, as shown in Figure 4C. DD levels increased steadily over time, while the TAT peaked after the ablation. The tPAI-1 levels tended to decrease from 1 day before ablation to immediately after the procedure but then began increasing the day after ablation. The degree of elevation of all markers was significantly higher in the conventional group than in the HP-SD group.

Cutoff values of thrombotic markers for the prediction of SS

ROC analysis was performed to determine cutoff values for predicting the occurrence of SS using the differences in periprocedural changes of each thrombotic marker, after identifying when the difference in perioperative changes of each thrombotic marker was most pronounced, such as the change in DD from 1 day after ablation to before ablation (1 day – Pre DD), the change in thrombin-antithrombin complex from immediately after the ablation to preprocedure (Post – Pre TAT), and the change in tPAI-1 from the day after ablation to immediately after the ablation (1 day – Post tPAI-1), as shown in Figure 5. As a result, 1 day – Pre DD >0.05, Post – Pre TAT >3.3, and 1 day – Post tPAI-1 >12.5 were defined as cutoff values.

Predictors for the incidence of SS

Univariate logistic regression analysis revealed that CHA_2DS_2-VASc (congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, prior stroke or transient ischemic attack or thromboembolism, vascular disease, age 65–74 years, sex category) score, HP-SD setting, LA dwelling time, 1 day – Pre DD >0.05, Post – Pre TAT >3.3, and 1 day – Post tPAI-1 >12.5 were related to SS occurrence (Table 3). In the multivariate analysis, CHA_2DS_2-VASc score, HP-SD setting, LA dwelling time, and Post – Pre TAT >3.3 remained as independent predictors (either negative or positive) of SS.

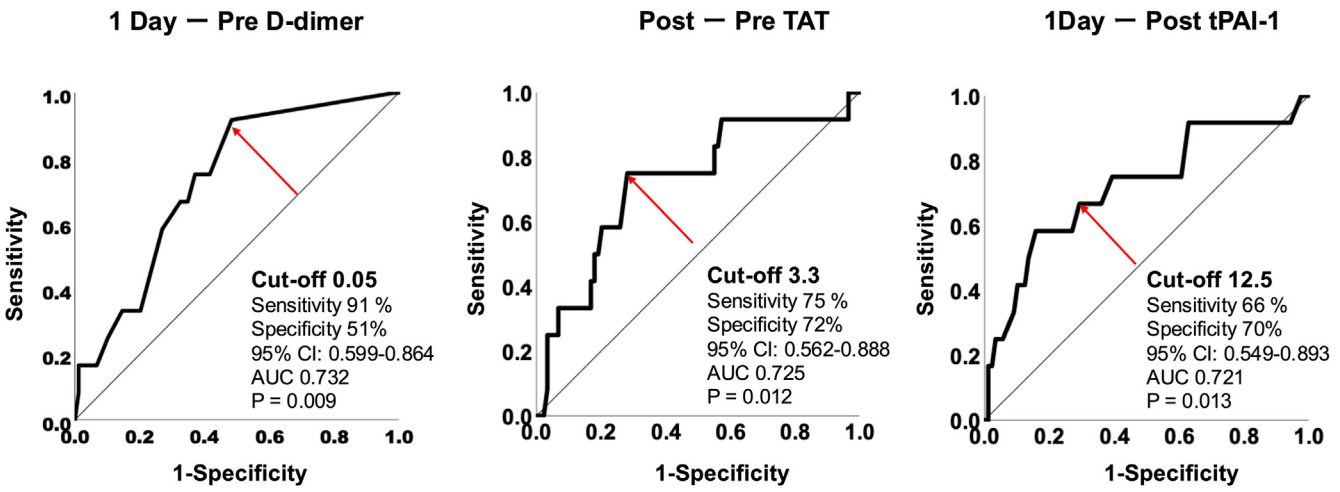

Figure 5 Receiver-operating characteristic analysis for the prediction of silent stroke occurrence using the periprocedural change of thrombotic markers. AUC = area under the curve; CI = confidence interval; TAT = thrombin-antithrombin complex; tPAI-1, total plasminogen activator inihibitor-1.

Discussion

The main findings of this study are as follows: (1) the ablation procedure using the HP-SD setting was associated with shorter LA dwelling time and RF application time, and with a better clinical outcome, compared with conventional ablation; (2) the incidence of SS was significantly higher in the conventional group than in the HP-SD group; and (3) significant periprocedural changes of thrombotic markers such as Post – Pre TAT >3.3, the HP-SD setting, and the LA dwelling time were proven to be independent markers of SS occurrence.

SS as a potential risk factor for cognitive dysfunction

Preventing thrombotic events like stroke/transient ischemic attack during the perioperative period of ablation for AF is important. Despite appropriate management of intraoperative ACT, thrombosis still occurs in 0.15% to 0.5% of cases.[11,12] In the present study, SS was used as a surrogate marker due to the low incidence of stroke/transient ischemic attack events. SS incidence is known to vary depending on ablation modality and anticoagulation protocols, ranging from approximately 5% to 30% postablation.[13,14] Schwarz and colleagues[15] reported that patients who underwent ablation for AF showed worse neurophysiological outcomes for verbal memory. On the other hand, according to the AXAFA-AFNET 5 (Anticoagulation Using the Direct Factor Xa Inhibitor Apixaban During Atrial Fibrillation Catheter Ablation: Comparison to Vitamin K Antagonist Therapy) trial, SS occurred in 26.1% of the patients following ablation for AF, and was not associated with cognitive dysfunction 3 months after the procedure.[16] While the data on SS predicting remote cerebral infarctions and affecting long-term cognitive function are mixed,

Table 3 Binominal logistic regression analysis for the prediction of SS.

	Univariate			Multivariate		
	Odds ratio	95% CI	*P* value	Odds ratio	95% CI	*P* value
Age	1.061	0.984–1.144	.124			
Nonparoxysmal AF	1.212	0.356–4.129	.758			
CHA_2DS_2-VASc score	1.629	1.106–2.400	.014	2.370	1.382–4.064	.002
LVEF	0.978	0.920–1.039	.466			
LAVI	0.991	0.954–1.028	.621			
NT-proBNP	1.000	1.000–1.001	.424			
eGFR	0.965	0.924–1.008	.106			
HP-SD setting	0.130	0.033–0.521	.004	0.141	0.023–0.852	.033
LA dwelling time	1.036	1.00–1.052	.014	1.036	1.001–1.072	.041
Post – Pre TAT >3.3	7.680	1.921–30.711	.045	5.838	1.039–32.815	.045
1 d – Pre DD >0.05	11.767	1.457–95.035	.021			
1 d – Post tPAI-1 >12.5	4.846	1.342–17.504	.045			

AF = atrial fibrillation; CHA_2DS_2-VASc = congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, prior stroke or transient ischemic attack or thromboembolism, vascular disease, age 65–74 years, sex category; CI, confidence interval; eGFR = estimated glomerular filtration rate; HP-SD = high power, short duration; LA = left atrial; LAVI = left atrial volume index; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro–B-type natriuretic peptide; Post – Pre TAT = change in thrombin-antithrombin complex from immediately after the ablation to preprocedure; 1 d – Post tPAI-1 = change in total plasminogen activator inhibitor-1 from the day after ablation to immediately after the ablation; 1 d – Pre DD = change in D-dimer from 1 day after ablation to before ablation.

preventing SS occurrence seems a reasonable goal because it partially predicts cerebral infarction and can potentially impact cognitive function.[17]

Correlation between AF ablation with the HP-SD setting and SS

In the present study, we aimed to validate the hypothesis that HP-SD ablation could reduce the perioperative thromboembolism risk through 2 potential mechanisms: (1) shortening the LA dwelling time to reduce periprocedural thrombosis risk and (2) reducing the risk of char and/or microbubble formation due to shorter RF application time, resulting in less periprocedural change of thrombotic markers compared with conventional ablation.

In terms of mechanism 1, longer procedure times are known to increase complications associated with ablation, including SS incidence.[18] Additionally, prolonged LA dwelling time predicts postprocedural cognitive dysfunction after AF ablation.[19] Thus, reducing RF application and LA dwelling times could lower thrombosis risk. Our study confirmed that shorter LA dwelling time is an independent negative risk factor for SS, consistent with previous findings mentioned previously. There are limited data comparing the incidence of SS between ablation methods using HP-SD and conventional settings.[20] Although the Short-AF (A Trial of High Power-Short Duration Versus Standard Power-Long Duration Radiofrequency Ablation for Treatment of Atrial Fibrillation) study is one of the randomized trials available, there are several significant differences from the present study.[21] First, the Short-AF study reported a tendency for a higher incidence of SS in the HP-SD group, which contrasts with the findings of our study. Additionally, the procedure time in the Short-AF study was reported to be drastically longer compared with that in our study. While the procedure and LA dwelling times in our study were comparable to those of previous studies,[22,23] those in the Short-AF study were approximately 3 times longer, which is likely responsible for the greater SS incidence.

In the comparison between patients who developed SS and those who did not, a significantly higher incidence of prior cerebral infarction was observed in patients with SS (Supplemental Table 2). While there were no significant differences in other background, procedural characteristics revealed that patients who developed SS had significantly longer LA dwelling times and a lower rate of PVI alone (Supplemental Table 3). The higher incidence of roof ablation and superior vena cava isolation in these patients likely contributed to the prolonged procedure time and may have influenced the occurrence of SS, supporting our hypothesis.

Regarding mechanism 2, the first-pass PVI ratio and ablation times differed between the Short-AF study and the current study. These differences, along with more RF applications, could lead to char or microbubble formation, increasing thrombosis risk. Even with HP-SD ablations, more RF applications could extend the total ablation time, negating the benefits of HP-SD. However, in the present study, perioperative thrombotic markers changed less with HP-SD than with conventional methods, and HP-SD ablation was proven to be an independent negative predictor of SS, indicating that HP-SD ablation does not increase thrombosis risk, even at high output. In the present study, Post – Pre TAT >3.3 remained an independent predictor for SS occurrence after multivariate correction. In comparison with TAT, DD and tPAI-1 have longer half-lives, requiring the observation of changes from 1 day after ablation to preablation for SS prediction.[24] Therefore, TAT is considered more useful as a predictive marker.

In recent years, ablation using 90 W output, known as very HP-SD, has become possible, and it has been reported to have the same safety profile as the historical control.[25,26] In previous studies, the SS frequency of this was reported to be low, at 8.2%, similar to our study, suggesting that the reduced procedural and RF application times in HP-SD therapy could lower the thrombotic risk.[27]

Influence of anticoagulation protocol on the incidence of SS

The perioperative anticoagulant therapy protocol is an important factor influencing the results of this study. Uninterruption of direct oral anticoagulants is recommended as a first-line protocol in the European and American guidelines.[12] In this study, we adopted a minimally interrupted anticoagulation protocol, as endorsed by guidelines and supported by 2 studies based on Japanese evidence.[28,29] Furthermore, a direct comparison between uninterrupted anticoagulation and minimally interrupted anticoagulation found no difference in the incidence of SS.[30] Therefore, the impact of this anticoagulation protocol on our study results is considered to be minimal.

Clinical implications

In AF ablation, it is very important to create a transmural lesion, and HP-SD ablation is one way to achieve this objective. However, at the same time, it is equally important to ensure the safety of the procedure. In addition to improving the treatment effect, HP-SD ablation has 2 significant benefits: (1) it decreases the risk of thrombus formation on treatment devices due to shorter LA dwelling times and (2) it suppresses char and microbubble formation during ablation by shortening the RF application time. These effects help suppress perioperative changes in thrombotic markers, potentially reducing the incidence of SS. Consequently, this approach may help prevent cerebral infarction and cognitive decline in the long term after ablation.

Limitations

There are several limitations of the present study. First, this is a nonrandomized single-center study. Therefore, the patient population in this study may have a different composition from the general population, which may affect the study results. A larger prospective randomized study would be needed to validate the results of the present study. Second,

because MRI was only taken postoperatively, small infarctions that spontaneously occurred immediately before ablation may have been counted as postprocedural SS.

Conclusion

Compared with conventional ablation, HP-SD ablation significantly reduces LA dwelling time and suppresses changes in perioperative thrombotic markers, thereby decreasing the incidence of SS.

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures: The authors have no conflicts to disclose.

Authorship: All authors attest they meet the current ICMJE criteria for authorship.

Patient Consent: All patients provided written informed consent prior to the procedure.

Ethics Statement: The present study was approved by the local ethics committee of the Jichi Medical University. The investigation conforms with the principles outlined in the Declaration of Helsinki, and reporting of the study conforms to STROBE and the broader EQUATOR guidelines.

References

1. Calkins H, Hindricks G, Cappato R, et al. 2017 HRS/EHRA/ECAS/APHRS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation. Heart Rhythm 2017;14:e275–e444.
2. Ouyang F, Tilz R, Chun J, et al. Long-term results of catheter ablation in paroxysmal atrial fibrillation: lessons from a 5-year follow-up. Circulation 2010;122:2368–2377.
3. Jaïs P, Cauchemez B, Macle L, et al. Catheter ablation versus antiarrhythmic drugs for atrial fibrillation: the A4 study. Circulation 2008;118:2498–2505.
4. Verma A, Jiang CY, Betts TR, et al. Approaches to catheter ablation for persistent atrial fibrillation. N Engl J Med 2015;372:1812–1822.
5. Tilz RR, Rillig A, Thum AM, et al. Catheter ablation of long-standing persistent atrial fibrillation: 5-year outcomes of the Hamburg Sequential Ablation Strategy. J Am Coll Cardiol 2012;60:1921–1929.
6. Pambrun T, Durand C, Constantin M, et al. High-power (40-50 W) radiofrequency ablation guided by unipolar signal modification for pulmonary vein isolation: experimental findings and clinical results. Circ Arrhythm Electrophysiol 2019;12:e007304.
7. Kaneshiro T, Kamioka M, Hijioka N, et al. Characteristics of esophageal injury in ablation of atrial fibrillation using a high-power short-duration setting. Circ Arrhythm Electrophysiol 2020;13:e008602.
8. Ravi V, Poudyal A, Abid QU, et al. High-power short duration vs. conventional radiofrequency ablation of atrial fibrillation: a systematic review and meta-analysis. Europace 2021;23:710–721.
9. Kamioka M, Hijioka N, Matsumoto Y, et al. Uncontrolled blood pressure affects atrial remodeling and adverse clinical outcome in paroxysmal atrial fibrillation. Pacing Clin Electrophysiol 2018;41:402–410.
10. Okumura K, Inoue K, Goya M, Origasa H, Yamazaki M, Nogami A. Acute and mid-term outcomes of ablation for atrial fibrillation with VISITAG SURPOINT: the Japan MIYABI registry. Europace 2023;25:euad221.
11. Joglar JA, Chung MK, Armbruster AL, et al. 2023 ACC/AHA/ACCP/HRS guideline for the diagnosis and management of atrial fibrillation: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation 2024;149:e1–e156.
12. Tzeis S, Gerstenfeld EP, Kalman J, et al. 2024 European Heart Rhythm Association/Heart Rhythm Society/Asia Pacific Heart Rhythm Society/Latin American Heart Rhythm Society expert consensus statement on catheter and surgical ablation of atrial fibrillation. Heart Rhythm 2024;21:e31–e149.
13. Nagao T, Suzuki H, Matsunaga S, et al. Impact of periprocedural anticoagulation therapy on the incidence of silent stroke after atrial fibrillation ablation in patients receiving direct oral anticoagulants: uninterrupted vs. interrupted by one dose strategy. Europace 2019;21:590–597.
14. Hohnloser SH, Camm J, Cappato R, et al. Uninterrupted edoxaban vs. vitamin K antagonists for ablation of atrial fibrillation: the ELIMINATE-AF trial. Eur Heart J 2019;40:3013–3021.
15. Schwarz N, Kuniss M, Nedelmann M, et al. Neuropsychological decline after catheter ablation of atrial fibrillation. Heart Rhythm 2010;7:1761–1767.
16. Haeusler KG, Eichner FA, Heuschmann PU, et al. MRI-detected brain lesions and cognitive function in patients with atrial fibrillation undergoing left atrial catheter ablation in the randomized AXAFA-AFNET 5 trial. Circulation 2022;145:906–915.
17. Calvert P, Kollias G, Pürerfellner H, et al. Silent cerebral lesions following catheter ablation for atrial fibrillation: a state-of-the-art review. Europace 2023;25:euad151.
18. Martinek M, Sigmund E, Lemes C, et al. Asymptomatic cerebral lesions during pulmonary vein isolation under uninterrupted oral anticoagulation. Europace 2013;15:325–331.
19. Medi C, Evered L, Silbert B, et al. Subtle post-procedural cognitive dysfunction after atrial fibrillation ablation. J Am Coll Cardiol 2013;62:531–539.
20. Chen WJ, Gan CX, Cai YW, et al. Impact of high-power short-duration atrial fibrillation ablation technique on the incidence of silent cerebral embolism: a prospective randomized controlled study. BMC Med 2023;21:461.
21. Lee AC, Voskoboinik A, Cheung CC, et al. A randomized trial of high vs standard power radiofrequency ablation for pulmonary vein isolation: SHORT-AF. JACC Clin Electrophysiol 2023;9:1038–1047.
22. Wielandts JY, Kyriakopoulou M, Almorad A, et al. Prospective randomized evaluation of high power during CLOSE-guided pulmonary vein isolation: the POWER-AF study. Circ Arrhythm Electrophysiol 2021;14:e009112.
23. Bunch TJ, May HT, Bair TL, et al. Long-term outcomes after low power, slower movement versus high power, faster movement irrigated-tip catheter ablation for atrial fibrillation. Heart Rhythm 2020;17:184–189.
24. Omote M, Asakura H, Takamichi S, et al. Changes in molecular markers of hemostatic and fibrinolytic activation under various sampling conditions using vacuum tube samples from healthy volunteers. Thromb Res 2008;123:390–395.
25. Reddy VY, Grimaldi M, De Potter T, et al. Pulmonary vein isolation with very high power, short duration, temperature-controlled lesions: the QDOT-FAST trial. JACC Clin Electrophysiol 2019;5:778–786.
26. Osorio J, Hussein AA, Delaughter MC, et al. Very high-power short-duration, temperature-controlled radiofrequency ablation in paroxysmal atrial fibrillation: the prospective multicenter Q-FFICIENCY trial. JACC Clin Electrophysiol 2023;9:468–480.
27. Boga M, Suhai FI, Orbán G, et al. Incidence and predictors of stroke and silent cerebral embolism following very high-power short-duration atrial fibrillation ablation. Europace 2023;25:euad327.
28. Nogami A, Harada T, Sekiguchi Y, et al. Safety and efficacy of minimally interrupted dabigatran vs uninterrupted warfarin therapy in adults undergoing atrial fibrillation catheter ablation: a randomized clinical trial. JAMA Netw Open 2019;2:e191994.
29. Okumura K, Aonuma K, Kumagai K, et al. Efficacy and safety of rivaroxaban and warfarin in the perioperative period of catheter ablation for atrial fibrillation – outcome analysis from a prospective multicenter registry study in Japan. Circ J 2016;80:2295–2301.
30. Nakamura K, Naito S, Sasaki T, et al. Uninterrupted vs. interrupted periprocedural direct oral anticoagulants for catheter ablation of atrial fibrillation: a prospective randomized single-centre study on post-ablation thrombo-embolic and haemorrhagic events. Europace 2019;21:259–267.