

Case Report

Echocardiographic Evaluation of Noncompaction Cardiomyopathy - Rare case report and literature review

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ABSTRACT:

Left ventricular non-compaction (LVNC) represents a diverse myocardial condition marked by exaggerated trabeculations extending into the left ventricular cavity and deep intertrabecular recesses. This pathology may occur as an isolated entity or in association with other cardiomyopathies. Its recognition in pediatric populations has increased, largely owing to advancements in cardiac imaging. The etiological spectrum of LVNC is broad, encompassing both hereditary and acquired mechanisms. Clinical presentation ranges widely: while some individuals remain asymptomatic, others exhibit manifestations such as heart failure, arrhythmias, or systemic embolic events. Diagnosis is predominantly based on structural cardiac assessment through imaging modalities, primarily echocardiography and cardiac magnetic resonance. Echocardiography remains the cornerstone for evaluating LVNC, with 2-dimensional, 3-dimensional, color Doppler, and contrast-enhanced techniques playing essential roles in both diagnosis and family screening. Although computed tomography and magnetic resonance imaging serve as additional diagnostic options, current evidence does not conclusively demonstrate their superiority over echocardiography. In this report, we describe a 2-month-old infant diagnosed with LVNC accompanied by severe left ventricular systolic dysfunction and marked pulmonary hypertension. A detailed transthoracic echocardiography (TTE) was performed to establish the definitive diagnosis.

Keywords: *Cardiomyopathy, Echocardiography, Left ventricular noncompaction, Cardiomyopathy, Myocardial disorder, Intertrabecular recesses.*

INTRODUCTION:

Left ventricular non-compaction (LVNC) is defined by the presence of a thin compact epicardial layer alongside a markedly thickened non-compact endocardial layer composed of prominent trabeculations and deep intertrabecular recesses. When LVNC occurs concurrently with impaired left ventricular (LV) systolic function, the condition is classified as LVNC cardiomyopathy; however, similar morphological features may also be observed in

individuals who maintain normal ventricular performance. LVNC cardiomyopathy is associated with a range of complications, including heart failure, arrhythmias, thromboembolic events, and sudden cardiac death [1–3].

Although LVNC is a relatively recent clinical entity, its hallmark feature consists of a heavily trabeculated myocardium with deep intertrabecular recesses that maintain continuity with the LV cavity (Figures 1, 2) [4].

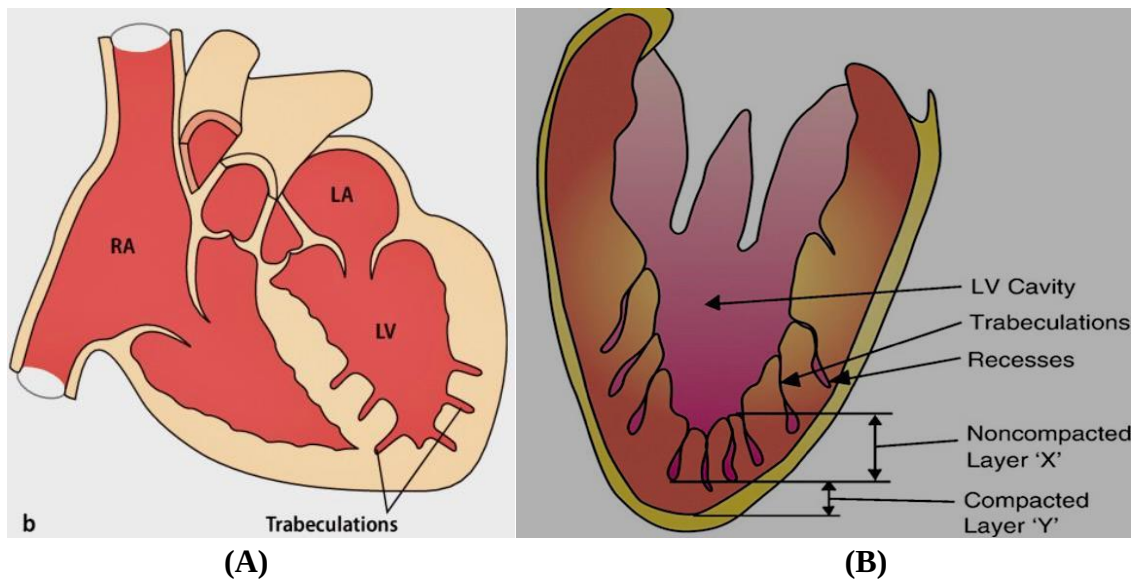


Figure 1: Schematic representation of LVNC. (A) Illustration highlighting left ventricular non-compaction (LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle). (B) Depiction of the compacted and non-compacted myocardial layers of the LV, demonstrating the characteristic deep trabeculations and intertrabecular recesses.

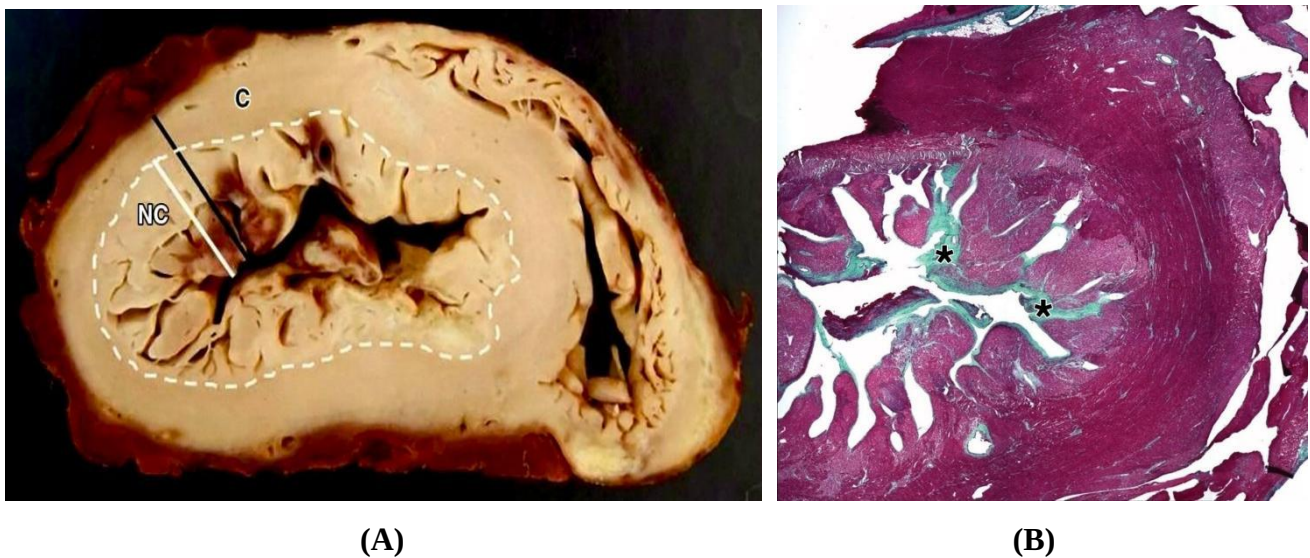


Figure 2: (A) Photograph of a necropsy specimen from an infant demonstrating the characteristic gross morphology of non-compaction. The coronal transection of both ventricles reveals the two-layered appearance of the left ventricular myocardium, consisting of a thin epicardial compacted layer (C) and a markedly thickened non-compacted endocardial layer (NC). The non-compacted layer (outlined by the dashed border) displays excessively prominent trabeculations and deep intertrabecular recesses that communicate directly with the left ventricular cavity. In several regions, the thickness of the non-compacted layer (solid white line) exceeds 75% of the overall myocardial wall thickness (black line). (B) Low-power photomicrograph (Masson trichrome, ×2) illustrating the histologic features. The cross-sectional view of the left ventricular myocardium demonstrates notable endocardial fibroelastosis (asterisks) overlying the trabeculated myocardial surface, with fibroelastosis extending into the intertrabecular recesses.

Prominent myocardial trabeculations were initially documented in association with several congenital heart

anomalies, as well as in cases without additional structural cardiac abnormalities [5,6]. Early

descriptions emphasized the persistence of embryonic myocardial components, historically referred to as persistent myocardial sinusoids. In 1990, Chin et al. [7] introduced the term “isolated non-compaction of the LV myocardium,” highlighting the failure of the normal compaction process during embryonic development. The regions most frequently involved include the apical and mid-ventricular segments of the inferior and lateral left ventricular walls [8].

The range of clinical manifestations is broad and often nonspecific at presentation [8,9]. Patients may be asymptomatic or may present with chest discomfort, heart failure symptoms, or arrhythmias. In advanced stages, more than half of affected individuals develop heart failure, with reduced systolic function being the most common abnormality observed [10]. LVNC is

associated with potentially life-threatening complications such as thromboembolic events, arrhythmias, and sudden cardiac death [11,12]. Early recognition and prompt management of high-risk patients can mitigate these outcomes. Preventive strategies include the use of anticoagulation in those predisposed to thromboembolism [13] and prophylactic implantation of an implantable cardioverter-defibrillator (ICD) in individuals at risk for malignant arrhythmias [8].

Progress in noninvasive cardiac imaging particularly high-resolution TTE (Figures 3–5) has significantly enhanced visualization of myocardial morphology in LVNC, enabling improved detection and diagnostic accuracy.

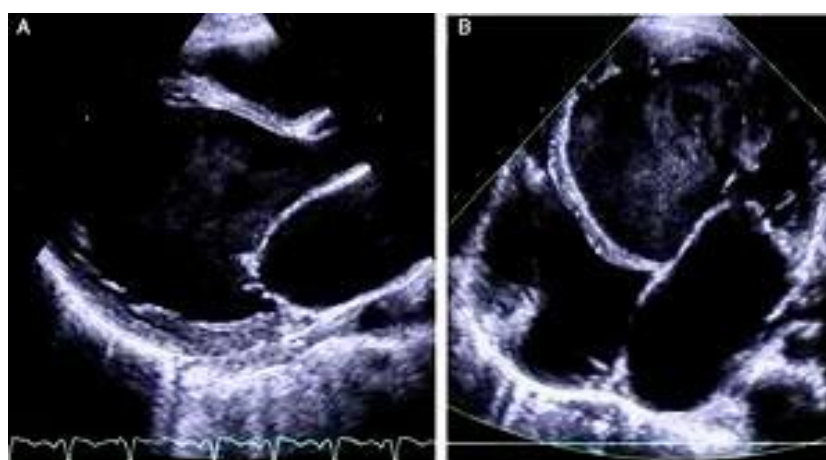


Figure 3: (A) Transthoracic echocardiographic long-axis parasternal view demonstrating prominent trabeculations along the left ventricular lateral wall, interspersed with deep intertrabecular recesses. (B) Apical four-chamber transthoracic echocardiographic view depicting marked trabeculation involving the left ventricular lateral wall and apex.

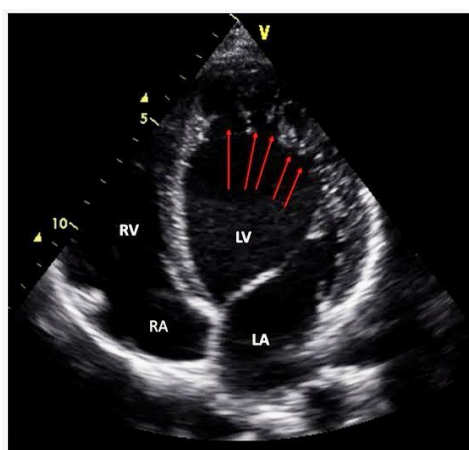


Figure 4: Apical four-chamber echocardiographic view from a patient with NCCM demonstrating mild dilation of the left ventricle (LV). The arrows indicate deep recesses within the non-compacted myocardial layer involving the apical and lateral LV walls. LV, left ventricle; LA, left atrium; RV, right ventricle; RA, right atrium.

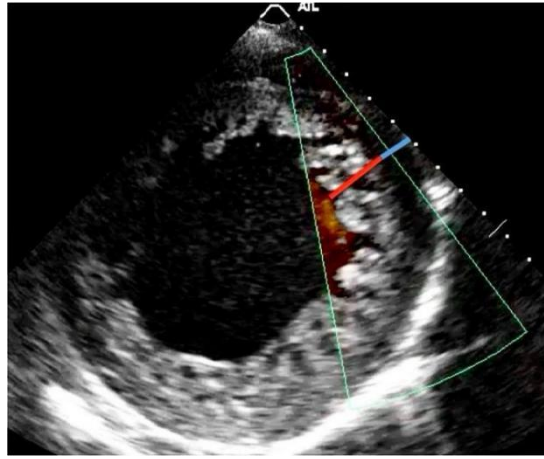


Figure 5: Systolic short-axis echocardiographic image from a patient with LVNC illustrating the measurement of myocardial layer thickness, where the ratio of the compacted layer (blue line) to the non-compacted layer (red line) exceeds 2. Doppler color flow imaging demonstrates blood flow within the intertrabecular recesses.

Echocardiography:

Several echocardiographic criteria have been introduced for diagnosing LVNC; however, the parameters established by Jenni et al. [14] remain the most widely accepted and routinely utilized (Table 1, Figure 6).

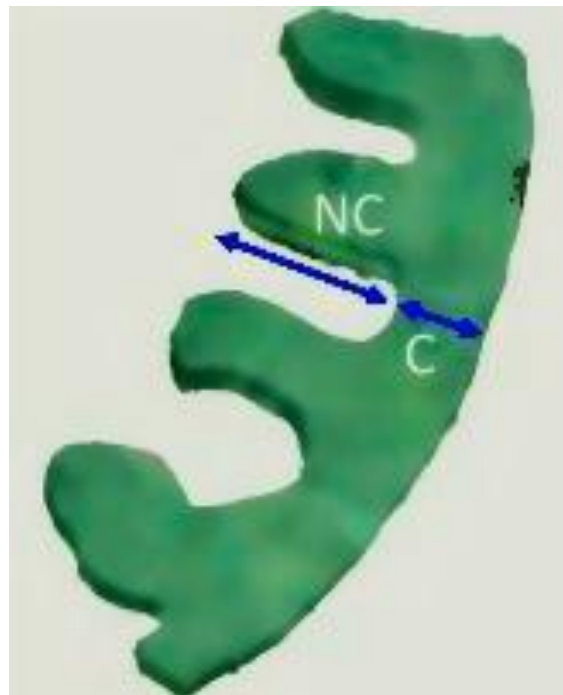


Figure 6: The criteria by Jenni et al. [14]

- 1) A two-layer structure with a thin, compacted layer (C) and a thickened non-compacted layer (NC) at end-systole.
- 2) A ratio of $NC/C > 2$.
- 3) Intertrabecular spaces are filled by blood flow from the ventricular cavity on color Doppler.

Table 1: Echocardiographic Diagnostic Criteria for Left Ventricular Noncompaction

Authors	Criteria
Jenni et al. [14]	Presence of prominent left ventricular trabeculations, predominantly in the apical and midventricular areas of both the inferior and lateral walls
	Two-layered appearance of the myocardium with a thin, compacted outer (epicardial) band and a thicker, noncompacted inner (endocardial) layer; end-systolic ratio between noncompacted and compacted myocardium is greater than 2
	Multiple deep intertrabecular recesses communicating with the ventricular cavity, as visualized at color Doppler imaging
	Absence of additional coexisting cardiac abnormalities
Chin et al. [7]	Presence of excessively prominent ventricular trabeculations and progressively increased total thickness of the myocardial wall from the mitral valve and towards the apical region, characterized by compacted myocardium/(noncompacted myocardium + compacted myocardium) ≤ 0.5 , assessed at end-diastole on short-axis parasternal views and/or apical views.
StÖllberger et al. [15]	End-diastolic ratio between noncompacted and compacted myocardium greater than 2 in apical four-chamber view
Gebhard et al. [16]	Systolic thickness of compacted myocardium less than 8 mm may facilitate differentiation of left ventricular noncompaction from normal hearts and potentially from those with myocardial hypertrophy

Differentiating the non-compacted from the compacted myocardial layers at end-diastole can be challenging on echocardiography; therefore, Jenni et al. recommended using the end-systolic ratio of compacted to non-compacted myocardium. A non-compacted to compacted myocardial ratio exceeding 2 in the short-axis view during end-systole is considered diagnostic for LVNC [14].

Chin et al. [7] proposed an alternative criterion, defining LVNC by a CM/(NCM + CM) ratio ≤ 0.5 measured in diastole (Table 2, Figure 7).

Table 2: Criteria proposed for the diagnosis of noncompacted myocardium [4].

Criterion	Chin et al [4]
Phase of the cardiac cycle	End-diastole
View	Short axis parasternal views and/or apical views
CM/(NCM+CM) ratio	≤ 0.5

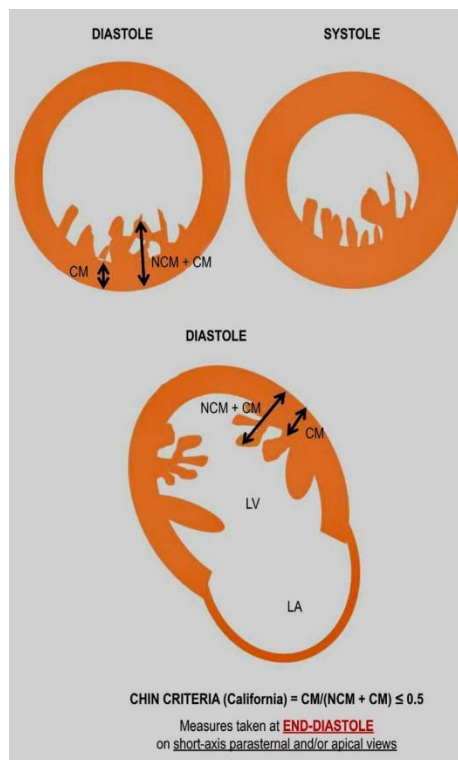


Figure 7: Criteria proposed by Chin for the diagnosis of noncompacted myocardium. NCM: noncompacted myocardium; CM: compacted myocardium; LV: left ventricle; LA: left atrium.

In 2012, Stöllberger et al. [15] refined echocardiographic diagnostic criteria (Table 1), establishing that the presence of more than three trabeculations and a double-layered myocardium are both essential features, optimally visualized in the end-diastolic four-chamber view. Gebhard et al. [16] suggested an additional parameter to enhance LVNC diagnosis by measuring the maximum systolic thickness of the compacted myocardium, noting that a thickness below 8 mm may aid in distinguishing LVNC from normal hearts and from myocardial hypertrophy. Further research is required to validate the added diagnostic utility of this criterion, particularly in patients with coexisting cardiomyopathies.

Echocardiography has several limitations. First, it is highly operator-dependent [17–19]. Second, the apex, which is most commonly affected in LVNC, can be difficult to visualize [17]. Third, trabeculations or the double-layered myocardium may not always be clearly delineated, raising the possibility of misdiagnosis as another cardiomyopathy, such as hypertrophic cardiomyopathy [20]. In such cases, contrast-enhanced echocardiography improves visualization of trabeculations, as the contrast medium fills the intertrabecular recesses that communicate with the

ventricular cavity [18]. Fourth, short-axis images may not be perpendicular to the LV long axis, potentially creating a false appearance of prominent trabeculations that mimic LVNC [17].

CASE REPORT:

A two-month-old male infant was referred to our center for a detailed evaluation of suspected congenital heart disease. According to his parents, he had experienced pronounced breathlessness since birth.

On examination, the infant appeared of average built but exhibited facial puffiness, marked respiratory distress, and profuse forehead sweating (Figure 8). Prominent intercostal retractions and notable sternal in drawing were observed. His anthropometric and vital signs were recorded as follows: weight 3.9 kg, length 56 cm, blood pressure 78/40 mmHg, heart rate 147/min, respiratory rate 88/min, and SPO₂ 99% on room air. Cardiovascular assessment revealed a grade 2/6 systolic murmur, most pronounced at the left infraclavicular region. The second heart sound was loud with a narrowly split component. The remainder of the systemic examination was unremarkable.



Figure 8: Facial appearance of our index patient. Marked breathlessness, sweating over forehead and puffiness of face was recognized.

The chest X-ray in the posteroanterior (PA) view (Figure 9) demonstrated marked cardiomegaly accompanied by pulmonary edema.



Figure 9: X-ray chest (PA view). Gross cardiomegaly with pulmonary edema was discerned.

The resting electrocardiogram (ECG) (Figure 10) showed markedly elevated QRS voltages in the precordial leads.

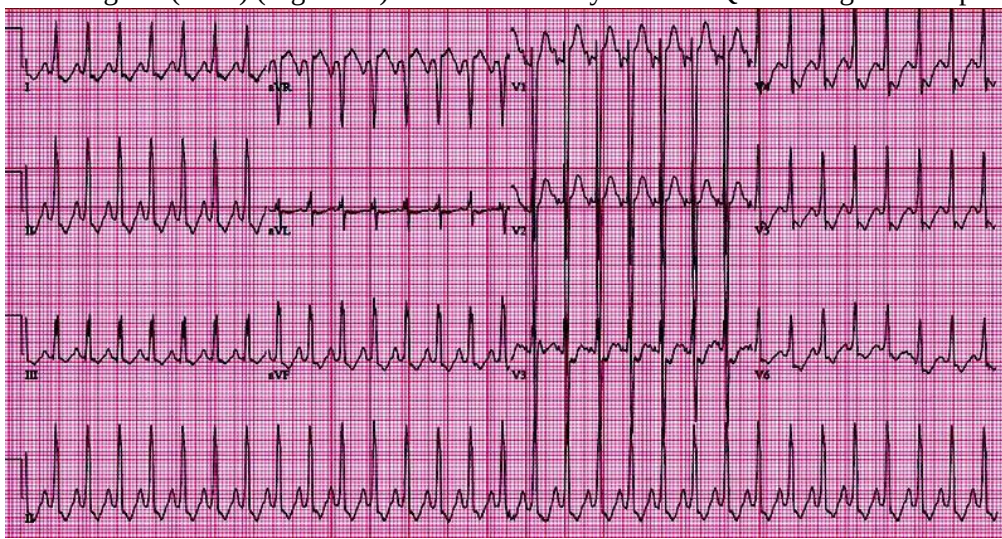


Figure 10: Resting ECG. There is sinus tachycardia (VR 140/min) with left ventricular hypertrophy with strain.

Transthoracic Echocardiography:

Transthoracic echocardiographic examination was carried out using the MyLab X7 4D XStrain system (Esaote, Italy) with a pediatric transducer. A sequential segmental analysis was performed through standard imaging windows, including subcostal, parasternal long-axis (LX), parasternal short-axis (SX), apical four-chamber (4CH), apical five-chamber (5CH), and suprasternal views.

M-mode Echocardiography:

M-mode echocardiography of both the left and right ventricles was obtained, and the corresponding measurements are summarized in Table 3 and Figure 11.

Table 3: Calculations of M-mode echocardiography.

Measurements	LV	RV
IVS d	2.9 mm	5.2 mm
ID d	34.2 mm	6.3 mm
PW d	5.0 mm	5.2 mm
IVS s	3.6 mm	4.6 mm
ID s	29.8 mm	5.5 mm
PW s	10.1 mm	5.7 mm
EF	28 %	30 %
% FS	13 %	12 %
EDV	48.2 ml	0.581 ml
ESV	34.5 ml	0.404 ml
SV	13.7 ml	0.176 ml
Mass	29 g	4 g

IVS, interventricular septum, ID, internal dimension; PW, posterior wall, d, diastole; s, systole; FS, fractional shortening; EDV, end-diastolic volume; ESV, end systolic volume; SV, stroke volume; EF, ejection fraction.

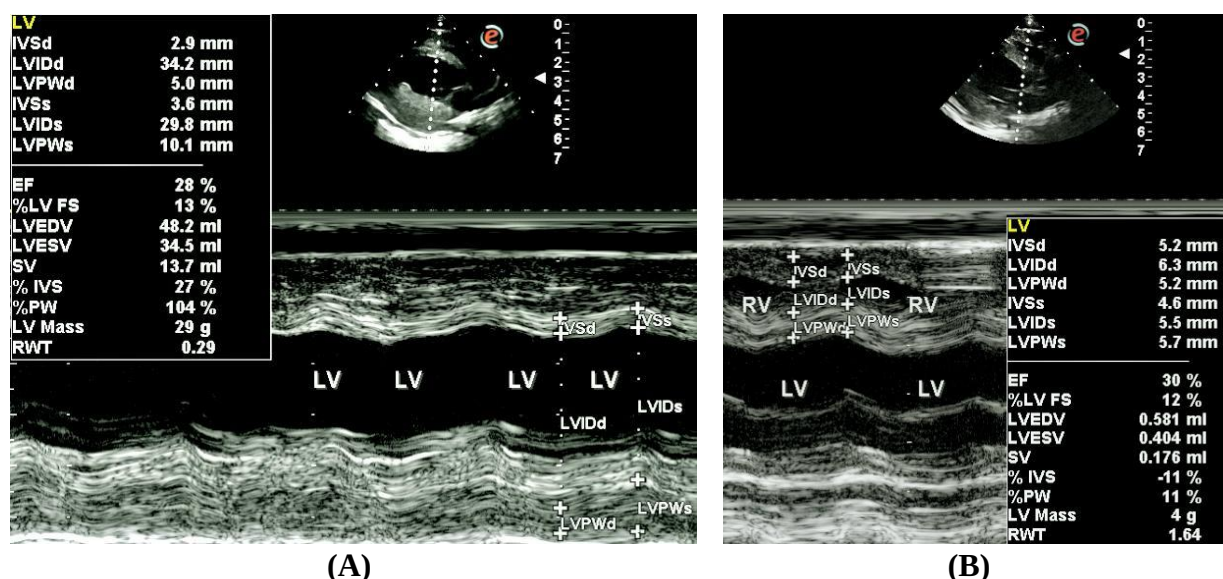


Figure 11: M- mode estimation of LV and RV indices - demarcated marked dilatation of LV, slit like RV with severely reduced bivenricular systolic function. LVEF & RVEF was 28 % & 30 %, respectively and LV mass & RV mass were 29 g & 4 g, respectively.

Summary of M-mode echocardiography:

The M-mode assessment demonstrated a dilated left ventricle with a slit-like right ventricle and markedly impaired biventricular systolic function. The LVEF and RVEF were 28% and 30%, respectively, while the LV and RV masses measured 29 g and 4 g, respectively.

2-Dimensional-Transthoracic Echocardiography:

Transthoracic echocardiography (TTE) was systemically performed by the sequential segmental approach (SSA) and the echocardiographic characteristics which were documented are enumerated below (Figure 12-21):

- Levocardia
- Situs solitus
- Atrioventricular concordance
- Ventriculo-arterial concordance

- **Concordant d-bulboventricular loop**
- **Normally related great arteries**
- **Left aortic arch**
- **Confluent pulmonary arteries**
- **Normal systemic and pulmonary venous drainage.**
- Dilated LV with global hypokinesia.
- Conspicuous dyssynchronised motion of LV.
- The LV posterior and lateral wall is distinctly divided into compacted and non compacted layers. The compacted portion is significantly thickened.
- Striking, multiple trabeculations and deep intertrabecular recesses were recognized on the posterior and lateral wall of LV.
- LVNC criteria (Chin, Jenni and Gebhard) photographs
- Mitral regurgitation (trace)
- Dilated LV, slit like RV.
- Biventricular concentric hypertrophy with severely reduced systolic functions.
- LVEF and RVEF were 28 % and 30 %, respectively.
- There was no evidence of ASD, VSD, PDA, COA, , AS, PS.



Figure 12: Situs solitus. Subcostal echocardiographic view showing the liver and inferior vena cava positioned on the right side, while the aorta and spleen are located on the left side. IVC, inferior vena cava; AO, aorta.



Figure 13: TTE in the LX view a salient LV non compacted zone of post wall is demonstrated. lv, left ventricle; la, left atrium; mv, mitral valve; ao, aorta; vs, ventricular septum; rv, right ventricle.

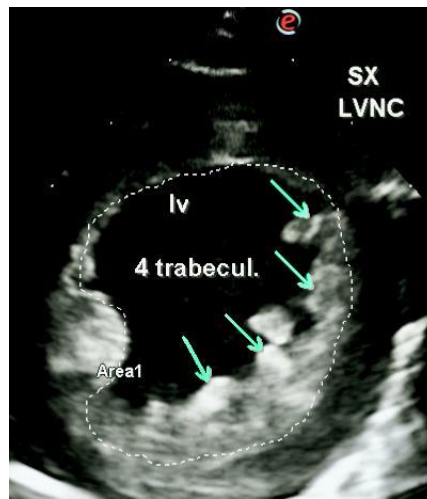


Figure 14: TTE in SX view depicted a large area of non compaction in the lateral wall of LV. At least 4 prominent trabeculations were delineated. LVNC, left ventricular non compaction; lv , left ventricle; trabecul, trabeculations.

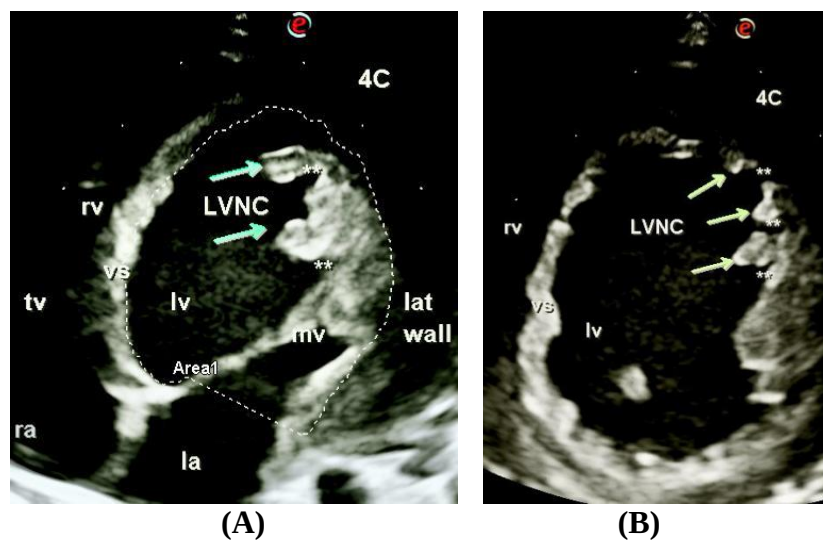


Figure 15: TTE in 4C view. Two images of apical 4C view are shown to highlight the presence of lv non compaction. Distinctive trabeculations and recesses were illustrated. LVNC, lv non compaction; lv, left ventricle; vs, ventricular septum; ** depict recesses; mv, mitral valve; la, left atrium; ra, right atrium.

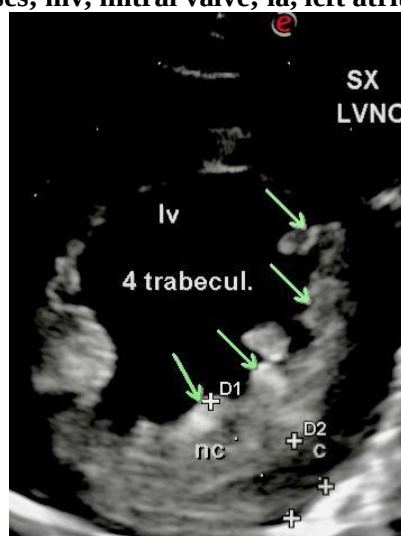


Figure 16: Chin criteria of LVNC - In the diastolic, SX view $c/nc + c$ ratio = < 0.37 c, compacted myocardium; nc, noncompacted myocardium.

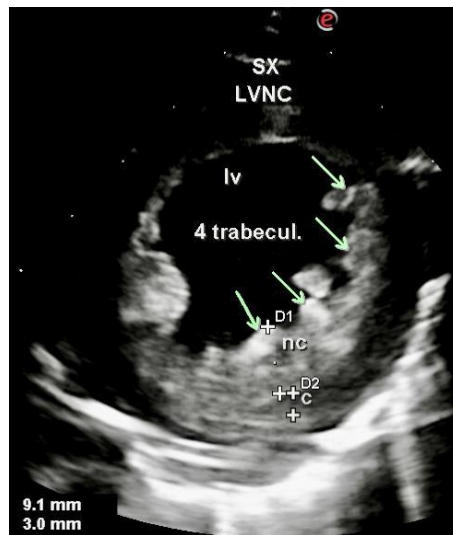


Figure 17: Jenni criteria of LVNC - In the systolic SX view nc/c ratio = 3.03. nc, noncompacted myocardium; c, compacted myocardium.

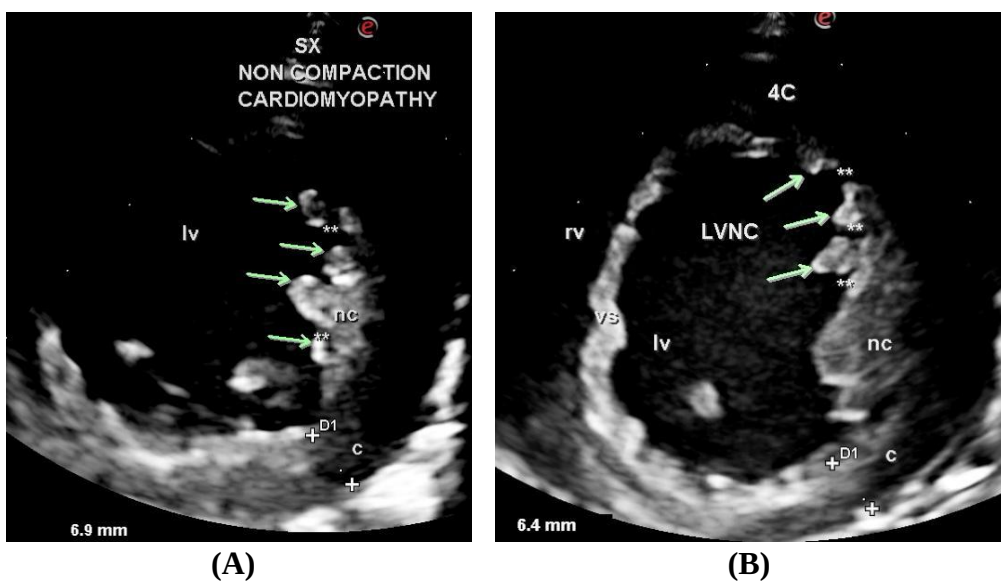


Figure 18: Gebhard criteria of LVNC. (A) In the systolic SX view thickness of compacted myocardium is 6.9 mm, and (B) In the apical 4C view the systolic thickness of compacted myocardium was 6.4 mm. c, compacted myocardium; LVNC, left ventricular noncompaction; lv , left ventricle; rv , right ventricle.

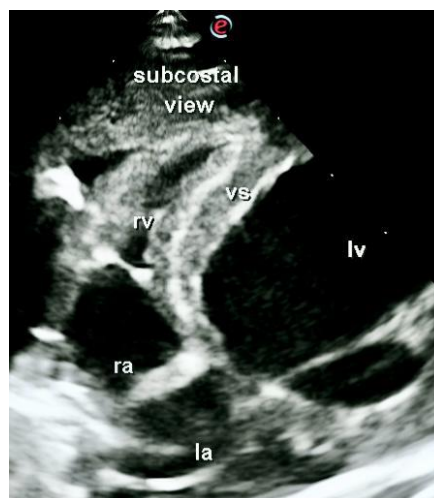


Figure 19: In the subcostal view a considerably dilated LV with a slit like RV was portrayed. rv, right ventricle; vs, ventricular septum; lv, left ventricle; la, left atrium; ra, right atrium.

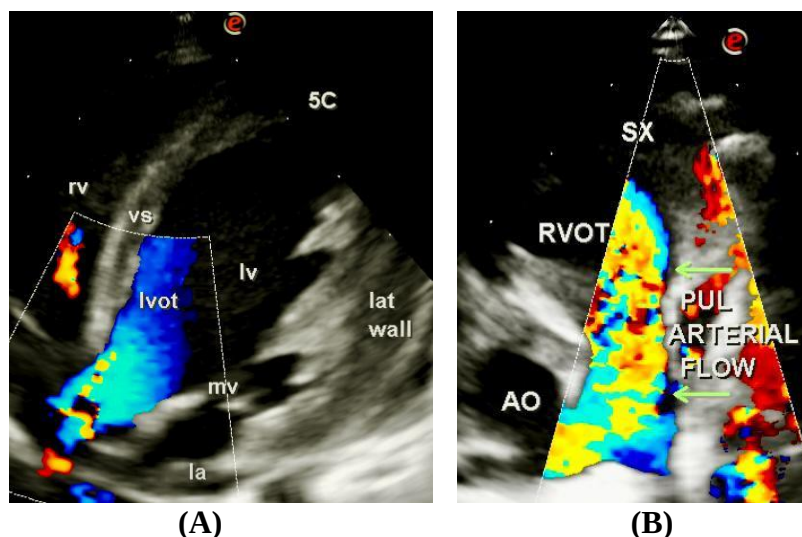


Figure 20: Color flow mapping (CFM) across left and right outflow tracts. The CFM was normal across both outflow tracts without any obstruction. lvot, left ventricular outflow tract; vs, ventricular septum; lv, left ventricle; mv, mitral valve; la, left atrium; RVOT, right ventricular outflow tract; AO, aorta.

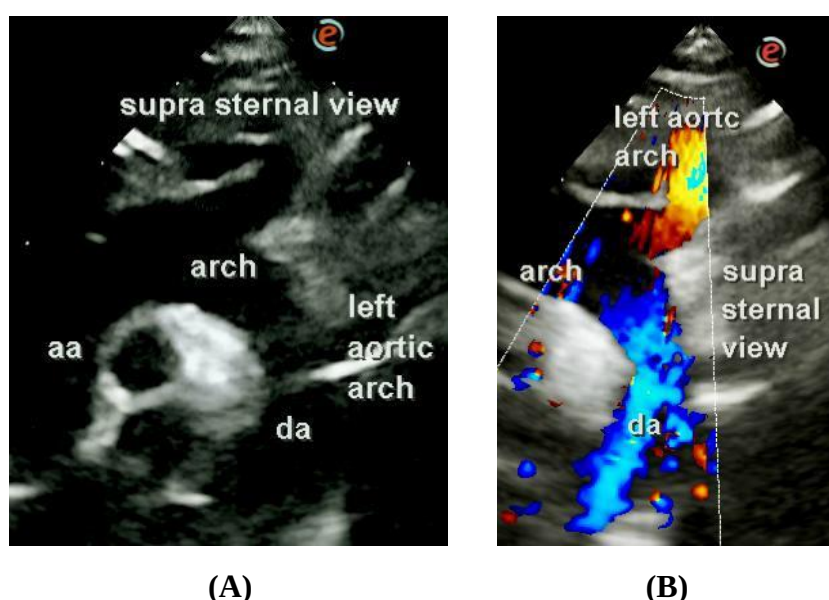


Figure 21: Suprasternal view depicting left aortic arch. (A) without and (B) with color flow mapping. aa, ascending aorta; da, descending aorta.

Summary of Color flow doppler echocardiography:

Color flow Doppler assessment identified extensive non-compacted regions along the posterior and lateral walls of the left ventricle, associated with marked LV dilation and significant dyssynchronous motion, leading to a severely reduced LVEF of 28%. Concentric hypertrophy of both ventricles was also observed. Despite the right ventricle appearing slit-like, the RVEF was significantly reduced at 30%.

DISCUSSION:

Noncompaction in children:

In pediatric patients, LVNC is most commonly linked to congenital anomalies, although cases of isolated

cardiomyopathy in neonates have also been reported [21]. The estimated prevalence of LVNC in children is approximately 0.14% [21]. Similar to adults, the condition primarily involves the left ventricle, but the right ventricle and interventricular septum may also be affected [21]. Morphologically, pediatric LVNC can present in various forms, ranging from a dilated to a hypertrophic ventricular appearance [22].

Clinical presentation in children may include chest pain, heart murmurs, abnormal heart sounds, electrocardiographic abnormalities, and/or echocardiographic findings [20,23]. The reported incidence of embolic events secondary to LVNC in this population varies from 0% to 38%, with the risk increasing in the presence of severe systolic dysfunction

[24]. McMahon et al. documented reduced tissue Doppler velocities in children with LVNC compared to age-matched controls [22]. Diagnosing isolated LVNC can be challenging, particularly when coexisting congenital heart defects are present [25]. If unrecognized or left untreated, LVNC may result in sudden cardiac death, especially in pediatric patients [25].

Approach to the Diagnosis of LVNC:

Accurate differential diagnosis of LVNC can be challenging, as several conditions may mimic its features [26]. To aid in this process, we propose a diagnostic flowchart (Figure 22) that emphasizes the critical role of imaging findings in establishing the diagnosis.

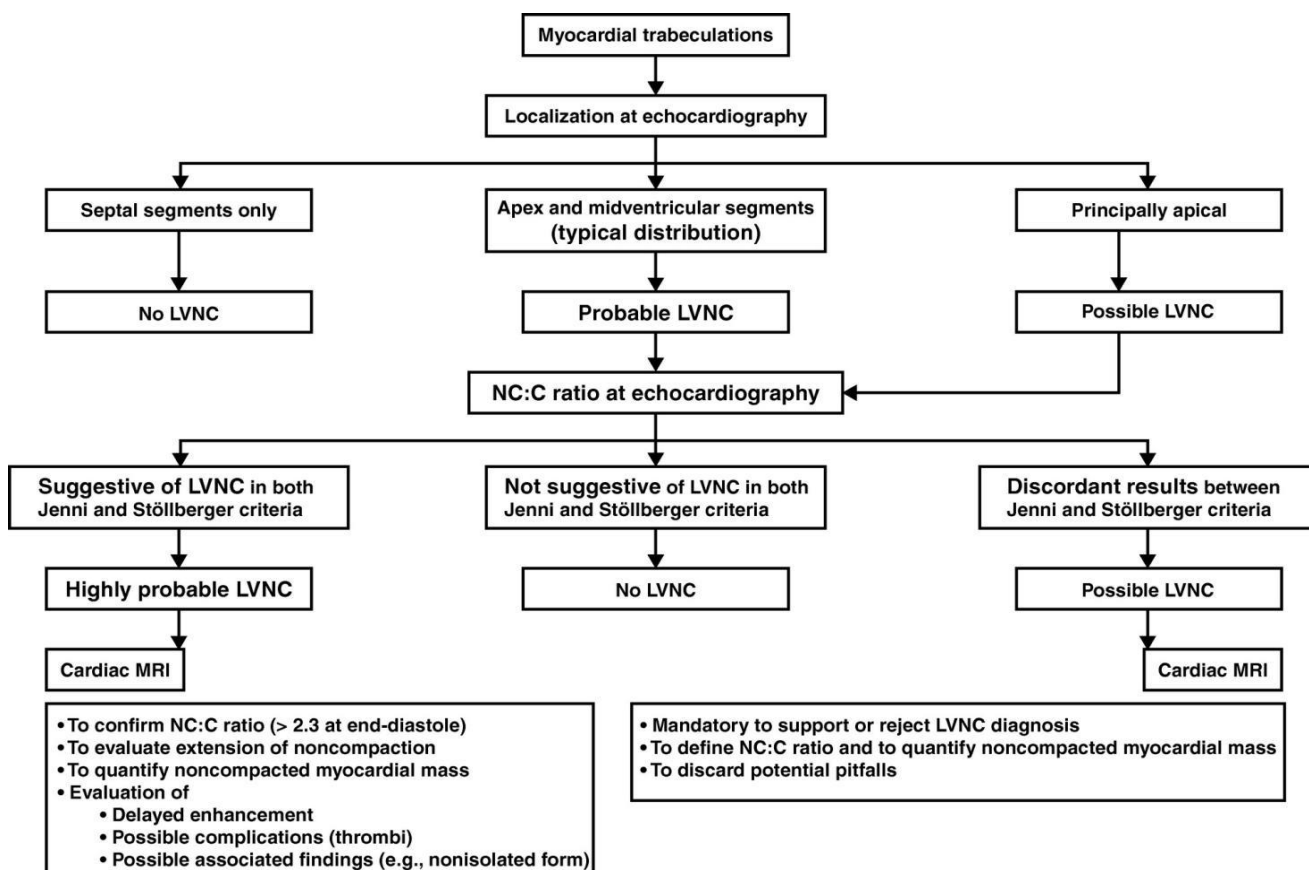


Figure 22: Proposed diagnostic flowchart for LVNC based on imaging findings. The initial step involves identifying the location of myocardial trabeculations; in LVNC, these are typically found in the apical or midventricular segments. If trabeculations are confined solely to the septal segments, LVNC can generally be excluded. Once an appropriate distribution of trabeculations is identified, the noncompacted-to-compacted (NC:C) ratio should be calculated. Given ongoing debate regarding echocardiographic criteria, it is recommended to assess the NC:C ratio using both end-systolic short-axis images (Jenni criteria) and end-diastolic four-chamber images (Stöllberger criteria). If the NC:C ratio is not indicative of LVNC on either view, the diagnosis can be excluded, potentially obviating the need for cardiac MRI. Conversely, if the NC:C ratio is suggestive of LVNC on both imaging planes, the diagnosis is highly probable, and cardiac MRI should be performed to evaluate the extent of noncompaction, quantify non-compacted myocardial mass, detect potential complications such as ventricular thrombi, and identify associated findings suggestive of a non-isolated form of LVNC. In cases where the NC:C ratio suggests LVNC but trabeculations are primarily apical, short-axis images may not be perpendicular to the left ventricular long axis, producing a false appearance of prominent trabeculations mimicking LVNC. In such instances, cardiac MRI is preferred for NC:C assessment in the four-chamber view, where measurements are perpendicular to the compacted myocardium.

Two-dimensional echocardiography (2DE) diagnosis of LVNC:

Two-dimensional echocardiography (2DE) remains the most widely utilized method for diagnosing LVNC. However, recent studies indicate that diagnosis is frequently delayed due to challenges in clearly identifying the characteristic features [11]. Often, multiple 2DE examinations are necessary before a definitive diagnosis can be established (Figure 23).

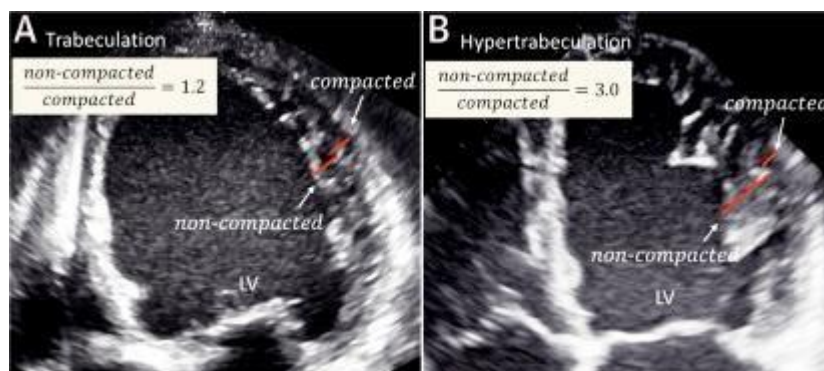


Figure 23: Echocardiographic four-chamber views differentiating prominent trabeculation from hypertrabeculation. (A) Four-chamber echocardiographic view of a patient with dilated cardiomyopathy showing prominent trabeculations in the LV apex and lateral wall; criteria for LVNC are not met in this case. (B) Four-chamber echocardiographic view of a patient with typical LVNC, demonstrating hypertrabeculation in the LV apex and lateral wall.

While 2DE provides excellent visualization of the left ventricular cavity, it may be insufficient for comprehensive assessment of the entire myocardium and endocardium. The characteristic prominent trabeculations in LVNC can extend into the LV cavity in a manner that is difficult to capture with the planar imaging inherent to 2DE. These limitations may result in misinterpretation of trabeculations as “false tendons” and can lead to underestimation of the extent of noncompaction. Consequently, the diagnosis of LVNC via 2DE is often delayed, sometimes requiring multiple examinations. In a study by Ichida et al. [27], over half of the pediatric patients had their diagnosis initially missed.

Color Doppler echocardiography Diagnosis of LVNC:

In some cases, the affected myocardial segments may exhibit hypokinesia, distinguishing them from certain forms of apical cardiomyopathy that can mimic LVNC. Differential echocardiographic considerations include prominent normal myocardial trabeculations, false tendons and aberrant bands, cardiac tumors, and left ventricular apical thrombi. In such scenarios, color Doppler echocardiography is particularly valuable, as it can demonstrate direct blood flow from the ventricular cavity into the deep intertrabecular recesses a feature absent in other echocardiographic abnormalities thereby aiding in the differentiation of LVNC [28] (Figure 24).

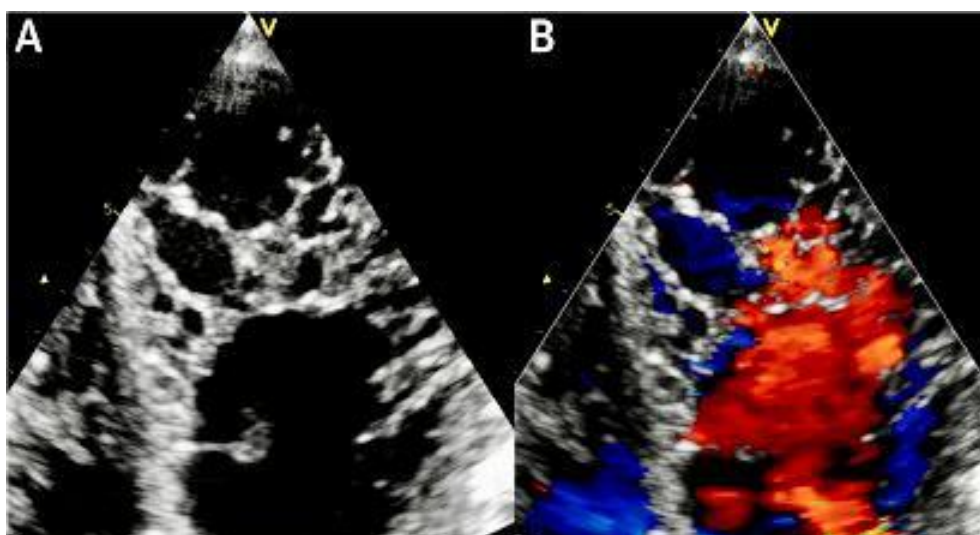


Figure 24: Echocardiographic and color Doppler images from a patient with LVNC. (A) Echocardiographic four-chamber view optimized to demonstrate noncompaction in the LV apex. (B) The same view with color Doppler, illustrating blood flow into the intertrabecular recesses from the LV cavity.

Three-dimensional echocardiography (3DE) Diagnosis of LVNC:

Three-dimensional echocardiography (3DE) enables precise diagnosis, detailed characterization, and assessment of the extent of myocardial involvement in noncompaction cardiomyopathy, as demonstrated in all four patients reported by Baker et al. [29]. This technique allows simultaneous visualization of the entire trabecular projections and intertrabecular recesses, clearly delineating the boundary between compacted and non-compacted left ventricular myocardium. Wall motion abnormalities across large

myocardial segments can also be evaluated concurrently.

Unlike 2DE, 3DE acquires pyramid-shaped volumetric datasets encompassing the entire left ventricle, permitting sectioning along user-defined planes with unlimited angles and orientations. Suspected intracavitary trabeculations can be tracked in multiple directions from base to apex, facilitating comprehensive visualization of each trabeculation. The deep intertrabecular recesses, as well as the distinction between the prominent non-compacted myocardium and the thin compacted layer, are well demonstrated using this modality [29] (Figures 25, 26).

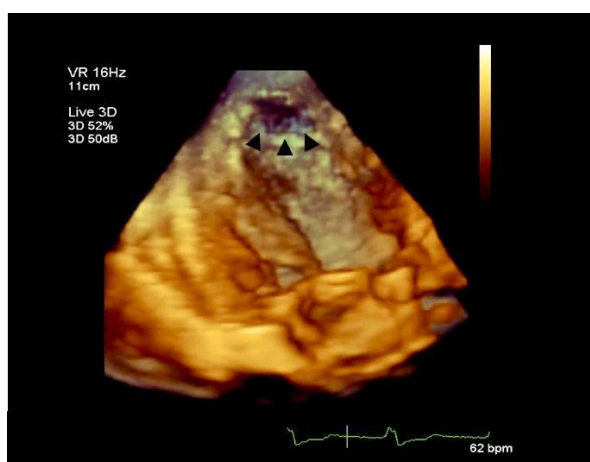


Figure 25: Real-time three-dimensional echocardiography showing the characteristic “honeycomb appearance” (arrowheads) in the apical lateral wall of our patient (Case 1).

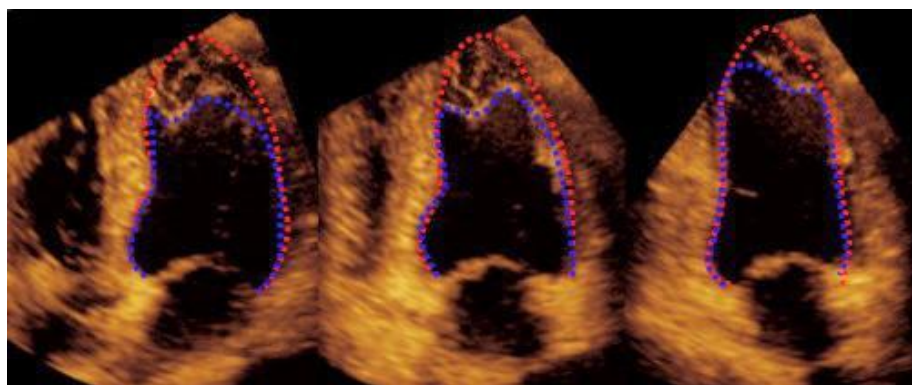


Figure 26: Owing to its high spatial resolution and precise volumetric capabilities, three-dimensional echocardiography enables accurate assessment of the extent of non-compacted myocardium and facilitates the identification of patients with LVNC.

Contrast echocardiography Diagnosis of LVNC:

Conventional echocardiography remains the primary diagnostic tool for LVNC. In typical cases, diagnosis is straightforward; however, subtle or incomplete noncompaction may be challenging to identify, necessitating consideration of differential diagnoses such as prominent normal myocardial trabeculations,

hypertrophic cardiomyopathy, dilated cardiomyopathy, and left ventricular apical thrombus. Conventional echocardiography can also be limited in patients with obesity or pulmonary disease due to poor acoustic windows.

Contrast-enhanced echocardiography, using various contrast agents, is increasingly employed to improve endocardial border delineation [30,31]. In situations where standard echocardiographic images are

suboptimal or the diagnosis is uncertain, contrast echocardiography can provide valuable clarification. This technique is minimally invasive, avoids additional

patient discomfort, and can reduce both time and costs (Figure 27).

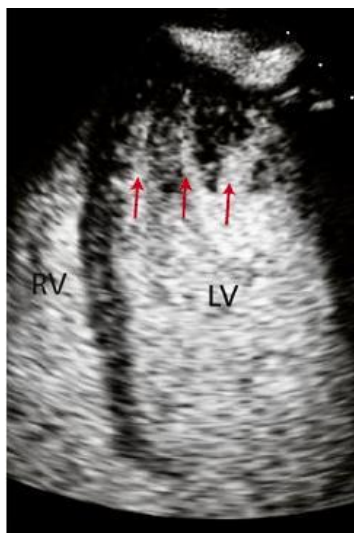


Figure 27: LVNC - Contrast echocardiography. Apical four chamber views in a patient with LV noncompaction, illustrating the value of contrast to delineate the deep recesses in the noncompacted LV apex (red arrows).

Differential diagnosis of LVNC:

In over 80% of patients, LVNC primarily affects the apical and midventricular segments of the inferior and lateral walls [10,32], whereas involvement of the midventricular anterior wall, basal segments, and interventricular septum is comparatively rare [10]. The segmental distribution is clinically significant, as prominent trabeculations can also be observed in hypertrophied myocardium [33].

Although echocardiography remains the preferred diagnostic modality for LVNC, alternative techniques including contrast ventriculography [34], computed tomography [34], and cardiac magnetic resonance imaging (MRI) [35] have been utilized. MRI correlates well with echocardiography regarding the localization and extent of noncompaction and is particularly useful when echocardiographic image quality is suboptimal [36]. Furthermore, differences in MRI signal intensity within non-compacted myocardium may help identify substrates for potentially lethal arrhythmias [37]. Despite technical advancements enhancing MRI's diagnostic value, echocardiography continues to be regarded as the gold standard for LVNC detection. In most instances, cardiac MRI provides no additional information beyond echocardiography, except for the identification of thrombi concealed within the sponge-like LVNC myocardium [38]. MRI offers advantages of reduced operator dependency and less susceptibility to technical limitations, although no large-scale comparative studies between the two modalities have been conducted [38].

Affected myocardial segments in LVNC may occasionally exhibit hypokinesia, distinguishing them from certain forms of apical cardiomyopathy that can

mimic LVNC. Differential echocardiographic considerations include prominent normal trabeculations, false tendons and aberrant bands, cardiac tumors, and left ventricular apical thrombi. In such cases, color Doppler analysis demonstrating direct blood flow from the ventricular cavity into deep intertrabecular recesses is particularly helpful, as this feature is absent in other echocardiographic abnormalities [10,38].

CONCLUSION:

Echocardiography remains the first-line and most widely employed imaging modality for diagnosing LVNC. Enhanced awareness and understanding of LVNC are essential to improve diagnostic accuracy within echocardiography laboratories. However, existing echocardiographic criteria are highly variable and have largely been derived from studies with limited patient populations.

Advanced imaging techniques including three-dimensional echocardiography, contrast-enhanced imaging to better delineate endocardial borders (particularly in the apical region for patients with suboptimal acoustic windows), and myocardial strain analysis via speckle-tracking offer promising avenues to increase the diagnostic precision of echocardiography in LVNC patients. The clinical adoption of these methods has grown in recent years; nevertheless, continuous refinement and validation of echocardiographic criteria remain necessary.

The observed high prevalence of LVNC features in low-risk populations, such as athletes and healthy Black individuals, suggests that increased LV trabeculations and intertrabecular recesses may represent an adaptive

response to chronically elevated preload. Accordingly, integrating clinical evaluation, electrocardiographic assessment, and a multimodality imaging approach including both echocardiography and cardiac MRI is recommended.

Future directions should focus on tailoring diagnostic criteria to account for ethnic variations, incorporating functional assessments, and leveraging multicenter and international collaborations that integrate genetic data to achieve a more precise and individualized diagnosis of LVNC.

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