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ORIGINAL RESEARCH

Prognostic Value of Spontaneous Potential in Left Atrial Posterior Wall Isolation and Radiofrequency Ablation for Non-Paroxysmal Atrial Fibrillation

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Objective: This study aimed to examine the long-term effects of left atrial posterior wall spontaneous potential (SP) in patients with non-paroxysmal atrial fibrillation (AF) who underwent pulmonary vein isolation (PVI) combined with left atrial posterior wall isolation (PVI+BOX) ablation.

Methods: Clinical data were retrospectively collected from 140 patients with symptomatic non-paroxysmal atrial fibrillation (NPAF) who underwent first-time radiofrequency ablation between 2022 and 2023. Based on the surgical procedure, patients were categorized into the pulmonary vein isolation group (PVI group) and the pulmonary vein isolation + left atrial posterior wall isolation group (PVI +BOX group). The PVI+BOX group was further subdivided into the spontaneous potential group (SP group) and the no-spontaneous potential group (no-SP group) based on the presence of SP after left atrial posterior wall isolation. Patients underwent monthly follow-ups in the clinic or via telephone and received 72-hour dynamic electrocardiography (ECG) at 3 and 12 months postoperatively. AF recurrence was compared among the groups, and factors associated with recurrence following AF ablation were analyzed.

Results: The PVI+BOX group included 78 cases, with 45 in the no-SP group and 33 in the SP group, while the PVI group comprised 62 cases. No significant difference was observed in the postoperative recurrence-free rate between the PVI+BOX and PVI groups. However, the SP group exhibited a higher postoperative recurrence-free rate compared to both the no-SP group ($p = 0.039$) and the PVI group ($p = 0.020$). No significant difference was found in the late recurrence-free rate between the no-SP and PVI groups. Multivariate logistic regression analysis indicated that the duration of AF and the presence of left atrial posterior wall SP were independent risk factors for post-ablation recurrence in patients with NPAF.

Conclusion: The presence of SP post-left atrial posterior wall isolation suggests a better long-term outcome in patients with NPAF following PVI+BOX catheter ablation.

Keywords: left atrial posterior wall, non-paroxysmal atrial fibrillation, pulmonary vein isolation, radiofrequency ablation, spontaneous potential

Introduction

Pulmonary vein isolation (PVI) serves as the cornerstone of atrial fibrillation (AF) catheter ablation and remains one of the most widely used treatments for paroxysmal atrial fibrillation (PAF). Currently, PVI combined with linear ablation represents the primary surgical approach for non-paroxysmal atrial fibrillation (NPAF).[1–4] In clinical practice, the long-term efficacy of standalone PVI for NPAF remains suboptimal. Advancements in cardiac electrophysiology suggest that

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AF development is associated with myocardial sleeves of the pulmonary veins, with the left atrial posterior wall also identified as a key abnormal site.[5] Pulmonary vein isolation combined with left atrial posterior wall isolation (PVI+BOX) ablation involves linear ablation of the left atrial apex line and left atrial posterior wall line in addition to bilateral pulmonary vein isolation, thereby isolating both the pulmonary veins and the left atrial posterior wall from other atrial regions. However, data on BOX ablation remain limited, and existing conclusions are inconsistent.[6–9]

Clinical findings indicate that spontaneous potential (SP) can be detected in the left atrial posterior wall of some patients with NPAF who have undergone PVI+BOX ablation. Previous studies indicated that most atrial premature beats (APBs) responsible for triggering AF originate from the pulmonary veins and induce AF through rapid discharges.[4] During embryonic development, the left atrial posterior wall and pulmonary veins originate from the same site.[10] However, whether the left atrial posterior wall SP is equivalent to spontaneous pulmonary vein potential and whether it can trigger AF remains unclear. Therefore, the relationship between left atrial posterior wall SP and long-term outcomes following PVI+BOX in patients with NPAF was investigated in this study.

Data and Methods

Objects

The clinical data in this study were retrospectively collected from 140 patients with symptomatic NPAF who underwent radiofrequency ablation for the first time between 2022 and 2023. Based on the surgical approach, patients were categorized into the PVI group and the PVI+BOX group. The PVI+BOX group was further subdivided into the SP group and the no-SP group based on the presence of left atrial posterior wall SP following BOX. All patients provided informed consent before treatment, and the study received approval from the local ethics committee.

NPAF that can be treated with catheter ablation includes persistent AF (PerAF) and long-standing persistent AF (LSPAF), as defined by the 2020 European Society of Cardiology (ESC) and European Association for Cardio-Thoracic Surgery (EACTS) guidelines for the management of AF.[11] Before catheter ablation, all patients underwent transesophageal echocardiography (TEE) or left atrial computed tomography angiography (CTA) to exclude left atrial and/or left auricular thrombosis. Additionally, pulmonary vein CTA was performed to assess the anatomical structure of the pulmonary veins. All antiarrhythmic drugs were discontinued for at least five half-life periods before the procedure.

Patients were excluded if they met any of the following criteria: (1) age younger than 18 years or older than 80 years; (2) diagnosis of valvular AF; (3) presence of hyperthyroidism; (4) history of cerebrovascular accidents or other neurological diseases within the past three months; (5) presence of other systemic diseases or tumors; (6) left atrial and/or left auricular thrombus; (7) AF of non-pulmonary vein origin; (8) prior cardiac surgery or heart disease requiring surgical intervention; (9) incomplete clinical data or failure to complete follow-up.

The radiofrequency ablation procedure was performed with the patient in a supine position under local anesthesia with 1% lignocaine. Bilateral femoral vein punctures were made, and a 10-pole electrode was advanced into the coronary sinus via the left femoral vein, while the interatrial septum was punctured via the right femoral vein. Two 8.5F Swartz sheaths were inserted into the left atrium for the administration of 100 μg/kg heparin. A ring electrode (Biosense-Webster, USA) and a saline-irrigated electrode catheter (Biosense-Webster, USA) were introduced into the left at rium via the Swartz sheath for modeling (CARTO 3D electroanatomic mapping system), mapping, and ablation. In the PVI group, linear ablation was performed around the circumferential bilateral pulmonary vein antrum to achieve PVI. In the PVI+BOX group, linear ablation targeted the circumferential bilateral PVI, left atrial apex line, and left atrial posterior wall line to achieve posterior wall isolation. Pulmonary vein isolation was confirmed by the absence of pulmonary vein potential or the presence of SP within the pulmonary vein, with pulmonary vein pacing unable to conduct to the left atrium. Left atrial posterior wall isolation was confirmed by the absence of atrial potential (Figure 1A) or the presence of SP (Figure 1B) in the left atrial posterior wall, with left atrial posterior wall pacing unable to conduct to other atrial sites. If AF persisted following PVI or BOX ablation, synchronized electrical cardioversion (100–150 J) was administered. Ablation parameters included power (35–45 W), saline flow rate (20–30 mL/min), and impedance (140–170 Ω).

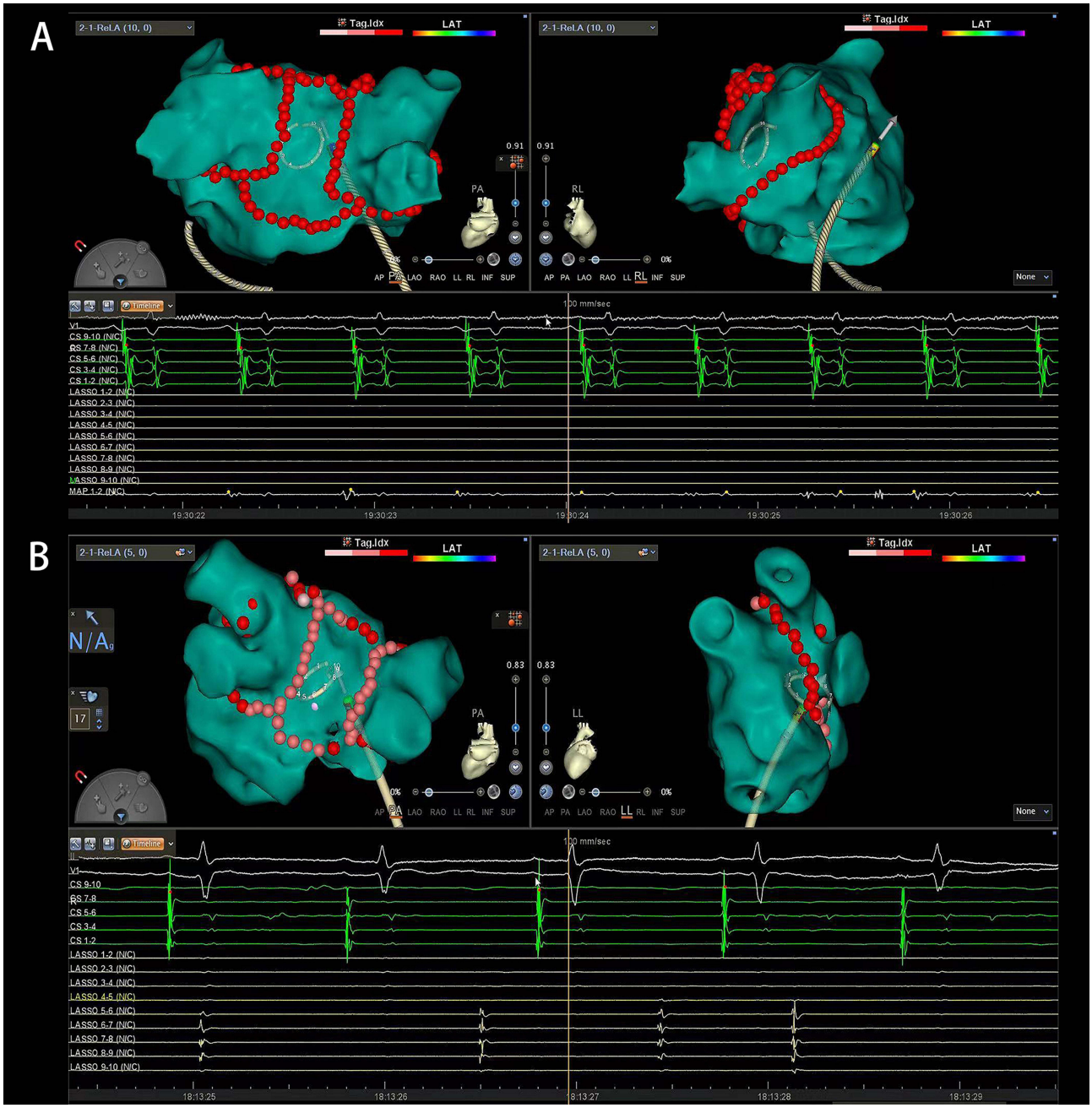

Figure 1 (**A**) Absence of spontaneous potential in the left atrial posterior wall following PVI + BOX. (**B**) Presence of spontaneous potential in the left atrial posterior wall following PVI + BOX.

Follow-Up

The clinical condition of all patients was continuously monitored through 24-hour electrocardiography (ECG), blood pressure measurement, and oxygen saturation assessment after the procedure. On the first postoperative day, patients were initiated to either warfarin or a novel oral anticoagulant based on personal preference, and were informed to use it mandatorily for at least three months. Anticoagulation therapy was then continued according to the CHA_2DS_2-VASc score (≥ 2 for males, ≥ 3 for females). Anti-arrhythmic medications, such as amiodarone or propafenone, with or without metoprolol, were routinely administered postoperatively and discontinued after three months. Regular follow-up was conducted through clinic visits or monthly telephone consultations to evaluate AF recurrence, assessed through symptoms including palpitations, chest discomfort, shortness of breath, and fatigue. Patients underwent routine ECG

examinations at local healthcare facilities, and in cases of the aforementioned symptoms, either a standard 12-lead ECG or dynamic ECG monitoring was performed. At three and twelve months postoperatively, cardiac ultrasound and 72-hour ECG monitoring were conducted at the clinic to detect AF recurrence. Follow-up data, including recurrence occurrences within three months postoperatively, were systematically documented.

The study's primary endpoint was the late recurrence of AF, defined as atrial tachyarrhythmia—including AF, atrial flutter, or atrial tachycardia—lasting more than 30 seconds on either a standard or dynamic ECG after all anti-arrhythmic medications were discontinued three months postoperatively.

Statistical Analysis

Categorical data were presented as frequency (percentage) and analyzed using the chi-square test. The Kolmogorov–Smirnov test was used to assess the normality of baseline characteristics. An independent sample *t*-test was conducted to compare data between the two groups, while non-normally distributed variables were expressed as median (25th–75th percentile) and analyzed using the Mann–Whitney *U*-test. The recurrence rate between the two groups was assessed using Kaplan-Meier survival analysis. Logistic regression was used to evaluate correlations between variables. A two-sided $P < 0.05$ was considered statistically significant. All statistical analyses were performed using SPSS 26.0 software.

Results

Basic Clinical Data

Among the 140 patients, 78 received PVI+BOX treatment, while 62 underwent PVI only. No significant differences were observed in the clinical characteristics between these groups (Table 1). Of the 78 patients treated with PVI+BOX, 33

Table 1 Baseline Characteristics of the PVI Group and PVI+BOX Group

Clinical Variables	PVI+BOX Group (n = 78)	PVI Group (n = 62)	*P* value
Male (%)	58(74.4%)	50(80.6%)	0.379
Age (year) (median, Q1-Q3)	63.0(53.5–69.0)	60.0(55.0–65.0)	0.490
Course of AF (month) (median, Q1-Q3)	14.5(7.0–42.0)	24.0(6.0–48.0)	0.761
BMI (kg/m^2) (mean±SD)	25.7±0.4	25.9±0.5	0.742
Echocardiographic parameters			
LVEDD (mm) (median, Q1-Q3)	47.25(44.0–50.0)	48.00(44.8–51.6)	0.296
LVESD (mm) (median, Q1-Q3)	30.05(27.3–32.9)	30.90(28.6–34.3)	0.143
LAD (mm) (mean±SD)	43.5±0.7	42.4±0.8	0.310
LVEF (%) (median, Q1-Q3)	65.15(59.9–70.6)	63.10(58.9–68.7)	0.350
Biochemical indexes			
NT-proBNP (pg/mL) (median, Q1-Q3)	499.0(310.0–1071.0)	484.0(322.0–780.0)	0.784
Scr (umol) (mean±SD)	75.1±2.0	78.5±1.6	0.192
WBC (×10^9/L) (median, Q1-Q3)	5.92(4.9–6.9)	6.07(5.3–7.3)	0.587
NE (×10^9/L) (median, Q1-Q3)	3.27(2.8–4.3)	3.16(2.8–4.3)	0.375
TG (mmol/L) (median, Q1-Q3)	1.20(0.9–1.7)	1.37(0.88–1.7)	0.469
TC (mmol/L) (mean±SD)	4.2±0.1	4.2±0.1	0.847
HDL-C (mmol/L) (mean±SD)	1.1±0.02	1.2±0.03	0.321
LDL-C (mmol/L) (mean±SD)	2.7±0.1	2.8±0.1	0.648
Concomitant disease			
Hypertension	40(51.3%)	29(46.8%)	0.596
Diabetes	11(14.1%)	10(16.1%)	0.739
CAD	7(9.0%)	2(3.2%)	0.168
HCM	3(3.8%)	1(1.6%)	0.431

Abbreviations: BMI, Body Mass Index; LVEDD, Left Ventricular End-diastolic Diameter; LVESD, Left Ventricular End-systolic Diameter; LAD, Left Atrial Diameter; LVEF, Left Ventricular Ejection Fraction; NT-proBNP, N-terminal pro-B-type Natriuretic Peptide; Scr, Serum creatinine; WBC, White Blood Cell Count; NE, Number of central granulocytes; TG, Triglyceride; TC, Total Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; LDL-C, Low Density Lipoprotein Cholesterol.

Table 2 Baseline Characteristics of the SP Group and No-SP Group

Clinical Variables	SP Group (n = 33)	No-SP Group (n = 45)	*P* value
Male (%)	27(81.8%)	31(68.9%)	0.196
Age (year) (median, Q1-Q3)	59.0(53.0–66.0)	66.0(53.0–70.0)	0.295
Course of AF (month) (median, Q1-Q3)	15.0(7.0–50.0)	14.0(9.0–36.0)	0.968
BMI (kg/m^2) (mean±SD)	25.77±0.5	25.70±0.5	0.933
Echocardiographic parameters			
LVEDD (mm) (median, Q1-Q3)	47.70(44.8–50.2)	47.20(44.00–48.7)	0.376
LVESD (mm) (median, Q1-Q3)	30.30(27.9–32.4)	29.90(27.00–32.9)	0.470
LAD (mm) (mean±SD)	44.17±1.22	42.98±0.85	0.416
LVEF (%) (median, Q1-Q3)	66.50(50.1–70.6)	65.00(59.9–69.7)	0.816
Biochemical indexes			
NT-proBNP (pg/mL) (median, Q1-Q3)	488.0(346.0–661.0)	522.0(294.0–1286.0)	0.685
Scr (umol) (median, Q1-Q3)	73.0(64.0–83.0)	76.0(64.0–86.0)	0.867
WBC (×10^9/L) (mean±SD)	5.94±0.7	6.23±0.3	0.470
NE (×10^9/L) (median, Q1-Q3)	3.15(2.8–3.7)	3.80(2.8–4.7)	0.390
TG (mmol/L) (median, Q1-Q3)	1.18(0.9–1.6)	1.20(1.0–1.7)	0.642
TC (mmol/L) (mean±SD)	4.0±0.1	4.3±0.2	0.183
HDL-C (mmol/L) (mean±SD)	1.1±0.04	1.1±0.03	0.869
LDL-C (mmol/L) (mean±SD)	2.6±0.1	2.8±0.1	0.464
Concomitant diseases			
Hypertension	16(48.5%)	24(53.3%)	0.672
Diabetes	6(18.2%)	5(11.1%)	0.375
CAD	2(6.1%)	5(11.1%)	0.441
HCM	2(6.1%)	1(2.2%)	0.384

Abbreviations: BMI, Body Mass Index; LVEDD, Left Ventricular End-diastolic Diameter; LVESD, Left Ventricular End-systolic Diameter; LAD, Left Atrial Diameter; LVEF, Left Ventricular Ejection Fraction; NT-proBNP, N-terminal pro-B-type Natriuretic Peptide; Scr, Serum creatinine; WBC, White Blood Cell Count; NE, Number of central granulocytes; TG, Triglyceride; TC, Total Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; LDL-C, Low Density Lipoprotein Cholesterol.

exhibited SP in the left atrial posterior wall, whereas 45 did not. Similarly, no significant differences were noted in the clinical characteristics between these subgroups (Table 2).

Long-Term Effect

Patients were monitored for 12 months postoperatively. In the PVI+BOX group, 24 patients (30.8%) experienced late recurrence, compared to 26 patients (41.9%) in the PVI group, with no statistically significant difference ($p = 0.145$; Figure 2A). In contrast, late recurrence occurred in 6 patients (18.2%) in the SP group and 18 patients (40%) in the no-SP group, showing a significant difference ($p = 0.041$; Figure 2B). Further analysis among the SP group, no-SP group, and PVI group indicated that the late recurrence rate was lower in the SP group than in the PVI group ($p = 0.020$), while no significant difference was found between the no-SP and PVI groups ($p = 0.780$; Figure 2B). Table 3 presents the late recurrence rates.

Complications

In the PVI+BOX group (n = 78), postoperative complications included pneumonia in 4 patients and pericardial effusion in 2 patients. In the PVI group (n = 62), 2 patients developed pneumonia, and 1 patient experienced pericardial effusion. No statistically significant difference in complication rates was observed between the groups ($p > 0.05$). All affected patients recovered before discharge. Table 4 presents the details on patient complications.

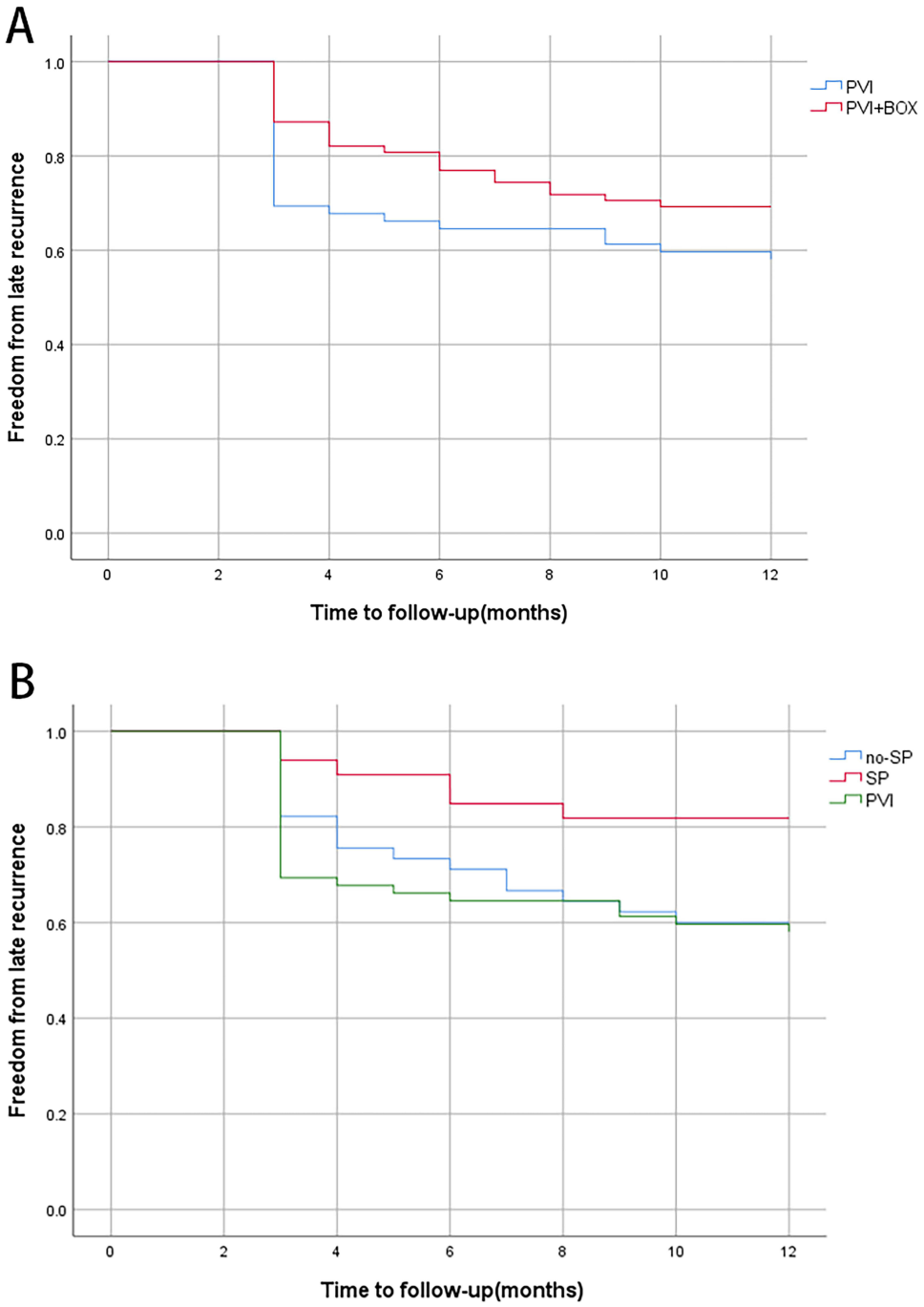

Figure 2 (**A**) Late recurrence-free survival curves comparing the PVI + BOX group and the PVI group. (**B**) Late recurrence-free survival curves comparing the SP group, no-SP group, and PVI group.

Analysis of Risk Factors for Postoperative Recurrence

Patients who underwent PVI+BOX treatment were categorized based on late AF recurrence. Univariate analysis results are presented in Table 5. The analysis identified AF duration ($p = 0.013$), total cholesterol (TC) ($p = 0.038$), white blood cell count (WBC) ($p = 0.032$), and SP ($p = 0.039$) as risk factors for post-ablation late recurrence of NPAF ($p < 0.05$).

No statistically significant differences were observed between the two groups in gender, age, body mass index (BMI), echocardiographic parameters, including left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), left atrial diameter (LAD), and ejection fraction (EF), as well as comorbidities and other factors.

Table 3 Late Recurrence Rate

Ablation Type		Recurrence
PVI+BOX group	SP group	6(18.2%)
	No-SP group	18(40%)
PVI group		26(41.9%)

Table 4 Complication Rate

	Postoperative Pneumonia	Pericardial Effusion	Total Case
PVI+BOX group	4(5.1%)	2(2.6%)	6(7.7%)
PVI group	2(3.2%)	1(1.6%)	3(4.8%)
P value	0.571	0.699	0.494

Table 5 Single-Factor Analysis of Postoperative Recurrence

Clinical Variables	Recurrence (n = 24)	No Recurrence (n = 54)	*P* Value
Male (%)	19(79.2%)	39(72.2%)	0.517
Age (year) (median, Q1-Q3)	65.0(52.0–70.0)	59.0(53.5–68.5)	0.795
Course of AF (month) (median, Q1-Q3)	30.5(12.0–90.0)	13.0(7.0–36.0)	0.013
BMI/(kg/m^2) (mean±SD)	25.4±0.7	25.9±0.5	0.549
Echocardiographic parameters			
LVEDD (mm) (median, Q1-Q3)	48.15(45.2–53.0)	47.10(43.5–49.7)	0.335
LVESD (mm) (median, Q1-Q3)	30.25(27.6–32.9)	30.05(27.2–32.8)	0.791
LAD (mm) (mean±SD)	42.9±1.2	43.8±0.9	0.568
EF (%) (median, Q1-Q3)	65.90(60.1–71.7)	63.55(59.9–69.6)	0.669
Biochemical indexes			
NT-proBNP (pg/mL) (median, Q1-Q3)	459.0(228.0–706.0)	532.5(345.5–1178.5)	0.208
Scr (umol) (mean±SD)	75.5±3.2	74.9±2.5	0.872
WBC (×10^9/L) (median, Q1-Q3)	5.47(4.3–6.7)	6.39(5.3–7.0)	0.032
NE (×10^9/L) (median, Q1-Q3)	3.03(2.6–4.0)	3.56(3.0–4.4)	0.104
TG (mmol/L) (median, Q1-Q3)	1.20(1.1–1.6)	1.19(0.9–1.7)	0.258
TC (mmol/L) (mean±SD)	4.5±0.2	4.1±0.1	0.038
HDL-C (mmol/L) (mean±SD)	1.1±0.04	1.1±0.03	0.905
LDL-C (mmol/L) (mean±SD)	2.8±0.2	2.6±0.1	0.350
Concomitant diseases			
Hypertension	13(54.2%)	27(50%)	0.734
Diabetes	3(12.5%)	8(14.8%)	0.786
CAD	3(12.5%)	4(7.4%)	0.468
HCM	1(4.2%)	2(3.7%)	0.922
SP	6(25%)	27(50%)	0.039

Abbreviations: BMI, Body Mass Index; LVEDD, Left Ventricular End-diastolic Diameter; LVESD, Left Ventricular End-systolic Diameter; LAD, Left Atrial Diameter; LVEF, Left Ventricular Ejection Fraction; NT-proBNP, N-terminal pro-B-type Natriuretic Peptide; Scr, Serum creatinine; WBC, White Blood Cell Count; NE, Number of central granulocytes; TG, Triglyceride; TC, Total Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; LDL-C, Low Density Lipoprotein Cholesterol; SP, Spontaneous potential).

Multivariate logistic regression analysis (Table 6) identified the course of AF (odds ratio (OR): 1.026, 95% CI: 1.007–1.046, p =0.006) and SP (OR: 0.219, 95% CI: 0.057–0.835, p =0.026) as independent predictors of late AF recurrence.

Table 6 Risk Factor Analysis for Late Recurrence

Multi-Factor Variable Regression		95% CI		*P* Value
		Lower Limit	Upper Limit	
Course of AF	1.026	1.007	1.046	0.006
WBC	0.750	0.379	1.485	0.410
NE	0.955	0.433	2.103	0.908
TC	1.599	0.767	2.981	0.233
SP	0.219	0.057	0.835	0.026

Abbreviations: WBC, White Blood Cell Count; NE, Number of Central Granulocytes); TC, Total Cholesterol.

Discussion

Application of PVI+BOX in AF Ablation

Since the identification of anomalous pulmonary venous activities as the primary trigger of AF, PVI has become the cornerstone of AF ablation. Over nearly two decades, it has been established as the standard procedure for AF catheter ablation, achieving a long-term success rate of 50% to 70% for paroxysmal AF. However, its effectiveness in NPAF remains limited, necessitating the incorporation of various linear ablation and substrate modification strategies. In recent years, growing recognition of the left atrial posterior wall's role in AF occurrence and maintenance has led researchers to explore left atrial posterior wall isolation, with BOX being a commonly used approach. Nevertheless, available data on BOX ablation remain scarce, and study conclusions are inconsistent. Yamaji et al reported that left atrial posterior wall isolation could reduce postoperative recurrence in patients with NPAF.[6,7] In contrast, Tamborero et al found no statistically significant difference between pulmonary vein isolation alone and its combination with left atrial posterior wall isolation in preventing arrhythmia recurrence.[8,9]

While recent studies, such as Yan et al, have compared radiofrequency ablation (RFA) guided by ablation index (RFCA-AI) and second-generation cryoballoon ablation (CBA-2) in AF treatment, the role of spontaneous potential (SP) in the left atrial posterior wall following PVI+BOX ablation remains unexplored.[12] Our study addresses this gap by evaluating SP as a novel predictor of long-term success after PVI+BOX, providing new insights into AF ablation strategies for NPAF patients.

Analysis of Risk Factors for Postoperative Recurrence

Several factors contribute to atrial substrate changes in patients with AF; however, the specific underlying mechanism remains unclear. Structural, electrical, and neural remodeling of the atrium are known to influence this process. Research has indicated a correlation between AF duration and post-ablation recurrence, with prolonged AF duration associated with a higher recurrence rate. The likely explanation is that an extended AF course increases the likelihood of atrial electrical and anatomical remodeling, making these changes irreversible and thereby significantly elevating the recurrence rate. In this study, univariate analysis of AF duration revealed that patients in the recurrence group had a significantly longer AF duration (30.5 [12–90] months) compared to the non-recurrence group (13 [7–36] months). Furthermore, multivariate analysis identified AF duration as an independent risk factor for post-ablation AF recurrence.

Distribution and Generation Mechanism of SP

Pulmonary veins exhibit distinct electrophysiological properties that contribute to the initiation and maintenance of AF. Pulmonary vein SP refers to spontaneous electrical activity generated by the pulmonary vein independent of the left atrium following PVI, with bidirectional PVI serving as a recognized endpoint of pulmonary vein ablation.[13,14] Pulmonary vein SP commonly manifests in three forms: sporadic isolated ectopic beats, slow and regular ectopic rhythms, and rapid fibrillation potential activity.[13,15] The primary mechanisms underlying pulmonary vein SP include (1) the focal mechanism, in which Jiang et al identified autorhythmic electrical activity as its principal cause.[16] Studies have indicated the presence of P cells, transitional cells, and Purkinje cells within the myocardial sleeves of pulmonary

veins, suggesting that pulmonary vein SP may result from electrical activity produced by autorhythmic cells.[17] (2) The reentrant mechanism, characterized by pronounced spatial heterogeneity in atrial action potential duration and a shortened plateau phase, elevates the risk of reentrant arrhythmias. (3) The AF trigger mechanism, as described by Yves et al, suggests that an AF-triggered pulmonary vein predicts AF recurrence following pulmonary vein isolation.[18] In such cases, pulmonary vein conduction is restored, leading to the occurrence of pulmonary vein SP, which has been frequently observed in AF-triggered pulmonary veins after circumferential PVI.[19]

There are currently limited studies on left atrial posterior wall SP. The left atrial posterior wall shares an embryological origin with the pulmonary vein, and its distinct histology and anatomical structures make it a crucial substrate for sustaining AF. It serves as a trigger for AF, with its electrophysiological properties contributing to AF maintenance. Prolonged AF episodes induce both electrophysiological and structural alterations, further facilitating AF persistence.[20] Embryologically, the smooth posterior wall is anatomically adjacent to the surrounding muscle trabecular tissue derived from the primitive left atrium. Due to this embryological origin, its electrophysiological characteristics more closely resemble the myocardial sleeves of pulmonary veins rather than the adjacent superior and inferior tissues.[20]

Electrophysiologically, pulmonary veins and cardiomyocytes in the posterior wall exhibit distinct electrophysiological and ion channel properties, which may contribute to arrhythmogenesis.[21] Anatomically, the cardiac muscle fibers of the left atrial posterior wall, particularly near the pulmonary vein junction, are oriented in varying directions. Consequently, conduction velocity and depolarization between adjacent tissues differ, and the transition between the epicardial and endocardial layers may exhibit heterogeneous anisotropy, potentially resulting in conduction delays, unidirectional blocks, and localized reentry.[22] These features of the posterior wall of the left atrium may cause SP in the left atrial posterior wall to trigger reentrant mechanism or focal mechanism similar to pulmonary vein potentials. These unique characteristics of the left atrial posterior wall may contribute to AF initiation and maintenance.

Analysis of Risk Factors for Postoperative Recurrence

In this study, pulmonary vein isolation combined with posterior wall isolation did not show a significant difference in reducing NPAF compared to pulmonary vein isolation alone. Among patients with NPAF who underwent pulmonary vein isolation with posterior wall isolation, the recurrence rate was lower in the left atrial posterior wall SP group than in the left atrial posterior wall no-SP group. Additionally, the recurrence rate in the SP group was lower than in the simple PVI group, whereas no significant difference was observed between the no-SP group and the PVI group. This finding suggests that left atrial posterior wall SP may indicate the presence of a trigger focus outside the pulmonary vein, leading to posterior wall isolation and subsequently reducing AF recurrence. Patients without SP of the posterior left atrial wall may have other unknown lesions or triggering mechanisms, and posterior wall isolation has a relatively unsatisfactory therapeutic effect on them.

Unlike Yan et al, which primarily focused on comparing different ablation techniques, our study introduces SP as a novel factor influencing AF recurrence after PVI+BOX ablation.[12] By identifying SP as a potential predictor of improved ablation success, this study provides new mechanistic insights into the role of left atrial substrate properties in AF recurrence.

White blood cells and neutrophils, as important indicators of the inflammatory system, their counts also reflect the degree of the inflammatory response. Previous studies have shown that patients with postoperative atrial fibrillation have significantly elevated peripheral blood white blood cells, and patients with significantly elevated white blood cells also have a longer duration of atrial fibrillation attacks. The increase of white blood cell count and neutrophil count, which are important indicators of the inflammatory system, is a risk factor for the maintenance of atrial fibrillation.

Limitations

In this study, SP has a certain degree of variability, the length of the recording time may affect the incidence of spontaneous potential. It is a single-center, small-sample, retrospective study, and postoperative AF recurrence was not recorded for some patients, potentially leading to discrepancies between the observed and actual long-term recurrence rates. Further multi-center studies are required to investigate the optimal degree of PVI+BOX.

Conclusion

SP following left atrial posterior wall isolation suggests a better long-term outcome for NPAF after PVI with BOX catheter ablation. The long-term outcome of non-paroxysmal atrial fibrillation refers to the free recurrence rate after 3 months of ablation.

Abbreviations

AF, Atrial Fibrillation; BMI, Body Mass Index; CAD, Coronary Artery Disease; CTA, Computerized Tomography Angiography; HCM, Hypertrophic Cardiomyopathy; HDL-C, High density lipoprotein cholesterol; LAD, Left Atrial Diameter; LDL-C, Low density lipoprotein cholesterol; LSPAF, Long-standing Persistent Atrial Fibrillation; LVEDD, Left Ventricular End-diastolic Diameter; LVEF, Left Ventricular Ejection Fraction; LVESD, Left Ventricular End-systolic Diameter; NE, Number of central granulocytes; NT-proBNP, N-terminal pro-B-type Natriuretic Peptide; PAF, Paroxysmal Atrial Fibrillation; PerAF, Persistent Atrial Fibrillation; PVI, Pulmonary Vein Isolation; Scr, Serum Creatinine; SP, Spontaneous potential; TC, Total cholesterol; TG, Triglyceride; WBC, White blood cell count.

Data Sharing Statement

All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethics Approval and Consent to Participate

This study was conducted with approval from the Ethics Committee of Fujian Medical University Union Hospital (Approval Number: 2024KY085). This study was conducted in accordance with the declaration of Helsinki. Written informed consent was obtained from all participants.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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