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CASE REPORT

Idiopathic dilated cardiomyopathy diagnosed by cardiac magnetic resonance imaging in a patient with new-onset heart failure: a case report

Miocardiopatía dilatada idiopática diagnosticada mediante resonancia magnética cardíaca en una paciente con insuficiencia cardíaca de reciente inicio: reporte de caso

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ABSTRACT

Idiopathic dilated cardiomyopathy (IDCM) is a myocardial disease characterized by ventricular dilation and systolic dysfunction in the absence of coronary artery disease, arterial hypertension, valvular heart disease, or congenital heart defects. Identifying its etiology poses a diagnostic challenge, and cardiac magnetic resonance (CMR) is a highly useful tool. We present the case of a 43-year-old woman with a history of bronchial asthma and overweight status who presented with progressive dyspnea, exercise intolerance, and clinical signs of congestive heart failure. The electrocardiogram showed sinus rhythm with complete left bundle branch block, while the echocardiogram revealed dilation of all four cardiac chambers and a left ventricular ejection fraction of 35%, with no segmental wall motion abnormalities. Coronary angiography was normal. CMR demonstrated a pattern of linear mid-wall late gadolinium enhancement in the interventricular septum, a characteristic finding of non-ischemic dilated cardiomyopathy consistent with IDCM. The patient showed a favorable clinical course following the initiation of medical therapy. Follow-up plans included Holter monitoring, assessment for primary prevention of sudden cardiac death via an implantable cardioverter-defibrillator, and clinical evaluation of first-degree relatives. This case highlights the incremental value of CMR in the etiological diagnosis of dilated cardiomyopathy, particularly after coronary artery disease has been ruled out. Characterizing the fibrosis pattern via late gadolinium enhancement improves diagnostic accuracy, provides relevant prognostic information, and guides therapeutic decisions as well as family screening in patients with suspected idiopathic or genetic disease.

Keywords: case report; dilated cardiomyopathy; gadolinium; heart failure; magnetic resonance imaging; myocardial fibrosis.

RESUMEN

La miocardiopatía dilatada idiopática (MCDI) es una enfermedad del miocardio caracterizada por dilatación ventricular y disfunción sistólica en ausencia de enfermedad coronaria, hipertensión arterial, valvulopatías o cardiopatías congénitas. Su identificación etiológica representa un desafío diagnóstico, siendo la resonancia magnética cardíaca (RMC) una herramienta de gran utilidad. Se presenta el caso de una mujer de 43 años, con antecedentes de asma bronquial y sobrepeso, que consultó por disnea progresiva, intolerancia al ejercicio y signos clínicos de insuficiencia cardíaca congestiva. El electrocardiograma mostró ritmo sinusal con bloqueo completo de rama izquierda, mientras que el ecocardiograma evidenció dilatación de las cuatro cavidades y una fracción de eyección del ventrículo izquierdo del 35%, sin alteraciones segmentarias de la contractilidad. La coronariografía fue normal. La RMC demostró un patrón de realce tardío con gadolinio mesocárdico lineal en el septo interventricular, hallazgo característico de miocardiopatía dilatada no isquémica, compatible con MCDI. La paciente presentó evolución clínica favorable tras el inicio de tratamiento médico. Se planificó seguimiento con monitorización holter, evaluación para prevención primaria de muerte súbita mediante desfibrilador automático implantable y estudio clínico de familiares de primer grado. Se destaca el valor incremental de la RMC en el diagnóstico etiológico de la miocardiopatía dilatada, particularmente cuando la enfermedad coronaria ha sido descartada. La caracterización del patrón de fibrosis mediante realce tardío con gadolinio permite mejorar la precisión diagnóstica, aporta información pronóstica relevante y orienta las decisiones terapéuticas, así como la evaluación familiar en pacientes con sospecha de enfermedad de origen idiopático o genético.

Palabras clave: miocardiopatía dilatada; insuficiencia cardíaca; imagen por resonancia magnética; gadolinio; fibrosis miocárdica; reporte de caso

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INTRODUCTION

Dilated cardiomyopathy (DCM) is a leading cause of heart failure with reduced ejection fraction and a frequent indication for heart transplantation in young adults. From the initial classifications proposed by the World Health Organization and the International Society and Federation of Cardiology to the recent update by the European Society of Cardiology (ESC) in 2023, the concept of cardiomyopathy has evolved from a definition based on morphological criteria to a comprehensive approach that incorporates clinical, genetic, molecular, and cardiovascular imaging aspects, allowing for a better etiological and prognostic characterization of these diseases (1,2).

Currently, DCM is defined as a myocardial disorder characterized by ventricular dilation and systolic dysfunction not explained by significant coronary artery disease, hypertension, valvular disease, or congenital heart disease. Its etiology is heterogeneous and includes genetic, inflammatory, infectious, metabolic, toxic, autoimmune, and infiltrative causes; however, a significant number of patients remain without an identifiable cause after a complete evaluation, a condition traditionally termed idiopathic dilated cardiomyopathy (1-3). The estimated prevalence is approximately 1 per 250-500 inhabitants and an annual incidence of 5 to 8 cases per 100 000 persons, making it an important cause of heart failure, ventricular arrhythmias, and sudden death, especially in young and middle-aged adults (2-5). Furthermore, the growing recognition of genetic variants has shown that between 20 % and 40 % of cases previously considered idiopathic exhibit familial aggregation, which has reinforced the importance of studying first-degree relatives (2,4,5).

The diagnosis of MCD requires a multimodal approach. Echocardiography remains the initial study to assess cardiac structure and function; however, cardiac magnetic resonance (RMC) has become the reference method for non-invasive tissue characterization. The identification of fibrosis by late gadolinium enhancement allows differentiation between ischemic and non-ischemic etiologies, establishes the degree of myocardial remodeling, stratifies the risk of ventricular arrhythmias and sudden death, and provides prognostic information that directly influences therapeutic decision-making (1,6,8). In particular, the pattern of linear mesocardial enhancement located in the interventricular septum is a highly suggestive finding of non-ischemic dilated cardiomyopathy and is associated with a less favorable clinical outcome (6,8).

In this context, the present case is relevant because it demonstrates the increasing contribution of cardiac magnetic resonance imaging (CMR) in confirming the etiology of a patient with newly diagnosed heart failure and ruled-out coronary artery disease. From a theoretical standpoint, it strengthens the evidence regarding the value of tissue characterization in the evaluation of cardiomyopathies; from a practical perspective, it highlights its usefulness for prognostic stratification, therapeutic planning, and the indication for family screening; and methodologically, it follows the international CARE guidelines for reporting clinical cases, promoting the quality and reproducibility of scientific information (9,10). Therefore, the objective of this report is to describe the diagnostic process and clinical course of a patient with idiopathic dilated cardiomyopathy, emphasizing the role of cardiac magnetic resonance imaging in etiological characterization, prognostic stratification, and guiding comprehensive clinical management.

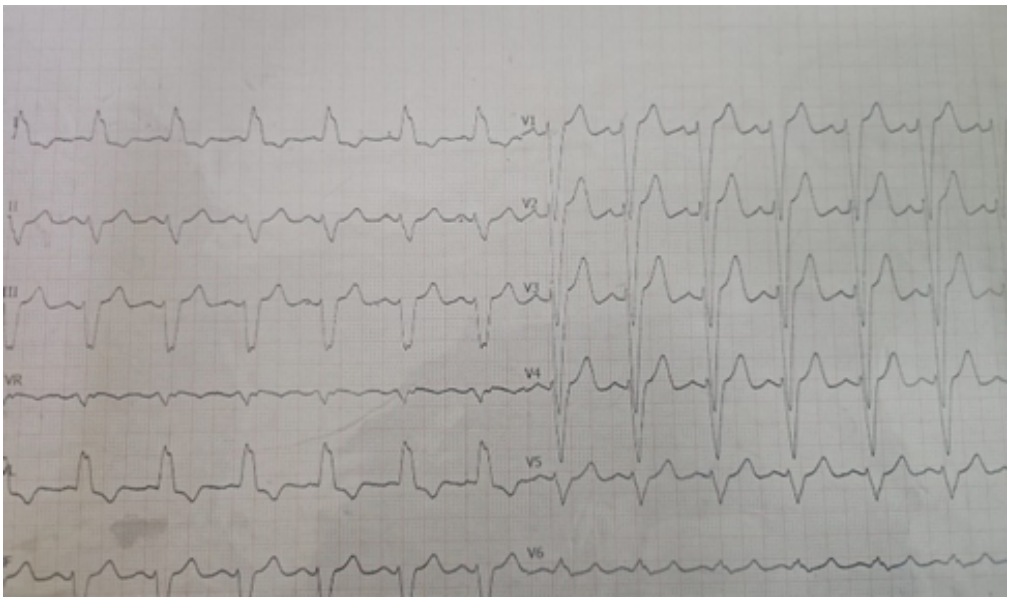
Case Presentation

We present the case of a 43-year-old woman with a history of bronchial asthma and overweight, with no known history of hypertension, diabetes mellitus, coronary artery disease, or previous structural heart disease. She presented with a condition of approximately three months' duration characterized by progressive dyspnea initially associated with significant exertion, which progressed to limit usual activities (New York Heart Association functional class III), accompanied by orthopnea, episodes of paroxysmal nocturnal dyspnea, exercise intolerance, asthenia, and progressive lower limb edema. The patient denied chest pain, syncope, fever, significant alcohol consumption, or known exposure to cardiotoxic agents.

Upon admission, the patient was hemodynamically stable. Physical examination revealed jugular distension, bibasilar crackles, pitting edema in both lower limbs, and lateral displacement of the apex beat, findings consistent with congestive heart failure. Cardiac auscultation revealed a regular rhythm without significant murmurs.

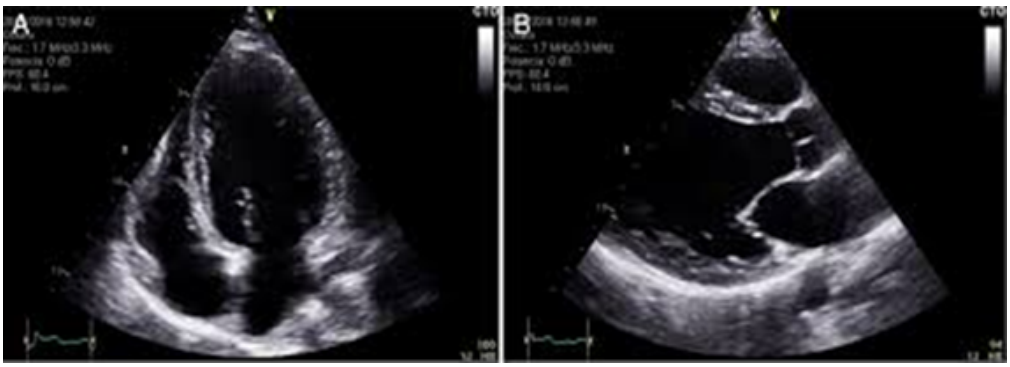
As part of the initial evaluation, an electrocardiogram (ECG) was performed, which showed sinus rhythm with complete left bundle branch block, without changes suggestive of acute ischemia (Figure 1).

Figure 1. ECG showing sinus rhythm with HR 100 bpm and complete left bundle branch block, with ventricular repolarization abnormalities.



The transthoracic echocardiogram showed dilation of all four cardiac chambers, predominantly involving the left ventricle, with a left ventricular ejection fraction estimated at 35% by the biplane Simpson method and global hypokinesis, without segmental wall motion abnormalities. No clinically significant valvular heart disease or findings compatible with congenital heart disease were identified, confirming a pattern of dilated cardiomyopathy with severe systolic dysfunction (Figure 2).

Figure 2. The 4-chamber view shows a spheroid pattern, increased volumes, and EF 35% by the Simpson method; the parasternal long-axis view shows increased diameters and decreased wall thicknesses.



Given the absence of segmental contractility alterations but the need to exclude coronary artery disease as a cause of ventricular dysfunction, diagnostic coronary angiography was performed. The study showed angiographically normal coronary arteries without significant obstructive lesions, ruling out an ischemic etiology (Figure 3).

Once ischemic heart disease was ruled out, the etiological study was completed using cardiac magnetic resonance imaging. The examination confirmed the left ventricular dilation and reduced systolic function previously documented by echocardiography. The late gadolinium enhancement sequence showed a mesocardial linear pattern located in the interventricular septum, without subendocardial or transmural distribution, a finding characteristic of non-ischemic dilated cardiomyopathy and highly suggestive of myocardial fibrosis (Figure 4a and b).

Figure 3. Coronary angiography without significant lesions with slow TIMI II flow, also confirms myopathy and deterioration of systolic function; anterior oblique view A and right coronary artery view B.

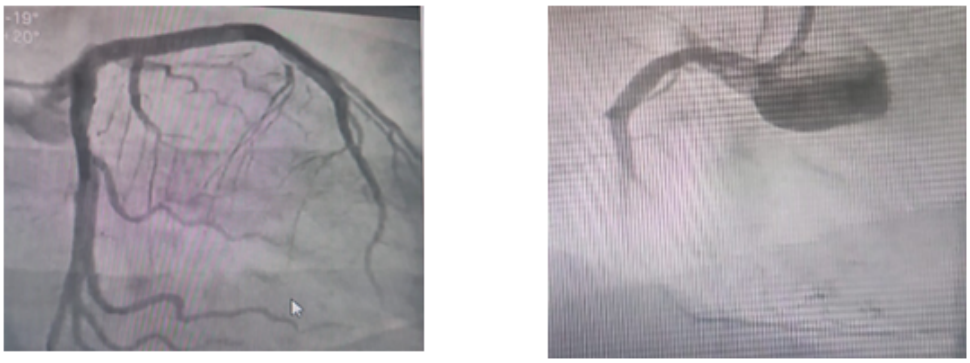
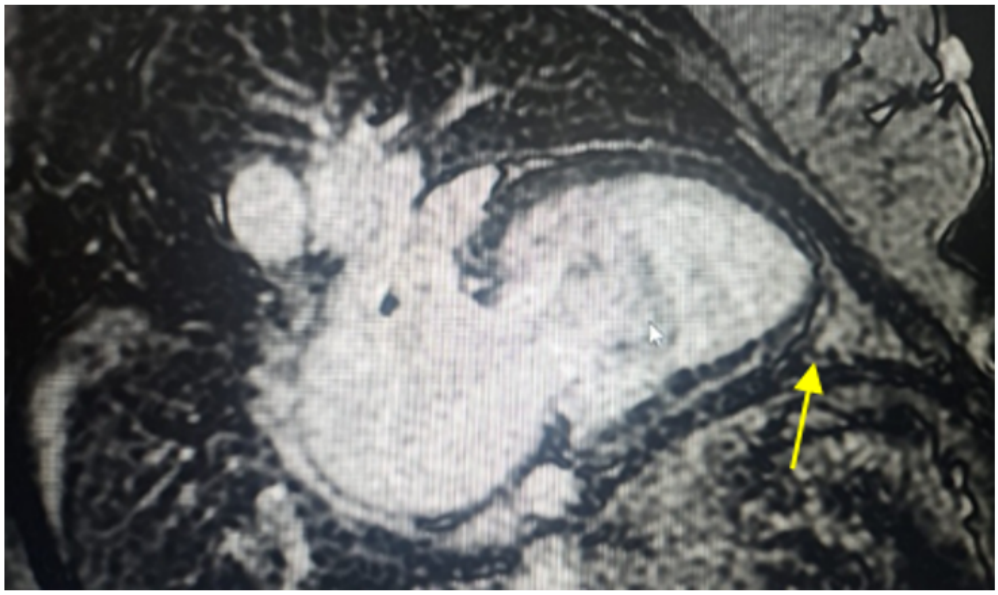


Figure 4a. Cardiac MRI with gadolinium. Abnormal myocardial viability study associated with idiopathic dilated cardiomyopathy with linear mid-septal mesocardial late enhancement; myocardial fibrosis of 0.9% corresponding to 1.65% of the mass; LVEF 28% (LV), RV 24% (see yellow arrow).

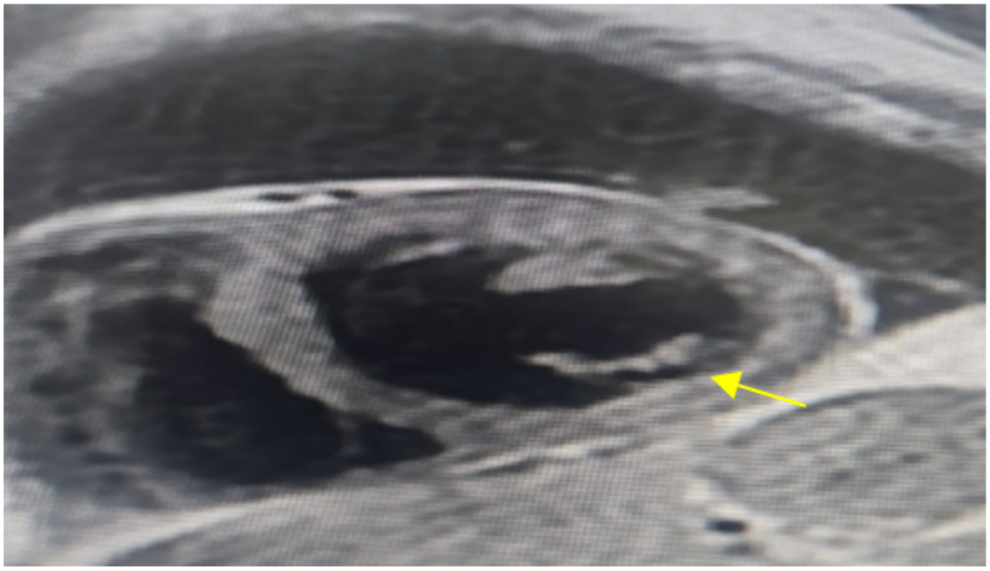


Clinical and imaging findings, together with the exclusion of coronary artery disease, valvular disease, and other structural causes, allowed the diagnosis of idiopathic dilated cardiomyopathy to be established. Guideline-directed medical therapy for heart failure with reduced ejection fraction was initiated, including a renin-angiotensin system inhibitor, beta-blocker, mineralocorticoid receptor antagonist, sodium-glucose cotransporter 2 inhibitor, and diuretics, with progressive adjustment based on clinical tolerance.

During hospitalization, the patient demonstrated progressive improvement in congestion, with an adequate response to diuretic therapy, resolution of dyspnea at rest, and reduction of peripheral edema, without episodes of sustained ventricular arrhythmias or other cardiovascular complications.

As part of prognostic stratification, ambulatory monitoring with 24-hour Holter was indicated to search for ventricular arrhythmias, as well as echocardiographic reassessment after 3 months of optimized medical therapy to evaluate the persistence of systolic dysfunction and determine the indication for an implantable cardioverter-defibrillator for primary prevention of sudden death, according to current international recommendations. Subsequent evolution was favorable. Likewise, considering the possibility of a genetic etiology, clinical and echocardiographic evaluation of first-degree relatives was recommended.

Figure 4b. Short-axis view shows late enhancement in the medial portion of the left ventricle (see yellow arrow).



Discussion

Dilated cardiomyopathy (DCM) is one of the most frequent causes of heart failure with reduced ejection fraction and remains a significant public health problem due to its high morbidity and mortality and its association with ventricular arrhythmias, sudden death, and heart transplantation. Although traditionally considered an idiopathic entity in a high percentage of patients, contemporary classifications have modified this concept by incorporating genetic, phenotypic, and imaging criteria that allow for a more precise etiological characterization. Consequently, current European Society of Cardiology guidelines recommend initially using the term non-ischemic dilated cardiomyopathy when obvious structural causes have been ruled out, reserving the term idiopathic for those cases in which a comprehensive clinical evaluation, including genetic testing when available, fails to identify a specific etiology (11,12). In our case, the absence of coronary, valvular, hypertensive, or congenital heart disease, along with the findings of cardiac magnetic resonance imaging, allowed us to establish the diagnosis of non-ischemic dilated cardiomyopathy with a high suspicion of idiopathic origin.

One of the main contributions of this case lies in highlighting the importance of a stepwise and comprehensive diagnostic approach. Transthoracic echocardiography remains the first-line tool for confirming ventricular dilation, quantifying systolic function, and identifying associated structural abnormalities; however, it has limitations in establishing the etiology of myocardial damage. In our patient, the echocardiogram documented dilation of all four chambers and a left ventricular ejection fraction of 35%, initially suggesting dilated cardiomyopathy, but without defining its origin. These findings are consistent with the recommendations of the American Society of Echocardiography, which recognizes echocardiography as the indispensable initial study, although insufficient for the tissue characterization of the myocardium (13).

The exclusion of coronary artery disease is an essential step in the evaluation of patients with recently diagnosed ventricular systolic dysfunction. In this context, coronary angiography remains the reference method for ruling out obstructive coronary disease when the clinical probability is intermediate or high, or when clinical findings cannot exclude an ischemic etiology. In the present case, the absence of significant coronary lesions allowed the investigation to be directed toward non-ischemic causes, avoiding misdiagnosis and facilitating the indication of targeted complementary studies (14).

The most relevant finding of the present report was the identification of mesocardial fibrosis by cardiac magnetic resonance imaging with late gadolinium enhancement. Over the last decade, CMR has ceased to be considered merely a complementary technique and has become a fundamental component of the diagnostic algorithm for cardiomyopathies.

In addition to providing highly reproducible quantification of ventricular volumes and ejection fraction, it allows characterization of myocardial tissue through the identification of edema, fatty infiltration, iron overload, and fibrosis. In nonischemic DCM, the pattern of linear mesocardial enhancement predominantly located in the interventricular septum is among the most characteristic findings and is associated with progressive ventricular remodeling and worse clinical outcomes (11,15).

Several studies have demonstrated that the presence of fibrosis detected by late gadolinium enhancement has significant prognostic value. Halliday and colleagues showed that patients with DCM and myocardial fibrosis have a significantly higher risk of malignant ventricular arrhythmias, sudden death, and hospitalization for heart failure, independently of left ventricular ejection fraction (16). Similarly, de Frutos F. et al. highlight that CMR has acquired a central role in risk stratification, allowing the identification of patients who could benefit from closer follow-up or advanced therapeutic strategies even before developing significant clinical deterioration (15). In our patient, the pattern of mesocardial fibrosis provided diagnostic and prognostic information that could not be obtained by echocardiography or coronary angiography.

Another relevant aspect is treatment. In recent years, the pharmacological management of heart failure with reduced ejection fraction has undergone a substantial change with the incorporation of guideline-directed medical therapy (GDMT), based on the early combination of renin-angiotensin system inhibitors or angiotensin receptor-neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter 2 inhibitors. Available evidence demonstrates that this strategy reduces cardiovascular mortality and hospitalizations, and promotes reverse ventricular remodeling when initiated early and progressively optimized (17). The favorable clinical course observed in our patient after starting medical therapy supports these findings and highlights the importance of early initiation of evidence-based therapies.

The follow-up of these patients represents another essential component of comprehensive management. Current guidelines recommend reassessing ventricular function after 3 to 6 months of optimized treatment before deciding on the implantation of an implantable cardioverter-defibrillator (ICD) for primary prevention of sudden death, except in particular situations. Likewise, ambulatory monitoring via Holter allows the detection of potentially malignant ventricular arrhythmias that can modify risk stratification and therapeutic approach (11,17). In this context, the planning carried out in our patient was consistent with current international recommendations.

The growing knowledge of the genetic basis of cardiomyopathies has also modified the diagnostic approach. It is now recognized that a considerable proportion of patients previously classified as idiopathic harbor pathogenic variants in genes related to the cytoskeleton, sarcomere, or nuclear envelope, with TTN, LMNA, DSP, and FLNC being some of the most frequently implicated genes. Therefore, recent guidelines recommend clinical and, when possible, genetic evaluation of first-degree relatives, a strategy that allows the identification of presymptomatic individuals and the establishment of early surveillance programs (12,18). Although genetic testing was not possible in the present case, this limitation does not diminish the relevance of the report; on the contrary, it highlights a common reality in middle-income countries, where the availability of these tests remains limited.

As limitations, it must be acknowledged that the absence of genetic confirmation prevented establishing the underlying etiological mechanism with certainty, so the diagnosis of idiopathic dilated cardiomyopathy must be interpreted within the available clinical and imaging context. Likewise, the nature of a case report limits the possibility of extrapolating the findings to other populations. However, the main strength lies in the sequential application of different diagnostic modalities, particularly cardiac magnetic resonance imaging, which allowed the integration of clinical and imaging findings to reach an etiological diagnosis with prognostic and therapeutic implications.

In conclusion, this case demonstrates that the contemporary approach to dilated cardiomyopathy transcends the simple demonstration of ventricular systolic dysfunction. The integration of echocardiography, systematic exclusion of coronary artery disease, and especially tissue characterization by cardiac magnetic resonance allows for a more precise diagnosis, optimal prognostic stratification, and guidance of decisions related to medical treatment, device indication, clinical follow-up, and family evaluation. These advances reflect the paradigm shift promoted by current classifications and consolidate cardiac magnetic resonance as an indispensable tool in the modern management of nonischemic dilated cardiomyopathy.

REFERENCES

1. Arbelo E, Protonotarios A, Gimeno JR, Arbustini E, Barriales-Villa R, Basso C, et al; ESC Scientific Document Group. 2023 ESC Guidelines for the management of cardiomyopathies. Eur Heart J. 2023 Oct 1;44(37):3503-3626. doi: 10.1093/eurheartj/ehad194.
2. Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, et al. Classification of the cardiomyopathies: a position statement from the European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2008 Jan;29(2):270-6. doi: 10.1093/eurheartj/ehm342.
3. Hershberger RE, Givertz MM, Ho CY, Judge DP, Kantor PF, McBride KL, et al. Genetic Evaluation of Cardiomyopathy-A Heart Failure Society of America Practice Guideline. J Card Fail. 2018 May;24(5):281-302. doi: 10.1016/j.cardfail.2018.03.004.
4. Schultheiss HP, Fairweather D, Caforio ALP, Escher F, Hershberger RE, Lipshultz SE, et al. Dilated cardiomyopathy. Nat Rev Dis Primers. 2019 May 9;5(1):32. doi: 10.1038/s41572-019-0084-1.
5. Ushasree B, Shivani V, Venkateshwari A, Jain RK, Narsimhan C, Nallari P. Epidemiology and genetics of dilated cardiomyopathy in the Indian context. Indian J Med Sci. 2009 Jul;63(7):288-96.
6. Yang EY, Shah DJ. Cardiac Magnetic Resonance in Nonischemic Cardiomyopathies. Methodist Debakey Cardiovasc J. 2020 Apr-Jun;16(2):97-105. doi: 10.14797/mdcj-16-2-97.
7. Morarji K, Brown TD, Ismail NA, Dweck MR, Di Pietro E, et al. Association of fibrosis with mortality and sudden cardiac death in patients with nonischemic dilated cardiomyopathy. JAMA. 2013 Mar 6;309(9):896-908. doi: 10.1001/jama.2013.1363.
8. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, et al. Cardiovascular Magnetic Resonance in Nonischemic Myocardial Inflammation: Expert Recommendations. J Am Coll Cardiol. 2018 Dec 18;72(24):3158-3176. doi: 10.1016/j.jacc.2018.09.072.
9. Ingles J, Goldstein J, Thaxton C, Caleshu C, Corty EW, Crowley SB, et al. Evaluating the Clinical Validity of Hypertrophic Cardiomyopathy Genes. Circ Genom Precis Med. 2019 Feb;12(2): e002460. doi: 10.1161/CIRCGEN.119.002460.
10. Riley DS, Barber MS, Kienle GS, Aronson JK, von Schoen-Angerer T, Tugwell P, et al. CARE guidelines for case reports: explanation and elaboration document. J Clin Epidemiol. 2017 Sep; 89:218-235. doi: 10.1016/j.jclinepi.2017.04.026.
11. Goff ZD, Calkins H. Sudden death related cardiomyopathies - Arrhythmogenic right ventricular cardiomyopathy, arrhythmogenic cardiomyopathy, and exercise-induced cardiomyopathy. Prog Cardiovasc Dis. 2019 May-Jun;62(3):217-226. doi: 10.1016/j.pcad.2019.04.002.
12. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al. ESC Scientific Document Group. 2023 Focused Update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) With the special contribution of the Heart Failure Association (HFA) of the ESC. Eur J Heart Fail. 2024 Jan;26(1):5-17. doi: 10.1002/ejhf.3024.
13. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015 Jan;28(1):1-39.e14. doi: 10.1016/j.echo.2014.10.003.
14. Knuuti J, Wijns W, Saraste A, Capodanno D, Barbato E, Funck-Brentano C, et al. ESC Scientific Document Group. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. Eur Heart J. 2020 Jan 14;41(3):407-477. doi: 10.1093/eurheartj/ehz425.
15. de Frutos F, Ochoa JP, Fernández AI, Gallego-Delgado M, Navarro-Peñalver M, Casas G, et al. Late gadolinium enhancement distribution patterns in non-ischaemic dilated cardiomyopathy: genotype-phenotype correlation. Eur Heart J Cardiovasc Imaging. 2023 Dec 21;25(1):75-85. doi: 10.1093/ehjci/jead184.
16. Halliday BP, Gulati A, Ali A, Guha K, Newsome S, Arzanauskaite M, et al. Association Between Midwall Late Gadolinium Enhancement and Sudden Cardiac Death in Patients with Dilated Cardiomyopathy and Mild and Moderate Left Ventricular Systolic Dysfunction. Circulation. 2017 May 30;135(22):2106-2115. doi: 10.1161/CIRCULATIONAHA.116.026910.
17. Al-Sadawi M, Aslam F, Tao M, Fan R, Singh A, Rashba E. Association of late gadolinium enhancement in cardiac magnetic resonance with mortality, ventricular arrhythmias, and heart failure in patients with nonischemic cardiomyopathy: A systematic review and meta-analysis. Heart Rhythm O2. 2023 Jan 13;4(4):241-250. doi: 10.1016/j.hroo.2023.01.001.
18. Eldemire R, Mestroni L, Taylor MRG. Genetics of Dilated Cardiomyopathy. Annu Rev Med. 2024 Jan 29; 75:417-426. doi: 10.1146/annurev-med-052422-020535.
19. Becker MAJ, van der Lingen ACJ, Cornel JH, van de Ven PM, van Rossum AC, Allaart CP, Germans T. Septal Midwall Late Gadolinium Enhancement in Ischemic Cardiomyopathy and Nonischemic Dilated Cardiomyopathy-Characteristics and Prognosis. Am J Cardiol. 2023 Aug 15; 201:294-301. doi: 10.1016/j.amjcard.2023.06.042.
20. Augusto JB, Eiros R, Nakou E, Moura-Ferreira S, Treibel TA, Captur G, et al. Dilated cardiomyopathy and arrhythmogenic left ventricular cardiomyopathy: a comprehensive genotype-imaging phenotype study. Eur Heart J Cardiovasc Imaging. 2020 Mar 1;21(3):326-336. doi: 10.1093/ehjci/jez188.

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