Impact of Baseline Right Bundle Branch Block on Outcomes After Pulmonary Vein Isolation in Patients With Atrial Fibrillation

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Right bundle branch block (RBBB) is one of the most frequent alterations of the electrocardiogram. Several studies have shown that RBBB is a risk factor of cardiovascular diseases. However, the clinical outcomes after pulmonary vein isolation (PVI) in patients with RBBB remain unclear. We enrolled consecutive atrial fibrillation (AF) patients who underwent PVI from the Osaka Rosai Atrial Fibrillation (ORAF) registry. We excluded patients with other wide QRS morphologies (left bundle branch block, ventricular pacing, and unclassified intraventricular conduction disturbances) and divided them into 2 groups: RBBB (QRS duration ≥120msec) and No-RBBB (QRS duration <120) groups. We compared the incidence of late recurrence of AF and/or atrial tachycardia (AT) (LRAF) between the 2 groups using a propensity score-matched analysis and evaluated the risk of LRAF using Cox regression model. We finally analyzed 671 consecutive AF patients. The RBBB group consisted of 50 patients (7.5%) and the No-RBBB group of 621 patients. Median follow-up duration was 734 [496, 1,049] days. Hypertension and diabetes mellitus were significantly higher in RBBB group than No-RBBB group. Among the 46 matched patients pairs, Kaplan-Meier analysis demonstrated that RBBB group had a significantly greater risk of LRAF than the No-RBBB group (p = 0.046). The Cox regression model revealed significantly higher risks of LRAF (HR, 2.30; 95% CI, 1.00 to 5.33; p=0.044) in RBBB group compared with No-RBBB group. Non-PV AF triggers were significantly higher in RBBB group than No-RBBB group (p = 0.048). In conclusion, RBBB can be an important predictor of LRAF after PVI. **(Am J Cardiol 2021;144:60−66)**

Catheter ablation has been established as a primary curative therapy for many arrhythmias. Pulmonary vein isolation (PVI) has become well-established as the standard therapy for patients with drug-refractory paroxysmal atrial fibrillation (PAF), because extrasystoles from the pulmonary veins (PV) are the most common AF trigger activity.[1] Good clinical outcomes of PVI are achievable but we occasionally experienced late recurrences of AF after PVI.[2−5] There have been many reports that have evaluated the predictors of recurrences after PVI. Several studies have shown that right bundle branch block (RBBB) is a risk factor of cardiovascular diseases and the appearance of RBBB in patients hospitalized for exacerbated heart failure (HF) is associated with a worse prognosis.[6,7] Various alternations in the electrocardiogram (ECG) such as left bundle branch block (LBBB) and an early repolarization pattern are associated with a high recurrence ratio of AF after PVI.[8,9] However, the clinical outcome after PVI in patients with RBBB remains unclear.

Methods

We enrolled consecutive AF patients who underwent primary PVI from May 2015 to November 2018 from the Osaka Rosai Atrial Fibrillation (ORAF) registry. We excluded patients with other wide QRS morphologies (LBBB, ventricular pacing, and unclassified intraventricular conduction disturbances). All patients received a detailed informed consent and the study protocol was approved by the hospital's institutional review board. The procedure were in accordance with the 'Declaration of Helsinki' and the ethical standards of the responsible committee on human experimentation. This study was granted an exemption from requiring ethics approval by Osaka Rosai Hospital Ethics Committee because this study was retrospective observational study and the permission for using the clinical data were obtained from all patients on admission.

RBBB was defined as a late R (R′) wave presenting in lead V_1 or V_2 with a slurred S wave in leads I and/or in lead V_6 with a prolonged QRS duration of ≥120 ms. Incomplete right bundle branch block (IRBBB) was defined as the above with a prolonged QRS duration >100ms and <120ms. The QRS duration was measured from the beginning of the QRS complex to the J point, which was defined as the point of transition from the R wave to the ST segment.[10−12]

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See page 65 for disclosure information.

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All patients underwent transthoracic echocardiography before the PVI. Transthoracic echocardiography was performed with a 5 MHz multiplane probe and live images were interpreted by experienced physicians who were blinded to the outcome of the PVI. Transesophageal echocardiography prior to the AF ablation was performed to exclude any left atrium (LA) and left atrial appendage thrombi.

All antiarrhythmic drugs (AADs) were discontinued at least 3 weeks before the ablation. In our hospital, all AADs were discontinued before the PVI because a previous study demonstrated that AADs, in particular Na^+ channel blockers, suppressed extra systoles from the PV.[13] Anticoagulation therapy was started at least 3 weeks before the PVI. A bolus infusion of hydroxyzine pamoate 25mg and pentazocine 15mg were intravenously administered before the PVI. The PVI was performed under mild sedation obtained with propofol and dexmedetomidine and the patients received adaptive servoventilation. An esophagus temperature monitoring catheter via the nose was placed. A duo-decapolar catheter (BeeAT, Japan Lifeline Co., Tokyo, Japan) was placed in the coronary sinus through the right internal jugular vein. If the patient was in AF, internal atrial cardioversion was performed with biphasic energy of 15 to 20J. We performed a transseptal puncture under guidance with the SOUNDSTAR 3-dimensional Ultrasound Catheter (Biosense Webster, Diamond Bar, California) from the right atrium. During the radiofrequency catheter ablation, after the transseptal puncture, one more long sheath (8.5Fr SL0, Abbott, Chicago, Illinois) was inserted into the LA. During the cryoballoon ablation after the puncture, a long sheath (8.5Fr SL0, Abbott, Chicago, Illinois) was exchanged for a steerable transseptal sheath (FlexCath, Medtronic, Minneapolis, Minnesota). A 100 IU/kg body weight bolus of heparin was administered following the transseptal puncture and heparinized saline was continuously infused to maintain the activated clotting time at 300 to 350 second. One circular mapping catheter was deployed in the superior and inferior PV, and the left-sided then right-sided ipsilateral PVs were circumferentially ablated guided by 3-dimensional LA mapping (CARTO3, Biosense-Webster, Diamond Bar, California). The PVI was performed with a 3.5 mm ablation catheter with an externally-irrigated tip (ThermoCool SmartTouch Catheter, Biosense-Webster, Diamond Bar, California). Radiofrequency current was delivered with a power of up to 30W and limited to 20W near the esophagus for 25 seconds. The end point of the PVI was the achievement of bidirectional conduction block between the LA and PVs, and any dormant PV conduction revealed by adenosine triphosphate and isoproterenol was eliminated. When AF persisted after the PVI or firing sites of atrial premature contraction triggers were detected, a substrate modification was sequentially performed. With the cryoballoon ablation, a 28mm cryoballoon was inserted into the left atrium over an inner-lumen circumferential mapping catheter (Achieve, Medtronic, Minneapolis, Minnesota). The cryoballoon was frozen at the ostium of the left and/or right superior and/or inferior PV with a freezing time of 180 to 240 seconds. The end point of the PVI was the achievement of bidirectional conduction block between the LA and PVs, and PV potential disappearance was confirmed using a circular mapping catheter after the procedure. In repeated ablation procedures, we confirmed the detection of non-PV foci (firing to AF), and we attempted to locate the spontaneous onset of the ectopic beats initiating AF in the baseline state or after an infusion of isoproterenol (up to 30 μg/min).

After the ablation, AADs were prescribed only in patients with early recurrence of AF (defined as the recurrence less than 3 months after ablation) and they were discontinued until 3 months after the ablation, regardless of AF recurrence. The patients underwent continuous electrocardiogram (ECG) monitoring for approximately 3 days (until discharge) after the ablation. They came to our cardiology clinic 1 month after the ablation. Subsequent follow-ups were performed every 3 months at the clinic. Patients were encouraged to have smartphone or tablet applications and check their pulse rate and rhythm every day and to visit our hospital if they experienced palpitations or other symptoms. The follow-up visits included a clinical interview, ECG, blood examination, 24-hour Holter monitoring or portable ECG (2-week cardiac event recording), and transthoracic echocardiography. Patients with palpitations or other chest symptoms underwent a portable ECG. Recurrence after the ablation was defined as AF and/or atrial tachycardia (AT) documented on the ECG or AF and/or AT continuing longer than 30 seconds on the Holter or portable ECG. AF and/or AT during the first 3 months after the ablation (blanking period) was considered as an early recurrence of AF and/or AT (ERAF), and AF and/or AT of more than 3 months after the ablation was considered as a late recurrence of AF and/or AT (LRAF).

JMP 15 statistical software (SAS Institute Inc., Cary, North Carolina) was used for the statistical analysis. Continuous variables were expressed as median [interquartile range]. Normality test was done for continuous variables by Shapiro-Wilk W test. Normal distribution was not confirmed in all variables. Two-group comparisons were analyzed by Mann-Whitney U test for continuous variables. Categorical data were expressed as the number (percentage) and were compared using the chi-square test or Fisher's exact test for categorical variables. A propensity score matching approach was used to adjust for potential confounding in the comparison of patients who had or did not have RBBB and the patients in the RBBB and the No-RBBB groups were matched 1:1 population. The matching variables included age, gender, hypertension, diabetes mellitus, AF subtypes, congenital heart disease and LA diameter. Kaplan-Meier curves were used for the incidence of an arrhythmia recurrence comparison and statistical significance was determined using the Log-rank test. Cox proportional hazards analysis was performed to compare hazard ratio of LRAF after ablation between the 2 groups. A value of $p < 0.05$ was considered to be statistically significant.

Results

The flowchart for the present analysis was shown in Figure 1. Finally, our study consisted of 671 patients and propensity score matching was performed (RBBB and No-RBBB groups consisted of 46 patients, respectively). The clinical characteristics of the patients, and echocardiographic parameters, medications at discharge

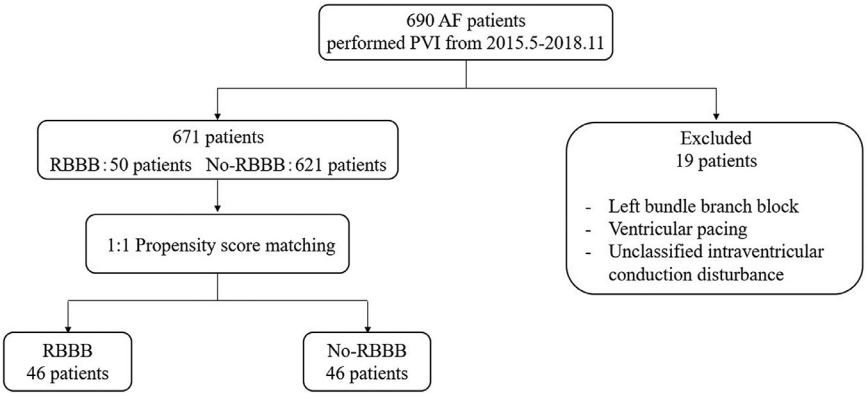

Figure 1. Flow chart of the study patients. Among 690 AF patients, we finally studied 671 patients (RBBB, 50 patients; No-RBBB, 621 patients). After propensity score-matching, 92 patients (RBBB, 46 patients; No-RBBB, 46 patients) were selected. AF = atrial fibrillation; PVI = pulmonary vein isolation; RBBB = right bundle branch block.

and procedure characteristics are shown in Table 1 and Table 2, respectively.

Kaplan-Meier analysis showed the no significant difference of the risk of ERAF between the RBBB and No-RBBB groups in overall population and in the propensity score-matched pairs ($p < 0.001$ and $p = 0.046$, Figure 2). Kaplan-Meier analysis demonstrated that the RBBB group had a significantly greater risk of LRAF after PVI than the No-RBBB group in overall population ($p < 0.001$, Figure 2). In the propensity score-matched pairs, the RBBB group had a significantly greater risk of LRAF after PVI than the No-RBBB group ($p = 0.046$, Figure 2). Kaplan-Meier analysis until 200 days which suggested the effect of electrical remodeling after PVI showed significant greater risk of LRAF after PVI than the No-RBBB group in overall population ($p = 0.023$, Supplementary Figure 1A), but no significant difference of the risk of LRAF after PVI in the propensity score-matched pairs ($p = 0.201$, Supplementary Figure 1B). The Cox regression model revealed significantly higher risks of LRAF in the RBBB group compared with the No-RBBB group (Table 3). The patients with IRBBB had no significant greater risk of LRAF after PVI compared with the patients with QRS duration ≤100ms (Supplementary Figure 2).

In the present study, the number of patients with LRAF after ablation was 20 in the RBBB group and 125 in the No-RBBB groups in overall population, respectively (40.0% vs 20.1%, $p = 0.001$). Repeated ablation was performed in 12 of 20 patients in the RBBB group and 82 of 125 patients in the No-RBBB group, respectively. The electrophysiological findings in the repeated ablation procedures are shown in Table 4. There was no significant difference in the ratio of pulmonary vein reconnections between the 2 groups. The incidence of non-PV AF triggers (atrial

Table 1
Clinical characteristics in patients with RBBB and No-RBBB

	Overall			Propensity score-matching		
	RBBB (n=50)	No-RBBB (n=621)	p value	RBBB (n=46)	No-RBBB (n=46)	p value
Age (years)	69 [66-76]	69 [62-75]	0.174	70 [66-76]	71 [66-77]	0.750
Male	34 (68.0%)	393 (63.3%)	0.505	32 (69.6%)	29 (63.0%)	0.508
Hypertension	37 (74.0%)	352 (56.7%)	0.017	36 (78.3%)	33 (71.7%)	0.470
Diabetes mellitus	16 (32.0%)	105 (16.9)	0.008	16 (34.8%)	17 (37.0%)	0.828
Chronic heart failure	3 (6.0%)	83 (13.4%)	0.134	3 (6.5%)	2 (4.3%)	0.646
Stroke	3 (6.0%)	63 (10.1%)	0.344	3 (6.5%)	4 (8.7%)	0.694
Ischemic heart disease	2 (4.0%)	49 (7.9%)	0.318	2 (4.3%)	3 (6.5%)	0.646
Congenital heart disease	4 (8.0%)	1 (0.2%)	<0.001	1 (2.2%)	1 (2.2%)	1.000
Chronic obstructive pulmonary disease	1 (2.0%)	7 (1.1%)	0.584	1 (2.2%)	1 (2.2%)	1.000
Paroxysmal atrial fibrillation	27 (54.0%)	397 (63.9%)	0.161	26 (56.5%)	25 (54.3%)	0.834
Chronic kidney disease	10 (20.0%)	91 (14.7%)	0.309	10 (21.7%)	9 (19.6%)	0.797
CHADS2-VASc score			0.347			0.199
0	3 (6.0%)	58 (9.4%)		3 (6.5%)	1 (2.2%)	
1	6 (12.0%)	129 (20.8%)		4 (8.7%)	9 (19.6%)	
2	12 (24.0%)	124 (20.0%)		10 (21.7%)	5 (10.9%)	
>3	29 (58.0%)	310 (49.9%)		29 (63.0%)	31 (67.4%)	
Albumin (g/dl)	4.1 [3.9-4.4]	4.1 [3.9-4.4]	0.871	4.1 [3.9-4.4]	4.2 [3.8-4.4]	0.937
Brain natriuretic peptide (pg/ml)	130 [65-205]	89 [40-198]	0.611	130 [70-196]	130 [62-268]	0.873

Continuous data are presented as median (interquartile range). Categorical variables are presented as numbers (percentage). RBBB = right bundle branch block.

Table 2
Electrocardiographic and/or echocardiographic parameters, medications at discharge, and procedure characteristics in patients with RBBB and No-RBBB

	Overall			Propensity score-matching		
	RBBB (n=50)	No-RBBB (n=621)	p value	RBBB (n=46)	No-RBBB (n=46)	p value
QRS width	144 [128-151]	96 [91-100]	<0.001	143 [128-151]	97 [94-100]	<0.001
Left ventricular diameter in diastole	48 [45-51]	48 [45-51]	0.810	48 [44-51]	48 [46-51]	0.657
Left ventricular diameter in systole	29 [27-32]	29 [27-33]	0.643	29 [27-32]	29 [27-34]	0.869
Left ventricular ejection fraction	69 [64-73]	68 [63-73]	0.495	69 [64-73]	69 [63-73]	0.970
Left atrial diameter	44 [42-49]	44 [40-48]	0.294	44 [41-49]	46 [41-49]	0.660
Tricuspid regurgitation pressure gradient	27 [24-32]	25 [21-30]	0.041	26 [24-32]	28 [24-31]	0.854
Pulmonary artery pressure	31 [27-35]	28 [24-33]	0.021	29 [27-35]	31 [27-34]	0.729
Tricuspid annular plane systolic excursion	20 [18-22]	22 [20-25]	<0.001	20 [18-22]	22 [20-26]	0.005
Right atrial diameter	40 [37-45]	38 [35-43]	0.039	40 [37-45]	38 [37-43]	0.269
Right ventricular diameter	36 [31-39]	33 [31-36]	0.050	36 [31-39]	32 [29-34]	0.043
Inferior vena cava diameter	14 [11-18]	12 [10-15]	0.001	14 [11-18]	13 [10-14]	0.086
Direct oral anticoagulant	41 (82.0%)	549 (88.4%)	0.181	38 (82.6%)	42 (91.3%)	0.216
Anti-arrhythmic drug (Ia Ib Ic III IV)	8 (16.0%)	100 (16.1%)	0.985	7 (15.2%)	6 (13.0%)	0.765
β-blocker (II)	18 (36.0%)	262 (42.2%)	0.393	16 (34.8%)	18 (39.1%)	0.666
ACEI/ARB	22 (44.0%)	228 (36.7%)	0.305	22 (47.8%)	21 (45.7%)	0.834
Digitalis	2 (4.0%)	21 (3.4%)	0.817	2 (4.3%)	1 (2.2%)	0.557
Cryoballoon ablation	5 (10.0%)	108 (17.4%)	0.179	5 (10.9%)	4 (8.7%)	0.726
Cavo tricuspid isthmus block	35 (70.0%)	366 (58.9%)	0.125	33 (71.7%)	30 (65.2%)	0.501
Superior vena cava isolation	2 (4.0%)	57 (9.2%)	0.214	2 (4.3%)	4 (8.7%)	0.398
Left atrial posterior wall isolation	5 (10.0%)	52 (8.4%)	0.691	4 (8.7%)	5 (10.9%)	0.726
Atrial premature contraction (AF trigger)	2 (4.0%)	32 (5.2%)	0.721	2 (4.3%)	3 (6.5%)	0.646

Continuous data are presented as median (interquartile range). Categorical variables are presented as numbers (percentage). ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin II receptor blocker; RBBB = right bundle branch block.

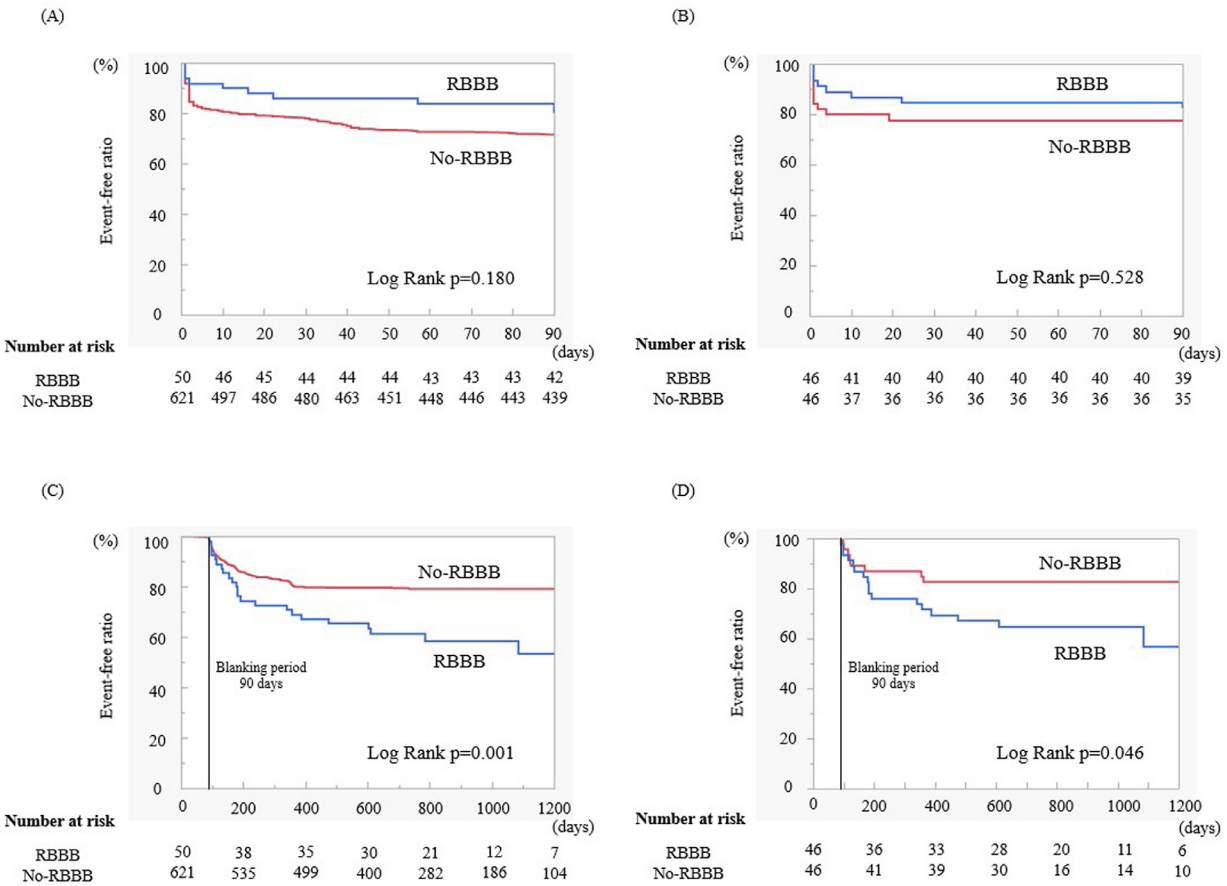

Figure 2. Kaplan-Meier curves illustrating the freedom from ERAF (*A*) in overall population (before propensity score matching) (*B*) among the propensity score-matched pairs between RBBB and No-RBBB groups. Kaplan-Meier curves illustrating the freedom from LRAF (*C*) in overall population (before propensity score matching) (*D*) among the propensity score-matched pairs between RBBB and No-RBBB groups. ERAF = early recurrence of atrial fibrillation and/or atrial tachycardia; LRAF = late recurrence of atrial fibrillation/atrial tachycardia; RBBB = right bundle branch block.

Table 3
The Cox regression model for LRAF

	Number of events				
	RBBB (n=46)	No-RBBB (n=46)	HR	95% CI	P value
LRAF	17	8	2.30	1.00-5.33	0.044

CI = confidence interval; HR = hazard ratio; LRAF = late recurrence of atrial fibrillation/atrial tachycardia; RBBB = right bundle branch block.

Table 4
Electrophysiological findings in repeated ablation

	RBBB (n=12)	No-RBBB (n=82)	P value
PV reconnection	7 (58.3%)	48 (58.5%)	0.989
Non-PV trigger (AF occurrence)	5 (41.7%)	14 (17.1%)	0.048
superior vena cava	3 (25.0%)	5 (6.1%)	0.028
left atrial posterior wall	2 (16.6%)	4 (4.9%)	0.119
Left atrial septum	-	3 (3.7%)	0.501
Right atrial septum	-	4 (4.9%)	0.434
High right atrium	-	1 (1.2%)	0.701
Mitral annulus	-	1 (1.2%)	0.701

Categorical variables are presented as numbers (percentage). AF = atrial fibrillation; RBBB = right bundle branch block.

premature contractions (APCs) confirmed firing to AF) was significantly higher in the RBBB group than the No-RBBB group, especially superior vena cava origin.

DISCUSSION

This study had following findings: (1) the prevalence of RBBB patients was 7.5% (50 patients, male; 68.0%) in AF patients underwent PVI enrolled from our registry; (2) RBBB patients had a significantly greater risk of LRAF after PVI than those without RBBB in the retrospective propensity score-matched analysis; (3) Non-PV AF triggered APCs were significantly higher in the RBBB group than the No-RBBB group.

The prevalence of RBBB is known to increase with age, to be approximately twice as high in males as in females, and to be higher in patients with hypertension and diabetes mellitus.[14,15] In the present study the prevalence of RBBB patients was 7.5% and RBBB patients was comprised of 50 patients (male, 34 patients; female, 16 patients). The prevalence of RBBB was higher as compared with the data in the other studies.[16-18] The Copenhagen City Heart Study showed that the prevalence of RBBB was 1.4% in males and 0.5% in females (more than 20 years old).[17] The Reykjavik Study demonstrated that the prevalence of RBBB was 1.4% in males and 0.7% in females (33 to 79 years old).[19] The higher age of our patients and history of AF could explain the higher incidence of RBBB in the present study.

We demonstrated that the incidence of LRAF after PVI was significantly higher in patients with RBBB than in those without RBBB. RBBB is generally considered as a healthy subjects.[17,20] However, many studies have shown that RBBB is associated with a poor clinical course and outcome in several diseases. Bussink et al reported that RBBB is associated with an increased cardiovascular risk and all-cause mortality.[17] The presence of RBBB has been associated with a poor prognosis in HF patients.[6,7] Pre-existing RBBB has been found in 10% of transcatheter aortic valve replacement recipients and is associated with poorer clinical outcomes.[21] RBBB accompanying an ST-elevation myocardial infarction of any location is an independent predictor of a high in-hospital mortality.[22]

Above mentioned poor outcomes in patients with cardiovascular disease may be related to cardiac function of RBBB. Several studies reported the relationship between RBBB and cardiac function. Oketona et al showed that right ventricular dysfunction is recognized in RBBB patients with HF and is an independent predictor of adverse outcomes in such patients.[23] They have been reported that RBBB is associated with left ventricular (LV) function as well as right ventricular (RV) function. Sillanmäki et al showed that RBBB caused LV mechanical dyssynchrony as evaluated by a myocardial perfusion imaging phase analysis.[24] In RBBB patients who received cardiac resynchronization therapy showed a significant larger percent reduction in LV end-diastolic volume, LV end-systolic volume and left atrial volume compared with the controls.[25] These studies demonstrated a prolonged QRS duration might suggest the existence of myocardial injury and interstitial collagen accumulation, which affects the cell-to-cell communication and these changes might enhance the LV dysfunction, left atrial remodeling and arrhythmogenicity. We showed the patients with IRBBB had no significant greater risk of LRAF after PVI compared with the patients with normal QRS width (Supplementary Figure 2), while the RBBB group had a significantly greater risk of LRAF after PVI than the No-RBBB group (Figure 2). Longer QRS width might have myocardial injury and interstitial collagen accumulation and induce substrate abnormality in atrium.

In the present study, RBBB group had higher incidence of hypertension and diabetes mellitus (Table 1). Tricuspid annular plane excursion (TAPSE) was significantly lower and RV diameter was significantly larger in RBBB group than No-RBBB group in the propensity score-matched pairs (Table 2). In the repeated ablation, the incidence of non-PV AF triggers (confirmed firing to AF), especially in superior vena cava, was significantly higher in the patients with RBBB than in those without RBBB, but PV reconnection was not significantly different between the both groups (Table 4). These results could suggest LA remodeling was not progressing in RBBB than No-RBBB group, but RBBB might have RV substrate abnormality and cause non-PV AF triggers such as superior vena cava. Possible mechanism of the relationship between RBBB and LRAF according to our results and previous reports was shown in Figure 3.

RBBB is one of the most frequent alterations of the electrocardiogram and usually judged to be within normal range. However, according to the present study, the prevalence of RBBB was higher as compared with the data in the other studies and RBBB patients could have significant greater risk of arrhythmia recurrence after PVI than those without RBBB. In patients with AF, RBBB had higher incidence of hypertension, diabetes mellitus and RV systolic dysfunction. Strict follow-up after PVI as well as the management of co-morbidities such as hypertension and diabetes mellitus should be required in AF patients with RBBB.

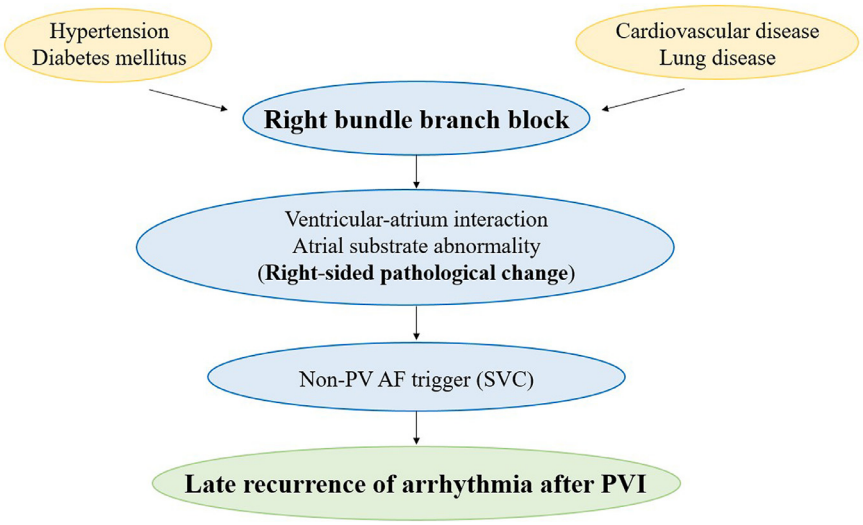

Figure 3. Possible mechanism of the relationship between RBBB and LRAF according to the present study. LRAF = late recurrence of atrial fibrillation and/or atrial tachycardia: RBBB = right bundle branch block.

Several limitations of the study need to be acknowledged. First, the present study was a single center and retrospective design. Second, few RBBB patients were enrolled in the present study. Previous reports showed the prevalence of RBBB was about 1.0% of total population. We believe that the present results are clinically important, despite of the small number of enrolled RBBB patients, considering the proportion of RBBB in the total population. Third, the degree and location of low voltage or scar tissue were not evaluated in RBBB and No-RBBB groups. The ratio of hypertension and diabetes mellitus were significantly higher, and TAPSE was significantly lower and RV diameter was significantly larger in the RBBB group than the No-RBBB group in our study. These factors might influence on the progression of LA substrate change and RV overload in RBBB group. Fourth, patients were followed by our cardiology clinic and their primary care doctors. Patients were encouraged to check their pulse rate and rhythm every day, have applications that can detect irregular pulse, and visit our hospital if they experienced palpitation or other symptoms, but the follow-up might not be complete.

In conclusion, RBBB patients could be a significantly greater risk of LRAF after PVI than those without RBBB. Non-PV AF triggered APCs were significantly higher in the RBBB group than the No-RBBB group.

Credit Author Statement

All authors substantially contributed to the work and met the authorship criteria as follows: Masamichi Yano: Conceptualization, Methodology, Investigation, Writing-Original Draft, Ryu Shutta, Koji Yasumoto: Methodology, Kohei Ukita: Investigation, Akito Kawamura: Investigation, Hitoshi Nakamura: Investigation, Naotaka Okamoto, Akihiro Tanaka: Investigation, Yasuharu Matsunaga-Lee: Conceptualization, Yutaka Matsuhiro, Masaki Tsuda: Visualization, Yasuyuki Egami: Supervision, Masami Nishino: Supervision, Jun Tanouchi: Supervision.

Declaration of Interests

The authors declare that they have no known competing financial interests or personal relations that could have appeared to influence the work reported in this paper.

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Disclosures

None

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Supplementary materials

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