Evaluation of myocardial deformation pattern of left ventricular noncompaction by cardiac magnetic resonance tissue tracking

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Introduction Left ventricular noncompaction (LVNC) is a rare dysplastic heart disease characterized by a large number of abnormal myocardial trabeculations and deep intertrabecular recesses. Its clinical manifestations vary extensively, and about two-thirds of patients with LVNC develop heart failure leading to poor prognosis.[1] At present, the diagnosis of LVNC is mainly based on the ratio of the thickness of noncompacted to compacted myocardium (NC/C). The echocardiographic criteria proposed by Jenni et al[2] and cardiac magnetic resonance (CMR) criteria proposed by Petersen et al[3] are most widely used in the diagnostic workup. However, this single dimension measurement based on morphology overlooks early cardiac dysfunction. Therefore, we need more accurate methods to evaluate left ventricular systolic function in patients with LVNC.

Cardiac magnetic resonance plays a major role in the diagnostic workup of cardiac disease due to its excellent versatility and postprocessing techniques compared with computed tomography and echocardiography.[4-6] Recently, CMR tissue-tracking (CMR-TT) can quantitatively reflect myocardial deformation in different directions based on routine cine sequences,[7] which has been widely used in clinical studies, such as hypertrophic cardiomyopathy, myocardial infarction, and valvular disease.[8] However, the application of CMR-TT in LVNC is relatively rare. Thus, the purpose of this study is to explore myocardial deformation pattern of LVNC and its correlation with cardiac function based on CMR-TT, so as to provide information of potentially incremental value in evaluating the prognosis of patients with LVNC.

Methods A total of 23 patients with LVNC (mean [SD] age, 48 [13] years; 17 men) who underwent echocardiography and CMR were retrospectively admitted to our hospital from January 2012 to March 2019. All of them met the echocardiography[2] and CMR criteria[3] and were divided into 2 groups according to left ventricular ejection fraction (LVEF) measured by CMR: 10 patients with LVEF ≥50% and 13 patients with LVEF <50%. The control group included 20 healthy subjects. This retrospective study complies with the Declaration of Helsinki and was approved by our institutional ethics committee (no. 20180226-60). Requirement for written informed consent was waived because of the retrospective nature of the study.

A detailed description of acquisition and analysis of echocardiography and CMR images can be found in Supplementary material.

Statistical analysis All baseline characteristics and cardiac function parameters analyses were performed using the SPSS 23.0 software (IBM, Armonk, New York, United States). Continuous variables were expressed by mean (SD) with the *t* test or the 1-way analysis of variance test, or by median (interquartile range [IQR]) with the Kruskal–Wallis test, and categorical data were expressed as frequency (percentage) and assessed by the χ^2 test. The Spearman rank correlation analysis was used to assess the correlation between myocardial strain and the number

of NC segments and the NC/C ratio. Intra- and interobserver variability for reproducibility was assessed using intraclass correlation coefficient. A 2-tailed *P* value of less than 0.05 was considered significant.

Results and discussion Compared with the control group, global longitudinal strain (GLS) and mid-longitudinal strain (LS) of all patients were decreased, but more obviously in those with LVEF less than 50% (*P* <0.05). Basal and apical LS were decreased in all patients regardless of LVEF. Global and regional radial strain (RS) and circumferential strain (CS) were reduced only in patients with LVEF less than 50%, while there was no decline in patients with LVEF 50% or greater (*P* >0.05) (TABLE 1 and Supplementary material, *Figure S2*).

The apical segments were involved in all patients, but no NC myocardium was found in the basal section. The NC/C ratio in patients with LVEF less than 50% was comparable with those with LVEF 50% or greater (*P* = 0.15). Late gadolinium enhancement (LGE) was only found in the group of patients with LVEF less than 50% (Supplementary material, *Table S1*). There was no correlation between myocardial strain and the number of NC segments and the NC/C ratio (*P* >0.05).

We found good reproducibility of all strain measurements for intra- and interobserver

TABLE 1 Baseline characteristics and cardiac function parameters of LVNC and control groups

Parameter	Control (n = 20)	LVNC + EF ≥50% (n = 10)	LVNC + EF <50% (n = 13)	*P* value
Age, y	48 (11)	42 (9)	54 (14)	0.05
Male, n (%)	10 (50)	7 (70)	10 (77)	0.26
Height, cm	168 (9)	168 (6)	166 (7)	0.99
Weight, Kg	69 (13)	65 (13)	64 (14)	0.57
BSA, m^2	1.8 (0.2)	1.7 (0.2)	1.7 (0.2)	0.60
LVEF, %	63.2 (59.3–65.6)	61.8 (52.5–64.7)	19.2 (15.7–32.7)[a,b]	<0.001
LVEDVi, ml/m^2	78.6 (69.5–88.7)	83.4 (72.2–99.8)	176.7 (126.0–200.7)[a,b]	<0.001
LVESVi, ml/m^2	31.0 (23.3–34.8)	35.2 (28.7–39.4)	139.1 (91.6–163.8)[a,b]	<0.001
SVi, ml/m^2	47.5 (43.3–55.3)	48.2 (41.9–66.6)	32.0 (28.5–42)[b,c]	0.005
GRS, %	30.9 (23.5–37.4)	21.2 (15.9–23.3)	6.7 (5–12.5)[a]	<0.001
GCS, %	–20.0 (–21.4 to –15.8)	–15.0 (–16.8 to –13.1)	–5.4 (–9.4 to –4.1)[a,b]	<0.001
GLS, %	–12.4 (2.4)	–9.2 (2.4)[c]	–5.4 (2.7)[a,b]	<0.001
Basal RS, %	45.1 (31.6–53.8)	35.3 (29.5–42.7)	11.6 (7.1–19)[a,b]	<0.001
Basal CS, %	–15.7 (3.8)	–12.8 (3.2)	–7.1 (2.9)[a,b]	<0.001
Basal LS, %	–8.8 (3.2)	–4.3 (4.4)[c]	–4 (4)[c]	0.001
Mid-RS, %	24.4 (18.9–28.3)	11.9 (10.4–15.2)	6 (4–9.8)[c]	<0.001
Mid-CS, %	–18.9 (–21.1 to –15.7)	–13.7 (–16.4 to –12.2)	–4.7 (–8.5 to –3.7)[a,b]	<0.001
Mid-LS, %	–11.7 (2.8)	–8.7 (2.5)[c]	–5.2 (2.4)[a,b]	<0.001
Apical RS, %	31.2 (20.7–37.3)	16.0 (11.2–18.4)[c]	7.2 (4.2–13.1)[a]	<0.001
Apical CS, %	–23.5 (–25.8 to –20.1)	–18.3 (–19.1 to –16.3)	–6.2 (–9.8 to –3.8)[a,b]	<0.001
Apical LS, %	–15.4 (–17.5 to –14.3)	–7.4 (–10.5 to –6.5)[c]	–5.3 (–8.4 to –4.4)[a]	<0.001

Data are presented as mean (SD) or median (interquartile range) unless otherwise indicated.

a *P* <0.001 vs control group

b *P* <0.05 vs LVNC + EF ≥50% group

c *P* < 0.05 vs control group

Abbreviations: BSA, body surface area; CS, circumferential strain; EF, ejection fraction; GCS, global circumferential strain; GLS, global longitudinal strain; GRS, global radial strain; LS, longitudinal strain; LVEDVi, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; LVESVi, left ventricular end-systolic volume index; LVNC, left ventricular noncompaction; RS, radial strain; SVi, stroke volume index

variability. Summaries of the intraclass correlation coefficient values are shown in Supplementary material, *Table S2*.

It has been reported that over half of patients with LVNC may develop heart failure leading to poor prognosis.[1] In this study, 57% of patients had decreased LVEF, and their cardiac function was significantly lower than that of healthy controls and patients with preserved LVEF. Therefore, early estimation of cardiac function is of particular importance in those patients.

We described patterns of global and regional strain in LVNC. In patients with preserved LVEF, GLS and regional LS decreased compared with controls. GLS and mid-LS were more decreased in patients with declined LVEF, which suggested that LS was impaired prior to heart failure. This was in accordance with data from Bellavia et al[9] who proposed that LS is the best strain index to differentiate those with LVNC from healthy people. As the disease progresses, the decrease in GLS and mid-LS becomes increasingly significant, and obvious reduction in global and regional RS and CS was observed in patients with decreased LVEF except for declined LS parameters. We speculate that patients with low LVEF are more likely to present with decreased RS and CS, which can potentially serve as prognostic markers of impending heart failure. This might be explained by the fact that contraction of subendocardial fibers contributes to longitudinal shortening, contraction of subepicardial fibers contributes to circumferential shortening, and both contribute to radial thickening.[7] During embryonic period, left ventricular myocardium gradually compacts from base to apex, epicardium to endocardium, septum to lateral wall, so NC segments always appear in the endocardial myocardium.[1] Thus, LS is first declined at the early stage in patients with LVNC, while RS and CS are within normal range.

The apex is more easily affected than the basal section, which is mainly related to the process of myocardial compaction.[1] In this study, the most commonly involved part was apical free wall. However, we did not find any correlations between morphological changes (the number of NC segments and the NC/C ratio) in patients with LVNC and myocardial strain and LVEF. This might be related to a broader range of myocardial abnormalities, possibly affecting compacted layer of myocardium near the noncompacted region, resulting in a more disorderly crossed array with increased interstitial collagen in subepicardial myofibers of patients with LVNC.[10] Moreover, decreased LS was also universal among patients regardless of their LVEF, thus indicating that systolic dysfunction was not just related to NC segments.

In addition, it is more likely that LGE existed in patients with impaired LVEF, suggesting that LGE was associated with left ventricular dysfunction. Nucifora et al[11] found that the presence and extent of LGE in patients with LVNC were correlated with the decrease in LVEF. In this study, LGE also appeared in compacted segments. Previous histopathology showed that both compacted and noncompacted myocardium in patients with LVNC could be accompanied by endocardial thickening, focal fibroelastic tissue proliferation, and fibrosis, and even ischemia in compacted segments.[10] It is indicated that LVNC is not a local cardiomyopathy with abnormal morphology, but a diffuse disease involving the entire myocardium.

In conclusion, patients with LVNC exhibit a strain pattern characterized by a decreased LS. Reduced RS and CS are more likely to occur in patients with LVEF less than 50%, which may be an important cause of heart failure in LVNC. Myocardial strain parameters based on CMR-TT may constitute potential prognostic markers for this disease.

The main limitation of the study is that the effect of LGE on myocardial strain of each segment needs further assessment.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

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CONFLICT OF INTEREST None declared.

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