Two-dimensional speckle tracking of the left ventricle in patients with systemic sclerosis for an early detection of myocardial involvement

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Aims
Myocardial involvement is associated with poor prognosis in patients with systemic sclerosis (SSc). Two-dimensional speckle-tracking echocardiography (STE) is a powerful novel modality for the assessment of subclinical cardiac left ventricular (LV) dysfunction that, so far, has not been investigated in SSc patients. The aim of this study was to evaluate deformation analyses derived from STE for early detection of LV systolic dysfunction in patients with SSc having preserved left ventricular ejection fraction (LVEF).

Methods and results
Twenty-two patients with SSc (57.1 ± 13.3 years, LVEF 64 ± 3.1%, mean time of 5.4 ± 4.6 years from diagnosis) and 22 gender- and age-matched healthy subjects (57.4 ± 14.0 years, LVEF 65 ± 2.7%) underwent echocardiography with STE to assess global and regional LV function. The global longitudinal 2D peak systolic strain (PSS) of the left ventricle was significantly lower in the SSc group compared with controls: −19.0 ± 2.4 vs. −21.1 ± 2.5% (P = 0.008). This was mainly driven by a reduced strain in the basal segments. Strain in the medial segments and in the apex did not differ significantly. In addition, there was a significant difference between both groups regarding the global longitudinal PSS rate of the left ventricle (−1.19 ± 0.18 vs. −1.43 ± 0.26 s⁻¹, P = 0.001).

Conclusion
LV deformation analysis by STE is a sensitive method to detect early LV systolic impairment primarily in the basal segments in patients with SSc having preserved LVEF.

Keywords
Systemic sclerosis • Speckle tracking • 2D strain • 2D strain rate • Myocardial involvement

Introduction
Systemic sclerosis (SSc) is a chronic, multi-system disease characterized by extensive fibrosis and vascular damage1 which, over time, can result in severe dysfunction of almost any internal organ. Cardiopulmonary involvement is frequently observed1 and associated with a poor prognosis.2 Histological studies detected myocardial involvement in up to 80% of patients with SSc,3,4 but clinical evidence of myocardial dysfunction is only recognized in 15–25%.2,5,6 Accordingly, cardiac manifestation in SSc is likely to be underdiagnosed.7 But even clinically evident cardiac involvement may often be underestimated due to an attribution of non-specific symptoms to non-cardiac causes like pulmonary fibrosis or pulmonary arterial hypertension. This could be fatal because the mortality rate of SSc is high8 and cardiac involvement is one of the leading causes of disease-related deaths (accountable for 20–26%), mainly due to heart failure and arrhythmias.2,9 Fortunately, significant advances in symptomatic organ-specific therapy have been made during recent years.9–12 Consequently, preclinical identification of myocardial manifestation is highly encouraged and a sensitive and specific non-invasive diagnostic approach is needed to identify patients who would benefit from early medical intervention. Echocardiography is a widely available, safe, and rapidly evolving technique that has already demonstrated the capability to diagnose preclinical cardiac manifestation of SSc: the left ventricular (LV) myocardial long-axis excursion measured by

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M-mode echocardiography is reduced in patients with SSc having normal left ventricular ejection fraction (LVEF).13 Newer echocardiographic methods, such as tissue Doppler imaging (TDI), allow assessment of myocardial deformation as a sensitive marker for regional and global LV systolic function14 and have already been established also in SSc patients.15–18 Similar to pulsed-wave Doppler blood flow velocity measurements, there are relevant restrictions to the use of TDI (e.g. relevant angle dependence and reliance on the positioning of the sample volume). In contrast, two-dimensional (2D) speckle-tracking echocardiography (STE) is a relatively new echocardiographic technique for obtaining Doppler-independent strain and strain rate (SR) analyses that may overcome some limitations of TDI. This modality has already shown a great clinical relevance and advantages in many clinical and subclinical diseases such as arterial hypertension, diabetes, ischaemic cardiomyopathy, or the assessment of the right heart function in SSc.19–22 Until now, however, no study has investigated the role of STE in detection of subclinical cardiac involvement of the left ventricle in patients with SSc.

The aim of this study was to assess global and regional LV systolic function in patients with SSc having preserved LVEF using STE as a new sensitive method for the detection of LV systolic dysfunction.

Methods

Study population

Twenty-two patients with SSc according to the American College of Rheumatology classification criteria21 (17 women, 5 men, mean age 57.1 ± 13.3 years, range 42–77 years) with a mean time of 5.4 ± 4.6 years (range: 1–13 years) from diagnosis were included into the study (Table 1). According to serological antibody analysis and the modified Rodnan Skin Score (mean: 9.9 ± 13.3; range: 3–26), 11 patients had a limited form and 11 patients suffered from a diffuse form of SSc. Serological antibody analysis revealed the presence of an anti-centromere pattern in 8 patients and anti-Scl-70 in 9 patients. All patients presented with normal pulmonary artery pressure as determined by transtricuspid conventional Doppler echocardiography. In nine patients, lung fibrosis was present. Patients with depressed cardiac involvement of the left ventricle in patients with SSc.

Table 1 Baseline characteristics

|                          | Systemic sclerosis (n = 22) | Control (n = 22) | P-value |
|--------------------------|-----------------------------|-----------------|---------|
| Age (years)              | 57.1 ± 13.3                 | 57.4 ± 14.0     | 0.93    |
| Sex (female) [n (%)]     | 17 (77.3)                   | 17 (77.3)       | 1.0     |
| Systolic BP (mmHg)       | 138 ± 19.1                  | 137 ± 18.8      | 0.95    |
| Diastolic BP (mmHg)      | 79 ± 9.4                    | 81 ± 9.4        | 0.78    |
| Body mass index (kg/m²)  | 25.1 ± 6.0                  | 23.4 ± 19       | 0.9     |
| Diabetes mellitus [n (%)]| 3 (13.6)                    | 2 (9.1)         | 1.0     |
| Arterial hypertension [n (%)] | 11 (50.0)           | 12 (54.5)       | 0.97    |
| Hypercholesterolaemia [n (%)] | 7 (31.2)               | 5 (22.7)        | 0.78    |
| LVEF (%)                 | 64 ± 3.1                    | 65 ± 2.7        | 0.31    |
| LV end-diastolic volume (mL) | 75.3 ± 31.6               | 66.5 ± 28.2     | 0.37    |
| LV end-systolic volume (mL) | 28.4 ± 13.6               | 23.6 ± 11.2     | 0.23    |
| Heart rate (bpm)         | 73.3 ± 8.3                  | 75.1 ± 12.9     | 0.66    |

Mean ± SD, except gender, hypertension, hypercholesterolaemia, and diabetes mellitus.

Conventional Doppler echocardiography

Tricuspid valve regurgitation was detected at the apical four-chamber view by colour Doppler echocardiography. Transthrucipid retrograde velocities were obtained using continuous-wave Doppler. Systolic pulmonary artery pressure was estimated from the peak pressure gradient calculated from three consecutive beats using the modified Bernoulli formula ($\Delta P = 4v^2$) and the right atrial pressure derived from the diameter of the inferior vena cava and the collapsibility index.24,25

2D speckle-tracking strain analysis

For assessment of the radial, circumferential and longitudinal speckle-tracking strain and SR of the left ventricle, standard 2D ultrasound images at the parasternal mid-ventricular short-axis view (at the level of the papillary muscles) and from the apical long-axis, and two- and four-chamber views with a frame rate between 60 and 80 fps were recorded and stored digitally for off-line analysis (EchoPac PC, GE Vingmed, Horton, Norway) as previously described.24,26,27

The timing of end-systole was defined by the aortic valve click that can be seen in Doppler flow recordings from the LV outflow tract. After manual tracing of endocardial borders, the software automatically traced the region of interest including the entire myocardial wall. In this process, every view of the left ventricle was divided into six segments. To optimize tracking, the width of the region of interest was adjusted if necessary. For each segment, the quality of speckle-tracking was analysed automatically. Segments with poor tracking were excluded from further measurements.

Peak systolic longitudinal strain and SR of the apical two-, four-chamber, and long-axis views were calculated averaging the peak systolic strain (PSS) and SR values of the six segments of the corresponding views. Finally, the global longitudinal PSS and global longitudinal peak systolic strain rate (PSSR) of the left ventricle were generated averaging peak systolic values of the three apical views. Peak systolic radial and circumferential strain and the SR were

Echocardiography and Doppler measurements

Standard echocardiography was performed in the left decubitus position using an ultrasound system (Vivid 7, GE Medical Systems, Horton, Norway) with a 3.4-MHz multi-frequency transducer. The LVEF was calculated according to the modified Simpson’s rule using the apical four- and two-chamber views.25 The diameter of the inferior vena cava and the collapsibility index was measured from the subcostal view.
2D speckle tracking of the left ventricle

Table 2  Speckle-tracking strain and strain rate data

|                      | SSC (n = 22)  | Control (n = 22) | P-value |
|----------------------|---------------|------------------|---------|
| Radial strain (%)    | 38.7 ± 21.3   | 48.3 ± 21.8      | 0.138   |
| Radial strain rate (s⁻¹) | 2.28 ± 0.86  | 2.54 ± 0.62      | 0.139   |
| Circumferential strain (%) | −20.4 ± 5.2  | −21.0 ± 7.6      | 0.893   |
| Circumferential strain rate (s⁻¹) | −1.81 ± 0.49  | −1.75 ± 0.40     | 0.776   |
| Global longitudinal PSS (%) | −19.0 ± 2.4  | −21.1 ± 2.5      | 0.008   |
| Global longitudinal PSSR (s⁻¹) | −1.19 ± 0.18  | −1.43 ± 0.26     | 0.001   |
| Longitudinal PSS—APLAX (%) | −18.7 ± 2.2  | −20.7 ± 3.3      | 0.02    |
| Longitudinal PSSR—APLAX (s⁻¹) | −1.16 ± 0.20  | −1.43 ± 0.35     | 0.003   |
| Longitudinal PSS—4CH (%) | −18.2 ± 2.8  | −20.8 ± 3.5      | 0.007   |
| Longitudinal PSSR—4CH (s⁻¹) | −1.14 ± 0.20  | −1.4 ± 0.35      | 0.001   |
| Longitudinal PSS—2CH (%) | −20.1 ± 3.9  | −22.1 ± 2.6      | 0.03    |
| Longitudinal PSSR—2CH (s⁻¹) | −1.28 ± 0.25  | −1.48 ± 0.31     | 0.043   |

PSS, peak systolic strain; PS, peak systolic; SR, strain rate; APLAX, apical long-axis view; 4CH, apical four-chamber view; 2CH, apical two-chamber view. Data are expressed as mean ± SD.

Inter- and intra-observer variability analysis

Three echocardiographers, blinded to clinical data, independently measured the strain and SR of 10 randomized patients for inter-observer variability analysis. One experienced observer calculated the strain and the SR twice on two consecutive days for analysis of the intra-observer variability.

Statistics and figures

Statistics were calculated with software (SPSS, Version 18.0, SPSS, Inc., Chicago, IL, USA). All results are presented as mean ± SD. The Mann–Whitney non-parametric test was used to compare echocardiographic data from patients and control subjects. Differences were considered statistically significant if the P-value was <0.05. The interclass correlation coefficient by Kolmogorov–Smirnov was used to calculate inter- and intra-observer variability.

Results

Clinical characteristics

Age, gender, LV end-diastolic volume and end-systolic volume, body mass index, systolic, and diastolic blood pressure as well as the incidence of cardiovascular risk factors such as hypertension, hypercholesterolaemia, and diabetes mellitus did not differ significantly between patients and the control group (Table 1).

Table 3  Regional longitudinal speckle-tracking strain data

|                      | SSC (n = 22)  | Control (n = 22) | P-value |
|----------------------|---------------|------------------|---------|
| Basal segments (%)   | −17.5 ± 3.0   | −20.2 ± 2.9      | 0.004   |
| APLAX (%)            | −18.0 ± 3.2   | −19.8 ± 4.1      | 0.21    |
| 4CH (%)              | −16.2 ± 3.7   | −19.9 ± 4.6      | 0.018   |
| 2CH (%)              | −19.3 ± 3.9   | −22.0 ± 3.2      | 0.039   |
| Medial segments (%)  | −18.4 ± 2.8   | −20.1 ± 3.2      | 0.065   |
| APLAX (%)            | −18.5 ± 2.2   | −19.4 ± 4.5      | 0.175   |
| 4CH (%)              | −17.7 ± 3.4   | −19.6 ± 3.6      | 0.173   |
| 2CH (%)              | −18.8 ± 4.7   | −22.0 ± 3.6      | 0.023   |
| Apical segments (%)  | −20.9 ± 4.6   | −23.0 ± 4.2      | 0.155   |
| APLAX (%)            | −19.7 ± 5.3   | −22.8 ± 5.2      | 0.054   |
| 4CH (%)              | −21.6 ± 5.3   | −23.8 ± 5.9      | 0.446   |
| 2CH (%)              | −21.7 ± 6.5   | −23.0 ± 4.2      | 0.318   |

PSS, peak systolic strain; PSSR, peak systolic strain rate; APLAX, apical long-axis view; 4CH, apical four-chamber view; 2CH, apical two-chamber view. Data are expressed as mean ± SD.

Standard echocardiographic measures

The mean systolic pulmonary artery pressure derived from the transtricuspid pressure gradient was 23 ± 4 mmHg in the SSC group. The LVEF was similar in both groups (64.3 ± 3.1 vs. 65 ± 2.7%, P = 0.31) (Table 1).

Left ventricular longitudinal strain and strain rate

Out of 396 analysed segments per group, 322 (81%) and 301 (76%) were acceptable for strain analysis in the SSC and control groups, respectively. The data of strain and SR analysis are shown in Tables 2 and 3.

The longitudinal PSS in the four-chamber view and two-chamber view as well as the global longitudinal PSS value was significantly different between both groups (−18.2 ± 2.8 vs. −20.8 ± 3.5%, P = 0.007; −20.1 ± 3.9 vs. −22.1 ± 2.6%, P = 0.03; −19.0 ± 2.4 vs. −21.1 ± 2.5%, P = 0.008) (Table 2 and Figure 1). This was mainly influenced by lower strain in the basal segments, while the strain in the medial segments and in the apex did not differ
significantly ($-17.5 \pm 3.0$ vs. $-20.2 \pm 2.9\%$, $P = 0.004$; $-18.4 \pm 2.8$ vs. $-20.1 \pm 3.2\%$, $P = 0.065$; $-20.9 \pm 4.6$ vs. $-23.0 \pm 4.2\%$, $P = 0.155$) (Table 3).

In addition, the longitudinal PSSR differed significantly between both groups in the four-chamber view, the two-chamber view, and the long-axis view as well as the global longitudinal PSSR ($-1.14 \pm 0.20$ vs. $-1.4 \pm 0.35 \text{s}^{-1}$, $P = 0.001$; $-1.28 \pm 0.25$ vs. $-1.48 \pm 0.31 \text{s}^{-1}$, $P = 0.043$; $-1.16 \pm 0.2$ vs. $-1.43 \pm 0.35 \text{s}^{-1}$, $P = 0.003$; $-1.19 \pm 0.18$ vs. $-1.43 \pm 0.26 \text{s}^{-1}$, $P = 0.001$) (Table 2 and Figure 2). This was driven by a lower SR in the basal and medial segments while the SR in the apex did not differ significantly ($-1.19 \pm 0.21$ vs. $-1.45 \pm 0.29 \text{s}^{-1}$, $P = 0.003$; $-1.10 \pm 0.14$ vs. $-1.27 \pm 0.21 \text{s}^{-1}$, $P = 0.003$; $-1.51 \pm 0.50$ vs. $-1.73 \pm 0.52 \text{s}^{-1}$, $P = 0.132$) (Table 3).

Significantly more patients had a pathologically reduced global longitudinal PSS (cut-off $-18\%$) and global longitudinal PSSR (cut-off $-1.1 \text{s}^{-1}$) value compared with the controls [9 (40.9) vs. 2 (9.1\%), $P = 0.037$; and 8 (36.4) vs. 1 (4.5\%), $P = 0.025$, respectively].

**Left ventricular radial and circumferential strain and strain rate**

In the parasternal mid-ventricular short-axis view, 107 segments (81.1\%) out of a total of 132 segments from the SSc group and 85 segments (64.4\%) out of a total of 132 segments from the control group showed sufficient image quality for radial and circumferential strain analysis (Figure 1). The data of strain and SR analysis are given in Table 2. Radial and circumferential strain and SR did not differ significantly between the SSc and the control groups (Table 2 and Figure 2).
Table 4  Comparison between scleroderma subtypes, data are expressed as mean ± SD

|                                | Diffuse-type SSc (n = 11) | Limited-type SSc (n = 11) | P-value |
|--------------------------------|----------------------------|---------------------------|---------|
| LVEF (%)                       | 63.2 ± 2.8                 | 64.9 ± 3.3                | 0.233   |
| Heart rate (bpm)               | 74.2 ± 7.6                 | 72.5 ± 9.3                | 0.550   |
| Radial strain (%)              | 36.4 ± 16.6                | 40.9 ± 25.5               | 0.973   |
| Radial strain rate (s⁻¹)       | 2.3 ± 0.5                  | 2.21 ± 1.11               | 0.290   |
| Circumferential strain (%)     | −21.5 ± 5.1                | −19.3 ± 5.3               | 0.282   |
| Circumferential strain rate (s⁻¹) | −1.85 ± 0.33              | −1.77 ± 0.60              | 0.616   |
| Global longitudinal PSS (%)   | −18.5 ± 2.4                | −19.6 ± 2.4               | 0.308   |
| Global longitudinal strain rate (s⁻¹) | −1.2 ± 0.2               | −1.19 ± 0.18              | 0.962   |
| Longitudinal PSS—APLAX (%)     | −18.6 ± 1.7                | −18.8 ± 2.7               | 0.756   |
| Longitudinal PSS—4CH (%)       | −17.2 ± 3.1                | −19.1 ± 2.3               | 0.151   |
| Longitudinal PSS—2CH (%)       | −19.6 ± 4.3                | −20.6 ± 3.5               | 0.438   |
| Longitudinal PSSR—APLAX (s⁻¹)  | −1.13 ± 0.21               | −1.19 ± 0.21              | 0.517   |
| Longitudinal PSSR—4CH (s⁻¹)    | −1.16 ± 0.18               | −1.12 ± 0.22              | 0.572   |
| Longitudinal PSSR—2CH (s⁻¹)    | −1.13 ± 0.21               | −1.28 ± 0.22              | 1.0     |

LVEF, left ventricular ejection fraction; PSS, peak systolic strain; PSSR, peak systolic strain rate; APLAX, apical long-axis view; 4CH, apical four-chamber view; 2CH, apical two-chamber view.
Data are expressed as mean ± SD.

Table 5  Regional longitudinal speckle-tracking strain data between scleroderma subtypes, data are expressed as mean ± SD

|                                | Diffuse-type SSc (n = 11) | Limited-type SSc (n = 11) | P-value |
|--------------------------------|----------------------------|---------------------------|---------|
| Longitudinal PSS (%)           |                           |                           |         |
| Basal segments                 | −17.7 ± 2.8                | −17.4 ± 3.4               | 0.784   |
| APLAX                          | −18.5 ± 3.6                | −17.5 ± 2.8               | 0.570   |
| 4CH                            | −16.8 ± 3.5                | −15.7 ± 4.0               | 0.719   |
| 2CH                            | −18.5 ± 4.4                | −20.1 ± 3.4               | 0.400   |
| Medial segments                | −17.4 ± 2.5                | −19.4 ± 2.8               | 0.133   |
| APLAX                          | −17.9 ± 1.0                | −19.1 ± 3.1               | 0.349   |
| 4CH                            | −16.6 ± 4.2                | −18.8 ± 2.2               | 0.332   |
| 2CH                            | −17.4 ± 5.0                | −20.2 ± 4.2               | 0.133   |
| Apical segments                | −20.0 ± 5.5                | −21.8 ± 3.6               | 0.300   |
| APLAX                          | −19.2 ± 4.9                | −20.1 ± 5.8               | 0.605   |
| 4CH                            | −19.8 ± 5.6                | −23.3 ± 4.6               | 0.132   |
| 2CH                            | −22.2 ± 7.6                | −21.2 ± 5.4               | 0.973   |
| Longitudinal PSSR (s⁻¹)        |                           |                           |         |
| Basal segments                 | −1.18 ± 0.20               | −1.21 ± 0.23              | 0.936   |
| Medial segments                | −1.10 ± 0.15               | −1.10 ± 1.15              | 0.508   |
| Apical segments                | −1.42 ± 0.52               | −1.61 ± 0.49              | 0.411   |

PSS, peak systolic strain; PSSR, peak systolic strain rate; APLAX, apical long-axis view; 4CH, apical four-chamber view; 2CH, apical two-chamber view.
Data are expressed as mean ± SD.

Figure 3  Colour coding of the average regional longitudinal PSS in the four-chamber view (A), two-chamber view (B), and the apical long-axis view (C) in patients with SSc (left panel) and controls (right panel). The echo templates were originally created by Patrick J. Lynch and C. Carl Jaffe, MD and used with permission under the Creative Commons Attribution 2.5 License 2006.
Comparison between diffuse and limited type of SSc

There were no significant differences among the echocardiographic parameters in patients with diffuse and limited type of SSc (Tables 4 and 5).

Inter- and intra-observer variability

The inter- and intra-observer variability for the longitudinal 2D strain was 5.0 and 10.3%, respectively. The inter- and intra-observer variability for the radial 2D strain was 8.4 and 16.3%, respectively.

Discussion

Myocardial damage is more frequent in SSc patients than clinically suspected. Since cardiac involvement is associated with a poor prognosis, the need for an early detection is evident. We could demonstrate that in patients with SSc having preserved LVEF global longitudinal PSS and SR of the left ventricle derived from 2D speckle-tracking analysis were impaired compared with a matched control group (Figures 1–3). Furthermore, significantly more patients had pathologically reduced global longitudinal PSS and global longitudinal PSSR values compared with the controls. Although it has been shown that STE-derived strain and SR are a sensitive marker for the detection of clinical and subclinical myocardial left heart dysfunction in a variety of pathologies, to our knowledge the present study is the first to describe such abnormalities in patients with SSc. STE is a relatively new technique with many advantages compared with TDI: e.g. lack of angle dependence since it can measure the myocardial motion in any direction within the image plane and capability of direct measurements of strain. In addition, speckle tracking can be performed off-line from recorded examinations using only standard 2D images. Furthermore, assessment of 2D strain is a semi-automatic method that is only minimally affected by inter- and intra-observer variability.

In the clinical routine, assessment of global systolic function is usually based on the LVEF. This parameter, however, is insufficient in describing the complex myocardial motion which is characterized by multiple three-dimensional movements: longitudinal shortening, radial thickening, and circumferential shortening, as well as a twist motion due to the helical nature of the heart muscle.

The longitudinal function is predominantly influenced by subendocardial fibres that are most susceptible to myocardial damage. In contrast, the mid-myocardial and epicardial fibres that are mostly responsible for the circumferential motion and twist remain relatively unaffected. Since the LVEF is mainly dependent on the radial and the circumferential deformation, the LVEF may remain normal despite relevant myocardial damage. Histological studies diagnosed myocardial fibrosis in up to 80% of patients with SSc. While autopsy studies are likely to represent patients with more advanced disease, the reduced regional and global longitudinal deformation detected in our study may also be caused by myocardial fibrosis. But as a limitation of our study, neither cardiac magnetic resonance imaging (MRI) nor a myocardial biopsy was performed to confirm this hypothesis.
In our patients, each LV segment had lower longitudinal strain and SR but not all segments were significantly divergent compared with the controls. The reduced global longitudinal strain was mainly caused by a reduced longitudinal strain in the basal segments (Figures 3–5). In contrast, the strain in the medial and apical segments did not differ significantly between both groups. Our results are in agreement with previously published data using TDI, which show reduced peak systolic longitudinal strain and SR of the LV lateral-free wall in 23 patients with SSC, despite normal LVEF and normal pulsed-wave Doppler tissue velocities. In addition, Kepez et al. reported a reduction in the strain in 6 out of 12 LV segments and of the SR in 2 out of 12 LV segments, which did not match any particular coronary artery distribution. In our patients, we found a significantly reduced longitudinal peak systolic SR in every apical view—mainly in the basal and medial segments but not in the apex. In conclusion, our results confirmed a heterogeneous distribution of segmental systolic dysfunction. Therefore, we agree with Kepez et al. to emphasize the use of global indices for the assessment of systolic function. Interestingly, these findings were independent from the disease subtype.

No patients had to be excluded due to poor image quality. This indicates an acceptable feasibility of echocardiographic assessment of LV function by speckle tracking in SSC patients. In contrast, for patients with SSC and advanced pulmonary involvement it could be demanding to tolerate an ultrasound examination lying mainly in a steep left lateral decubitus position. Furthermore, respiratory diseases often lead to a significant impairment of the echocardiographic acoustic window due to air trapping or due to pulmonary fibrosis, but the free right ventricular wall should be more susceptible to this limitation than the left ventricle.

**Limitations**

As a limitation of our study, no coronary angiography was performed to rule out coronary artery disease as a reason for reduced longitudinal function. However, all patients were asymptomatic in this regard and the pre-test probability was low based on clinical atherosclerotic risk factors, and no significant difference compared with the control could be found (Table 1). In addition, as mentioned myocardial fibrosis was not assessed by cardiac MRI or myocardial biopsies.

Our study included only long-term scleroderma patients with a mean time of 5.4 ± 4.6 years from diagnosis. Therefore, 5 (22.7%) of the included patients had already received potentially cardiotoxic disease-modifying agents (azathioprine, methotrexate, or cyclosporine A). Accordingly, we cannot rule out that the reduced longitudinal strain in SSC patients was, at least in part, due to side effects of the medical therapy.

**Conclusion**

In summary, global longitudinal strain and SR derived from STE are reduced in asymptomatic patients with SSC having preserved LVEF compared with matched controls. Since cardiac involvement is common, our results may be explained by a subclinical myocardial fibrosis. In addition to analysing previously published studies using TDI, 2D speckle tracking can also be practically used to assess global and regional deformation parameters in SSC.
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Conflict of interest: none declared.

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