# Impaired myocardial deformation in psoriatic arthritis patients assessment by speckle tracking echocardiography

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## ABSTRACT

**Objective:** We aimed to evaluate left ventricular (LV) mechanics of patients diagnosed with PsA and no clinical evidence for cardiovascular disease (CVD) using a novel, more sensitive technique, which evaluates myocardial deformation in multidimensional planes for the detection of impaired LV function.

**Methods:** The study enrolled 31 PsA patients and sex-age matched 19 healthy controls. All participants underwent conventional echocardiography and 2-dimensional speckle tracking echocardiography (STE). Global longitudinal, circumferential, and radial strain were measured.

**Results:** Although patients with PsA had normal LV ejection fraction, the myocardial deformation in multidimensional planes was impaired. STE analysis results showed that PsA patients had significantly lower global longitudinal strain (mean±S.D. -17.11±2.83 % and -19.29±2 % respectively, p=0.005), global circumferential strain (mean±S.D. -14.28±3 % and -20.34±4.78 % respectively, p<0.001) and global radial strain (mean±S.D. 29.26±10 % and 46.54±17 % respectively, p<0.001) versus control group. However, no correlation was found between longitudinal, radial, and circumferential strains and disease-related risk factors.

**Conclusion:** Subclinical impaired myocardial deformation was common in patients with PsA even with no clinical evidence for CVD. Thus, the use of this novel imaging technique could provide additional benefits for determining cardiovascular involvement at an early stage and risk stratification in PsA patients.


**Keywords:** Cardiovascular risk; Echocardiography; Psoriatic arthritis.

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## INTRODUCTION

Psoriatic arthritis (PsA) is a type of chronic inflammatory arthritis associated with psoriasis and affects 0.3%--1% of the general population[1]. Increased mortality has been reported in PsA patients and cardiovascular disease (CVD) was found to be the leading cause of morbidity and mortality among these patients[2]. Currently, inflammation is believed to have a major role in the increased risk of CVD in rheumatic diseases, particularly rheumatoid arthritis (RA)[3]. Inflammation may precipitate atherosclerosis by affecting CVD risk factors directly and/or indirectly[3,4]. A mortality study reported a 1.3-fold higher rate of CVD-related death among PsA patients compared to control group[2]. In another study with prospective follow-up, psoriasis was identified as a potential independent risk factor for myocardial infarction based on a comparison between psoriatic and non-psoriatic patients who had a myocardial infarction[5]. Thus, earlier detection of myocardial involvement and development of individualized treatment strategies for patients are important because such efforts may have a potential limiting effect on long-term morbidity and mortality. Gonzalez-Juanetey et al. showed that actively treated PsA patients without cardiovascular risk factors or clinically evident cardiovascular disease do not exhibit silent subclinical echocardiographic abnormalities[6]. On the other hand, Shang et al. showed that by using conventional echocardiography and tissue Doppler imaging (TDI) among PsA patients without established CVD disease and in the absence of traditional CV risk factors have a high prevalence of subclinical LV dysfunction[7]. Limitations of conventional echocardiography include its dependency on angle and its ability to detect only the deformations within the ultrasound beam associated with sound waves even though myocardial deformation is actually three-dimensional[8].

Recently, speckle-tracking echocardiography (STE),

an angle-independent technique, was proposed as a reliable and sensitive method for assessment of subclinical myocardial dysfunction[9]. In recent studies, STE was shown to be a sensitive method for detection of preclinical myocardial dysfunction before changes in LV ejection fraction in patients with systemic inflammatory conditions9, particularly in those with RA[10], systemic sclerosis[11] and PsA[12].

In our study, STE was used to measure LV deformation and function in 3 dimensions (longitudinal, radial, and circumferential strain) and their relationships to PsA were evaluated.

## METHODS

### STUDY POPULATION

Thirty one patients who admitted to Rheumatology outpatient clinic between November 2011 and April 2012 and met CASPAR Working Group criteria for classification of PsA[13] were enrolled cross-sectionally in the study. Patients with known diabetes mellitus (ie., patients with a history of diabetes who were on a diabetic diet or on treatment with oral hypoglycemic drugs or using insulin, fasting blood glucose >126 mg/dl), chronic renal disease (creatinine> 1.3 mg/dl), a history of angina pectoris, coronary artery disease, acute coronary syndrome or coronary revascularization, atrioventricular block or bundle branch block, atrial fibrillation, valvular heart disease, heart valve replacement or peripheral artery disease were excluded. Hypertension was defined as a systolic blood pressure >140 mmHg or diastolic blood pressure >90 mmHg. Positive family history, smoking, obesity and hypertension were considered as CVD risk factors. Randomly selected, sex- and age-matched 19 subjects without any known cardiovascular disease were enrolled as controls. The study was approved by ethics committee and conducted in accordance with the World Health Organization- Declaration of Helsinki principles. Written informed consent was obtained from all patients and healthy controls.

### CLINICAL EVALUATION OF PSA PATIENTS

A 100-mm visual analogue scale was used for patient and physician global assessments. Joint activity was assessed using Disease Activity Score in 28 joints (DAS 28) in patients with peripheral joint involvement[14]. Skin disease activity was assessed using Psoriasis Area Severity Index *(PASI)* and recorded in the protocol form[15]. The Health Assessment Questionnaire (HAQ) was completed by all patients and results were recorded in the form[16].

### CONVENTIONAL DOPPLER ECHOCARDIOGRAPHY AND TISSUE DOPPLER IMAGING

All subjects were imaged in the left lateral decubitus position with a commercially available system (VIVID 7, General Electric-Vingmed Ultrasound, Horten, Norway). Ejection fraction (EF) was measured with the modified biplane Simpson's method from the apical 4- and 2-chamber views[17]. LV mass was calculated according to Devereux formula[18]. LV hypertrophy was defined as an LV mass indexed (LVMI) to body surface area (BSA) that exceeded 110 g/m$^2$ for women and 125 g/m$^2$ for men[19]. Concentric geometry was diagnosed if the RWT (relative wall thickness) exceeded 0.44 for both men and women. Concentric remodeling was diagnosed if LVMI was normal but RWT was high[20]. The early filling (E) and atrial (A) filling peak velocities, E/A ratio, and deceleration time (DT) of early filling and isovolumic relaxation time were measured from transmitral flow. Peak mitral annular myocardial velocity of wall of the LV (lateral) was recorded with real-time pulsed wave tissue Doppler method. The early diastolic mitral annular velocity (e ) was measured from the lateral mitral annulus, and the ratio of early mitral diastolic inflow velocity to early diastolic mitral annular velocity (E/ e ), which is an index of LV filling pressure, was calculated[21].

### SPECKLE TRACKING ECHOCARDIOGRAPHY

STE was measured using a commercially available speckle tracking system in an ECHOPAC (ver. 6.3, GE Vingmed, Horten, Norway) workstation. In this system, the displacement of speckles of myocardium in each spot was analysed and tracked from frame to frame. We selected the best quality digital 2D image cardiac cycle, and the left ventricle endocardium was traced. Regarding adequate tracking quality, the system automatically generates an acceptable or unacceptable tracking quality. We systematically accepted only segments that received an acceptable tracking quality for analysis. To optimize speckle tracking, 2D grey-scale harmonic images were obtained at a frame rate of 60--90 frames/s. Longitudinal strain was assessed with AFI (automaticall functional imaging). At first, the end-systolic frame was defined in the apical long-axis view. The closure of the aortic valve was marked, and the AFI software measured the time interval between the R-wave and aortic valve closure, which was used as

event timing. We manually defined 3 index points (2 points at the base of LV and one at apex). AFI algorithm automatically traced 3 concentric lines on the endocardial border, mid-myocardial layer, and epicardial border, and followed the endocardium from this single frame throughout the cardiac cycle. The left ventricle in each apical image is divided into 6 segments, and the tracking quality for each segment is validated by the operator. Then, the AFI algorithm tracks the percent of wall lengthening and shortening in a set of 3 longitudinal 2D-image planes. The peak systolic longitudinal strain for each segment was displayed based on a 17-segment model for each plane, and the results of all 3 planes were combined in a single bull's-eye summary. Global longitudinal peak strian was automatically calculated as an averaged value of peak longitudinal strain in all 3-image planes (apical 2-, 4- chamber and long axis views). For circumferential-radial strain, event timing was marked. Images were obtained in parasternal short axis view and basal and papillary muscle level for strain velocity. Automatically traced 3 concentric lines on the endocardial border, mid-myocardial layer, and epicardial border, and followed the endocardium from this single frame throughout the cardiac cycle. The left ventricle in parasternal short axis image is divided into 6 segments (septum, lateral, anterior, inferior, anterior septum, posterior), and the tracking quality for each segment is validated by the operator. The average of peak systolic circumferential and radial strain values from the three short-axis views was calculated to derive the global LV circumferential and radial strain[22].

### INTRA-OBSERVER VARIABILITY

All echocardiographic studies and measurements were performed by an experienced cardiologist (T.S.) who was blinded to previously obtained data. In our laboratory, the intra-observer variability was as follows: r=0.98 for two dimensional and M-mode echocardiographic measurements; r=0.97 for Doppler measurements; and r=0.98 for speckle tracking echocardiographic measurements.

### LABORATORY STUDIES

C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) was obtained from venous blood samples of patients. CRP level was quantified using an Abbott Architect c16000 device by immunoturbidimetric method. ESR was measured using an Alifax test1 device by laser method.

### STATISTICAL METHOD

Statistical analyses of study data were conducted using "SPSS for Windows 20.0 (SPSS, Chicago, IL, USA) software package. Results were expressed as mean±SD in case of normal distribution and median (intrequartile range [IQR]) in the absence of normal distribution. Pairwise comparisons of groups were made using independent t-test for numerical variables with normal distribution and chi-square test or Fisher's exact test as appropriate for categorical variables. Numerical data without normal distribution were analyzed using Mann--Whitney U test. Associations between echocardiographic and clinical variables were evaluated using Pearson's correlation test. All tests were two-way and a p value below 0.05 was considered statistically significant.

## RESULTS

### CLINICAL FINDINGS AND TREATMENT OF PSA PATIENTS

Demographic and clinical findings of 31 patients and 19 controls enrolled in the study are shown in Table I. The mean age of PsA patient group was 41 years and 68% were females. The median duration of PsA condition was 5 years with a mean DAS 28 score of 2.05. None of the patients in patient or control groups had clinical evidence for CVD. In the study, 6 patients (19%) used a non-steroidal antiinflammatory drug (NSAID), 25 (80%) used methotrexate, 1 patient (3%) used leflunomide and 12 patients (39%) received an anti-tumor necrosis factor-alpha (TNF) agent and 8 patients (26%) received combination therapy with methotrexate and anti-TNF alpha. Among PsA patients, 27 patients (87%) were using corticosteroid. In the study, 10 PsA patients (32%) and 4 control subjects (21%) were smoking and 15 PsA patients (48%) and 8 control subjects (42%) had hypertension. As for body mass index (BMI), those patients with a BMI of <25 kg/m2 were considered normal, 25-29.9 kg/m2 overweight and ≥30 kg/m2 obese. Among PsA patients, eleven were obese and there were four obese patients in the control group. There was no difference between patient and healthy control groups in age, gender, BMI, smoking, presence of hypertension and the prevalence of body surface area variables ($p > 0.05$).

### CONVENTIONAL DOPPLER ECHOCARDIOGRAPHY AND TDI RESULTS

Results of standard conventional echocardiography and

**TABLE I. DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF THE STUDY POPULATION**

| Variables | Control group (n=19) | PsA patients (n=31) | p |
|---|---|---|---|
| Age, years±SD | 41±8 | 41.3±11 | 0,888 |
| Gender (Male/Female), n | 9/10 | 10/21 | 0,285 |
| Age at PsA onset, years ±SD | | 33.7±11.8 | |
| Duration of psoriasis, years±SD | | 13 (7-20) | |
| Duration of PsA, years±SD | | 5 (3-10) | |
| Body surface area, $m^2$±SD | 1.8±0.13 | 1.8±0.16 | 0,719 |
| Body mass index, $kg/m^2$±SD | 27±4.2 | 28±4.6 | 0,454 |
| Smoking status, n (%) | 4 (21) | 10 (32) | 0,392 |
| Presence of hypertension, n (%) | 8 (42) | 15 (48) | |
| Systolic blood pressure, mmHg (IQR) | 120 (120-130) | 130 (120-140) | 0,156 |
| Diastolic blood pressure, mmHg(IQR) | 80 (80-85) | 80 (80-90) | 0,347 |
| HAQ score (IQR) | | 0.2 (0-0.6) | |
| DAS28 score ±SD | | 2±0.7 | |
| PASI score(IQR) | | 0 (0-0.4) | |
| Sedimentation, mm/h (IQR) | | 9 (5-15) | |
| C-reactive protein, mg/dl (IQR) | | 0.4 (0.2-0.7) | |
| Involvement pattern of the disease, n (%) | | | |
| Peripheral/Peripheral and axial | | 21 (68)/10 (32) | |
| Treatment | | | |
| DMARDs, n (%) | | 19 (61) | |
| Biologics, n (%) | | 12 (39) | |
| History of steroid use, n (%) | | 27 (87) | |

Values are expressed as Mean±SD or Median (IQR) unless otherwise indicated. PsA: Psoriatic arthritis; HAQ: Health Assessment Questionnaire; DAS28: Disease Activity Score in 28 Joints; PASI: Psoriasis Area and Severity Index; DMARD: Disease-modifying antirheumatic drugs.

TDI measurements are shown in Table II for patient and control groups. No significant difference was found between the two groups with respect to LV diameters, LV wall thickness and mass (p>0.05). LV ejection fraction, an indicator of systolic function, was found to be at normal range in both groups. No significant difference was found between the two groups with respect to early filling (E) and atrial (A) filling peak velocities, E/A ratio, and deceleration time (DT), early diastolic mitral annular velocity (e ) and the ratio of early mitral diastolic inflow velocity to early diastolic mitral annular velocity (E/ e ) (p>0.05).

### RESULTS OF SPECKLE TRACKING ANALYSIS

Myocardial strain was measured in three different directions using speckle-tracking analysis to investigate impaired myocardial deformation and function (Table III). Compared to control groups, PsA patients were found to have markedly lower values of global longitudinal strain, global circumferential strain and global radial strain (Table III) (Figure I). STE analysis results showed that PsA patients had significantly lower global longitudinal strain (mean±S.D. -17.11±2.83 % and -19.29±2 % respectively, p=0.005), global circumferential strain (mean±S.D. -14.28±3 % and -20.34± 4.78% respectively, p<0.001) and global radial strain (mean±S.D. 29.26±10 % and 46.54±17 % respectively, p<0.001) versus control group. However, there was not any association of global longitudinal, global circumferential and global radial strain analysis results with disease-related risk factors.

## DISCUSSION

In this study STE showed that in PsA patients there may be impaired myocardial deformation before the clinical findings related to myocardial dysfunction appeared.

STE technique provides myocardial motion in multiple directions[23-25]. In the earlier stages of heart failure,

**TABLE II. COMPARISON OF LEFT VENTRICULAR FUNCTIONS OF PSA PATIENT AND CONTROL GROUPS AS DEMONSTRATED BY CONVENTIONAL DOPPLER ECHOCARDIOGRAPHY AND TISSUE DOPPLER ECHOCARDIOGRAPHY**

| Doppler echocardiography parameters | Control group (n=19) | PsA patients (n=31) | p |
|---|---|---|---|
| Interventricular septal thickness, cm±SD | 1±0.1 | 1±0.1 | 0,059 |
| Posterior wall thickness, cm±SD | 0.9±0.1 | 1±0.1 | 0,200 |
| Left ventricular end-diastolic diameter, cm±SD | 4.8±0.4 | 4.7±0.5 | 0,687 |
| Left ventricular end-systolic diameter, cm±SD | 2.9±0.3 | 2.7±0.3 | 0,129 |
| Relative wall thickness±SD | 0.4±0.04 | 0.4±0.06 | 0,055 |
| Left ventricular mass, g±SD | 161.5±34 | 171.6±42 | 0,583 |
| Left ventricular mass index, g/m²±SD | 87.4±17.4 | 93.7±21.5 | 0,289 |
| Presence of left ventricular hypertrophy | | | 0,864 |
| Normal geometry n (%) | 14 (74) | 18 (58) | |
| Concentric remodelling n (%) | 4 (21) | 11 (36) | |
| Eccentric hypertrophy n (%) | 1 (5) | 1 (3) | |
| Concentric hypertrophy n (%) | 0 | 1 (3) | |
| Left ventricular ejection fraction, %±SD | 71.4±4.4 | 72.6±4 | 0,317 |
| MV E, m/sec±SD | 0.8±0.1 | 0.8±0.2 | 0,174 |
| MV A, m/sec±SD | 0.7±0.1 | 0.7±0.1 | 0,638 |
| E/A ratio±SD | 1.2±0.3 | 1.1±0.4 | 0,238 |
| MV deceleration time, msec±SD | 202±31 | 200±24.4 | 0,967 |
| e′ at mitral lateral annulus, cm/sec±SD | 9.9±1.9 | 9±2.9 | 0,176 |
| E/e′ (lateral annulus)±SD | 9.5±3.4 | 8.9±2.5 | 0,968 |

Values are expressed as Mean±SD unless otherwise indicated. MV E: early peak mitral inflow velocity; MV A: late peak mitral inflow velocity; e′: early peak diastolic velocity of mitral annulus.

the subendocardial longitudinal fibres are mainly affected[24]. Because of this in the earlier stages while LV longitudinal and radial strains are reduced, circumferential strain is preserved[24]. During the disease process the whole ventricul is affected by macrovascular and microvascular abnormalities and interstitial fibrosis. So, these effects result in impaired global myocardial function[23]. Shang *et al.* have showed that subclinical impaired myocardial deformation was common in patients with PsA even without CV risk factors[12].

Also in this study, we found that PsA patients even with normal ejection fraction had evidence of early impairment of longitudinal, circumferential and radial deformation. All these studies suggest that there were early changes of myocardial deformation in PsA.

These new speckle tracking echocardiography techniques demonstrated that there is higher prevalence of subclinical myocardial dysfunction in PsA than previously considered[3]. All these studies suggested that patients with PsA had global myocardial involvement. This involvement may share a different pathologic mechanism from ischemic heart disease in the early stages[12].

Shang *et al.* have showed the relationship between impaired myocardial deformation and disease activity (DAS 28 and ESR)[12]. However, we could not find a correlation between the global longitudinal circumferential and radial strain analysis results and disease activity in our patients, possibly because of the relatively low levels of DAS 28, ESR, CRP, PASI scores in our cohort.

It was previously reported that inflammation has been verified to accelerate CV damage by contributing to atherosclerosis, cardiac fibrosis, necrosis, and apoptosis[26]. In the present study, although PsA patients had low disease activity, they had subclinical impaired LV deformation. We suggest that existing impaired myocardial deformation may be associated with irreversible damage resulting from myocardial fibrosis due to long-standing inflammation. However, it remains to be clarified whether LV dysfunction is reversible when PsA activity and disease-related inflammation are well controlled[12].

Study limitations include small sample size and the inability to evaluate possible causality due to its cross-sectional/observational design. Despite the fact that the patients with clinical evidence for CVD were excluded from the study, some patients had CVD risk factors

**TABLE III. COMPARISON OF LEFT VENTRICULAR FUNCTIONS OF PSA PATIENT AND CONTROL GROUPS USING STE ANALYSIS**

| STE analysis results | Control group (n=19) | PsA patients (n=31) | p |
|---|---|---|---|
| Longitudinal long-axis strain,% (IQR) | -18.7 (17.8-19.8) | -15.7 (14.3-18.1) | 0,001 |
| Longitudinal four-chamber strain,%±SD | -19±2.5 | -17.5±3.4 | 0,084 |
| Longitudinal two-chamber strain, %±SD | -20.2±2.6 | -18±3.4 | 0,024 |
| Longitudinal global strain, %±SD | -19.3±2 | -17.1±2.8 | 0,005 |
| Circumferential antero septal strain, %±SD | -20.5±5.6 | -20.1±4.6 | 0,806 |
| Circumferential anterior strain, %±SD | -19.2±5.4 | -14.5±4.9 | 0,003 |
| Circumferential lateral strain, %±SD | -19.3±7 | -9.7±5.4 | <0,001 |
| Circumferential posterior strain, %±SD | -19.9±6.8 | -10.8±5.8 | <0,001 |
| Circumferential inferior strain, %±SD | -21.2±6 | -13.2±5.4 | <0,001 |
| Circumferential septal strain, %±SD | -22±5.5 | -17.3±5.1 | 0,003 |
| Circumferential global strain, %±SD | -20.3 ±4.8 | -14.3 ±3 | <0,001 |
| Radial antero septal strain, % (IQR) | 35.8 (25.1-53.5) | 31.6 (19.5-37.7) | 0,086 |
| Radial anterior strain, % (IQR) | 38.9 (29.4-54.6) | 26.7 (22.1-32.3) | 0,002 |
| Radial lateral strain, % (IQR) | 41.4 (33-61) | 25.5 (21.7-32) | <0,001 |
| Radial posterior strain, % (IQR) | 55.3 (32.7-69.3) | 28.2 (23.6-38) | <0,001 |
| Radial inferior strain, % (IQR) | 55.3 (32.7-69.3) | 28.2 (23.6-38) | <0,001 |
| Radial septal strain, % (IQR) | 43.4 (29.6-56.8) | 26.7 (18.8-33.5) | 0,004 |
| Radial global strain, %±SD | 46.5±17 | 29.3±10 | <0,001 |

Values are expressed as Mean±SD or Median (IQR) unless otherwise indicated.

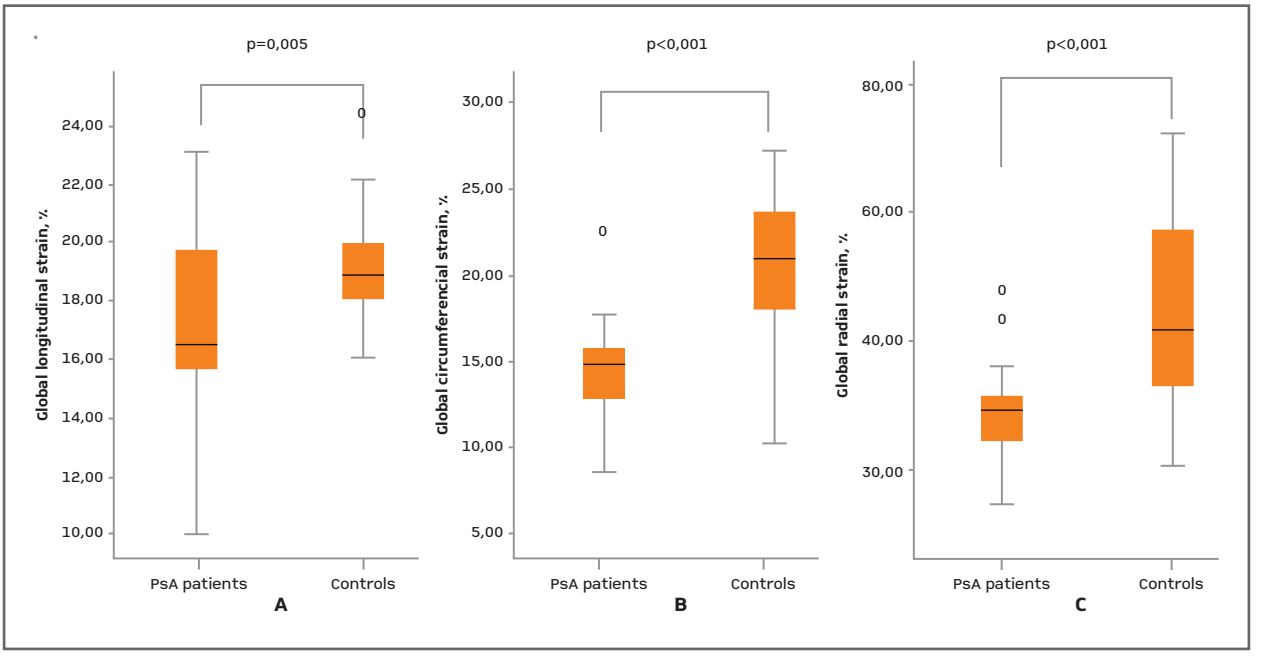


**FIGURE 1.** **A)** Global longitudinal strain in PsA patients and controls. **B)** Global circumferential strain in PsA patients and controls. **C)** Global radial strain in PsA patients and controls. In A,B and C, boxes represent the 25$^{th}$ to 75$^{th}$ percentiles. Lines inside the boxes represent the median.

which could have a major effect on the myocardial function such as hypertension, obesity and smoking status. When the existence of these confounding factors with a similar rate considered in the control group, PsA patients (via its inherent complex mechanisms) are considered to have a potential adverse effect on LV functions.

In our study, we demonstrated a common impairment of myocardial deformation in PsA patients. Thus, the use of this new imaging technique may provide additional benefits in monitoring of cardiovascular involvement and risk stratification in PsA patients.

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