IJC Heart & Vasculature 5 (2014) 9–14

Contents lists available at ScienceDirect

IJC Heart & Vasculature

journal homepage: http://www.journals.elsevier.com/ijc-heart-and-vasculature

Myocardial deformation pattern in left ventricular non-compaction: Comparison with dilated cardiomyopathy

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ARTICLE INFO

Article history:
Received 12 October 2014
Accepted 4 November 2014
Available online 12 November 2014

Keywords:
Left ventricular noncompaction
Echocardiography
2D speckle tracking
Dilated cardiomyopathy

ABSTRACT

Introduction: Left ventricular (LV) systolic dysfunction is the most frequent initial presentation of patient with LV noncompaction (NC). Our objectives were to evaluate myocardial contraction properties in patients with LVNC and the relationship of non-compacted segments with the degree of global and regional systolic deformation.
Methods: We included 50 LVNC with an echocardiography and speckle imaging calculation of peak longitudinal strain (PLS). Each of the 16 LV myocardial segments was defined as NC (ratio NC/compacted layer > 2), borderline (NC/C 0–2) and compacted (NC/C = 0). Basal, median and apical strain values were calculated as the average of segmental strain values. For comparison a group of 50 patients with dilated cardiomyopathy (DCM) underwent the same measurements.
Results: There was no statistical difference between the 2 groups for any conventional LV systolic parameters. A characteristic deformation pattern was observed in LVNC with higher strain values in the LV apical segments (-12.8 ± 5.9 vs -10.7 ± 5.7) and an apical–basal ratio (1.52 ± 0.73 vs 1.12 ± 0.42; $p < 0.001$). There was no correlation between LV function and the degree of NC. Among 726 segments, compacta thickness was thinner in NC vs C segments (6.4 ± 1.4 vs 7.7 ± 1.8 mm; $p < 0.05$). There was no difference in WMS but regional strain values were significantly higher in NC compared to C segments (-13.1 ± 6.1 vs -10.2 ± 6.3; $p < 0.05$).
Conclusions: Compared to DCM, LVNC presented with relatively preserved apical deformation as compared to basal segments. Lower regional deformation values in compacted segments confirm the concept that LVNC is a phenotypic marker of an underlying diffuse cardiomyopathy involving both C and NC myocardium.

1. Introduction

According to the European Society of Cardiology Working Group on Myocardial and Pericardial Disease, left ventricular noncompaction (LVNC) is still an unclassified cardiomyopathy [1]. This cardiomyopathy is characterized by trabeculations and recesses within the ventricular myocardium. LV systolic dysfunction associated with heart failure is the main predictor of outcome and the most frequent initial presentation of patient with LVNC [2–4]. But its mechanism and relation to non-compaction is not clearly established.

In the context of dilated and hypokinetic left ventricles, it is unclear whether LVNC is a morphologic trait rather than a distinct cardiomyopathy. Pronounced trabeculations are present in both LVNC and dilated cardiomyopathy (DCM), which sometimes makes the differentiation difficult.

Although cardiac magnetic resonance imaging (CMR) has emerged as the gold standard modality in the diagnosis and evaluation of this disease [5], echocardiography is critical to perform and is still used as the first-line imaging. The echocardiographic criteria of Chin, Jenni and Stöllberger [6–8] are used to confirm the diagnosis. Furthermore, Paterick and Tajik proposed to introduce the concept of myocardium contraction of the NC segments as an additional diagnosis criterion [9], so that abnormal ventricular function and myocardial deformation patterns, on top of criteria of pathologic hypertrabeculation may lead to diagnose LVNC.

Deformation imaging with peak systolic longitudinal strain (PLS) calculation is able to evaluate the degree and the extent of LV dysfunction and differentiate mechanisms of segmental abnormalities. Dilated cardiomyopathy is associated with reduction of strains in all directions

Abbreviations: LV, left ventricle; LVNC, left ventricular noncompaction; DCM, dilated cardiomyopathy; NC, noncompacted; C, compacted; 2D, two dimensional; 2DSI, two dimensional speckle imaging; PLS, peak longitudinal strain; GLS, global longitudinal strain; CMR, cardiovascular magnetic resonance.

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http://dx.doi.org/10.1016/j.ijcha.2014.11.001

with attenuation of LV twist [10]. This character is a marker of global systolic dysfunction and helps to understand the physiological nonuniformity of regional LV performance [11–12]. Furthermore, global longitudinal strain value is a prognostic marker of heart failure in cardiomyopathy [13–14].

The purpose of this study was to illustrate the role of deformation imaging to evaluate the relationship between the degree and the extent of NC with regional and global systolic dysfunction. The second objective was to define a new tool using functional approach to help in the classification of patients with similar dilated phenotypes and discriminate primitive from non-compacted dilated cardiomyopathy.

2. Material and methods

2.1. Populations

Fifty patients newly referred for LVNC were screened between January 2001 and December 2013. The diagnosis of isolated LVNC was based on the presence of the following criteria: (1) visual appearance of two distinct compacted epicardial layer and a non-compacted endocardial layer; (2) marked trabeculation and deep intertrabecular recesses within the non-compacted layer; (3) non-compacted to compacted end-diastolic myocardial ratio >2, and (4) absence of other associated congenital or acquired heart disease. [6–9]. Atrial fibrillation was an exclusion criterion, because it made it impossible to perform 2D speckle analysis.

In addition, 50 age- and LVEF-matched nonischemic dilated cardiomyopathy (DCM) patients were enrolled in our study (mean age 51.9 ± 15.2 years).

Oral informed consent was obtained from each participant.

2.2. Echocardiographic imaging and analysis

A complete 2-dimensional and Doppler echocardiography (Vivid 7 and Vivid 9, General Electric Medical Systems, Horten, Norway) with 2D speckle tracking analysis was performed in all patients.

2.2.1. 2D standard echocardiography

Echocardiographic images were obtained from the parasternal short axis views at the basal, median and apical levels and from the 3 standard LV apical views (4-, 2- and 3-chambers). All images were acquired at a frame rate of 50 to 70 frames/s for 2D views. For each patient we measured 2D LVEF (Simpson biplane method). Left ventricular mass was calculated by the Teichholz method. Diastolic function was assessed by the E/A and E/E′ ratio and the LA area according to guidelines [15].

LV wall motion was assessed according to a 17 segment model (ACC/AHA) [16]. Each segment was analyzed individually and scored on the basis of its motion and systolic thickening ranging from 1 to 4: 1 = normokinetic or hyperkinetic, 2 = hypokinetic, 3 = akinetic, and 4 = dyskinetic. The wall motion score index (WMSI) was calculated as the sum of all scores divided by the number of segments visualized. Apical segment (apical cap-segment 17) was excluded from the segmental analysis.

2.2.2. Extent and degree of non-compaction

The severity of NC in each of the 16 LV segments was assessed quantitatively by measuring the non-compacted and compacted myocardial layer thickness from the 3 parasternal short axis view in end-diastole (millimeter and percentage of extent). We calculated the ratio between NC and C layer to obtain the value of NC/C ratio. The segmental degree of non-compacted myocardium was categorized into 3 grades: NC if the ratio was above 2; borderline if the NC/C ratio was between 0 and 2 and compacted if there was no trabeculation (NC/C ratio = 0) (Fig. 1).

2.2.3. 2D longitudinal peak systolic strain

Strain measurements were performed offline with a dedicated automated software (Automated Function Imaging, EchoPAC PC, version 110.1.0, GE Healthcare). Only good quality images were used. From each apical view, 3 sample points were placed manually along the endocardium to define the LV base and the apex at the end-systolic frame. Each LV wall was divided into 3 segments (basal, median and apical) and bull's eye according to the 17 segment classification was displayed. The values of longitudinal systolic strain of all the segments were averaged to obtain a 2D global longitudinal strain (GLS) value. Peak longitudinal strain (PLS) was defined as the lowest strain value obtained for the longitudinal direction during systole (before the reference time point of the end of systole). Basal, median and apical strain values were respectively calculated as the average of the strain values of the 6 basal, 6 median, and 4 apical segments.

2.3. Statistical analysis

The statistical analysis was performed using SPSS for Windows (SPSS version 17, Chicago, Illinois). Quantitative values are expressed as the mean value ± SD. Intergroup comparisons were made by the independent samples Student's paired sample *t*-test or Mann–Whitney *U* test when appropriate. We assessed the association of LVEF with the degree of NC by the use of Pearson correlation analysis. A p value < 0.05 was considered to indicate statistical significance. Agreements were assessed for PLS measurement using the method proposed by Bland and Altman for the inter/intra-observer variability repeated by two independent observers in 30 patients.

3. Results

3.1. Baseline TTE characteristics

The mean age was 51.8 ± 15.1 years (39 male/76%). There was no statistical difference in 2D conventional systolic function parameters between LVNC and DCM patients. The mean LVEF was 33.7 ± 11.2% in LVNC and 34.0 ± 10.9% in DCM group. LV tends to be more dilated in LVNC group (LVEDV 190.9 ± 73.0 vs 173.7 ± 69.6 ml, $p = 0.067$). The degree of diastolic dysfunction was similar in the two groups. Only the left atrial area was significantly greater in LVNC patients (26.5 ± 8.2 vs 23.5 ± 8.7 cm^2, $p < 0.005$). Right ventricular function was preserved in both groups.

Conventional echocardiographic characteristics are shown in Table 1.

3.2. Global and regional deformation parameters (Table 2)

The comparison between LVNC and DCM patients showed no significant differences for the global PLS (−10.6 ± 4.9% vs −10.1 ± 4.9%).

The analysis of PLS values at basal and mid-level showed no significant differences between the 2 groups. We observed a significant correlation between LVEF and GPLS ($r = -0.886$; $p < 0.001$) but there was no correlation between the degree and the extent of the non-compaction with the level of systolic dysfunction (LVEF or GPLS).

The analysis of basal, median and apical levels of the LV showed a significant gradient from the base toward the apex among patients with LVNC. In patient with similar LVEF, LVNC group demonstrated a relatively preserved and significantly higher apical LV PLS values compared to DCM patients (−12.8 ± 5.9 vs −10.7 ± 5.7 $p = 0.025$). This difference was confirmed with a ratio of apex/basal strain values of 1.5 ± 0.7 in LVNC group compare to 1.1 ± 0.4 in DCM group ($p < 0.001$).

3.2.1. Segmental analysis (Table 3)

A total of 726 segments were analyzed on 3 parasternal short axis views (90.1% of all segments) in LVNC group. The remaining segments

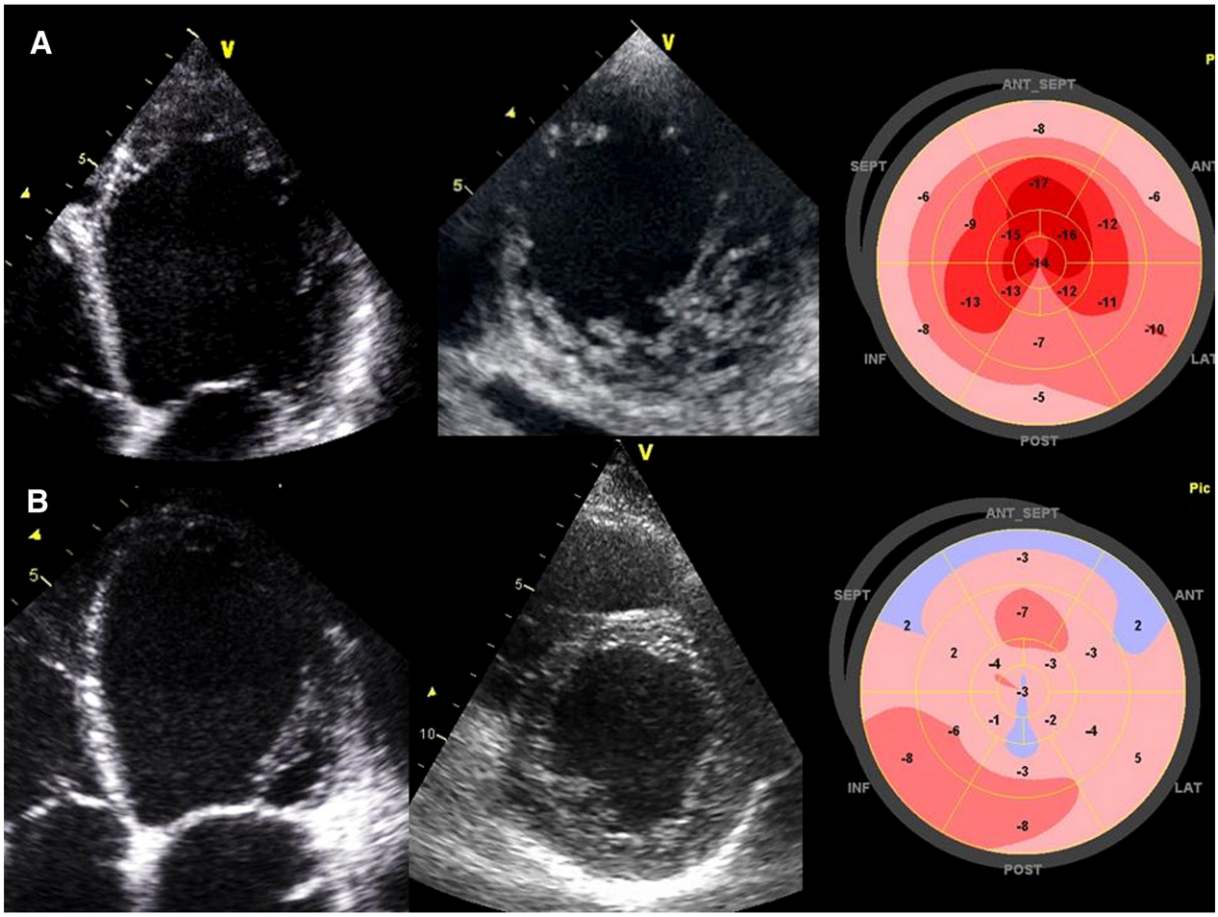

Fig. 1. 2D Myocardial deformation pattern of a dilated cardiomyopathy and a left ventricular noncompaction. 2D Transthoracic echocardiography of two patients with moderate LV systolic dysfunction (LVEF: 35%): Apical 4 chambers and parasternal short axis views and Bulls-eye of peak systolic strain values recorded from 3 apical chambers views. (A) Patient with confirmed LV non-compaction demonstrating trabeculations and deep sinusoids within non-compacted layer; higher regional peak longitudinal strain at the level of the apical non-compacted segment; lower values of systolic deformation in mid- and basal septal and inferior wall with apex/basal ratio > 1.5. (B) Dilated cardiomyopathy with overall segmental low peak longitudinal strain values.

Table 1
Comparison of conventional echocardiographic characteristics between LVNC and DCM groups.

	LVNC n = 50	DCM n = 50	p Value
Age	51.8 ± 15.1	51.9 ± 15.2	NS
Sex (male)	39 (76)	39 (76)	NS
LVEF (%)	33.7 ± 11.2	34.0 ± 10.9	NS
LVEDV (ml)	190.9 ± 73.0	173.7 ± 69.6	0.067
LVESV (ml)	128.4 ± 56.7	118.4 ± 58.8	NS
LVEDD (mm)	65.1 ± 11.4	63.9 ± 7.5	NS
LVM index ASE (g/m2)	154.9 ± 70.4	163.8 ± 82.8	NS
Cardiac output (l/min/m2)	2.6 ± 0.9	2.3 ± 0.8	NS
Diastolic function			
LA area (cm^2)	26.5 ± 8.2	23.5 ± 8.7	0.005
E/A	1.7 ± 1.2	1.5 ± 1.0	NS
E/E′	10.9 ± 6.7	10.9 ± 6.0	NS
RV Function			
RA area (cm^2)	17.6 ± 6.2	16.8 ± 6.2	NS
TR (mm Hg)	28.9 ± 10.8	27.0 ± 11.6	NS
TAPSE (mm)	21.4 ± 6.5	19.5 ± 4.5	NS
S′ (cm/s)	10.8 ± 2.8	10.6 ± 3	NS

LVNC = Left ventricular noncompaction; DCM = dilated cardiomyopathy; LS = longitudinal strain; LVEF = left ventricular ejection fraction; LVESV = left ventricular end-systolic volume; LVEDV = left ventricular end-diastolic volume; LVEDD left ventricular end-diastolic diameter; LVM = left ventricular mass; RV = right ventricle; TAPSE = tricuspid annular plane systolic excursion; TR = tricuspid regurgitation; LA = left atrium; RA = right atrium; S′ = peak systolic lateral right ventricle.

were excluded from the analysis because accurate measurement of NC or C layer was not possible on the SAX views.

2D TTE identified 511 (68.7%) compacted segments, 138 (16%) borderline segments and 77 (10.6%) NC segments. Localization of non-compacted myocardial segments is shown in Fig. 2. The borderline NC segments were more common in the apical and mid-lateral segments (11th, 12th 13th and 15th segments) than in basal segments. NC localization was the most frequent in the 15th and 16th segments in percentage (Fig. 2). The end-diastolic thickness of compacted myocardium of the segments appeared lower in NC segment (6.4 ± 1.4 mm) compared to borderline and C segments (7.5 ± 1.6 and 7.7 ± 1.8 mm; $p < 0.05$). Among the NC, C and borderline segments there was no

Table 2
Comparison of 2D deformation parameters between LVNC and DCM: (LS: longitudinal strain).

Parameters (%)	LVNC n = 50	DCM n = 50	p Value
Global LS	−10.6 ± 4.9	−10.1 ± 4.9	NS
2 chambers LS	−11.1 ± 4.9	−10.5 ± 4.9	NS
4 chambers LS	−10.3 ± 4.8	−9.9 ± 4.7	NS
3 chambers LS	−11.0 ± 5.1	−10.4 ± 4.9	NS
Basal LS	−9.3 ± 4.7	−9.9 ± 4.6	NS
Mid LS	−10.7 ± 5.1	−10.4 ± 4.9	NS
Apical LS	−12.8 ± 5.9	−10.7 ± 5.7	0.025
Apical/basal LS Ratio	1.5 ± 0.7	1.1 ± 0.4	0.001

LVNC = Left ventricular noncompaction; DCM = dilated cardiomyopathy; LS = longitudinal strain.

Table 3
Segmental analysis of 726 segments of the LVNC patients according to the degree of trabeculations (3 grades: non-compacted: NC/C ratio above 2; borderline: NC/C ratio between 0–2 and compacted–no trabeculations: NC/C ratio = 0).

Segments	Non-compacted	Borderline	Compacted
Ratio NC/C	>2	0–2	0
Number of segments n (%)	77 (10.6)	138 (19.0)	511 (70.4)
Mean NC/C thickness ratio	2.5 ± 0.5	1.3 ± 0.4*,†	0*
Compacta thickness (mm)	6.4 ± 1.4	7.5 ± 1.6*	7.7 ± 1.8*
Noncompacta thickness (mm)	16.1 ± 3.7	10.2 ± 3.6*,†	0*
Akinesia/dyskinesia n (%)	4 (5.2)	8 (5.8)†	71 (13.9)
Hypokinesia n (%)	54 (70.1)	95 (68.8)	317 (62.0)
Normokinesia n (%)	19 (24.7)	35 (25.4)	123 (24.1)
WMSI	1.8 ± 0.5	1.8 ± 0.5	1.9 ± 0.6
Segmental PLS value (%)	−13.1 ± 6.1	−11.3 ± 6.5	−10.2 ± 6.3*

NC = non-compacted; C = compacted; WMSI = Wall motion score index; PLS = Peak longitudinal strain.
* $p < 0.05$, Vs NC segments.
† $p < 0.05$ vs compacted segments.

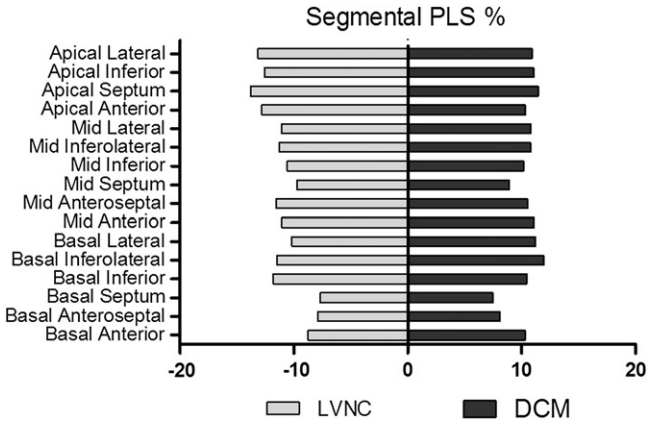

Fig. 3. Segmental deformation values in LVNC and DCM groups according to the 16-segment left-ventricular model (ACC/AHA) (PLS: Peak longitudinal strain values; %, LVNC = left ventricular non-compaction. DCM = dilated cardiomyopathy).

difference on the visual assessment of myocardial contraction using the WMS (Fig. 3).

Segmental PLS values were significantly higher in NC compared to C segments (−13.1 ± 6.1 vs −10.2 ± 6.3; $p < 0.05$). Regional longitudinal LV deformation was intermediate in the borderline segments group but not statically different (−11.1 ± 6.5%) (Fig. 4).

3.2.2. Reproducibility

The intra-observer and inter-observer limits of agreement for peak longitudinal strain measurements by speckle tracking echocardiography were 3.9 ± 3.7% and 2.6 ± 2.8% respectively.

4. Discussion

The mechanisms of myocardial dysfunction in LVNC are not clearly elucidated. Early reports and recent studies suggest that LVNC is associated with more severe LV dysfunction and a higher incidence of ventricular arrhythmias and thrombo-embolic complications than DCM. This suggests that LVNC cardiomyopathy is a disease that requires close surveillance and aggressive treatment. As the management may differ from other causes of cardiomyopathy, LVNC therefore needs early and reliable diagnosis [4,17]. We observed a specific LV systolic deformation pattern with relatively preserved strain values in NC segments despite an overall hypokinesia that allows the differentiation from DCM among patients with decreased LVEF.

4.1. Differentiation between LVNC and DCM

The morphological appearance of the myocardium in LVNC is characteristic and suggests a distinct cardiomyopathy. But pronounced trabeculation may be presented in both LVNC and DCM, which sometimes makes the differentiation difficult. In our experience, none of the standard LV systolic dysfunction parameters was able to discriminate a DCM from a LVNC. Previous studies tried to find useful echocardiographic discrepancies and demonstrated the same results [18]. According to Tufekcioglu et al., we found that most of the conventional echocardiography findings for the two conditions were statistically similar [19]. Also, they noted more dilated LV with more severe LV diastolic dysfunction in DCM patients. Unfortunately, this study was a small sample of patients who do not match with LVEF between the 2 groups. Other authors described a distinct form of spherical LV remodeling in LVNC compared to DCM [20].

Despite new advances in imaging modalities, clinicians' dream of perfect diagnostic tools for the identification of pathologic hypertrabeculation and predictive factors of LV dysfunction still did not come true . Accurate evaluation and measurement of LV volumes

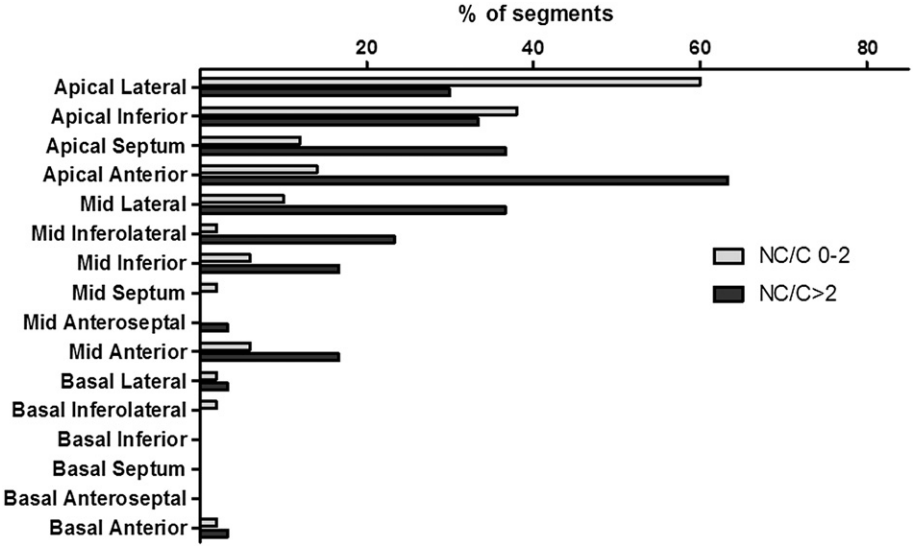

Fig. 2. Distribution of non-compaction according to the 16 segment left-ventricular model: The bars represent the location and the percentage of trabeculations (non-compacted; NC/C > 2 and borderline segments: NC/C 0–2) according to the 16-segment left-ventricular model (ACC/AHA); NC = Non-compacted; C = Compacted.

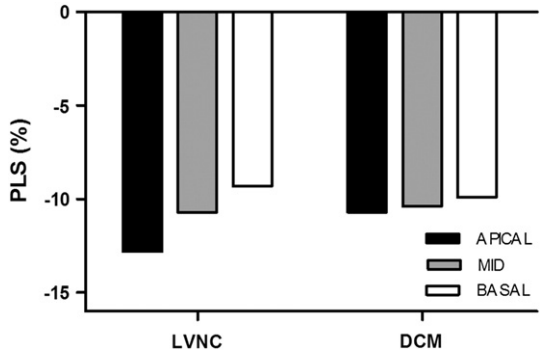

Fig. 4. Basal, median and apical strain values in LVNC and DCM groups. Mean PLS in LVNC and DCM groups according to basal, median and apical strain values higher apical LV PLS values compared to DCM patients (-12.8 ± 5.9 vs -10.7 ± 5.7 $p < 0.05$) (PLS: Peak longitudinal strain values; %); LVNC = left ventricular non-compaction. DCM = dilated cardiomyopathy.

and EF are sometimes difficult both by 2D or 3D echocardiography because of endocardial border delineation limitations [21]. Moreover the reproducibility of measurements to diagnose LVNC by accepted criteria is poor [22].

4.2. Mechanisms of myocardial dysfunction in NC segments

It was demonstrated that LVNC with preserved LVEF have subclinical myocardial dysfunction with impairment of deformation parameters [23]. LV solid body rotation/twist was also described as a potential quantitative functional diagnostic criterion for non-compaction [24]. In our study we observed a significant increase in longitudinal function from the base to the apex with more preserved longitudinal strain at the level of the apical NC segments. This specific regional deformation pattern may help to distinguish this population of cardiomyopathy from the classical idiopathic DCM. In the normal heart, we know that myocardial velocities decrease from the mitral annulus toward the LV apex. However, when the ventricle was subdivided into basal, midventricular, and apical regions the mean difference of strain values was only 1 percentage point and the difference was not significant [25–26]. However, contradictory results are reported in the literature regarding regional deformation distribution in LVNC. In a small study using tissue Doppler imaging for regional LV deformation assessment, Niemann et al. reported preserved deformation in basal segments of LVNC [18]. Our pattern of myocardial strain with the apex/basal ratio of 1.5 may be related to the shape of the LV and the usual distribution of NC segments. Sengupta et al. hypothesized that the spongy myocardial architecture of the LV apex likely resists to the dilatation and leads to the particular form of ventricular remodeling and causes asynergy in these areas [20]. In addition, a reduced thickness of the compacted layer in the NC area was noted as previously described. Previous studies defined a maximal compacted thickness <8 mm as a specific marker that allows the differentiation of pathological trabeculation of non-compaction [27]. Despite a thinning of the compacted layer of NC segments, the regional thickening and deformation seems to be relatively preserved in NC segments due to the myocardial deformation of the NC layer.

4.3. Relation between global systolic function and the degree of segmental NC

Visually studying wall motion is problematic in NC myocardium. Cardiac imaging with the use of 2D speckle tracking echocardiographic may help for tissue characterization and confirm etiology and localization of the WMA. Regional morphology, motion and deformation analyses differ in NC segments and explain this pattern. In our study we found similar WMS in the 3 categories of C, NC and borderline segments. It is admitted that the most trabeculated segments are not responsible for the overall LV systolic dysfunction. Global PLS low values are caused by impairment of myocardial contraction in the affected ventricular segments but also in the compacted myocardium. However the correlation between the number of NC segments with LVEF and LV end-diastolic volume remains controversial. Several papers suggested that the degree of NC and the extent of the NC myocardium is related to the severity of LV function [28–29] and are independent predictors of global LV dysfunction [30–31]. In contrast, a recent study published by Habib and Fazio et al. reported opposite findings, identifying no relationship between the extent of NC myocardium and LV systolic function [2,32]. In our study the extent (number of NC segments) and the severity (maximal NC layer thickness) of myocardial non-compaction was not correlated with the LVEF, LV end-diastolic volume index or the global PLS.

The first study evaluating myocardial function in LVNC used tissue Doppler imaging to estimate longitudinal wall velocity and concluded that longitudinal LV wall velocity is impaired in LVNC but not related to the extent or severity of NC [33]. We demonstrated significantly higher absolute segmental PLS values in NC as compared to C segments. Relative chronic myocardial ischemia caused by impaired microcirculation can lead to segmental LV dysfunction and can account for the histologically subendocardial fibrosis or ischemic lesions in prominent trabeculae [32,34]. Coronary angiography reveals usually no significant abnormalities but positron emission tomography shows a decreased reserve of coronary blood flow in the compacted and NC segments [35–36]. Myocardial fibrosis is the second hypothesis and is observed in half of the patients with isolated LVNC by Nucifora et al. with an average extent of 5% of the overall LV myocardium. Fibrosis involved both compacted and NC myocardium with a similar prevalence. In this study a significant association was observed between the degree of LV dysfunction and extent of LGE [37].

4.4. Study limitations

Paradoxical contributions of NC and C segments and better understanding of LV dysfunction mechanisms in LVNC should come from speckle tracking echocardiographic studies. Although due to the nature of the NC myocardium with a very unusual and nonspecific fiber orientation, the calculation for regional deformation may be difficult [38].

Even if we took particular attention to endocardial border tracings, some of the trabeculations are necessary in the AFI region of interest. As a consequence evidence of direct blood flow coming from the ventricular cavity into deep intertrabecular recesses may change the deformation values.

5. Conclusion

Left ventricular systolic dysfunction associated with heart failure is the main predictor of outcome and the most frequent initial presentation of patient with LVNC.

Speckle analysis of echocardiography improves information and may help to understand the mechanism of LV systolic dysfunction in LVNC. We observed a base–apex gradient with relatively preserved deformation values in the apical region. Despite an overall regional hypokinesia with alteration of deformation values we noted a significantly higher segmental PLS values in NC compared to C segments. Moreover the extent and severity of NC was not correlated with LV dysfunction.

The functional impact of an abnormal myocardial architecture induced change in deformation pattern that could help the clinician to distinguish between normally trabeculated myocardium of DCM from LVNC.

This study confirms that NC may be a part of a more generalized cardiomyopathy and helps to understand the pathophysiology of the LV dysfunction involving both the morphologically normal and abnormal segments.

Conflict of interest

There is no conflict of interest.

References

[1] Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, et al. Classification of the cardiomyopathies: a position statement from the European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J 2008 Jan; 29(2):270–6.

[2] Habib G, Charron P, Eicher J-C, Giorgi R, Donal E, Laperche T, et al. Isolated left ventricular non-compaction in adults: clinical and echocardiographic features in 105 patients. Results from a French registry. Eur J Heart Fail 2011 Feb;13(2):177–85.

[3] Murphy RT, Thaman R, Blanes JG, Ward D, Sevdalis E, Papra E, et al. Natural history and familial characteristics of isolated left ventricular non-compaction. Eur Heart J 2005 Jan;26(2):187–92.

[4] Carrilho-Ferreira P, Almeida AG, Pinto FJ. Non-compaction Cardiomyopathy: Prevalence, Prognosis, Pathoetiology, Genetics, and Risk of Cardioembolism. Curr Heart Fail Rep 2014 Dec 1;11(4):393–403.

[5] Jacquier A, Thuny F, Jop B, Giorgi R, Cohen F, Gaubert J-Y, et al. Measurement of trabeculated left ventricular mass using cardiac magnetic resonance imaging in the diagnosis of left ventricular non-compaction. Eur Heart J 2010 May;31(9): 1098–104.

[6] Chin TK, Perloff JK, Williams RG, Jue K, Mohrmann R. Isolated noncompaction of left ventricular myocardium. A study of eight cases. Circulation 1990 Aug;82(2):507–13.

[7] Jenni R, Oechslin E, Schneider J, Jost CA, Kaufmann PA. Echocardiographic and pathoanatomical characteristics of isolated left ventricular non-compaction: a step towards classification as a distinct cardiomyopathy. Heart 2001 Dec 1;86(6): 666–71.

[8] Stöllberger C, Finsterer J. Left ventricular hypertrabeculation/noncompaction. J Am Soc Echocardiogr 2004 Jan;17(1):91–100.

[9] Paterick TE, Tajik AJ. Left ventricular noncompaction: a diagnostically challenging cardiomyopathy. Circ J 2012;76(7):1556–62.

[10] Meluzin J, Spinarova L, Hude P, Krejci J, Poloczkova H, Podrouzkova H, et al. Left ventricular mechanics in idiopathic dilated cardiomyopathy: systolic–diastolic coupling and torsion. J Am Soc Echocardiogr 2009 May;22(5):486–93.

[11] Mistry N, Beitnes JO, Halvorsen S, Abdelnoor M, Hoffmann P, Kjeldsen SE, et al. Assessment of left ventricular function in ST-elevation myocardial infarction by global longitudinal strain: a comparison with ejection fraction, infarct size, and wall motion score index measured by non-invasive imaging modalities. Eur J Echocardiogr 2011 Sep;12(9):678–83.

[12] Zeng S, Zhou Q, Peng Q, Cao D, Tian L, Ao K, et al. Assessment of regional myocardial function in patients with dilated cardiomyopathy by velocity vector imaging. Echocardiography 2009 Feb;26(2):163–70.

[13] Kalam K, Otahal P, Marwick TH. Prognostic implications of global LV dysfunction: a systematic review and meta-analysis of global longitudinal strain and ejection fraction. Heart 2014 May;23.

[14] Nahum J, Bensaid A, Dussault C, Macron L, Clémence D, Bouhemad B, et al. Impact of longitudinal myocardial deformation on the prognosis of chronic heart failure patients. Circ Cardiovasc Imaging 2010 May;3(3):249–56.

[15] Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005 Dec;18(12):1440–63.

[16] Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Int J Cardiovasc Imaging 2002 Feb;18(1):539–42.

[17] Stanton C, Bruce C, Connolly H, Brady P, Syed I, Hodge D, et al. Isolated left ventricular noncompaction syndrome. Am J Cardiol 2009 Oct 15;104(8):1135–8.

[18] Niemann M, Liu D, Hu K, Cikes M, Beer M, Herrmann S, et al. Echocardiographic quantification of regional deformation helps to distinguish isolated left ventricular non-compaction from dilated cardiomyopathy. Eur J Heart Fail 2012 Feb;14(2): 155–61.

[19] Tufekcioglu O, Aras D, Ozeke O, Maden O, Topaloglu S. Comparison of regional systolic myocardial velocities in patients with isolated left ventricular noncompaction and patients with idiopathic dilated cardiomyopathy. J Am Soc Echocardiogr 2006 Nov;19(11):1320–5.

[20] Sengupta PP, Mohan JC, Mehta V, Jain V, Arora R, Pandian NG, et al. Comparison of echocardiographic features of noncompaction of the left ventricle in adults versus idiopathic dilated cardiomyopathy in adults. Am J Cardiol 2004 Aug 1;94(3):389–91.

[21] Nemes A, Forster T. Three-dimensional echocardiography for the evaluation of left ventricular noncompaction. J Am Soc Echocardiogr 2012 Jul;25(7):805–6.

[22] Saleeb SF, Margossian R, Spencer CT, Alexander ME, Smoot LB, Dorfman AL, et al. Reproducibility of echocardiographic diagnosis of left ventricular noncompaction. J Am Soc Echocardiogr 2012 Feb;25(2):194–202.

[23] Tufekcioglu O, Aras D, Yildiz A, Topaloglu S, Maden O. Myocardial contraction properties along the long and short axes of the left ventricle in isolated left ventricular non-compaction: pulsed tissue Doppler echocardiography. Eur J Echocardiogr 2008 May;9(3):344–50.

[24] Vandalen B, Caliskan K, Soliman O, Nemes A, Vletter W, Tencate F, et al. Left ventricular solid body rotation in non-compaction cardiomyopathy: a potential new objective and quantitative functional diagnostic criterion? Eur J Heart Fail 2008 Nov; 10(11):1088–93.

[25] Dalen H, Thorstensen A, Aase SA, Ingul CB, Torp H, Vatten LJ, et al. Segmental and global longitudinal strain and strain rate based on echocardiography of 1266 healthy individuals: the HUNT study in Norway. Eur J Echocardiogr 2010 Mar; 11(2):176–83.

[26] Leitman M, Lysyansky P, Sidenko S, Shir V, Peleg E, Binenbaum M, et al. Two-dimensional strain — a novel software for real-time quantitative echocardiographic assessment of myocardial function. J Am Soc Echocardiogr 2004 Oct; 17(10):1021–9.

[27] Gebhard C, Stähli BE, Greutmann M, Biaggi P, Jenni R, Tanner FC. Reduced left ventricular compacta thickness: a novel echocardiographic criterion for non-compaction cardiomyopathy. J Am Soc Echocardiogr 2012 Oct;25(10):1050–7.

[28] Aras D, Tufekcioglu O, Ergun K, Ozeke O, Yildiz A, Topaloglu S, et al. Clinical features of isolated ventricular noncompaction in adults long-term clinical course, echocardiographic properties, and predictors of left ventricular failure. J Card Fail 2006 Dec;12(9):726–33.

[29] Belanger AR, Miller MA, Donthireddi UR, Najovits AJ, Goldman ME. New classification scheme of left ventricular noncompaction and correlation with ventricular performance. Am J Cardiol 2008 Jul 1;102(1):92–6.

[30] Dellegrottaglie S, Pedrotti P, Roghi A, Pedretti S, Chiariello M, Perrone-Filardi P. Regional and global ventricular systolic function in isolated ventricular non-compaction: pathophysiological insights from magnetic resonance imaging. Int J Cardiol 2012 Jul 26;158(3):394–9.

[31] Lofiego C, Biagini E, Ferlito M, Pasquale F, Rocchi G, Perugini E, et al. Paradoxical contributions of non-compacted and compacted segments to global left ventricular dysfunction in isolated left ventricular noncompaction. Am J Cardiol 2006 Mar 1;97(5): 738–41.

[32] Fazio G, Novo G, Casalicchio C, Di Gesaro G, Sutera L, Grassedonio E, et al. Left ventricular non-compaction cardiomyopathy in children: is segmental fibrosis the cause of tissue Doppler alterations and of EF reduction? Int J Cardiol 2009 Feb 20; 132(2):278–80.

[33] Caliskan K, Soliman OI, Nemes A, van Domburg RT, Simoons ML, Geleijnse ML. No relationship between left ventricular radial wall motion and longitudinal velocity and the extent and severity of noncompaction cardiomyopathy. Cardiovasc Ultrasound 2012 Mar 19;10:9.

[34] Ritter M, Oechslin E, Sütsch G, Attenhofer C, Schneider J, Jenni R. Isolated noncompaction of the myocardium in adults. Mayo Clin Proc 1997 Jan;72(1):26–31.

[35] Junga G, Kneifel S, Von Smekal A, Steinert H, Bauersfeld U. Myocardial ischaemia in children with isolated ventricular non-compaction. Eur Heart J 1999 Jun;20(12): 910–6.

[36] Sato Y, Matsumoto N, Matsuo S, Kunimasa T, Yoda S, Tani S, et al. Myocardial perfusion abnormality and necrosis in a patient with isolated noncompaction of the ventricular myocardium: evaluation by myocardial perfusion SPECT and magnetic resonance imaging. Int J Cardiol 2007 Aug 21;120(2):e24–6.

[37] Nucifora G, Aquaro GD, Pingitore A, Masci PG, Lombardi M. Myocardial fibrosis in isolated left ventricular non-compaction and its relation to disease severity. Eur J Heart Fail 2011 Feb;13(2):170–6.

[38] Perk G, Tunick PA, Kronzon I. Non-Doppler two-dimensional strain imaging by echocardiography — from technical considerations to clinical applications. J Am Soc Echocardiogr 2007 Mar;20(3):234–43.