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CLINICAL PRESENTATION OF LEFT VENTRICULAR NONCOMPACTION DIAGNOSED IN ADULTS

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ABSTRACT

Left ventricular noncompaction (LVNC) is a rare, morphologically and genetically heterogeneous form of cardiomyopathy, the pathogenesis, classification, and clinical significance of which remain subjects of ongoing debate. The aim of this review is to present the current state of knowledge regarding the classification, pathophysiology, diagnostics, genetic determinants, clinical presentation, and therapeutic management of LVNC. The European Society of Cardiology (ESC) classifies LVNC as an unclassified cardiomyopathy, whereas the American Heart Association (AHA) considers it a genetically determined cardiac disorder. The characteristic morphology arises from abnormal compaction of the left ventricular trabeculae during embryogenesis, although an acquired or adaptive origin of these changes is also possible. Diagnostic evaluation relies primarily on echocardiography and cardiac magnetic resonance imaging; however, the lack of uniform morphological criteria contributes to both overdiagnosis and underdiagnosis. From a genetic perspective, sarcomeric gene mutations predominate and are associated with more severe clinical outcomes. Clinical manifestations include heart failure, arrhythmias, and thromboembolic complications, with reduced left ventricular ejection fraction being the principal prognostic factor. Therapeutic management follows general heart failure treatment strategies, thromboembolism prevention, and implantation of cardiac devices; in advanced cases, heart transplantation or mechanical circulatory support may be considered. An integrated diagnostic approach and the MOGE(S) classification system may serve as a foundation for future standardization.

KEYWORDS

Left Ventricular, Noncompaction, LVNC, Cardiomyopathy, Echocardiography, Magnetic Resonance Imaging, Heart Failure, Arrhythmias

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Classification and Epidemiology of Left Ventricular Noncompaction

Left ventricular noncompaction (LVNC) is a disorder of left ventricular wall maturation. According to the ESC classification, it is categorized as an unclassified cardiomyopathy [1], whereas the AHA includes it among genetically determined cardiomyopathies [2]. The clinical and genetic presentations of LVNC exhibit marked heterogeneity [3]. Some authors—including Monserrat— emphasize that LVNC may represent a phenotypic variant of other cardiomyopathies, such as hypertrophic, restrictive, or dilated forms, and that currently used diagnostic criteria often overlap [4].

The description of this morphology has a long history. As early as 1926, Grant noted the characteristic structure of the myocardial wall [5], and in 1932, Bellet and Gouley provided the first post-mortem description [6]. A major milestone occurred in 1984, when Engberding and Bender documented the first echocardiographically confirmed case in an adult woman [7]. The term *"isolated non-compaction left ventricular myocardium"* was later popularized by Chin [5].

The prevalence of LVNC remains difficult to determine precisely. Echocardiographic studies have reported rates ranging from 0.014% to 0.24% [1,8]. A Swiss cohort from 1984–1998 found a prevalence of 0.014%, which is now considered underestimated, particularly among patients with symptoms of heart failure [9]. In populations with reduced ejection fraction, the frequency of diagnosis increases: up to 3.7% in individuals with LVEF ≤45% [10] and as high as 8.7% with LVEF <35% [11]. In a series by Ronderos comprising 10,857 echocardiographic examinations, 26 cases of LVNC were identified (0.24%); the mean age was 52.6 years, women predominated (16/10), and isolated LVNC was found in 24 patients, while two had concomitant ASD II. Among asymptomatic individuals with preserved LVEF, the prevalence was only 0.05%, whereas in patients with idiopathic DCM it reached as high as 24% [12]. Despite differences among studies, overall LVNC is observed more frequently in men (56–82%) [13].

Structural Morphology of LVNC

The phenotype of LVNC is characterized by a two-layered structure within the affected segments of the left ventricular wall: an inner, trabeculated (endocardial) layer containing deep recesses that communicate with the ventricular cavity, and an outer, thin, compacted (epicardial) layer. Together, these features produce a characteristic "spongy" appearance [14]. The abnormalities most commonly involve the apex, as well as the apical and mid-ventricular segments of the inferior and lateral walls. The left ventricle may be enlarged, with evidence of hypertrophy and both systolic and diastolic dysfunction [15]. The morphological spectrum is broad and heterogeneous [16]. Additional findings reported in some cases include facial dysmorphism (prominent forehead, strabismus, low- set ears, mandibular hypoplasia) and coexisting Wolff–Parkinson–White syndrome—primarily in pediatric patients, as it is rare in adults [17,9].

Diagnostic Methods and Diagnostic Criteria

The diagnosis of LVNC is primarily based on transthoracic echocardiography (TTE) and cardiac magnetic resonance imaging (CMR), with computed tomography (CT), transesophageal echocardiography (TEE), contrast-enhanced echocardiography, and, less frequently, ventriculography or intracardiac echocardiography (ICE), serving as supplementary tools [18–21]. Echocardiographic abnormalities are present in all patients with LVNC [22].

Classical Echocardiographic Criteria

Chin (1990):

Presence of a bilayered myocardial structure (a compacted epicardial layer and a non-compacted endocardial layer), with an X/Y ratio ≤ 0.5 (X – distance from the epicardial surface to the base of the intertrabecular recess; Y – distance from the epicardial surface to the apex of the trabeculations). Measurements should be obtained in the parasternal short-axis view during late diastole (PSAX) [17].

Jenni:

Measurements should be performed in end-systole in the parasternal short-axis view. A non- compacted to compacted layer ratio (N/C) > 2 must be present, along with color Doppler evidence of blood flow into the intertrabecular recesses and the absence of other structural cardiac abnormalities in cases of isolated LVNC [23].

Stöllberger/Finsterer:

These authors proposed criteria based on the presence of at least three trabeculations located apically to the papillary muscles, exhibiting echogenic characteristics identical to the myocardium, with visible blood flow between them. Additionally, a non-compacted to compacted layer ratio (N/ C) > 2 in late diastole is required. Evaluation is recommended primarily in apical views, with the use of additional projections permitted to improve image quality [24].

Limitations of Echocardiography

Echocardiographic criteria for LVNC were developed on small patient cohorts and differ in measurement timing and imaging projections; concordance among these criteria is poor. In heart

failure (HF) populations, 24% of patients met at least one definition, whereas only 30% fulfilled all three. Chin's criteria were the most sensitive but also yielded the highest rate of false positives. Up to 8% of healthy individuals (predominantly Black participants) met at least one criterion, suggesting potential overdiagnosis in this population [25,26].

Echocardiography is operator-dependent; imaging planes that are not orthogonal to the left ventricular axis tend to overestimate trabeculation, and interobserver agreement is approximately 67% [27]. No definitive "gold standard" exists, and the risk of both overdiagnosis and underdiagnosis remains significant [28].

Advances in Echocardiographic Imaging

Assessment of left ventricular (LV) function in patients with excessive trabeculation is crucial, and modern echocardiographic techniques—such as ultrasound contrast agents, three-dimensional (3D) imaging, tissue Doppler imaging, strain analysis, speckle-tracking echocardiography, and evaluation of LV twist mechanics—provide significant diagnostic support [29]. Contrast agents enhance visualization of intertrabecular recesses, particularly in the apical region, and require harmonic imaging with a high frame rate and a moderate mechanical index (MI ≈ 0.3) [30].

Under normal conditions and in dilated cardiomyopathy (DCM), the LV base rotates clockwise while the apex rotates counterclockwise. In contrast, in LVNC both segments rotate in the same direction, which may facilitate diagnosis [31,32]. Rudolecka demonstrated that the degree of apical rotation correlates with systolic function, and that its reduction—assessed using speckle-tracking techniques—may aid in both

diagnosis and risk stratification [33]. Compared with hypertrophic cardiomyopathy, patients with LVNC exhibit more severely impaired longitudinal strain in apical segments and reduced apical rotation [34].

An additional distinguishing criterion is a compacted myocardial layer thickness < 8 mm in systole within segments exhibiting non-compaction, which improves differentiation between LVNC and conditions such as aortic stenosis or nonspecific endocardial hypertrabeculation [35].

Cardiac Magnetic Resonance (CMR)

CMR provides superior visualization of all myocardial segments and is recommended when echocardiographic image quality is poor or when false-negative results are suspected (e.g., in young patients with left bundle branch block, LBBB) [36,37].

Most Commonly Used CMR Criteria

Petersen:

Petersen proposed an MRI-based criterion based on the ratio of non-compacted to compacted myocardial thickness in end-diastole (N/C > 2.3), demonstrating a sensitivity of 86% and specificity of 99% [38].

Jacquier:

Jacquier suggested that the mass of the non-compacted myocardium should exceed 20% of the total LV mass in end-diastole [39–41].

Stacey:

Stacey developed another set of criteria, based on an NC/C ratio > 2 measured in the short-axis view during end-systole. This parameter showed better correlation with the risk of heart failure and adverse cardiac events than end-diastolic measurements or the Petersen and Jacquier criteria [42].

However, studies by Ivanov did not confirm an association between LVNC diagnosed using any of the existing CMR criteria and the occurrence of adverse clinical events during a 7-year follow-up [43].

Significance of the Extent of Non-Compaction and Late Gadolinium Enhancement (LGE)

The presence of ≥5 non-compacted segments has been associated with lower right ventricular ejection fraction (RVEF) and a higher incidence of ventricular arrhythmias [46]. However, other studies indicate that prognosis is more strongly determined by ventricular dilation and reduction in left ventricular ejection fraction (LVEF) than by the mere “extent” of trabeculation. LGE is present in approximately 40% of patients, exhibits heterogeneous patterns, and correlates with ventricular function and clinical course; it may also reveal subclinical disease in individuals with preserved LVEF [47–50].

Cardiac MRI has also been used to evaluate longitudinal fiber function in non-compacted myocardium. Analysis of mitral annular plane systolic excursion (MAPSE) in the medial and lateral segments demonstrated that impairment of longitudinal fiber contractility significantly contributes to reduced global systolic function in these ventricles [51].

Computed tomography (CT) serves as an alternative to CMR, primarily in patients with contraindications to MRI. CT additionally allows assessment of coronary anatomy during the same examination [19,20].

Diagnostic Perspective

Excessive trabeculation alone does not necessarily indicate abnormal embryogenesis— it may instead reflect remodeling secondary to hemodynamic overload [52]. Therefore, the diagnosis of LVNC should integrate morphology and clinical context (symptoms, reduced LVEF), family history, and abnormal ECG findings to minimize false-positive results. In patients who meet morphological criteria but have normal LV geometry and function, individualized follow-up is recommended, taking into account variable genetic penetrance and age-dependent phenotypic expression [53,54].

Etiology

The etiology of LVNC remains incompletely understood. The most widely cited genetic theory proposes disrupted embryogenesis. In the normally developing heart, trabeculae form a spongy myocardial structure until the coronary circulation develops (weeks 5–8 of gestation). Myocardial compaction begins around week 10 and progresses from base to apex and from epicardium to endocardium [55]. Abnormal arrest of this process leads to persistent trabeculation and formation of a bilayered ventricular wall. A genetic basis is further supported by the frequent coexistence of LVNC with congenital heart defects [56–61].

However, the embryologic hypothesis does not explain acquired and potentially reversible forms of LVNC. A non-compaction–like phenotype has been described in athletes [62–65]. Gati reported increased trabeculation in 18.3% of 1,146 athletes, with 8.3% fulfilling diagnostic criteria for LVNC, suggesting an adaptive response to left ventricular overload [66–69]. An acquired phenotype has also been observed in

pregnancy: approximately 25% of pregnant women exhibit increased trabeculation, which regresses in 73% after delivery [70]. Hypertrabeculation has also been documented in chronic kidney disease and sickle-cell anemia [29,71]. Prenatal and postnatal imaging studies have shown that the LVNC phenotype may develop only after birth. These findings do not exclude a genetic substrate, as sarcomeric disorders also demonstrate delayed phenotypic expression, and similar mutations have been identified in HCM, DCM, and LVNC [5].

Non-compaction may coexist with hypertrophic, dilated, or restrictive cardiomyopathy phenotypes [72–74], as well as numerous genetic syndromes, including Emery–Dreifuss muscular dystrophy, and Barth, Leopard, Noonan, Turner, DiGeorge, and Pierre Robin syndromes, along with various metabolic and mitochondrial diseases [75–80]. The presence of LVNC in patients with neuromuscular disorders is associated with worse prognosis [81]. Cases have also been reported of LVNC coexisting with isolated apical hypoplasia, leading to combined systolic and diastolic dysfunction [82].

Genetic Determinants

Left ventricular non-compaction may occur sporadically or in familial forms, most commonly with autosomal dominant inheritance, and less frequently as X-linked or autosomal recessive; familial occurrence is estimated at 18–50% [5,83], which justifies screening—primarily via echocardiography—among relatives of affected individuals [84]. In patients with LVNC, numerous mutations have been described in genes encoding sarcomeric proteins, cytoskeletal elements, mitochondrial proteins, and Z-line components [85]. Experimental studies (including mouse models with FKBP-12 deficiency) indicate that disturbances in signaling pathways regulating proliferation and apoptosis may lead to non-compaction [86–88]. Likewise, mutations in Notch1 signaling genes —both in humans and animal models—result in excessive trabeculation and abnormal myocardial development [89–91]. However, no single mutation is specific to isolated LVNC.

In a large analysis by van Waning involving 327 patients with LVNC, genetic testing (a 45-gene panel) distinguished three groups: genetically confirmed cases (30%), likely familial cases (16%), and sporadic cases (54%) [92]. The most frequent mutations involved MYH7, TTN, and MYBPC3. The presence of pathogenic variants correlated with impaired systolic function and a higher risk of major adverse cardiac events (MACE); this association applied to mutation-positive patients but was not observed in sporadic forms. Similar findings were reported in a Chinese cohort by Li, where mutations (primarily in TTN, MYH7, MYBPC3, and DSP) were associated with worse ejection fraction and an increased risk of death or heart transplantation (50% vs. 23.5%) [93]. The authors concluded that a positive genotype is an independent predictor of adverse prognosis.

The OMIM registry continually updates the list of gene variants associated with the LVNC phenotype; however, no mutation has yet been identified as definitively responsible for a pure, isolated form of this cardiomyopathy without overlap with other phenotypes or congenital defects [94].

Classifications, Taxonomy, and Clinical Types of LVNC

For many years, cardiomyopathies were categorized primarily by their morphological phenotype, a strategy that from today's perspective has proven insufficient: phenotype does not always reflect the genetic substrate, is often of limited value for risk stratification, and does not facilitate early diagnosis in asymptomatic relatives. Advances in genetic research therefore support an integrated approach that combines the morphological–functional cardiac profile with information on extracardiac involvement, etiology, and specific genetic variants. This conceptual framework is encompassed in the MOGE(S) system [95].

In clinical practice, LVNC exists along a continuum—from forms with normal left ventricular dimensions and preserved systolic function, through genetically determined variants (associated with chromosomal aberrations, single-gene mutations, or coexisting congenital heart defects or other cardiomyopathies), to nongenetic or acquired, potentially reversible phenotypes observed in elite athletes, during pregnancy, and in the context of chronic kidney disease, sickle cell anemia, or hematologic disorders [69,96,97]. Noncompaction confined to the left ventricle may also coexist with LV dilation and dysfunction, meet simultaneous diagnostic criteria for DCM/HCM/RCM/

ARVC, or involve the right ventricle. Some authors, such as Stöllberger, question the usefulness of the term *"isolated LVNC"*, noting that most patients demonstrate additional cardiac morphological abnormalities [98].

Conversely, Oechslin and Jenni propose that after obtaining a detailed clinical history (syncope, heart failure symptoms, family history, arrhythmias), ECG analysis, imaging, and genetic testing, patients should be categorized into one of three groups: cardiomyopathy with LVNC, LVNC associated with pathological remodeling, or LVNC representing a physiological and reversible adaptive response [99]. Such a narrative, multidimensional classification better reflects the true phenotypic heterogeneity and—crucially—helps link the diagnosis to prognosis and therapeutic decision-making.

Clinical Presentation

Left ventricular noncompaction (LVNC) may remain asymptomatic and can be diagnosed at any age—from the fetal period to over 90 years of age—most commonly during imaging performed for unrelated indications, screening examinations, or in patients with heart failure of unclear etiology [8, 9, 100, 101]. The absence of pathognomonic symptoms means that the interval between the onset of complaints and final diagnosis may span many months or even years. Three major categories of clinical manifestations predominate: symptoms of heart failure (dyspnea, fatigue, palpitations, chest pain), arrhythmias—including sudden cardiac death—and thromboembolic complications [6, 8, 102–104].

The presence of deep intertrabecular recesses, particularly in the setting of reduced LVEF, favors thrombus formation and systemic embolization; reported rates vary widely (0–38%) [5, 8, 71, 83, 105]. In a large Japanese cohort (n = 272; median age 55 years, LVEF 35%), all-cause mortality reached 8.1% over 42 months, and the most frequent adverse event was hospitalization for heart failure (21.7%); event rates did not differ significantly between isolated LVNC and LVNC coexisting with other cardiac conditions [106].

Electrocardiographic abnormalities are detected in approximately 90% of patients, although they are nonspecific: QRS widening, signs of hypertrophy, repolarization disturbances, and pre-excitation; features suggestive of Brugada syndrome have also been described (≈3%) [107–109]. A positive T wave in aVR is associated with increased risk of arrhythmias, heart failure hospitalization, and mortality, and constitutes an independent marker of complex arrhythmic events [110,111]. Myocardial ischemia—assessed, among others, by SPECT—correlates with worse systolic function, LV dilation, and heightened neurohumoral activation [112].

Ventricular arrhythmias are of particular clinical relevance: they may occur in up to 62% of patients, often as monomorphic tachycardia, though polymorphic forms are also seen; the incidence of malignant tachyarrhythmias and aborted cardiac arrest reaches 38–47%, and that of sudden death 13–18% [8, 113–115]. Atrial fibrillation occurs with a frequency comparable to other cardiomyopathies (≈13–25%), although device-based monitoring may detect it far more often (up to 66%); risk increases in the presence of LBBB and fragmented QRS complexes. However, isolated LVNC has not been consistently demonstrated as an independent determinant of supraventricular arrhythmias [5, 20, 116–118].

Treatment

No dedicated, disease-specific guidelines for the management of LVNC currently exist; therefore, therapeutic decisions are guided by the clinical presentation—heart failure, ventricular arrhythmias, and thromboembolic complications—which informs the choice of pharmacological therapy and implantable devices. Although prospective trials are lacking, early initiation of guideline-directed

medical therapy for heart failure is recommended in asymptomatic patients with declining systolic function, as prognosis is determined by LV dysfunction rather than by the noncompaction phenotype itself. Prevention of thromboembolic events is essential: deep intertrabecular recesses promote thrombogenesis, and some authors advocate anticoagulation when LVEF < 40% [5]. Thromboembolic risk increases in the presence of atrial fibrillation or impaired systolic function [119]. Anticoagulation is also indicated when intracardiac thrombus is present, for secondary prevention, and in patients with atrial fibrillation [120,121].

Indications for ICD and CRT follow general cardiomyopathy guidelines. The efficacy of ICD therapy in LVNC has been demonstrated: in a study of 30 patients, appropriate shocks occurred in 37% over a 40-month follow-up [122–124]. CRT may improve NYHA functional class in patients with mechanical dyssynchrony and LVEF ≤ 35% [120]. Procedural risk is low, with lead dislodgement representing the most common complication [125].

In selected cases, surgical intervention may be considered; successful reconstruction of the left ventricle and valves has been reported in a patient with severe heart failure and intraventricular thrombi [126]. In advanced heart failure, referral for heart transplantation may be necessary [127], and mechanical circulatory support can serve as a bridge to transplant [128–130].

Summary

Left ventricular noncompaction (LVNC) is a heterogeneous entity with diverse genetic, morphological, and clinical substrates. Despite advances in imaging techniques and molecular genetics, diagnosis remains challenging due to the absence of unified diagnostic criteria. An integrated approach—encompassing morphological, functional, genetic, and clinical data—is essential for accurate assessment and optimal management. Further research is required to standardize diagnostic frameworks, refine classification systems, and individualize therapeutic strategies for patients with LVNC.

REFERENCES

1. Elliott, P., Andersson, B., Arbustini, E., Bilinska, Z., Cecchi, F., Charron, P., et al. (2008). Classification of the cardiomyopathies: A position statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *European Heart Journal, 29*, 270–276.
2. Maron, B. J., Towbin, J. A., Thiene, G., Antzelevitch, C., Corrado, D., Arnett, D., et al. (2006). Contemporary definitions and classification of the cardiomyopathies: An American Heart Association scientific statement. *Circulation, 113*, 1807–1816.
3. Paszkowska, A., & Ziółkowska, L. (2020). Kardiomiopatia z niescalenia mięśnia lewej komory. In A. Dobrzańska, Ł. Obrycki, & P. Socha (Red.), *Choroby rzadkie* (s. 324–326). Media Press.
4. Monserrat, L., Hermida-Prieto, M., Fernandez, X., Rodríguez, I., Dumont, C., Cazón, L., et al. (2007). Mutation in the alpha-cardiac actin gene associated with apical hypertrophic cardiomyopathy, left ventricular non-compaction, and septal defects. *European Heart Journal, 28*, 1953–1961.
5. Oechslin, E., & Jenni, R. (2011). Left ventricular non-compaction revisited: A distinct phenotype with genetic heterogeneity? *European Heart Journal, 32*, 1446–1456.
6. Udeoji, D. U., Philip, K. J., & Morrissey, R. P. (2013). Left ventricular noncompaction cardiomyopathy: Updated review. *Therapeutic Advances in Cardiovascular Disease, 7*, 260–273.
7. Engberding, R., & Bender, F. (1984). Identification of a rare congenital anomaly of the myocardium by two-dimensional echocardiography: Persistence of isolated myocardial sinusoids. *American Journal of Cardiology, 53*, 1733–1734.
8. Sarma, R. J., Chana, A., & Elkayam, U. (2010). Left ventricular noncompaction. *Progress in Cardiovascular Diseases, 52*, 264–273.
9. Hussein, A., Karimianpour, A., Collier, P., & Krasuski, R. A. (2015). Isolated noncompaction of the left ventricle in adults. *Journal of the American College of Cardiology, 66*, 578–585.
10. Sandhu, R., Finkelhor, R. S., Gunawardena, D. R., & Bahler, R. C. (2008). Prevalence and characteristics of left ventricular noncompaction in a community hospital cohort of patients with systolic dysfunction. *Echocardiography*. https://doi.org/10.1111/j.1540-8175.2007.00560.x
11. Grant, J. F., Harris, J., Helmcke, F., & Smart, F. (2017). Increased prevalence of left ventricular noncompaction in certain populations may confer increased risk of catastrophic events. *Journal of the American College of Cardiology, 69*, 853.
12. Ronderos, R., Avegliano, G., Borelli, E., Kuschnir, P., Castro, F., Sanchez, G., et al. (2016). Estimation of prevalence of the left ventricular noncompaction among adults. *American Journal of Cardiology, 118*, 901–905.
13. Vizzardi, E., Nodari, S., Metra, M., & Dei Cas, L. (2006). Non-compaction of the ventricular myocardium. *Heart International, 3–4*, 178–183.
14. Szczeklik, A., & Tendera, M. (Red.). (2009). *Kardiologia* (t. 1, s. 654–655). Medycyna Praktyczna.
15. Towbin, J. A., Lorts, A., & Jefferies, J. L. (2015). Left ventricular non-compaction cardiomyopathy. *The Lancet, 386*, 813–825.
16. Ezon, D., Maskatia, S., Jeewa, A., & Denfield, S. (2012). Morphologic heterogeneity in left ventricular noncompaction resulting in accessory left ventricular chambers. *Circulation: Heart Failure, 5*(6), 94–95. https://doi.org/10.1161/CIRCHEARTFAILURE.112.969469
17. Chin, T. K., Perloff, J. K., Williams, R. G., Jue, K., & Mohrmann, R. (1990). Isolated noncompaction of left ventricular myocardium: A study of eight cases. *Circulation, 82*, 507–513.
18. Rangin, K., Thampinathan, B., & Thavendiranathan, P. (2019). Imaging-specific cardiomyopathies: A practical guide. *Heart Failure Clinics, 15*, 275–295.
19. Bhat, T., Lafferty, J., Teli, S., Abou Rjaili, G., Olkovsky, Y., & Costantino, T. (2011). Isolated left ventricular noncompaction cardiomyopathy diagnosed by transesophageal echocardiography. *Clinical Medicine Insights: Cardiology, 5*, 23–27.
20. Carrilho-Ferreira, P., Almeida, A. G., & Pinto, F. J. (2014). Non-compaction cardiomyopathy: Prevalence, prognosis, pathology, genetics, and risk of cardioembolism. *Current Heart Failure Reports, 11*, 393–403.
21. Ficili, S., Pandozi, C., Galeazzi, M., Kol, A., Russo, M., Lavalle, C., et al. (2011). Noncompacted ventricular myocardium: Characterization by intracardiac echo. *Journal of Cardiovascular Medicine, 12*, 294–296. https://doi.org/10.2459/JCM.0b013e32833e5892
22. Konka, M., & Hoffman, P. (2008). Niescalenie mięśnia sercowego. In *Echokardiograficzna ocena chorób mięśnia sercowego* (s. 63–67). Via Medica.
23. Jenni, R., Oechslin, E., Schneider, J., Attenhofer Jost, C., & Kaufmann, P. A. (2001). Echocardiographic and pathoanatomical characteristics of isolated left ventricular non-compaction: A step towards classification as a distinct cardiomyopathy. *Heart, 86*, 666–671.
24. Stöllberger, C., & Finsterer, J. (2004). Left ventricular hypertrabeculation/noncompaction. *Journal of the American Society of Echocardiography, 17*, 91–100.

25. Kohli, S. K., Pantazis, A. A., Shah, J. S., Adeyemi, B., Jackson, G., McKenna, W. J., et al. (2008). Diagnosis of left ventricular non-compaction in patients with left ventricular systolic dysfunction: Time for a reappraisal of diagnostic criteria? *European Heart Journal, 29*, 89–95.
26. Val-Bernal, J. F., Garijo, M. F., Rodriguez-Villar, D., & Val, D. (2010). Non-compaction of the ventricular myocardium: A cardiomyopathy in search of a pathoanatomical definition. *Histology and Histopathology, 25*, 495–503. https://doi.org/10.14670/HH-25.495
27. Saleeb, S. F., Margosian, R., Spencer, C. T., Alexander, M. E., Smoot, L. B., Dorfman, A. L., et al. (2012). Reproducibility of echocardiographic diagnosis of left ventricular noncompaction. *Journal of the American Society of Echocardiography, 25*, 194–202.
28. Morcos, P., Andersson, U., & Adler, E. (2014). Left ventricular non-compaction: Current controversy and new insights. *Journal of Genetic Syndromes & Gene Therapy, 6*, 255. https://doi.org/10.4172/2157-7412.1000255
29. Mirea, O., Berceanu, M., Constantin, A., & Târtea, G. C. (2017). Non-compaction cardiomyopathy – brief review. *Journal of Mind and Medical Sciences, 4*, 115–124.
30. Porte, T. R., Aggarwal, S., Xie, F., & Morad, A. (2018). Unmasking of isolated non-compaction of the left ventricle with real time contrast echocardiography and intermediate mechanical index. *Journal of the American College of Cardiology, 71*(11, Suppl.), A2145.
31. van Dalen, B. M., Caliskan, K., Soliman, O. I., Nemes, A., Vletter, W. B., Ten Cate, F. J., & Geleijnse, M. L. (2008). Left ventricular solid body rotation in non-compaction cardiomyopathy: A potential new objective and quantitative functional diagnostic criterion? *European Heart Journal – Cardiovascular Imaging, 10*, 1088–1093.
32. Cortés, M., Oliva, M. R., Orejas, M., Navas, M. A., Rábago, R. M., Martínez, M. E., et al. (2016). Usefulness of speckle myocardial imaging modalities for differential diagnosis of left ventricular non-compaction of the myocardium. *International Journal of Cardiology, 223*, 813–818.
33. Rudolecka, J., Veiser, T., Plasek, J., Homza, M., & Fűrstová, J. (2014). Ventricular twist in isolated left ventricular noncompaction. *Cor et Vasa, 56*, 471–477.
34. Rao, M. S., & Devasia, T. (2018). Regional cardiac function in left ventricular non-compaction: Focus on strain and torsion analysis and differentiation from hypertrophic cardiomyopathy. *Indian Heart Journal, 70*, 14–23.
35. Gebhard, C., Stähli, B. E., Greutmann, M., Biaggi, P., Jenni, R., & Tanner, F. C. (2012). Reduced left ventricular compacta thickness: A novel echocardiographic criterion for non-compaction cardiomyopathy. *Journal of the American Society of Echocardiography, 25*, 1050–1057.
36. Thuny, F., Jacquier, A., Jop, B., Giorgi, R., Gaubert, J. Y., Bartoli, J. M., et al. (2010). Assessment of left ventricular non-compaction in adults: Side-by-side comparison of cardiac magnetic resonance imaging with echocardiography. *Archives of Cardiovascular Diseases, 103*, 150–159.
37. Khan, F., Fretz, A. E., Khan, S., Kono, A. T., & Gilstrap, L. G. (2019). Clinical predictors of discordant imaging between cardiac MRI and echocardiography in the diagnosis of left ventricular noncompaction cardiomyopathy. *Journal of Cardiac Failure, 25*, 39. https://doi.org/10.1016/j.cardfail.2019.07.109
38. Petersen, S. E., Selvanayagam, J. B., Wiesmann, F., Robson, M. D., Francis, J. M., Anderson, R. H., et al. (2005). Left ventricular non-compaction: Insights from cardiovascular magnetic resonance imaging. *Journal of the American College of Cardiology, 46*, 101–105.
39. Jacquier, A., Thuny, F., Jop, B., Giorgi, R., Cohen, F., Gaubert, J. Y., et al. (2010). Measurement of trabeculated left ventricular mass using cardiac magnetic resonance imaging in the diagnosis of left ventricular non-compaction. *European Heart Journal, 31*, 1098–1104. https://doi.org/10.1093/eurheartj/ehp595
40. García, G. B., Gonzales-Carrillo, J., Sanche, J. C., Sánchez, R. D., Espín, S. D., & Blanes, B. J. R. (2017). Performance of a new software tool for automatic quantification of left ventricular trabeculations. *Revista Española de Cardiología, 70*, 405–407.
41. Cai, J., Bryant, J. A., Le, T. T., Su, B., de Marvao, A., O'Regan, D. P., et al. (2017). Fractal analysis of left ventricular trabeculations is associated with impaired myocardial deformation in healthy Chinese. *Journal of Cardiovascular Magnetic Resonance, 19*, 102.
42. Stacey, R. B., Andersen, M. M., St Clair, M., Hundley, W. G., & Thohan, V. (2013). Comparison of systolic and diastolic criteria for isolated LV non-compaction in CMR. *JACC: Cardiovascular Imaging, 6*, 931–940.
43. Ivanov, A., Dabiesingh, D. S., Bhumireddy, G. P., Mohamed, A., Asfour, A., Briggs, W. M., et al. (2017). Prevalence and prognostic significance of left ventricular noncompaction in patients referred for cardiac magnetic resonance imaging. *Circulation: Cardiovascular Imaging, 10*(9), e006174. https://doi.org/10.1161/CIRCIMAGING.117.006174
44. Ross, S. B., Jones, K., Blanch, B., Puranik, R., McGeechan, K., Barratt, A., & Semsarian, C. (2020). A systematic review and meta-analysis of the prevalence of left ventricular non-compaction in adults. *European Heart Journal, 41*, 1428–1436. https://doi.org/10.1093/eurheartj/ehz317
45. Dreisbach, J. G., Mathur, S., Houbois, C. P., Oechslin, E., Ross, H., Hanneman, K., & Wintersperger, B. J. (2020). Cardiovascular magnetic resonance–based diagnosis of left ventricular non-compaction cardiomyopathy: Impact of cine bSSFP strain analysis. *Journal of Cardiovascular Magnetic Resonance, 22*, 9. https://doi.org/10.1186/s12968-020-0599-3

46. Verma, R., Memon, S., Newman, J., Turin, A., Bibliowicz, N., Bakhos, L., & Syed, M. (2018). Prognostic value of extent of non-compaction in non-ischemic cardiomyopathy. *Journal of the American College of Cardiology, 71*, 737.
47. Banerji, D., Mendoza, D., Ghoshhajra, B. B., & Hedgire, S. S. (2019). The role of contrast-enhanced cardiac magnetic resonance in the assessment of patients with malignant ventricular arrhythmias. *Magnetic Resonance Imaging Clinics of North America, 27*, 475–490.
48. Andreini, D., Pontone, G., Bogaert, J., Roghi, A., Barison, A., Schwitter, J., et al. (2016). Long-term prognostic value of cardiac magnetic resonance in left ventricular noncompaction: A prospective multicenter study. *Journal of the American College of Cardiology, 68*, 2166–2181.
49. Dodd, J. D., Holmvang, G., Hoffmann, U., Ferencik, M., Abbara, S., Brady, T. J., & Cury, R. C. (2007). Quantification of left ventricular noncompaction and trabecular delayed hyperenhancement with cardiac MRI: Correlation with clinical severity. *AJR American Journal of Roentgenology, 189*, 974–980.
50. Nucifora, G., Aquaro, G. D., Pingitore, A., Masci, P. G., & Lombardi, M. (2011). Myocardial fibrosis in isolated left ventricular non-compaction and its relations to disease severity. *European Journal of Heart Failure, 13*, 170–176.
51. Tandon, N., Morgenstern, D., Mikolich, B., & Mikolich, R. J. (2018). Left ventricular longitudinal fiber dysfunction in left ventricular non-compaction cardiomyopathy. *Journal of the American College of Cardiology, 71*, 734.
52. Anderson, R. H., Jensen, B., Mohun, T. J., Petersen, S. E., Aung, N., Zemrak, F., et al. (2017). Key questions relating to left ventricular noncompaction cardiomyopathy: Is the emperor still wearing any clothes? *Canadian Journal of Cardiology, 33*, 747–757. https://doi.org/10.1016/j.cjca.2017.01.017
53. Oechslin, E., & Jenni, R. (2017). Nosology of noncompaction cardiomyopathy: The emperor still wears clothes! *Canadian Journal of Cardiology, 33*, 701–704.
54. Aung, N., Zemrak, F., Mohiddin, S. A., & Petersen, S. E. (2017). LV noncompaction cardiomyopathy or just a lot of trabeculations? *JACC: Cardiovascular Imaging, 10*, 704–707.
55. Sedmera, D., Pexieder, T., Vuillemin, M., Thompson, R. P., & Anderson, R. H. (2000). Developmental patterning of the myocardium. *The Anatomical Record, 258*, 319–337.
56. Stähli, B. E., Gebhard, C., Biaggi, P., Klaassen, S., Valsangiacomo Buechel, E., Attenhofer Jost, C. H., et al. (2013). Left ventricular non-compaction: Prevalence in congenital heart disease. *International Journal of Cardiology, 167*, 2477–2481.
57. Sinkovec, M., Kozelj, M., & Podnar, T. (2005). Familial biventricular myocardial noncompaction associated with Ebstein's malformation. *International Journal of Cardiology, 102*, 297–302.
58. Das, B. B., Chrisant, M., Lavandosky, G., Zakrzewski, M., Winchester, R., Turner, I., et al. (2018). An adolescent with left ventricular noncompaction and Ebstein anomaly presenting with advanced heart failure: Discharge from hospital with a biventricular assist device. *Journal of Pediatrics, 202*, 304–310. https://doi.org/10.1016/j.jpeds.2018.07.038
59. Kumor, M., Lipczyńska, M., Biernacka, E. K., Klisiewicz, A., Wójcik, A., Konka, M., et al. (2018). Cardiac arrest and ventricular arrhythmia in adults with Ebstein anomaly and left ventricular non-compaction. *Journal of Cardiology, 71*, 484–488.
60. Sleurs, E., De Catte, L., & Benatar, A. (2005). Prenatal diagnosis of isolated ventricular noncompaction of the myocardium. *Journal of Ultrasound in Medicine, 24*(9), 1325–1329.
61. Ferreira, C., Thompson, R., & Vernon, H. (2014). Barth syndrome. In M. P. Adam, H. H. Ardinger, R. A. Pagon, et al. (Red.), *GeneReviews*. University of Washington, Seattle. https://www.ncbi.nlm.nih.gov/books/NBK247162/
62. Karch, I., Tomkiewicz-Pająk, L., Komar, M., Olszowska, M., Rubiś, M., & Podolec, P. P. (2012). Isolated left ventricular noncompaction in an asymptomatic athlete (RCD code: III-5A.1.0). *Journal of Rare Cardiovascular Diseases*. https://doi.org/10.20418/jrcd.vol1no7.149
63. Peritz, D. C., Vaughn, A., Clocca, M., & Chung, E. H. (2014). Hypertrabeculation vs left ventricular noncompaction on echocardiogram: A reason to restrict athletic participation. *JAMA Internal Medicine*. https://doi.org/10.1001/jamainternmed.2014.1066
64. Coris, E. E., Moran, K. B., De Cuba, R., Farrar, T., & Curtis, A. B. (2016). Left ventricular non-compaction in athletes: To play or not to play. *Sports Medicine, 46*, 1249–1259.
65. D'Ascenzi, F., Pelliccia, A., Natali, B. M., Bonifazi, M., & Mondillo, S. (2015). Exercise-induced left-ventricular hypertrabeculation in athlete's heart. *International Journal of Cardiology, 181*, 320–322.
66. Gati, S., Chandra, N., Bennett, R. L., Reed, M., Kervio, G., Panoulas, V. F., et al. (2013). Increased left ventricular trabeculation in highly trained athletes: Do we need more stringent criteria for the diagnosis of left ventricular non-compaction in athletes? *Heart, 99*, 401–408.
67. Abela, M., & D'Silva, A. (2018). Left ventricular trabeculations in athletes: Epiphenomenon or phenotype of disease? *Current Treatment Options in Cardiovascular Medicine, 20*, 100.
68. Caselli, S., Ferreira, D., Kanawati, E., Di Paolo, F. M., Pisicchio, C., Attenhofer Jost, C., et al. (2016). Prominent left ventricular trabeculations in competitive athletes: A proposal for risk stratification and management. *International Journal of Cardiology, 223*, 590–595.

69. Arbustini, E., Favalli, V., Narula, N., Serio, A., & Grasso, M. (2016). Left ventricular noncompaction: A distinct genetic cardiomyopathy. *Journal of the American College of Cardiology, 68*, 949–966.
70. Gati, S., Papadakis, M., Papamichael, N. D., Zaidi, A., Sheikh, N., Reed, M., et al. (2014). Reversible de novo left ventricular trabeculations in pregnant women: Implications for the diagnosis of left ventricular noncompaction in low-risk populations. *Circulation, 130*, 475–483.
71. Rani, M., Rajput, R., Mishra, S., & Garg, R. (2020). Non-compaction of ventricular myocardium with polycystic kidney disease with cardiogenic cerebral embolism. *BMJ Case Reports, 13*, e232458. https://doi.org/10.1136/bcr-2019-232458
72. Jefferies, J. L., Wilkinson, J. D., Sleeper, L. A., Colan, S. D., Lu, M., Pahl, E., et al. (2015). Cardiomyopathy phenotypes and outcomes for children with left ventricular myocardial noncompaction: Results from the Pediatric Cardiomyopathy Registry. *Journal of Cardiac Failure, 21*, 877–884. https://doi.org/10.1016/j.cardfail.2015.06.381
73. Siroky, G., Alam, A., Patel, A., Dobrescu, D., Iyer, D., Mody, K., & Huang, M. (2019). Left ventricular hypertrophic noncompaction: A genetic overlap. *Journal of the American College of Cardiology, 73*, 2713.
74. Espinola-Zavaleta, N., Oregel-Camacho, D. O., Alexanderson-Rosas, E., Flores-Garcia, A., & Camacho-Camacho, G. (2019). Restrictive cardiomyopathy type C and left ventricular non-compaction: A rare association. *Journal of the American College of Cardiology, 73*, 2208.
75. Rigopoulos, A., Rizos, I. K., Aggeli, C., Kloufetos, P., Papacharalampous, X., Stefanadis, C., & Toutouzas, P. (2002). Isolated left ventricular noncompaction: An unclassified cardiomyopathy with severe prognosis in adults. *Cardiology, 98*, 25–32.
76. Oryszczyn, N., Pawlyk, S., & Iwaniw, J. (2004). Izolowane niescalenie mięśnia lewej komory. *Polski Przegląd Kardiologiczny, 4*, 461–464.
77. Pignatelli, R. H., McMahon, C. J., Dreyer, W. J., Denfield, S. W., Price, J., Belmont, J. W., et al. (2003). Clinical characterization of left ventricular noncompaction in children: A relatively common form of cardiomyopathy. *Circulation, 108*, 2672–2678.
78. Ergul, Y., Nisil, K., & Demirel, A. (2011). Left ventricular non-compaction in children and adolescents: Clinical features, treatment, and follow-up. *Cardiology Journal, 18*, 176–184.
79. Paszkowska, A., Kowalczyk-Domagała, M., Turska-Kmieć, A., Brzezińska-Rajszys, G., & Ziółkowska, L. (2017). Obraz kliniczny kardiomiopatii pod postacią niescalenia mięśnia lewej komory u dzieci — doświadczenia własne. *Postępy Nauk Medycznych, 30*(9), 471–477.
80. Aypar, E., Sert, A., Gokmen, Z., Aslan, E., & Odabas, D. (2013). Isolated left ventricular noncompaction in a newborn with Pierre–Robin sequence. *Pediatric Cardiology, 34*(2), 452–454. https://doi.org/10.1007/s00246-012-0294-8
81. Stöllberger, C., & Finsterer, J. (2019). Understanding left ventricular hypertrabeculation/noncompaction: Pathomorphologic findings and prognostic impact of neuromuscular comorbidities. *Expert Review of Cardiovascular Therapy, 17*(2), 95–109. https://doi.org/10.1080/14779072.2019.1561280
82. Skidan, V. I., Kuznecova, T., Pavlyukova, E. N., & Nartsissova, G. P. (2020). Isolated left ventricular apical hypoplasia with myocardial non-compaction: A case report. *European Heart Journal – Case Reports, 4*, 1–6. https://doi.org/10.1093/ehjcr/ytz215
83. Oechslin, E. N., Attenhofer Jost, C. H., Rojas, J. R., Kaufmann, P. A., & Jenni, R. (2000). Long-term follow-up of 34 adults with isolated left ventricular noncompaction: A distinct cardiomyopathy with poor prognosis. *Journal of the American College of Cardiology, 36*, 493–500. https://doi.org/10.1016/S0735-1097(00)00755-5
84. Basson, A. L., Essop, M. R., Libhaber, E., & Peters, F. (2020). Family screening in black patients with isolated left ventricular non-compaction: The Chris Hani Baragwanath experience. *Cardiovascular Journal of Africa, 31*, 1–5. https://doi.org/10.5830/CVJA-2020-003
85. Nguyen, Q., Lim, K. R. Q., & Yokota, T. (2020). Genome editing for the understanding and treatment of inherited cardiomyopathies. *International Journal of Molecular Sciences, 21*(3), 733. https://doi.org/10.3390/ijms21030733
86. Shou, W., Aghdasi, B., Armstrong, D. L., Guo, Q., Bao, S., Charng, M. J., et al. (1998). Cardiac defects and altered ryanodine receptor function in mice lacking FKBP12. *Nature, 391*, 489–492. https://doi.org/10.1038/35146
87. Ichida, F. (2020). Left ventricular noncompaction – Risk stratification and genetic consideration. *Journal of Cardiology, 75*(1), 1–9. https://doi.org/10.1016/j.jjcc.2019.09.011
88. Kochel, I., & Strządała, L. (2004). Rola białek wiążących FK506 w regulacji aktywności czynników transkrypcyjnych w komórkach T. *Postępy Higieny i Medycyny Doświadczalnej, 58*, 118–127.
89. Arbustini, E., Narula, N., Tavazzi, L., Serio, A., Grasso, M., Favalli, V., et al. (2014). Left ventricular noncompaction: A distinct cardiomyopathy or a trait shared by different cardiac diseases? *Journal of the American College of Cardiology, 64*, 1840–1850.
90. Luxán, G., Casanova, J. C., Martínez-Poveda, B., Prados, B., D'Amato, G., MacGrogan, D., et al. (2013). Mutations in the NOTCH pathway regulator MIB1 cause left ventricular noncompaction cardiomyopathy. *Nature Medicine, 19*, 193–201. https://doi.org/10.1038/nm.3046
91. Sitnik, K., & Cichy, J. (2006). Udział techniki warunkowej inaktywacji genów opartej na systemie Cre-loxP w postępie wiedzy na temat roli receptorów Notch. *Postępy Biochemii, 52*, 49–55.

92. van Waning, J. I., Caliskan, K., Hoedemaekers, Y. M., van Spaendonck-Zwarts, K. Y., Baas, A. F., Boekholdt, S. M., et al. (2018). Genetics, clinical features, and long-term outcome of noncompaction cardiomyopathy. *Journal of the American College of Cardiology, 71*, 711–722.
93. Li, S., Zhang, C., Liu, N., Bai, H., Hou, C., Wang, J., et al. (2018). Genotype-positive status is associated with poor prognoses in patients with left ventricular noncompaction cardiomyopathy. *Journal of the American Heart Association, 7*, e009910. https://doi.org/10.1161/JAHA.118.009910
94. Online Mendelian Inheritance in Man. (n.d.). *OMIM*. https://www.omim.org/
95. Arbustini, E., Narula, N., Tavazzi, L., Serio, A., Grasso, M., Favalli, V., et al. (2014). The MOGE(S) classification of cardiomyopathy for clinicians. *Journal of the American College of Cardiology, 64*, 304–318. https://doi.org/10.1016/j.jacc.2014.05.027
96. Gupta, S. B., & D'Souza, E. (2015). Biventricular non-compaction cardiomyopathy. *Journal of the Association of Physicians of India, 63*, 77–78.
97. Włodarska, E. K., Woźniak, O., Konka, M., Piotrowska-Kownacka, D., Walczak, E., & Hoffman, P. (2010). Isolated ventricular noncompaction mimicking arrhythmogenic right ventricular cardiomyopathy – A study of nine patients. *International Journal of Cardiology, 145*(1), 107–111. https://doi.org/10.1016/j.ijcard.2009.05.062
98. Stöllberger, C., Finsterer, J., & Blazek, G. (2002). Left ventricular hypertrabeculation/noncompaction and association with additional cardiac abnormalities and neuromuscular disorders. *American Journal of Cardiology, 90*, 899–902.
99. Oechslin, E., & Jenni, R. (2018). Left ventricular noncompaction: From physiologic remodeling to noncompaction cardiomyopathy. *Journal of the American College of Cardiology, 71*, 723–726. https://doi.org/10.1016/j.jacc.2017.12.031
100. Zemrak, F., Ahlman, M. A., Captur, G., Mohiddin, S. A., Kawel-Boehm, N., Prince, M. R., et al. (2014). The relationship of left ventricular trabeculation to ventricular function and structure over a 9.5-year follow-up: The MESA study. *Journal of the American College of Cardiology, 64*, 1971–1980. https://doi.org/10.1016/j.jacc.2014.08.035
101. Winer, N., Lefevre, M., Nomballais, M. F., David, A., Rival, J. M., Gournay, V., et al. (1998). Persisting spongy myocardium: A case indicating the difficulty of antenatal diagnosis. *Fetal Diagnosis and Therapy, 13*, 227–232.
102. Gajewski, P. (Red.). (2019). *Interna Szczeklika 2019/2020* (s. 378–390). Medycyna Praktyczna.
103. Sotiris, S. (2018). Isolated form of left ventricular non-compaction cardiomyopathy (LVNC) as a rare cause of heart failure. *Current Perspectives on Cardiomyopathies, 6*. https://doi.org/10.5772/intechopen.76379
104. Graça, S. L., Carvalho, R., Montenegro, F., Saraiva, F., & Guardado, J. (2020). Left ventricular noncompaction: A rare cause of effort angina unveiled during left heart catheterization. *Journal of Invasive Cardiology, 32*(2), E42.
105. Taghavi, M., Ghaemian, A., Nabati, M., Farsavian, A., & Shokri, M. (2019). The coexistence of left ventricular non-compaction cardiomyopathy, significant coronary artery disease and massive thrombus formation in left ventricular cavity: A rare case report. *Journal of Clinical Ultrasound, 47*(2), 107–110. https://doi.org/10.1002/jcu.22659
106. Minamisawa, M., Koyama, J., Kuwahara, K., & Ikeda, U. (2018). Characteristics and clinical outcomes in patients with left ventricular noncompaction in adults: A nationwide survey in Japan. *Journal of the American College of Cardiology, 71*, 714.
107. Hotta, V. T., Tendolo, S. C., Rodrigues, A. C., Fernandes, F., Nastari, L., & Mady, C. (2017). Limitations of the diagnosis of noncompaction cardiomyopathy by echocardiography. *Arquivos Brasileiros de Cardiologia, 109*, 483–488.
108. Shoji, M., Yamashita, T., Uejima, T., Asada, K., Semba, H., Otsuka, T., et al. (2010). Electrocardiography characteristics of isolated non-compaction of ventricular myocardium in Japanese adult patients. *Circulation Journal, 74*, 1431–1435.
109. Steffel, J., Kobza, R., Oechslin, E., Jenni, R., & Duru, F. (2009). Electrocardiographic characteristics at initial diagnosis in patients with isolated left ventricular noncompaction. *American Journal of Cardiology, 104*, 984–989.
110. Ekizler, F. A., Cay, S., Ulvan, N., Tak, B. T., Cetin, E. H. O., Kafes, H., et al. (2020). Importance of lead aVR in predicting adverse cardiac events in patients with noncompaction cardiomyopathy. *Annals of Noninvasive Electrocardiology, 25*(3), e12719. https://doi.org/10.1111/anec.12719
111. Towbin, J. A. (2010). Left ventricular noncompaction: A new form of heart failure. *Heart Failure Clinics, 6*, 453–469.
112. Cerar, A., Jaklic, M., Frljak, S., Poglajen, G., Zemljic, G., & Guzic Salobir, B. (2020). Impairment of myocardial perfusion correlates with heart failure severity in patients with non-compaction cardiomyopathy. *ESC Heart Failure, 7*(3), 1161–1167. https://doi.org/10.1002/ehf2.12631
113. Tumolo, A. Z., & Nguyen, D. T. (2017). Spectrum of cardiac arrhythmias in isolated ventricular non-compaction. *Journal of Innovations in Cardiac Rhythm Management, 8*(7), 2774–2783. https://doi.org/10.19102/icrm.2017.080701
114. Bhaskaran, A., Campbell, T., Trivic, I., Virk, S., & Kumar, S. (2019). Electrophysiologic substrate of ventricular arrhythmias in ventricular noncompaction – A systematic review. *Heart, Lung and Circulation*. https://doi.org/10.1016/j.hlc.2019.06.184

115. Jordaens, L., & Towbin, J. A. (2018). Arrhythmias and conduction disturbances in noncompaction cardiomyopathy. In D. P. Zipes, J. Jalife, & W. G. Stevenson (Red.), *Cardiac electrophysiology* (s. 870–877). https://doi.org/10.1016/B978-0-323-44733-1.00090-0
116. Kobza, R., Steffel, J., Erne, P., Schoenenberger, A. W., Hürlimann, D., Lüscher, T. F., et al. (2010). Implantable cardioverter-defibrillator and cardiac resynchronization therapy in patients with left ventricular noncompaction. *Heart Rhythm, 7*, 1545–1549.
117. Vaidya, V., Lyle, M., Nadipalli, A., Michelena, H., Asirvatham, S., Connolly, H., et al. (2018). Electrocardiographic variables associated with atrial arrhythmias in patients with left ventricular noncompaction. *Journal of the American College of Cardiology, 71*, 425.
118. Fazio, G., Corrado, G., Pizzuto, C., Zachara, E., Rapezzi, C., Sulafa, A. K., et al. (2008). Supraventricular arrhythmias in noncompaction of left ventricle: Is this a frequent complication? *International Journal of Cardiology, 127*, 255–256.
119. Stöllberger, C., Blazek, G., Dobias, C., Hanafin, A., Wegner, C., & Finsterer, J. (2011). Frequency of stroke and embolism in left ventricular hypertrabeculation/noncompaction. *American Journal of Cardiology, 108*, 1021–1023.
120. Bennett, C. E., & Freudenberger, R. (2016). The current approach to diagnosis and management of left ventricular noncompaction cardiomyopathy: Review of the literature. *Cardiology Research and Practice, 2016*, 5172308. https://doi.org/10.1155/2016/5172308
121. Subahi, A., Hassan, A. A. L., Abubakar, H., & Ibrahim, W. (2017). Isolated left ventricular non-compaction (LVNC) and recurrent strokes: To anticoagulate or not to anticoagulate, that is the question. *BMJ Case Reports, 2017*, bcr2017220954. https://doi.org/10.1136/bcr-2017-220954
122. Caliskan, K., Szili-Torok, T., Theuns, D. A. M. J., Kardos, A., Geleijnse, M. L., & Balk, A. H. (2011). Indications and outcome of implantable cardioverter-defibrillators for primary and secondary prophylaxis in patients with noncompaction cardiomyopathy. *Journal of Cardiovascular Electrophysiology, 22*, 898–904.
123. Ozben, B., Mutlu, B., & Erdogan, O. (2011). ICD implantation in left ventricular noncompaction: A case report and review of the literature. *Cardiology Journal, 18*(6), 691–694.
124. Coppola, G., Guttilla, D., Corrado, E., Falletta, C., Marrone, G., & Farulla, R. A. (2009). ICD implantation in noncompaction of the left ventricular myocardium: A case report. *Pacing and Clinical Electrophysiology, 32*(8), 1092–1095.
125. Gleva, M. J., Wang, Y., Curti, J. P., Berul, C. I., Huddleston, C. B., Poole, J. E., et al. (2017). Complications associated with implantable cardioverter-defibrillators in adults with congenital heart disease or left ventricular noncompaction cardiomyopathy: From the NCDR ICD Registry. *American Journal of Cardiology, 120*, 1891–1898.
126. Shimamoto, T., Marui, A., Yamanaka, K., Shikata, N., Tambara, K., Ikeda, T., & Komeda, M. (2007). Left ventricular restoration surgery for isolated left ventricular noncompaction: Report of the first successful case. *Journal of Thoracic and Cardiovascular Surgery, 134*, 246–247.
127. Ikeda, U., Minamisawa, M., & Koyama, J. (2015). Isolated left ventricular non-compaction cardiomyopathy in adults. *Journal of Cardiology, 65*, 91–97.
128. Hawatmeh, A., Thawabi, M., Strueb, M., Gidea, C., & Cohen, M. (2018). LVAD as a bridge to heart transplantation in a patient with left ventricular noncompaction cardiomyopathy and advanced heart failure. *Journal of the American College of Cardiology, 71*, 2424.
129. Bansal, A., Akhtar, F., Bansal, M., Desai, S., Jacks, D., & Gilliland, Y. (2019). Safety of mechanical circulatory support for left ventricular non-compaction: Single-center experience. *Journal of Heart and Lung Transplantation, 38*, 442.
130. Uribarri, A., Rojas, S. V., Avsar, M., Hanke, J. S., Napp, L. C., Berliner, D., et al. (2015). First series of mechanical circulatory support in non-compaction cardiomyopathy: Is LVAD implantation a safe alternative? *International Journal of Cardiology, 197*, 128–132. https://doi.org/10.1016/j.ijcard.2015.04.046