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# Predictors of Progression of Tricuspid Regurgitation in Patients with Persistent Atrial Fibrillation

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Previous studies have shown that tricuspid regurgitation (TR) can be developed in patients with atrial fibrillation (AF) due to annular dilatation. This study aimed to investigate the incidence and predictors of the progression of TR in patients with persistent AF. A total of 397 patients (66.9±11.4 years, 247 men; 62.2%) with persistent AF were enrolled between 2006 and 2016 in a tertiary hospital, and 287 eligible patients with follow-up echocardiography were analyzed. They were divided into two groups according to TR progression (progression group [n=68, 70.1±10.7 years, 48.5% men] vs. non-progression group [n=219, 66.0±11.3 years, 64.8% men]). Among 287 patients in the analysis, 68 had worsening TR severity (23.7%). Patients in the TR progression group were older and more likely to be female. Patients with left ventricular ejection fraction $<50\%$ were less frequent in the progression group than those in the non-progression group (7.4% vs. 19.6%, p=0.018). Patients with mitral valve disease were more frequent in the progression group. Multivariate analysis with COX regression demonstrated independent predictors of TR progression, including left atrial (LA) diameter $>54$ mm (HR 4.85, 95%CI 2.23-10.57, $p<0.001$), E/e' (HR 1.05, 95%CI 1.01-1.10, p=0.027), and no use of antiarrhythmic agents (HR 2.20, 95%CI 1.03-4.72, p=0.041). In patients with persistent AF, worsening TR was not uncommon. The independent predictors of TR progression turned out to be greater LA diameter, higher E/e', and no use of antiarrhythmic agents.

**Key Words:** *Tricuspid Valve Insufficiency; Atrial Fibrillation; Patients*



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## INTRODUCTION

The etiologies of tricuspid regurgitation (TR) are comprised of primary and secondary or functional. Primary TR usually develops from congenital heart disease or the impairment of the valve due to inflammation such as endocarditis. However, significant TR may develop in the absence of structural abnormalities of the tricuspid valve (TV) and its apparatus, namely functional or secondary TR.[1,2] Actually, functional etiology is most frequent, occupying 80-90% of all TR with morphologically normal leaflets with impaired coaptation. The mechanism of Functional TR involves two main mechanisms, including annulus dilatation and leaflet tethering, and three main groups, including left-sided heart disease, pulmonary disease, and chronic atrial fibrillation (AF).[3] Functional TR is relatively common in patients with left-sided heart disease or pulmonary hypertension. The incidence of late significant functional TR is reported in up to 40% of the patients who underwent left-sided valve surgery.[4] AF is a common arrhythmia in the elderly or patients with structural heart disease. AF with a long duration usually results in the enlargement of both atria and/or annular dilatation of atrioventricular

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valves, and these kinds of atrial remodeling are also known to be a risk factor for developing significant functional TR.[5,6]

Contrary to the general expectation, significant tricuspid regurgitation (TR) is known to be an independent predictor of long-term mortality and increasing severity of TR is associated with poor prognosis regardless of etiology, left ventricular ejection fraction (LVEF), or pulmonary artery pressure.[7] Since functional TR due to chronic AF is not uncommon and could cause adverse outcomes when progressing to significant TR, it is important to understand what kind of factors affect TR progression. However, there is a paucity of data regarding the incidence and the predictors of TR progression in patients with AF. Therefore, the present study aimed to investigate the incidence and predictors of TR progression in patients with persistent AF.

## MATERIALS AND METHODS

### 1. Study population

Between 2006 and 2016, a total of 397 patients (66.9± 11.4 years, 247 men; 62.2%) with persistent AF visited a tertiary hospital and underwent at least one echocardiogram. Among these, 110 patients were eliminated, and the reasons for elimination were as follows: no follow up (FU) echocardiography (n=84), the presence of prosthetic mitral valve at baseline (n=19), and the presence of intracardiac device with right ventricular lead (n=10). The 287 eligible patients with FU echocardiography were divided into 2 groups according to TR progression: the progression group (n=68, 65.2±10.8 years, 52.9% men) vs. the non-progression group (n=219, 63.1±11.0 years, 64.2% men) (Fig. 1). The median follow-up duration was 4.9 years. The change of TR was defined as different TR severity measured between two separate echocardiographic examinations. The change of TR severity to an upper degree on FU echocardiography was considered progressive TR in the present study and mild TR was not considered as a progressive case. Persistent AF was diagnosed when the arrhythmic episodes endured beyond 7 days or require cessation with pharmacological or direct current cardioversion of between 48 hours to 7 days duration.[8] This study was approved by the ethics committee and institutional review board of Chonnam National University Hospital (IRB number: CNUH-2022-047).

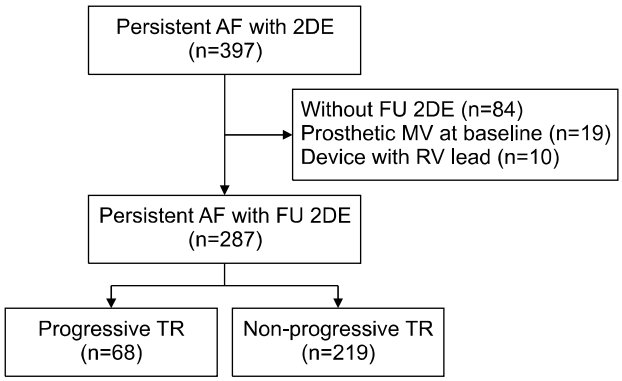


**FIG. 1.** Study flow and clinical outcomes of the study patients. AF: atrial fibrillation, 2DE: 2D-echocardiography, FU: follow up, MV: mitral valve, RV: right ventricle, TR: tricuspid regurgitation.

### 2. Echocardiographic measurements

Echocardiographic examinations were performed at the initial presentation and during the FU period. Images were taken while patients were in the left lateral decubitus position. Conventional echocardiographic studies, including Doppler studies, were performed according to the recommendations of the American Society of Echocardiography (ASE).[1] Left ventricular end-systolic and end-diastolic dimensions, interventricular septal and posterior wall thicknesses, and left-atrial anteroposterior diameters were determined from 2-dimensional images. The LVEF was calculated using the conventional Teicholz's and biplane Simpson's methods. A severe TR was defined as a TR with a distal jet area $\geq 10\ cm^2$, vena contracta width greater than 0.7 cm, and systolic flow reversal in hepatic veins. Mild and moderate TR was defined according to the report from the ASE.[9] Pulmonary artery systolic pressure was assessed by the maximal velocity of the TR jet using a modified version of Bernoulli's equation.[10] Right atrial pressure was estimated as 5 mmHg if the inferior vena cava (IVC) was not dilated ($<1.7$ cm) and there was a 50% decrease in the diameter during inspiration, 10 mmHg if the IVC was dilated with normal inspiratory collapse and 15 mmHg if the IVC was dilated and did not collapse with inspiration.[11]

### 3. Study endpoints

The primary endpoint was the development of any TR progression in the follow-up echocardiography during the study period.

### 4. Statistical analysis

Continuous variables with normal distributions are presented as mean±standard deviation and were compared using Student's t-test or Mann-Whitney U test when group distributions were skewed. Categorical variables were compared using the Chi-square test or Fisher's exact test, where appropriate. A regression analysis using logistic regression was performed to identify independent predictors of TR progression. The variables with $p<0.1$ on univariate logistic regression analysis and clinically relevant ones were tested in the model. All statistical tests were 2-tailed, and p values $<0.05$ were considered significant. The receiver operating characteristics (ROC) curve analysis was performed to determine the cut-off values of left atrial (LA) diameter and right ventricular systolic pressure (RVSP). All analyses were performed using the Statistical Package for Social Sciences, version 21.0 (SPSS-PC, Chicago, Illinois) and the ROC-curve analysis were performed using R version 4.1.1 (R Foundation for Statistical Computing, Vienna, Austria).

## RESULTS

### 1. Baseline clinical characteristics

Baseline clinical characteristics are summarized in Table 1. The patients in the progression group were older, more likely to be female, to have trended toward longer duration of AF, and to have concomitant mitral regurgitation (MR) as well as stenosis. Patients with MR had a higher progression rate than those without MR (28.0% vs. 15.8%, p=0.021). However, the severity of MR was not associated with TR progression. Interestingly, an index LVEF of less than 50% showed less progression (10.4% vs. 26.4%, p= 0.018). Of note, treatment for AF showed an impact on the progression of TR. AF treatment strategies toward rhythm conversion showed less frequent progression. However, diuretics were more frequently used in the progression group suggesting more intensive therapy in patients with higher grade TR (Table 1).

### 2. Echocardiographic findings

Echocardiographic findings are summarized in Table 2. LV end-diastolic and end-systolic dimensions were not different between the two groups at both initial and follow-up echocardiography. Baseline LVEF was significantly lower in the non-progression group than in the progression group. Also, initial LVEF was improved about 4% at follow-up only in the non-progression group. LA chambers of both groups were significantly enlarged at follow-up. However, the LA diameter of the progression group was significantly greater than that of the non-progression group in both initial and follow-up echocardiography (Table 2). The difference of LA diameter between initial and follow-up echocardiography in the progression group was significantly greater than in the non-progression group (7.8±8.4 mm vs. 2.8±6.4 mm, $p<0.001$). However, peak TR velocity and estimated RVSP of the progressive group were greater than those of the non-progressive group only in the follow-up echocardiography apparently due to not yet progressed TR in the initial period. The size of tricuspid annulus and right atrium were not different between the two groups and neither were the parameters of right ventricular function.

### 3. Predictors of progressive TR

Progressive TR was observed in 68 out of 287 patients with chronic AF (23.7%). The cut-off value for LA diameter and RVSP was determined using the ROC curve analysis. It showed that an initial LA diameter >54 mm and RVSP >35 mmHg identified the patients with TR progression with an area under the curve of 0.737 and 0.620 respectively (sensitivity/specificity 69.3%/70.1% and 69.1%/62.3%, respectively). Male sex, initial low LVEF (<50%), concomitant mitral stenosis (MS), MR, and RVSP were predictors of progressive TR only in the univariate analysis. However, LA diameter >54 mm and no use of antiarrhythmic agents were independent predictors of progressive TR also in the multivariate analysis (Table 3).

## DISCUSSION

The present study was aimed to investigate the incidence, baseline characteristics and predictors of TR pro-

**TABLE 1.** Comparison of baseline clinical characteristics between progressive and non-progressive tricuspid regurgitation group

| n, (%) | Progression (n=68) | Non-progression (n=219) | p |
|---|---|---|---|
| Age (years) | 70.1±10.7 | 66.0±11.3 | 0.009 |
| Female sex | 35 (51.5) | 77 (35.2) | 0.016 |
| Diabetes mellitus | 13 (19.1) | 53 (24.9) | 0.329 |
| Hypertension | 35 (51.5) | 102 (47.7) | 0.584 |
| Smoking history | 10 (14.7) | 46 (21.5) | 0.221 |
| Dyslipidemia | 14 (20.6) | 39 (18.2) | 0.664 |
| Cerebrovascular accident | 8 (11.8) | 26 (12.1) | 0.932 |
| Prior myocardial infarction | 3 (4.4) | 12 (5.6) | 0.488 |
| Duration of Atrial fibrillation >3 years | 45 (66.2) | 110 (53.1) | 0.060 |
| Left ventricular ejection fraction <50% | 5 (7.4) | 43 (19.6) | 0.018 |
| Concomitant mitral regurgitation | 52 (76.5) | 134 (61.2) | 0.021 |
| Concomitant mitral stenosis | 12 (17.6) | 19 (8.7) | 0.037 |
| Blood urea nitrogen (mg/dL) | 19.0±6.7 | 16.7±5.6 | 0.021 |
| Creatinine (mg/dL) | 1.01±0.37 | 0.97±0.41 | 0.546 |
| NT-proBNP (pg/mL) | 1324.0±1544.7 | 2446.1±4677.1 | 0.167 |
| Antiarrhythmic agents | 21 (31.8) | 113 (54.3) | 0.001 |
| Cardioversion | 2 (3.0) | 32 (15.4) | 0.007 |
| Ablations for atrial fibrillation | 1 (1.5) | 20 (9.5) | 0.031 |
| Diuretics use | 51 (77.3) | 129 (61.4) | 0.018 |
| Loop diuretics | 50 (73.5) | 118 (53.9) | 0.004 |
| Spironolactone | 32 (48.5) | 92 (44.0) | 0.525 |

NT-proBNP: N-terminal pro-B type natriuretic peptide.

**TABLE 2.** Comparison of initial and follow-up echocardiographic findings between patients with progressive and non-progressive tricuspid regurgitation

| n, (%) | Progression (n=68) | Non-progression (n=219) | p | Progression (n=68) | Non-progression (n=219) | p |
|---|---|---|---|---|---|---|
| | Initial | | | Follow-up | | |
| Left ventricular end-diastolic dimension (mm) | 51.1±6.3 | 52.4±27.2 | 0.625 | 51.2±7.4 | 49.8±5.4 | 0.333 |
| Left ventricular end-systolic dimension (mm) | 34.3±6.5 | 34.7±7.1 | 0.614 | 33.4±6.4 | 33.7±21.4 | 0.706 |
| Left ventricular ejection fraction (%) | 61.7±8.5 | 59.1±11.3 | 0.031 | 63.3±7.8 | 63.7±8.6** | 0.581 |
| Interventricular septum (mm) | 9.3±2.1 | 9.3±1.6 | 0.831 | 9.6±1.9 | 9.4±1.6 | 0.582 |
| Left ventricular posterior wall (mm) | 9.4±1.2 | 9.2±1.6 | 0.237 | 9.4±1.7 | 9.2±1.2 | 0.537 |
| Left atrial diameter (mm) | 52.9±8.1 | 48.3±8.2 | 0.003 | 60.5±10.1** | 51.1±8.2** | <0.001 |
| E velocity (m/s) | 1.1±0.5 | 0.9±0.4 | 0.043 | 1.2±0.5 | 0.9±0.4 | 0.006 |
| Deceleration time (sec) | 288.3±198.5 | 221.2±155.7 | 0.081 | 259.5±257.0 | 223.1±151.6 | 0.425 |
| e' velocity (cm/s) | 8.6±10.1 | 8.0±6.3 | 0.684 | 7.4±2.2 | 8.1±2.8 | 0.115 |
| s' velocity (cm/s) | 5.8±1.4 | 6.9±5.9 | 0.256 | 6.3±1.5* | 6.8±1.5 | 0.049 |
| E/e' | 16.1±10.9 | 11.9±5.5 | 0.174 | 16.3±7.6 | 15.5±27.1 | 0.952 |
| Aortic valve peak velocity | 1.5±0.7 | 1.3±0.5 | 0.188 | 1.7±0.8* | 2.2±10.8 | 0.688 |
| Pulmonic valve peak velocity (m/s) | 0.8±0.2 | 0.7±0.2 | 0.263 | 0.84±0.23 | 0.77±0.24 | 0.169 |
| Tricuspid Regurgitation peak velocity (m/s) | 2.7±1.0 | 2.4±1.1 | 0.263 | 3.9±1.7 | 2.7±0.9 | 0.001 |
| Right ventricular systolic pressure (mmHg) | 38.5±10.1 | 37.0±21.6 | 0.772 | 47.9±15.7** | 35.8±9.1 | <0.001 |
| Tricuspid annular diameter (mm) | 38.7±5.1 | 37.3±3.6 | 0.532 | 41.7±11.1 | 38.2±5.5 | 0.454 |
| Right atrial area ($cm^2$) | 24.7±5.8 | 24.2±6.1 | 0.876 | 30.5±11.0 | 22.6±5.8 | 0.183 |
| TAPSE (mm) | 15.7±2.6 | 17.2±2.0 | 0.197 | 17.0±4.6 | 15.5±3.2 | 0.474 |
| Tricuspid valvular s' velocity (cm/s) | 9.8±3.4 | 10.5±2.2 | 0.613 | 0 10.7±1.0 | 11.3±1.9 | 0.442 |

*p<0.05, **p<0.001 compared to initial echocardiography. E: early diastolic wave, e': early diastolic tissue Doppler wave, s': systolic excursion velocity, TAPSE: tricuspid annular plane systolic excursion.

**TABLE 3.** Independent predictors of progressive tricuspid regurgitation in patients with persistent atrial fibrillation

| Variables | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|
| | Odd ratio (95% confidence interval) | p value | Odd ratio (95% confidence interval) | p value |
| Age >65 years | 1.80 (0.99-3.26) | 0.053 | 1.53 (0.80-2.92) | 0.197 |
| Male sex | 0.51 (0.30-0.89) | 0.017 | 0.71 (0.39-1.28) | 0.255 |
| LVEF <50% | 0.33 (0.12-0.86) | 0.023 | 0.51 (0.15-1.76) | 0.289 |
| Concomitant mitral stenosis | 2.26 (1.03-4.93) | 0.041 | 1.95 (0.83-4.54) | 0.126 |
| Concomitant mitral regurgitation | 2.06 (1.11-3.84) | 0.023 | 1.86 (0.89-3.88) | 0.101 |
| Left atrial diameter >54 mm | 4.29 (2.50-7.35) | <0.001 | 4.85 (2.23-10.57) | <0.001 |
| E/e' | 1.04 (1.01-1.07) | 0.015 | 1.05 (1.01-1.10) | 0.027 |
| RVSP >35 mmHg | 2.18 (1.32-3.60) | 0.002 | 1.68 (0.82-3.43) | 0.071 |
| Use of loop diuretics | 1.53 (0.89-2.63) | 0.125 | | |
| Prior AF duration >3 years | 1.40 (0.84-2.32) | 0.194 | | |
| No use of antiarrhythmic agents | 2.25 (1.30-3.87) | 0.004 | 2.20 (1.03-4.72) | 0.041 |

LVEF: left ventricular ejection fraction, E: early diastolic wave, e': early diastolic tissue Doppler wave, RVSP: right ventricular systolic pressure, AF: atrial fibrillation.

gression in patients with persistent AF. First, the progression of TR was observed in 23.7% of the study population during the study period. Second, LA diameters over 54 mm, E/e', and no use of antiarrhythmic agents were independent predictors of progression of TR in patients with persistent AF. Even though prior duration of AF was not an independent predictor of TR progression, LA diameter may reflect AF duration and diastolic dysfunction at the same time. In addition, treatment of AF with rhythm conversion strategies might be beneficial in the prevention of significant TR development.

Fibrous skeleton in the heart has strong resistance to elongation or dilatation. The mitral valve annulus has a completed fibrous ring and is firmly anchored along the circumference of the anterior leaflet by the tough fibrous skeleton of the heart. However, less fibrous tissue is developed in the tricuspid valve and has distensible fibro-adipose tissue.[12] Therefore, the right atrium (RA) usually enlarges

more easily than LA in the setting of AF because there is less fibrous skeleton in tricuspid annulus than mitral valve. Then, there is a vicious cycle that tricuspid annular dilatation contributes to development of TR and it causes further dilation of RA. AF turned out to be one of the most important determinants of TR late after surgery.[13] Many reported that AF was identified as a predictor of severe TR after mitral valve surgery,[14-18] however, Zhou et al.[19] found that lone AF without mitral valve surgery could also cause significant TR.

In a study of 170 patients with AF, Zhao et al.[20] divided patients into severe vs. non-severe TR groups and reported that predictors for severe TR were age, female gender, and RV systolic pressure. The definition of severe TR is different from worsening progressive TR, but similarly in the present study female patients showed more progression and older patients had a tendency toward TR progression. At the baseline in our study, there were no differences in RVSP between the two groups. However, mean RVSP in the progression group was increased from 38.7 mmHg to 47.8 mmHg ($p < 0.001$) and follow-up RVSP was higher in the progression group. This is because our study is not merely cross-sectional but also observational. RVSP seems to rise gradually as AF and MR persists. Also, RVSP can be usually underestimated when TR progress because of the decompression effect of severe TR.

Relative to the treatment modality of AF, antiarrhythmic agents, cardioversion, ablations for atrial fibrillation were performed more in the non-progression group. Patients had AF at both the time of index and the follow-up echocardiography. The rate control strategy for AF might have reduced AF burden for a limited time duration in the non-progression group. This might be why AF duration was not one of the independent predictors while antiarrhythmic agents were.

Our group once analyzed 76 patients with severe TR and AF and demonstrated that an improvement of LVEF of more than 10% was the only independent predictor of reversible TR.[21] Similarly, in the present study, an initial LVEF $< 50\%$ showed lower progression risk in the univariate analysis (Table 3). In this situation, improving left-sided heart disease seems to be the main inhibitor of TR progression. If we look into it in detail, it is interesting that mean LVEF was not that much lower in the non-progression group than in the progression group (59.1±11.3 vs. 61.7±8.5, p=0.031). Since left heart disease due to cardiomyopathy or valvular heart disease usually has volume overload, small differences in LVEF might cause a bigger change. In this regard, NT-proBNP levels might be higher in the non-progression group than in the progression group in the present study though they are not statistically significant (Table 1).

Despite the fact that RA enlargement tends to more easily develop than LA enlargement, as discussed above, LA diameter is also generally increased in the setting of AF. In the meantime, LA enlargement often develops due to the LV stiffness and diastolic dysfunction.[22-24] In the present study E/e' was associated with TR progression and this suggests that diastolic dysfunction may synergistically affect the pressure of right-side heart and TR.

There are several limitations in this study. First, the present study was conducted in a single center in a retrospective fashion. As a result, all variables needed for analysis could not be collected. Second, there could be a selection bias because only the patients with both initial and follow-up echocardiography could be involved in the final analysis. Third, the follow-up duration of each patient was varied so that the event rate could be affected by that. Fourth, we could only measure the duration of AF based on patient-reported history, though it was not an independent predictor of TR progression in the multivariable analysis. Therefore, there could be an issue as to the accuracy in AF burden. Finally, this study could not observe patients with atrial fibrillation from the onset. Therefore, some patients could have longer AF durations as in the other study.[25] A longitudinal prospective observational study looking at the TR progression in AF patients from the onset is warranted.

In conclusion, the progression of TR was not uncommon, and the incidence was 23.7% in patients with persistent AF. LA diameter over 54 mm, E/e', and no use of antiarrhythmic agents were independent predictors of progression of TR in patients with persistent AF.

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## CONFLICT OF INTEREST STATEMENT

None declared.

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