Original Article

Prevalence of Left Ventricular Non-Compaction and Factors Associated with Left Ventricular Systolic Dysfunction in Patients Who Underwent Cardiac Magnetic Resonance Imaging

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Background: Left ventricular non-compaction [LVNC] cardiomyopathy is a cause of left ventricular systolic dysfunction [LVSD].

Objective: To investigate the prevalence of LVNC, and the factors associated with LVSD in patients who underwent cardiac magnetic resonance [CMR] imaging.

Materials and Methods: This retrospective chart and imaging review included consecutive adult patients who underwent CMR at Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand between January and March 2012. Non-compact layer [NC] and compact layer [C] diameters were measured in each of three long-axis views. The maximum value of NC/C was used. LVNC was diagnosed if the NC/C value was greater than 2.3. Univariate and multivariate analysis were performed to identify factors associated with LVSD, which was defined as left ventricular ejection fraction of less than 50%.

Results: Four hundred thirteen patients were included. The mean age of patients was 66.12±14.43 years, and 241 were male. The prevalence of LVNC was 3.6% (95% CI 2.2% to 5.9%). NC/C ratio in the LVNC and non-LVNC group was 3.34±0.84 and 1.56±0.47, respectively. Multivariate analysis revealed the five factors that significantly associated with LVSD (all $p<0.05$), which were male, body mass index, heart rate, hypertension, and LVNC.

Conclusion: Prevalence of LVNC was 3.63%. Factors significantly associated with LVSD were male gender, BMI, heart rate, hypertension, and maximum NC/C greater than 2.3.

Keywords: Thailand, Prevalence, Left ventricular non-compaction, Left ventricular systolic dysfunction, Cardiac magnetic resonance imaging

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Non-compaction cardiomyopathy, also known as isolated left ventricular non-compaction [LVNC], is a congenital heart disease that is caused by the inability of the intertrabecular recesses to compress into compact myocardium during organogenesis during the first trimester of pregnancy[1,2]. The morphologic reason of this tissue compaction failure is tissue consisting of a sponge-like meshwork that is located predominantly in the left ventricular [LV] cavity. According to one systematic review, this condition was associated with sudden cardiac death (7.6%), congestive heart failure (38%), and thromboembolism (8%), with overall mortality of 14% during a mean follow-up period of 39 months[3]. Previous transthoracic echocardiography [TTE] studies using echocardiographic criteria to evaluate for this condition[4] reported prevalence rates of 3.7%[5] and 6.1%[6]. However, these figures may represent an underestimation of the condition due to the inherent problem of near-field clutter that can obstruct or distort the visualization of structures close to the echocardiographic transducer, which makes diagnosis of LVNC by TTE difficult. Cardiac magnetic resonance [CMR] imaging produces an image with better spatial resolution than images produced by TTE[7,8]. Accordingly, diagnosis of LVNC by CMR is more sensitive and specific than LVNC diagnosis by TTE. Another benefit of CMR is the use of late gadolinium enhancement [LGE], which has proved useful in the diagnosis and prognostication of many conditions, and it may also have diagnostic and prognostic value in LVNC[9]. The prevalence of LVNC has not been studied in Thailand using CMR criteria[10].

Accordingly, the aim of the present study was to investigate the prevalence of LVNC in patients who underwent CMR, and to identify factors associated with

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left ventricular systolic dysfunction [LVSD], which was defined as LVEF of less than 50%.

Materials and Methods

Study population

This retrospective chart and imaging review included consecutive adult patients (older than 18 years) who underwent CMR at the Division of Cardiology, Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand between January and March 2012. Siriraj Hospital is Thailand's largest university-based national tertiary referral center. Subjects with complex congenital heart disease were excluded. The protocol for the present study was approved by the Siriraj Institutional Review Board [SIRB].

CMR imaging protocol

CMR was performed for assessment of cardiac function, myocardial perfusion, and LGE using a 1.5 Tesla Gyroscan NT Philips scanner (Philip Medical Systems, Best, the Netherlands). Functional study was performed by acquisition of images using the steady-state free-precession technique in vertical long axis, 4-chamber, and multiple slice short-axis views. Parameters for cardiac function were, as follows: repetition time/echo time/number of excitations 3.7 ms/1.8 ms/2,390×312 mm field of view, 256×240 matrix, 1.52×1.21 reconstruction pixel, 8 mm slice thickness, and 70° flip angle.

Three short-axis slices at the apical, mid, and basal LV level were selected for perfusion imaging with an electrocardiograph [ECG]-triggered, T1-weighted, inversion recovery single-shot turbo gradient echo sequence (prepulse delay 360 ms, acquisition duration 360 ms, flip angle 15°, echo time 1.7 ms, repetition time 9 ms), with a slice thickness of 8 mm and a spatial resolution of 1.7 to 1.9 mm. First-pass perfusion study was performed via administration of 0.05 mmol/kg gadolinium contrast agent (Magnevist, Bayer Schering Pharma, Berlin, Germany) immediately following a 4-minute infusion of 0.56 mg/kg adenosine. During adenosine infusion, blood pressure, and oxygen saturation (by pulse oximetry) was measured once per minute, with continuous monitoring of ECG rhythm. The adenosine infusion was discontinued prematurely upon patient request or when progressive/severe angina, dyspnea, decrease in systolic pressure of 40 mmHg or more, severe arrhythmias, or other adverse effects occurred. Another injection of 0.1 mmol/kg gadolinium was administered immediately after the acquisition of perfusion images. LGE images were acquired approximately 10 minutes after this injection by segmented 3D gradient echo and phase sensitive inversion recovery sequence. LGE images were acquired in multiple short-axis slices at the level similar to the functional images, long-axis and 4-chamber view. Parameters for LGE study were, as follows: echo time 1.25 ms, repetition time 4.1 ms, 15° flip angle, 303×384 mm field of view, 240×256 matrix, in-plane resolution 1.26×1.5 mm, slice thickness 8 mm, and 1.5 Sensitivity-Encoding [SENSE] factor.

CMR imaging analysis

CMR imaging analysis was performed on a "ViewForum" workstation (Philips Medical Systems, Best, the Netherlands). All analyses were performed by one reviewer (Khlangpremchit S).

Assessment of non-compaction and compaction dimension was performed on three long-axis views in cine-CMR images (i.e., 2-chamber view, 3-chamber view, and 4-chamber view). In each view, the reviewer first identified the essential related cardiac anatomy to facilitate measurement, which included the a) non-compact layer, b) compact layer, and c) papillary muscles. The papillary muscles were not measured. The apex (segment 17) was also excluded from measurement. The reviewer then measured the non-compact and compacted layer of the myocardium in the end-diastolic frame. The segment in which the non-compaction layer was most prominent was used as a reference point for measurement of both the non-compaction and compaction layer. The ratio of non-compact layer to compact layer [NC/C ratio] was obtained in each view, three times in each patient, with the highest of the three values included in our analysis. LVNC was diagnosed if the highest NC/C ratio exceeded 2.3[10]. If a non-compaction layer could not be identified, the NC/C ratio was recorded as 0.

Statistical analysis

All data analyses were performed using SPSS Statistics version 22 (SPSS Inc., Chicago, IL, USA). Patient characteristics are summarized using descriptive statistics. Prevalence data are presented as percentage. Categorical data are presented as frequency and percentage, and continuous data are shown as mean ± standard deviation. Comparison of categorical data was performed using Chi-square test or Fisher's exact test. Student's t-test was used to compare continuous data. Evaluation of factors associated with LVSD (from demographic, cardiovascular risk factors, and

CMR variables), defined as LVEF of less than 50%, was performed using multiple logistic regression analysis. Factors with a *p*-value of less than 0.05 in univariate analysis were entered into the logistic regression analysis. The results of multivariate analysis are presented as odds ratio [OR] and 95% confidence interval [CI], and adjusted OR [AOR] and 95% CI. Missing data were excluded from analysis. A two-tailed *p*-value of less than 0.05 was considered statistically significant for all tests.

Results

Four hundred thirteen patients were included. The mean age of patients was 66.12±14.43 years, and 241 (58.35%) were male. There were 15 patients with LVNC for a prevalence of 3.63% (95% CI 2.2% to 5.9%). Forty-eight patients (14.8%) had history of heart failure. Maximum NC/C ratio in the LVNC and non-LVNC group was 3.34±0.84 and 1.56±0.47, respectively. Baseline clinical characteristics of the overall study population and by LVNC group are shown in Table 1. Baseline CMR data are given in Table 2. CMR images of patients with and without LVNC are shown in Figure 1 and 2, respectively.

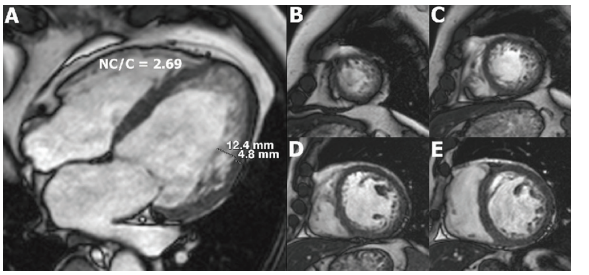

Figure 1. Cardiac magnetic resonance [CMR] images of left ventricular non-compaction [LVNC] showing a non-compaction to compaction [NC/C] ratio of 2.69 with prominent trabeculation in 4-chamber view (A) and short-axis slices (B-E).

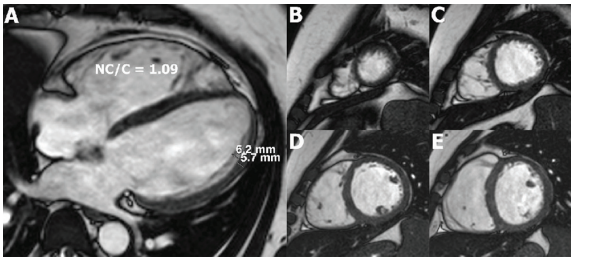

Figure 2. Cardiac magnetic resonance [CMR] images showing no presence of left ventricular non-compaction [LVNC], with a non-compaction to compaction [NC/C] ratio of 1.09 (A) and short-axis slices (B-E).

LVSD

The following factors were statistically significant

Table 1. Baseline clinical characteristics of the overall study population and by LVNC group

Clinical variables	Overall population (n = 413)	LVNC (n = 15)	No LVNC (n = 398)
Age (years)	66.12±14.43	56.55±18.93	66.48±14.14
Male gender	241 (58.35)	7 (46.7)	234 (58.8)
Body mass index (kg/m^2)	25.17±4.34	22.85±4.21	25.26±4.33
SBP (mm Hg)	133.66±21.40	115.91±19.07	134.27±21.23
DBP (mm Hg)	67.03±12.52	59.64±11.13	67.28±12.51
Heart rate (bpm)	78.40±15.14	82.18±14.97	78.27±15.15
Diabetes mellitus	126 (37.0)	5 (41.7)	121 (36.8)
Hypertension	261 (76.5)	6 (50.0)	255 (77.5)
Dyslipidemia	246 (72.4)	5 (41.7)	241 (73.5)
Chronic kidney disease	54 (15.9)	1 (8.3)	53 (16.2)
Stroke/transient ischemic attack	20 (5.9)	2 (16.7)	18 (5.5)
Heart failure	48 (14.8)	5 (41.7)	43 (13.8)
Syncope	2 (0.6)	0 (0.0)	2 (0.6)
Diagnosis			
Coronary artery disease	181 (43.8)	5 (33.3)	176 (44.2)
Dilated cardiomyopathy	32 (7.7)	4 (26.7)	28 (7.0)
Hypertrophic cardiomyopathy	8 (1.9)	0 (0.0)	8 (2.0)
Congenital heart disease	10 (2.4)	1 (6.7)	9 (2.3)
Valvular heart disease	8 (1.9)	0 (0.0)	8 (2.0)
Thalassemia	20 (4.8)	2 (13.3)	18 (4.5)

Data presented as number and percentage or mean ± standard deviation

LVNC = left ventricular non-compaction; SBP = systolic blood pressure; DBP = diastolic blood pressure; bpm = beats per minute

Table 2. Baseline CMR characteristics of the overall study population and by LVNC group

CMR variables	Overall population (n = 413), mean ± SD	LVNC (n = 15), mean ± SD	No LVNC (n = 398), mean ± SD
Maximum NC/C ratio	1.63±0.59	3.34±0.84	1.56±0.47
LVDd (2)	57.08±9.23	66.57±13.19	56.72±8.88
LVDs (2)	39.67±14.6	53.20±18.66	39.16±14.20
LVEF (%)	58.41±18.71	40.95±22.34	6
LVEDV (2)	156.14±69.54	226.38±124.58	153.49±65.40
LVESV (2)	74.82±69.99	148.21±112.68	72.05±66.52
LVSV (2)	81.33±22.12	78.17±38.80	81.45±21.31
LV mass (g)	94.11±36.22	105.74±51.26	93.67±35.54
LVEDV index (ml/m)	93.72±41.19	136.95±62.76	92.09±39.36
LVESV index (ml/m)	45.12±42.41	89.13±61.30	43.46±40.72
LVSV index (ml/m)	48.61±11.82	47.81±21.43	48.64±11.35
LV mass index (g/m)	56.14±20.47	64.14±25.76	55.84±20.22

CMR = cardiac magnetic resonance; LVNC = left ventricular non-compaction; NC/C = non-compaction to compaction ratio; LVDd = left ventricular diastolic dimension; LVDs = left ventricular systolic dimension; LVEF = left ventricular ejection fraction; LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; LVSV = left ventricular stoke volume; LV = left ventricular

in univariate analysis: male gender, body mass index [BMI], systolic blood pressure [SBP], heart rate, hypertension, heart failure, maximum NC/C exceeding 2.3, left ventricular diastolic dimension [LVDd], left ventricular systolic dimension [LVDs], left ventricular ejection fraction [LVEF], left ventricular end-diastolic volume [LVEDV], left ventricular end-systolic volume [LVESV], left ventricular stoke volume [LVSV], LV mass, LVEDV index, LVESV index, LVSV index, LV mass index (all $p<0.05$) (Table 3). Of those, male gender (AOR 1.85, 95% CI 1.06 to 3.24; $p = 0.03$), BMI (AOR 0.926, 95% CI 0.87 to 0.99; $p = 0.023$), heart rate (AOR 1.04, 95% CI 1.02 to 1.06; $p<0.001$), hypertension (AOR 2.674, 95% CI 1.296 to 5.52; $p = 0.008$), and maximum NC/C exceeding 2.3 (AOR 3.821, 95% CI 1.05 to 13.95; $p = 0.042$) remained statistically significant in multivariate analysis (Table 4).

Table 3. Univariate analysis for factors significantly associated with LVSD

Variables	LVSD (n = 108)	No LVSD (n = 305)	*p*-value
Age (years)	66.06±12.09	66.14±15.19	0.962
Male gender	72 (66.7)	169 (55.4)	0.041*
Body mass index (kg/m^2)	24.45±3.90	25.43±4.46	0.043*
SBP (mm Hg)	127.76±21.18	135.85±21.10	0.002*
DBP (mm Hg)	68.97±13.16	66.31±12.23	0.087
Heart rate (bpm)	84.06±15.59	76.31±14.45	<0.001*
Diabetes mellitus	38 (41.3)	88 (35.3)	0.310
Hypertension$^+$	78 (84.8)	183 (73.5)	0.029*
Dyslipidemia	70 (76.1)	176 (71.0)	0.348
Chronic kidney disease	19 (20.7)	35 (14.2)	0.147
Stroke/transient ischemic attack	9 (9.8)	11 (4.5)	0.064
Heart failure	31 (36.0)	17 (7.1)	<0.001*
Syncope	1 (1.1)	1 (0.4)	0.467
CMR variables			
Maximum NC/C >2.3	9 (8.3)	6 (2.0)	0.005*
LVDd	67.72±9.65	53.31±5.31	<0.001*
LVDs	59.05±11.38	32.80±7.90	<0.001*
LVEF	31.04±11.00	68.11±8.45	<0.001*
LVEDV	236.53±84.19	127.68±30.82	<0.001*
LVESV	168.26±77.71	41.73±17.71	<0.001*
LVSV	68.28±23.96	85.95±19.46	<0.001*
LV mass	119.62±39.19	85.07±30.40	<0.001*
LVEDV index	143.14±49.27	76.23±16.46	<0.001*
LVESV index	102.07±46.76	24.95±10.40	<0.001*
LVSV index	41.073±13.40	51.27±9.95	<0.001*
LV mass index	72.19±22.46	50.46±16.31	<0.001*

Data presented as number and percentage or mean ± standard deviation

LVSD = left ventricular systolic dysfunction; LV = left ventricular; SBP = systolic blood pressure; DBP = diastolic blood pressure; bpm = beats per minute; CMR = cardiac magnetic resonance; NC/C = non-compacted to compacted ratio; LVDd = left ventricular diastolic dimension; LVDs = left ventricular systolic dimension; LVEF = left ventricular ejection fraction; LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; LVSV = left ventricular stoke volume

* A *p*-value <0.05 indicates statistical significance, $^+$ Hypertension: available data = 341

Discussion

This is the first study to report the prevalence of LVNC in Thailand using CMR criteria. The 3.6% prevalence that the authors report here is consistent with the 3.7% and 6.1% rates reported in other previous studies. The authors also found a strong correlation between LVNC and LVSD. The authors findings suggest that LVNC may be more prevalent than previously thought. Accordingly, it is recommended that LVNC be investigated in cases of LVSD with unclear etiology.

The strength of the present study is that the authors used CMR to diagnose LVNC, which differed from previous studies that used TTE to diagnose

Table 4. Multivariate analysis for factors significantly associated with LVSD

Factors	Crude OR (95% CI)	*p*-value	Adjusted OR (95% CI)	*p*-value
Male gender	1.61 (1.02 to 2.55)	0.04	1.85 (1.06 to 3.24)	0.03*
Body mass index (kg/m^2)	0.95 (0.90 to 1.00)	0.044	0.926 (0.87 to 0.99)	0.023*
Heart rate (bpm)	1.04 (1.02 to 1.05)	<0.001	1.04 (1.02 to 1.06)	<0.001*
Hypertension	2.01 (1.07 to 3.79)	0.02	2.674 (1.296 to 5.52)	0.008*
Maximum NC/C >2.3	4.53 (1.57 to 13.05)	0.005	3.821 (1.05 to 13.95)	0.042*

LVSD = left ventricular systolic dysfunction; OR = odds ratio; CI = confidence interval; bpm = beats per minute; NC/C = non-compacted to compacted ratio

* A *p*-value<0.05 indicates statistical significance

LVNC. Despite the lower temporal resolution of CMR compared to that of TTE, CMR has higher spatial resolution, which results in better tissue clarification and border identification. Good border identification is essential for non-compaction and compaction layer discrimination, as shown in Figure 2. The echocardiographic criteria proposed by Jenni et al suggested a ratio of non-compaction to compaction of 2[11]. In contrast, Petersen et al proposed a ratio of 2.3 or higher to diagnose LVNC. Some experts have suggested the use of trabeculated LV mass for LVNC diagnosis[12,13]. No consensus agreement has been reached regarding the most accurate criteria for diagnosing this condition. Based on our experience, the Petersen et al criteria is the most suitable and easy to measure[10].

The prevalence of LVNC in the present study was 3.6%, which should not reflect the prevalence of LVNC the general population since the authors selected only patients who were referred for CMR. In contrast to the CMR imaging criteria used in our study, a previous study reported a 0.1% prevalence of LVNC using echocardiographic criteria[4]. CMR has a better image resolution, which makes it a better method for detecting LVNC than echocardiography[8]. Although echocardiogram criteria use an NC/C ratio of 2:1[11] and CMR criteria use an NC/C ratio of 2.3:1[10] for diagnosis of LVNC, the prevalence of LVNC in our study was greater than the prevalence reported in the study that used echocardiographic criteria. Our study also showed that the presence of LVNC is an independent predictor of LVSD.

The present study has some mentionable limitations. First and consistent with the retrospective nature of the present study, some patient data may have been missing or incomplete. Second, the size of the study population in the LVNC group was relatively small. As a result, our study may have lacked sufficient power to identify all significant differences and associations. Third, the patients enrolled in the present study were from a single center. Fourth, our center is Thailand's largest tertiary referral hospital, which means that the authors are often referred patients with complicated and intransigent conditions. As such, it is possible that our findings may not be generalizable to patients with the same condition in other settings. Finally, CMR images were evaluated by only one reviewer, so there was no interobserver agreement to strengthen the validity of the measurements. However, the non-compaction and compaction layers of the myocardium are clearly visible by CMR, so there is a low likelihood that erroneous measurements would be made. Importantly, the strength of the present study is that this data reflects diagnostic imaging results obtained in a real-world clinical setting.

Conclusion

This is the first study in Thailand to evaluate the prevalence of LVNC using CMR. The prevalence of LVNC was 3.63%. Factors found to be significantly associated with LVSD were male gender, BMI, heart rate, hypertension, and maximum NC/C exceeding 2.3.

What is already known on this topic?

LVNC is a cause of cardiomyopathy and LVSD. The yield of CMR is greater than that of echocardiogram for diagnosis of LVNC.

What this study adds?

The prevalence of LVNC was 3.63% in patients who were referred for CMR. Male gender, BMI, heart rate, hypertension, and maximum NC/C exceeding 2.3 were factors found to be significantly associated with LVSD.

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Potential conflicts of interest

The authors declare no conflict of interest.

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