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A SYSTEMATIC REVIEW: SIGNS AND SYMPTOMS, CLINICAL PRESENTATION, AND DIAGNOSIS OF NONCOMPACTION MYOCARDIUM

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ABSTRACT

Objective: The purpose of this study is to present an updated view of the noncompaction myocardium, in addition to providing a detailed analysis of the clinical manifestations and diagnostic methods. **Methodology:** To conduct this research, the following question was formulated: "What are the main signs and symptoms of Left Ventricular Noncompaction (LVNC), what are the methods used for its diagnosis and what are the therapeutic resources used in clinical practice?" The searches were carried out in the PubMed Central (PMC) database. Using the Boolean term "AND", the following 5 descriptors were combined: Isolated Noncompaction of the Ventricular Myocardium, Thrombosis, Signs and Symptoms, Pathological Conditions and Cardiomyopathies. In all, 45 articles were found, of which, after applying the inclusion and exclusion criteria, 10 studies were selected to compose the review. **Results:** LVNC is characterized by the occurrence of heart failure, arrhythmias, and a high risk of embolic events. The prevalence of atrial fibrillation and

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ventricular tachyarrhythmias is noteworthy. The diagnosis is based on specific morphological criteria, with transthoracic echocardiography and cardiac magnetic resonance imaging being the main diagnostic methods. **Conclusion:** The variations in the echocardiographic criteria used to diagnose LVNN underscore the urgent need for a more standardized consensus. The correct identification of LVNC is essential to ensure effective clinical management of patients, aiming at reducing complications and significantly improving quality of life.

Keywords: Diagnosis. Cardiology. Uncompacted myocardium.

INTRODUCTION

Left ventricular noncompaction (LVNC) or also known as noncompaction myocardium (NCM) is a specific type of cardiomyopathy that is characterized by having gross myocardial trabeculae and deep intertrabecular crypts inside the ventricle, communicating with the ventricular cavity and with the development of systolic and diastolic insufficiency. (TIAN. et al; 2023) Although LVNC is considered a genetic cardiomyopathy by the American Heart Association, the European Society of Cardiology classifies it as an unclassified cardiomyopathy. (AUNG. et al; 2020)

LVNC demonstrates a prevalence of 1:5,000 individuals in the general population and in 3 to 4% of adults with heart failure. (ICHIDA. et al; 2020) Noncompaction of the left ventricle can be diagnosed at any age, but in many individuals it remains undiagnosed until later in life. (MASSO. et al; 2020)

LVNC is a cardiomyopathy defined by the presence of a layer with a prominent trabecular network and deep intertrabecular recesses, which communicate directly with the left ventricular cavity. These recesses can affect coronary artery circulation by creating areas of impaired intramural perfusion, particularly in the subendocardium, leading to subendocardial ischemia (ICHIDA. et al; 2020) During embryonic myocardial development, the normal process involves the transformation of the initial spongy myocardium into a more compact musculature, with reduced trabeculation. LVNC occurs due to disruption or failure in this process, resulting in a myocardium that does not compact properly. This phenomenon can be observed in both children and adults and is associated with an estimated prevalence of 1:5,000 in the general population (ICHIDA. et al; 2020).

Left ventricular hypertrabeculation (LVHT) is a cardiac phenotype that constitutes a morphological abnormality of the myocardium, standing out for the presence of two distinct structural layers that perform specific functions and have unique characteristics. The first layer, known as the trabecular layer, is the thickest, made up of multiple prominent trabeculae that protrude into the left ventricular cavity, creating deep, complex recesses that often communicate with the ventricular cavity. On the other hand, the compacted layer is a significantly thinner subepicardial structure, whose reduced thickness contrasts directly with the trabecular, and which, in cases of LVHT, is particularly reduced. The origin of LVHT is often associated with an interrupted or altered process of myocardial compaction during embryonic development, resulting in the preservation of this increased trabecular architecture that characterizes the phenotype. In terms of prevalence, advances in cardiac imaging technologies, such as cardiac magnetic resonance imaging (CMR) and advanced echocardiograms, have played a crucial role in identifying and diagnosing the condition,

which explains the increase in its detection in recent years. From a functional standpoint, the exaggerated trabeculations seen in hearts affected by LVHT can significantly impair both contractile and diastolic left ventricular function, making it a potential source of cardiac dysfunction. In addition, the etiologies of LVHT are varied, encompassing congenital causes, such as genetic mutations related to cardiac development, or lifelong acquired factors, such as those observed in individuals during pregnancy, in conditions of heart failure or hypertension, and even in athletes subjected to intense physical exertion. This multifaceted phenotype illustrates not only the diagnostic and etiological complexity of LVHT, but also its clinical relevance in modern cardiology, given its potential impact on cardiac health. (ADABIFIROUZJAEI. et al; 2021)

The gross anatomy of the myocardium is called "spongy" and has two layers, comprising a shallow and dense layer on the surface and a thick, loose and non-dense layer on the inside. (TIAN. et al; 2023) The endocardial trabecular layer is usually imperceptible, as intertrabecular clefts are largely minimized during maturation. (ADABIFIROUZJAEI. et al; 2021) Emerging evidence suggests that excessive trabeculation may be a result of a disturbance in the compaction process during early myocardial development. (FEMIA. et al; 2020) LV hypertrabeculation (LVHT) is an abnormality of the LV myocardium consisting of multiple elongated trabeculae, separated by widened and deep intertrabecular fissures, which protrude prominently into the LV cavity and are usually accompanied by a decreased compressed myocardium. (ADABIFIROUZJAEI. et al; 2021)

Trabeculae are transient embryonic structures that play a role in myocardial oxygenation through the intertrabecular pathway, a space that precedes the development of coronary vascularization. The trabecular remodeling process is located around the 8th week of gestation and the increase in ventricular volume compresses the fissures, increasing the thickness of the compacted myocardium. Myocardial maturation generates two myocardial zones: a trabecular zone adjunct to the endocardium and a compact subepicardial zone. The compaction process is critical to provide rotational and contractile function of the LV myocardium. In LVHT, the persistence or reappearance of the intertrabecular crypts occurs, generating deep recesses that communicate with the ventricular cavity. (ADABIFIROUZJAEI. et al; 2021)

LVNC can occur in isolation or be associated with other congenital heart diseases, neuromuscular diseases, genetic syndromes, and other types of cardiomyopathies. Most trabeculae are found in the apical region of the ventricle, being seen in both ventricles and rarely accumulate only in the right ventricle. (TIAN. et al; 2023) LVNC, like other forms of hereditary cardiomyopathy, is genetically heterogeneous and can be inherited through an

X-linked autosomal dominant or recessive disorder. Evidence from genetic studies has shown that the main cause of LVNC is the result of mutation of genes that encode sarcomeric proteins, symbolizing up to 30% of all cases. Other related mutations occurred in genes responsible for encoding cytoskeletal, Z-line, and mitochondrial proteins. The most common mutations were in myosin heavy chain 7 (MYH7), myosin protein-binding protein C genes (MYBPC3), tropomyosin alfa (TPM1), myocardial actin (ACTC1), troponin T (TNNT2), and cardiac troponin I (TNNI3). Familial LVN, when accompanied by hypertrophic or dilated cardiomyopathy, is typical of the MYH7 mutation. (DO RÊGO AQUINO. et al; 2021)

This condition was first demonstrated in 1926 through autopsy with a spongy appearance of the myocardium, and the term LVNC was first mentioned in 1990. (TIAN. et al; 2023) In general, there is heterogeneity in clinical manifestations, from asymptomatic to ventricular arrhythmias, left ventricular (LV) dysfunction, stroke, and/or cardiac death. (FEMIA. et al; 2020) It is detected only through medical examination or family screening. However, it can also present end-stage heart failure, arrhythmias, and systemic embolic events. (TIAN. et al; 2023) It is reported that athletes and pregnant women have higher prevalence rates of VEN, which suggests a physiological adaptation related to pressure overload and not to an actual pathological process in these patients. (AUNG. et al; 2020)

The purpose of this literature review was to present the current knowledge about the main signs and symptoms, methods used in the diagnosis of noncompaction myocardium, and to provide a critical analysis. Given the frequent uncertainty regarding the diagnosis, an evidence-based review of the current literature was performed to group the most recent information and contribute to a better understanding of noncompaction myocardium among medical professionals.

METHODS

Objective: This study consists of a systematic review with the objective of exploring the most relevant clinical manifestations of Left Ventricular Noncompaction (LVNC), unraveling the methods used for its diagnosis, and identifying innovative therapeutic strategies that promote early diagnosis and effective interventions. To guide the research, the following guiding question was formulated based on the PVO (population, variable and objective) methodology: **"What are the predominant clinical manifestations of Left Ventricular Noncompaction (LVNC), the most effective diagnostic methods and the innovative therapies applied in current medical practice?"** The search for articles was performed in the PubMed Central (PMC) database, using 5 descriptors combined with the

Boolean operator "AND": Isolated Noncompaction of the Ventricular Myocardium, Thrombosis, Signs and Symptoms, Pathological Conditions and Cardiomyopathies. The search strategy followed the combinations: 45 articles were identified. After applying the inclusion and exclusion criteria, a total of 10 studies were selected to compose this review. **Results:** The studies analyzed indicated that the symptoms most frequently associated with LVNC include breathing difficulties, such as dyspnea, extreme tiredness, and irregularities in the heartbeat. These signs can have varying degrees of intensity, depending on the individual. To establish an accurate diagnosis, advanced imaging methods stand out, including cardiac magnetic resonance imaging and three-dimensional echocardiography, tools that allow a detailed and reliable analysis of the structure and function of the myocardium. As for therapeutic approaches, the personalization of pharmacological treatment is emphasized, complemented by the use of implantable devices, such as defibrillators, especially in patients at high risk of severe cardiac complications. In addition, preventive strategies have gained relevance, including active surveillance of genetically predisposed patients and continuous medical follow-up programs to identify early changes before they progress to critical conditions. **Conclusion:** The findings of this review emphasize the importance of an integrated clinical approach, combining high-precision diagnostic imaging and therapies adapted to the patient's needs. Promoting early diagnosis and strategic management of LVNC is essential to improve patients' quality of life, in addition to reducing associated complications. This study reinforces the need for continuous advances in research to expand the possibilities of diagnosis and treatment of LVN, consolidating a more personalized and effective care.

DISCUSSION

The clinical manifestations of LVNC are variable, ranging from total absence of symptoms to severe complications such as congestive heart failure, potentially fatal arrhythmias, systemic thromboembolism, and even sudden cardiac death. These manifestations can vary greatly even among members of the same family, highlighting the genetic and phenotypic heterogeneity of the condition (ICHIDA. et al; 2020).

The triad of heart failure, arrhythmias, and embolic events is the main clinical manifestation in patients with LVNC and is comparable in the adult and pediatric populations. Various patterns of arrhythmia can be seen, ranging from atrial fibrillation to sustained ventricular tachycardia. (ICHIDA. et al; 2020) Atrial fibrillation can affect 25% of adult patients and ventricular tachyarrhythmias up to about 50%, in addition to clinical

symptoms reported by the patients themselves, such as weakness and palpitations. (DO RÊGO AQUINO. et al; 2021)

Left ventricular noncompaction (LVNC) is a genetically heterogeneous condition, often associated with mutations in genes that are fundamental for myocardial development and function, especially sarcomeric genes such as MYH7, ACTC1, TNNT2, MYBPC3, TPM1 and TNNI3, which encode the sarcomere components responsible for cardiac muscle contraction, and whose mutations are responsible for a significant portion of LVNC cases. It also involves the TAZ/G4.5 gene, which encodes the protein tafazzin, essential in mitochondrial lipid metabolism, whose mutation was initially identified in Barth syndrome, related to failures in mitochondrial energy production and sarcomeric dysfunctions. In addition, cytoskeletal and Z-disc genes, such as desmin and lamin A/C, are implicated in the cellular structural integrity of the myocardium, while the NOTCH signaling pathway, through the Notch1 receptor and its ligands, plays an essential role in trabeculae formation and ventricular compaction during embryonic development. The etiology of LVNC is related to an interruption in the embryonic myocardial compaction process, resulting in a spongy and trabecular pattern that should have been transformed into compact muscle. Studies show that between 20-40% of cases are hereditary, and genetic screening is recommended in first-degree relatives when there is a confirmed diagnosis, with inheritance being possible as an autosomal dominant, recessive, or X-linked disorder, highlighting the complexity and importance of ongoing investigations into the genetic mechanisms involved (ICHIDA. et al; 2020).

A large retrospective study of 169 adult patients with noncompaction myocardium 15% experienced thromboembolic events: 92% of them had stroke and 8% had a peripheral embolism. (CHIMENTI. et al; 2022) There is a notion of an increased risk of systemic thromboembolism attributable to slow blood flow in the deep intertrabecular recesses in patients with LVNC. (AUNG. et al; 2020) The cause of thromboembolism was cardioembolic (69%), atherosclerotic (19%) and indeterminate (12%); among the 18 patients with cardioembolism and stroke, only 39% had atrial fibrillation (AF), while 78% had LV systolic dysfunction. In the cardioembolic group, 50% of patients were receiving acetylsalicylic acid (ASA) 100 mg/day, 6% were receiving vitamin K antagonists, 6% were receiving low molecular weight heparin, and 38% were not on antithrombotic or anticoagulant therapy. (CHIMENTI. et al; 2022)

Currently, the morphological diagnosis of LVNC is mainly based on echocardiography or cardiac magnetic resonance imaging (CMR) (TIAN. et al; 2023). Echocardiography is the main test used to diagnose LVNC. The morphological criteria for

LVN analyzed by transthoracic echocardiography were suggested by Chin, Stollberger, Jenni, and Paterick. The most used criterion is the one proposed by Jenni. (DO RÊGO AQUINO. et al; 2021) Based on the criteria, proposed by Chin, it postulates that a relationship between the distance from the epicardial surface to the trabecular recess and the distance from the epicardial surface to the trabecular peak less than 0.5 is suggestive of LVNC. In the criteria proposed by Stollberger, the presence of more than three trabeculations in the left ventricular wall with apical location of the papillary muscles visible in a single image plane and perfused intertrabecular spaces of the ventricular cavity visualized by color Doppler is suggestive of LVNC. The suggestive criteria for LVNC proposed by Jenni are based on a maximum ratio between uncompressed myocardium and compacted myocardium greater than 2 at the end of systole of the short parasternal axis and, finally, the criteria proposed by Paterick the ratio between the thickness of the uncompressed and compacted myocardium should be greater than 2; The measurement should be performed at the end of diastole in the transverse parasternal section. (DO RÊGO AQUINO. et al; 2021)

The best criterion for identifying LVNN on cardiac magnetic resonance imaging was developed by Petersen, This criterion requires a relationship between trabecular thickness and compact myocardial thickness >2.3 at the end of diastole in long-axis sections, with a sensitivity of 83% and specificity of 99% at diagnosis. Another way to diagnose this entity by CMRI is to verify whether the trabeculate mass of the left ventricle is greater than 20% of the total mass, with a sensitivity of 91.6% and specificity of 86.5% for LVS (DO RÊGO AQUINO. et al; 2021)

In an ambitious initiative led by the Texas Heart Institute, researchers conducted a fascinating study involving thousands of young students, ages 13, in Harris County, Texas. The main objective was to estimate the prevalence of congenital cardiovascular conditions associated with sudden cardiac death in young people. During the process, one piece of data drew attention: an unexpectedly high prevalence of left ventricular noncompaction (LVNC), detected by cardiac magnetic resonance imaging (CMR) scans, as indicated by Masso et al. (2020).

LVNC, a rare and complex condition, occurs when the muscle trabeculae of the left ventricle do not compact properly in the development of the heart. This process is essential for transforming the heart muscle from a spongy structure to a solid, functional one. In the study, applying diagnostic criteria such as Petersen's, about 18.6% of the participants showed signs of the condition. However, a question arose: were all these cases really abnormal or just normal anatomical variations?

Moved by this doubt, the researchers analyzed the images more deeply and used other diagnostic criteria, such as those proposed by Jacquier and Choi. The results suggested that many of the identified cases could not be pathological, while others presented potential physiological risks, such as ventricular dysfunctions or arrhythmias. It is especially relevant to note that, in children and adolescents, such characteristics may go unnoticed, requiring longitudinal follow-up to understand their clinical impact (Masso et al. 2020). The study revealed more than numbers. It has brought to light important challenges for the medical community: from the limitations of current diagnostic criteria to the need for more in-depth studies that can better distinguish what is a normal variant and what requires clinical attention, as pointed out in the analysis by Masso et al. (2020).

Comparing the three main echocardiographic criteria in a study of 199 patients and 60 healthy people in the control group, 47 patients met at least one of the criteria. Approximately 79% of this sample met the criteria for noncompaction established by Chin, about 64% were diagnosed by Jenni's criteria, and less than 54% met Stollberger's criteria. Only about 30% of this sample fully met the three criteria, evidencing the lack of consensus among them. (DO RÊGO AQUINO et al., 2021)

In another study, the authors evaluated more than 1000 asymptomatic athletes and found that 18% had increased left ventricular trabeculation and 8% met the criteria for LVNC. In a second study, the same authors examined 102 women with normal echocardiograms and found that 25% developed de novo trabeculation during pregnancy. Interestingly, at 24 months postpartum, 73% had complete resolution of trabeculations. (FEMIA et al., 2020)

Color Doppler can detect that the intertrabecular crypts communicate with the left ventricle, but not with the circulation of the coronary artery, which excludes the combination of other congenital or acquired heart diseases. CMR is another method for diagnosing LVNN. (TIAN et al., 2023)

Increased awareness and improved imaging techniques have led to inaccurate diagnoses, clinical challenges, and unwarranted restrictions on competitive sport. Currently, there are several criteria based on two-dimensional echocardiography and cardiac MRI, but there is no diagnostic "gold standard" or specific clinical guidelines to help differentiate physiological hypertrabeculation from pathological LVNC. (FEMIA et al., 2020)

Compared to echocardiography, CMR has a longer examination time and a high cost. However, it can be used as a complementary tool due to the good spatial resolution of the cardiac segment. According to current clinical research, patients with dilated and hypertrophic cardiomyopathy often present with enlargement and thickening of the

myocardial trabeculae. Some scholars believe that this may contribute to the overdiagnosis of the disease in the population. (TIAN et al., 2023) CMR is used when echocardiographic findings are inconclusive.

Echocardiography does not always completely visualize the apical region and may underestimate the degree of LVNN. Therefore, CMR has become the method of choice to confirm or rule out LVN. CMR is generally used to complement and confirm 2D echo findings, providing better spatial resolution in all LV segments, detailed visualization of cardiac morphology, robust volumetry, and the ability to identify fibrosis with delayed gadolinium enhancement (GTR). CMR can more easily differentiate compacted from noncompacted myocardium throughout the LV cavity, and the diagnostic rate has been shown to be higher when compared to 2D echo. (FEMIA et al., 2020) The criteria have been limited by the rarity of the disease, which makes it difficult to recruit large and heterogeneous patient cohorts. (ROSS et al., 2020)

All current methodologies used to establish a diagnosis have strengths and weaknesses in terms of how they are derived, ease of use, time to acquisition of the relevant images and diagnostic accuracy. There is no evidence to suggest the superiority of any criterion or imaging modality in comparison with each other. However, as image quality and knowledge of diagnostic criteria evolves, the phenotype in question emerges as an increasingly recognized finding, with the inherent risk of overdiagnosis being a significant concern. (AUNG et al., 2020)

The rate of diagnosis of LVNC has gradually increased. However, there are still many controversies about its pathogenesis, with the main theories currently including the embryogenesis hypothesis and the molecular genetic mechanism. In recent years, with the advancement of genetic research methods, related pathogenic genes have been confirmed. (TIAN et al., 2023) Despite this knowledge, the yield in diagnosing genetic tests remains low for some individuals; Abnormal trabeculations may develop concomitantly with other cardiovascular or systemic conditions. (FEMIA et al., 2020) Considering that LVNC is classified as a genetic cardiomyopathy, there is a need for family screening, given that diagnostic issues can cause harm beyond the individual patient, causing inadequate diagnoses if there is overdiagnosis in family members. (ROSS et al., 2020)

Although most patients with LVNC remain symptom-free, it is important to regularly review patients with cardiac imaging, as some may be at risk for heart failure, stroke, and/or sudden cardiac death. In particular, those with reduced LV function should be reviewed frequently and treated with evidence-based, guideline-guided pharmacological therapy. According to the guidelines, an intracardiac defibrillator should be offered to those who

survive an episode of sustained ventricular tachycardia or sudden cardiac arrest. Heart transplantation has been reported to be successful in some patients and should be considered for those with end-stage heart failure. Although the rate of stroke events is 1–2% per year, the optimal medical strategy in those who do not meet standard anticoagulation criteria remains uncertain, given the paucity of data. However, patients with a prior cardioembolic event, evidence of intracardiac thrombus, and/or documented atrial fibrillation should be treated with anticoagulation consistent with standard recommendations for cardiogenic embolism. (FEMIA et al., 2020)

Left ventricular noncompaction (LVNC) has distinct subtypes that reflect different morbidity and mortality profiles, and is classified as isolated LVNC (with normal cardiac function), hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DMC), and indeterminate cardiomyopathy phenotype. These subtypes help in risk stratification, which is essential to better understand heterogeneous phenotypes. Among the classifications, children with isolated LVNC and preserved cardiac function have the best prognosis, followed by those with LVNC associated with HCM. On the other hand, children with LVNC accompanied by CMD or indeterminate cardiomyopathy generally exhibit the worst clinical outcomes, demonstrating a higher risk of serious complications such as heart failure, arrhythmias, and sudden death (ICHIDA. et al; 2020).

The prognosis of patients with LVNC is closely related to the presence of myocardial dysfunction or significant arrhythmias, both of which are often associated with higher mortality. In adults with LVNC, up to 47% of cases (75% of symptomatic cases) can lead to death within six years of diagnosis. In children, studies indicate better outcomes in individuals diagnosed after the first few years of life, while affected neonates often have a poorer prognosis due to associated systemic diseases, such as mitochondrial disorders. In addition, factors such as congestive heart failure at the time of diagnosis and hypoplasia of the ventricular compacted layer are significant predictors of adverse outcomes (ICHIDA. et al; 2020).

To improve disease management, subtype classification and risk stratification offer key insights. This allows for more targeted therapeutic interventions, such as the prevention of arrhythmias, the use of implantable defibrillators in severe cases, and continuous clinical follow-up. Thus, the importance of long-term monitoring, even in apparently asymptomatic patients, is crucial to identify future complications and improve quality of life. If you want to explore any more nuance, I can drill down or expand on related topics. (ICHIDA. et al; 2020).

The optimal management to prevent thromboembolic events has not yet been fully elucidated. Some studies recommend the prophylactic use of oral anticoagulants, such as warfarin, for all patients diagnosed with noncompaction myocardium. However, the use of anticoagulants is currently recommended only for patients with reduced systolic function, ejection fraction less than 40%, and a history of previous thromboembolism or atrial fibrillation. (DO RÊGO AQUINO. et al; 2021)

ICD implantation for the primary prevention of sudden cardiac death is indicated in patients with left ventricular ejection fraction of up to 35% and HF functional class II or III (New York Heart Association), in addition to the presence of diagnosed noncompaction myocardium. Patients with a history of sustained ventricular tachycardia or who have recovered from cardiac arrest require ICD implantation for secondary prevention. (DO RÊGO AQUINO. et al; 2021) The treatment of HF in these patients should be the same as for other etiologies. Thus, to control systolic or diastolic dysfunction, beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, mineralocorticoid receptor antagonists, and/or diuretics can be used (DO RÊGO AQUINO. et al; 2021)

CONCLUSION

Based on the data analyzed, it is concluded that Left Ventricular Noncompaction (LVNC) is a complex condition characterized mainly by the triad of heart failure, arrhythmias, and embolic events. The high prevalence of atrial fibrillation and ventricular tachyarrhythmias, as well as the significant risk of thromboembolic events of cardioembolic origin, highlight the need for a vigilant clinical approach.

The diagnosis of LVNC is based on precise morphological criteria, in which transthoracic echocardiography, governed by the Jenni criteria, and cardiac magnetic resonance imaging (CMR), with the Petersen criteria, play essential roles due to their superior sensitivity and specificity. The variations observed in the diagnostic criteria reinforce the urgency of a uniform consensus, which allows greater precision in the identification of the disease and greater standardization in clinical management.

Thus, the study emphasizes the importance of early diagnosis combined with individualized therapeutic strategies, including pharmacological management and the use of implantable devices in situations of high cardiac risk. Accurate detection of LVNC not only prevents serious complications but also contributes significantly to improving patients' quality of life. Continuous investments in research are crucial for the advancement of diagnostic and therapeutic practices, consolidating an integrated and personalized approach to the treatment of this condition.

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