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ORIGINAL ARTICLE

Diagnostic Utility of Speckle Tracking Echocardiography in Detection of Subclinical Left Ventricular Dysfunction in Patients with Rheumatoid Arthritis

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ABSTRACT

Background: Patients with rheumatoid arthritis (RA) have a shorter life expectancy and a higher risk of cardiovascular death than the general population. Speckle tracking echocardiography may detect early myocardial dysfunction in RA patients.The present study aimed to detect early systolic & diastolic myocardial impairment in RA patients and effects of disease on left ventricular remodeling**Methods:**This case control study that was conducted at cardiology department, Zagazig university hospital on 100 individuals were recruited from rheumatology & cardiology clinics during the period from August 2022 to February 2023. Patients were subdivided into two groups; Study group included 50 patients already diagnosed as Rheumatoid arthritis and Control group included 50 sex- and age-matched healthy adults.**Results:**There was a statistically significant positive correlation between disease duration and the age of the analyzed cases, AP4C LS, AP2C LS, AP3C LS, GLS, and PASP, but not with the other studied parameters. Global longitudinal strain (GLS) was significantly impaired compared to healthy control of the same age and sex group, rather than diastolic dysfunction, and it should be considered the first sign of subclinical cardiac dysfunction in adults.**Conclusion:**GLS measurement using Speckle Tracking Echocardiography (STE) is valuable and reproducible tool in detecting impairment of left ventricle systolic function in RA patients, even in the presence of normal ejection fraction. The degree of systolic function impairment is correlated to RA duration.

Keywords: Left ventricle dysfunction, Rheumatoid Arthritis, Speckle-tracking Echocardiography.

INTRODUCTION

The majority of synovial joints are affected by the chronic, systemic, inflammatory disease known as rheumatoid arthritis (RA). As a disease progresses, the erosion of bone and cartilage destroys the joints, resulting in deformities. While the synovium of the diarthrodial joints is the only area where the basic pathology of RA is found, chronic cases can also affect other non articular organs. Cytokines that promote synovial illness and the inflammatory response are the causes of pathophysiological changes in extra-articular tissues over the course of the disease, over 40% of RA patients develop extra-articular symptoms [1].Heart failure (HF), rheumatoid nodules, atrial fibrillation, myocarditis, pericarditis, and secondary amyloidosis are all symptoms of RA's cardiac involvement. Patients with RA have a twofold increase in the risk of developing HF. Accelerated atherosclerosis, which is facilitated by inflammatory cytokines, leads to increased cardiovascular

morbidity. However, the established risk factors do not fully explain the higher risk of HF. Amyloidosis, inflammatory mediators, antirheumatic medication, and ischemic heart disease are a few of the etiopathogenetic routes that cause HF in RA Previous research from industrialized nations have revealed that HF is linked to a higher chance of dying from RA. Indian RA patients present later and have greater morbidity rates than their Western counterparts [2].

Early systolic dysfunction can be identified by measuring the strain (% of the myocardium deformed relative to its initial shape) and strain rate of the whole myocardium or certain cardiac segments in specified planes, which is the pace at which the deformation happens. Doppler is an angle-dependent method, so it is unsuitable for detecting strain (deformation) parameters. Using an angle-independent method, speckle-tracking echocardiography (STE) detects and tracks minute natural reflection patterns in ultrasound B-mode pictures to measure local heart strain and strain rate within predetermined intervals[3].Current study aimed to detect early systolic & diastolic myocardial impairment in RA patients and effects of disease activity on left ventricular remodeling.

METHODS

This case control study that was conducted at cardiology department Zagazig University Hospitals on 100 patients were recruited from rheumatology & cardiology clinics during the period from August 2022 to February 2023.

Inclusion criteria: Patients diagnosed with RA by rheumatologists upon clinical findings and investigations with preserved left ventricular systolic function.

Exclusion criteria:Congenital heart disease, Impaired LV systolic function (LVEF < 50%), ischemic heart disease (diagnosed by stress test or Echocardiography), prior MI, prior PCI or CABG, valvular dysfunction, any rhythm other than sinus rhythm, and poor Echo window.

Sample size:The present study that was conducted on 100 patients, patients were subdivided into two groups; study group included 50 patients already diagnosed as rheumatoid arthritis and control group included 50 sex- and age-matched healthy adults.

Data Collection and Procedures:

All patients were subjected to full history taking involving; age, gender, hypertension, DM, smoking, dyslipidemia, any cardiac symptoms e.g. dyspnea, chest pain, palpitation., etc.General examination includingnon-invasive arterial blood pressure using sphygmomanometer, pulse rate using the radial artery pulsations, neck veins, lower limbs for bilateral pitting edema of the heart failure, chest examination for excluding chest infections, abdominal examination for detecting of hepatomegaly was performed. Cardiac examination inspection and palpation to detect if cardiomegaly present.

Investigations included electrocardiography standard 12 leads, the two groups underwent ECGs (at a voltage of 10 mm/mV and a speed of 25 mm sec) in order to rule out ischemia alterations and identify rhythm. Echocardiography, Two-dimensional speckle tracking echocardiogram and traditional tissue Doppler ultrasound were conducted for the 2 groups using S5-1 probe of Vivid GE 95 machine.

The Tissue Doppler imaging (TDI):

The following parameters were obtained using pulsed wave TDI over the lateral and spatial mitral annulus in the apical four chamber view:

The mean (e') of the peak diastolic velocity at the septal and lateral mitral annuli (e') was computed.

E/e' velocities on average.

The mean (S) of the septal and lateral mitral annuli's peak systolic myocardial velocity (S) was calculated [4].

Two-dimensional speckle tracking:

With ECG gating, images were obtained from the views of the apical four, two, and three chambers. In order to maintain a grayscale frame rate between 30 and 70 frames per second, the gain settings were tuned. Every heart cycle was recorded for three loops. To prevent breathing artifacts, every image was taken while holding still[5].

The three-chamber view, or apical long-axis image, was examined first. The complete endocardial border, starting at one end of the mitral annulus and finishing at the other, was traced in the end-systolic frame. The whole myocardial thickness was included in the region-of-interest (ROI) that the software produced. The ROI's width was changed by hand. The apical two- and four-chamber pictures underwent the same procedure twice. The global longitudinal strain (GLS) was calculated by averaging the strain values for each section.

Ethical and administrative considerations: A written informed consent was taken from all patients and the study was accepted by the Research Ethical Committee of Faculty of Medicine, Zagazig University (ZU-IRB#9633-2-7-2022). The study was carried out according to the code of Ethics of the World Medical Association (Declaration of Helsinki) for studies including humans.

STATISTICAL ANALYSIS

The data was analyzed using SPSS (Statistical Program for Social Science) version 24. Quantitative data was expressed using the mean ±SD. The qualitative data were expressed using percentages and frequencies the mean, or average, is the central value that is, the total number of values divided by the sum of the values of a discrete set of integers. The standard deviation (SD) is a measure of the dispersion of a set of data. A high SD indicates that the values are scattered across a wider range, whereas a slow SD indicates that the values normally tend to be near the given mean. A significant t-test was employed to compare two means. The chi-square test was employed to compare non-parametric data sets. A P-value of less than 0.05 was deemed significant. P-values less than 0.001 were regarded as extremely significant. P-value greater than 0.05 was deemed not significant. Correlation by Pearson's correlation agreement by Kappa.

RESULTS

Table (1) revealed that there was no statistically significant difference in the mean age of the RA group (30.8 ±7.6) and the control group (30.4 ±7.4) years between both studied groups as regards demographic data ($p > 0.05$).

The difference in the number was not statistically significant of diabetic patients between the three patients (6%) in the RA group and the six patients (12%) in the control group (P value 0.486). The number of hypertension patients in the control and RA groups was the same (18). In terms of risk factor, there was no statistically significant difference between the two groups tested ($p > 0.05$) (Table 2).

Table (3) illustrated that there was a highly statistically significant difference between the studied groups in terms of LVESV, but no statistically significant difference in terms of LVEDV or EF.

Table (4) showed that the Mean S wave velocity of the control group was [9.74 ± 0.56] cm/s and [9.82 ±0.58] in the RA group (P value 0.498), showing no statistically significant difference. With a statistically significant difference (P value <0.05), the mean e~ velocity was [15.7 ± 1.8] cm/s in the RA group and [16.5 ± 1.07] cm/s in the control group. With no statistically significant difference (P value 0.646), the Mean E wave velocity was [95.19 ± 14.31] cm/s in the control group and [93.80 ± 15.85] cm/s in the RA group. The control group's mean a wave velocity was [70.1 ± 11.21] cm/s, whereas the RA group was [72.96 ± 17.54] cm/s. There was no statistically significant difference

between the two groups (P value 0.476). With no statistically significant difference (P value 0.664), the mean E/A ratio for the RA group was [1.33 ± 0.13] and for the control group it was [1.35 ± 0.14]. There was no statistically significant difference (P value 0.167) in the Mean Average E/e for the RA group and the control group, which was [5.7 ± 1.25] and [6.42 ± 1.1], respectively.

There was no statistically significant difference between the two groups in Mean TR velocity, which was comparable at [2.12 ± 0.26] cm/s for the RA group and [2.4 ± 0.35] cm/s for the control group(P value 0.132). For both the control and RA groups, the mean LAVI was [21.62 ± 2.89] ml/m^2 and [21.62 ± 3.82] ml/m^2, respectively, with no statistically significant difference (P value 1) (Table 4).The RA group's AP4C LS values were lower than those of the control group; the RA group's mean AP4C LS was [-19.19 ± 3.18] %, whereas the control group's was [-21.4 ± 2.69] % (P value <0.001).

The RA group's mean AP2C LS was [-19.27 ± 2.21] % for the RA group and [-22.8 ± 3.0] % for the control group, indicating a significant difference in AP2C LS values (P value <0.001).

The RA group's mean AP3C LS was [-17.47 ± 2.17] % for the RA group and [-20.1 ± 2.14] % for the control group, indicating a significant difference in AP3C LS values (P value <0.001).

The GLS values of the RA group were lower than those of the control group; the mean GLS was [-18.95 ± 2.02] % for the RA group and [-21.4 ± 2.1] % for the control group (P value <0.001) (table 4).

Table (5) showed that there was a statistically significant positive correlation between disease duration and age (P 0.001, R = 0.480) as well as PASP (P 0.001, R = 0.628). There was also a statistically significant positive association between the duration of the disease AP4C LS (%) (P< 0.05, R = 0.341), AP2C LS (%) (P< 0.05, R = 0.389), AP3C LS (%) (P< 0.05, R = 0.403) and GLS (%) (P< 0.05, R = 0.417), whereas there is no statistically significant difference between disease duration and the other parameters.

Table (1): Comparison between control group and RA group regarding demographic data.

Variable	**RA group N=50**		**control group N=50**		**T test**	**P value**
Age: (years):						
Mean ± SD Range	30.8 ± 7.6 18-45		30.4±7.4 18-45		0.125	0.923
	No.	**%**	**No.**	**%**	**χ^2**	**P value**
Sex:						
Female	50	100	50	100	Fisher test	1

χ^2 =chi square test

Table (2): Comparison between control group and RA group regarding cardiovascular risk factors.

Variable	RA group N=50		control group N=50		χ^2	P value
	No.	%	No.	%		
DM:						
Diabetic Non diabetic	47 3	94.0 6.0	44 6	88.0 12.0	Fisher test	0.486
Hypertension:						
Hypertensive Non hypertensive	32 18	64.0 36.0	32 18	64.0 36.0	0.0	1.0

Table (3): Comparison between control group and RA regarding conventional echocardiography parameters.

Variable	RA group N=50	control group N=50	T test	P value
LVEDV(LV):				
Mean ± SD Range	109.4±24.04 35-159	103.7±11 60-130	1.51	0.134
LVESV(LV):				
Mean ± SD Range	46.42±7.6 24.4-58	40.89±3 38-54	0.399	<0.05
EF (%):				
Mean ± SD Range	67.3 ± 3.34 60 – 73	67.56 ± 3.77 59 – 73	-0.25	0.801

Table (4): Comparison between control group and RA regarding tissue Doppler, diastolic function and strain parameters.

Variable	RA group N=50	control group N=50	T test	P value
Mean S(cm/s)				
Mean ± SD Range	9.82 ±0.58 6.2 – 10.7	[9.74 ± 0.56] 7.1 – 10.7	0.73	0.498
AP4C LS(%):				
Mean ± SD Range	-19.19 ± 3.18 -25 – -12.5	-21.4 ± 2.69 -27.1 – -15.2	3.78	**<0.001 (HS)**
AP2C LS(%):				
Mean ± SD Range	-19.27 ± 2.21 -23.2 – -11	-22.8 ± 3.0 -29.1 – -15.4	4.25	**<0.001 (HS)**
AP3C LS(%):				
Mean ± SD Range	-17.47 ± 2.17 -23 – -11	-20.1 ± 2.14 -24.2 – -15.4	6.02	**<0.001 (HS)**
GLS (%):				
Mean ± SD Range	-18.95 ± 2.02 -23.63 – -14.83	-21.4 ± 2.1 -24.8 – -15.8	5.56	**<0.001 (HS)**
E velocity(cm/s):				
Mean ± SD Range	93.8 ± 15.85 67 – 132	95.19 ± 14.31 69 – 124	0.46	0.646
A velocity(cm/s):				
Mean ± SD Range	72.96 ± 17.54 39 – 116	70.1 ± 11.21 47 – 89	0.234	0.476
Mean e`(cm/s):				
Mean ± SD Range	15.7±1.8 9.35-19.7	16.5±1.07 14.9-20	2.55	**<0.05 (S)**
E/A ratio:				
Mean ± SD Range	1.33 ± 0.13 1.16 – 1.92	1.35 ± 0.14 1.1 – 1.6	0.436	0.664
Average E/e`:				
Mean ± SD Range	6.42 ± 1.1 4.08 – 9.17	5.7 ± 1.25 3.52 – 8.5	1.39	0.167
PASP				
Mean ± SD Range	31.98±10 18-68.5	29.6±9.3 18-62	1.16	0.245
TR velocity(m/s)				
Mean ± SD Range	2.12 ± 0.26 1.6 – 2.6	2.4 ± 0.35 1.8 – 3.4	1.51	0.132
LAVI (ml/m2)				
Mean ± SD Range	21.62 ± 3.82 16 – 31	21.62 ± 2.89 16 – 31	0.0	1

Table 5: Correlation of duration of RA with the other studied parameters in RA group.

Variable	Duration of the disease	
	R	**P**
Age(years)	**0.480**	**<0.001 (HS)**
Weight(kg)	0.078	0.592
Height(cm)	-0.182	0.206
BMI(Kg/m2)	0.227	0.112
SBP (mmHg)	-0.075	0.606
DBP (mmHg)	-0.059	0.683
LVEDV (lv)	-0.245	0.087
LVESV (lv)	-0.213	0.137
EF (%)	0.000	0.999
AP4C LS (%)	**0.341**	**<0.05 (S)**
AP2C LS (%)	**0.389**	**<0.05 (S)**
AP3C LS (%)	**0.403**	**<0.05 (S)**
GLS (%)	**0.417**	**<0.05 (S)**
Mean e`	0.145	0.314
E velocity(cm/s)	-0.105	0.467
A velocity(cm/s)	0.011	0.939
E/A	-0.095	0.511
AverageE/e`	0.141	0.329
PASP	0.628	**<0.001 (HS)**

r is for Pearson's correlation co-efficient

DISCUSSION

Extra-articular manifestations of RA include neurological disorders such as Multiplex polyneuropathy and mononeuropathy, hematological disorders (anemia, eosinophilia, thrombocytosis), cardiac disorders (pericarditis, left ventricular dysfunction, and pericardial effusion), lung diseases (including nodules, fibrosis, and pleural effusion), and ocular conditions (including scleritis, episcleritis, and scleromalacia perforans). The presence of extra-articular involvement not only suggests that the underlying disease is severe and progressing, but it also indicates a higher rate of morbidity and mortality. The death rate for RA patients is 1.5–1.6 times higher than the overall population. The most frequent cause of death that is identified is cardiovascular disease, which is followed by infections, lung, and kidney diseases [6].Patients with RA are more than 50% more likely than the general population to die from cardiovascular causes, develop cardiovascular events earlier in life, and have shorter life expectancies. Furthermore, it is possible that chronic inflammation and inflammatory myocardial infiltration are the cause of these clinical occurrences, which are only partially reliant on traditional cardiovascular risk factors [7].

An accessible and noninvasive technique for evaluating cardiac involvement in RA patients is echocardiography. It can be used to quantify global longitudinal strain (GLS), screen for valve dysfunction, and detect early myocardial dysfunction in order to prevent early mortality[8].In this investigation, we found that the demographic data for the two groups under consideration did not have statistically significant difference from one another ($p > 0.05$).Ali *et al.* [9]concluded thus, in a comparison study of groups with connective tissue illnesses, the demographic information for the cases and control groups does not differ statistically significantly ($p>0.05$).Naseem *et al.* [10] found that the variations between the three groups in his study' Systolic, diastolic blood pressure, age, sex, and smoking status were not statistically significant.

In this study we proved that between the two study groups, the Risk factor did not differ substantially ($P>0.05$). Ali et al. [9] found that the risk factors like (smoking, hypertension, and dyslipidemia) is not statistically significant ($p>0.05$) between two groups . Benacka et al. [11] found that the traditional risk factors for cardiovascular disease (triacylglycerol, blood pressure, smoking glycaemia, and total cholesterol) were identical between RA patients and controls.

In this study we found that there is high notable variation in LVESV between the groups under study ($p <0.05$).Midtbø *et al.* [12]have demonstrated that, in comparison to patients with active illness, those with RA better left ventricular performance (as determined by end-systolic volume, global longitudinal strain, and stress-corrected mid-wall shortening) in remission. Several mechanisms have been proposed to explain LV systolic failure in RA, including endothelial dysfunction leading to decreased perfusion, myocyte dysfunction, oxidative stress, and interstitial fibrosis. These could explain our finding that disease activity dictated subclinical LV systolic impairment in our patient cohort.Ji*et al.* [13] found that the RA Group had much lower LVGLS and significantly higher LVESV compared to the control group (all $P < 0.05$).Patients with rheumatoid arthritis had longer IVCTs (Isovolumic Contraction time), poorer LVEFs, and larger LV end systolic volumes in research by Benacka *et al.* [11**]** Additionally, a greater E/E' ratio and IVRT prolongation indicated a significantly higher degree of diastolic cardiac dysfunction in the individuals. A noteworthy decrease in the GLS was also noted in the STE assessment.

In this study we illustrated that the Mean e` velocity was [16.5 ± 1.07] cm/s in the control group and [15.7 ± 1.8] cm/s within the RA group when the difference was statistically significant (P value <0.05).These findings are consistent with those of Du Toit *et al.* [14], they found that the patients in the RA group had a significant decrease in the mean é velocity wave ($P = 0.01$) and ($P < 0.001$), respectively.

The results of this study showed that the RA group's AP4C LS values were lower than those of the control group; the RA group's mean AP4C LS was [-19.19 ± 3.18] %, while the control group's was [-21.4 ± 2.69] % (P value <0.001). The RA group's mean AP2C LS was [-19.27 ± 2.21] % for the RA group and [-22.8 ± 3.0] % for the control group, indicating a significant difference in AP2C LS values (P value <0.001). The RA group's mean AP3C LS was [-17.47 ± 2.17] % for the RA group and [-20.1 ± 2.14] % for the control group, indicating a significant difference in AP3C LS values (P value <0.001).These findings are consistent with those of Nikdoust *et al.* [15], they found that, in comparison to the healthy controls, the RA group's AP2C LS findings significantly decreased ($P = 0.005$), while the AP3C LS results demonstrated a significant drop ($P = 0.006$) and the LV GLS results showed a significant decrease ($P = 0.02$).

In this investigation, we demonstrated that, with a P value <0.001, the mean GLS was lower in the RA group as compared to the control group, coming in at [-21.4 ± 2.1] % for the former and [-18.95 ± 2.02] % for the latter.Ali *et al*. [9] found that, in a comparison analysis, there was a significant difference in GLS between the Cases and Control groups (P<0.001).Nikdoust *et al*. [15] revealed a noteworthy drop in the case group's LV GLS as compared to the healthy controls.Naseem*et al*. [10] found that, in comparison to active patients, the LV GLS value was considerably lower than that of the control group and RA patients who were in remission (less negative) (p = <0.001).Tański *et al*.[16] concluded that, in terms of global values, patients with RA had statistically significantly lower peak GLS and peak GCS values than patients in the control group. Meune *et al*. [17], who looked at 27 cases of RA found that there was no discernible difference in systolic strain between the patients and the control group, which is in contrast to our findings. Their technique for measuring the strain could be one reason for this result. STE, a more automated and sensitive method of detecting strain, was not used in favor of tissue Doppler.

In this study we illustrated that a positive statistically significant correlation was discovered between the duration of the illness and AP4C LS (%) (P < 0.05, R = 0.341), AP2C LS (%) (P < 0.05, R = 0.389), AP3C LS (%) (P < 0.05, R = 0.403) and GLS (%) (P < 0.05, R = 0.417).Conversely, Du Toit *et al*. [15] did not find a significant relationship between GLS and the length of the disease. Their inclusion criteria provide an explanation for this. Patients with clinically obvious myocarditis who were not included in our trial were included in theirs.

Limitations:

Because this was a single-center trial, the current findings need to be verified in a larger multicenter study with a small number of patients before the use of speckle tracking echocardiography on RA patients becomes normal clinical practice. We did not compare the LV function produced by 2D-STI to the LV function acquired by 3D-STI, but we expect that this comparison will be incorporated in future studies.

CONCLUSION

Global longitudinal strain (GLS) testing in cases where the ejection fraction is normal, STE is a useful and reliable technique for identifying worsening in the left ventricular systolic performance in patients with RA. The duration of RA disease is correlated with the degree of impairment of systolic function.

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