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Case Reports

The Forgotten Spongy Myocardium: Clinical Trajectory of Left Ventricular Noncompaction Cardiomyopathy in an Asymptomatic Adult

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Abstract

Background: Left Ventricular Noncompaction (LVNC) is a rare cardiomyopathy characterized by a thin compacted epicardial layer and an extensive noncompacted endocardial layer with prominent trabeculations and deep intertrabecular recesses that communicate with the Left Ventricular (LV) cavity. The classic triad of complications includes chronic heart failure, ventricular arrhythmias, and systemic embolic events. At present, evidence-based management guidelines remain limited.

Case Illustration: We report a 42-year-old man with LVNC, initially detected as an incidental Left Bundle Branch Block (LBBB) on Electrocardiogram (ECG) during a routine medical checkup. Although he remained asymptomatic, LV Ejection Fraction (LVEF) progressively declined, accompanied by rising N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP) levels. Coronary artery disease was excluded by coronary computed tomography angiography. Given worsening LV systolic function over 2 years, Cardiac Magnetic Resonance (CMR) demonstrated an LVNC phenotype consistent with cardiomyopathy. Guideline-Directed Medical Therapy (GDMT) for heart failure was initiated, along with oral anticoagulation for primary prevention of LV thrombus. After medication optimization, LVEF improved markedly, and NT-proBNP normalized.

Conclusions: This case illustrates the value of comprehensive evaluation and multimodality imaging in patients with unexplained LBBB, even when asymptomatic. Early diagnosis, phenotype-guided treatment, and longitudinal surveillance may help prevent clinical progression and future heart-failure, arrhythmic, or thromboembolic complications.

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Introduction

Left Ventricular Noncompaction (LVNC) is a rare cardiomyopathy characterized by prominent trabeculations and deep intertrabecular recesses, producing a spongy myocardial appearance.[1] The American Heart Association (AHA) classifies LVNC as a primary genetic cardiomyopathy, whereas the European Society of Cardiology (ESC) considers it an unclassified cardiomyopathy.[2-3] LVNC predominantly involves the left ventricle and features a two-layered myocardium, with a thick noncompacted trabecular layer over a thinner compacted layer. It can present at any age, including mid-life, but reported prevalence in imaging series is low (approximately 0.01-0.26%), and it is identified more often in men, potentially due in part to under-recognition of asymptomatic cases.[4-5] Patients with LVNC are at increased risk of progression to heart failure, life-threatening ventricular arrhythmias, and thromboembolic events related to intracardiac thrombus formation.[6]

Diagnosing LVNC can be difficult because clinical presentation ranges from incidental findings to advanced heart failure, and Electrocardiography (ECG) findings are non-specific.[7-8] Common ECG features include increased QRS complex voltage, Left Bundle Branch Block (LBBB), and ST-T wave abnormalities. Arrhy thmias are also frequent.[9-10] LVNC is typically suspected on Transthoracic Echocardiography (TTE) and can be confirmed by Cardiac Magnetic Resonance imaging (CMR). On echocardiography, LVNC is classically recognized by a two-layered myocardium, consisting of a thin compacted epicardial layer and a thicker noncompacted endocardial layer with prominent trabeculations and deep intertrabecular recesses communicating with the LV cavity. Established echocardiographic frameworks include the Jenni, Chin, and Stöllberger criteria, which assess compacted and noncompacted myocardial layers, trabeculation morphology, and intertrabecular recesses. On CMR, prominent trabeculations commonly involve the apical and mid-lateral left ventricular (LV) segments, and a maximal Noncompacted-to-Compacted (NC/C) myocardial thickness ratio >2.3 at end-diastole fulfills the Petersen criterion.[11] While echocardiography remains the first-line, widely accessible imaging modality, CMR provides complementary value through higher spatial resolution and more reproducible quantification of myocardial layers, particularly when echocardiographic windows are limited or diagnostic uncertainty persists.[12]

In addition to diagnostic challenges, evidence guiding treatment strategies for LVNC remains limited, and current approaches are largely individualized. Management typically focuses on associated clinical manifestations such as heart failure, atrial fibrillation, thromboembolism, and arrhythmias.[3,13] Prognosis is highly variable and is influenced by the timing of detection and the development of complications, including ventricular arrhythmias, heart failure, and thromboembolic events. In adults, mortality rates of up to 50% within 6 years after diagnosis have been reported.[6]

In Indonesia, the relatively limited availability of magnetic resonance imaging, together with documented barriers to clinical CMR implementation in Asia and other low- and middle-income settings, likely contributes to delayed diagnosis and under-recognition of cardiomyopathies that require advanced imaging, including LVNC.[14-15] To our knowledge, published case reports describing the clinical course of LVNC in Indonesia remain limited. Herein, we present an asymptomatic adult patient with LVNC who developed LV systolic dysfunction and complete LBBB.

Case Illustration

A 42-year-old Chinese man with long-standing hypertension and metabolic syndrome presented to our outpatient clinic in October 2024 for further evaluation of previously identified complete LBBB and reduced LV Ejection Fraction (LVEF). He remained asymptomatic during activities of daily living and denied exertional dyspnea, chest pain, palpitations, syncope, presyncope, orthopnea, paroxysmal nocturnal dyspnea, or peripheral edema. He denied tobacco or alcohol use and reported no known family history of cardiomyopathy, heart failure, sudden cardiac death, unexplained syncope, arrhythmia, device implantation, or premature cardiovascular disease. Vital signs and physical examination were unremarkable.

The initial abnormality was incidentally detected in March 2021, when a routine medical checkup showed complete LBBB on ECG (Figure 1). Coronary artery disease was excluded by Coronary Computed Tomography Angiography (CCTA). Standard laboratory tests and Transthoracic Echocardiography (TTE) were unremarkable, with preserved LVEF (62%). Over time, LVEF declined to 46% by October 2023, without evidence of LV thrombus, and he remained asymptomatic. No specific therapy was initiated at that time.

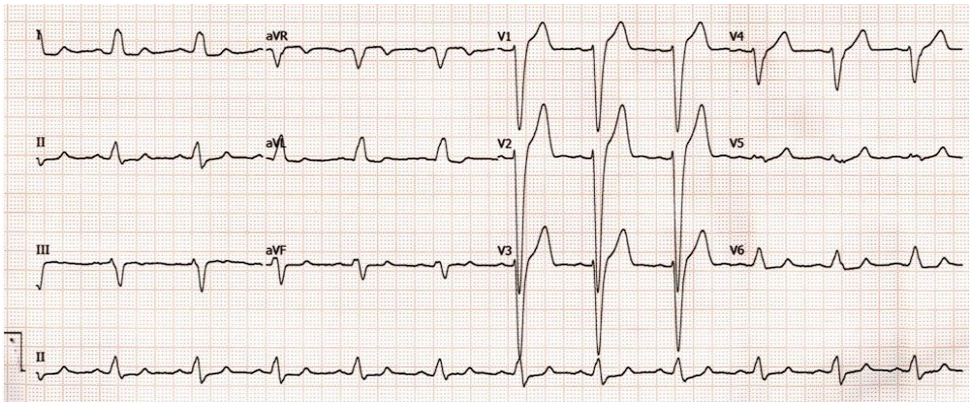

Figure 1. ECG showing complete LBBB.

At presentation in October 2024, repeat TTE showed a markedly reduced LVEF of 25% with global LV hypokinesia (Figure 2), accompanied by an elevated N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP) level of 531 pg/mL. Repeat CCTA during the same period demonstrated only mild, non-obstructive coronary plaque. CMR revealed an LVNC phenotype with a maximal NC/C ratio of 3.5, fulfilling the Petersen criterion (>2.3), together with global hypokinesia and no late gadolinium enhancement or evidence of fibrosis or myocardial inflammation.

Guideline-Directed Medical Therapy (GDMT) was initiated, consisting of candesartan 16 mg once daily, bisoprolol 2.5 mg twice daily, spironolactone 25 mg once daily, dapagliflozin 10 mg once daily, rosuvastatin 20 mg once daily, and metformin 1,000 mg once daily. Given the markedly reduced LVEF and extensive trabeculation, low-dose edoxaban (30 mg once daily) was prescribed for primary prevention of thromboembolism. Candesartan was subsequently switched to sacubitril/valsartan 100 mg twice daily to promote LV reverse remodeling.

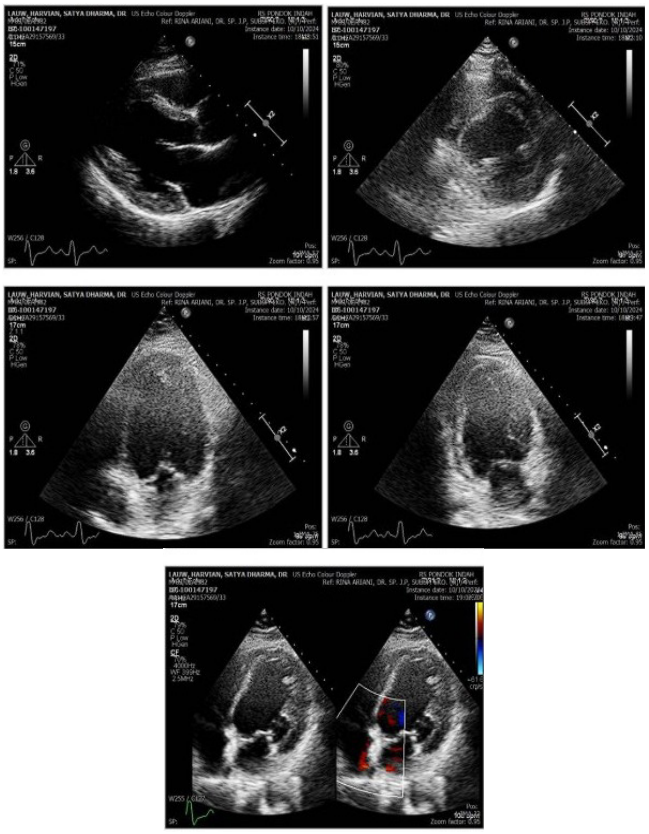

Figure 2. Initial TTE in October 2024.

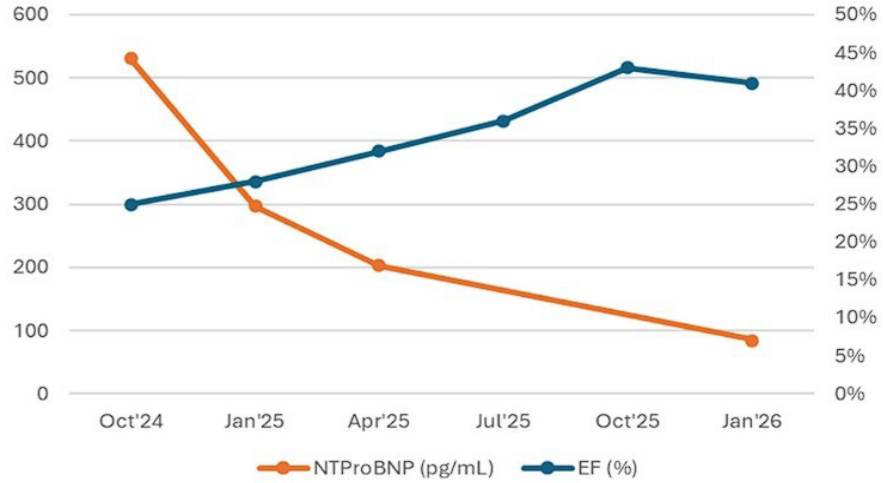

Figure 3. Trends in LVEF and NT-proBNP during GDMT.

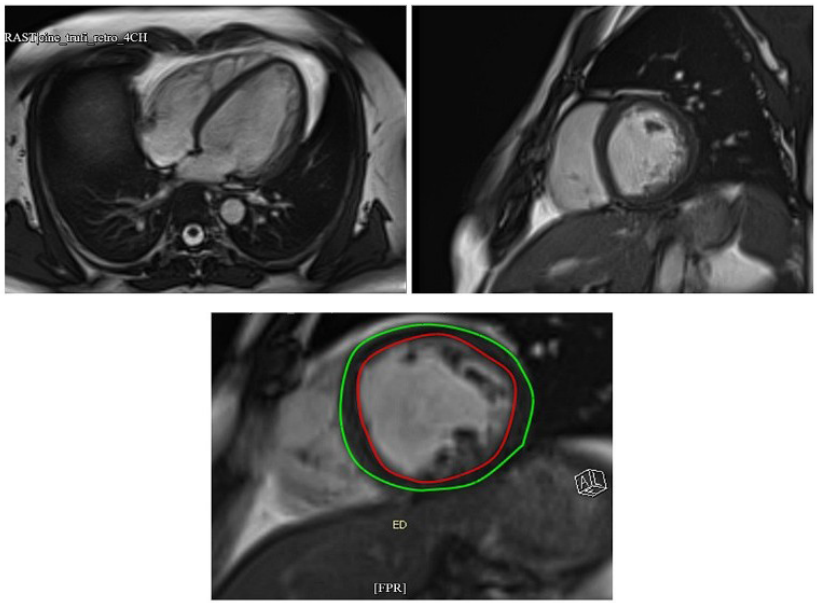

Figure 4. A follow-up CMR in October 2025 showed improved LV systolic function following GDMT optimization.

Guideline-Directed Medical Therapy (GDMT) was initiated, consisting of candesartan 16 mg once daily, bisoprolol 2.5 mg twice daily, spironolactone 25 mg once daily, dapagliflozin 10 mg once daily, rosuvastatin 20 mg once daily, and metformin 1,000 mg once daily. Given the markedly reduced LVEF and extensive trabeculation, low-dose edoxaban (30 mg once daily) was prescribed for primary prevention of thromboembolism. Candesartan was subsequently switched to sacubitril/valsartan 100 mg twice daily to promote LV reverse remodeling.

Over the subsequent 12 months, the patient remained clinically asymptomatic, with progressive improvement in LVEF and a decline in NT-proBNP levels (Figure 3). Serial ECGs and 24-hour Holter monitoring showed no significant arrhythmias. Repeat CMR in October 2025 (Figure 4) demonstrated partial reverse remodeling, with LVEF of 43%, an NC/C ratio of 3.3, and no LV thrombus. Cardiopulmonary exercise testing showed adequate functional capacity, with a peak VO_2 of 19.9 mL/kg/min and a normal hemodynamic response to exercise. Given the improvement in LVEF and persistent absence of symptoms, GDMT was continued, and consideration of cardiac resynchronization therapy was deferred.

Discussion

LVNC describes a myocardial phenotype characterized by prominent trabeculations and deep intertrabecular recesses, and may be associated with LV dysfunction, arrhythmias, and thromboembolic complications.[16] LVNC remains diagnostically challenging because reported prevalence varies by population, imaging modality, and diagnostic criteria. Meta-analyses suggest higher prevalence estimates when CMR criteria are applied, raising concern for overdiagnosis of physiological hypertrabeculation and potential overtreatment.[17] Clinical features that prompt careful evaluation are therefore important. Although ECG abnormalities are common and often non-specific, observational data indicate that intraventricular conduction disturbances, including LBBB, are frequently encountered in LVNC cohorts.[18] In our patient, incidentally detected complete LBBB preceded recognition of LV systolic impairment, and diagnosis was delayed until serial imaging showed progressive LVEF decline and CMR confirmed LVNC.

CMR was pivotal for confirmation and risk stratification. CMR-based morphologic thresholds, such as an NC/C ratio >2.3 at end-diastole (Petersen criterion), are widely used, with the understanding that no single criterion is definitive and that imaging findings require clinical correlation. Other CMR-based approaches, including Jacquier, Stacey, Captur/fractal analysis, and indexed non-compacted mass criteria, have also been proposed; however, these criteria vary in diagnostic yield and are not uniformly applied in routine clinical reporting.[12] In our patient, CMR demonstrated marked trabeculation (NC/C 3.5) and global hypokinesia, without late gadolinium enhancement or evidence of myocardial inflammation. Late gadolinium enhancement, as a surrogate for myocardial fibrosis, has prognostic relevance across cardiomyopathies and in LVNC. Meta-analysis data in LVNC cohorts have associated late gadolinium enhancement with a higher risk of major adverse cardiovascular events, supporting its role as a clinically useful risk marker.[19] Accordingly, absence of these CMR features may be favorable, although reduced LVEF remains a key determinant of outcome in LVNC.[20-21]

In the absence of robust randomized evidence and LVNC-specific therapeutic algorithms, management is typically individualized and guided by the dominant clinical phenotype (heart failure, arrhythmia burden, and thromboembolic risk).[22] In patients with LV systolic dysfunction, GDMT for heart failure with reduced ejection fraction remains the cornerstone, including renin-angiotensin system inhibition, beta-blocker, mineralocorticoid receptor antagonist, and Sodium-Glucose Cotransporter-2 (SGLT2) inhibitor.[2,10,23] The patient in this report received comprehensive GDMT (Angiotensin Receptor-Neprilysin Inhibitor [ARNI], beta-blocker, mineralocorticoid receptor antagonist, and SGLT2 inhibitor), with objective improvement in LVEF and NT-proBNP over 12 months and preserved functional status. Case-based reports and reviews suggest that ARNI therapy may be associated with reverse remodeling in LVNC, including improvement in LVEF and ventricular dimensions.[7] In parallel, a prospective cohort of 30 LVNC patients on stable GDMT reported that addition of an SGLT2 inhibitor was associated with LV reverse remodeling, improved hemodynamic indices, and NT-proBNP reduction at 12 months, particularly in less advanced disease.[24] Taken together, these observations support early initiation and careful optimization of GDMT in patients with LVNC and LV systolic dysfunction.

Device therapy decisions in LVNC should follow standard indications based on LVEF, symptoms, and electrical or mechanical dyssynchrony, rather than trabeculation morphology alone.[2,10,23] Persistent LBBB raised consideration of resynchronization strategies; however, because LVEF improved with GDMT and no sustained ventricular arrhythmias were documented, cardiac resynchronization therapy and implantable cardioverter-defibrillator implantation were deferred. Continued rhythm surveillance remains reasonable, given the association of LVNC with atrial and ventricular arrhythmias and the potential for late arrhythmic events.[16]

Thromboembolism prevention remains a key management question in LVNC and is an important consideration in this case. Thromboembolic events occur in LVNC, and systematic review data suggest a higher risk in the presence of atrial fibrillation and/or LV systolic dysfunction.[25] In the absence of atrial fibrillation, intracardiac thrombus, or prior embolic events, prophylactic anticoagulation is not established by randomized evidence. Nonetheless, expert opinion and observational experience support considering anticoagulation in selected higher-risk LVNC phenotypes, particularly in patients with markedly reduced LVEF, after individualized bleeding-risk assessment.[9] Therefore, reduced-dose edoxaban in this case should be interpreted as a risk-mitigation strategy in the setting of severe systolic dysfunction and extensive trabeculation, rather than a standard-of-care approach.

Several limitations should be acknowledged. First, LVNC diagnostic criteria are heterogeneous and overlap with physiological trabeculation; in this patient, the diagnosis was supported by concordant imaging findings and a trajectory of progressive LV dysfunction. Echocardiographic assessment of quantitative LVNC indices, including the Jenni, Chin, and Stöllberger criteria, was limited by suboptimal acoustic windows, which precluded reliable retrospective measurement of myocardial layer thickness and trabeculation ratios. Therefore, TTE was used primarily for serial assessment of LV systolic function, while LVNC morphology was quantified and diagnostically supported by CMR using the Petersen criterion.[12,17] Second, the mechanism underlying LV systolic dysfunction remains uncertain. Obstructive coronary artery disease was not supported by CCTA, and CMR showed no late gadolinium enhancement, myocardial inflammation, or scar, making ischemic, inflammatory, or scar-mediated cardiomyopathy less likely. Because complete LBBB preceded overt systolic dysfunction, LBBB-related dyssynchrony may have contributed, although LVNC-associated cardiomyopathy remains a plausible primary substrate.[26] Although prior TTE documented preserved LVEF in 2021, no earlier CMR or LVNC-focused echocardiographic assessment was available; therefore, we could not determine whether the noncompaction morphology was already present, became more apparent with LV remodeling, or was previously under-recognized. Third, genetic testing and structured family evaluation were not performed. This is relevant because LVNC may show familial clustering, with prior studies reporting cardiomyopathy among relatives in approximately 17% to 50% of affected patients.[27] Accordingly, genetic counseling and clinical screening of first-degree relatives, including focused history, ECG, and echocardiography, should be considered, particularly because affected relatives may be asymptomatic.[3,16] Longer follow-up is needed to confirm the durability of reverse remodeling, arrhythmia-free status, and thromboembolic risk over time.

Conclusion

This case illustrates that otherwise unexplained complete LBBB may serve as an early clinical clue to an underlying cardiomyopathy, such as LVNC, before overt clinical manifestations develop. Timely, comprehensive evaluation, including multimodal imaging, is important for early detection and diagnostic confirmation, enabling earlier initiation of appropriate therapy and potentially improving clinical outcomes. Although evidence for LVNC-specific therapy remains limited, phenotype-guided management with contemporary GDMT was associated with substantial improvement in LVEF and NT-proBNP in this patient. Continued follow-up remains important to monitor the durability of reverse remodeling and to reduce the risk of future heart-failure progression, ventricular arrhythmias, and thromboembolic complications.

List of Abbreviations

AHA	American Heart Association
ARNI	Angiotensin Receptor-Neprilysin Inhibitor
CCTA	Coronary Computed Tomography Angiography
CMR	Cardiac Magnetic Resonance
ECG	Electrocardiogram
ESC	European Society of Cardiology
GDMT	Guideline-Directed Medical Therapy
LBBB	Left Bundle Branch Block
LV	Left Ventricular
LVEF	Left Ventricular Ejection Fraction
LVNC	Left Ventricular Noncompaction
MeSH	Medical Subject Headings
NC/C	Noncompacted-to-compacted myocardial thickness ratio
NT-proBNP	N-terminal pro-B-type Natriuretic Peptide
SGLT2	Sodium-Glucose Cotransporter-2
TTE	Transthoracic Echocardiography
VO_2	Oxygen uptake

Ethical Clearance

Ethics committee or institutional review board approval was not required for publication of this single case report in accordance with local institutional policy. The report was prepared using anonymized clinical information. Written informed consent for publication of the clinical details and any accompanying images was obtained from the patient. Identifying information has been removed to protect privacy

Publication Approval

The corresponding author confirms that all authors reviewed and approved the final version of the manuscript and consent to its submission. The

work is original and is not under consideration by another journal, nor has it been published previously, in whole or in part.

Authors Contributions

SSI, JWH, and HSD collected and curated the clinical data, performed the literature review, and prepared the first draft. LPS contributed to the clinical management and helped frame the clinical narrative. DYG provided electrophysiology and ECG/Holter interpretation. LPS and DYG provided final revisions and critical intellectual input. All authors contributed to the manuscript revision, approved the final version, and agreed to be accountable for the accuracy and integrity of the work.

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The authors have no acknowledgements to declare.

Conflict of Interest

All authors declare that they have no competing interests (financial or non-financial) relevant to this work.

Availability of Data and Materials

All data relevant to this case are included in the manuscript. Additional de-identified information may be made available by the corresponding author upon reasonable request, subject to patient consent and institutional/privacy restrictions.

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Generative AI and AI-Assisted Technologies in the Writing Process

Generative AI (ChatGPT 5.4 Pro, OpenAI) was used only to improve language, grammar, and clarity. The tool was not used to generate or modify clinical data, analyses, interpretations, or conclusions, and no identifiable patient information was entered. All authors review and take full responsibility for the final content.

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