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Left ventricular twist mechanics in patients with apical hypertrophic cardiomyopathy: assessment with 2D speckle tracking echocardiography

S-A Chang,1 H-K Kim,2 D-H Kim,2 J-C Kim,3 Y-J Kim,2 H-C Kim,3 D-W Sohn,2 B-H Oh,2 Y-B Park2

ABSTRACT

Objective: Left ventricular (LV) apical rotation significantly contributes to LV twist, which has been reported to have a vital role in maintaining LV systolic and diastolic function. Apical hypertrophic cardiomyopathy (ApHCM) is a unique disease with pathological LV hypertrophy at the apex. We aimed (1) to evaluate LV twist mechanics in ApHCM and (2) to demonstrate the influence of predominantly local, pathological involvement of the apical myocardium on LV twist mechanics.

Methods: 21 patients diagnosed with ApHCM were consecutively enrolled and compared with normal controls. After a standard echocardiographic examination, we scanned parasternal basal and apical short-axis planes to quantify LV rotations and LV twist using the speckle tracking technique. For better understanding of LV twist mechanics in ApHCM, LV radial and biplanar strains and LV twist-volume curve were also evaluated.

Results: Compared with the normal controls, apical rotation was markedly decreased in ApHCM patients (p<0.001), but the decreases in basal rotation were not significant. As a consequence, LV twist was significantly lower in ApHCM patients (p = 0.007). Apical radial (p = 0.01) and biplanar (p<0.001) strains in ApHCM were also significantly decreased. Compared to normal controls, LV twist-volume and twist-radial displacement curves clearly showed a decrement in the slope of the linear systolic phase and a loss of an inflection point separating the early from late untwisting phase in ApHCM patients.

Conclusion: LV twist in ApHCM was significantly decreased due to a reduction in apical rotation, suggesting that regional myocardial changes in ApHCM can modify the global LV twist mechanics. Given the close interconnection between LV systolic and diastolic function, impairment of LV twist may lead to the loss of early diastolic suction and finally generate diastolic dysfunction in ApHCM.

Left ventricular (LV) twist is a complex motion of the LV myocardium, which is caused by a close interplay between clockwise rotation of the base and counter-clockwise rotation of the apex during systole.1 LV twist has an important role in maintaining efficient myocardial contraction during systole3 and aids in generating early suction power during the isovolumic relaxation period.3 Since the importance of LV twist was first realised, a number of studies have been conducted on this topic in a variety of different disease settings, such as hypertension,1 LV diastolic dysfunction,9,10 myocardial infarction,11 mitral regurgitation,12 aortic stenosis13,14 and asymmetric septal hypertrophic cardiomyopathy.11

Hypertrophic cardiomyopathy (HCM) is a genetic disorder that is characterised by a hypertrophied and non-dilated LV with variable myocardial involvement. Although HCM is classified based on the anatomical location of myocardial involvement, apical hypertrophic cardiomyopathy (ApHCM) was not included in the conventional classification.12 Recently, ApHCM has been widely accepted to be a unique phenotype of HCM with a giant negative T wave in anterior leads of the electrocardiogram, pathological hypertrophy of the LV apex and a “spade-like” configuration of the LV cavity at end-diastole by two-dimensional (2D) echocardiography.13 Although LV twist mechanics have been investigated in HCM patients with asymmetrical septal hypertrophy,14 our understanding of LV twist mechanics in ApHCM patients is lacking. Furthermore, interrogation of LV twist mechanics in ApHCM patients is of clinical importance because ApHCM is being increasingly diagnosed worldwide14,15 and because it is now recognised that ApHCM may cause morbidities such as atrial fibrillation, myocardial infarction, heart failure, stroke and ventricular arrhythmia.16–18

Theoretically, given the predominant contribution of apical rotation to LV twist, involvement of the LV apex by a pathological myocardial disarray and/or fibrosis is expected to reduce the extent of LV twist. This is in sharp contrast to asymmetrical septal HCM, which mainly involves the basal and mid-septal myocardium and was reported to have a significant increment or at least preservation of LV twist, compared to normal controls.6,10,19 In addition, because ApHCM is mainly manifested by a localised involvement of the apical myocardium,20 usually without compromising LV systolic function, it is an ideal model for investigating the isolated effect of apical myocardial pathological involvement on global LV twist. Therefore, the objectives of this study were (1) to evaluate LV twist mechanics in patients with ApHCM, and (2) to determine the influence of local myocardial involvement by a pathological process on LV twist mechanics.

METHODS

Patient enrolment

Patients diagnosed with ApHCM were consecutively recruited. Diagnosis of ApHCM was based on 2D echocardiography findings. Hypertrophy of

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apical segments was defined as more than 12 mm at end-diastole in the apical short-axis plane. Great care was taken to enrol ApHCM patients with involvement of the apex only.22 Patients in normal sinus rhythm were exclusively recruited. Patients with any clinical evidence of ischaemic heart disease or significant valvular disease by echocardiography were systematically excluded. Other types of HCM were also excluded. Apart from ApHCM patients, age-matched and gender-matched normal volunteers, who attended a healthcare centre at our hospital, were also invited in a consecutive manner and served as the control group. Normal controls had no cardiac symptoms, no relevant medical history and normal electrocardiography, computed tomographic coronary angiography and echocardiography findings. The study protocol was approved by the institutional review board of our hospital and written informed consent was obtained from all participants before enrolment.

### Standard echocardiography

Transthoracic echocardiography was performed using a commercially available echocardiography machine (Vivid 7, GE Medical System, Horten, Norway) with subjects in the left lateral decubitus position. A routine standard echocardiographic examination was performed, which included measurements of LV systolic and diastolic dimensions, wall thickness, LV ejection fraction (EF), mitral inflow velocities and mitral annular velocities. LV EF was calculated using the biplane Simpson’s method. Peak early (E) and late (A) diastolic velocities of mitral inflow were measured using a pulsed-wave Doppler at the tip of mitral valve leaflets. Systolic (S') and early (E') and late (A') diastolic mitral annular velocities were acquired at the septum in apical four-chamber view.

### Speckle tracking echocardiography (STE)

After standard routine echocardiography, scanning of apical and basal short-axis planes was performed to quantify basal and apical LV rotation using an MSS probe without a dual-focusing tool. Frame rate (range 80–100 frames/s) and probe frequency (range 1.7–2.0 MHz) were adjusted during end-expiratory breath hold for optimal image acquisition. Sector width and image depth were optimised to maintain an adequate frame rate without losing 2D image quality. Basal and apical levels were defined as the point of the tips of mitral valve leaflets and just proximal to the level with LV luminal obliteration at the end-systolic period, respectively.23 Great care was taken to make the LV cross-section as circular as possible. To obtain reliable values, three consecutive heart beats were digitally stored in cine-loop format and were analysed.

### Image analysis

Image analysis was performed by one independent cardiologist using a customised dedicated software package (EchoPac 7.05 for PC, GE Medical System). LV endocardial borders were manually traced at end-systolic phase for STE analysis. The reliability of tracking was confirmed by the reliability parameter offered by the system (V = valid tracking; X = unacceptable tracking), and was again visually checked. LV rotations and rotation rates at the basal or apical short-axis planes were determined as average angular displacement of six myocardial segments. Curves of the averaged basal and apical rotation/twisting rate in six segments and LV twist/twisting rate can be directly generated from this version of the EchoPac program, and transported to the worksheet (Microsoft Excel, 2007). The systolic duration was measured from the onset of the QRS to the aortic valve closure, and the diastolic duration was calculated from the RR interval and systolic duration. To account for variable heart rates between subjects, the time values were normalised to the percentage of systolic duration (%) and then the parameters associated with LV twist mechanics were calculated. Peak positive twisting rate, peak untwisting rate, time to peak positive twisting velocity and time to peak untwisting velocity were measured. LV peak radial and circumferential strains were obtained by averaging the peak radial and circumferential strain of six myocardial segments at the apex and base.

For calculation of biplanar strain, circumferential and longitudinal components of strain were measured at the septal and lateral LV walls. Biplanar strain magnitude and vector angle (relative to the short-axis plane) were calculated using the method given by Carasso et al.9 LV twist-volume and twist-radial displacement loops were also generated, as previously described. Briefly, time was normalised to the RR interval. LV instantaneous volume was calculated using the formula employed in a study by Notomi et al, with an assumption of a prolate-ellipsoid LV configuration.

\[ V(t) = \frac{\pi}{6} \times L(t) \times S(t^2) \]

in which L(t) is the long-axis length and S(t) is the short-axis dimension at time of t. L(t) and S(t) were calculated from longitudinal and radial displacement, respectively, as previously described.

### Statistical analysis

Data are expressed as means (SD) or percentages, as appropriate. Independent t test was adopted for inter-group comparisons after normality testing. Correlations between LV twist and other parameters were assessed by Pearson’s correlation analysis. Inter-observer and intra-observer variabilities were evaluated from 10 randomly selected samples using the Bland-Altman analysis. All statistical analyses were performed using SPSS 13.0 and p values of <0.05 were considered statistically significant.

### RESULTS

#### Study population

Thirty-two ApHCM patients were initially recruited for this study, but 11 patients were excluded owing to inadequate image quality for analysis. Consequently, the final study population consisted of 21 ApHCM patients. The same number of age-matched and sex-matched healthy subjects served as normal controls. Table 1 summarises the characteristics of the study population. In brief, LV EF was slightly higher in ApHCM patients; however, LV stroke volume was lower in ApHCM patients than in controls. As was expected, E’ was significantly lower in ApHCM patients.

### Comparison of LV twist, rotation and twisting and untwisting rate between the two groups

LV twist, apical and basal rotation and twisting rates for normal controls and ApHCM patients are summarised in table 2. Peak apical rotation was markedly lower in ApHCM patients (19.5° (5.0°) for normal controls vs 12.0° (4.3°) for ApHCM patients, p<0.001), and peak basal rotation tended to be lower in patients, without statistical significance (−3.1° (2.6°) for normal controls vs −6.6° (3.3°) for ApHCM patients, p = 0.11). As a result, peak LV twist was significantly lower in ApHCM patients (22.6° (5.5°) for normal controls vs 18.1° (5.0°) for ApHCM patients, p = 0.007). Whereas, a slight fall in peak
LV twisting rate was observed in ApHCM patients with borderline statistical significance (p = 0.07). Peak LV untwisting rate during early diastole was significantly lower in ApHCM patients (2143.5 (39.4)°/s for normal controls vs 2101.0 (37.0)°/s for ApHCM patients, p = 0.001). Of interest, a decrease in peak apical untwisting rate during early diastole appeared to be more pronounced than a decrease in peak basal untwisting rate in ApHCM patients (table 2). Time to peak untwisting rate was significantly delayed in ApHCM patients (14.6 (8.9) for normal controls vs 22.8 (12.5) for ApHCM patients, p = 0.009), which suggested global LV diastolic dysfunction in ApHCM patients. Representative examples for LV rotation, twist and twisting and untwisting rate are demonstrated for a normal subject and an ApHCM patient in figure 1.

Correlations with conventional echocardiographic parameters
LV EF showed no correlation with LV twist for all study participants (R = 0.04, p = 0.81). E and E’ was correlated with peak early diastolic untwisting rate (R = 0.43, p = 0.004, and R = 0.48, p = 0.001, respectively), however E/E’ did not (R = 0.25, p = 0.11). Neither LV end-diastolic volume nor LV wall thickness was associated with the extent of LV twist in ApHCM (p > 0.05).

Circumferential, longitudinal and biplanar strains
The magnitude of circumferential, longitudinal and biplanar strains was significantly reduced in apical segments of ApHCM patients in comparison with normal controls, as shown in figure 2. However, biplanar strain angle was comparable in all segments between the two groups and demonstrated no

**Table 1** Characteristics of the study population

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Normal controls (n = 21)</th>
<th>ApHCM patients (n = 21)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51.2 (14.0)</td>
<td>54.0 (12.4)</td>
<td>0.50</td>
</tr>
<tr>
<td>Male gender, n (%)</td>
<td>18 (86)</td>
<td>21 (100)</td>
<td>0.23</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>63.9 (11.00)</td>
<td>63.3 (6.4)</td>
<td>0.85</td>
</tr>
<tr>
<td>LV ESV (ml)</td>
<td>42.6 (9.1)</td>
<td>29.0 (10.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV EDV (ml)</td>
<td>114.2 (20.9)</td>
<td>90.9 (23.6)</td>
<td>0.002</td>
</tr>
<tr>
<td>LV SV (ml)</td>
<td>71.7 (12.9)</td>
<td>61.9 (16.7)</td>
<td>0.04</td>
</tr>
<tr>
<td>LV EF (%)</td>
<td>62.8 (3.0)</td>
<td>68.5 (6.8)</td>
<td>0.002</td>
</tr>
<tr>
<td>E (m/s)</td>
<td>0.64 (0.19)</td>
<td>0.59 (0.14)</td>
<td>0.38</td>
</tr>
<tr>
<td>A (m/s)</td>
<td>0.77 (1.11)</td>
<td>0.55 (0.17)</td>
<td>0.36</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.17 (0.55)</td>
<td>1.19 (0.53)</td>
<td>0.94</td>
</tr>
<tr>
<td>DT (ms)</td>
<td>208 (45)</td>
<td>201 (43)</td>
<td>0.51</td>
</tr>
<tr>
<td>S’ (cm/s)</td>
<td>7.6 (1.4)</td>
<td>6.8 (1.4)</td>
<td>0.08</td>
</tr>
<tr>
<td>E’ (cm/s)</td>
<td>7.4 (2.2)</td>
<td>4.9 (1.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>A’ (cm/s)</td>
<td>7.8 (1.5)</td>
<td>7.7 (2.2)</td>
<td>0.79</td>
</tr>
<tr>
<td>E/E’ ratio</td>
<td>8.8 (2.2)</td>
<td>12.9 (4.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV diastolic function†</td>
<td>8 (38%)</td>
<td>1 (4.8%)</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>13 (62%)</td>
<td>8 (38%)</td>
<td></td>
</tr>
<tr>
<td>Pseudonormalisation</td>
<td>0 (0%)</td>
<td>12 (57%)</td>
<td></td>
</tr>
</tbody>
</table>

*By biplane Simpson’s method.
†Based on the combined assessment of the mitral inflow and mitral annular velocity patterns.
A, late diastolic mitral inflow velocity; A’, late diastolic mitral annular velocity; ApHCM, apical hypertrophic cardiomyopathy; DT, deceleration time; E, early diastolic mitral inflow velocity; E’, early diastolic mitral annular velocity; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; LV, left ventricle; S’, systolic mitral annular velocity; SV, stroke volume.

**Table 2** LV twist mechanics of study population

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Normal controls (n = 21)</th>
<th>ApHCM patients (n = 21)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak apical rotation (°)</td>
<td>19.5 (5.0)</td>
<td>12.0 (4.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak basal rotation (°)</td>
<td>-8.1 (2.6)</td>
<td>-6.6 (3.3)</td>
<td>0.11</td>
</tr>
<tr>
<td>Peak LV twist (°)</td>
<td>22.6 (5.5)</td>
<td>18.1 (5.0)</td>
<td>0.007</td>
</tr>
<tr>
<td>Time to peak systolic twist (%)†</td>
<td>96.6 (6.2)</td>
<td>95.6 (16.4)</td>
<td>0.82</td>
</tr>
<tr>
<td>Peak apical twisting rate (°/s)</td>
<td>98.8 (12.7)</td>
<td>78.4 (25.4)</td>
<td>0.002</td>
</tr>
<tr>
<td>Peak basal twisting rate (°/s)</td>
<td>-68.7 (27.3)</td>
<td>-58.5 (22.8)</td>
<td>0.20</td>
</tr>
<tr>
<td>Peak LV twisting rate (°/s)</td>
<td>130.3 (38.8)</td>
<td>110.7 (29.8)</td>
<td>0.07</td>
</tr>
<tr>
<td>Time to peak LV twisting rate (%)†</td>
<td>52.4 (13.1)</td>
<td>49.4 (14.1)</td>
<td>0.49</td>
</tr>
<tr>
<td>Peak apical untwisting rate during early diastole (°/s)</td>
<td>-104.0 (30.0)</td>
<td>-65.9 (24.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak basal untwisting rate during early diastole (°/s)</td>
<td>71.2 (21.3)</td>
<td>56.1 (23.5)</td>
<td>0.04</td>
</tr>
<tr>
<td>Peak LV untwisting rate during early diastole (°/s)</td>
<td>-143.5 (39.4)</td>
<td>-101.0 (37.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>Time to peak untwisting rate during early diastole (%)†</td>
<td>14.6 (6.9)</td>
<td>22.8 (12.5)</td>
<td>0.009</td>
</tr>
</tbody>
</table>

*Normalised to systolic duration, †normalised to diastolic duration.
regional variability within each group. When the entire study population was combined without breakdown according to disease status, biplanar strain of the apical segments displayed a significant positive correlation with apical rotation (apicoseptal strain: $R = 0.57$, $p < 0.001$, apicolateral strain: $R = 0.72$, $p < 0.001$) and LV twist (apicoseptal strain: $R = 0.47$, $p = 0.003$, apicolateral strain: $R = 0.42$, $p = 0.01$), but not with basal rotation ($p > 0.05$), underlining a more crucial role of apical segments in maintaining LV systolic performance.

LV twist-volume and twist-radial displacement loops

Average LV twist-volume curves for controls or ApHCM patients throughout a cardiac cycle were constructed (fig 3A). It assumed the figure-of-eight configuration in both groups, but a slight difference in shape could be noted. In normal controls, three principal phases could be clearly identified: a linear systolic phase from mitral valve closure to aortic valve closure, a rapid early untwisting phase from aortic valve closure to an inflection point between mitral valve opening and peak early filling velocity and, lastly, a late untwisting phase between the inflection point to mitral valve closure, similar findings to those previously described. Contrary to normal controls, there were significant differences in both systolic and early diastolic untwisting phases of the LV twist-volume curve in ApHCM patients. The slopes of the systolic phase were significantly smaller in ApHCM patients (0.54 (0.14) for normal controls vs 0.36 (0.12) for ApHCM, $p < 0.001$). This is also true for the slope of early diastolic untwisting phase, in which the slope was characterised by the loss of the inflection point between mitral valve opening and peak early filling velocity in ApHCM patients.

Figure 2  Circumferential, longitudinal and biplane left ventricular (LV) strain was significantly decreased in apical segments of ApHCM (apical hypertrophic cardiomyopathy) patients. Biplanar strain angle was no different between groups. *$p < 0.001$; †$p < 0.05$. 

Heart failure and cardiomyopathy

Figure 1  Left ventricular (LV) twist and twisting rate in normal and apical hypertrophic cardiomyopathy (ApHCM). LV twist was lower in ApHCM patients (right upper) than normal controls, and this was mainly attributed to a relative decrease in the apical rotation. Peak LV twisting and untwisting rates were also significantly lower in ApHCM patients (right lower) than the normal controls (left lower). Green line, LV twist and LV twisting rate; blue line, apical rotation and rotation rate; red line, basal rotation and rotation rate. AUR, peak apical untwisting rate; AVC, aortic valve closure; AVO, aortic valve opening; BUR, peak basal untwisting rate during early diastole; MVC, mitral valve closure; MVO, mitral valve opening; PAR, peak apical rotation; LVUV, LV untwisting velocity; PATR, peak apical twisting rate; PBR, peak basal rotation; PBTR, peak basal twisting rate; PLVT, peak LV twist; PLVTR, peak LV twisting rate.

Heart failure and cardiomyopathy

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Heart failure and cardiomyopathy

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as shown in figure 3. The value of the slope between peak twist and mitral valve opening was $1.47^\circ$ (0.42\textdegree) for controls and 0.69$^\circ$ (0.34\textdegree) for ApHCM ($p<0.001$). The slope of late untwisting phase did not different between the two groups (0.55$^\circ$ (0.69\textdegree) for controls vs 0.54$^\circ$ (0.58\textdegree) for ApHCM, $p = 0.05$). The slope of early untwisting phase was correlated with peak twist ($R = 0.38$, $p = 0.001$), apical rotation ($R = 0.55$, $p = 0.001$), peak untwisting rate during early diastole ($R = -0.58$, $p = 0.001$), apical biplanar strain ($R = 0.49$, $p = 0.01$) and $E/E'_R$ ($R = -0.41$, $p = 0.02$).

The LV twist-radial displacement loop showed a similar pattern of the figure-of-eight configuration (fig 3B). The slopes of the systolic phase were significantly smaller in ApHCM patients (3.98$^\circ$ (1.52\textdegree)/mm for normal controls vs 2.78$^\circ$ (0.95\textdegree)/mm for ApHCM, $p = 0.01$). In ApHCM patients, the loss of the inflection point between mitral valve opening and peak early filling velocity was again observed during the early diastolic untwisting phase (9.48$^\circ$ (5.68\textdegree)/mm for normal controls vs 5.75$^\circ$ (2.76\textdegree)/mm for ApHCM, $p = 0.01$), leading to the simultaneous occurrence of LV untwisting and chamber dilation. Similar to LV twist-volume loop, we failed to find a significant difference in the slope of late diastolic untwisting loop between controls and ApHCM (2.24$^\circ$ (1.43\textdegree)/mm for normal controls vs 2.84$^\circ$ (1.48\textdegree)/mm for ApHCM, $p = 0.26$).

**Measurement reproducibility**

Inter-observer and intra-observer variabilities for measurement of LV twist were determined by two independent blinded cardiologists who analysed 10 randomly selected patients. Inter-observer variability showed a correlation coefficient of 0.92 (standard error of the estimate = 1.6). In terms of intra-observer variability, a correlation coefficient was found to be 0.96 (standard error of the estimate = 1.2). Bland-Altman analysis also showed good agreements for intra-observer and inter-observer variabilities (limits of agreement 0.48$^\circ$ (2.51\textdegree) and 0.51$^\circ$ (3.27\textdegree), respectively).

**DISCUSSION**

We analysed LV twist mechanics of ApHCM patients in association with temporal changes in LV volume, strains and displacement. LV twist was found to be markedly reduced in ApHCM patients, compared to the normal controls. Radial and biplanar strains were also significantly reduced in the apical segments, possibly being linked with predominant involvement of myocardial hypertrophy and fibrosis in these segments. The decline in the extent of the slopes of systolic and early diastolic untwisting phases according to LV twist-volume and twist-radial displacement loops in ApHCM patients can be regarded as LV diastolic functional impairment in ApHCM patients who had an even higher LV EF (a conventional, widely used echocardiographic variable representing LV systolic function).

**Effects of apical myocardial dysfunction on LV twist**

From an anatomical standpoint, the orientation of LV myofibres changes smoothly from a right-handed helix in the subendocardium to a left-handed helix in the subepicardium. As was shown by Taber et al, the contraction of the subepicardial fibres caused the apex to rotate counterclockwise and base in the clockwise direction, whereas the contraction of the subendocardial fibres causes the apex and base to rotate in exactly the opposite direction. Since the larger radius of the outer epicardial layer gives subepicardial fibres a mechanical advantage over subendocardial fibres, the overall direction of rotation is determined by the outer subepicardial fibres—that is, counterclockwise rotation at the apex and clockwise rotation at the base. Because most myocardial diseases initially cause subendocardial fibre dysfunction, and attenuate clockwise rotation, which is known to be generated by subendocardial fibres, it is anticipated in conjunction that this dysfunction is associated with a corresponding increase in unopposed counter-clockwise motion caused by the subepicardial fibres, and a significant augmentation of LV twist. Indeed, a significant increment in LV twist is observed in those with an impaired subendocardial function—that is, subendocardial ischaemia and aortic stenosis.

ApHCM, a subtype of HCM, is a unique myocardial disease, in which apical involvement is a predominant feature regardless of disease stage. This stands in sharp contrast to most other cardiac diseases, in which subendocardial dysfunction is the initial manifestation. Although a significant increase in or at least preservation of LV twist was identified in HCM patients with asymmetrical septal hypertrophy, little information is available on LV twist mechanics in patients with ApHCM. Owing to the clear difference in myocardium primarily encroached by pathological processes, LV twist observed in ApHCM is likely to differ from that observed in asymmetrical septal HCM, in which the basal and mid-septal myocardium are the main foci of involvement. Furthermore, given the fact that the apex is known to be a key player that actively participates in the decision process of LV twist mechanics, we assumed that a localised, pathological hypertrophy at the LV apex would exert a remarkable influence on global LV twist mechanics and possibly reduce LV twist. The present study demonstrates that predominant invasion of a myocardial pathology into the apex significantly decreases the global LV twist. The impact of a localised apical pathology on global LV twist was investigated in ApHCM patients.
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a model of anterior myocardial infarction by Takeuchi et al. They reported a significant correlation between LV twist and LV EF, but their study population had a rather heterogeneous infarct size and transmurality for effectively demonstrating the effect of apical dysfunction on global LV twist and global LV systolic function. Furthermore, they did not address the correlation between apical myocardial function and apical rotation. In contrast, based on morphological considerations and a previous report using delayed enhancement of cardiac magnetic resonance imaging, ApHCM can be considered a model of predominant apical pathological involvement, and thus can provide an opportunity to obtain information about the effect of apical myocardial pathology on the global LV twist.

LV twist and untwist mechanics in HCM

HCM is a primary myocardial disease with heterogeneous clinical and morphological characteristics. Myofibre disarray, which increases in loose connective tissue, and fibrosis are known to be essential components of this disease. As demonstrated by cardiac magnetic resonance imaging studies, delayed gadolinium enhancement tends to be found at the mostly thickened segments, which suggests predominant involvement of myocardial fibrosis at hypertrophied segments. This concept is further supported by the findings of a pathological study, which clearly demonstrated that matrix collagen is more prominent in the hypertrophied legion and that this is associated with primary morphological abnormalities, like myocardial hypertrophy. In ApHCM patients, delayed enhancement of cardiac magnetic resonance imaging showed that pathological fibrotic involvement was found only in the LV apex and not in the basal or mid regions.

LV twist in HCM was first reported by Young et al using cardiac magnetic resonance imaging with tagging technique. They included patients with asymmetrical septal hypertrophy and unexpectedly found a significant rise in LV twist mainly the result of the augmentation of the apical rotation. In another study, Carasso et al demonstrated that LV twist was well preserved in HCM patients with asymmetrical septal hypertrophy, but that systolic twist was apically displaced, which is possibly the result of LV stiffness at the base to mid-level of the myocardium. Despite several publications regarding LV twist mechanics in HCM patients, ApHCM has attracted less attention and LV twist mechanics in ApHCM has not been scrutinised. In the present study, patients with a pure type of ApHCM were exclusively recruited, and we found that apical rotation and circumferential, longitudinal and biplanar types of strain were markedly lower in ApHCM patients than in normal controls. A recent report by Yamada et al showed an involvement of myocardial pathological fibrosis only in the LV apex. Accordingly, we believe that regional pathological hypertrophy at the apex may be responsible for the decline in LV apical rotation and different types of LV strain. Global LV twist is predominantly determined by LV apical rotation. Hence, it is not surprising that LV apical hypertrophy with associated myocardial stiffness induced by fibrotic change markedly diminishes apical rotation, and thus, reduces global LV twist.

In addition, given the close interconnection between the systolic and diastolic function, a reduction of LV twist in ApHCM is expected to contribute to an impairment of LV diastolic function. Impairment of LV diastolic function was further advocated by average plots of LV twist against LV volume or radial displacement, in which untwisting occurs more slowly relative to chamber expansion, producing a simultaneous occurrence of untwisting and chamber expansion in ApHCM patients (fig 3). Also, LV twisting and untwisting curves in figure 1 illustrate a significant reduction in LV early untwisting rate and its delay in time even after mitral valve opening in ApHCM patients. A decrease in the rate of early LV untwisting in association with volume expansion is known to be caused by an increment of LV preload. However, this mechanism is unlikely to explain a decline and slowing of LV untwisting observed in ApHCM patients because LV end-diastolic volume, a representative of LV preload, is much smaller in ApHCM than in normal controls. Therefore, myocardial pathology itself is likely to be responsible for this decrease, instead of the influence of haemodynamic change. Considering a cardinal role of the LV apex in LV systolic and diastolic performance, it is not surprising that apical pathological involvement itself can be enough to generate the global reduction in LV systolic and diastolic function.

Traditionally, LV twist is known to contribute to uniform distribution of the LV fibre stress across the wall during systole. The clinical significance of a reduced LV twist in ApHCM patients is uncertain, but it is possible that a non-uniform distribution of the LV fibre stress stemming from a decrease in the LV twist may be associated with a series of morbid events, sometimes found in patients with ApHCM—that is, atrial fibrillation, myocardial infarction without epicardial coronary artery disease, heart failure and ventricular arrhythmia. Further research is warranted on the possible relation between a decline in LV twist and future morbid events in patients with ApHCM.

Study limitation

Several limitations of the present study require consideration. First, the number of subjects enrolled was relatively small. Second, we did not include other types of HCM, and thus, we cannot directly compare the findings in ApHCM with other type of HCM, such as asymmetrical septal hypertrophy. Nevertheless, given the results of Young et al, we can indirectly presume the possible differences of LV twist mechanics between ApHCM and asymmetrical septal HCM.

Conclusions

Unlike the situation observed HCM patients with asymmetrical septal hypertrophy, LV twist decreased significantly in ApHCM patients primarily because of a remarkable fall in apical rotation. Given the beneficial effect that LV twist has on the distribution of LV wall stress, the prognostic implications of a decline in LV twist in ApHCM patients warrants further studies. Because the localised involvement of a myocardial pathology can significantly influence global LV twist mechanics, LV twist should be considered a myocardial morphological characteristic, especially in HCM patients.

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