Impaired Left Ventricular Apical Rotation is Associated with Disease Activity of Psoriatic Arthritis

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ABSTRACT. ***Objective.*** Although early cardiovascular (CV) involvement has been found in patients with psoriatic arthritis (PsA), few studies have related this to PsA disease activity. The aim of our study was to evaluate left ventricular (LV) mechanics using novel, more sensitive techniques based on assessment of LV rotation for the detection of impaired LV function in patients with PsA correlated with disease-related risk factors.

Methods. Seventy-six patients with PsA and 24 healthy control subjects were enrolled, including 33 patients without any CV risk factors. All participants underwent conventional echocardiography and 2-dimensional speckle tracking imaging. Global longitudinal, apical circumferential, and radial strain, and apical rotation and maximal untwisting rate during early diastole were measured.

Results. Although patients with PsA had normal LV ejection fraction, the myocardial deformation in multidimensional planes was impaired. Based on the cutoff point derived from the apical rotation of control subjects, 81% of the patients had subclinical systolic and/or diastolic dysfunction. Similar prevalence was found in patients without CV risk factors. Spearman correlation demonstrated a relationship between Disease Activity Score in 28 joints ($r = 0.299$, $p = 0.011$), erythrocyte sedimentation rate ($r = 0.309$, $p = 0.008$), and impaired apical rotation, even after adjusting for age and hypertension. No correlation was found between longitudinal, radial, and circumferential strain and disease activity.

Conclusion. Subclinical impaired myocardial deformation was common in patients with PsA even without CV risk factors. Apical rotation was associated with the status of PsA disease activity. These new speckle tracking echocardiography techniques can detect subclinical myocardial involvement in PsA. (J Rheumatol First Release March 1 2014; doi:10.3899/jrheum.130589)

Key Indexing Terms:
PSORIATIC ARTHRITIS DISEASE ACTIVIT
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Patients with psoriatic arthritis (PsA) have a high risk of developing cardiovascular diseases (CVD) compared to the healthy population. This includes diseases such as hyperlipidemia, hypertension, ischemic heart disease, congestive heart failure, peripheral vascular disease, cerebrovascular disease, and type II diabetes[1]. Tam, *et al* found that PsA is an independent risk factor of subclinical atherosclerosis and that the metabolic abnormalities in PsA may be related to a shared inflammatory pathway[2,3]. Gonzalez-Juanatey, *et al* found evidence of endothelial dysfunction and macrovascular diseases in patients with PsA even without traditional CV risk factors or clinically evident CVD[4,5]. Costa, *et al* reported increased arterial stiffness in patients with PsA who did not have known CV risk factors[6]. Shang, *et al* recently demonstrated a high prevalence of subclinical left ventricular (LV) dysfunction and increased ventricular-arterial stiffness in patients with PsA[7,8]. Nonetheless, few studies have assessed the relationship between cardiac function and any specific PsA-related risk factors.

Myocardium consists of inner oblique, middle, and outer oblique layers created by a single myocardial muscle band helically folding upon itself. In systole, ventricles move inward and the wall thickens; the base moves toward the apex and ventricles shorten in long axis; the apex rotates counterclockwise and the base rotates clockwise. Speckle tracking echocardiography (STE) is a new noninvasive ultrasound imaging based on a frame-to-frame tracking of ultrasonic speckles on greyscale 2-dimensional (2-D) images and a relatively angle-independent technology. STE can judge the direction of movement of any points in the

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myocardium and record the speed, path, and distance of such movement. Compared to conventional echocardiography, STE provides new insights in deciphering cardiac physiology and mechanics in cardiomyopathy[9,10,11] and identifying early subclinical changes in various pathologies by accurate evaluation of global and regional myocardial deformation and function[12,13,14]. In addition, STE can detect myocardial ischemia and viability objectively and accurately by showing reduced deformation[15,16], differentiate transmural from subendocardial infarction by demonstrating lower circumferential strain in the former[17], and detect early myocardial impairments before changes in LV ejection fraction in systemic diseases[18,19,20,21] and chemotherapy-induced cardiotoxicity[22]. In our study, STE was applied to measure LV deformation and function in 3 dimensions (longitudinal, radial, and circumferential strain) and rotation, and their relationships to PsA were assessed.

MATERIALS AND METHODS

Study population. One hundred five patients were consecutively screened at the rheumatology clinic of Prince of Wales Hospital affiliated to the Chinese University of Hong Kong from January 2007 to May 2008. Seventy-six patients, who were aged > 18 years and who fulfilled the Classification of Psoriatic Arthritis criteria[23], were recruited for this study, including 33 patients without traditional CV risk factors. Exclusion criteria included pregnancy, hypothyroidism, clinically significant renal disease (serum creatinine level ≥ 270 μmol/l), history of angina, stable coronary artery disease, previous acute coronary syndromes, coronary revascularization, bundle-branch block, second-degree or higher atrioventricular block, atrial fibrillation, valvular stenosis or at least moderate valvular regurgitation, valvular replacement or repair, or mitral annular calcification. Twenty-four healthy control subjects without a history of overt CVD were recruited from the community through advertisement or from other clinics. They were matched to the patients without CV risk factors for age, sex, body mass index, and blood pressure. The Ethics Committee of The Chinese University of Hong Kong and the Hong Kong Hospital Authority approved our study, and it was conducted in compliance with the Declaration of Helsinki (2000) of the World Medical Association. All participants provided written informed consent.

Clinical assessment. PsA patients with predominant peripheral arthritis were included in the category of peripheral arthritis, and those with predominant inflammatory arthritis of the back were included in the category of spondyloarthritis (axis arthritis)[24].

Physician and patient global assessments of pain were measured using a 10-point visual analog scale (0, excellent well-being; 10, feeling extremely unwell). Physical examination recorded the number of tender and swollen joints using the 68 tender/66 swollen joint count, the presence of dactylitis, and the number of permanently deformed joints. Disease activity was assessed using the Disease Activity Score in 28 joints (DAS28)[25]. Cutaneous lesions were examined and the Psoriasis Area and Severity Index (PASI) was calculated to evaluate the severity[26]. Erythrocyte sedimentation rate (ESR) was measured by the Westergren method. High-sensitivity C-reactive protein (hsCRP) level was measured using an immunoturbidimetric assay performed with Olympus OSR6185 (Olympus Diagnostics). Apolipoprotein A and B were tested by automated analyzer (Cobas-Mira Plus, Hoffman-LaRoche Diagnostics) using a turbidimetric assay.

Echocardiography. Comprehensive echocardiography was performed using Vivid 7 systems with a 3.5-MHz probe (GE Medical Systems). Standard echocardiographic assessments included the measurements of LV dimensions, wall thickness, LV mass index, ejection fraction, and color Doppler imaging of all valves, and were done according to the recommendations of the American Society of Echocardiography[27]. The LV ejection fraction was evaluated by modified biplane Simpson's method. Ventricular stiffness was derived from echocardiographic measurements, including LV end-systolic elastance (0.9 × systolic blood pressure/LV end-systolic volume) and diastolic elastance [(lateral E/E')/LV stroke volume][28]. Real-time pulsed-wave tissue Doppler velocities were recorded from the septal and lateral sites of the mitral annulus in the apical 4-chamber view for measurements of early peak (E') diastolic velocity. Subclinical LV diastolic dysfunction was identified by lateral E' < 11.5 cm/s and/or the ratio of the mitral inflow early diastolic filling velocity (E) to lateral E' (E/E') > 10[29,30,31]. In our laboratory, the intraobserver and interobserver variability for tissue Doppler velocity data were 3% and 5%, respectively, as reported[32].

With STE, 2-D images of basal, mid-papillary, and apical short-axis views, as well as apical 2-chamber, 3-chamber, and 4-chamber views were gathered using a high frame rate (50–80 frames/s). Offline analysis was performed using customized software (EchoPac-PC SW-only, Version 6.0.0, Vingmed-GE). An 18-segmental LV model was used in longitudinal strain analysis[33]. In short-axis view, apical segmental circumferential and radial strain and apical rotation were measured. In our laboratory, the intraobserver and interobserver variability were 8.2% and 9.8% for rotation, 5.9% and 9.4% for mean circumferential strain, 6.1% and 8.1% for mean radial strain, as well as 2.3% and 3.1% for mean longitudinal strain, respectively[33].

Statistical analysis. SPSS 17.0 was used for the analyses. Results are expressed in mean (SD) for normally distributed data and the median [interquartile range (IQR)] for non-normally distributed data. To find PsA-related cardiac involvement, the comparisons of conventional echocardiography and STE between patients without CV risk factors and control group were performed using independent t test for continuous variables and chi-square test for categorical variables. The cutoff points of LV apical rotation were derived from control group by using mean +2 SD, because 95% of all the data values in the control group can be found between the mean +2 SD and the mean -2 SD. Then all patients were placed in 2 subgroups based on the cutoff point of apical rotation. To find possible risk factors of abnormal LV apical rotation, the comparisons of PsA-related clinical and conventional echocardiographic measures were done between 2 patient subgroups using the t test, chi-square, and Mann-Whitney U test. Spearman correlation and chi-square were performed to further assess the relationship between apical rotation and disease activity. All tests were 2-tailed, and $p < 0.05$ was considered statistically significant.

RESULTS

Clinical characteristics. In 76 patients with PsA, the mean age was 47.9 years, the mean age at PsA diagnosis was 38.7 years, and the median of disease duration was 8.2 years. Fifty-six patients (73.7%) had psoriasis with the median of PASI equal to 2.0. Sixty-three patients (82.9%) had peripheral arthritis and 13 (17.1%) had spondyloarthritis. Based on the DAS28 score, 29 patients (38.2%) had inactive disease (DAS28 ≤ 3.2), 30 (39.5%) had moderate disease activity (DAS28 > 3.2 to ≤ 5.1), and 17 (22.4%) had very active disease (DAS28 > 5.1). Twenty-eight (36.8%) patients had hypertension, 14 (18.4%) had diabetes mellitus, and 8 (10.5%) had hyperlipidemia. More than half the patients had taken nonsteroidal antiinflammatory drugs (63.2%) or disease-modifying antirheumatic drugs (56.6%), fewer patients had corticosteroid (10.5%) or antihypertensive therapy (19.7%), and no patients received antirheumatic biologic agents.

Early impaired LV deformation and function. Comparison of cardiac structure, compliance, and function were performed between 33 PsA patients without CV risk factors and 24 healthy control subjects. Age, sex, and blood pressure were comparable. Although patients without CV risk factors had normal ejection fraction, LV structure, and mass index compared to control groups, they still showed significantly increased LV diastolic elastance, decreased LV deformation, and high prevalence of subclinical systolic and/or diastolic dysfunction (Table 1). In the whole patient group, 81% had subclinical systolic and/or diastolic dysfunction. Among them, 25 (32.9%) had only subclinical diastolic dysfunction, 15 (19.7%) had only subclinical systolic dysfunction, and 21 (27.6%) had both subclinical systolic and diastolic dysfunction. In addition, patients with PsA only showed higher proportion of the subclinical LV systolic dysfunction but lower proportion of subclinical LV diastolic dysfunction than those with CV risk factors (Table 2). Figure 1 illustrates normal and impaired LV deformation.

Relationship between impaired LV deformation and disease activity. Table 3 shows the comparison of cardiac structure and PsA-related clinical data between 2 patient subgroups. The groups were comparable to the healthy controls in age, blood pressure, history, PsA disease duration, disease patterns, and medical therapies. Moreover, there was no difference in cardiac structure, compliance, and subclinical myocardial diastolic dysfunction between them. However, patients with impaired apical rotation showed significantly higher DAS28 and ESR than controls (Table 3) and patients with active disease had a high proportion of abnormal apical rotation (Table 4). Spearman correlation demonstrated a relationship between DAS28 ($r = 0.253$, $p = 0.027$), ESR ($r = 0.275$, $p = 0.253$), and impaired apical rotation. After adjusting for age and hypertension, the correlations were still significant (DAS28: $r = 0.299$, $p = 0.011$; ESR: $r = 0.309$, $p = 0.008$). No correlation was found between longitudinal, radial, and circumferential strain and disease activity.

DISCUSSION

Our study evaluated myocardial function in multidimensional planes in patients with PsA and is the first, to our knowledge, to describe the relationship between impaired myocardial deformation and disease activity.

Early impairments of myocardial deformation. The application of STE enables considerable insight into myocardial

Table 1. Comparisons of left ventricle between patients without cardiovascular risk factors and control subjects.

	PsA Without CV RF, n = 33	Controls, n = 24	p
Age, yrs, mean ± SD	43.9 ± 12.8	46.6 ± 8.9	0.375
Sex (female/male), n	18/15	13/11	0.977
Systolic blood pressure, mmHg, mean ± SD	119 ± 9	119 ± 13	0.934
Diastolic blood pressure, mmHg, mean ± SD	74 ± 7	71 ± 8	0.152
Heart rate, bpm, mean ± SD	72 ± 9	71 ± 11	0.563
Conventional echocardiography, mean ± SD			
Structure			
Interventricular septum thickness, cm	0.85 ± 0.15	0.83 ± 0.13	0.689
LV posterior wall thickness, cm	0.81 ± 0.13	0.75 ± 0.08	0.055
LV end-diastolic diameter, cm	4.6 ± 0.5	4.5 ± 0.4	0.220
Relative wall thickness	0.35 ± 0.07	0.33 ± 0.04	0.166
LV mass index, g/m^2	77.0 ± 14.4	70.7 ± 13.9	0.111
LV end-systolic elastance, mm Hg/ml	4.60 ± 1.12	4.55 ± 1.17	0.865
LV diastolic elastance	0.20 ± 0.05	0.14 ± 0.03	< 0.001
Deformation and function, mean ± SD			
Global longitudinal strain, %	20.0 ± 3.4	21.7 ± 2.5	0.048
Apical radial strain, %	34.5 ± 20.0	37.7 ± 18.4	0.562
Apical circumferential strain, %	21.3 ± 5.1	33.6 ± 4.8	< 0.001
Apical rotation, degree	9.2 ± 3.9	19.4 ± 4.9	< 0.001
Maximal untwisting rate during early diastole, degree(s)	61.7 ± 32.3	103.7 ± 44.0	< 0.001
Ejection fraction, %	66.0 ± 4.8	68.4 ± 4.9	0.072
Speckle tracking imaging			
Subclinical LV diastolic dysfunction, n (%)			
Lateral E' < 11.5 cm/s and/or (E/E') > 10	14 (42.4)	3 (17.6)	0.015
Subclinical LV systolic dysfunction, n (%)			
Speck tracking (apical rotation < 9.6)	21 (63.6)	0	< 0.001
Subclinical LV dysfunction, n (%)	27 (81.8)	0	< 0.001

CV RF: cardiovascular risk factors; LV: left ventricle/left ventricular; PsA: psoriatic arthritis.

Table 2. Comparisons of left ventricle between patients with and without cardiovascular risk factors.

	CV Risk Factors		p
	No, n = 33	Yes, n = 43	
Age, yrs, mean ± SD	43.9 ± 12.8	50.9 ± 11.7	0.015
Sex (female/male), n	18/15	21/22	0.622
Systolic blood pressure, mmHg	119 ± 9	137 ± 20	< 0.001
Diastolic blood pressure, mmHg	74 ± 7	82 ± 10	< 0.001
Heart rate, bpm	72 ± 9	74 ± 12	0.670
Conventional echocardiography			
Structure			
Interventricular septum thickness, cm	0.85 ± 0.15	0.92 ± 0.18	0.073
LV posterior wall thickness, cm	0.81 ± 0.13	0.86 ± 0.14	0.104
LV end-diastolic diameter, cm	4.6 ± 0.5	4.7 ± 0.3	0.487
Relative wall thickness	0.35 ± 0.07	0.37 ± 0.06	0.368
LV mass index, g/m^2	77.0 ± 14.4	85.6 ± 21.2	0.052
LV end-systolic elastance, mm Hg/ml	4.60 ± 1.12	4.28 ± 1.27	0.256
LV diastolic elastance	0.20 ± 0.05	0.24 ± 0.10	0.035
Deformation and function			
Global longitudinal strain, %	20.0 ± 3.4	19.3 ± 2.3	0.292
Apical radial strain, %	34.5 ± 20.0	32.2 ± 17.3	0.641
Apical circumferential strain, %	21.3 ± 5.1	22.7 ± 5.2	0.262
Apical rotation, degree	9.2 ± 3.9	11.2 ± 4.4	0.040
Maximal untwisting rate during early diastole, degree/s	61.7 ± 32.3	67.57 ± 25.3	0.421
Ejection fraction, %	66.0 ± 4.8	61.0 ± 5.2	< 0.001
Speckle tracking imaging			
Subclinical LV diastolic dysfunction, n (%)			
Lateral E' < 11.5 cm/s and/or (E/E') > 10	14 (42.4)	32 (74.4)	0.005
Subclinical LV systolic dysfunction, n (%)			
Speck tracking (apical rotation < 9.6)	21 (63.6)	15 (34.9)	0.013
Subclinical LV dysfunction, n (%)	27 (81.8)	34 (79.1)	0.865

CV: cardiovascular; LV: left ventricle/left ventricular.

motion in multiple directions[34,35,36]. In earlier stages of heart failure, LV longitudinal and radial strains are reduced while LV rotation and circumferential strain are preserved because the subendocardial longitudinal fibers are primarily affected[35]. With the disease progress, the macrovascular and microvascular abnormalities and interstitial fibrosis involve the whole ventricular and LV rotation, and circumferential deformation becomes weak, with impaired global myocardial function[34]. In patients with hypertension, the effect on LV twist differs at different stages of LV remodeling. Patients with concentric remodeling and hypertrophy have increased LV twist while those with eccentric hypertrophy show decreased twist[37]. In this study, we found that patients even with normal ejection fraction and without CV risk factors still had evidence of early impairment of longitudinal and circumferential deformation. Further, LV rotation was sharply decreased, which has been considered as a compensatory mechanism in heart failure, hypertensive heart disease, or in the elderly[38]. In addition, our results showed that the subclinical LV diastolic dysfunction was prevalent in patients with CV risk factors while the subclinical LV systolic dysfunction was common in patients with only PsA. All these studies suggested that patients with PsA had multilayer myocardial involvements and may share a different pathologic mechanism from ischemic heart disease in the early stages.

Relationship between apical rotation and disease activity. In addition to radial thickening and longitudinal shortening, LV rotation is indispensable for effective pumping function[39]. Normal LV ejection fraction cannot be achieved by radial and longitudinal deformation alone without rotation[40,41]. Moreover, rotation is sensitive to changes in both regional and global LV functions but insensitive to alterations in preload and afterload[42,43,44,45]. It was reported that apical rotation was highly correlated with the maximum rate of LV pressure increase under a variety of LV inotropic conditions, irrespective of coronary ligation and development of regional wall motion abnormality[46,47]. Therefore, rotation is an important measurement for a comprehensive assessment of cardiac function. Our previous publications reported the presence of increased ventricular stiffness and early involved cardiac function (detected by tissue Doppler imaging) in patients with PsA, which were associated with disease duration and age at PsA diagnosis, respectively[7,8]. Our study demonstrated that there were early changes of myocardial deformation and higher prevalence of

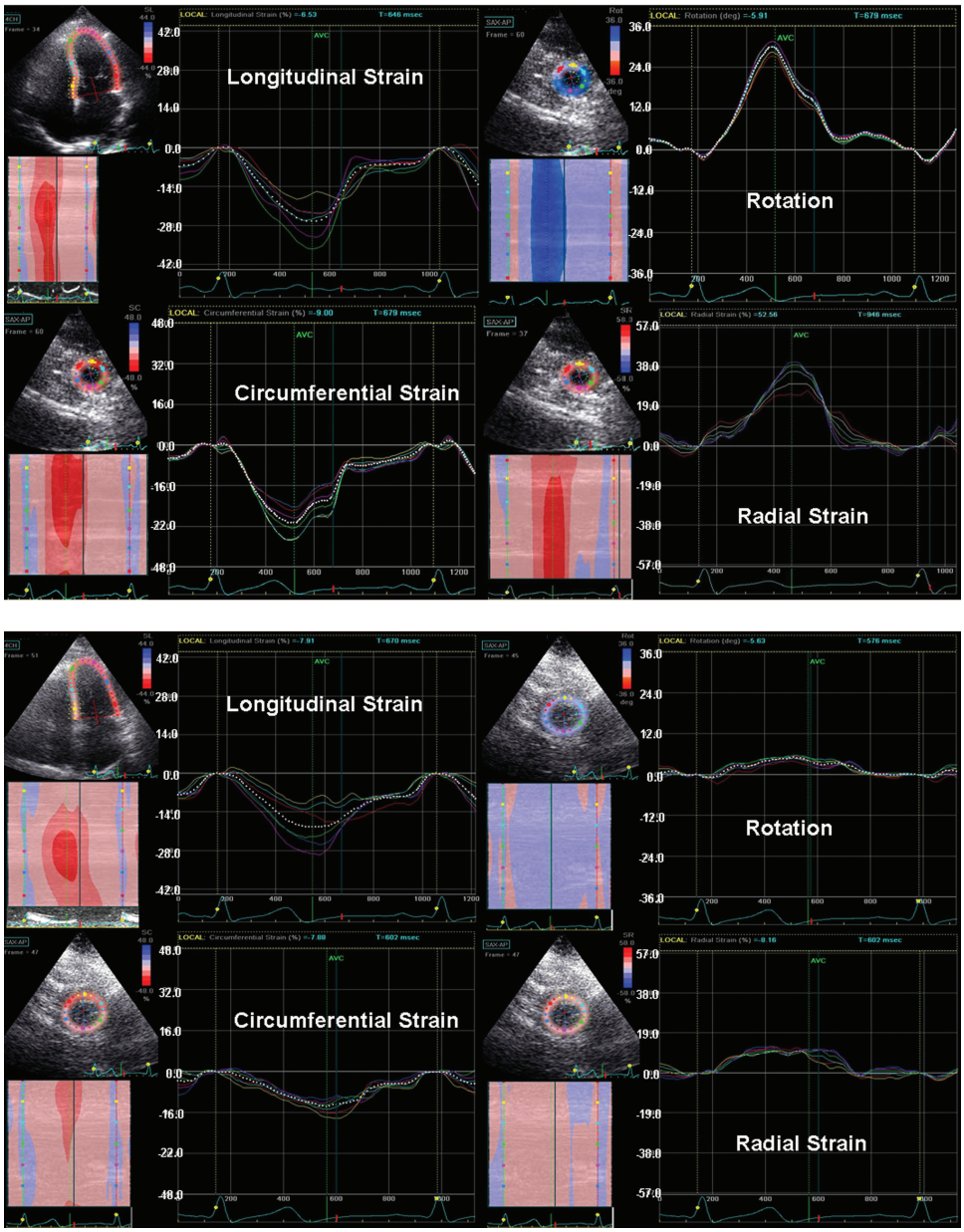

Figure 1. Illustration of normal and impaired left ventricular (LV) deformation. Normal (upper) and impaired (lower) LV deformation (longitudinal, circumferential, and radial strain) and rotation are shown.

subclinical myocardial dysfunction (detected by STE) than previously considered[7]. The relationship between apical rotation and disease activity (DAS28 and ESR) were also explored even after adjusting for age, female sex, and LV elastance. We could not find a correlation between the apical rotation and CRP in our patients, possibly because of the relatively low levels of CRP in our cohort. Kimhi, *et al* reported that atherosclerosis in patients with PsA was significantly correlated with ESR but not CRP[48]. In addition, a mortality study of patients with PsA reported that higher inflammatory burden as reflected by an ESR > 15 mm/h, medications used prior to initial clinic visit, radiologic damage, and the absence of nail lesions were associated with an increased overall mortality rate[49]. On the whole, our results suggested a trend that patients with active disease may have an impaired myocardial deformation (rotation). It may require further studies to determine whether ESR is a better indicator of inflammatory burden than CRP in patients with PsA.

On the other hand, inflammation has been verified to accelerate CV damage by contributing to atherosclerosis, cardiac fibrosis, necrosis, and apoptosis[50]. Our results further verified that inflammation plays an important role in myocardial involvement in PSA. However, it remains to be

Table 3. Comparisons between patients with normal and abnormal apical rotation.

	Apical Rotation		p
	Normal, n = 40	Abnormal, n = 36	
Age, yrs, mean ± SD	49.0 ± 12.3	46.6 ± 12.9	0.412
Sex, F/M	17/23	22/14	0.105
Systolic BP	131 ± 20	127 ± 15	0.282
Diastolic BP	79 ± 11	78 ± 8	0.408
Heart rate, bpm	72 ± 11	75 ± 11	0.306
Hypertension, n (%)	11 (42.5)	17 (30.6)	0.281
Hyperlipidemia, n (%)	5 (12.5)	3 (8.3)	0.555
Diabetes mellitus, n (%)	9 (22.5)	5 (13.9)	0.334
Structure, compliance, and function			
Interventricular septum thickness, cm	0.93 ± 0.17	0.84 ± 0.16	0.033
LV posterior wall thickness, cm	0.86 ± 0.15	0.81 ± 0.12	0.198
LV end-diastolic diameter, cm	4.6 ± 0.4	4.7 ± 0.5	0.564
Relative wall thickness	0.37 ± 0.07	0.35 ± 0.06	0.185
LV mass index, g/m^2	82.8 ± 20.0	80.8 ± 18.4	0.649
LV end-systolic elastance, mm Hg/ml	4.35 ± 1.25	4.50 ± 1.17	0.623
LV diastolic elastance	0.21 ± 0.07	0.24 ± 0.10	0.090
LV ejection fraction, %	62.9 ± 5.6	63.5 ± 5.7	0.628
Subclinical LV diastolic dysfunction, n (%)	25 (62.5)	21 (58.3)	0.711
Disease-related clinical variables			
Age at PsA diagnosis, yrs, mean ± SD	39.1 ± 12.7	38.2 ± 11.5	0.776
Age at psoriasis diagnosis, yrs, mean ± SD	35.6 ± 14.1	34.2 ± 13.7	0.686
Disease duration, yrs, median (IQR)	10.3 (1.5–16.4)	7.3 (2.6–11.4)	0.517
Disease pattern (peripheral/ spondyloarthritis), n	33/7	30/6	0.723
DAS28	3.3 ± 1.1	4.1 ± 1.6	0.013
No. tender joints	1.5 (0–4.5)	3 (0–7.0)	0.151
No. swollen joints	0 (0–1.8)	2 (0–3.0)	0.015
PASI, median (IQR)	1.9 (0.7–7.9)	3.1 (1.2–9.2)	0.281
Apo A-1, mg/dl	152.8 ± 32.2	139.9 ± 31.9	0.168
Apo B, mg/dl	82.4 ± 19.3	80.5 ± 13.1	0.715
ESR, median (IQR) mm/h	17 (8–37)	21 (15–60)	0.019
hs-CRP, median (IQR) mg/l	4.0 (1.4–10.2)	9.9 (1.9–21.6)	0.087
Current NSAID, n (%)	26 (65.0)	22 (61.1)	0.726
Current DMARD, n (%)	22 (55.0)	21 (58.3)	0.770
Sulfasalazine, n (%)	7 (29.2)	8 (38.1)	—
Hydroxychloroquine, n (%)	3 (12.5)	2 (9.5)	0.807
Methotrexate, n (%)	14 (58.3)	11 (52.4)	—
Corticosteroid ever, n (%)	4 (10.0)	4 (11.1)	0.875
Current antihypertensive therapy, n (%)	9 (22.5)	6 (13.9)	0.334

BP: blood pressure; IQR: interquartile range; DAS28: Disease Activity Score in 28 joints; PASI: Psoriasis Area and Severity Index; Apo A-1: apolipoprotein A 1; Apo B: apolipoprotein B; ESR: erythrocyte sedimentation rate; hs-CRP: high-sensitivity C-reactive protein; NSAID: nonsteroidal antiinflammatory drugs; DMARD: disease-modifying antirheumatic drugs.

Table 4. Distribution of abnormal apical rotation among different disease activity groups. Data are n (%).

	Disease Activity			p
	Inactive (DAS28 ≤ 3.2)	Moderately Active (3.2 < DAS28 ≤ 5.1)	Very Active (DAS28 > 5.1)	
Apical rotation ≥ 9.6°	18 (62.1)	18 (60.0)	4 (23.5)	0.024
Apical rotation < 9.6°	11 (37.9)	12 (40.0)	13 (76.5)	

DAS28: 28-joint Disease Activity Score.

clarified whether LV dysfunction is reversible when PsA activity and disease-related inflammation are well controlled. Finally, we used apical rotation rather than torsion because it was difficult to obtain stable curve and reliable measurements of basal rotation[51].

The obvious limitations of our study are the relatively

small sample size and no intervention to improve the impaired myocardial deformation. Although our results suggested that patients with active disease had lower apical rotation, it still needs to be studied whether apical rotation can be used as a monitor of disease activity.

This is the first study, to our knowledge, to demonstrate a common impairment of myocardial deformation and the apical rotation in patients with PsA that is sensitive to the status of disease activity.

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