Original Article
Left atrial pressure, proBNP and hsCRP during periablation period in atrial fibrillation patients

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Abstract: The clinical impact of left atrial pressure (LAP) in patients with atrial fibrillation (AF) undergoing successful radiofrequency catheter ablation remains largely unknown. In this study, we explored the impact of LAP on the incidence of post-cardiac ablation syndrome in 116 patients with paroxysmal (group P, n = 72) or persistent (group N, n = 44) AF. Patients in both groups received either pulmonary vein antrum isolation or stepwise ablation. LAP was measured before ablation and immediately after ablation. Post-cardiac ablation syndrome were collected and pro-BNP, hsCRP were detected respectively. Post-cardiac ablation syndrome was diagnosed in 14 patients (2 in group P, 12 in group N). Group N had a higher incidence rate (27.3% vs. 2.8%, P = 0.001). There is a significant difference of LAP in both groups, whether before ablation (12.4 ± 4.8 mmHg vs. 16.7 ± 4.1 mmHg; P = 0.044) or after ablation (17.5 ± 6.7 mmHg vs. 23.6 ± 3.3 mmHg; P = 0.042). Compared with pre-ablation, the post-ablation LAP had significant increase in both group P (P = 0.001) and N (P = 0.01). After ablation, in patients with the post-cardiac ablation syndrome, the level of ΔLAP was higher than that in patients without the post-cardiac ablation syndrome (8.4 ± 3.7 mmHg vs. 4.2 ± 2.7 mmHg, P = 0.003). The level of proBNP, hsCRP and WBC increased gradually during first several days after the ablation treatment, and then declined. In conclusions, AF patients with increased post-ablation left atrial pressure, paired with increased proBNP and hsCRP were more likely to have a post-cardiac ablation syndrome.

Keywords: Atrial fibrillation, left atrial pressure, catheter ablation, post-cardiac ablation syndrome

Introduction

Curative catheter ablation for atrial fibrillation (AF) has become an effective therapeutic option for patients with drug-refractory symptomatic AF. Radio frequency catheter ablation (RFCA) strategies for paroxysmal AF were mainly focused on isolating the pulmonary venous (PVs) antrum from the rest of the left atrium (LA). Meanwhile, persistent AF seems to be less dependent on the pulmonary venous antrum for both initiation and perpetuation [1], therefore a stepwise approach to ablate persistent AF is highly effective for maintaining sinus rhythm [2]. However, when following a cardiac ablation procedure, clinical outcomes such as fever, pericarditis and pericardial effusion were reported [3, 4]. These adverse clinical outcomes caused by these ablation techniques may lead to life-threatening complications such as atrio-esophageal fistula and congestive heart failure (CHF) [5].

Left atrial pressure (LAP), an intra-procedural parameter, could be easily obtained after transseptal puncture during ablation period of AF. Elevated level of LAP indicates a stretched left atrium and pulmonary, which is considered as one of the determinant factors in the development and maintenance of AF. Atrial diseases in which LA compliance is markedly reduced are associated with impaired LA filling while increase in diastolic stiffness resulting in increased LAP can maintain stroke volume in the absence of atrial booster pump function [6].

However, the interaction of such extensive ablation and the changing level of LAP and their relative impact on clinical outcomes are not clear-cut. The aim of this study was to determine whether LA ablation was associated with the changing level of LAP, and how these factors might affect clinical outcomes after catheter ablation of AF.

Materials and methods

Study subjects

We conducted a prospective, longitudinal observation study to examine the trend of change in LAP level, LA remodeling, and the incidence rate of post-cardiac ablation syndrome among AF patients having undergone RFCA.

Patients with symptomatic AF referring for catheter ablation at our centre between October 2013 and March 2014 were considered in this study.

Patients were excluded if they had one or more of the following conditions: hyperthyroidism, moderate or severe valvular disease, pregnancy, ongoing infections, intracardiac thrombosis, contra indications to anticoagulation, history of myocardial infarction or unstable angina pectoris within the last 3 months, life-expectancy < 6 months, repeat ablation and refusal to give written informed consent. Patients with LA diameter > 5.5 cm, advanced heart failure (left ventricular ejection fraction < 50%), abnormal blood pressure peri-procedure and cardiac tamponade were also excluded. Before ablation, patients with an obscured costophrenic angle or pericardial effusion were also excluded.

In this study, all eligible patients were divided into two groups: P group and N group. P group contains all patients with paroxysmal AF who underwent pulmonary venous antrum isolation (PVAI) ablation, and N group contains all non-paroxysmal AF patients who underwent step-wise ablation.

The type of AF was defined according to the guidelines suggested by the American College of Cardiology [7]. Paroxysmal atrial fibrillation was defined as attacks of arrhythmia lasting < 7 days and separated by periods of normal sinus rhythm. The study complies with the Declaration of Helsinki and was performed according to the recommendations of the hospital's Ethics Committee including written informed consent.

Pre-ablation treatment

Oral anticoagulation (target international normalized ratio, INR, 2~3) was given for at least 1 month before admission and was discontinued at least 5 days prior to the ablation. Weight-adjusted low molecular weight heparin (LMWH) was given on the day of intervention treatment. The use of antiarrhythmic drugs (AAD) such as propafenone was continued during peri-procedural period and last for three months after the procedure. On admission, a medical history check, a physical examination, a 12-lead ECG exam, an upright chest x-ray, a transesophageal echocardiogram, and left atrial computed tomography and standard laboratory measurements were obtained or completed. Systemic inflammatory response (SIR) parameters [white blood count (WBC) and high sensitive C-reactive protein (hsCRP)], prohormone of brain natriuretic peptide (proBNP), serum cardiac troponin I (cTnI) and echocardiography, an upright chest x-ray were detected at 1, 3, 5, 7 day after ablation. Before the procedure, blood pressure (BP) and blood sugar were monitored and controlled to normal levels, heart function to I or II grade (NYHA classification).

When pharmacologic antihypertensive therapy was necessary, intravenous drip nitroglycerin may be administered to acute perioperative blood pressure elevation. Meanwhile vasopressors (e.g. dopamine) were administered to perioperative hypotension.

Electrophysiological study

The electrophysiologic (EP) study was performed in the fasting conscious state. Electrograms were displayed and recorded at filter settings of 30 to 500 Hz in a specialized EP recording system (EP Med, MN, US). Vascular access was obtained through the right femoral and internal jugular veins. A steerable decapolar catheter (Biosense Webster, Diamond Bar, CA, USA) was positioned in the coronary sinus (CS) for mapping and pacing. After two transseptal punctures with a BRK™ needle (St Jude Medical, USA), systemic anticoagulation was achieved with intravenous heparin to maintain an activated clotting time of 300~350 s. A 7 Fr catheter with 3.5-mm deflectable irrigated tip (Navistar Thermocool, Biosense Webster, USA) and a Lasso catheter (Biosense Webster, USA) were introduced into the LA for mapping and ablation.

The LA and PVs were re-constructed in CARTO 3 system, a computerized 3-dimentional electro-anatomical mapping system (Biosense Webster, Inc., USA).

Pulmonary venous antrum isolation: We used the Lasso catheter for mapping the LA and pulmonary veins. The irrigated ablation catheter was used to perform ablation outside the antrum of pulmonary veins in order to isolate electrograms emit from the inside.

Stepwise ablation approach: The stepwise ablation strategy consisted of PVAI and linear ablation following PVAI. Linear ablation was performed at the LA roof and mitral isthmus [8]. If AF persisted after linear ablation, ablation of CFAEs was performed in the LA [9]. Favorable areas in the LA are continuous electrograms with complex fractionated potentials that are noted at first sight, especially when the AF cycle length (CL) is around 140~150 ms [10].

If AF persisted after these steps, ablation was performed in the right atrium targeting sites exhibiting CFAEs, at the discretion of the physician [11]. The end point of the ablation procedure was the termination of AF or completion of the ablation strategy within a time limit of 5.5 hours. When AF was converted to atrial tachycardia (AT), the tachycardia was mapped and ablated with an approach described in detail elsewhere [12].

Internal electrical cardioversion was used to restore sinus rhythm if AF or AT could not be terminated by radiofrequency ablation. RF energy was delivered at 30~35 W, a maximum flow rate of 30 ml/min, and a maximum temperature of 43°C. The power was reduced to 20 W during RF energy delivery within the CS.

Internal cardioversion

Briefly, it was performed under sedation, in the fasting state. One electrode served as the cathode was positioned in the right atrium so that the electrodes had contact with the anterolateral wall. The other electrode as the anode was positioned in the LA. One shock was given at each voltage, R wave was synchronized by a right ventricular bipolar lead. The defibrillation protocol included a single conversion and an initial test shock of 5 V, the energy was then increased in 5-V steps until restoration of sinus rhythm.

LAP measurement

A 5F multipurpose angiography catheter (MPA) was used for pressure measurement. A contin-uous pressure monitoring system (MacLab: GE Healthcare, Horten, Norway) was connected to the lumen to allow for pressure waveform analysis. Specifically, a 3-way Manifold (Navylist Medical, CA) was attached via a Touchy (Merit Medical, IRE) to the MPA. The three ports of the manifold were saline flush, contrast and pressure monitoring. The pressure monitoring port was then connected to a standard cardiac catheterization laboratory pressure transducer. The pressure waveforms were displayed on the EP recording system live monitor.

The pressure recordings were set at a scale of 25 mmHg and sweep speed at 25 mm/sec. PV pressure (PVP) was measured at 1 cm in PV from PV ostia. LAP was measured at central section in LA. Removal of air bubbles from the system was critical and care was taken to thoroughly flush the system prior to utilization. Thereafter, pressure monitoring sensor was zeroed, calibrated. The timings of measurement were respectively preprocedure, and the half hour post procedure. Five consecutive beats were averaged at the end of expiration. The initial rhythm when measured LAP, whether sinus rhythm (SR) or AF, was recorded. We analyzed LAP_{mean} $[LAP_{nadiri}+1/2(LAP_{peak}-LAP_{nadiri})]$ [13, 14].

Post-ablation care

After the procedure, all patients were continuously monitored and received intravenous heparin for 24 hours, warfarin was resumed and subcutaneous LMWH was administered until the INR is ≥ 2.0. Warfarin was continued for at least 3 months. Patients were discharged 10 days after the procedure. Clinical follow-up was performed at the first, third, sixth and twelfth month. Long term antiarrhythmic and anticoagulation therapy was determined by clinical outcomes. Postcardiac ablation clinical outcome was evaluated according to the symptoms, signs and assistant examination.

Post cardiac ablation syndrome

Post cardiac ablation syndrome was characterized by features of low-grade fever, pericarditis/pericardial effusion, pleuritis/pleural effusion following a cardial ablation [15, 16]. Symptoms maybe short of breath or chest distress. In the evaluation of pleural effusion, obtuse angulation was a sign in an upright x-ray. Echocardiography was the imaging modality of choice for the diagnosis of pericardial effusion.

Table 1. Baseline clinical characteristics ($\bar{x} \pm$ SD) (n = 116)

Items	P group (n = 72)	N group (n = 44)	P value
Age (years)	62.6 ± 9.5	61.2 ± 9.9	P = 0.273
Male (n = 68)	43 (59.7%)	25 (56.8%)	P = 0.758
AF history (months)	54.2 ± 55.8	37.8 ± 38.6	P = 0.298
Hypertension (n = 80) (%)	49 (68.1%)	31 (70.5%)	P = 0.786
Ischemic stroke (n = 12) (%)	5 (6.9%)	7 (15.9%)	P = 0.124
Diabetes mellitus (n = 14) (%)	7 (9.7%)	7 (15.9%)	P = 0.321
Dyslipidemia (n = 17) (%)	9 (12.5%)	8 (18.2%)	P = 0.401
ProBNP (pg/mL)	387.4 ± 658.9	864.2 ± 947.5	P = 0.001
hsCRP (mg/L)	6.1 ± 8.2	5.4 ± 7.4	P = 0.595
cTnI (ng/mL)	0.02 ± 0.10	0.01 ± 0.01	P = 0.285
WBC	5.8 ± 1.2	5.4 ± 0.8	P = 0.545
Left atrial diameter (mm)	37.5 ± 4.8	42.4 ± 4.5	P = 0.001
LAWT (mm)	5.8 ± 1.3	4.9 ± 0.6	P = 0.375
LVEF (%)	64.9 ± 5.0	63.9 ± 6.3	P = 0.254

Abbreviations: AF, atrial fibrillation; proBNP, brain natriuretic peptide; hsCRP, high sensitive C-reactive protein; cTnI, cardiac troponin I; WBC, white blood cell; LAWT, left atrial anterior wall thickness; LVEF, left ventricular ejection fraction.

Echocardiography protocol

Transthoracic echocardiography was performed with an available echocardiography system (Philips SONOS iE33, Aliso Viejo, USA) with a 1.0~5.0 MHz phased-array transducer by a sonographer credentialed in cardiac ultrasound. Two-dimensional imaging was performed from standard parasternal and apical transducer positions with frame rates of 60~100 frames per second from left lateral decubitus position. The measurement should be done at end-systole, on the frame just before the opening of the mitral valve. The LA shape should be roughly square. LA wall thickness was measured at four planes. The measured sites were selected at the posterior wall, the anterior wall (LAWT), the lateral wall and the roof wall.

All data were stored digitally, and offline data analysis was performed by 2 cardiologists blinded to study time point.

Statistical analysis

The study data were analyzed by using PASW Statistics 18 (IBM, USA). An independent sample t-test was used to analyze the difference between the two groups in SIR levels, LAP, LAWT, LVEF, Left atrial diameter. The difference in LAP between preablation and postablation

was analyzed with a paired sample t-test. Preablation and postablation (1 d, 3 d, 5 d, 7 d) SIR levels and LAWT were analyzed with ANOVA. Categorical variables such as gender, early recurrence, incidence of post cardiac ablation syndrome were presented as proportions and were compared by use of the chi-square test. Relationship between variables was checked by Pearson correlation test. All tests were two-tailed. P value was accepted as significant when it was equal or below 0.05.

Results

Fifty-eight of the 176 patients were excluded for the study when exclusion criteria were applied. Consequently, 118 patients were registered in the AF ablation cohort. Two of these patients were excluded from the analysis because of cardiac tamponade (1 in each group). Finally, 116 patients with either paroxysmal (n = 72) or non-paroxysmal (n = 44) AF were analyzed. Patients with paroxysmal AF had a mean of 19 episodes of AF per month.

Baseline characteristics

Baseline characteristics of the final study patients were presented in **Table 1**. The mean age was 62.2 ± 9.6 years, and 68 patients (58.6%) were male. Patients with hypertension accounted for 68.9% (80 patients) of the whole population. Ischemic stroke, diabetes mellitus, and dyslipidemia were found in 12 patients (10.3%), 14 patients (12.1%), 17 patients (14.7%) respectively. At the preprocedure assessment, all participants had normal blood pressure. The difference in all demographic and baseline characteristics between two groups were not statistically significant.

Procedure parameters

Table 2 summarizes the procedural parameters. PVAI in this series was performed with a mean procedure time of 173.9 ± 38.6 min, and fluoroscopy time of 27.6 ± 11.4 min in group P. We achieved stepwise ablation in group N with

Table 2. Ablation and clinical outcome ($\bar{x} \pm$ SD) (n = 116)

Items	P group (n = 72)	N group (n = 44)	P value
Procedure time (min)	173.9 ± 38.6	213.9 ± 47.9	0.000
Ablation time (min)	94.6 ± 28.2	148.3 ± 34.7	0.008
Fluoroscopic time (min)	27.6 ± 11.4	38.2 ± 15.9	0.000
Post cardiac ablation syndrome, n (%)	2 (2.8)	12 (27.3)	0.000
ΔLeft atrial diameter (mm)	2.48 ± 9.56	7.51 ± 16.66	0.263
ΔLAWT (mm)	1.11 ± 0.66	0.55 ± 0.21	0.291
ΔProBNP (pg/mL)	-132.9 ± 909.1	392.4 ± 786.6	0.107
ΔHsCRP (mg/L)	22.6 ± 20.1	44.2 ± 56.2	0.107
ΔcTnI (ng/L)	2.0 ± 1.48	2.3 ± 2.20	0.833
Early recurrence, n (%)	5 (6.9)	11 (25.0)	0.006

Abbreviations: LAWT, left atrial anterior wall thickness; LAP, left atrial pressure; proBNP, brain natriuretic peptide; hsCRP, high sensitive C-reactive protein; cTnI, cardiac troponin I; BP, blood pressure.

Table 3. The change of LAP during ablation period ($\bar{x} \pm$ SD) (n = 116)

Items		Preablation	Postablation	P value
LAP_{mean} (mmHg)	P group (n = 72)	12.4 ± 4.8	17.5 ± 6.7	0.000
	N group (n = 44)	16.7 ± 4.1	23.6 ± 3.3	0.01
P value		0.044	0.042	-

Abbreviations: LAP, left atrial pressure.

Table 4. The change of LAP during ablation period across post-cardiac ablation syndrome ($\bar{x} \pm$ SD)

Items		PCAS +(n = 14)	PCAS -(n = 102)	P value
LAP_{mean} (mmHg)	Preablation	14.9 ± 3.7	12.6 ± 5.2	0.263
	Postablation	23.3 ± 4.5	16.9 ± 6.5	0.02
ΔLAP (mmHg)		8.4 ± 3.7	4.2 ± 2.7	0.003

Abbreviations: LAP, left atrial pressure; PCAS, post-cardiac ablation syndrome.

a mean procedure time of 213.9 ± 47.9 min and fluoroscopy time of 38.2 ± 15.9 min. The total procedure time and radiofrequency energy delivery time (ablation time) were significantly shorter in P group than those in N group (all P < 0.01).

Clinical outcome

During ablation period, only two patients, 1 in each group, had cardiac tamponade and need pericardiocentesis and drainage. After ablation, pericardial effusion was found in 7 patients, pleural effusion was found in 3 patients, and 4 patients had both pericardial and pleural effusion. All of these 14 patients were diagnosed with post-cardiac ablation syn-

drome. Comparisons between group P and group N showed that the latter had higher incidence rate of post-cardiac ablation syndrome (2.8% vs. 27.3%, P = 0.001) or early recurrence rate of atrial flutter/fibrillation (6.9% vs. 25.0%, P = 0.006).

LAP

In P group, the initial rhythm was SR before ablation in 51 patients, AF in 21 patients. At baseline, there was a significant difference in LAP_{mean} (10.6 ± 3.4 mmHg vs. 16.2 ± 5.3 mmHg; P = 0.002) between SR patients and AF patients. After ablation, the LAP_{mean} in AF patients was higher than that in SR patients (15.1 ± 5.3 mmHg vs. 23.3 ± 6.5 mmHg; P = 0.007).

In N group, the LAP_{mean} was higher than that in P group at baseline (16.7 ± 4.1 mmHg vs. 12.4 ± 4.8 mmHg; P = 0.044) or postablation (23.6 ± 3.3 mmHg vs. 17.5 ± 6.7 mmHg; P = 0.042).

Compared with preablation, the postablation LAP_{mean} had increased significantly in group P (P = 0.001) and in group N (P = 0.01) (**Table 3**).

These results suggested that the group with higher LAP_{mean} was more likely to have persistent AF, longer and more extensive LA ablation, and a greater LA dimension.

To examine the potential association between post-cardiac ablation syndrome and biochemical examination, we dichotomized the patients across the post-cardiac ablation syndrome and found significant difference in the change of LAP (ΔLAP). In patients with the post-cardiac ablation syndrome, the level of ΔLAP was higher than that in patients without the post-cardi-
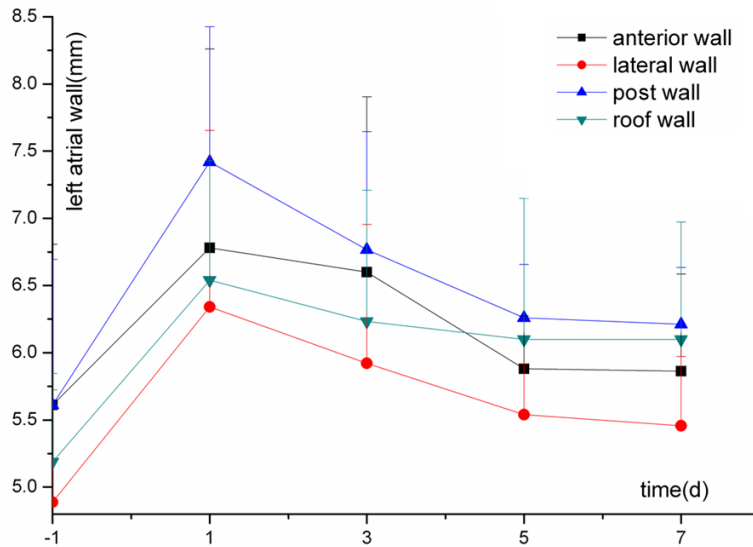
Figure 1. The curve of left atrial wall thickness over the first 7 d of follow-up.
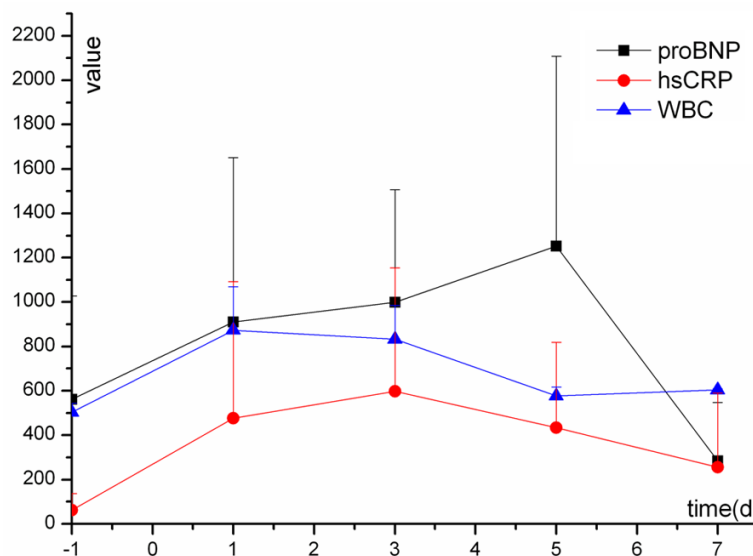

Figure 2. The curve of systemic inflammatory response in patients with the post-cardiac ablation syndrome over the first 7 d of follow-up.

ac ablation syndrome (8.4 ± 3.7 mmHg vs. 4.2 ± 2.7 mmHg, *P* = 0.003) (**Table 4**).

Left atrial wall thickness

At baseline, the mean LA diameter (anteroposterior) and LAWT measured by echocardiography was 38.9 ± 5.2, 5.6 ± 1.2 mm respectively. The LA diameter in N groups was larger than in group P (42.4 ± 4.5 vs. 37.5 ± 4.8 mm, *P* < *0.01*) (**Table 1**).

The measurements showed considerable difference in thickness of LA wall at different lev-

els (**Figure 1**). In general, the lateral wall was found to be the thinnest among all measured sites. After ablation, the LA wall thickness increased first, then decreased at all two measured planes, which displayed a changing trend.

Biochemical examination results

At baseline, there were no significant differences in WBC counts and hsCRP values between two groups (*P* > 0.05). Following ablation, the level of both values increased gradually in both groups. The levels increased to their peak level in a few days before it began to decline. Baseline proBNP level was significantly higher in group N patients compared with group P patients (*P* = 0.001) (**Table 1**). Following ablation, the proB-NP level decreased at first in both groups, and then increased significantly, but in the end returned to baseline values after several days. There were no statistically differences in the perioperative change of the three between two groups (*P* > 0.05).

To examine the potential association between post-cardiac ablation syndrome and biochemical examination, we dichotomized the patients across the post-cardiac ablation syndrome and found significant difference in proBNP, but no difference in hsCRP and WBC level (**Figures 2**, **3**).

Discussion

Discovery

In this prospective trial, we found that the postablation LAP increased significantly compared to preablation. The patients with higher postablation LAP were more prone to have post-cardiac ablation syndrome. In patients with the post-
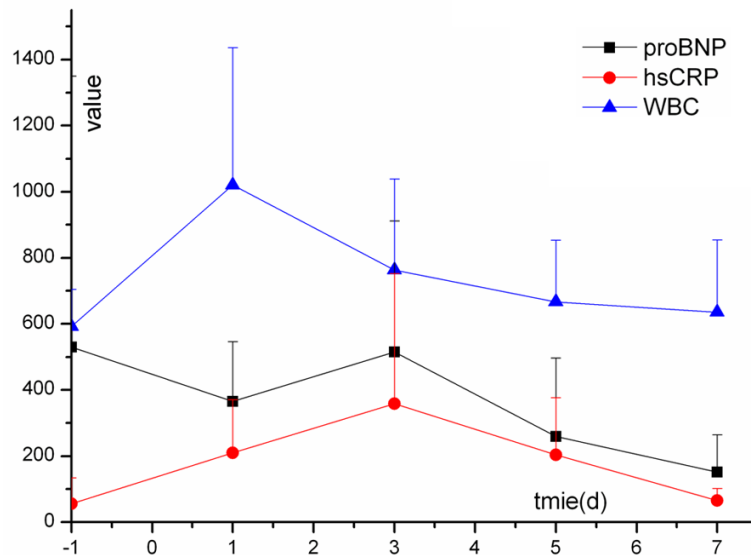
Figure 3. The curve of systemic inflammatory response in patients without the post-cardiac ablation syndrome over the first 7 d of follow-up.

cardiac ablation syndrome, the level of proBNP, hsCRP and WBC increased gradually during first several days and can be used as the predictor of the prognosis of post-cardiac ablation syndrome when the three increased simultaneously.

Possible mechanisms

The increase of postablation LAP maybe caused by the left atrial wall edema and left atrial systolic dysfunction caused by the ablation: In 2006, Steel et al. firstly reported that one patient with AF underwent severe left atrial wall edema and heart failure after catheter ablation [5]. They thought that mechanical stunning attributable to atrial edema also caused increased LAP, even severe CHF [5]. In 2007, another study reported that the severity of the LA edema might be associated with the difference in the amount of damaged myocardium resulting from the different PV ablation techniques [15].

In recent years, the extensive PV ablation has achieved better clinical results than the PVI techniques. It is considered that such extensive ablation is required in patients with persistent AF as a result of adverse structural and electrical remodeling.

Previous studies have suggested that LA enlargement and scarring [17, 18] and shorter

AF cycle length are associated with AF recurrence [16]. Diastolic stiffness caused by LA enlargement and scarring can increase LAP. ProBNP is proposed for evaluating and monitoring heart pathologies characterized by myocardial wall stress [19].

Present study show that, after ablation, the ejection fraction detected by transthoracic echocardiography was normal, but cardiac atrium contractile function was lost when Doppler mitral inflow wave A disappeared. Left atrial wall annular edema shown by echocardiography was obvious. The left atrial wall thickness increased to 7.4 mm from preoperative 5.1 mm. The proBNP level increased 547 pg/mL in patients who undergone stepwise ablation.

The postablation inflammation contributes significantly to the post-cardiac ablation syndrome: Catheter ablation could induce a moderate SIR in patients with AF [20]. Wood et al. suggested pericarditis was caused by the extensive transmural atrial or pericardial injury from the linear LA lesions [21].

Okada et al. reported that the edematous changes in the LA wall often extended to regions remote from the PVs, where the RF ablation was not applied [15]. The mechanism is still unclear. However, it may be related to post injury inflammation.

In present study, peak point values of SIR (WBC and hsCRP) occurred at 48 vs. 72 h after RFCA, pro-BNP and the LAWT reached a peak, and then declined over time after ablation. All these support that induced inflammation lead to the post-cardiac ablation syndrome.

Clinical implications

This study identified that increased postablation LAP, paired with increased proBNP and hsCRP during periablation period, as a novel modifiable risk factor for AF and post-cardiac ablation syndrome. It can be used to identify

"high-risk" patients. Further studies are necessary to determine whether targeting of LAP with anti-hyperpressure medications may modify the burden or progression of AF and reduce the incidence of post-cardiac ablation syndrome.

Study limitations

In this study, only the left atrial pressure and pulmonary venous pressure were measured. The level of proBNP was also investigated. The pressure was only measured by the large end. In addition, the study could be improved by a longer study period, a larger sample size, and a more extended follow-up period.

Conclusions

After ablation, the patients with increased postablation LAP, accompanied by increased proBNP and hsCRP were more likely to have post-cardiac ablation syndrome, and the three indicators could be used as the predictors of the prognosis.

Acknowledgements

Disclosure of conflict of interest

None.

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