Journal of Interventional Cardiac Electrophysiology (2019) 54:209–215
https://doi.org/10.1007/s10840-018-0489-x

The relationship between obstructive sleep apnea and recurrence of atrial fibrillation after pulmonary vein isolation using a contact force–sensing catheter

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Received: 30 July 2018 / Accepted: 12 November 2018 / Published online: 20 November 2018
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Abstract

Purpose Our aim was to elucidate the relationship between obstructive sleep apnea (OSA) and atrial fibrillation (AF) recurrence after repeated pulmonary vein isolation (PVI).

Methods We conducted a non-randomized observational study, with the data prospectively collected. One hundred patients (paroxysmal AF, $n = 89$) underwent PVI using a contact force–sensing catheter. All patients underwent an electrophysiological study and additional ablation for left atrium-pulmonary vein (PV) reconnection and non-PV foci, 6 months after the first treatment session, regardless of AF recurrence. Those with an apnea-hypopnea index ≥ 15 were diagnosed with OSA. Continuous positive air pressure (CPAP) therapy was initiated after the second treatment session, based on results of a sleep study. For analysis, patients were classified into the non-OSA ($n = 66$), treated OSA (OSA patients undergoing CPAP; $n = 11$), and untreated OSA ($n = 23$) groups, and between-group differences evaluated.

Results After the first session, AF recurrence was observed in 18.2% (12/66) and 14.7% (5/34) of patients without and with OSA, respectively ($P = 0.678$). After the second procedure, the rate of AF recurrence was 12.1% (8/66) in the non-OSA group, 9.1% (1/11) in the treated OSA group, and 8.7% (2/23) in the untreated OSA group (log-rank $P = 0.944$).

Conclusions The rate of AF recurrence might not be greater in patients with untreated OSA than in those without OSA and those with treated OSA after repeated PVI, using a contact force–sensing catheter, for patients with paroxysmal or short-term persistent AF.

Keywords Atrial fibrillation · Pulmonary vein isolation · Obstructive sleep apnea · Continuous positive air pressure · Contact force–sensing catheter

1 Introduction

Obstructive sleep apnea (OSA) has a high association with cardiovascular diseases [1]. Gami et al. reported that OSA increased the incidence of atrial fibrillation (AF) and that the severity of OSA predicted the risk of AF [2]. Pulmonary vein isolation (PVI) is a standard treatment for patients with AF. However, previous studies have indicated that patients with OSA had a high recurrence rate of AF after PVI [3–7]. The use of continuous positive airway pressure (CPAP) has been shown to improve AF-free periods among patients with OSA [4–8]. In these studies, PVI was performed with a non-contact force–sensing catheter (non-CF-C). Moreover, the mechanism underlying AF improvement after PVI was not explored.

Electrical reconnection between the PV and the left atrium (LA) is believed to be the principal mechanism underlying AF recurrence after PVI [9]. Accordingly, multiple sessions of PVI could improve the outcomes for AF ablation [9, 10]. Moreover, CF-C is superior to non-CF-C for providing greater durability of PVI [11]. Considering that repeated PVI performed using CF-C improves the durability of PVI and reduces AF recurrence after PVI, we hypothesized that this treatment strategy could lower the incidence of AF recurrence among patients with OSA after PVI. Accordingly, our aim in

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this study was to evaluate the impact of OSA on the recurrence of AF after multiple sessions of PVI using CF-C.

2 Methods

2.1 Study population

This study was a non-randomized observational study and the data were prospectively collected. The study group included 141 patients (paroxysmal AF, $n = 118$) who underwent PVI using CF-C, between August 2013 and September 2015, at the Tokyo Metropolitan Hiroo Hospital, noting that CF-C was not available between October 2013 and December 2013. Patients with persistent AF (> 1 year) were excluded as these patients underwent additional procedures, including linear ablation and/or ablation using complex atrial fractionated electrograms (CAFE) during the first treatment session. All patients underwent a follow-up electrophysiological study and additional ablation for LA-PV reconnection and/or non-pulmonary vein foci (NPVF), if present, 6 months after the first treatment session, regardless of AF recurrence. The 100 patients who underwent a second treatment session, 6 months after the first, including 87 patients with paroxysmal AF and 13 with persistent AF lasting < 1 year, were entered into the analysis.

OSA was diagnosed as an apnea-hypopnea index (AHI) > 15. For analysis, patients were classified into the following groups: non-OSA group (patients without OSA), treated OSA (patients with OSA who underwent CPAP therapy), and untreated OSA (patients with OSA who did not undergo CPAP therapy). The AF recurrence rate was compared after the first and second treatment sessions among the three groups.

The study design and procedures were approved by the institutional review board of our hospital. All patients provided written informed consent before undergoing the first procedure.

2.2 Sleep study and CPAP therapy

Apnea was defined as the cessation of inspiration for ≥ 10 s. Hypopnea was defined as a reduction in airflow of ≥ 30%, with a decrease in oxygen saturation ≥ 4% for ≥ 10 s, in the presence of a thoracoabdominal ventilation effort. The AHI was calculated as the sum of the apneic and hypopneic events per hour, as per the methods described in the type 3 sleep study (LS-300, Fukuda Denshi, Tokyo) of the American Academy of Sleep Medicine, which monitored arterial oxygen saturation, heart rate, respiratory movement, and airflow. The criterion for OSA was an AHI ≥ 15, with at least 80% of all events being obstructive in nature [7].

A sleep study was performed when patients were admitted to our hospital for the first treatment session. According to the health assurance system in Japan, the cost of CPAP therapy is covered for patients with an AHI > 40, recorded during a type 3 sleep study, or > 20 when using polysomnography (PSG). As access to PSG was not available for all patients undergoing PVI in our institution, OSA was diagnosed using a stepwise screening process, with PSG (Compumedics E Series, Abbotsford, Australia) recommended for patients with a mild AHI (between 10 and 40). Finally, we recommended CPAP therapy for patients with a severe AHI (≥ 40 on the type 3 sleep study or ≥ 20 on PSG), according to the criteria of the Japanese health insurance system, with CPAP therapy initiated after the second treatment session.

2.3 Protocol for catheter ablation

Anti-arrhythmic drugs were discontinued for ≥ 5 half-lives prior to ablation, and no patients used amiodarone during this study. Oral anticoagulant therapy was initiated at least 1 month prior to the procedure. Transesophageal echocardiography and multi-detector computed tomography were performed to rule out the formation of an intra-atrial thrombus. The electrophysiological study was performed under continuous anesthesia, using an intravenous administration of propofol.

In the first treatment session, all patients underwent circumferential isolation of the ipsilateral PVs at the antrum, as per standard technique. Briefly, a decapolar catheter (Inquiry Luma-Cath; St. Jude Medical, St. Paul, MN, USA) or a 20-polar superior vena cava (SVC) right atrium coronary sinus electrode catheter (BeeAT, Japan Lifeline, Tokyo, Japan) was inserted via the right subclavian vein into the coronary sinus. A trans-septal puncture was performed using a radiofrequency-powered trans-septal needle, under fluoroscopic and/or intracardiac echocardiography guidance. The PVs were mapped using a circular mapping catheter (Lasso, Biosense Webster, Diamond Bar, CA, USA). A 3.5-mm contact force–sensing irrigated-tip catheter (ThermoCool Smart touch Navistar, Biosense Webster) and a three-dimensional anatomical mapping system (CARTO, Biosense Webster) were used for non-pulmonary vein foci (NPVF) mapping and ablation.

PV antral isolation was performed using double circular mapping catheters that were placed within the ipsilateral ostia of the superior and inferior PVs. Radiofrequency energy, delivered at 30 W (25 W for posterior wall ablation) and with a maximal temperature limit of 43 °C, was applied point-by-point for 30 s. The contact force was controlled to achieve a > 450 force-time integral at each point.

The endpoint of PVI was the achievement of a bidirectional conduction block between the LA and PVs. After confirming the complete bidirectional block, we continuously administered intravenous isoproterenol (4 μg/min), followed by a bolus injection of 40 mg of adenosine triphosphate (ATP), with isoproterenol infusion, to exclude reconnection or ATP-provoked dormant conduction between the PVs and LA. Catheter ablation was performed to eliminate the presence of reconnection and/or dormant conduction. If NPVFs were identified during drug infusion, catheter ablation was applied

to the foci, targeting the foci which induced AF. To locate the NPVFs, the mapping catheters (two ring catheters and one ablation catheter) were originally placed in the right atrium, atrial septum, and LA. After detection of reproducible foci (i.e., occurring at least twice in the same area), the catheters were placed around the earliest electrode of these catheters to identify the precise focus, with the site first localized by the earliest electrodes defined as the earliest site of NPVFs. After ablation of this earliest NPVF site, we reinitiated NPVF to confirm the effect of the ablation. When NPVFs were located in the SVC, the SVC was electrically isolated. The endpoint of catheter ablation for NPVF was confirmed elimination of all NPVFs, using ATP and an isoproterenol infusion. Ablation was not performed in patients with NPVF of multiple origins or unmappable foci.

All patients underwent a second electrophysiological study, 6 months after the first treatment session, regardless of AF recurrence. The presence or absence of a bidirectional conduction block between the LA and PVs was explored, and any reconnected PV was ablated. Then, continuous, intravenous administration of isoproterenol (4 μg/min), followed by a bolus injection of ATP (40 mg) with an isoproterenol infusion, was applied to reveal possible LA-PV reconnection and NPVFs. Additional ablations were performed for LA-PV reconnection and/or NPVFs. If patients had a recurrence of AF manifested by additional arrhythmic substrates, we performed further ablation targeting CFAE and roof as well as mitral linear ablation.

2.4 Follow-up

After undergoing PVI, patients were discharged from the hospital on oral anticoagulants. The use of anti-arrhythmic drugs could be discontinued at 3 months after ablation, at the physician's discretion. The rhythm and presence of arrhythmias were evaluated based on the patient's symptoms and a resting 12-lead electrocardiogram, which was recorded during regular visits to our outpatient clinic. To detect atrial tachyarrhythmia (ATa), we performed a 24-h Holter monitoring at 1, 3, and 6 months after the first treatment session, as well as at 1, 3, 6, 12, 18, and 24 months after the second treatment session. In this cohort, compliance with Holter monitoring was 92% after the first treatment session and 88.7% after the second. The number of patients using anti-arrhythmic drugs at the time of the first and second treatment sessions and at 6 months after the second session was 51 (51%), 15 (15%), and 9 (9%), respectively. Recurrent AF and ATa were defined as documented tachycardia lasting longer than 30 s.

2.5 Statistical analysis

Continuous and categorical data are presented as the mean ± standard deviation and numbers and percentages, respectively. Categorical variables were analyzed using the chi-squared (χ^2) test, where appropriate, with Fisher's exact test otherwise used. Continuous variables were compared using Student's t test or Mann-Whitney's U test, as appropriate for the data distribution. The cumulative incidence of AF recurrence and cardiac events were analyzed using the Kaplan-Meier method and a log-rank test. A multivariate Cox regression analysis was used to determine the risk factors for AF recurrence, with hazard ratios and 95% confidence intervals calculated. All analyses were performed using SPSS version 23.0J (SPSS Inc., Chicago, IL, USA). The level of significance was set at $P < 0.05$.

3 Results

3.1 Patient characteristics

The classification of patients into the non-OSA ($n = 66$), treated OSA ($n = 11$), and untreated OSA ($n = 23$) groups is shown in Fig. 1. The rate of obstructive events accounted for 93.4% of the total apneic events identified among patients with OSA. Fifteen patients among the 34 with an AHI between 10 and 40 underwent PSG to determine the indication for CPAP therapy, with an increase in the AHI indicated by PSG in these patients from 22.6 ± 8.9 to 34.0 ± 14.1 ($P = 0.017$). CPAP therapy was started, on average, 1.7 months after the second treatment session.

The characteristics of the patients forming our study group are reported in Table 1. Age, the body mass index, and the AHI were higher in the non-OSA than OSA groups. The numbers of patients with hypertension and diabetes were higher in the treated OSA and OSA groups than in the non-OSA group.

3.2 The outcome of the first treatment session

After the first session, AF recurrences were noted in 18.2% (12/66) of patients without OSA and 14.7% (5/34) of those with OSA. Patients underwent the second session at an average of 7 months after the first. The rate of reconnected PVs was 17% (45/264) in the non-OSA group (mean 0.6 PVs), 15.9% (7/44) in the treated OSA group (mean 0.5 PVs), and 12.0% (11/92) in the untreated OSA group (mean 0.3 PVs). NPVFs were identified in 19 (28.8%) patients in the non-OSA group, 4 (36.4%) in the treated OSA group, and 7 (30.4%) in the untreated OSA group at the second session (Table 2). The rate of reconnected PVs in the chronic phase and the incidence rate of NPVFs at the second session were similar for patients with and without OSA (reconnected PVs, 17% (45/364) versus 13.2% (18/136), respectively; NPVFs, 28.8% (19/66) versus 32.4% (11/34)). The distribution of NPVFs among the 19 patients without OSA was as follows: 3 in the SVC; 6 in the

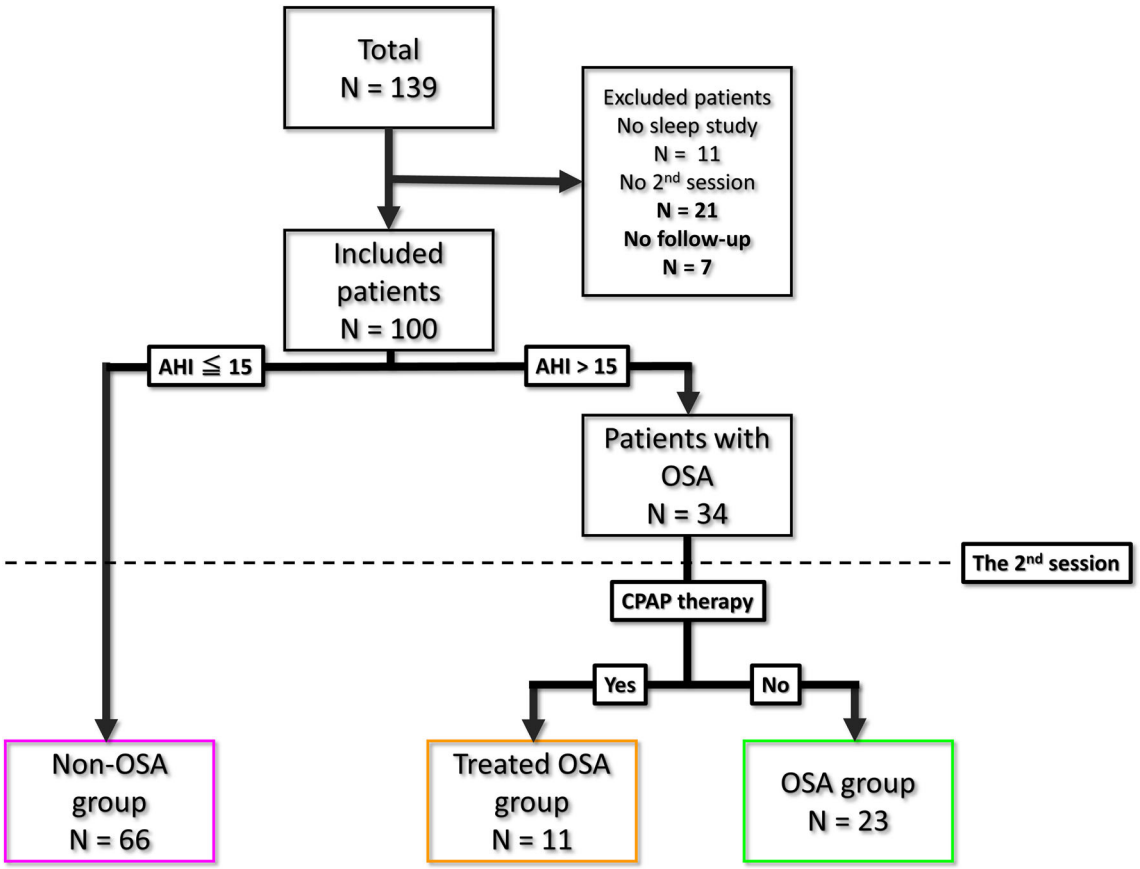

Fig. 1 Flow chart of the study. AHI apnea-hypopnea index, OSA obstructive sleep apnea

interatrial septum; 8 in the right atrium; 2 in the left atrium; and 3 unmappable foci. By comparison, for the 11 patients with OSA, 1 NPVF was in the SVC, 2 in the interatrial septum, 7 in the right atrium, and 3 in the left atrium.

3.3 The outcome of the second session

After the second treatment session, ATa recurred in 18.2% (12/66) of patients in the non-OSA group, 9.1% (1/11) in the treated OSA group, and 8.7% (2/23) in the untreated OSA group (log-rank, $P = 0.746$). The distribution of AF recurrence was 12.1% (8/66), 9.1% (1/11), and 8.7% (2/23), respectively, across the groups (log-rank, $P = 0.944$; Fig. 2a, b).

On multivariate analysis, NPVF and AF recurrence after the first session were identified as independent predictors of ATa recurrence after the second session (Table 3). Treated OSA and untreated OSA, however, were not related to ATa recurrence after the second treatment session.

4 Discussion

The main findings of our study were as follows: (1) the rate of LA-PV reconnection was similar for patients with and without OSA; (2) the incidence of NPVF at the first and second session was similar for patients with and without OSA; and (3) the outcome of repeated ablation for an AF trigger, using a CF-C, was not different for the non-OSA, treated OSA, and untreated OSA groups. On multivariate analysis, OSA and untreated OSA were not predictors of ATa recurrence after the second treatment session.

Several mechanisms might underlie the progression of OSA into AF, including abnormal gas exchange, autonomic nervous system imbalance, atrial stretch, and inflammation [12]. Atrial remodeling has previously been reported in patients with OSA [13, 14], with CPAP therapy improving changes in various parameters [14]. Therefore, OSA might cause a delay in atrial conduction, leading to an increased risk of AF development; CPAP therapy might be effective in modifying the remodeling process.

Previous studies have indicated a negative impact of OSA on outcomes of PVI. Fein et al. [7] prospectively evaluated patients with AF and OSA, who underwent PVI. In this study group, CPAP therapy was associated with a high success rate, after both single and multiple procedures for catheter ablation, compared to the non-treated group. Of note, 53.5% of patients in their study group presented with persistent AF, a ratio that was higher than in our study group. There are several other

Table 1 Patient characteristics

	Non-OSA group, $N = 66$	OSA group, $N = 34$		
		Treated OSA group, $N = 11$	Untreated OSA group, $N = 23$	*P* value
Male	44 (66.7)	8 (72.7)	19 (82.6)	NS
Age	60.4 ± 12.2	66.3 ± 9.6	68.3 ± 8.8	0.011
CHADS2 score	0.9 ± 1.4	1.6 ± 1.8	2.1 ± 1.9	NS
Heart failure	5 (7.6)	0	4 (17.4)	NS
Hypertension	25 (37.9)	8 (72.7)	16 (69.6)	0.008
Diabetes	4 (6.1)	3 (27.3)	7 (30.0)	0.006
Stroke	5 (7.6)	1 (9.1)	4 (17.4)	NS
Body mass index	23.5 ± 3.5	25.6 ± 2.8	26.1 ± 3.1	0.005
Left atrial diameter (mm)	37.4 ± 9.7	41.5 ± 6.2	41.7 ± 7.4	NS
Ejection fraction (%)	62.7 ± 7.1	63.6 ± 10.3	62.4 ± 14.1	NS
E/*e*′	12.8 ± 10.5	11.4 ± 3.2	17.6 ± 14.2	NS
BNP (pg/ml)	80.4 ± 157.6	40.9 ± 24.3	154.9 ± 210.6	N5
Cre (g/dl)	0.89 ± 0.22	0.8 ± 0.1	1.3 ± 1.8	NS
Left atrial volume (ml)	103.7 ± 25.1	107.5 ± 30.0	120.3 ± 40.1	NS
Persistent AF	7 (10.6)	3 (27.3)	3 (13.0)	NS
AF history (month)	34.0 ± 58.3	13.4 ± 20.5	25.8 ± 40.5	NS
Apnea hypopnea index	5.8 ± 3.8	26.0 ± 15.8	28.1 ± 10.9	0.0001
Follow-up period (month)	28.4 ± 6.2	23.3 ± 8.8	25.8 ± 7.7	NS

OSA obstructive sleep apnea; *CHADS2 score* congestive heart failure, hypertension, age ≥ 75 years, diabetes, and stroke score; *BNP* brain natriuretic peptide; *Cre* creatinine level; *AF* atrial fibrillation

reasons to explain the differences between our findings and those reported in previous studies. First, our study exclusively included patients with paroxysmal AF and persistent AF lasting < 1 year who could be treated effectively with triggering substrates by ablation. Relatively short history of AF might produce mild effects on atrial remodeling and this factor could abbreviate the influence OSA. Anter et al. reported a similar 1-year arrhythmia-free survival between patients with and without OSA who underwent PVI and additional NPVF ablation for PAF [15]. Their results are comparable with our results. Second, using a CF-C and repeated procedures improved the ablation outcome. Since the LA-PV reconnection rates in our study were similar among the non-OSA, treated OSA and untreated OSA group, we hypothesized that the use of CF-C

Table 2 The outcome of pulmonary vein isolation

	Non-OSA group, $N = 66$	Treated OSA group, $N = 11$	Untreated OSA group, $N = 23$	*P* value
Interval between 1st and 2nd session (month)	7.0 ± 1.5	7.0 ± 0.9	6.8 ± 1.2	NS
ATa recurrence between 1st and 2nd session (%)	15 (22.7)	3 (27.3)	6 (26.1)	N5
Patients with PV reconnection	32 (48.5)	5 (45.5)	8 (34.8)	NS
PV reconnection (%)	45/264 (17.0)	7/44 (15.9)	11/92 (12.0)	
LSPV	11 (16.7)	1 (9.1)	4 (17.4)	
LIPV	11 (16.7)	2 (18.2)	3 (13.0)	
RSPV	12 (18.2)	2 (18.2)	2 (8.7)	
RIPV	11 (16.7)	2 (18.2)	2 (8.7)	
Number of reconnected PV	0.6 ± 0.8	0.5 ± 0.7	0.3 ± 0.5	NS
Cava-tricuspid isthmus ablation	24 (36.4)	4 (36.4)	8 (34.8)	NS
Non-PV foci in 1st session	9 (13.6)	1 (9.1)	3 (13.0)	NS
Non-PV foci in 2nd session	19 (28.8)	4 (36.4)	7 (30.0)	NS

AF atrial fibrillation, *PV* pulmonary vein

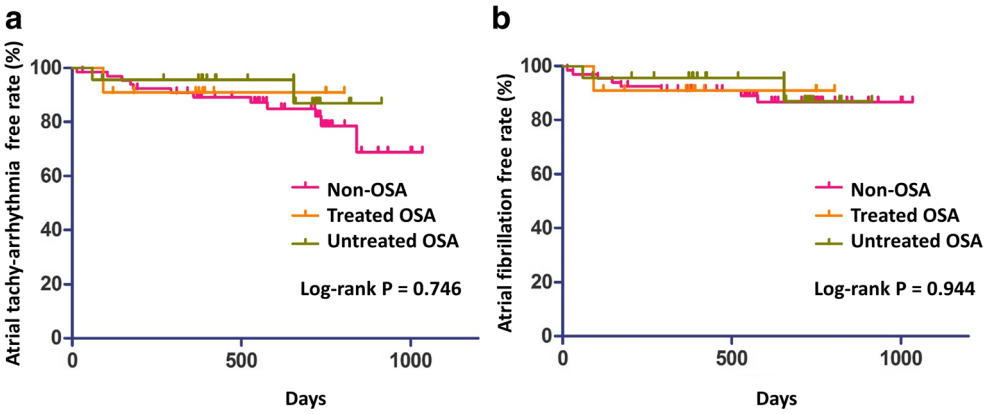

Fig. 2 Atrial tachyarrhythmia and atrial fibrillation recurrence after the second treatment session. **a** The atrial tachyarrhythmia-free rate after the second session. **b** The atrial fibrillation-free rate after the second session. OSA obstructive sleep apnea

could stabilize catheter manipulation, although a previous study, however, described that catheter instability using CF-C occurred during PVI [16]. The reasons for these discrepant results are not known. However, we propose that OSA might exacerbate catheter instability and that contact force information might help operators to stabilize the catheter.

A previous study reported that the incidence of NPVF was higher among patients with OSA than those without OSA [5, 15]. In this study, the prevalence of NPVF was similar among the three groups. There are several reasons why our findings differ from those of previous reports. First, the dose of isoproterenol used in previous studies was higher than those in our study [5, 15]. The optimal dose of isoproterenol is controversial, and a high dose of isoproterenol may cause non-clinical NPVF. Second, the cut-off value of AHI was also different, which would influence outcomes. Finally, in our study, all patients underwent PVI using CF-C. The use of CF-C may have a more intense and longer effect on the cardiac autonomic system, compared to procedures that did not use CF-C [17]. A previous study also reported that NPVF ablation added to PVI may improve the outcomes of ablation in patients with OSA [15].

CPAP therapy reduces sleepiness and improves the quality of life of patients with OSA. A recent study demonstrated that, compared to usual care alone, CPAP therapy in addition to usual care did not prevent cardiovascular events in patients with moderate-to-severe OSA and established cardiovascular disease [18]. The effect of CPAP therapy on AF recurrence after PVI, using new technology, should be revisited, especially for patients with a short history of AF.

4.1 Limitations

This was a single-center study. As well, the recurrence rate after the first treatment session might have been underestimated because of the short (7-month) follow-up period. For PSG, we used a type 3 sleep study method, which does not include an electroencephalogram and attendant, which might underestimate the incidence of OSA. In this

Table 3 Univariate and multivariate analyses of ATa recurrence after the second treatment session

	Univariate analysis		Multivariate analysis	
	HR	*P* value	HR	*P* value
Age	1.011 (0.966–1.059)	0.624		
Sex (male)	0.768 (0.262–2.247)	0.629		
Hypertension	0.911 (0.330–2.515)	0.857		
Body mass index	1.038 (0.900–1.199)	0.607		
AF history (month)	1.003 (0.996–1.011)	0.358		
PV reconnection	0.785 (0.279–2.208)	0.646		
LA volume (ml)	1.010 (0.994–1.026)	0.213		
OSA	1.092 (0.372–3.201)	0.873		
Untreated OSA	1.280 (0.407–4.021)	0.673		
ATa recurrence after theist session	6.039 (2.145–17.001)	0.001	4.596 (1.761–15.113)	0.006
Persistent AF	0.937 (0.211–4.154)	0.931		
NPVF	3.883 (1.326–11.367)	0.013	2.589 (0.843–7.948)	0.097

AF atrial fibrillation, *ATa* atrial tachyarrhythmia, *LA* left atrium, *OSA* obstructive sleep apnea, *PV* pulmonary vein

study, the patients with mild OSA (5 < AHI < 15) were included in the non-OSA group. We did not perform additional PSG for patients with an AHI between 10 and 40, which introduces a clear bias for CPAP therapy indication. Whether the patients with OSA underwent CPAP therapy or not was dependent on the patient's and physician's decision. As CPAP therapy was prescribed and introduced by respiratory medicine, with follow-up by each patient's physician, we do not have data on patients' compliance with CPAP therapy. As such, the effect of CPAP therapy might be underestimated. Outcomes may also have been influenced by differences in baseline patient characteristics between the groups, which could not be controlled for due to the relatively small sample size. Similarly, the relatively small number of patients with OSA was also limitation. These limiting factors could explain differences between our findings and those of previous studies. The effect of CPAP therapy on suppressing the recurrence of AF after PVI in patients with long-lasting persistent AF could not be evaluated using our study design. Finally, because of the small size of our study group, there was insufficient statistical power to reveal the non-inferiority of non-treated OSA to the treated OSA and non-OSA groups. A further study evaluating this factor should be performed.

5 Conclusions

In patients with PAF or persistent AF lasting < 1 year, OSA and CPAP therapy might not influence the recurrence of ATa after repeated catheter ablation using CF-C.

Author contributions Rintaro Hojo designed the study and wrote the initial draft of the manuscript. Seiji Fukamizu and Masayasu Hiraoka contributed to analysis and interpretation of data. All other authors have contributed to data collection and interpretation and critically reviewed the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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