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CLINICAL PRESENTATION OF LEFT VENTRICULAR NONCOMPACTION IN CHILDREN

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ABSTRACT

Left ventricular noncompaction (LVNC) is a heterogeneous form of cardiomyopathy associated with abnormal myocardial maturation and characteristic prominent trabeculation of the left ventricular wall. The classification of the disease remains controversial, and some authors consider LVNC to represent a phenotypic manifestation of other cardiomyopathies. The clinical presentation, particularly in children, is highly variable, ranging from asymptomatic forms to heart failure, arrhythmias, and thromboembolic events. Echocardiography remains the cornerstone of diagnosis, complemented by cardiac magnetic resonance imaging; however, the lack of universally accepted diagnostic criteria contributes to the risk of overdiagnosis. Treatment is primarily symptomatic and depends on the disease phenotype and the degree of left ventricular dysfunction. Prognosis is determined mainly by cardiac function and the presence of arrhythmias, whereas isolated LVNC with preserved systolic function is generally associated with a favorable clinical course.

KEYWORDS

Left Ventricular Noncompaction, Noncompaction Cardiomyopathy, Pediatric LVNC, Ventricular Dysfunction

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Classification and Epidemiology of Left Ventricular Noncompaction

Left ventricular noncompaction (LVNC) is a developmental abnormality characterized by impaired maturation of the left ventricular myocardial wall [1,2]. According to the classification of the European Society of Cardiology (ESC), this condition is categorized as an unclassified cardiomyopathy, whereas the American Heart Association (AHA) classifies it as a genetically determined cardiomyopathy [3–5]. The clinical presentation and genetic background of LVNC are highly heterogeneous [1,2]. Some investigators, including Monserrat, have suggested that LVNC may not represent a distinct disease entity but rather a phenotypic manifestation of other cardiomyopathies, such as hypertrophic, restrictive, or dilated cardiomyopathy, with substantial overlap among the diagnostic criteria used for these conditions [6–10].

Structural Features of LVNC

The LVNC phenotype is characterized by a two-layered structure of the affected segments of the left ventricular wall [1,2]. An inner endocardial layer with prominent trabeculations and deep intertrabecular recesses communicating directly with the ventricular cavity can be distinguished, along with an outer epicardial layer that is thin and normally compacted, collectively giving the myocardium a characteristic “spongy” appearance. The abnormalities are most commonly localized at the cardiac apex and within the apical and mid-ventricular segments of the inferior and lateral left ventricular walls [1,2]. Left ventricular dilatation is frequently observed and may be accompanied by hypertrophy as well as systolic and diastolic dysfunction. The morphological presentation of LVNC is highly heterogeneous and variable. The literature also describes the coexistence of facial dysmorphic features, such as frontal bossing, strabismus, low-set ears, and mandibular hypoplasia, as well as the presence of Wolff–Parkinson–White syndrome, which is diagnosed more frequently in children than in adults [11–14].

Clinical Presentation of LVNC in Children

The clinical presentation of left ventricular noncompaction (LVNC) in the pediatric population is highly heterogeneous, similarly to that observed in adults [11–15]. The disease may remain asymptomatic or present with a broad spectrum of symptoms and complications. Three principal groups of manifestations are recognized: heart failure, cardiac arrhythmias, and thromboembolic events, which may occur individually or in various combinations [1,11,14–16].

Heart failure represents the most common mode of presentation of LVNC in children [11–15]. Available studies indicate that it is the predominant initial manifestation, whereas a substantial proportion of diagnoses are established in asymptomatic patients in whom the disease is detected incidentally or during family screening, for example after cardiomyopathy has been diagnosed in a relative [11,14,15]. This underscores the importance of family history assessment and heightened diagnostic vigilance in at-risk populations.

LVNC may occur in isolation or coexist with ventricular dysfunction involving both systolic and diastolic impairment [1,11,14,15]. Importantly, the degree of trabeculation does not necessarily correlate directly with the severity of cardiac dysfunction [17,18]. In structurally normal hearts with preserved function, no clear association has been demonstrated between the extent of trabeculation and functional parameters. These findings suggest that, in some patients, excessive trabeculation may have limited hemodynamic significance, and that clinical assessment should focus not only on the “quantitative” severity of the morphological abnormality but also on associated ventricular dysfunction, remodeling, or other cardiomyopathic features.

Cardiac arrhythmias constitute another important group of manifestations [15,19]. Arrhythmias and electrocardiographic abnormalities are observed more frequently in children with LVNC, particularly in the presence of heart failure or impaired left ventricular systolic function [11–15,19]. Although the abnormal myocardial architecture might intuitively suggest an increased arrhythmogenic potential, available evidence indicates that, after adjustment for associated factors such as left ventricular dilatation, systolic dysfunction, or myocardial fibrosis, excessive trabeculation alone may not represent an independent risk factor for arrhythmias. From a clinical perspective, it is also important to recognize that some ventricular arrhythmias may originate from remodeled myocardial regions, including scarred segments or the ventricular outflow tract.

Electrocardiographic abnormalities are common in pediatric LVNC, although they are generally nonspecific. Clinical observations most frequently demonstrate increased QRS voltages consistent with left ventricular hypertrophy, as well as repolarization abnormalities involving ST-segment and T-wave changes [11–13,15]. Both ventricular and supraventricular tachyarrhythmias have been reported in children, particularly forms associated with atrioventricular re-entry mechanisms or focal atrial tachycardia. Notably, the incidence of sudden cardiac death varied across pediatric cohorts; however, it was not observed in children without prior arrhythmias or in patients with normal cardiac dimensions and preserved cardiac function, highlighting the importance of risk stratification based on clinical and functional parameters [15].

The third major category of complications in LVNC comprises thromboembolic events [1,16]. In childhood, a reduced left ventricular ejection fraction constitutes a major risk factor for these complications, particularly when the ejection fraction falls below 40% [16]. Cases of left ventricular thrombus formation, including thrombi located within intertrabecular recesses, have been described. At the same time, cohort studies suggest that in children with preserved ejection fraction, excessive trabeculation alone may not be associated with an increased risk of adverse events, again emphasizing that left ventricular functional parameters and concomitant cardiomyopathic features are the key determinants of prognosis [17,18].

Diagnostic Criteria for Left Ventricular Noncompaction

The diagnosis of left ventricular noncompaction (LVNC), both in children and adults, is based primarily on morphological criteria [20–32]. Echocardiography remains the fundamental diagnostic modality and also plays a central role in longitudinal clinical follow-up [20–25]. Cardiac magnetic resonance imaging (CMR) serves as a complementary imaging technique, used mainly to confirm the diagnosis and assess prognosis, including the identification of myocardial fibrosis by means of late gadolinium enhancement (LGE) imaging [27–32].

Echocardiography

The absence of a universally accepted “gold standard” for the diagnosis of LVNC has led to the development of multiple echocardiographic criteria, not all of which have been adequately validated [20–25]. In the pediatric population, several sets of criteria are used, differing both in measurement methodology and in the phase of the cardiac cycle during which assessment is performed.

The most commonly cited are the criteria proposed by Chin, Jenni, Stöllberger, and Pignatelli [11,20–22]. These criteria are based either on the assessment of the ratio between the trabeculated (noncompacted) and compacted myocardial layers of the left ventricle (NC/C ratio) or on the measurement of the distance between trabecular structures and the epicardium. Measurements are performed in different echocardiographic views, most commonly in the apical four-chamber view and the left ventricular short-axis view.

In clinical practice, the criteria proposed by Jenni et al. are most widely applied. These criteria require the presence of a two-layered myocardial structure of the left ventricle and a noncompacted-to-compacted myocardial thickness ratio exceeding 2:1, measured during systole in the short-axis view [21]. The most prominent trabeculation is typically observed in the apical segments as well as in the mid-lateral and mid-inferior wall segments. An important assumption of these criteria is the absence of concomitant congenital heart defects.

Population-based neonatal studies have demonstrated that an increased NC/C ratio may be present in a substantial number of clinically asymptomatic children, most commonly within the apical region of the left ventricle [26]. These findings emphasize the limited specificity of some diagnostic criteria and suggest the possibility of LVNC overdiagnosis, particularly in asymptomatic populations [23–26,30,31].

Comparative analyses of echocardiographic criteria have shown that not all parameters exhibit equal reproducibility and diagnostic value. In pediatric cohorts, the X/Y ratio (describing the relationship between the depth of intertrabecular recesses and trabecular height) measured during diastole has been reported to demonstrate greater sensitivity and specificity than the conventional NC/C ratio. In contrast, the NC/C ratio has shown lower measurement reproducibility, limiting its usefulness in certain patient groups [24,25].

Additional diagnostic challenges arise from difficulties in clearly delineating the border between the compacted and noncompacted myocardial layers, as well as from the relatively small study populations on which the original criteria were established. In cases where visualization of the left ventricular cavity is suboptimal, contrast-enhanced echocardiography, three-dimensional echocardiography, and advanced imaging techniques such as speckle-tracking strain analysis may prove helpful [1,33]. These methods have demonstrated the ability to detect subclinical left ventricular dysfunction in children with LVNC and preserved ejection fraction before abnormalities become apparent in conventional echocardiographic parameters.

Cardiac Magnetic Resonance Imaging

Cardiac magnetic resonance imaging (CMR) is considered a second-line modality in the diagnosis of LVNC. It provides superior visualization of cardiac structures, particularly in patients with suboptimal acoustic windows on echocardiography. In addition, CMR enables the assessment of myocardial fibrosis, which may have important prognostic implications.

Several CMR-based diagnostic criteria for LVNC in adults have been proposed. The most widely used are the Petersen criteria, which define LVNC as a ratio of noncompacted to compacted myocardial thickness greater than 2.3 during diastole [27–32]. Other approaches include assessment of the proportion of trabeculated myocardial mass relative to total left ventricular mass, quantification of trabecular volume, or fractal analysis of endocardial border irregularity [28–31].

Population-based studies have demonstrated, however, that fulfillment of CMR criteria may also occur in individuals without clinical features of cardiomyopathy, once again raising concerns regarding the limited specificity of morphological criteria [17,30,31]. Some authors have suggested that measurements obtained during systole may correlate more closely with the risk of heart failure and cardiovascular events than the conventional diastolic criteria [29].

It should be emphasized that dedicated, validated CMR criteria for the pediatric population are lacking, and the currently applied diagnostic principles are largely extrapolated from studies conducted in adults [32].

Treatment and Management of Patients with Left Ventricular Noncompaction

There is no causal treatment for left ventricular noncompaction (LVNC); therefore, therapeutic management is primarily symptomatic and phenotype-oriented [1,5,33]. According to current clinical practice, treatment should be tailored to the patient's clinical presentation, the degree of left ventricular dysfunction, and the presence of complications such as heart failure, arrhythmias, or thromboembolic events. Particular emphasis is placed on individualized therapy and long-term clinical follow-up.

Pharmacological Treatment

Pharmacotherapy in LVNC is based on standard principles of heart failure management [5,33,34]. In patients with reduced left ventricular ejection fraction, treatment includes angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, β -blockers, and mineralocorticoid receptor antagonists. Diuretics are indicated in the presence of congestive symptoms. The choice of therapy depends on the severity of clinical symptoms, treatment tolerance, and the patient's age.

In the pediatric population, pharmacological treatment should be administered cautiously, taking into account the child's somatic development. In asymptomatic patients with preserved left ventricular systolic function, management may be limited to regular clinical monitoring without initiation of pharmacotherapy [1,17,18,33].

Management of Complications

One of the major clinical challenges in patients with LVNC is the occurrence of cardiac arrhythmias, which are more frequent in individuals with significant left ventricular dysfunction. In the pediatric population, arrhythmias and heart failure constitute the principal risk factors for adverse outcomes, including sudden cardiac death. In selected cases, following comprehensive risk stratification, device therapy, including implantation of an implantable cardioverter-defibrillator (ICD), may be considered [5,15,19,33,35].

Another important complication of LVNC is thromboembolic events. In childhood, the primary risk factor for their occurrence is reduced left ventricular ejection fraction, particularly below 40%. Anticoagulant therapy is recommended in patients with a history of thromboembolic events, intracardiac thrombi, or atrial fibrillation [5,16,33]. Routine anticoagulant prophylaxis is not recommended in patients with preserved left ventricular systolic function [16,33].

In cases of end-stage heart failure refractory to conservative treatment, advanced therapeutic strategies may be considered in selected patients, including mechanical circulatory support and heart transplantation.

Patient Monitoring

Patients with LVNC require systematic long-term follow-up regardless of the presence or absence of clinical symptoms [1,5,33]. Echocardiography remains the cornerstone of follow-up assessment, enabling evaluation of left ventricular function, the degree of ventricular remodeling, and potential disease progression [20–25,33]. Electrocardiographic investigations, including Holter monitoring, constitute an important adjunctive tool, particularly in patients with suspected arrhythmias [15,19,33].

Cardiac magnetic resonance imaging represents an important modality in prognostic assessment, especially through the detection of myocardial fibrosis using late gadolinium enhancement (LGE) imaging. The presence of LGE is associated with a higher risk of cardiovascular events and may influence therapeutic decision-making [27–32].

The Role of Long-Term Care

LVNC is a chronic condition requiring continuous specialist care. A multidisciplinary approach and continuity of care are of paramount importance, particularly during the transition from pediatric to adult healthcare services. In cases where a familial form of the disease is suspected, genetic counseling and screening of relatives are recommended [5,35–38]. The pathogenesis of some LVNC cases has been linked to abnormalities in signaling pathways regulating myocardial development, including the NOTCH signaling pathway [39].

Education of patients and their families regarding warning symptoms, the importance of regular follow-up, and the principles of safe physical activity constitutes an integral component of long-term care and may significantly influence both prognosis and quality of life.

Prognosis

The prognosis of patients with left ventricular noncompaction (LVNC) is heterogeneous and largely depends on concomitant cardiac functional abnormalities rather than on the mere presence of excessive left ventricular trabeculation. Numerous studies have demonstrated that left ventricular dysfunction is the most important prognostic factor associated with an adverse clinical course [14,15,17,18,32]. Patients with reduced ejection fraction exhibit significantly higher cardiovascular mortality compared with individuals with preserved left ventricular systolic function.

Cardiac magnetic resonance imaging provides important prognostic information, particularly through the assessment of late gadolinium enhancement (LGE), which reflects myocardial fibrosis. Studies involving

patients with excessive left ventricular trabeculation have shown that the presence of LGE is associated with an increased risk of cardiac events [32]. At the same time, data comparing the prognosis of patients with LVNC and those with dilated cardiomyopathy remain inconclusive. Some observations suggest a higher risk of cardiovascular events in patients with excessive trabeculation; however, multivariable-adjusted analyses have not consistently demonstrated significant differences in event-free survival between these groups.

In asymptomatic individuals with normal cardiac function, the isolated phenotypic feature of excessive trabeculation does not appear to have independent prognostic significance [17,18,40]. In contrast, in patients in whom LVNC coexists with another cardiomyopathic phenotype, the risk of adverse events is determined primarily by the nature and severity of the associated cardiomyopathy rather than by the degree of trabeculation itself.

The prognosis of children with LVNC is particularly variable and depends on multiple clinical factors. Pediatric studies have demonstrated that impaired left ventricular systolic function is the strongest predictor of death or the need for heart transplantation. Children with preserved cardiac function generally have an excellent prognosis, whereas significantly worse outcomes are observed in patients with systolic dysfunction.

Another important determinant of disease course in the pediatric population is the cardiomyopathy phenotype. The most favorable prognosis is observed in children with isolated LVNC and normal cardiac geometry, whereas poorer outcomes are associated with hypertrophic, mixed, or dilated phenotypes. Children with concomitant dilated cardiomyopathy exhibit the highest risk of death or heart transplantation [14,15].

Additional adverse prognostic factors in children include electrocardiographic abnormalities, particularly repolarization disturbances and the occurrence of arrhythmias [11,12,15]. Especially unfavorable outcomes are observed in patients whose disease manifests during the first year of life and in those presenting with ventricular arrhythmias early in the disease course [11,14,15].

Echocardiographic parameters assessing diastolic function also carry prognostic significance. Pediatric studies have shown that reduced lateral mitral annular velocity (Ea), assessed by Doppler imaging, is a sensitive marker of increased risk of death or heart transplantation. Furthermore, the presence of myocardial fibrosis detected by cardiac magnetic resonance imaging may represent an additional factor associated with poorer prognosis [32].

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