Catheter Ablation of Atrial Fibrillation in Patients With Chronic Lung Disease

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Background—Chronic lung disease (CLD) is one of the important underlying diseases of atrial fibrillation (AF). The outcomes after radiofrequency catheter ablation of AF in patients with CLD have not yet been reported. We investigated the electroanatomic alterations in pulmonary veins (PVs) in CLD patients with AF and assessed their effect on the outcomes of radiofrequency catheter ablation of AF.

Method and Results—We assessed 15 patients who had CLD and underwent radiofrequency catheter ablation of AF. CLD included chronic obstructive pulmonary disease, a tuberculosis-destroyed lung, and interstitial lung disease. For controls, we selected 60 sex-, age-, and procedure era–matched non-CLD patients who received radiofrequency catheter ablation for AF (4 controls for each CLD patient). Eight patients had chronic obstructive pulmonary disease, 6 had a tuberculosis-destroyed lung, and 1 had interstitial lung disease. PV morphology in the affected lung was altered significantly, ie, obliteration, pulling of the PVs toward the destroyed lung, or compensatory bulging of the PV antrum. These alterations were related to arrhythmogenicity in 6 (40%) of 15 patients with CLD. Non-PV foci were more common in the CLD group (4/15, 26.7%) than in the control group (3/60, 5.0%; P = 0.025). All non-PV foci were located in the right atrium. The AF recurrence rate in the CLD group (26.7%, 4/15) was similar to that in the control group (18.3%, 11/60; P = 0.45).

Conclusions—Significant alteration of PV anatomy was related to arrhythmogenicity, and non-PV foci from the right atrium were commonly observed in the CLD group. Radiofrequency catheter ablation can be performed safely for AF in CLD patients with a comparable success rate to that in patients with normal lungs. (Circ Arrhythm Electrophysiol. 2011;4:815-822.)

Key Words: ablation | atrial fibrillation | lung | catheter ablation | pulmonary heart disease

Cardiac arrhythmias, including atrial fibrillation (AF), occur frequently in patients with chronic lung disease (CLD). In addition, changes in pulmonary vascular structure and hemodynamics can result from chronic hypoxia in lung diseases such as chronic obstructive pulmonary disease (COPD) or can be caused by the destruction of lung parenchyma by the chronic inflammation that characterizes diseases such as tuberculosis and interstitial lung disease (ILD). These changes in pulmonary vascular structure and hemodynamics may constitute the basis for AF perpetuation or the triggers that initiate AF. Furthermore, antiarrhythmic drugs used to control AF, such as sotalol and propafenone, have the potential to provoke bronchial spasm and decrease lung function. One of the detrimental side effects of amiodarone is pulmonary fibrosis. Accordingly, pharmacological treatment of AF in patients with CLD requires special caution and is of limited value.

Recently, radiofrequency catheter ablation (RFCA) has been demonstrated to be an effective therapy for various subsets of AF; however, the outcomes for RFCA of AF in patients with CLD have not yet been reported. The purpose of the present study, therefore, was to investigate anatomic alterations in the pulmonary veins (PVs) of CLD patients with AF and to compare the clinical outcomes of RFCA for AF between CLD and control patients.

Methods

Patients

A total of 752 consecutive patients who underwent RFCA for paroxysmal or persistent AF between April 2000 and May 2009 were
reviewed. Patient history and chest radiographic findings were reviewed retrospectively, and 15 patients with chronic lung disease, including COPD (n=8), tuberculosis-destructive lung (TDL, n=6), and ILD (n=1), were included in the present study. COPD was defined according to the GOLD (Global Initiative for Chronic Obstructive Lung Disease) criteria: Forced expiratory volume in 1 second/forced vital capacity <0.70 in a pulmonary function test, or emphysematous changes on chest radiography with a smoking history of more than 20 pack-years or medical treatment history. Patients with a destroyed lung or lung involvement greater than 30% of the total lung volume, as observed on chest radiography and chest computed tomography (CT) images, were defined as having TDL or ILD (Figure 1). For each CLD patient, we selected 4 control patients who were matched for sex, age (±2 years), and procedure era (±1 year). After reviewing the chest radiographs and clinical history of all patients, we selected patients who did not have lung disease to be in the control group. In total, the control group included 60 patients without CLD who underwent AF ablation.

Imaging Studies
All PV angiographic CT and 3-dimensional (3D) reconstruction images were performed within 24 hours before RFCA in all patients (Figure 1). The 3D PV CT image was merged with 3D left atrial (LA)-PV geometry acquired by use of a multielectrode catheter on NavX (St Jude Medical, Inc). Transthoracic echocardiography and transesophageal echocardiography were performed before RFCA in all AF patients. We also assessed whether a thrombus was present without CLD who underwent AF ablation.

Catheter Ablation Procedure
In the case of 1 patient who was treated by catheter ablation in 2000, we used focal ablation at the PV, guided by a Lasso catheter (Biosense Webster Inc). From 2005 to 2007, we used noncontact mapping–guided 4-PV ablation and non-PV foci ablation for 2 patients. Starting in 2007, we performed circumferential ablation of 4 PVs guided by NavX (St.Jude Medical, Inc, St. Paul, MN) with PV isolation for 12 patients. Since 2008, we added multiple linear ablations at the LA roof and perimtrial isthmus and also used 3D automated complex fractionated atrial electrogram mapping–guided ablation for 3 patients with sustained AF after PV isolation (PVI). Multipolar catheters were positioned at the His recording area, the right atrial (RA) lateral wall, and the coronary sinus. Double transseptal punctures and multiview pulmonary venuograms were performed. We then inserted a quadripolar catheter into the ascending aorta as a reference catheter for the NavX mapping system and mapped the LA geometry.

Before PVI, we tried to identify arrhythmogenic foci if the ectopic foci spontaneously initiated paroxysmal AF. In patients with a sinus rhythm at baseline, AF was induced by burst pacing at the high RA and coronary sinus from 250 ms to the atrial refractory period under the effect of isoproterenol (10 μg/min). Internal DC cardioversion was subsequently performed with a decapolar catheter in the high RA as an anode, with another decapolar catheter inside the coronary sinus acting as a cathode. The arrhythmogenic foci were identified if they caused immediate reinitiation of AF within 2 minutes after cardioversion. We then investigated the relationship between the arrhythmogenic foci and anatomic distortion of the PVs. After PVI or multiple linear ablations at the LA, we repeated the same protocol at least 3 times to identify consistent non-PV foci–initiated AF. We applied the same protocol in patients with persistent AF to localize arrhythmogenic foci.

The end point of PVI was elimination of PV potentials on the 10-bipole ring-shaped catheter at each PV. PVI was not performed at the obliterated PVs in which the PV potential was not recorded (Figure 2). Efficient localization of non-PV foci at the specific areas

Figure 1. Chest radiographs and pulmonary vein computed tomography 3-dimensional reconstruction images of the representative patients in the chronic lung disease group. A and B, Images of the patients with a lung destroyed by tuberculosis. C and D, Images of patients with chronic obstructive pulmonary disease (COPD). A red dot indicates the site of arrhythmogenic focus-initiated atrial fibrillation in each patient.
in the RA was achieved by keeping a decapolar catheter at the RA that covered the area from the RA septum to the high crista terminalis and another duo-decapolar catheter at the RA that covered high and low crista terminalis and the cavotricuspid isthmus, including the coronary sinus ostium. A quadripolar catheter was inserted into the superior vena cava (SVC; Figure 2C). Then, we performed an internal DC cardioversion of the induced AF during infusion and washout of the isoproterenol and waited for 2 minutes to define immediate reinitiation of AF.

If AF was sustained after PVI, 3D automated complex fractionated atrial electrograms were mapped during 6 seconds of AF with NavX software. Complex fractionated atrial electrogram map settings were a refractory period of 49 ms, P-P sensitivity >0.1 mV, and duration of 30 ms. Additional ablation was performed guided by the complex fractionated atrial electrogram map until AF was terminated or organized to an atrial tachycardia or the fractionated activity was eliminated.

We documented the occurrence of postprocedure complications, including bleeding or cardiac tamponade, in both groups. In patients with CLD, respiratory-related complications related to deep sedation, hypercapnia, or respiratory suppression during or after the procedures were carefully monitored while sedation was maintained or when inhalation of a low to high content of oxygen was continued. We also measured and compared the procedure time, oxygen saturation, and drug dosage of thiopental sodium, midazolam, and fentanyl to assess safety in both groups. Bleeding events, including pulmonary hemorrhage that occurred during or after anticoagulation, were compared between the 2 groups.

Follow-Up
Patients were followed up by means of clinical assessment, ECG, ambulatory ECG monitoring, and event-triggered monitoring. Initial postablation follow-up took place at 1 and 3 months and every 6 months thereafter. Beyond this interval, outpatient clinic visits every 6 months with 24 to 48 hours of Holter monitoring were scheduled. All patients were instructed to maintain their personal records with descriptions of every episode of symptomatic palpitations and, in the case of persistent arrhythmia episodes, to obtain ECG documentation of the underlying rhythm. A successful outcome over the follow-up period was defined as the lack of electrocardiographically recorded supraventricular tachycardia or AF, and no paroxysmal AF (duration >30 seconds) on Holter monitoring. We compared AF recurrence rate, the interval to the recurrence of AF, and the rate of taking antiarrhythmic drugs after ablation in both groups.

Statistical Analysis
Variables were expressed as mean±SD or number and percentages, as appropriate. To account for our matched sampling design, continuous variables were compared with a linear mixed-effects regression model in which the group variable (ie, the CLD or control groups) was considered as a fixed effect and the subject identifica-
Table 1. Clinical Characteristics of Patients With Atrial Fibrillation in CLD

<table>
<thead>
<tr>
<th>Control (n=60)</th>
<th>CLD (n=15)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female, n</td>
<td>52/8</td>
<td>13/2</td>
</tr>
<tr>
<td>Age, y</td>
<td>60.2±10.6</td>
<td>60.2±11.0</td>
</tr>
<tr>
<td>Paroxysmal</td>
<td>40 (66)</td>
<td>10 (66)</td>
</tr>
<tr>
<td>Persistent</td>
<td>20 (33)</td>
<td>5 (33)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>8 (13)</td>
<td>2 (13)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2 (3)</td>
<td>2 (13)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>2 (3)</td>
<td>1 (7)</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>56.8±5.4</td>
<td>54.6±5.5</td>
</tr>
<tr>
<td>LA diameter/BSA, mm/m²</td>
<td>23.7±4.9</td>
<td>23.0±6.2</td>
</tr>
<tr>
<td>LA volume/BSA, mL/m²</td>
<td>33.6±12.5</td>
<td>35.9±16.1</td>
</tr>
<tr>
<td>E/E</td>
<td>9.6±5.3</td>
<td>10.2±4.2</td>
</tr>
<tr>
<td>PA systolic pressure, mm Hg</td>
<td>31.6±4.8</td>
<td>28.4±8.9</td>
</tr>
<tr>
<td>Na, mmol/L</td>
<td>140.5±5.2</td>
<td>140.4±2.4</td>
</tr>
<tr>
<td>K, mmol/L</td>
<td>4.2±0.4</td>
<td>4.1±0.4</td>
</tr>
</tbody>
</table>

CLD indicates chronic lung disease; LVEF, left ventricular ejection fraction; LA, left atrium; BSA, body surface area; PA, pulmonary artery; and Hb, hemoglobin.

Data are presented as mean±SD or n (%).

*Calculated by linear mixed model.

Results

The clinical characteristics of the CLD and control groups are compared in Table 1. No significant differences were noted between the 2 groups in terms of echocardiographic parameters of the LA or laboratory findings before the procedure, including hemoglobin, electrolyte, and transaminase. No significant difference was observed in the mean LA diameter/body surface area between the 2 groups (23.7±4.9 mm/m² in the control group versus 23.0±6.2 mm/m² in the CLD group, P=0.51). The mean pulmonary arterial systolic pressure measured by pulsed-wave Doppler was significantly higher in the CLD group (35.4±8.9 versus 31.6±4.8 mm Hg, P=0.04). The clinical characteristics of the 15 patients in the CLD group are summarized in Table 2. The mean age was 60.2±11.0 years, and 13 patients (86%) were male. Ten of the 15 patients had paroxysmal AF, whereas 5 patients had persistent AF.

Electrophysiological Characteristics of Atrial Fibrillation in Patients With CLD

Figure 1 shows 4 representative chest radiographs and 3D PV CT images. Figure 1A shows the chest radiograph and CT of patient 6 (Table 2). The chest radiograph (posteroanterior view) shows a destroyed left lung, and the CT image shows obliteration of the left superior PV (LSPV) and distal tributaries of the left inferior PV. The right PVs of the corresponding lung were normal. The obliterated LSPV had residual electric PV potentials, which functioned as the arrhythmogenic focus initiating AF in this patient. Figure 1B shows the results for patient 11 (Table 2). The chest radiograph shows a destroyed left lung and left-deviated major bronchi. The corresponding left PVs were obliterated on CT, whereas the right PVs were normal, with increased vascularity of the distal tributaries. The obliterated left PVs did not exhibit PV potentials on the ring-shaped catheter. The right superior PV had an arrhythmogenic focus in this patient; therefore, PVI was performed only at the right PVs (Figure 2). Figure 1C

Table 2. Baseline Characteristics of Each Patient in the Chronic Lung Disease Group

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Age, y</th>
<th>Type of AF</th>
<th>Lung Disease</th>
<th>Other Diseases</th>
<th>FVC</th>
<th>FEV₁</th>
<th>FEV₁/FVC</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Female</td>
<td>70</td>
<td>Paroxysmal</td>
<td>TDL</td>
<td>...</td>
<td>86</td>
<td>107</td>
<td>1.24</td>
</tr>
<tr>
<td>2</td>
<td>Male</td>
<td>56</td>
<td>Paroxysmal</td>
<td>COPD</td>
<td>Old MI</td>
<td>116</td>
<td>62</td>
<td>0.53</td>
</tr>
<tr>
<td>3</td>
<td>Female</td>
<td>65</td>
<td>Paroxysmal</td>
<td>COPD</td>
<td>...</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>4</td>
<td>Male</td>
<td>42</td>
<td>Paroxysmal</td>
<td>TDL</td>
<td>...</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>5</td>
<td>Male</td>
<td>71</td>
<td>Paroxysmal</td>
<td>COPD</td>
<td>...</td>
<td>89</td>
<td>65</td>
<td>0.73</td>
</tr>
<tr>
<td>6</td>
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<td>49</td>
<td>Paroxysmal</td>
<td>TDL</td>
<td>...</td>
<td>37</td>
<td>39</td>
<td>1.05</td>
</tr>
<tr>
<td>7</td>
<td>Male</td>
<td>75</td>
<td>Paroxysmal</td>
<td>ILD</td>
<td>...</td>
<td>76</td>
<td>87</td>
<td>1.14</td>
</tr>
<tr>
<td>8</td>
<td>Male</td>
<td>67</td>
<td>Paroxysmal</td>
<td>COPD</td>
<td>MR</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>9</td>
<td>Male</td>
<td>57</td>
<td>Paroxysmal</td>
<td>COPD</td>
<td>HTN, DM</td>
<td>92</td>
<td>58</td>
<td>0.63</td>
</tr>
<tr>
<td>10</td>
<td>Male</td>
<td>42</td>
<td>Paroxysmal</td>
<td>COPD</td>
<td>...</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>11</td>
<td>Male</td>
<td>49</td>
<td>Persistent</td>
<td>TDL</td>
<td>...</td>
<td>73</td>
<td>66</td>
<td>0.90</td>
</tr>
<tr>
<td>12</td>
<td>Male</td>
<td>75</td>
<td>Persistent</td>
<td>COPD</td>
<td>...</td>
<td>92</td>
<td>69</td>
<td>0.75</td>
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<tr>
<td>13</td>
<td>Male</td>
<td>67</td>
<td>Persistent</td>
<td>TDL</td>
<td>HCMP</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<tr>
<td>14</td>
<td>Male</td>
<td>57</td>
<td>Persistent</td>
<td>COPD</td>
<td>DM</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<tr>
<td>15</td>
<td>Male</td>
<td>63</td>
<td>Persistent</td>
<td>TDL</td>
<td>...</td>
<td>86</td>
<td>80</td>
<td>0.93</td>
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</tbody>
</table>

AF indicates atrial fibrillation; FVC, forced vital capacity; FEV₁, forced expiratory volume in the first second of expiration; TDL, tuberculosis destructive lung; COPD, chronic obstructive pulmonary disease; MI, myocardial infarction; NA, not available; ILD, interstitial lung disease; MR, mitral regurgitation; HTN, hypertension; DM, diabetes mellitus; and HCMP, hypertrophic cardiomyopathy.
shows the images of patient 9. The posteroanterior chest radiograph revealed an emphysematous lung, and CT revealed a dilated LSPV ostium. Both upper PVs were pulled down by overinflated emphysematous lung, forming a “V” shape on the roof of the LA body. An arrhythmogenic focus was identified at the dilated LSPV in this patient. Figure 1D shows the images for COPD patient 8. A bulging and dilated LSPV was seen on CT, and the right inferior PV originated from the left common pulmonary trunk. The bulged LSPV was arrhythmogenic.

As illustrated in the representative examples, the PV morphology was altered in the affected lungs, eg, they bulged, shrank, or were obliterated. The significant alterations in PV morphology were related to arrhythmogenicity; for example, all 3 COPD patients had arrhythmogenic foci from bulged PVs. In patients with TDL, 2 shrunken PVs that had clear PV potentials were arrhythmogenic; however, 1 of the obliterated PVs without PV potential was not arrhythmogenic, whereas the opposite site of the PVs showing compensatory enlargement functioned as trigger sites for AF (Figure 2).

Thus, we concluded that severely modified and bulged PVs were arrhythmogenic as long as the PV potential was existed, whereas obliterated PVs without PV potential were not arrhythmogenic. Sixty percent of patients (9 of 15) demonstrated arrhythmogenic foci before ablation, whereas PVs were arrhythmogenic in only 40% of patients (6 of 15). Moreover, the number of CLD patients was too small to determine whether anatomic alterations in the PVs were truly responsible for the initiation and maintenance of AF. We failed to engage the catheter into 1 right inferior PV, in which the ostium was acutely angulated, so we ablated the whole circumference of both PV antra under guidance by a deflectable size-adjustable Lasso catheter.

Non-PV foci were provoked in 4 (26.7%) of 15 patients with CLD after PVI, for which the incidence was higher than that in the control group (3/60, 5.0%; P = 0.025). Interestingly, non-PV foci in all patients were exclusively from the RA, including the coronary sinus, the SVC, the low crista terminalis, and the neck of the RA appendage. Patient 2 showed atrial tachycardia, the earliest activation site of which was the high RA near the SVC and RA junction. We mapped the SVC with a Lasso catheter and performed the SVC isolation by segmental ablation 1 to 2 cm above the junction between the SVC and RA. In patient 10, we performed linear ablation on the cavitricuspid isthmus and confirmed the bidirectional block across it; thereafter, tachycardia was no longer inducible. In patient 12, atrial flutter after PVI was induced and sustained, which was terminated and no longer induced after linear ablation at the crista terminalis. In patient 13, the earliest activation site of the atrial tachycardia was low crista terminalis, which was targeted and terminated during ablation.

### Effect of Catheter Ablation on Symptoms and Outcome

Many symptoms of CLD and AF are often confused, and patients cannot clearly distinguish them. We performed catheter ablation for the patients who complained of symptoms after sufficient medical treatment of CLD and for those who had symptoms that worsened without aggravating factors of CLD, for example, superimposed infection. We also investigated the correlation of AF with symptoms recorded on Holter monitoring.

Symptom status before and after ablation is illustrated in Table 3. The rate of relief was high for patients who complained of palpitation or fluttering (8 of 11) or syncope (2 of 2) as the main symptom, but dyspnea (2 of 4) and chest pain (3 of 3) persisted after the procedure. On the contrary, dyspnea or shortness of breath was attenuated or relieved in 82% (49/60) of control group patients. The rate of symptom relief was clearly different between the paroxysmal AF group (8/10, 80%) and persistent AF group (1/5, 20%). We believe
that distinguishing between symptoms is more difficult in patients with persistent AF than in patients with paroxysmal AF.

The mean total procedure time for RFCA was similar in both groups (253±94 minutes in CLD versus 255±77 minutes in the control group, \(P=0.98\)). The doses of sedative drugs required to maintain sedation without respiratory suppression in the CLD group were not significantly different from those in the control group (thiopental sodium 1569±597 versus 1327±374 mg, \(P=0.01\); midazolam 6.1±4.5 versus 5.9±2.8 mg, \(P=0.83\); fentanyl 59±77 versus 84±51 mg, \(P=0.15\)). In the present study, ablation of AF in CLD patients was performed safely with no clinical signs of oversedation or respiratory suppression due to hypercapnia. During sedation, supplemental oxygen (0–3 L/min) was used to maintain optimal \(O_2\) saturation (92% to 100%).

We applied the same anticoagulation strategy in CLD patients as in the control subjects. Differences between the 2 groups in terms of the rate of complications after anticoagulation or the incidence of thrombosis in CLD patients were not evident. We performed postprocedure chest radiographs to observe potential pulmonary hemorrhage or congestion, but there were no pulmonary parenchymal changes in any of the CLD patients.

The rate of antiarrhythmic drug use was not significantly different between the 2 groups (44.1% in the control group versus 46.7% in the CLD group, \(P=0.85\)). We performed a follow-up CT at 7 months after the first procedure in only 1 patient (Figure 3). On preprocedure CT, we found LSPV enlargement and a relatively shrunken left inferior PV. On follow-up CT, bulging was attenuated after circumferential antral ablation. Unfortunately, follow-up CT data were not available for all patients; however, none of the patients complained of symptoms suggestive of PV stenosis, such as aggravation of dyspnea, hemoptysis, chest pain, or pneumonia-like infiltration on chest radiograph.

During an average follow-up of 46.6±25.5 months, the AF recurrence rate in the CLD group (26.7%, 4/15) was somewhat higher than that in the control group (18.3%, 11/60), but this difference was not statistically significant (\(P=0.45\)). In 9 patients who were followed up for more than 3 years, the recurrence rate of the CLD group was higher than that of the control group (4/9 [44%] versus 11/36 [31%], \(P=0.45\)). We analyzed the recurrence using the Kaplan-Meier method, as shown in Figure 4. The recurrence rate of the CLD group was higher than that of the control group in the first 46 months of follow-up. Thereafter, there was not a significant difference in recurrence rate between the 2 groups (\(P=0.49\)). Nevertheless, the last 2 recurrent events in the CLD group occurred at 15 months and 109 months, whereas in the control group, the last event was at 47 months. This meant the 2 survival curves crossed over each other at the later follow-up periods. The nonsignificance of recurrence rates between groups was due in part to those events, so that a definite interpretation of the test result should not be made until more data are available.

**Discussion**

The major findings of the present study are that (1) AF patients with CLD have significant alterations in PV anatomy
due to lesions in the affected lung, and these morphological alterations are closely related to AF arrhythmogenicity; (2) non-PV foci in the RA are observed more often in patients with CLD than in those with normal lungs; and (3) RFCA can be performed safely for AF in CLD patients with a comparable success rate to that in patients with normal lungs.

PV Anatomic Variations in Patients With AF

Several reports have indicated that specific anatomic and morphological features of PVs, such as a common ostium or bulging of the PV, are associated with AF induction. A few studies have reported that a unique PV morphology is related to the occurrence of AF, and therefore, the ablation strategy needs to be customized according to the type of PV anatomic alteration. Hof et al reported that the most commonly observed PV variation in patients with lone AF was a right-sided middle PV in 23%, followed by a common left trunk in 16%. They found, however, that PV anatomy did not have any effect on the outcome of RFCA, for AF because PV variations were equally distributed among the different ablation procedures, and therefore, PV anatomy was not a predictor of outcome.

The presence of a common inferior trunk, either individually or combined with the left superior PV, is an uncommon anatomic variety of PV, as shown in the present study. In a larger patient series, the incidence of a common inferior trunk was 0.9% in patients with AF, which was the most infrequent anomaly. The present study confirms the importance of assessing PV anatomy in patients with CLD before ablation. Identification of alterations in PV anatomy and coexisting tachyarrhythmias allows for the tailoring of ablation strategies, which may minimize unnecessary ablation and result in a reduction in procedure times and procedure-related complications. 3D imaging before ablation was certainly required in these patients.

Pulmonary Hypertension and PV Morphology

CLD causes hypoxemia and acidosis, and these lead to increased pulmonary vascular resistance. Increased levels of inflammatory factors such as angiotensin, prostacyclin, endothelin, and serotonin also induce pulmonary arterial and venous hypertension. These inflammatory factors may also become triggers of fibrosis and cause structural remodeling of pulmonary vessels. In the present study, pulmonary arterial pressure was higher in the CLD group than in the control group (35.4±8.9 versus 31.6±4.8 mm Hg, P=0.04). This hemodynamic factor may contribute to altering the morphology of the LA and PV structures and subsequently lead to AF.

Non-PV Foci in Patients With CLD

Non-PV foci initiate AF with an incidence ranging from 3.2% to 55%. Non-PV foci are known to be associated with a higher recurrence of AF after RFCA. Identification of non-PV foci in AF ablation is therefore important to increase the long-term efficacy of catheter ablation. In the present study, the incidence of non-PV foci-initiated AF in CLD patients was higher than that in the control group. Interestingly, all non-PV foci originated from the RA side. Hemodynamic overloading or stretching of the RA side due to pulmonary hypertension may contribute to a higher prevalence of non-PV foci from the RA in CLD patients. It is not yet clear whether the high prevalence of AF in patients with CLD is directly attributable to the higher incidence of non-PV foci in these patients. In the present study, most AFs in CLD patients were successfully treated by PV isolation in conjunction with ablation of non-PV foci.

Ablation Strategy

The specific characteristics of AF have been noted in CLD patients, but in the present study, the AF recurrence rate and postablation antiarrhythmic drug use rate in the CLD group were not significantly different from those in the control group. This indicates that RFCA is as effective in the CLD group as it is in the control group. However, anatomic alterations of PVs require additional consideration when ablation is performed. In CLD patients with an obliterated PV, special caution is needed to avoid stenosis of the ipsilateral or contralateral sites of normal PVs by more proximal ablation at the PV antrum rather than ostial ablation. Furthermore, early identification and effective elimination of non-PV foci may increase the efficacy of RFCA for AF in these patients.

Study Limitations

In the present study, the number of CLD patients with AF was too small to draw definitive conclusions. Fifteen (2.0%) of 752 consecutive patients who underwent RFCA were found to have CLD. Four (50%) of 8 COPD patients underwent a pulmonary function test within at least 6 months before RFCA, which fulfilled the GOLD criteria, but the other 4 patients were defined as having COPD on the basis of significant emphysematous changes on chest radiography with a long-standing history of heavy smoking and a treatment history of oral bronchodilators. Assessment of the relationship between LA-PV electroanatomic alteration and arrhythmogenicity was limited because only 60% of the patients demonstrated spontaneous or induced arrhythmogenic foci before ablation.

Conclusions

Significant alterations in PV anatomy provide proarrhythmia grounds for AF in CLD patients. Non-PV foci in the RA were observed more frequently in the CLD group than in the control group. RFCA can be performed safely for AF in CLD patients with a comparable success rate to that in patients with normal lungs.

Disclosures

None.

References


The therapeutic options for patients with atrial fibrillation (AF) who have underlying chronic lung disease (CLD) have been known to be limited. CLD includes chronic obstructive pulmonary disease, a tuberculosis-destroyed lung, and interstitial lung disease. Patients who complained of symptoms after optimal medical treatment of CLD or those whose symptoms that worsened despite the absence of aggravating factors of CLD were selected for catheter ablation of AF in the present study, because we believed elimination of AF would result in relief of symptoms attributable to AF and yield a better clinical outcome in this subset of CLD patients. Most symptoms, including palpitation, fluttering, or syncope, were relieved after successful ablation of AF; and this was demonstrated more clearly in the group with paroxysmal AF. The AF-free rate was comparable to that in AF patients with normal lungs. From the ablation experience in these CLD patients, we also found that significant alterations in pulmonary vein anatomy provided proarrhythmic grounds associated with the perpetuation of atrial fibrillation. Circ Arrhythm Electrophysiol. 2010;3:39-45.
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