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Pediatric Left Ventricular Non-compaction: A Single-center Experience from Anatolia

Taner Kasar[1], Emine Yurdakul Ertürk[2], Yeliz Kaşko Arıcı[3]

[1]Ordu University Faculty of Medicine, Department of Pediatric Cardiology, Ordu, Türkiye
[2]Ordu University Faculty of Medicine, Department of Pediatrics, Ordu, Türkiye
[3]Ordu University Faculty of Medicine, Department of Biostatistics and Medical Informatics, Ordu, Türkiye

What is known on this subject?

Left ventricular non-compaction (LVNC) is a rare but clinically important cardiomyopathy characterized by the coexistence of compact and noncompact layers, deep intertrabecular recesses, and prominent trabeculations. The phenotypic diversity of LVNC in childhood is remarkable. Dilated, hypertrophic, restrictive forms associated with congenital heart diseases, and isolated LVNC forms have been described, and these phenotypes can occur alone or in combination.

What this study adds?

This pediatric series shows that the isolated form is relatively common in LVNC, but clinical risk varies by phenotype. Arrhythmias are common, and the dilated and mixed-restrictive forms have a poorer prognosis.

ABSTRACT

Objective: To describe the phenotypic diversity, arrhythmia burden, and short-to-medium term clinical outcomes in pediatric left ventricular non-compaction (LVNC) cases, in light of a single-center experience from Anatolia.

Material and Methods: Sixteen children diagnosed with LVNC and followed at the Pediatric Cardiology Department of Ordu University Training and Research Hospital between December 2017 and July 2025 were retrospectively evaluated. Demographic characteristics, presenting symptoms, transthoracic echocardiography (TTE) and cardiac magnetic resonance (CMR) findings, 12-lead electrocardiogram and 24-hour Holter results, and treatments were recorded. The diagnosis was made based on TTE criteria and confirmed by CMR in all cases. Genetic analysis results were recorded for the cases.

Results: The median age at diagnosis was 7 years (range: 15 days-16 years), and 68.8% of the patients were male; the median follow-up was 33 months (range: 6-82). At presentation, 62.5% of cases were asymptomatic, whereas heart failure was observed in 37.5% of cases. The phenotypic distribution was predominantly isolated LVNC (n=8, 50%); among the non-isolated forms, the dilated type (n=5) was most common, while hypertrophic (n=1), restrictive (n=1), and mixed types (n=1) were also observed. The overall arrhythmia rate was 37.5% (n=6): ventricular tachycardia (VT) was observed in two cases, Wolff-Parkinson-White associated supra-VT was observed in two cases, and frequent ventricular extrasystoles were observed in two cases. Two cases required intervention: sympathetic denervation in one case (diagnosed with catecholamine-sensitive polymorphic VT) and implantation of an implantable cardioverter-defibrillator in the other. Three patients (18.8%) died during follow-up, and the fatalities were concentrated among the dilated and mixed-restrictive subtypes. Pathogenic variants were detected in two of the three cases that underwent genetic testing: PLEKHM2 in two cases and RYR2 in one. The RYR2 carrier had catecholaminergic polymorphic VT. Acetylsalicylic acid and beta-blockers formed the basis of medical management; antiarrhythmics and advanced interventions were applied in selected cases.

Corresponding Author: Prof. Assoc. Emine Yurdakul Ertürk, Ordu University Faculty of Medicine, Department of Pediatrics, Ordu, Türkiye
E-mail: eyurdakul52@hotmail.com **ORCID ID:** orcid.org/0000-0002-6741-3323

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ABSTRACT

Conclusion: This single-center pediatric series shows that the isolated form is relatively dominant, but clinical risk changes depending on the phenotype. Arrhythmias are common, and dilated and mixed-restrictive forms have a less favorable course. Confirming the TTE-based diagnosis with CMR and closely monitoring pediatric cases based on phenotype-rhythm findings may facilitate accurate classification and risk management.

Keywords: Pediatrics, left ventricular non-compaction, cardiomyopathy, arrhythmia, heart failure

Introduction

Left ventricular non-compaction (LVNC) is a rare but clinically important cardiomyopathy characterized by the coexistence of compact and non-compact layers, deep intertrabecular recesses, and prominent trabeculation (1,2). The American Heart Association classifies LVNC as a genetic cardiomyopathy, attributing it to an arrest of myocardial development during embryogenesis (3). In contrast, the European Society of Cardiology still categorizes it as an "unclassified cardiomyopathy" (4). However, recent studies have shown that acquired mechanisms, such as a physiological increase in trabeculation observed in athletes or pregnant women, can also produce phenotypes that mimic LVNC (5).

The phenotypic diversity of LVNC in childhood is remarkable. Dilated, hypertrophic, and restrictive forms (often associated with congenital heart disease), as well as isolated LVNC forms, have been described; these phenotypes can occur alone or in combination (6,7). While isolated LVNC generally has a better prognosis (8), LVNC accompanied by a dilated phenotype (LVNC-D) is associated with poor short-term outcomes (9). This indicates that the risks of mortality and heart transplantation are particularly high among pediatric patients.

The clinical presentation of LVNC is highly heterogeneous, ranging from asymptomatic cases to heart failure, arrhythmias, and thromboembolic events, which constitute the most common manifestations (3). Although transthoracic echocardiography (TTE) is the most commonly used diagnostic tool (1), cardiac magnetic resonance (CMR) imaging also improves diagnostic sensitivity and provides prognostic information, such as late gadolinium enhancement (10).

With respect to etiopathogenesis, LVNC is associated with a genetic basis. It has been associated with more than 100 genes to date, with sarcomere gene mutations (MYH7, TTN, MYBPC3) being the most frequently detected (11). The overlap between these mutations and genes associated with dilated and hypertrophic cardiomyopathy continues the debate about whether LVNC is a distinct cardiomyopathy or a common phenotype (12).

Although recent developments in symptomatic treatments are promising, there is still no targeted treatment for LVNC in children. Current treatments are primarily aimed at managing complications such as heart failure, arrhythmia, and thromboembolism (13). Therefore, sharing experiences from different centers is of great importance for both understanding the natural history of the disease and developing clinical management strategies. Data on LVNC from centers in Anatolia are particularly limited. Our study aims both to contribute to the literature by presenting pediatric LVNC experiences from a regional center and to guide clinicians in our country regarding the diagnosis and management of this rare cardiomyopathy.

Material and Methods

This retrospective descriptive study included patients diagnosed with LVNC who were followed at the Pediatric Cardiology Outpatient Department of Ordu University Training and Research Hospital between December 2017 and July 2025. A total of 16 pediatric patients with regular follow-ups during this period were included in the study.

Patients' electronic records and files were reviewed retrospectively. Demographic data (age, gender, weight), presenting complaints, family history, and physical examination findings were recorded. Cardiac imaging findings, including TTE and CMR results, were evaluated, and results of genetic tests performed on patients were also recorded.

The presence of heart failure, surface electrocardiogram (ECG) findings, 24-hour Holter ECG findings, arrhythmia detection rates, and treatment approaches were recorded. Additionally, medical treatments (e.g., for heart failure and antiarrhythmic therapies), interventional procedures, mechanical support devices, and advanced treatments (e.g., heart transplantation) were reviewed. Mortality and clinical outcomes during the follow-up period were analyzed.

The diagnosis of LVNC was based on TTE and/or CMR findings. TTE was performed with a Philips EPIQ 7 (Philips Healthcare, Andover, MA, USA). Because CMR images were obtained using various devices across different hospitals, information on device brand and model could not be reported uniformly.

The diagnosis of LVNC was established based on TTE and/or CMR findings.

- **TTE Criteria:** The three most commonly used TTE criterion sets in the literature were considered in the study:

Chin Criteria: Defined as a ratio of the distance from the epicardial surface to the trough of the trabeculation (X) to the distance from the epicardial surface to the peak of the trabeculation (Y) of X/Y ≤0.5 at end-diastole (2).

Jenni Criteria: Defined as an end-systolic ratio of non-compacted to compacted (NC/C) myocardium of ≥2 (≥1.4 in children), with a typical location in the apical and lateral/inferior walls, the presence of blood flow communicating with the intertrabecular recesses on Doppler imaging, and the absence of other coexisting structural heart anomalies (1).

Stöllberger Criteria: Defined by the presence of three or more prominent trabeculations located apically, distinct from the compact myocardium but with similar echogenicity and synchronous movement, and blood flow within the intertrabecular recesses visualized on color Doppler. An NC/C ratio of ≥2 at end-diastole was later incorporated as an additional quantitative criterion (14).

• **CMR Criteria:** CMR was used in cases where the diagnosis could not be confirmed by echocardiography or when detailed tissue characterization was required.

Petersen Criteria: Defined as an end-diastolic ratio of NC/C myocardium of >2.3 (10).

Jacquier Criteria: Defined as trabeculated myocardial mass representing >20% of total left ventricular (LV) mass (15).

Grothoff Criteria: Defined by a global NC mass index of >15 g/m² and a ratio of NC mass to total LV mass of >25%, in addition to the presence of an NC/C ratio ≥3:1 in segments 4-6 (16).

The three most commonly used echocardiographic criteria sets were considered in the study (1,2,14). CMR was used to support and confirm diagnoses in patients diagnosed using TTE (10,15,16).

The study was approved by the Ordu University Ethics Committee (decision number: 2025-282, date: 12.09.2025). Due to the retrospective design, informed consent was not obtained. All procedures were conducted in accordance with the Declaration of Helsinki.

Statistical Analysis

All analyses were descriptive and performed using IBM SPSS Statistics for Windows, Version 30.0 (IBM Corp., Armonk, NY, USA). Given the small sample size and the predominantly non-normal distribution of continuous variables, continuous data were summarized as median (minimum-maximum), and categorical variables were presented as frequencies (n) and percentages (%).

Results

A total of 16 pediatric LVNC cases were included in the study. The median age at diagnosis was 7 years (range: 15 days-16 years); 68.8% (n=11) of patients were male. The median body weight was 19.5 kg (range: 3.8-58 kg), and the median follow-up duration was 33 months (range: 6-82 months). Most cases were asymptomatic at presentation (n=10; 62.5%). In symptomatic patients, easy fatigability (n=5) and palpitations (n=3) were most frequently observed; shortness of breath (n=2), chest pain (n=2), and syncope (n=1) were recorded less frequently. Some cases presented with multiple complaints concurrently. Family history was positive in four patients (25%). One case had genetically confirmed (PLEKHM2-positive) LVNC in their mother; two cases had asymptomatic LVNC in their siblings; and one case had LVNC in their father accompanied by dilated cardiomyopathy and heart failure. Phenotypic distribution analysis revealed that the most common type was isolated LVNC (n=8, 50%); the dilated type was observed in five patients, the hypertrophic type in one patient, the restrictive type in one patient, and the mixed type in one patient. Heart failure was detected clinically in six cases (37.5%). The demographic and clinical characteristics of the patients are presented in Table 1.

Table 1. Demographic and clinical characteristics of the patients

Parameters	
Age (years), median (min-max)	7 years (15 days-16 years)
Sex, n (%)	Male: 11 (68.8%) Female: 5 (31.2%)
Weight (kg) Median (min-max)	19.5 (3.8-58)
Follow-up duration (months) Median (min-max)	33 (6-82)
Presenting symptoms*, n	None: 10 Fatigue: 5 Palpitation: 3 Dyspnea: 2 Chest pain: 2 Syncope: 1

Table 1. Continued

Parameters	
Family history, n (%)	Absent: 12 (75) Present: 4 (25)
LVNC type, n (%)	Isolated: 8 (50) Dilated: 5 (31.2) Hypertrophic: 1 (6.2) Restrictive: 1 (6.2) Mixed type (H+R):1 (6.2)
Heart failure, n (%)	6 (37.5)
Arrhythmia, n (%)	Absent: 10 (62.5) Present: 6 (37.5)
Thrombus, n (%)	Absent: 15 (93.8) Present: 1 (6.2)
Death, n (%)	3 (18.8)

**Some patients presented with multiple complaints. LVNC: Left ventricular non-compaction cardiomyopathy, H: Hypertrophic, R: Restrictive, min: Minimum, max: Maximum*

All patients were diagnosed with LVNC by TTE, and CMR confirmed the diagnosis in all patients. Pathogenic mutations were detected in three cases by genetic testing; PLEKHM2 variants were found in two cases, and an RYR2 variant in one case. The case harboring the RYR2 mutation was also diagnosed with CPVT (Table 2).

Arrhythmia was observed in six patients (37.5%). Ventricular tachycardia (VT) was detected in two cases, one of whom was a patient diagnosed with CPVT. Wolff-Parkinson-White (WPW) syndrome-associated supra-VT was observed in two cases, and frequent ventricular extrasystoles were observed in two cases. On 12-lead surface ECG and 24-hour Holter ECG assessments, sinus tachycardia, left bundle branch block, and biatrial dilatation were also recorded in two patients each (Table 2).

Two patients underwent interventional procedures. Sympathetic blockade (sympathectomy) was performed in one patient diagnosed with catecholaminergic polymorphic VT, and an implantable cardioverter-defibrillator (ICD) was implanted in another patient. The patient who received an ICD also underwent heart transplantation and died on the third postoperative day. Three patients (18.8%) died during the follow-up period: the first, a 14-year-old male, had dilated-type LVNC, congestive heart failure, and a PLEKHM2 mutation; the second, a four-year-old patient, had mixed-type LVNC (dilated-restrictive); and the third, a three-year-old child, had restrictive-type LVNC (Table 2).

As medical treatment, acetylsalicylic acid was initiated in all patients, and beta-blockers were added in most patients. Antiarrhythmic agents such as flecainide and amiodarone, as well as anticongestive treatments, were used when clinically necessary. Multiple-drug combinations were preferred, especially in patients with arrhythmia or who had developed heart failure.

Discussion

In this single-center pediatric series, we presented the phenotypic diversity, arrhythmia burden, and short- to medium-term outcomes of LVNC based solely on our data, and compared our findings with the current literature.

Phenotypic heterogeneity in pediatric LVNC and its impact on prognosis are well known (a relatively better course in the isolated form and a worse course in the dilated form) (1,7). The predominance of isolated LVNC in our series (50%) was consistent with the phenotype distributions reported in the literature for pediatric patients; however, the clustering of death and the need for advanced intervention in the dilated/mixed-restrictive subtypes support the view that phenotype-based risk distinction may be determinative in clinical management (7,9). The higher symptom burden and more advanced stage of disease among cases presenting to our clinic, compared with the general population, may have contributed to the high observed rate of heart failure (37.5%) (9,17).

Our total arrhythmia rate is 37.5%. Our VT frequency was 12.5% (2/16), close to rates reported in pediatric series: Stanton et al. (18) reported 13.3%, Brescia et al. (9) reported 17%, and Chin et al. (2) reported VT rates up to 38%. Our WPW association rate was 12.5% (2/16), which is highly consistent with the findings of Howard et al. (19), who reported a WPW association of approximately 10% in the pediatric LVNC population. These findings support the need for close rhythm monitoring in pediatric LVNC and for early rhythm-

Table 2. Diagnosis, follow-up, and outcomes of pediatric LVNC patients

Case	Age (year)	Echo findings	MRI confirmation	Genetics confirmation	ECG-Holter findings	Thrombus	Medical treatment	Interventional procedure	Transplantation	Death
1	4 y	LVNC, RCM-LVH, MR (moderate)	+	-	LBBB, BAD, sinus tachycardia	+	ASA, β1-BB, AC	-	-	+
2	14 y	LVNC+D	+	PLEKHM2	VT	-	ASA , β1-BB, FLEC, AC	ICD	Yes	+
3	15 day	LVNC+D, Ebstein anomalisi	+	-	SVT, WPW	-	ASA, AMIO, β1-BB, AC	Sympathectomy	-	-
4	8 y	LVNC	+	-	Normal	-	ASA	-	-	-
5	12 y	LVNC+H	+	RYR2	VT	-	ASA, β1-BB, FLEC	-	-	-
6	2 y	LVNC+D	+	-	Normal	-	ASA	-	-	-
7	4 y	LVNC	+	-	Normal	-	ASA	-	-	-
8	13 y	LVNC	+	-	Normal	-	ASA	-	-	-
9	5 y	LVNC+D, MR (mild)	+	-	Frequent VES	-	ASA, β1-BB, AC	-	-	-
10	1 y	LVNC	+	-	Normal	-	ASA	-	-	-
11	6 y	LVNC	+	-	Normal	-	ASA	-	-	-
12	16 y	LVNC+D	+	-	Frequent VES, LBBB	-	ASA, β1-BB, AC	-	-	-
13	14 y	LVNC	+	PLEKHM2	Normal	-	ASA	-	-	-
14	6 y	LVNC	+	-	SVT, WPW	-	ASA	-	-	-
15	3 y	LVNC+R	+	-	Sinus tachycardia, BAD	-	ASA, β1-BB, AC	-	-	+
16	4 y	LVNC	+	-	Normal	-	ASA	-	-	-

MRI: Magnetic resonance imaging, ECG: Electrocardiogram, LVNC: Left ventricular non-compaction, LVH: Left ventricular hypertrophy, D: Dilated, H: Hypertrophic, R: Restrictive MR: Mitral regurgitation, LBBB: Left bundle branch block, BAD: Biatrial dila, VT: Ventricular tachycardia, SVT: Supraventricular tachycardia, WPW: Wolff-Parkinson-White syndrome, VES: Ventricular extrasystole, ASA: Acetylsalicylic acid, β1-BB: Beta-blocker, AMIO: Amiodarone, FLEC: Flecainide, AC: Anticongestive, ICD: Implantable cardioverter-defibrillator, RCM: Restrictive cardiomyopathy

management strategies in patients with conduction defects or accessory pathways (19,20).

In our study, TTE criteria (1,2,14) were systematically applied; given the interpretive differences of the criteria in pediatric cases and the controversial nature of NC/C, confirmation with CMR was performed in all patients (1,2,10,14). The structural and confirmatory power of CMR [e.g., Petersen et al. (10) NC/C >2.3; trabeculated mass ratio as described by Jacquier et al. (15); global NC mass criteria proposed by Grothoff et al. (16)] is particularly valuable in reducing the risk of overdiagnosis or missed diagnoses, especially in childhood. Furthermore, since increased physiological trabeculation in athletes has been shown to mimic LVNC, it is prudent to avoid making a diagnosis based solely on morphology and to adopt a multiparametric TTE + CMR + ECG + family history ± genetic) approach (3,4,5).

In our series, two PLEKHM2 variants and one RYR2 variant were detected, and the RYR2 carrier presented with CPVT. Although sarcomere gene variants (MYH7, TTN, MYBPC3) are most frequently reported in LVNC, genetic heterogeneity is substantial, and the same genes can be associated with different cardiomyopathy phenotypes; these findings support individualized screening and family investigation in pediatric cases (1,3,11). Our small sample size is insufficient for statistical testing of genotype-outcome relationships; however, the VT event in the case of CPVT underscores the arrhythmic risk among channelopathy-related phenotypes (20).

In our series, with a median follow-up of 33 months, mortality was 18.8%, a rate consistent with the 12.8-22% range reported in the pediatric literature, and fatalities were concentrated in the dilated/mixed-restrictive subtypes, an expected pattern (9,17). Our experience yields three clinical

messages: 1) Phenotype-focused monitoring, particularly close clinical follow-up for the dilated and mixed-restrictive subtypes. 2) Rhythm strategies, pathways for early intervention in the presence of WPW/conduction defects and VT. 3) Diagnostic assurance, confirmation of TTE criteria with CMR in pediatric patients (10,19,20). Treatment in pediatric LVNC is focused on the management of heart failure, arrhythmias, and thromboembolism rather than on targeted therapy; in our practice, treatment with acetylsalicylic acid and beta-blockers was emphasized, with antiarrhythmics and interventions applied in selected cases (3,13).

Study Limitations

This study has several limitations that should be considered. The retrospective and single-center design with a small sample size limits the statistical power, particularly for phenotype-outcome and genotype-outcome relationships. Rhythm assessment is based on ECG/Holter; invasive electrophysiological data and long-term device outcomes are not available for all patients.

Conclusion

This pediatric series reported from Anatolia shows that the isolated form predominates in LVNC, but clinical risk varies in a phenotype-dependent manner. The burden of arrhythmia and heart failure is notable; fatalities were concentrated in the dilated and mixed-restrictive subtypes. In pediatric patients, confirming TTE criteria with CMR and implementing close monitoring based on phenotype-rhythm findings may facilitate accurate classification and risk management. Larger, multicenter, and genetically integrated pediatric cohorts will contribute to the development of phenotype-focused risk models.

Ethics

Ethics Committee Approval: The study was approved by the Ordu University Ethics Committee (decision number: 2025-282, date: 12.09.2025).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: T.K., E.Y.E., Concept: T.K., Design: T.K., Data Collection or Processing: T.K., E.Y.E., Analysis or Interpretation: T.K., E.Y.E., Y.K.A., Literature Search: T.K., E.Y.E., Writing: T.K., E.Y.E., Y.K.A.

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