Protective effect of Catheter Ablation of Atrial Fibrillation on the Renal Function in Patients with Hypertrophic Cardiomyopathy

Rei Mimuro[1], Hiroshi Hayashi[1], Yu-ki Iwasaki[1], Masato Hachisuka[1], Yuhi Fujimoto[1], Eiichiro Oka[1], Hiroshige Murata[1], Teppei Yamamoto[1], Kenji Yodogawa[1], and Wataru Shimizu[1]

[1]Nippon Medical School

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Abstract

Introduction: Atrial fibrillation (AF) is a common arrhythmia in patients with hypertrophic cardiomyopathy (HCM) and is associated with renal function deterioration. The protective effects of catheter ablation (CA) of AF on the renal function in HCM patients remains unsolved. Methods: From 2009 to 2020, 169 consecutive patients with HCM and AF (age 70±12, 87 males) were retrospectively evaluated. The estimated glomerular filtration rate (eGFR) was evaluated at the study enrollment or one month before the CA and reevaluated three months and 12 months later. Results: Among the 169 patients, 63 underwent CA of AF (ablation group) and the remaining 106 did not (control group). After propensity score matching, 45 pairs were matched. The baseline eGFR was similar between the two groups (P=0.83). During a mean follow-up period of 34±27 months, sinus rhythm was maintained in 36 (80%) patients after 1.7±0.8 ablation procedures. The eGFR significantly decreased from baseline to three months (P<0.01) and from baseline to one year (P<0.01) in the control group, while the eGFR in the ablation group was maintained both from baseline to three months (P=0.94) and from baseline to one year (P=1.00) after the CA. The change in the eGFR between baseline and 12 months was significantly smaller in the ablation group than control group (P<0.01). After a logistic regression analysis, CA of AF was the independent predictor of an improvement of eGFR (OR: 2.81; 95% CI: 1.08-7.36 P=0.04). Conclusions: CA of AF had a protective effect on the renal function in patients with HCM.

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Rei Mimuro, M.D.[*1], Hiroshi Hayashi, M.D., PhD[*1], Yu-ki Iwasaki, M.D., PhD[*1], Masato Hachisuka, M.D.[*1], Yuhi Fujimoto, M.D., PhD[*1],

Eiichiro Oka, M.D., PhD[*1], Hiroshige Murata M.D., PhD[*1],

Teppei Yamamoto, M.D., PhD[*1], Kenji Yodogawa, M.D., PhD[*1],

Wataru Shimizu M.D., PhD[*1].

1. Department of Cardiovascular Medicine, Nippon Medical School, Tokyo, Japan

Short tile: Efficacy of AF Ablation on the Renal Function in HCM Patients

Address for correspondence:

Hiroshi Hayashi, M.D., PhD.

Department of Cardiovascular Medicine, Nippon Medical School

1-1-5 Sendagi, Bunkyo-ku

Tokyo, 113-8603, Japan

Phone: 81-3-3822-2131 (Ext 24007)

Fax: 81-3-5685-0987

E-mail: *s00-067@nms.ac.jp*

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Methods: From 2009 to 2020, 169 consecutive patients with HCM and AF (age 70±12, 87 males) were retrospectively evaluated. The estimated glomerular filtration rate (eGFR) was evaluated at the study enrollment or one month before the CA and reevaluated three months and 12 months later.

Results: Among the 169 patients, 63 underwent CA of AF (ablation group) and the remaining 106 did not (control group). After propensity score matching, 45 pairs were matched. The baseline eGFR was similar between the two groups (P=0.83). During a mean follow-up period of 34±27 months, sinus rhythm was maintained in 36 (80%) patients after 1.7±0.8 ablation procedures. The eGFR significantly decreased from baseline to three months (P<0.01) and from baseline to one year (P<0.01) in the control group, while the eGFR in the ablation group was maintained both from baseline to three months (P=0.94) and from baseline to one year (P=1.00) after the CA. The change in the eGFR between baseline and 12 months was significantly smaller in the ablation group than control group (P<0.01). After a logistic regression analysis, CA of AF was the independent predictor of an improvement of eGFR (OR: 2.81; 95% CI: 1.08-7.36 P=0.04).

Conclusions: CA of AF had a protective effect on the renal function in patients with HCM.

Keywords Hypertrophic cardiomyopathy, Ablation, Atrial fibrillation, Kidney

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common hereditary cardiomyopathy characterized by left ventricular hypertrophy and a spectrum of clinical manifestations, with a prevalence of approximately 1:500 in the general population[1]. Atrial fibrillation (AF) is the most common sustained arrhythmia in HCM, which occurs in up to 20% of patients over their lifetime[2]. The occurrence of AF is associated with thromboembolisms, progression of heart failure and mortality[3,4].

Chronic kidney disease (CKD) is a progressive and irreversible renal dysfunction lasting more than three months caused by various etiologies, and it is an important risk factor for cardiovascular disease and heart failure[5,6]. Several heart diseases are reported to be a risk factor for developing CKD[7], and the HCM is one of the significant predictors of end-stage renal disease[8]. In addition, a previous study showed that renal dysfunction was a common comorbidity and independent predictor of the outcomes in patients with HCM[9]. The management of CKD in patients with HCM is an essential issue in terms of the maintenance of quality of life as well as the improvement of prognosis.

Recently, catheter ablation (CA) has become one of the therapeutic options for AF, and several studies have shown that patients maintaining sinus rhythm after CA improved their renal function[8,10,11]. However, to the best of our knowledge, the protective effect of AF ablation on the renal function remains unsolved in patients with HCM. The purpose of the study was to investigate the protective effects of CA of AF on the renal function in HCM patients.

Methods

Study population

The present study retrospectively evaluated 169 consecutive patients (age 70±12, 87 males, 98 paroxysmal AF) with HCM and AF who were followed up at Nippon Medical School hospital from January 2009 to December 2020. The diagnosis of HCM was based on two-dimensional echocardiographic evidence of a hypertrophied, non-dilated left ventricle (maximum wall thickness [?]15 mm) in the absence of systemic or cardiac disease capable of making the magnitude of the hypertrophy evident. Among the 169 patients with HCM and AF, 63 underwent CA of drug-refractory AF (ablation group) and the remaining 106 did not (control group). The patients in both groups were followed up every one to three months with optimal medical therapy including oral anticoagulants during the study period. Patients with chronic hemodialysis, a previous history of catheter ablation, and percutaneous coronary intervention within six months before starting the observation were excluded.

Propensity score matching was used to match the patient's clinical characteristics between the ablation group and control group[12,13]. The propensity score was generated from a multivariate logistic regression model using four variables associated with changes in the estimated glomerular filtration rate (eGFR): age, gender, body mass index (BMI), and baseline eGFR. After the propensity score generation, the ablation group and control group underwent 1:1 nearest neighbor matching of the logit of the propensity score with a caliper width of 0.25. The patients who did not meet the matching criteria were excluded. All patients gave their written informed consent for the ablation procedure and they were enrolled in the study notified of the opportunity to opt out. This study protocol was approved by the institutional review board of our institution.

Electrophysiologic study and catheter ablation

All antiarrhythmic drugs except for amiodarone were discontinued for at least five half-lives before the ablation. Warfarin was administered with a target international normalized ratio of 2.0-3.0 in patients under 75 years old or 1.6-2.5 in patients 75 years or more for at least one month before the procedure and continued during the periprocedural period. Direct oral anticoagulants (DOAC) were used for at least one month before the procedure and continued during the periprocedural period except for the procedure morning. The absence of atrial thrombi was confirmed by transesophageal echography or enhanced computed tomography. CA was performed under deep sedation using midazolam and dexmedetomidine. We deployed a multielectrode catheter into the coronary sinus from the jugular vein and circumferential decapolar electrode catheter in the pulmonary veins. Boluses of 80 and 50 IU/kg heparin were administered after venous and transseptal punctures. The activated clotting time was evaluated at least every 30 minutes and maintained at [?]300 seconds during the procedure. Pulmonary vein isolation was performed guiding by a circumferential decapolar electrode catheter. We monitored the surface electrocardiogram (ECG) and bipolar intracardiac electrograms on a computer-based digital amplifier recording system (RMC-5000, Nihon Kohden). Ablation was performed with a non-irrigation catheter (Navistar, Biosense Webster Inc) or irrigated-tip catheter (SmartTouch, Biosense Webster Inc. or FlexAbility, Abbott). A CARTO electroanatomical mapping system (Biosense Webster Inc) or Ensite Velocity system (Velocity Abbott) was used. The endpoint of the ablation was the complete isolation of all four pulmonary veins (PVs). Whether to create linear lesions, isolate the superior vena cava, and ablate the complex fractionated atrial electrograms (CFAEs) and non-PV triggers were left to the discretion of each operator.

Follow-up

After discharge from the hospital, the patients in the ablation group were followed in the outpatient clinic every month for the first 12 months, and every two or three months thereafter. Twelve lead electrocardiograms were performed every hospital visit. A 24-hour Holter monitoring was recorded three months after the procedure and every 12 months thereafter. The patients were advised to come to the hospital and undergo an additional electrocardiogram in the event of palpitations or dyspnea. The first three months after the ablation were designed as the blanking period. Recurrent atrial tachyarrhythmias, which continued for >30 seconds after the blanking period, were defined as a recurrence of AF. The patients in the control group were also followed up in the outpatient clinic every one to three months with optimal medical therapy.

Assessment of the renal function

In the present study, the eGFR calculated by 194 x age$^{-0.287}$ x serum creatinine$^{-1.094}$(x 0.739 for women)[14] was evaluated as the renal function in all the subjects. In the ablation group, the eGFR within one month before the CA was defined as the baseline eGFR and evaluated three months and 12 months after the ablation. Among the patients who had recurrence of AF and underwent repeat ablation sessions, the post-ablation eGFR was evaluated after the last session. In the control group, the baseline eGFR was evaluated at the date of the AF documentation and evaluated three months and 12 months thereafter. Among the patients whose AF onset was unknown, the baseline eGFR was evaluated on the day the follow-up was started at the study institute. The ΔeGFR was defined as the change in the eGFR between baseline and 12 months and compared between the two groups. The difference in the ΔeGFR between the patients with paroxysmal AF (PAF) and non-PAF in the ablation group was also evaluated. Specifically, patients with an eGFR [?]90 mL/min/1.73 m^2 were classified as a CKD stage G1, an eGFR of 60-89 mL/min/1.73 m^2 as a CKD stage G2, an eGFR of 45-59 mL/min/1.73m^2 as a CKD stage G3a, an eGFR of 30-44 mL/min/1.73 m^2 as a CKD stage G3b, an eGFR of 15-29 mL/min/1.73 m^2 as a CKD stage 4, and an eGFR <15 mL/min/1.73 m^2 as a CKD stage G5.

Statistical analysis

The data are expressed as the mean ± standard deviation or median (interquartile range) for continuous variables, and as absolute frequencies and percentages for categorical variables. For continuous variables, the differences between the groups were compared using a Student *t* test or Mann-Whitney *U* test, and for categorical variables, the Fisher exact test. The comparison of the eGFR between the two groups at baseline and one year was performed by a paired Student t test. A two-way repeated-measures ANOVA was used to compare the changes in the eGFR from baseline to three months and one year after between the two groups. A multivariate logistic regression analysis was performed to investigate the predictors associated with the change in the renal function. All tests were two-sided and a p value of < 0.05 was considered statistically significant. A Kaplan-Meier analysis and log-rank test were performed to analyze the probability of the freedom from arrhythmia recurrence after CA. The propensity score creation and all statistical analyses were conducted using SPSS 26.0 software (IBM Inc., Armonk, NY, USA).

Results

Characteristics of the study subjects

The study flow chart of the present study is shown in Figure 1. After excluding the patients whose follow-up period was less than one year and performing a propensity score matching, 45 pairs were matched. The baseline clinical characteristics, echocardiographic variables, and biomarkers after the propensity score matching are shown in Table 1. The age, body mass index and baseline eGFR did not differ between the two groups while the serum NT-proBNP level was significantly higher (P=0.04), and more patients were taking β-blockers (P<0.01) in the control group than ablation group. The number of patients with hypertrophic obstructive cardiomyopathy (HOCM) was larger in the ablation group compared to the control group (P=0.02).

Catheter ablation outcomes

A complete pulmonary vein isolation was performed in all subjects. A left atrial (LA) posterior wall isolation was achieved in 36 (80%) patients. A mitral isthmus linear ablation, cavotricuspid isthmus linear ablation, and superior vena cava isolation were performed in 15 (33%), 29 (64%), and one (2%) patient, respectively. The ablation procedural characteristics and periprocedural complications are shown in Suppl 1. After the first ablation, 10 (22%) patients had recurrences of AF within one year and 28 (62%) had an AF recurrence during the study period. Among the patients with AF recurrences, 25 (89%) received a second procedure. Eventually, during a mean follow-up period of 34 ± 27 months after 1.7 ± 0.8 ablation procedures, sinus rhythm was maintained in 36 (80%) patients (Figure 2). Of the 36 patients free from AF recurrence after the last ablation, 21 were taking antiarrhythmics including amiodarone (n=19) and cibenzoline (n=7). During the follow-up, 4 of 45 (9%) patients in the ablation group and 7 of 45 (16%) patients in the control group had hospitalizations due to heart failure (log-rank P=0.49). Death occurred in one (2%) patient in the ablation group and six (13%) in the control group (log-rank P=0.01). The cause of death was a neoplasm (N=1) in the ablation group, interstitial pneumonia (N=1), a neoplasm (N=1), unknown (N=3), and heart failure (N=1) in the control group. During the follow up, an angiotensin converting enzyme inhibitor (ACEI) (one vs. zero patient P=0.32) and diuretics (four vs. three patients P=0.70) were discontinued in the CA and control group, respectively. On the other hand, ACEIs (three vs. one patient P=0.31), angiotensin II receptor blockers (two vs. four patients P=0.40), and diuretics (two vs. two patients P=1.00) were started in the CA and control groups during the study period.

Changes in the renal function

The mean eGFR among the 169 HCM patients with AF at baseline was 54 ± 18 mL/min/1.73 m^2. One hundred and six (44%) of 169 patients had an eGFR of less than 60 mL/min/1.73 m^2. After a propensity score matching, the baseline eGFR was 55.0 ± 16.3 mL/min/1.73 m^2 in the ablation group and 55.7 ± 13.9 mL/min/1.73 m^2 in the control group (P=0.83). The number of patients in each CKD stage was similar between the two groups and the prevalence of an eGFR<60 mL/min/1.73 m^2 at baseline was 67% in the ablation group and 56% in the control group (P=0.28) (Table 1). The changes in the eGFR during the study in both groups are shown in Figure 3. In the control group, the eGFR significantly decreased from baseline to three months (55.7 ± 13.9 to 52.1 ± 12.8 mL/min/1.73 m^2, P<0.01) and from baseline to one year (55.7 ± 13.9 to 50.5 ± 13.5 mL/min/1.73 m^2, P<0.01) after the study enrollment. On the other hand, the eGFR in the ablation group remained unchanged during the study period both from baseline to three months (55.0 ± 16.3 to 55.3 ± 16.4 mL/min/1.73 m^2, P=0.94) and from baseline to one year (55.0 ± 16.3 to 55.0 ± 15.7 mL/min/1.73 m^2, P=1.00) after the ablation. The changes in the eGFR from baseline to three months and one year after the study enrollment were similar between the two groups (P=0.49). A comparison of the ΔeGFR between the two groups is shown in Figure 4. The ΔeGFR in the ablation group was significantly smaller than that in the control group (0 ± 7.6 vs. -5.2 ± 9.1 mL/min/1.73 m^2, P<0.01). In the ablation group, the ΔeGFR tended to be smaller in the patients with persistent AF than in those with PAF (1.7 ± 7.4 vs. -2.1 ± 7.4 mL/min/1.73 m^2, P=0.10).

The predictors of an improvement in the eGFR

The patient characteristics including echocardiographic variables, biomarkers, and drug therapy were compared between the patients whose eGFR increased (N=37) and those the eGFR decreased (N=53)(Table 2) during the study. When comparing the two groups, the ratio of male patients was larger in the patients whose eGFR increased than the patients whose eGFR decreased (P=0.03). The patients whose eGFR increased had a lower left ventricular ejection fraction (LVEF)(P=0.02), larger left atrial diameter (P=0.04), higher serum creatinine level (P<0.01) and lower eGFR (P=0.02) than those whose eGFR decreased. In the control group (CA (-)), the number of patients whose eGFR decreased tended to be larger than that whose eGFR increased (P=0.054). A multivariable logistic regression analysis was performed to investigate the predictors associated with an improvement in the renal function. Catheter ablation of AF (OR: 2.81; 95% CI: 1.08-7.36 P=0.04) and the baseline LVEF (OR: 0.95; 95% CI: 0.91-1.00 P=0.04) were independent predictors of the improvement in the eGFR at one year of follow-up (Table 3). The change in the eGFR over one year was evaluated and stratified by the CKD stages (Figure 5). The eGFR decreased in all CKD stages except CKD stage G4 in the control group, while the eGFR increased in CKD stage G3a, stage G3b, and stage G4 in the ablation group. In the patients with CKD stage G3a, the change in the eGFR in the ablation group was significantly larger than that in the control group (2.7 ± 7.7 vs. -5.1 ± 6.8 mL/min/1.73 m^2, P<0.01).

Discussion

The main findings of this study can be summarized as follows: (1) the patients with HCM complicated with AF had a high prevalence of CKD, (2) HCM patients with AF who underwent CA of AF had maintenance of their renal function for one year of follow-up, and (3) the ablation outcome in the patients with HCM was favorable, although multiple procedures were required.

HCM and the renal function

Several previous studies examined the association between HCM and the incidence of CKD including end stage renal disease (ESRD). From the Korean nationwide population-based cohort study[8], the presence of HCM was associated with approximately a 7-fold increased risk of newly diagnosed ESRD. Huang et al.[9] reported that approximately 15% of the HCM patients complicated with CKD whose eGFR was <60 mL/min/1.73 m^2 and HCM patients with CKD had an increased risk of all-cause mortality. In the present study, the prevalence of an eGFR<60 mL/min/1.73 m^2 at baseline in the two groups was 56-67%, which was higher than that in the above previous report[9]. In HCM patients, the renal dysfunction may be attributed to impaired LV diastolic dysfunction caused by LV hypertrophy and stiffness, which are pathological features of HCM. A reduced LV diastolic function and cardiac output leads to an increased LV filling pressure and renal venous pressure, which eventually attenuates the renal function. As there are few solutions for preventing the progression of LV diastolic dysfunction in patients with HCM, the risk of developing CKD in this patient population is a crucial problem. AF is the most common arrhythmia in patients with HCM,[3,15] in whom the incidence is reported to be 20% in their lifetime[2]. The loss of the atrial kick and irregular beats due to AF reduce the stroke volume and cardiac output[16]. Several studies have indicated the influence of CKD on the increased risk of AF,[17,18] and its mechanism is assumed to be persistent inflammation related to CKD[19] and mechanical stress caused by a high atrial pressure due to hypertension and/or heart failure associated with CKD[20]. The high prevalence of CKD in the HCM patients of the present study indicated that the coexistence of AF accelerates the deterioration in the renal function in patients with HCM, and therefore, maintenance of sinus rhythm in patients with HCM is highly desired.

Protective effect of AF ablation on the renal function

Previously, several studies examined the protective effect of AF ablation on the renal function[8,10,21]. Takahashi et al.[10] evaluated the change in the eGFR after AF ablation and reported that the eGFR increased three months after CA and was maintained until one year in patients free from AF recurrence, whereas the eGFR decreased over one year in patients with AF recurrences. In the present study, the HCM patients who underwent AF ablation had the maintenance of their renal function, however, the HCM patients without CA had a decrease in their eGFR over one year of follow-up. The number of patients, in whom pharmacotherapy changed during the study, was small in both groups and the incidence of hospitalizations due to heart failure did not differ between the two groups. These data indicated that those factors did not influence the change in the eGFR in this study. Therefore, we considered that the maintenance of the renal function was achieved by AF ablation. The mechanism of the protective effect of AF ablation on the renal function has been discussed in previous studies[10,21]. The improvement in the atrial contractile function after a successful ablation will increase the cardiac output, leading to the protection of renal function. An echocardiography and MRI study showed that a successful AF ablation was associated with LA reverse remodeling and LA functional recovery[22,23]. As an impaired LA function is associated with cardiovascular hospitalizations in patients with heart failure with a preserved ejection fraction[24], the LA dysfunction may have a significant negative impact on the renal function in patients with HCM.

In the present study, another independent predictor of renal function improvement (increased eGFR) was a lower LVEF at baseline. The reason for the baseline lower LVEF predicting the renal function improvement remains unsolved. An elevation of central venous pressure, which is observed in patients with impaired LV systolic function and heart failure, lowers the gradient in the pressure across the arteries and veins of the kidneys which eventually reduces the renal function[25,26]. As CA of AF is associated with LVEF improvement[27], the AF ablation might have lowered the central venous pressure and had a positive effect on renal function. The patients with reduced LVEF at baseline may receive more benefit from AF ablation than patients with normal LVEF. However, the data supporting the decrease in the renal venous pressure after the ablation was lacking in the present study.

Ablation outcomes in patients with HCM

Atrial fibrillation with a rapid ventricular response causes an increase in the heart rate, shorter diastolic filling time, and loss of an effective atrial contraction, which contributes to a reduction in the cardiac output, especially in patients with an impaired diastolic function such as HCM patients. Pozzoli et al.[28]reported that the onset of AF was associated with a significant worsening of the NYHA class, cardiac index, and increased mitral regurgitation. Therefore, rhythm control for AF in patients with HCM is of great importance. A previous systematic review showed that the overall rate of the freedom from an AF/AT recurrence with a single CA procedure was 38.7% and that with multiple CA procedures was 51.8% in HCM patients, while those were 49.8% and 71.2% respectively in non-HCM patients[29]. In the present study, the overall rate of the freedom from AF/AT recurrences with a single CA procedure in HCM patients was 38%, which was comparable with that reported in the systematic review. However, the arrhythmia free rate after multiple CA procedures was 80%, which was better than the previous studies[29,30]. The reason for better outcome may be due to the difference in the number of CA procedures, CA strategy, and usage of antiarrhythmic therapy after the ablation. The mean number of ablation procedure in the present study was 1.7 ± 0.8, which was more frequent than that in the previous reports[29]. Regarding the ablation strategy, a posterior wall isolation was performed in 80% of the subjects and additional mitral isthmus ablation was performed in one-third of the subjects. Previously, Santangeli et al.[31] reported that non-PV triggers were observed in the majority of the HCM patients with a late recurrence of AF after ablation, and elimination of non-PV triggers were associated with an improvement in the ablation outcome. As non-PV triggers are commonly observed on the posterior wall and in the coronary sinus[32], those additional ablation lesions might have improved the success rate[33]. In the present study, antiarrhythmic therapy, mostly amiodarone, was continued in 58% of the HCM patients after the last ablation. Amiodarone contains a risk of life-threatening side effects including interstitial pneumonia, thyroid dysfunction, and liver dysfunction, and therefore its discontinuation is desirable. However, the use of long-term antiarrhythmics was often required according to the meta-analysis of AF ablation in patients with HCM[29]. As atrial remodeling is ongoing due to the pathologically progressive nature of HCM, an aggressive therapeutic approach by catheter ablation and anti-arrhythmic agents may be needed for the maintenance of sinus rhythm in the HCM patients.

Limitations

There are several limitations to the present study. First, this was a retrospective single-center study with a small study population. Although, there were several clinical backgrounds that influenced the renal function, we consider that the propensity score matching reduced the biases of the variables related to the eGFR. A further large-scale prospective study will be needed to clarify these issues. Second, the freedom from AF more than 3 months after the ablation might have been possibly overestimated, as asymptomatic episodes of recurrence of AF could not have been evaluated by regular twelve lead electrocardiograms and 24-hour Holter monitoring.

Conclusions

CKD was frequently observed in patients with HCM associated with AF. The successful CA of AF resulted in the maintenance of the renal function for one year of follow-up.

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Figure legends

Figure 1.

The flow chart of the study.

AF = atrial fibrillation, HCM = hypertrohic cardiomyopathy, BMI = body mass index, eGFR = estimated glomerular filtration rate

Figure 2.

Kaplan-Meier analysis of the long-term freedom from recurrent AF after the first and last catheter ablation procedures. Events were not counted during the first 3 month blanking period. The solid line and dotted line indicate the arrhythma free rate after the first and last catheter abaltion procedures, respectively.

CA = catheter ablation

Figure 3.

Two-way ANOVA analysis of the changes in the eGFR from baseline to three months and baseline to one year between the ablation and control groups.

eGFR = estimated glomerular filtration rate

Figure 4.

Comparison of the ΔeGFR between the ablation and control groups. The ΔeGFR was defined as the change in the eGFR between baseline and 12 months.

eGFR = estimated glomerular filtration rate

Figure 5.

Comparison of the ΔeGFR between the ablation and control groups for each CKD stage. The ΔeGFR was defined as the change in the eGFR between baseline and 12 months.

eGFR = estimated glomerular filtration rate , *CKD = chronic kidney disease*

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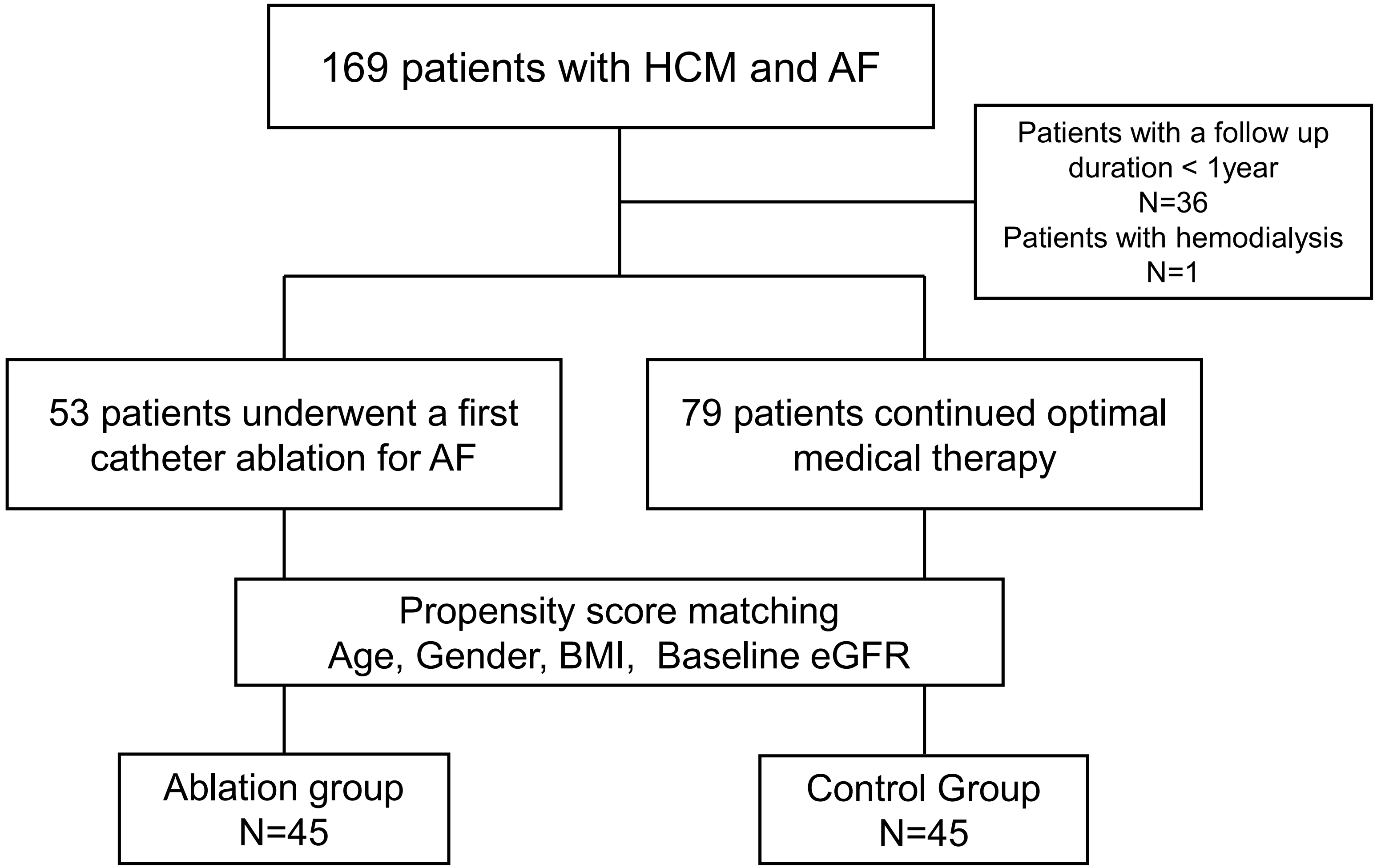
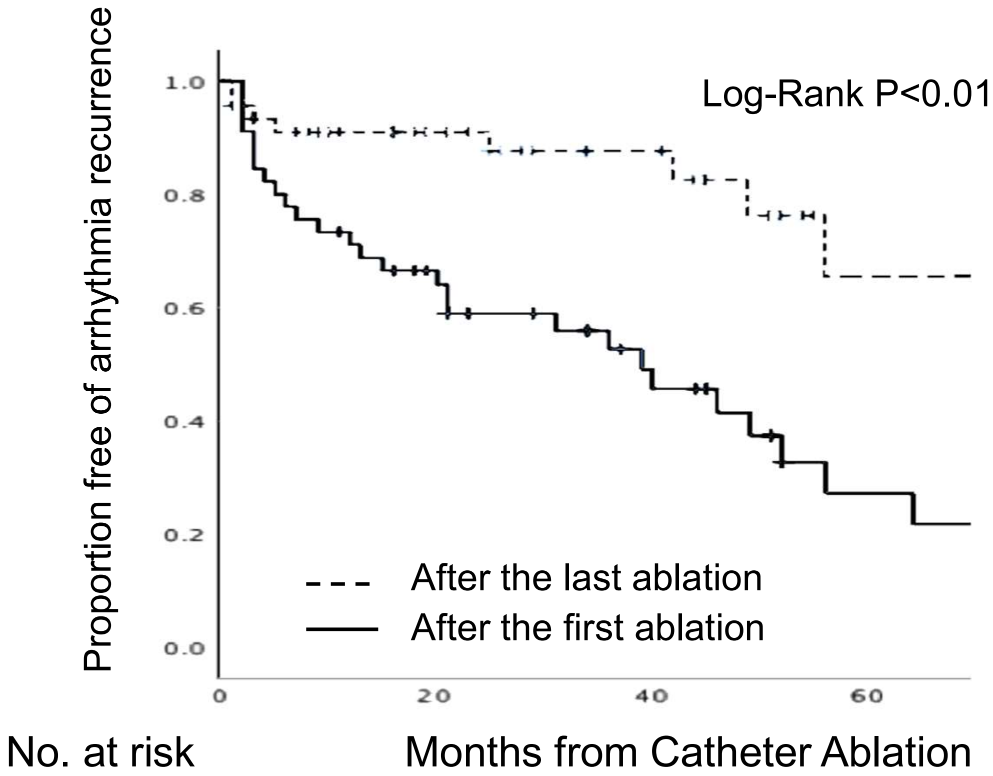
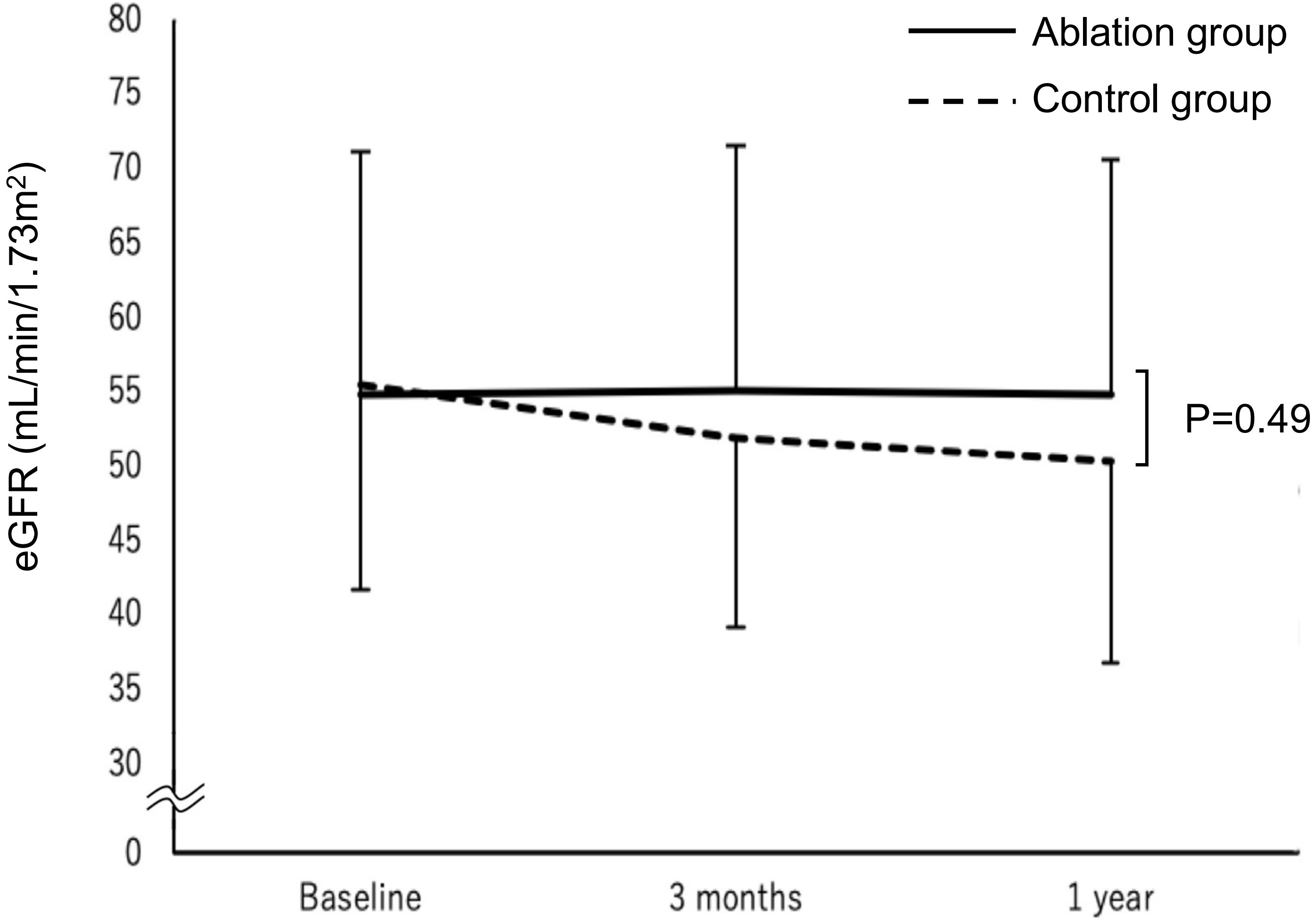
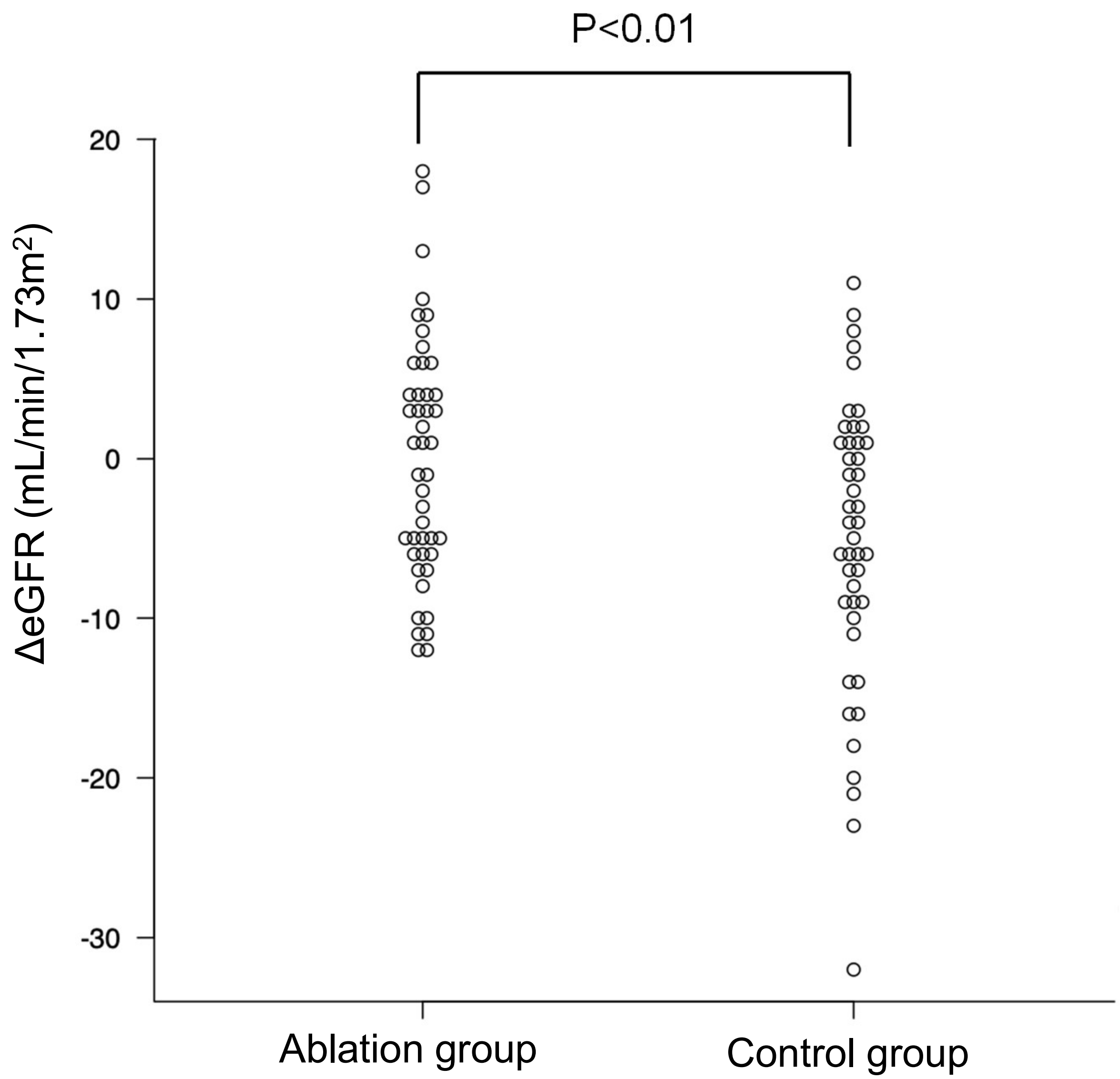
`table1101.docx` available at https://authorea.com/users/737518/articles/712434-protective-effect-of-catheter-ablation-of-atrial-fibrillation-on-the-renal-function-in-patients-with-hypertrophic-cardiomyopathy


169 patients with HCM and AF
Patients with a follow up duration < 1year
N=36
Patients with hemodialysis
N=1
53 patients underwent a first catheter ablation for AF
79 patients continued optimal medical therapy
Propensity score matching
Age, Gender, BMI, Baseline eGFR
Ablation group
N=45
Control Group
N=45

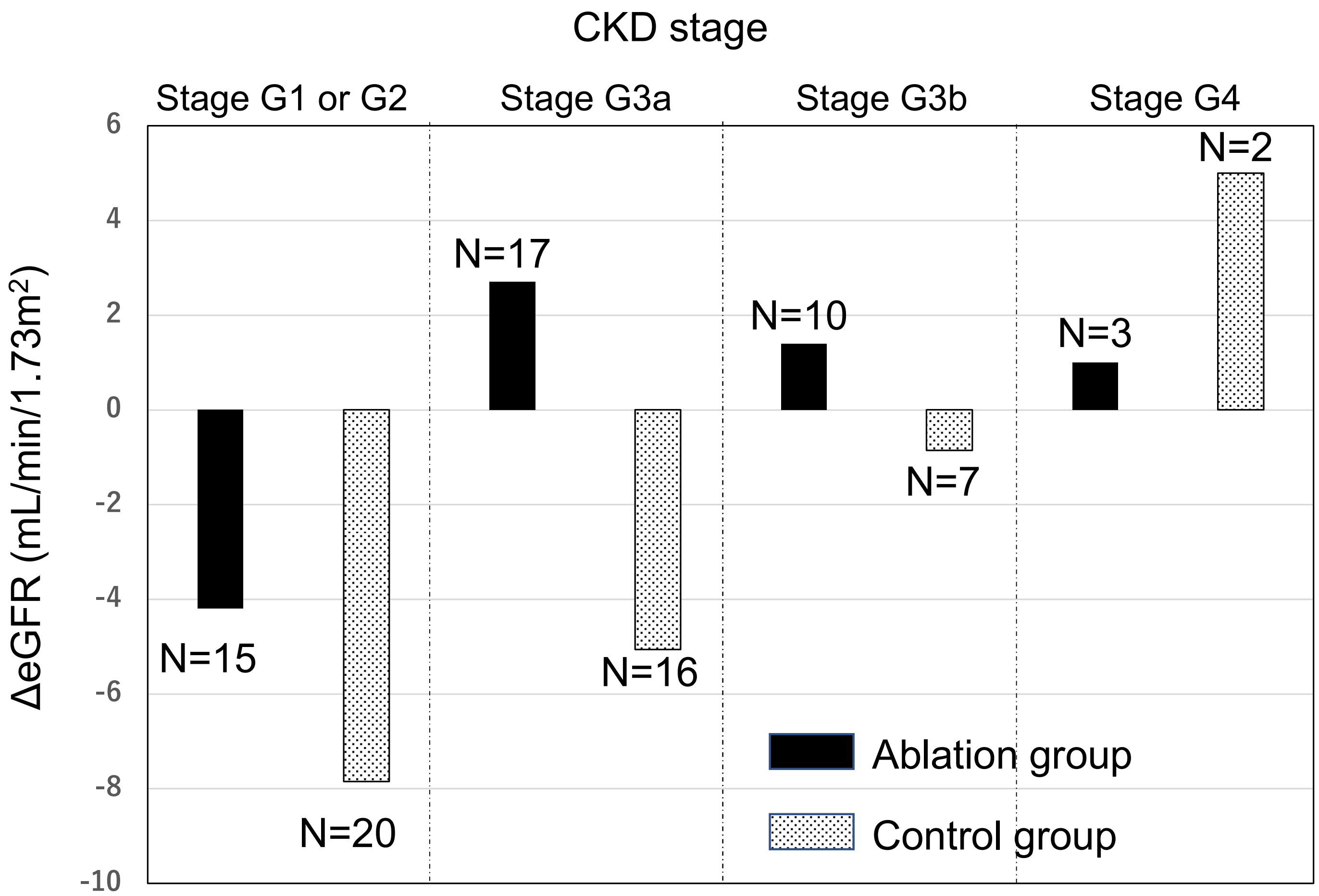


1st CA	**45**	**25**	**13**	**5**
Last CA	**45**	**29**	**19**	**6**


Ablation group
Control group
eGFR (mL/min/1.73m2)
80
75
70
65
60
55
50
45
40
35
30
0
P=0.49
Baseline
3 months
1 year



P<0.01
20
10
0
-10
-20
-30
ΔeGFR (mL/min/1.73m²)
Ablation group
Control group


CKD stage
Stage G1 or G2
Stage G3a
Stage G3b
Stage G4
ΔeGFR (mL/min/1.73m²)
6
4
2
0
-2
-4
-6
-8
-10
N=15
N=20
N=17
N=16
N=10
N=7
N=3
N=2
Ablation group
Control group