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Cardiac Image

Evaluation of Right Ventricular Function by Speckle-Tracking Echocardiography in Patients with Ankylosing Spondylitis: A Case-Control Study

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Background: We aimed to evaluate the right ventricular (RV) systolic function in patients with ankylosing spondylitis (AS) compared to healthy subjects by using standard echocardiography and speckle-tracking echocardiography (STE) methods.

Methods: This was a case-control study in which 64 patients (mean age, 55.7 ± 9.2 years; male/female, 53/11), who had AS for at least five years (mean disease duration, 7.1 ± 2.6 years) and 70 age-matched healthy subjects (mean age, 54.9 ± 8.5 years; male/female 55/15) were included. Clinical and laboratory signs of cardiac disease were recorded. The RV systolic function was assessed by standard echocardiography and two-dimensional STE method.

Results: Case and control groups did not show significant difference in terms of clinical and laboratory signs of cardiac disease. RV function parameters in standard echocardiography were statistically similar between AS patients and control subjects. However, RV parameters in STE revealed significantly impaired RV function in AS patients compared to control group. RV-free wall longitudinal strain, RV-free wall longitudinal systolic strain rate, RV-free wall longitudinal early diastolic strain rate, RV-free wall longitudinal late diastolic strain rate were lower, and RV-early diastolic strain rate/RV-late diastolic strain rate ratio was higher for the patients in the AS group ($p < 0.001$ for all).

Conclusions: AS is associated with impaired RV function as shown by STE even if there is no clinical or laboratory sign of cardiac abnormality. STE is more effective than standard echocardiography to detect RV function. Therefore we suggest regular evaluation of RV function in patients with AS.

Key Words: Ankylosing spondylitis • Right ventricle function • Speckle-tracking echocardiography

INTRODUCTION

Ankylosing spondylitis (AS) is a chronic inflammatory rheumatic disease involving the sacroiliac joints and vertebrae.[1] However, it can also impair the functions of other organs over time including the digestive system, eyes, kidneys, and cardiac structures.[2] Cardiac involvement is primarily observed in the form of aortic valve insufficiency and conduction problems, and it is generally asymptomatic. Cardiac failure may develop in some patients, particularly due to deterioration of diastolic function.[3,4] The presence of cardiac involvement worsens the prognosis in patients with AS.[5] An early diagnosis of any cardiac dysfunction associated with AS would allow for the timely treatment and prevention of its progression to cardiac failure.

Transthoracic echocardiography is an imaging method commonly used in clinical practice to evaluate cardiac function. In addition to transthoracic echocardiography, myocardial function can be evaluated in a sensitive and detailed manner with new echocardiographic

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techniques such as speckle-tracking echocardiography (STE). STE is a Doppler-based method that allows for the evaluation of longitudinal myocardium movements during systole,[6] and it is not very different from transthoracic echocardiography in terms of operator experience and repeated measurements.[7,8] However, STE can provide information about the segmental dislocation of myocardial segments, and strain analysis can express the degree of deformation against the forces applied to the myocardium. In addition, STE can measure the degree of dimensional changes of the segment in question, which can then be evaluated as the percent change compared with the initial dimension. Strain rate expresses the temporal change in local deformation rate, e.g. the shortening-elongation function, and both strain and strain rate can be calculated using STE. Negative strain and strain rate values obtained as measurements of the evaluated plane are in the direction of systolic wave shortening. Goebel et al. determined strain and strain rate values for clinical use among various age ranges in healthy adults.[9] Furthermore, two-dimensional (2D) STE allows for the determination of changes at a subclinical level which do not readily lead to clinical manifestations.[10,11]

Although previous studies have reported left ventricular (LV) function in patients with AS,[12] to the best of our knowledge, no study has investigated right ventricular (RV) function in such patients. Thus, in the present study, we aimed to evaluate RV function using STE in patients with AS and compare the results with those of healthy subjects. The results of this case-control study mainly demonstrated a subclinical deterioration in RV function among the patients with AS compared with the healthy subjects.

METHODS

Study population

This case-control study was conducted between March 2013 and June 2014 at the rheumatology outpatient clinic of our center. The AS group included 64 patients (mean age, 55.7 ± 9.2 years; male/female, 53/11) who had AS for at least 5 years. The diagnosis of AS was based on the modified New York criteria.[13] The control group included 70 age-matched subjects (mean age, 54.9 ± 8.5 years; male/female, 55/15). Patients with collagen tissue disease, pregnancy or any known systemic disease including hypertension, hyperlipidemia, diabetes mellitus, chronic renal disease, peripheral vascular disease, coronary heart disease, cardiac failure, significant cardiac valve disease, and rhythm disorders were excluded. Demographics, clinical and laboratory parameters indicative of cardiac diseases (body mass index, blood pressure, heart rate, smoking status, blood creatinine, low-density lipoprotein, and hemoglobin levels), and echocardiographic findings were recorded for all subjects.

Written informed consent was obtained from all patients prior to inclusion in the study. This study was conducted in accordance with the Helsinki Declaration, and the protocol was approved by the Institutional Ethics Committee of our institute.

Echocardiography

An HD15 (Philips, The Netherlands) system and 3.5 MHz transducer were used for transthoracic echocardiographic evaluations. Device adjustments were made to provide the highest frame rate during the evaluation (50-90 frames/s). To reach high frame rates during these procedures, the image window was narrowed to 15-30 degrees, and the recordings were obtained such that the wall under evaluation was in the middle and parallel to the ultrasound beams. Ejection fraction was calculated using the modified Simpson's method. Mitral and tricuspid inflow velocities were taken from apical 4-chamber images using pulse-wave Doppler.

Mitral annulus systolic (Sm) and early diastolic (Em) tissue velocities were measured at the lateral corner of the annulus using tissue Doppler imaging using the pulse-wave Doppler mode from the apical 4-chamber view. The early mitral inflow velocity (E)/Em ratio was calculated to evaluate the LV filling pressure. Tissue Doppler imaging recordings were also obtained from the RV apical 4-chamber focused view with the pulse-wave Doppler sample volume placed on the tricuspid lateral annulus, and the tricuspid annulus peak systolic tissue velocity was then measured. To determine the motion and excursion of the annulus, the M-mode cursor was oriented to the junction of the tricuspid valve plane with the RV free wall using images in the apical 4-chamber view. Maximal tricuspid annular plane systolic excursion

(TAPSE) was determined as the total excursion of the tricuspid annulus from its highest position after atrial ascent to the peak descent during ventricle systole. None of the patients had echocardiographic evidence of pulmonary stenosis or RV outflow tract obstruction; therefore, the RV systolic pressure was considered to be equivalent to the systolic pulmonary artery pressure. The RV systolic pressure was estimated by calculating the systolic pressure gradient between the right ventricle and right atrium according to the maximum velocity of the tricuspid regurgitation jet using the modified Bernoulli equation. This value was then added to the estimated right atrial pressure based on both the size of the inferior vena cava and change in caliber of this vessel with respiration. The sum of RV isovolumic contraction and relaxation time was obtained by subtracting the RV ejection time from the interval between the cessation and onset of the tricuspid inflow velocities using pulse-wave Doppler. The RV myocardial performance index (MPI) was obtained by dividing the sum of both isovolumic intervals by the ejection time. RV isovolumic acceleration (IVA) was measured using tissue Doppler imaging at the lateral tricuspid annulus, and calculated by dividing the isovolumic contraction peak velocity by the time interval between the onset of this wave and its peak velocity.

Assessment of right ventricle using STE

Assessment of RV speckle-tracking strain longitudinal deformation in the RV free wall was conducted with 2D speckle-tracking longitudinal strain using a routine grayscale right ventricle-focused view image, and performed offline using dedicated software (QLab version 7.1, Philips, The Netherlands). Briefly, a region of interest (ROI) was traced with a point-and-click approach on the endocardium at end-diastole in the right ventricle from the right ventricle-focused view. A second, larger ROI was then generated and manually adjusted near the epicardium. The right ventricle was divided into 6 standard segments (basal, middle, and apical), and 6 corresponding time-strain curves were generated. RV free wall longitudinal strain (RV-FWLs) was calculated by averaging each of the 3 regional peak systolic strains along the entire RV free wall. RV free systolic strain rate (RV-Ssr), early diastolic strain rate (RV-Esr), and late diastolic strain rate (RV-Asr) were calculated in the same manner. The RV-Esr/RV-Asr ratio was also calculated.

Statistical analysis

The study data were summarized using descriptive statistics (mean ± standard deviation for continuous variables, percentage for categorical variables). The Kolmogorov-Smirnov test was used to assess the normality of distribution of continuous variables. An independent sample t-test was used for inter-group comparisons of continuous variables. The chi-square test was used to compare categorical variables. All analyses were performed using SPSS software Windows version 17.0 (SPSS Inc., Chicago, Illinois, USA). A two-tailed p value < 0.05 was considered to indicate statistical significance.

RESULTS

Clinical findings

There were no significant differences in age, gender, body mass index, smoking status, vital signs, and laboratory parameters between the AS and control groups (Table 1).

No extra-articular signs or symptoms were noted in the AS group. The mean disease duration was 7.1 ± 2.6 years. Fifty-eight patients (90.6%) received anti-rheumatic drugs, and 6 patients used anti-tumor necrosis factor-alpha (TNFα) agents.

Echocardiographic findings

The echocardiographic parameters are summarized in Table 2. There was no significant difference between the control and AS groups in LV ejection fraction (63.5 ± 4.5 vs. 64.9 ± 5.1, respectively; p = 0.450). In addition, LV end-systolic diameter, LV end-diastolic diameter, interventricular septum thickness, and posterior wall thickness were also similar between the groups (all p > 0.05, Table 2).

The echocardiographic assessment of RV function showed that the mean values of RV ejection fraction, TAPSE, RV-IVA, RV-MPI, RV-fractional area change (FAC), E, Sm, Em, E/Em, deceleration time (DT), and peak systolic tissue velocity were similar between the groups (all p > 0.05, Table 2).

STE findings

The mean systolic pulmonary artery pressure was

Table 1. Demographic, clinical and laboratory characteristics of the study population

Variables	Control group (n = 70)	AS group (n = 64)	p value
Age (years)	54.9 ± 8.5	55.7 ± 9.2	0.480
Male gender	55 (78.0%)	53 (82.8%)	0.085
Smoking	9 (12.0%)	11 (17.2%)	0.186
Body mass index (kg/m^2)	23.8 ± 2.5	22.9 ± 2.6	0.398
Vital signs			
Systolic blood pressure (mmHg)	125.5 ± 12.5	116.3 ± 13.2	0.550
Diastolic blood pressure (mmHg)	75.1 ± 8.0	72.6 ± 7.1	0.520
Heart rate (beats/min)	76.8 ± 9.5	75.2 ± 11.3	0.365
Blood biochemistry			
Creatinine (mg/dL)	0.98 ± 0.35	1.02 ± 0.21	0.755
Hemoglobin (mg/dL)	13.5 ± 2.8	14.1 ± 2.3	0.425
LDL-cholesterol (mg/dL)	115.5 ± 18.5	113.6 ± 16.4	0.678
ESR	6.5 ± 2.5	6.2 ± 1.8	0.350
CRP	0.63 ± 1.4	0.55 ± 1.2	0.485

AS, ankylosing spondylitis; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; LDL, low-density lipoprotein.
Data are presented as mean ± standard deviation or n (%).

Table 2. Echocardiographic parameters of the study population

Variables	Control group (n = 70)	AS group (n = 64)	p value
RV ejection fraction (%)	62.5 ± 15.4	61.4 ± 8.9	0.385
RV-TAPSE (mm)	22.8 ± 5.5	23.6 ± 5.1	0.335
RV-IVA (m/s^2)	2.7 ± 0.9	2.9 ± 0.5	0.550
RV E/e'	7.6 ± 2.1	7.3 ± 1.5	0.085
RV-MPI	0.25 ± 0.08	0.28 ± 0.04	0.455
RV-FAC (%)	41 ± 3.7	40 ± 4.2	0.220
E (cm/s)	84 ± 3.0	82 ± 4.4	0.345
Sm (cm/s)	14.0 ± 2.0	13.9 ± 1.9	0.475
Em (cm/s)	11.0 ± 0.5	11.2 ± 0.9	0.550
E/Em	7.2 ± 1.8	7.3 ± 2.3	0.752
DT (ms)	192.0 ± 35.0	204.2 ± 40.6	0.120
Peak systolic tissue velocity (cm/s)	14.8 ± 2.9	13.6 ± 1.8	0.340
LV ejection fraction (%)	63.5 ± 4.5	64.9 ± 5.1	0.450
LV end-systolic diameter (mm)	25.9 ± 3.8	26.3 ± 3.1	0.325
LV end-diastolic diameter (mm)	46.0 ± 3.5	47.2 ± 3.8	0.425
Inter-ventricular septum thickness (mm)	9.8 ± 1.8	10.0 ± 1.2	0.110
Posterior wall thickness (mm)	9.2 ± 1.5	9.7 ± 1.1	0.185

AS, ankylosing spondylitis; DT, deceleration time; E, early mitral inflow velocity; Em, mitral annulus early diastolic tissue velocity; FAC, fractional area change; IVA, isovolumic acceleration; LV, left ventricular; MPI, myocardial performance index; RV, right ventricular; Sm, mitral annulus systolic tissue velocity; TAPSE, tricuspid annular plane systolic excursion.

higher in the AS group than in the control group (Table 3). 2D STE analysis of the right ventricle revealed that RV-FWLs, RV-Ssr, RV-Esr, and RV-Asr were lower, and RV-Esr/RV-Asr ratio was higher in the AS group than in the control group (all p < 0.001, Table 3).

Independent predictors of RV function

Multivariable analysis controlling for all univariate predictors revealed that the independent predictors of RV (Table 4) function in the patients with AS were male gender (p < 0.001), systolic pulmonary artery pressure (p = 0.01), RV-Ssr (p < 0.001), RV-Esr (p < 0.001), RV-Asr (p < 0.001), and RV-Esr/RV-Asr ratio (p < 0.001).

DISCUSSION

The main finding of this case-control study is subclinical deterioration in RV function among the AS patients compared with the healthy individuals. Rheumatic

Table 3. Right ventricle STE parameters of the study population

Variables	Control group (n = 70)	AS group (n = 64)	p value
Systolic pulmonary artery pressure	18.5 ± 3.5	19.3 ± 3.8	< 0.001
RV-FWLs	18.5 ± 2.0	16.6 ± 2.9	< 0.001
RV-Ssr (/s)	1.4 ± 0.5	1.2 ± 0.2	< 0.001
RV-Esr (/s)	1.3 ± 0.7	1.2 ± 0.1	< 0.001
RV-Asr (/s)	1.5 ± 0.5	1.1 ± 0.2	< 0.001
RV-Esr/RV-Asr ratio	0.98 ± 0.45	1.12 ± 0.32	< 0.001

AS, ankylosing spondylitis; FWLs, free wall longitudinal strain; RV, right ventricular; RV-Asr, RV-free wall longitudinal late diastolic strain rate; RV-Esr, RV-free wall longitudinal early diastolic strain rate; RV-Ssr, RV-free wall longitudinal systolic strain rate.

Table 4. Odds ratios for the likelihood of a deterioration in RV function

	Odds ratio (95%)	p value
Individual components in the adjusted model		
RV-Esr	0.75 (0.40, 0.90)	p < 0.001
Male gender	3.85 (2.75, 5.75)	p < 0.001
Systolic PAPs	4.55 (3.15, 7.60)	p = 0.01
RV-Ssr	0.60 (0.30, 0.95)	p < 0.001
RV-Asr	0.85 (0.55, 0.95)	p < 0.001
RV-Esr/RV-Asr ratio	3.25 (2.95, 5.15)	p < 0.001

Adjusted for age, gender, TAPSE, RV ejection fraction, and RV MPI. Abbreviations are in Tables 3.

inflammatory diseases can cause cardiovascular dysfunction,[3-5,12] and regression of cardiac function may occur with a longer duration of the disease. Various studies have investigated cardiac function levels in AS, and demonstrated systolic and diastolic dysfunction using 2D, M-mode, and Doppler methods at an early stage.[14,15] AS reportedly decreases LV compliance, particularly in diastolic function.[16,17] Systemic inflammation has been suggested to be a cause of damage in cardiac myocytes and to increase collagen deposition in patients with AS.[18] In addition, increased cardiac amyloid levels and cardiac valve insufficiency have been reported to precipitate diastolic dysfunction.[17,19] Previous studies have also reported a deterioration in diastolic function in patients with AS.[17,20] Shang et al. used conventional echocardiography and Doppler methods and found that systolic function is regressed in spondylosis-arthritis.[21] The introduction of STE has allowed clinicians to diagnose subclinical changes which do not cause clinical manifestations and cannot be determined using standard echocardiographic methods. STE has been shown to be able to identify subclinical systolic and diastolic dysfunctions in patients with AS in the absence of clinical signs and symptoms.[22-26]

The opportunity for high resolution evaluations has become possible with the development of imaging modalities, and particularly 2D STE, which is less dependent on the operator and image quality.[27] As in single dimensional STE, LV systolic and diastolic dysfunctions have also been demonstrated by 2D STE in patients with AS compared with healthy individuals. Cardiac dysfunction has even been determined by STE in patients with normal findings in Doppler and tissue Doppler techniques.[12,28]

However, all of the aforementioned studies evaluated LV function, and no previous study has directly assessed RV in patients with AS. Therefore, we evaluated the RV function of 64 patients with AS and 70 healthy volunteers using standard and 2D STE methods with good image quality. Using standard echocardiographic methods, no significant differences were determined between the groups in terms of RV ejection fraction, RV-FAC, TAPSE, RV-MPI, RV-IVA, LV ejection fraction, and cardiac dimensions. This may be because the clinical findings of cardiac dysfunction caused by AS require a longer disease duration than in our cases (7.1 ± 2.6 years). Among the standard echocardiographic parameters, only mean systolic pulmonary artery pressure was higher in the AS group (p < 0.001). Increased pulmonary artery pressure also increases RV afterload and wall strain, and even a slight increase in the pulmonary ar-

tery pressure levels may lead to a dyssynchronous state of the right ventricle.[29,30] Increased end-diastolic pressure of the left ventricle increases the pulmonary artery pressure, and the left ventricle is known to have systolic and particularly diastolic dysfunction in AS.[12,15] Although the presence of AS did not cause any apparent clinical cardiac findings in our study, cardiac changes may have occurred at a subclinical level. In addition, the myocardium of the right ventricle is exposed to inflammation in rheumatic inflammatory diseases, and amyloid deposition, collagen accumulation, and decreased RV compliance are also among the possible causes of subclinical RV dysfunction. As mentioned, STE may provide subclinical findings related to RV dysfunction even at a very early stage.[10,11] Lower mean values of RV-FWLs and RV-Ssr were observed in the AS group in our study (both $p < 0.001$). Increased pulmonary artery pressure can lead to a decrease in RV-Esr and RV-Asr, and this increased pulmonary artery pressure acts like an increase in RV afterload and causes RV diastolic dysfunction leading to a decrease in RV-Esr and RV-Asr.[31-35] In addition, the mean RV-Esr and RV-Asr values of diastolic speckle-tracking parameters were significantly lower in the AS group, and the ratio of these two parameters was higher in the AS group ($p < 0.001$).

The main limitation of this study is the small sample size, which precludes us reaching a definitive conclusion. Moreover, it is not always feasible to obtain appropriate echocardiographic windows to evaluate all of the RV walls by 2D STE analysis. More definitive results may have been obtained if all the ventricular walls had been evaluated in detail and with more samples.

CONCLUSIONS

In conclusion, subclinical systolic and diastolic RV dysfunction may present in patients with AS. 2D STE can be used to determine this dysfunction at an early stage. The clinical findings may become evident with a prolonged disease duration. Meanwhile, monitoring both RV and LV function is important in such patients. Therefore, cardiac function including both RV and LV function should be evaluated using 2D STE as well as standard echocardiographic techniques in patients with AS with a disease duration of at least 5 years.

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COMPETING INTERESTS

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