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Inflammation response post-radiofrequency ablation for atrial fibrillation: implications for early atrial fibrillation recurrence

Running title: Inflammatory predictors of early AF recurrence

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WHAT'S NEW?

In our study, the extent of the inflammatory response escalation after radiofrequency ablation serves as a predictive indicator for early atrial fibrillation recurrence. Furthermore, high expression of prothrombotic markers at 1 week after radiofrequency ablation for atrial fibrillation might explain the increased risk of early thromboembolic events. The magnitude and specific alterations in postoperative elevation of inflammation subsequent to radiofrequency ablation for atrial fibrillation was investigated with an aim to help guide the timing and regimen of postoperative interventions to prevent recurrence of atrial fibrillation.

ABSTRACT

Background: Inflammation is associated with early recurrence after atrial fibrillation (AF) ablation and is linked to stroke in AF.

Aims: The aim of this study is to investigate the time course of inflammatory biomarkers following radiofrequency ablation (RFA) for AF and its relation to early AF recurrence.

Methods: Ninety patients who underwent successful RFA for AF were enrolled. Blood samples were collected from the median cubital vein preoperatively and on postoperative days 1, 2, 3, 7, and 14 to measure levels of high-sensitivity C-reactive protein (hs-CRP), fibrinogen, creatine kinase isoenzyme (CKMB), and cardiac troponin I (cTnI). Follow-up evaluations of AF recurrence were conducted at weeks 1, 2, 3, 4 and months 2 and 3 postoperatively.

Results: The hs-CRP levels significantly increased on postoperative days 2 and 3 compared to preoperative values, followed by a decline on days 7 and 14 (P <0.05). Fibrinogen level peaked between postoperative days 2 and 7 (P <0.05), and then declined by day 14. CKMB and cTnI levels peaked between postoperative days 1 and 3 (P <0.05). The escalation of hs-CRP following RFA was identified as an independent risk factor for early AF recurrence (OR, 2.948; 95% CI, 1.892–4.602; P = 0.004).

Conclusions: The degree of inflammatory response following RFA serves as a predictive marker for early AF recurrence, and the significant inflammatory response and hypercoagulable state are evident within the first week post-RFA for AF, potentially explaining the heightened risk of early thromboembolic events.

Key words: atrial fibrillation, inflammation, myocardial injury, radiofrequency catheter ablation, recurrence of atrial fibrillation

INTRODUCTION

Atrial fibrillation (AF) is a prevalent form of rapid cardiac arrhythmia encountered in clinical practice, associated with heart failure, an increased risk of stroke, and heightened mortality and disability rates [1]. Radiofrequency ablation (RFA) is a crucial therapeutic approach used to restore sinus rhythm and manage AF [2]. Despite its benefits, this technique is often challenged by a high incidence of postoperative recurrence. Although early AF recurrence does not fully reflect the procedural success,

it is a significant predictor of late recurrence, which is critical for guiding clinical treatment strategies for AF [3, 4]. Therefore, attention must be directed towards this “blank period” following RFA for AF.

Recent studies have demonstrated a correlation between inflammatory responses and early recurrence following catheter ablation for AF [5]. However, the relationship between preoperative and postoperative inflammatory responses and both early and late recurrence following ablation has produced inconsistent results [6–8]. Detailed research on the underlying mechanisms has shown that postoperative inflammation plays a significant role in the initiation and progression of AF [9]. Patients with AF undergoing ablation are at an increased risk of thromboembolic events, particularly within the first two weeks post-procedure, although the precise mechanisms remain unclear [10].

In this study, we aimed to investigate the specific temporal profile of inflammatory response following ablation, examining its relationship with prothrombotic risk and AF recurrence. By utilizing blood biomarkers as therapeutic targets, we seek to mitigate the inflammatory response after ablation, thereby providing theoretical evidence for evaluating operation outcomes and reducing early recurrences.

MATERIAL AND METHODS

General information

In this prospective study, 98 patients aged less than 75 years with AF were consecutively enrolled between March 2015 and May 2016. All patients successfully underwent a circumferential pulmonary vein isolation (CPVI) procedure. The exclusion criteria were acute or chronic infections, left ventricular dysfunction, left atrial diameter greater than 50 mm, atrial thrombosis, valvular heart disease, hyperthyroidism, history of AF ablation, previous prosthetic heart valve replacement, malignant tumors, blood disorders, contraindications to anticoagulation therapy, or inability to adhere to anticoagulant medication. This study was approved by the local ethics committee (Approval number: 2016122).

Method

Operation procedure

Prior to the procedure, transesophageal echocardiography was performed to exclude left atrium thrombus. All antiarrhythmic agents were discontinued for five half-lives before the procedure. All patients underwent CPVI treatment following a standardized

surgical protocol, without any additional ablation beyond the pulmonary veins unless they had been diagnosed with atrial flutter prior to the operation. A computed tomography scan of the left atrium and pulmonary veins was conducted preoperatively to assess their size and morphology.

During the procedure, under three-dimensional anatomical mapping (Carto 3, Biosense Webster), a pressure-infused ablation catheter was inserted through the interatrial septum into the left atrium for RFA. A circular mapping catheter (Lasso® NAV eco, Biosense Webster) was utilized before, during, and after CPVI treatment to record pulmonary vein potentials. The standard approach involved creating an encircling lesion around two adjacent ipsilateral pulmonary veins as the endpoint for successful ablation. Following the achievement of entrance and exit block in pulmonary vein potentials, a monitoring period of 20 to 30 minutes was implemented before reassessing conduction status. If recovery was observed during this period, the ablation procedure was continued. If sinus rhythm did not resume despite achieving entrance block, unidirectional cardioversion was performed using a 360 J shock.

Sample collection

Blood samples were collected from the median cubital vein preoperatively and on postoperative days 1, 2, 3, 7, and 14. High-sensitivity C-reactive protein (hs-CRP), fibrinogen, creatine kinase-MB (CKMB), and troponin-I levels in peripheral blood were measured at each time point and were promptly analyzed. Hs-CRP was quantified using an immunoturbidimetric latex CRP assay, fibrinogen levels were determined using the STAR coagulation analyzer, CKMB concentrations were assessed using an immunoinhibition method, and troponin-I measurements relied on chemiluminescent immunoassay. The normal reference ranges for these biomarkers are: hs-CRP 0–0.08 mg/l, fibrinogen 2.38–4.98 g/l, CKMB 0–24 U/l, and troponin-I 0–0.04 ng/ml. The extent of biomarker elevation is defined as the maximum value observed (between day 1 and day 14) minus the baseline value recorded preoperatively, such as hs-CRP elevation, fibrinogen elevation, CKMB elevation, and troponin-I elevation.

Postoperative care and follow-up

All patients received standard postoperative care, including immobilization, fluid therapy, and continuous cardiac monitoring for three days. Outpatient follow-up visits were scheduled at weeks 1, 2, 3, and 4 post-operation, and at months 2 and 3. Weeks

1–4 postoperation suggest four times of follow-up in the first month. After 1 month post-procedure, follow-up was conducted twice a month, and patients underwent electrocardiogram (ECG) examinations and 48-hour ambulatory ECG recordings-to-assess arrhythmia recurrence. AF recurrence was defined as any episode of atrial arrhythmia lasting ≥30 seconds. Early recurrence was defined as any episode of atrial arrhythmia lasting ≥30 seconds within 3 months post-ablation based on ECG examination and ambulatory ECG recordings. Anticoagulation therapy with oral anticoagulant medication was continued for at least three months post-ablation. For patients with a $CHADS_2$ score ≤1 and no recurrence, oral anticoagulant medication was discontinued. The $CHADS_2$ scoring system includes congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, and previous stroke/transient ischemic attack, and is used to predict stroke risk in patients with non-valvular AF. For patients with a CHADS2 score >1, anticoagulation therapy was continued for 12 months before considering discontinuation based on mutual agreement with the patient.

Statistical analysis

Data analysis was performed using SPSS 13.0 statistical software. Normally distributed continuous variables are presented as mean (standard deviation). Continuous variables that were not normally distributed are expressed as median (interquartile range). Categorical data are displayed as frequencies and percentages. The χ^2 test or Fisher's exact test was used to analyze categorical data. A significance level of P <0.05 was considered statistically significant. Intra-group indicators following ablation were compared using a repeated measures mixed linear model. Logistic regression analysis models assessed the impact of various risk factors on early recurrence post-ablation. The Enter method in single-factor logistic regression analysis was utilized to calculate unadjusted odds ratios (OR) for initial screening of risk factors associated with early recurrence. Significant variables identified in the single-factor analysis, along with clinically relevant variables believed to impact early recurrence, were incorporated in the multiple-factor logistic regression analysis model to determine the risk factors for early recurrence.

RESULTS

General characteristics of baseline data

The baseline characteristics of the patients and procedural characteristics are

summarized in Table 1. The CHADS2 score was 1 (0–1). Among the 98 patients initially screened. Two patients met the exclusion criteria (Figure 1). At least 90 patients were examined, six of whom failed to appear for more than one visit or Holter recording and were excluded from this study. The follow-up period was 3 months, during which 35 patients experienced recurrence, resulting in an early recurrence rate of 38.9%. Among the patients, 53 (58%) had paroxysmal AF, and 37 (42%) had persistent AF.

Changes in inflammatory biomarkers at different time points following RFA for AF

hs-CRP

Following RFA for AF, hs-CRP levels significantly increased over time compared to preoperative levels. The hs-CRP levels were significantly higher on postoperative days 1, 2, 3, 7, and 14 (P <0.05), with peak levels observed on postoperative days 2 and 3 (P <0.05). Subsequently, hs-CRP levels decreased but remained slightly elevated compared to preoperative levels on postoperative days 7 and 14 (Figure 2A).

Fibrinogen

Fibrinogen levels showed significant variations pre- and post-RFA on postoperative days 1, 2, 3, 7, and 14 (P <0.05). There was a significant increase in fibrinogen levels on postoperative days 2, 3, and 7 compared to preoperative levels (P <0.05), with a peak between days 3 and 7. By day 14, fibrinogen levels had returned to preoperative levels (P >0.05) (Figure 2B).

CKMB

CKMB levels significantly increased on the first postoperative day, reaching a peak (P <0.05). Levels then decreased from the second to the third postoperative day and returned to preoperative levels by day 3 (P >0.05). This decreasing trend continued through the seventh and fourteenth days (Figure 2C).

Troponin-I

Troponin-I levels significantly increased on the first postoperative day, reaching their highest level. Levels gradually decreased during follow-up visits but remained higher than preoperative levels on the second and third postoperative days (P <0.05). By the seventh day, levels had returned to preoperative values (P >0.05), and a further reduction was noted by the fourteenth day (Figure 2D).

Follow-up and recurrence of AF

During the three-month follow-up period, patients attended outpatient appointments at specified intervals, including postoperative weeks 1, 2, 3, and 4, and at the second and third months. Of the 90 patients who underwent RFA for AF, 35 experienced early recurrence, resulting in an early recurrence rate of 38.9%. Univariate logistic regression analysis results indicated difference in age, hypertension, electric cardioversion and RF ablation time in relation to early recurrence of AF (P >0.05). However, elevations in inflammatory biomarkers such as CKMB, troponin-I, fibrinogen, and hs-CRP were identified as risk factors for early recurrence. The extent of elevation in CKMB (OR, 1.207; 95% CI, 1.020–1.428; P = 0.029), troponin-I (OR, 2.834; 95% CI. 1.116–7.196; P = 0.028), fibrinogen (OR, 3.564; 95% CI, 1.090–11.654; P = 0.035), and hs-CRP (OR, 2.864; 95% CI, 1.493–5.493; P = 0.002) showed a positive correlation with early recurrence of AF. Multivariable logistic regression analysis revealed that only the degree of elevation in hs-CRP was an independent predictor of early recurrence following RFA for AF (OR, 2.948; 95% CI, 1.892–4.602; P = 0.004) (Table 2).

Post-procedure complications

All patients recovered following treatment, with no withdrawals from the study. Two patients developed sinus bradycardia and required pacing. Four patients experienced mild fluid overload, necessitating diuretic therapy.

DISCUSSION

The efficacy of RFA in the treatment of AF is widely recognized. However, postoperative recurrence remains a significant clinical concern [11]. Several factors have been identified as being associated with the recurrence of AF following RFA, including left atrial diameter, body mass index, and inflammatory biomarkers. Specifically, there has been an increasing focus on inflammatory biomarkers. Most arrhythmias that occur within three months following ablation for AF can resolve spontaneously, likely due to procedure-induced inflammation [12]. Early recurrence of AF serves as a reliable predictor for late recurrence and is one of the most significant predictive factors [3, 4]. In our study, we applied a three-month post-ablation blanking period, though the recent expert consensus statement has shortened this period to two months [13]. Early recurrence may be attributed to secondary inflammation subsequent

to the ablation procedure but does not necessarily indicate treatment failure. Evaluation of operation outcomes should be conducted once inflammation has subsided. The aim of this study was to investigate the relationship between inflammatory changes following RFA for AF and early recurrence.

In this study, the levels of CKMB and troponin-I were observed to increase and reach their peak on the first day following ablation for AF. Subsequently, they exhibited a gradual decline after three days, returning to preoperative levels within the first week. These findings suggest mild myocardial injury subsequent to the procedure, attributed to cardiac tissue damage during ablation rather than myocardial ischemia [14]. Lim et al. [15] reported that troponin-I level peaked on the first day following RFA for AF and continued to rise until the third postoperative day, while CKMB levels also peaked on the first day, consistent with our findings. Furthermore, the levels of CK-MB and potentially troponin I might be influenced to some extent by cardioversion. The magnitude of elevation in cardiac muscle proteins may be associated with the intensity of radiofrequency pulses delivered during the operation and the ablation site.

Our study revealed that hs-CRP levels peaked on the second- and third-day following ablation and remained elevated for 14 days before gradually declining. Previous studies have demonstrated that localized myocardial tissue damage and edema occur following ablation for AF [16]. This results in an acute inflammatory response characterized by increased white blood cell count, hs-CRP levels, coagulation biomarkers, and other markers. Additionally, early post-ablation inflammation induces a prothrombotic state in patients undergoing ablation for AF within two weeks postoperatively, significantly contributing to complications such as thromboembolism associated with AF [17]. The results of this study revealed a notable elevation in fibrinogen levels from the second or third day post-operation, peaking on the third and seventh days, and persisting until the seventh day compared to preoperative levels. This phase of heightened coagulation marker expression coincides with most thromboembolic complications arising within two weeks following RFA, potentially explaining the increased risk of early thromboembolic events. Therefore, monitoring the timing and degree of anticoagulation after ablation for AF may effectively prevent thromboembolic events and reduce anticoagulant costs and bleeding risk.

Emerging studies have demonstrated a close correlation between oxidative stress, inflammatory reactions, and the occurrence, progression, and maintenance of AF. Frustaci et al. [18] observed lymphocyte infiltration in atrial myocardium biopsies from

patients with paroxysmal AF, accompanied by surrounding necrotic cardiac tissue, which provided pathological evidence supporting the presence of inflammation in AF development. Yuksel et al. [19] enrolled 427 patients undergoing isolated off-pump coronary artery bypass grafting in this retrospective observational cohort study and for the first time demonstrated that multi-inflammatory indices predicted new-onset AF after off-pump coronary artery bypass grafting. The mechanisms underlying AF recurrence following RFA are complicated and not fully elucidated. Previous studies have indicated that preoperative baseline hs-CRP levels independently predict AF recurrence following RFA [20]. This study revealed that the extent of postoperative rise in hs-CRP levels (defined as the difference between maximum postoperative value and preoperative level) is an independent risk factor for early AF recurrence. Koyama et al. [21] found that a higher inflammatory response following ablation for AF is associated with a greater rate of early AF recurrence, consistent with our findings. However, there is controversy regarding factors influencing early recurrences following ablation for AF. Richter et al. [22] found that recurrences within 48 hours post ablation cannot reliably predict late recurrences.

Studies have demonstrated that the use of corticosteroids and anti-inflammatory medications post-catheter ablation for AF can mitigate inflammation, thereby reducing the occurrence of early postoperative arrhythmias [23, 24]. Colchicine, a medication with anti-inflammatory, immunosuppressive, and antifibrotic properties, is primarily used to treat acute attacks of arthritic gout and has anti-carcinogenic properties. It exerts a potent cardioprotective effect by reducing the incidence of cardiovascular diseases through its anti-inflammatory action, such as minimizing in-stent restenosis in coronary arteries and decreasing the likelihood of pericardial effusion and pleural effusion following cardiac operations. Studies have shown that oral administration of colchicine for three months following RFA for AF reduces early recurrence rates [23]. Additionally, a mere three-day regimen of corticosteroid use following RFA for AF not only reduces early recurrences but also lowers the incidence of midterm arrhythmias [24]. However, an experimental animal study found that prophylactic steroids did not alter the systemic inflammatory response or lesion healing in pigs undergoing atrial ablation [25]. Furthermore, numerous studies have confirmed that inflammation is a significant contributing factor in the development of AF [9, 26].

Limitations

A limitation of this study is follow-up and recurrence of AF-the probing frequency following RFA might be low, failing to show the peak of fibrinogen levels. Thus, the extent of this biomarker may be underestimated. Inflammation presents a novel target for clinical treatment strategies aimed at combating AF development [27]. Further research is warranted to elucidate the complex mechanisms linking inflammation with AF [28].

CONCLUSION

In this study, the extent of the inflammatory response escalation after RFA serves as a predictive indicator for early atrial fibrillation recurrence. The results of our study revealed that high expression of prothrombotic markers one-week following RFA for AF which may explain the increased risk of early thromboembolic events seen in other studies, which can guide the timing and degree of anticoagulation to effectively prevent thromboembolic events while reducing anticoagulant costs and bleeding risk. The results may assist in determining the optimal timing and regimen of postoperative interventions to prevent the AF recurrence. They may also help mitigate the inflammatory response after AF ablation, providing theoretical evidence for evaluating procedural outcomes and reducing early recurrences.

Article information

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REFERENCES

1. Trohman RG, Huang HD, Sharma PS. Atrial fibrillation: Primary prevention, secondary prevention, and prevention of thromboembolic complications: part 1. Front

Cardiovasc Med. 2023; 10: 1060030, doi: 10.3389/fcvm.2023.1060030, indexed in Pubmed: 37396596.
2. Hussain S, Sohrabi C, Providencia R, et al. Catheter ablation for the management of atrial fibrillation: An update of the literature. Life (Basel). 2023; 13(8): 1784, doi: 10.3390/life13081784, indexed in Pubmed: 37629641.
3. Liu J, Fang PH, Liu Y, et al. Relationship between occurrence of atrial arrhythmia within three months after atrial fibrillation ablation and long-term success rate. Chin J Cardiac Pacing Electrophysiol. 2011; 25(2): 115–118, doi: 10.13333/j.cnki.cjcpe.2011.02.011.
4. Wu Na, Xu B, Xiang Y, et al. Association of inflammatory factors with occurrence and recurrence of atrial fibrillation: A meta-analysis. Int J Cardiol. 2013; 169(1): 62–72, doi: 10.1016/j.ijcard.2013.08.078, indexed in Pubmed: 24095158.
5. Richter B, Gwechenberger M, Socas A, et al. Markers of oxidative stress after ablation of atrial fibrillation are associated with inflammation, delivered radiofrequency energy and early recurrence of atrial fibrillation. Clin Res Cardiol. 2012; 101(3): 217–225, doi: 10.1007/s00392-011-0383-3, indexed in Pubmed: 22102100.
6. Lellouche N, Sacher F, Wright M, et al. Usefulness of C-reactive protein in predicting early and late recurrences after atrial fibrillation ablation. Europace. 2009; 11(5): 662–664, doi: 10.1093/europace/eup077, indexed in Pubmed: 19351632.
7. Carballo D, Noble S, Carballo S, et al. Biomarkers and arrhythmia recurrence following radiofrequency ablation of atrial fibrillation. J Int Med Res. 2018; 46(12): 5183–5194, doi: 10.1177/0300060518793807, indexed in Pubmed: 30178684.
8. Budzianowski J, Hiczkiewicz J, Burchardt P, et al. Predictors of atrial fibrillation early recurrence following cryoballoon ablation of pulmonary veins using statistical assessment and machine learning algorithms. Heart Vessels. 2019; 34(2): 352–359, doi: 10.1007/s00380-018-1244-z, indexed in Pubmed: 30140958.
9. Issac TT, Dokainish H, Lakkis NM. Role of inflammation in initiation and perpetuation of atrial fibrillation: a systematic review of the published data. J Am Coll Cardiol. 2007; 50(21): 2021–2028, doi: 10.1016/j.jacc.2007.06.054, indexed in Pubmed: 18021867.
10. Oral H, Chugh A, Ozaydin M, et al. Risk of thromboembolic events after percutaneous left atrial radiofrequency ablation of atrial fibrillation. Circulation. 2006; 114(8): 759–765, doi: 10.1161/CIRCULATIONAHA.106.641225, indexed in Pubmed: 16908760.
11. Tomaiko E, Su WW. Comparing radiofrequency and cryoballoon technology for the ablation of atrial fibrillation. Curr Opin Cardiol. 2019; 34(1): 1–5, doi: 10.1097/HCO.0000000000000578, indexed in Pubmed: 30444763.
12. Kong XQ, Yu XD, Li WH, et al. Relationship between inflammatory factors and matrix metalloproteinases and early recurrence of atrial fibrillation after catheter ablation. J Clin Intern Med. 2018; 12(35): 848–849.
13. Tzeis S, Gerstenfeld EP, Kalman J, et al. 2024 European Heart Rhythm Association/Heart Rhythm Society/Asia Pacific Heart Rhythm Society/Latin American Heart Rhythm Society expert consensus statement on catheter and surgical ablation of atrial fibrillation. Europace. 2024; 26(4): euae043, doi: 10.1093/europace/euae043, indexed in Pubmed: 38587017.
14. Schmidt M, Marschang H, Clifford S, et al. Trends in inflammatory biomarkers during atrial fibrillation ablation across different catheter ablation strategies. Int J Cardiol. 2012; 158(1): 33–38, doi: 10.1016/j.ijcard.2010.12.084, indexed in Pubmed: 21276621.
15. Lim HS, Schultz C, Dang J, et al. Time course of inflammation, myocardial injury, and prothrombotic response after radiofrequency catheter ablation for atrial fibrillation. Circ Arrhythm Electrophysiol. 2014; 7(1): 83–89, doi: 10.1161/CIRCEP.113.000876, indexed in Pubmed: 24446024.

16. Yano M, Egami Y, Yanagawa K, et al. Comparison of myocardial injury and inflammation after pulmonary vein isolation for paroxysmal atrial fibrillation between radiofrequency catheter ablation and cryoballoon ablation. J Cardiovasc Electrophysiol. 2020; 31(6): 1315–1322, doi: 10.1111/jce.14475, indexed in Pubmed: 32250506.
17. Oral H, Chugh A, Ozaydin M, et al. Risk of thromboembolic events after percutaneous left atrial radiofrequency ablation of atrial fibrillation. Circulation. 2006; 114(8): 759–765, doi: 10.1161/CIRCULATIONAHA.106.641225, indexed in Pubmed: 16908760.
18. Frustaci A, Chimenti C, Bellocci F, et al. Histological substrate of atrial biopsies in patients with lone atrial fibrillation. Circulation. 1997; 96(4): 1180–1184, doi: 10.1161/01.cir.96.4.1180, indexed in Pubmed: 9286947.
19. Yuksel A, Velioglu Y, Atasoy MS, et al. Multi-inflammatory index as a novel predictor of new-onset atrial fibrillation after off-pump coronary artery bypass grafting. Pol Heart J. 2024; 82(7-8): 733–740, doi: 10.33963/v.phj.100847, indexed in Pubmed: 38845422.
20. Lin YJ, Tsao HM, Chang SL, et al. Prognostic implications of the high-sensitive C-reactive protein in the catheter ablation of atrial fibrillation. Am J Cardiol. 2010; 105(4): 495–501, doi: 10.1016/j.amjcard.2009.10.019, indexed in Pubmed: 20152244.
21. Koyama T, Sekiguchi Y, Tada H, et al. Comparison of characteristics and significance of immediate versus early versus no recurrence of atrial fibrillation after catheter ablation. Am J Cardiol. 2009; 103(9): 1249–1254, doi: 10.1016/j.amjcard.2009.01.010, indexed in Pubmed: 19406267.
22. Richter B, Gwechenberger M, Socas A, et al. Frequency of recurrence of atrial fibrillation within 48 hours after ablation and its impact on long-term outcome. Am J Cardiol. 2008; 101(6): 843–847, doi: 10.1016/j.amjcard.2007.11.021, indexed in Pubmed: 18328850.
23. Deftereos S, Giannopoulos G, Kossyvakis C, et al. Colchicine for prevention of early atrial fibrillation recurrence after pulmonary vein isolation: A randomized controlled study. J Am Coll Cardiol. 2012; 60(18): 1790–1796, doi: 10.1016/j.jacc.2012.07.031, indexed in Pubmed: 23040570.
24. Koyama T, Tada H, Sekiguchi Y, et al. Prevention of atrial fibrillation recurrence with corticosteroids after radiofrequency catheter ablation: A randomized controlled trial. J Am Coll Cardiol. 2010; 56(18): 1463–1472, doi: 10.1016/j.jacc.2010.04.057, indexed in Pubmed: 20951321.
25. Nascimento T, Mota F, dos Santos LF, et al. Impact of prophylactic corticosteroids on systemic inflammation after extensive atrial ablation in pigs. Europace. 2012; 14(1): 138–145, doi: 10.1093/europace/eur259, indexed in Pubmed: 21846643.
26. Vlachos K, Letsas KP, Korantzopoulos P, et al. Prediction of atrial fibrillation development and progression: Current perspectives. World J Cardiol. 2016; 8(3): 267–276, doi: 10.4330/wjc.v8.i3.267, indexed in Pubmed: 27022458.
27. Chen YC, Liu CC, Hsu HC, et al. Systemic immune-inflammation index for predicting postoperative atrial fibrillation following cardiac surgery: A meta-analysis. Front Cardiovasc Med. 2024; 11: 1290610, doi: 10.3389/fcvm.2024.1290610, indexed in Pubmed: 38374999.
28. Yuksel A, Velioglu Y, Atasoy MS, et al. Multi-inflammatory index as a novel predictor of new-onset atrial fibrillation after off-pump coronary artery bypass grafting. Pol Heart J. 2024; 82(7-8): 733–740, doi: 10.33963/v.phj.100847, indexed in Pubmed: 38845422.

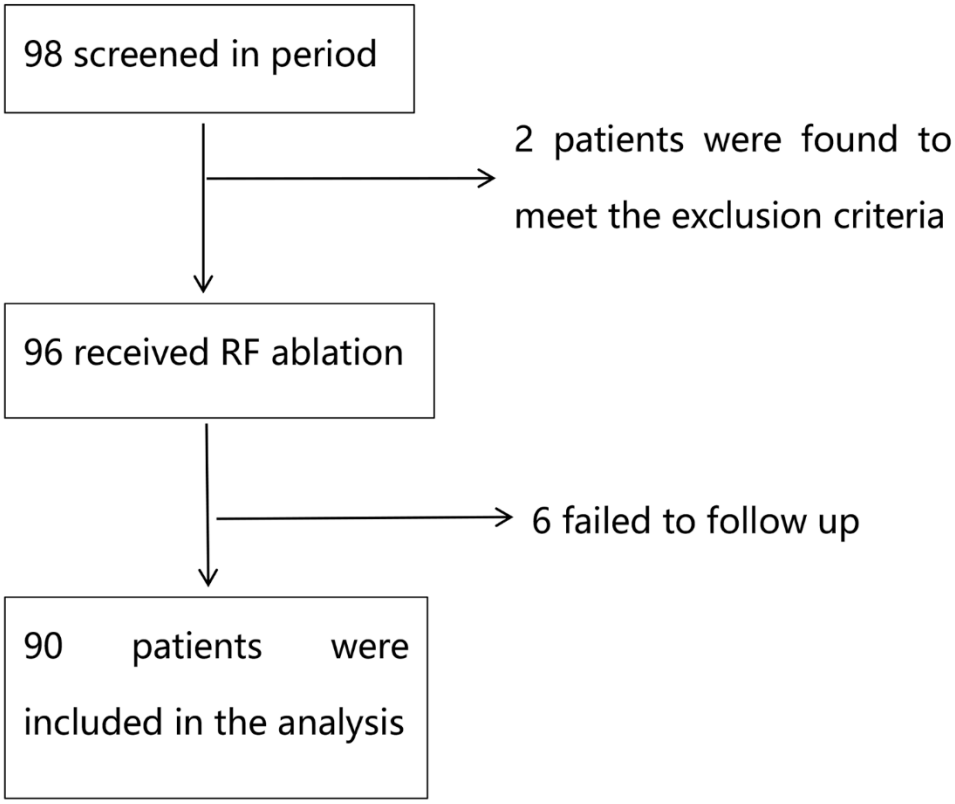

Figure 1. Flow diagram of the study

Abbreviation: RF, radiofrequency

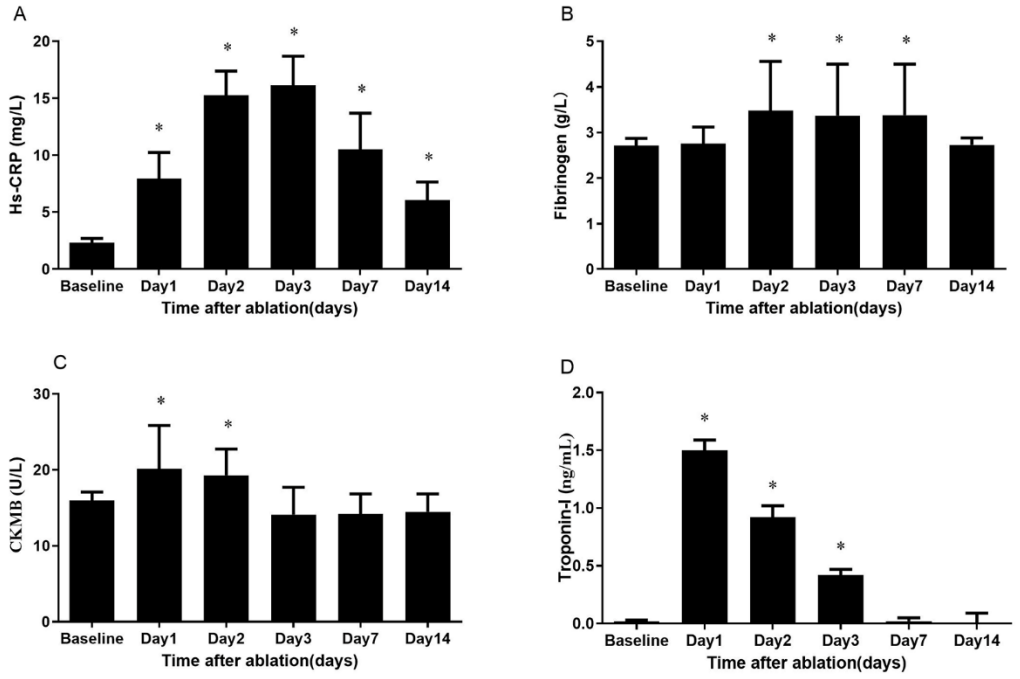

Figure 2. Duration with respect to variations in inflammatory markers following radiofrequency ablation for AF. **A.** High-sensitivity C-reactive protein (hs-CRP); **P* <0.05 (compared to baseline values). **B.** Fibrinogen; **P* <0.05 (compared to baseline values). **C.** Creatine kinase isoenzyme (CKMB); **P* <0.05 (compared to baseline values). **D.** Troponin-I; **P* <0.05 (compared to baseline values)

Table 1. Baseline information of the enrolled patients with atrial fibrillation

Characteristics	Patient cohort (n = 90)
Age, years	56 (11.6)
Male	42 (46.7%)
Comorbidities	
Hypertension	41 (45.6%)
Diabetes	7 (7.8%)
Coronary artery disease	12 (13.3%)
Previous stroke/TIA	8 (8.9%)
Low-density lipoprotein (mmol/l)	2.77 (0.62)
Blood creatinine (umol/l)	68.5 (11.2)
Baseline Hs-CRP	11.9 (6.5–17.8)

Baseline fibrinogen	0.8 (0.5–1.2)
Baseline CKMB	4.8 (2.8–7.1)
Baseline troponin-I	1.4 (0.9–1.6)
$CHADS_2$ score	
0	32 (35.6%)
1	43 (47.8%)
2	8 (8.9%)
≥3	7 (7.7%)
$CHADS_2$ score	1 (0–1)
Type of AF	
Paroxysmal AF	53 (58%)
Persistent AF	37 (42%)
Usual medications	
β-blockers	32 (35.6%)
Propafenone	30 (33.3%)
ACE inhibitors or ARB	33(36.7%)
Echocardiographic parameters	
Left ventricular ejection fraction, %	62.5 (8.2)
RF ablation time, min	126.5 (22.6)

Results are mean (SD), median (interquartile range), or n (%)

Abbreviations: AF, atrial fibrillation; $CHADS_2$, congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, and previous stroke/TIA; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; RF, radiofrequency; SD, standard deviation; TIA, transient ischemic attack

Table 2. Predictor of early AF recurrence by multivariable analysis

Variable	P	OR	OR 95% CI	
			Lower limit	Upper limit
Age	0.081	1.031	1.012	1.441
Hypertension	0.201	0.506	0.427	0.801
electric cardioversion	0.159	3.071	1.056	8.938

RF ablation time	0.102	1.116	1.025	1.243
CKMB elevation	0.753	1.390	0.479	2.765
Troponin-I elevation	0.052	2.550	0.993	6.546
Fibrinogen elevation	0.097	1.176	0.856	3.256
Hs-CRP elevation	0.004	2.948	1.892	4.602

Abbreviations: CI, confidence interval; OR, odds ratio; other — see Figure 2