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Utilities and Limitations of Cardiac Magnetic Resonance Imaging in Dilated Cardiomyopathy

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Dilated cardiomyopathy (DCM) is one of the most common types of non-ischemic cardiomyopathy. DCM is characterized by left ventricle (LV) dilatation and systolic dysfunction without coronary artery disease or abnormal loading conditions. DCM is not a single disease entity and has a complex historical background of revisions and updates to its definition because of its diverse etiology and clinical manifestations. In cases of LV dilatation and dysfunction, conditions with phenotypic overlap should be excluded before establishing a DCM diagnosis. The differential diagnoses of DCM include ischemic cardiomyopathy, valvular heart disease, burned-out hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, and non-compaction. Cardiac magnetic resonance (CMR) imaging is helpful for evaluating DCM because it provides precise measurements of cardiac size, function, mass, and tissue characterization. Comprehensive analyses using various sequences, including cine imaging, late gadolinium enhancement imaging, and T1 and T2 mapping, may help establish differential diagnoses, etiological work-up, disease stratification, prognostic determination, and follow-up procedures in patients with DCM phenotypes. This article aimed to review the utilities and limitations of CMR in the diagnosis and assessment of DCM.

Keywords: Cardiomyopathy, Dilated; Myocardium; Heart failure; Ventricular dysfunction; Magnetic resonance imaging

Historical Review of the Various Classifications of Cardiomyopathy and the Definitions of Dilated Cardiomyopathy

The classifications of cardiomyopathy and the definition of dilated cardiomyopathy (DCM) have changed in recent decades, with several addendums and updates to various international guidelines [1-7]. The 1980 World Health Organization (WHO)/International Society and Federation of Cardiology (ISFC) Task Force defined cardiomyopathy as a heart muscle disease of unknown cause and classified it into three major types, namely, DCM, hypertrophic cardiomyopathy (HCM), and restrictive cardiomyopathy (RCM) [1]. In 1995, the WHO/ISFC Task Force included arrhythmogenic right ventricular cardiomyopathy (ARVC) as a new condition. DCM is characterized by the dilatation

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and impaired contraction of the left ventricle (LV) or both ventricles that cannot be explained by abnormal loading conditions or ischemic damage [2].

In 2006, the American Heart Association (AHA) proposed a classification system that divided cardiomyopathies into two groups according to the predominantly involved organ: primary cardiomyopathy (genetic, non-genetic, and acquired; confined to the heart muscle) and secondary cardiomyopathy (myocardial involvement in patients with systemic disorders) [3]. DCM was classified as a primary cardiomyopathy, which could be genetic or non-genetic, and showed ventricular enlargement and systolic dysfunction with a normal LV wall thickness. In 2008, the European Society of Cardiology (ESC) Working Group proposed a clinically oriented classification based on morphological and functional phenotypes, including HCM, DCM, ARVC, RCM, and unclassified cardiomyopathy, which were subclassified into familiar and non-familial forms [4]. The ESC Working Group defined DCM as the presence of LV dilatation and systolic dysfunction in the absence of abnormal loading conditions (such as hypertension or valvular disease) or coronary artery disease that is sufficient to cause global systolic impairment.

In 2016, the ESC Working Group updated the clinicopathological definition of DCM: it was defined as a progressive disease with LV or biventricular systolic dysfunction and dilatation that cannot be explained by abnormal loading conditions or coronary artery disease [5]. Considering the clinical spectrum and progressive expression of the phenotype in some cases of DCM, they created a new category called hypokinetic nondilated cardiomyopathy, which was defined as systolic dysfunction without dilatation. In 2013, the World Heart Federation proposed the MOGE(S) nosology system, which incorporated the morpho-functional phenotype (M); organ(s) involvement (O); genetic inheritance pattern (G); etiologic annotation (E), including genetic defects or underlying disease/substrate; and functional status (S) of the disease, by using both the ACC/AHA heart failure stages and the New York Heart Association functional class [6]. Owing to advances in the knowledge of the genetic basis of cardiomyopathy, they insisted that the conventional cardiomyopathy classification based on morphological phenotypes may not be correlated with the results of gene analysis and pre-clinical diagnoses in family members. Furthermore, they suggested that this descriptive genotype–phenotype nosology system might provide flexibility for such potential transitions.

The latest guideline by ESC was released in 2023, which included HCM, DCM, non-dilated left ventricular cardiomyopathy (NDLVC), RCM, and ARVC for cardiomyopathy phenotype classification [7]. This new guideline adopted both morphological and functional traits to describe the cardiomyopathy phenotypes and highlighted the key roles of ventricular myocardial scar assessment and myocardial tissue characterization using cardiac magnetic resonance (CMR) imaging. It specifically stated the clinical values of CMR in diagnosis, monitoring of disease progression, and risk stratification in each of the main cardiomyopathy phenotypes.

Regarding the specific diagnostic criteria for "systolic dysfunction" and "LV dilatation," the 2016 ESC position statement defined systolic dysfunction as an LV ejection fraction (LVEF) of < 45% and defined LV dilatation as an LV end-diastolic volume or diameter of more than two standard deviations from the normal [5]. However, absolute cutoff values have not been clearly documented in other classification systems (Table 1). The 2021 ESC guidelines on heart failure divided LV dysfunction into three categories: reduced LVEF (≤ 40%), mildly reduced LVEF (41%–49%), and preserved LVEF (≥ 50%) [8].

In the 2023 ESC guidelines for the management of cardiomyopathies, LV dilatation is defined by LV end-diastolic dimensions or volumes > 2 z-scores above population mean values corrected for body size, sex, and/or age. For adults, this represents an LV end-diastolic diameter of > 58 mm in males and > 52 mm in females and an LV end-diastolic volume index of ≥ 75 mL/m^2 in males and ≥ 62 mL/m^2 in females by echocardiography. LV global systolic dysfunction is defined by LVEF < 50% [7].

Table 1 presents the evolution of various proposed classification systems for cardiomyopathies and the definitions and descriptions of DCM. These classification systems aim to improve the understanding of the disease and facilitate communication with physicians.

CMR: Findings and Clinical Usefulness

CMR imaging can be used to comprehensively evaluate and understand DCM by using various sequences (Table 2).

Cine Imaging

Echocardiography or CMR is recommended for assessing LV or biventricular systolic dysfunction [5]. For example, in the diagnostic criteria suggested by the ESC, systolic

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Table 1. Historical review of the classification of cardiomyopathies and definitions of DCM

Guidelines	Classification of cardiomyopathies	Definition of DCM
1980 WHO/ISFC task force	DCM HCM RCM	Dilatation of LV or RV, or both Impaired systolic ventricular function
1995 WHO/ISFC task force	DCM HCM RCM ARVD	Dilatation and impaired contraction of LV or both ventricles, not explained by the abnormal loading conditions or the extent of ischemic damage
2006 AHA Scientific Statement: From the Council on Clinical Cardiology, Heart Failure, and Transplantation Committee	Primary: solely or predominantly confined to the heart muscle Secondary: myocardial involvement of generalized systemic (multiorgan) disorders	Primary cardiomyopathy, showing ventricular chamber enlargement and systolic dysfunction with normal LV wall thickness
2008 ESC working group on myocardial and pericardial diseases	HCM DCM RCM ARVC Unclassified	LV dilatation and LV systolic dysfunction in the absence of abnormal loading conditions (hypertension, valve disease) or coronary artery disease sufficient to cause global systolic impairment RV dilation and dysfunction may be present but are unnecessary for the diagnosis
2013 World Heart Federation	MOGE(S) classification M: morpho-functional phenotype O: Organ involvement G: Genetic inheritance pattern E: Etiological annotation S: Functional status	Presence of LV dilation and LV systolic dysfunction in the absence of other disorders sufficient to cause global systolic impairment Right ventricular dilation and dysfunction can be present, but not necessary, for the diagnosis Example for MOGE(S) nomenclature for DCM: $M_{D[AVB]}O_{H}G_{AD}E_{G\text{-}LMNA[p.Leu197ProfsX2]}S_{C\text{-}II}$ - M: dilated cardiomyopathy (D) with an atrioventricular block (AVB) - O: heart (H) involvement - G: autosomal dominant (AD) transmission - E: genetic and caused by the *p.Leu197Pro fs X2* mutation in the *LMNA* gene - S: ACC/AHA stage C, NYHA II
2016 ESC working group on myocardial and pericardial diseases	A new concept of hypokinetic non-dilated cardiomyopathy (HNDC)	- LV or biventricular systolic dysfunction (LVEF < 45%) - LV dilatation (LV end-diastolic volumes of diameters > 2 SD from normal, corrected by age and body surface area) - Not explained by abnormal loading conditions or coronary artery disease
2023 ESC guidelines for the management of cardiomyopathies	HCM DCM NDLVC RCM ARVC	LV dilatation and global or regional systolic dysfunction unexplained solely by abnormal loading conditions (hypertension, valve disease, congenital heart disease) or coronary artery disease Right ventricular dilatation and dysfunction may be present but are not necessary for the diagnosis LV dilatation with normal LVEF in the absence of athletic remodeling or other environmental factors is not in itself a cardiomyopathy but may represent an early manifestation of DCM, and the preferred term for this is isolated left ventricular dilatation LV dilatation for adults is defined by LV end-diastolic diameter > 58 mm in males and > 52 mm in females and LV end-diastolic volume index of ≥ 75 mL/m^2 in males and ≥ 62 mL/m^2 in females by echocardiography LV global systolic dysfunction is defined by LVEF < 50%

DCM = dilated cardiomyopathy, WHO = World Health Organization, ISFC = International Society and Federation of Cardiology, HCM = hypertrophic cardiomyopathy, RCM = restrictive cardiomyopathy, LV = left ventricle, RV = right ventricle, ARVD = arrhythmogenic right ventricular dysplasia, AHA = American Heart Association, ESC = European Society of Cardiology, ARVC = arrhythmogenic right ventricular cardiomyopathy, ACC = American College of Cardiology, NYHA = New York Heart Association, LVEF = left ventricular ejection fraction, SD = standard deviation, NDLVC = non-dilated left ventricular cardiomyopathy

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Table 2. Clinical usefulness of CMR in patients with DCM

CMR sequence	Usefulness
Cine imaging	Precise measurement of ventricular volume and function
	Precise measurement of EF for ICD implantation
	CMR-FT for diagnosis and prognostication
LGE imaging	Mid-wall LGE as a well-known outcome predictor
	Differential diagnosis based on LGE patterns
T1 mapping	ECV as an independent predictor of poor outcomes
	ECV as an incremental prognostic factor to LGE
	Native T1 value and ECV as markers for differentiating DCM from phenocopies

CMR = cardiac magnetic resonance, DCM = dilated cardiomyopathy, EF = ejection fraction, ICD = implantable cardioverter-defibrillator, FT = feature tracking, LGE = late gadolinium enhancement, ECV = extracellular volume

dysfunction, which is defined by abnormal LVEF, can be measured using any modality; however, LVEF abnormality should be shown either by two independent imaging modalities or on two distinct occasions using the same technique (preferably echocardiography or CMR) [9]. Cine imaging can be used to assess segmental wall-motion abnormalities in the LV or in LV dilatation and LVEF. In patients with DCM that are expected to survive substantially longer than one year, have good functional status, and show LVEF ≤ 35% despite more than three months of optimal medical therapy, an implantable cardioverter-defibrillator (ICD) is recommended to reduce the risk of sudden death and all-cause mortality [8]. According to the literature, the use of CMR to measure LVEF (CMR-LVEF) is an accurate and useful approach for ICD implantation [10]. The mean CMR-LVEF was significantly lower than the mean LVEF measured using transthoracic echocardiography (TTE-LVEF) (24% vs. 28%, respectively), and only the CMR-LVEF was associated with the risk of the primary composite outcome [11]. Rijnierse et al. [12] showed that CMR-LVEF measurements resulted in more patients being eligible for ICD implantation than LVEF measurements using 2D echocardiography, which showed a relatively low event rate during follow-up. Certain studies revealed a discrepancy between CMR-LVEF and TTE-LVEF measurements, and the selection of a particular imaging technique could cause the reclassification of a significant number of patients with DCM for ICD insertion [13]. These results suggest the critical role of CMR, and further prospective studies are needed to implement new guidelines for appropriate indications for ICD insertion in DCM.

CMR strain parameters such as global longitudinal strain (GLS), circumferential strain, and radial strain have emerged as novel biomarkers for detecting early myocardial changes [14]. Myocardial strain can be accomplished by feature tracking (FT) using cine sequences or dedicated strain-encoded sequences with high accuracy without intravenous gadolinium injection [15]. The clinical utility of CMR-FT has broadened with increasing reproducibility, and a growing body of evidence shows a significant correlation with CMR combined with late gadolinium enhancement (LGE) [16]. Recent studies have shown that CMR strain parameters can be used as early markers for the diagnosis and prediction of myocardial fibrosis in DCM [17]. Kammerlander et al. [18] reported that the GLS from CMR-FT was associated with the extracellular volume (ECV) fraction and was related to major adverse cardiac events in patients with heart failure and preserved LVEF. Similarly, Buss et al. [19] showed that GLS is an independent prognostic index for patients with non-ischemic DCM.

LGE Imaging

LGE patterns help identify various types of cardiomyopathy, and the underlying etiology of non-ischemic cardiomyopathy has important prognostic implications [20]. Approximately 60%–90% of DCM cases show no hyperenhancement on LGE images, whereas hyperenhancement in the mid-wall unrelated to the specific coronary perfusion territory is the most common pattern (10%–35%). Mid-wall LGE is a well-known predictor of all-cause mortality, cardiovascular hospitalization, sudden cardiac death (SCD), and ventricular tachycardia [21]. However, multiple LGE patterns (subepicardial, focal, or multifocal) and LGE locations (septum and free wall of the LV) have been identified in DCM cases. The risk of SCD has been reported to be greatest with concomitant septal and free-wall LGE. Furthermore, the presence of septal LGE was associated with a significant increase in the risk of death and SCD events even when the extent was small [22]. Careful evaluation using LGE imaging is necessary for the differential diagnosis and stratification of patients with LV dilatation and dysfunction.

Mapping Imaging

Recently, T1 mapping techniques have provided quantitative T1 values, and the ECV fractions of the LV could be calculated as follows [23]:

$$ECV = (\Delta R1 \text{ of myocardium} / \Delta R1 \text{ of LV blood pool}) \times (1 - \text{hematocrit level}),$$

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where R1 = 1 / T1, and ΔR1 = (post-contrast R1 - pre-contrast R1). ECV reflects the changes in the interstitial space, and increased ECV is associated with diffuse myocardial fibrosis [24]. DCM cases that have been histologically validated for diffuse fibrosis show increased ECV fraction in the LV [25]. ECV has been correlated with adverse cardiac events and demonstrated to be an independent indicator of poor outcomes in patients with DCM [26]. One study reported that ECV offered prognostication for outcomes incremental to LGE [27]; among 240 patients with DCM, LGE patterns were detected in 34% of patients, whereas ECV was elevated (≥ 1 location) in 58% of patients. ECV values from all six locations were associated with adverse events and death, with the anteroseptal wall values being the most significant. There was a 2.8-fold adjusted increase in the risk of adverse outcomes for every 10% increase in mean ECV.

Patients with early DCM can be differentiated from those with an athlete's heart (both show LV dilatation and mildly decreased LVEF [45%–55%]) by using native T1, ECV, and T2 relaxation times, all of which are significantly increased in patients with DCM compared with controls and exercisers (native T1 values showed the best differentiation) [28].

Causes of DCM

DCM has various etiologies, and an etiological work-up is essential for proper management, relapse prevention, and prognosis determination. Although etiological evaluation based on CMR findings may be limited owing to similarities in histological changes, it is necessary to inform patients of the possible causes and for clinicians to understand the diagnostic criteria and characteristics of DCM.

Idiopathic DCM

DCM is the second most common type of cardiomyopathy after HCM. However, Codd et al. [29] reported that the age- and sex-adjusted prevalence rates of DCM and HCM were 36.5/100000 and 19.7/100000, respectively. Hershberger et al. [30] reported that the prevalence might be higher than that reported in previous studies. The underlying causes of DCM are diverse, with approximately 50% of cases being idiopathic. Advances in genetic sequencing and diagnostic methods have decreased the proportion of idiopathic DCMs [31] because cases are reclassified when the underlying etiologies can be precisely evaluated (Fig. 1).

Genetic Causes

In the early 1990s, familial DCM was reported to constitute approximately 20% of all DCM cases [32]; this proportion further increased to 30%–50% after the systematic cardiac screening of the relatives of patients with DCM [33]. If more than one relative has DCM that meets the clinical criteria or if a relative of a patient with DCM aged > 30 years old has an unexplained sudden death, familial DCM is diagnosed [34]. The most common form of inherited DCM is the autosomal dominant form with variable expressivity and penetrance. X-linked, autosomal recessive, and mitochondrial inheritance types are less common but may still occur [33]. Approximately 40% of patients with familial DCM have an identifiable genetic cause [35], and the prevalence of genetic mutations is greater than 10% in non-familial DCM.

To date, more than 40 chromosomal loci and DCM-related genes have been identified. DCM is genetically heterogeneous, and DCM genes encode proteins with broad cellular functions. Mutations in the genes that encode cytoskeletal proteins (e.g., desmin, metavinculin, and muscle LIM protein), sarcomeres (e.g., β-myosin heavy chain, troponin T, titin, and α-tropomyosin), desmosomal proteins (e.g., desmoplakin, desmoglein, and plakophilin), nuclear membranes (e.g., lamin A/C), mitochondria, and RNA-binding proteins have been linked to DCM [36]. Truncating mutations in the gene encoding the sarcomere protein TTN are the most frequent causes of DCM (25% of familial DCM cases and 18% of sporadic cases) [37]. Other common autosomal dominant genetic mutations included mutations in *LMNA*, which encodes lamins A and C (5%–8% of patients with DCM); *PLN*, which encodes phospholamban; *RBM20*, which encodes RNA-binding motif 20; and *SCN5A*, which encodes the sodium channel alpha unit.

To diagnose familial DCM, the evaluation should begin with an extensive and accurate assessment of the patient's family history for at least three generations, including clinical history, electrocardiogram data, and echocardiography screening [38]. The confirmation of DCM-causing mutations should lead to the genetic counseling and screening of relatives under certain circumstances, such as *LMNA* mutations, to establish the prompt monitoring and management of arrhythmias and conduction abnormalities [5]. Future family planning discussions should be conducted for patients with DCM and causative genetic mutations and their partners.

The increased clinical utilization of genetic screening makes the identification of pre-clinical or early stage

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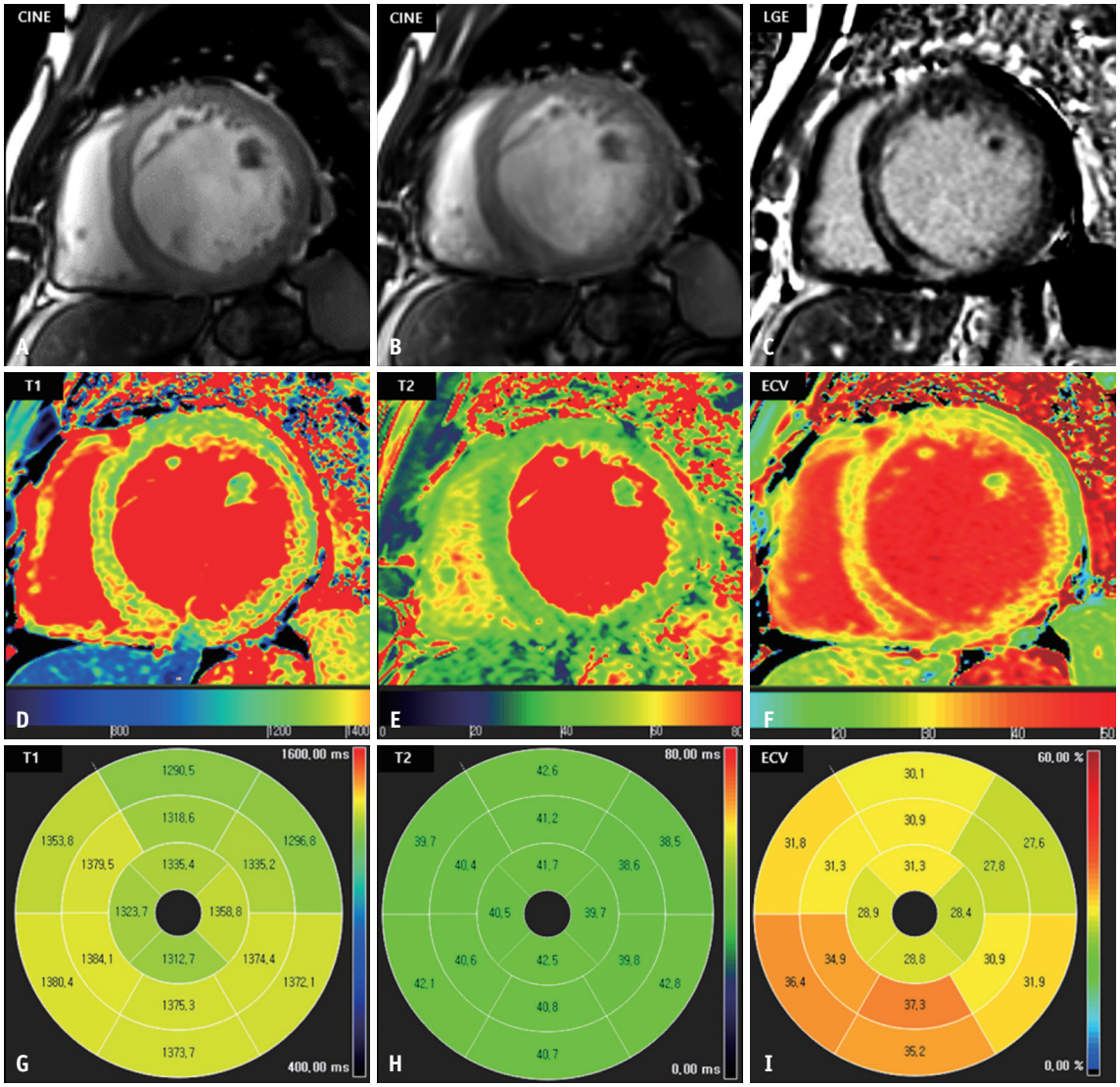

Fig. 1. Idiopathic dilated cardiomyopathy. A 44-year-old man underwent 3T cardiac MRI because of dyspnea on exertion. On cine images (**A:** short-axis image of the end-diastolic phase, **B:** short-axis image of the end-systolic phase), the LV shows diffuse severe hypokinesia. The LV end-diastolic diameter is 8.8 cm, and its ejection fraction is 16.5%. **C:** The LGE image reveals mid-wall LGE in the basal septum. The native T1 map **(D)** and bull's eye diagrams **(G)** reveal diffusely increased native T1 values of up to 1384.1 ms, and the T2 map **(E)** and bull's eye diagram **(H)** show minimally increased T2 values of up to 42.8 ms. The ECV map **(F)** and bull's eye diagram **(I)** show heterogeneously increased myocardial ECV fractions of up to 36.4%. MRI = magnetic resonance imaging, LV = left ventricle, LGE = late gadolinium enhancement, ECV = extracellular volume

DCM possible. The early diagnosis of mutation carriers who may develop overt DCM requires advanced imaging techniques that can detect subtle structural and functional abnormalities. CMR is a reference standard for evaluating ventricular size and function. During the cardiac cycle, the dyssynchrony of the septal wall motion can occur in patients with blockage of the left bundle branch [36]. Focal LGE or abnormal myocardial T1 and T2 values may be present even when the heart appears normal, thus suggesting disease involvement. Segmental LGE has been visualized in carriers of *LMNA* mutations with preserved LVEF [39]. Additionally, elevated ECV is found in *LMNA* mutation carriers with normal LV systolic function and in some individuals without LGE [40].

Toxins

Alcohol

Chronic excessive alcohol consumption can lead to non-ischemic DCM. Since Münzinger first described

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alcohol- or ethanol-induced cardiomyopathy in 1877, its pathophysiology and epidemiology have been extensively studied [41]. However, currently available data are inconclusive [42]. Although the exact amount and duration of alcohol abuse needed to produce cardiac dysfunction are unknown, it has been proven that the development of alcohol-induced cardiomyopathy is related to the amount and duration of alcohol intake (prevalence of 23%–47% in individuals with alcohol consumption > 80 g/day for at least five years) [43]. Moderate excess alcohol consumption, which is defined as > 21 units/week for men and > 14 units/week for women (1 unit of alcohol = 10 mL or 8 g of pure alcohol, an amount the average adult metabolizes in 1 h) but < 80 g/day for five years, was associated with adverse cardiac structure and function, particularly in males [44]. However, abstinence can reverse LV systolic dysfunction [45].

According to the literature, alcohol-induced cardiomyopathy is caused by intracellular structure dysfunction, myocyte hypertrophy, apoptosis, necrosis, contractile protein modification, calcium homeostasis, mitochondrial degeneration, and fibrosis [46]. These factors lead to decreased cardiac contractility and DCM with impaired function in one or both ventricles [42].

On CMR, patients with alcohol-induced cardiomyopathy showed a significantly lower LVEF and larger LV end-diastolic volume than those with idiopathic DCM [47]. LGE patterns were observed in 43% of patients with alcohol-induced cardiomyopathy (n = 22), and LGE was associated with a trend toward a higher rate of arrhythmic events; however, it was not associated with adverse outcomes. The overall outcomes of patients with alcohol-induced cardiomyopathy were comparable to those of patients with idiopathic DCM despite more advanced cardiac remodeling at baseline in the former (Fig. 2).

Anticancer Drugs

Cancer therapy–related cardiac dysfunction (CTRCD) refers to cardiac injury caused by a broad range of cancer therapies, including chemotherapy and targeted therapies (e.g., trastuzumab, proteasome inhibitors, immune checkpoint inhibitors, and vascular endothelial growth factor inhibitors) [48].

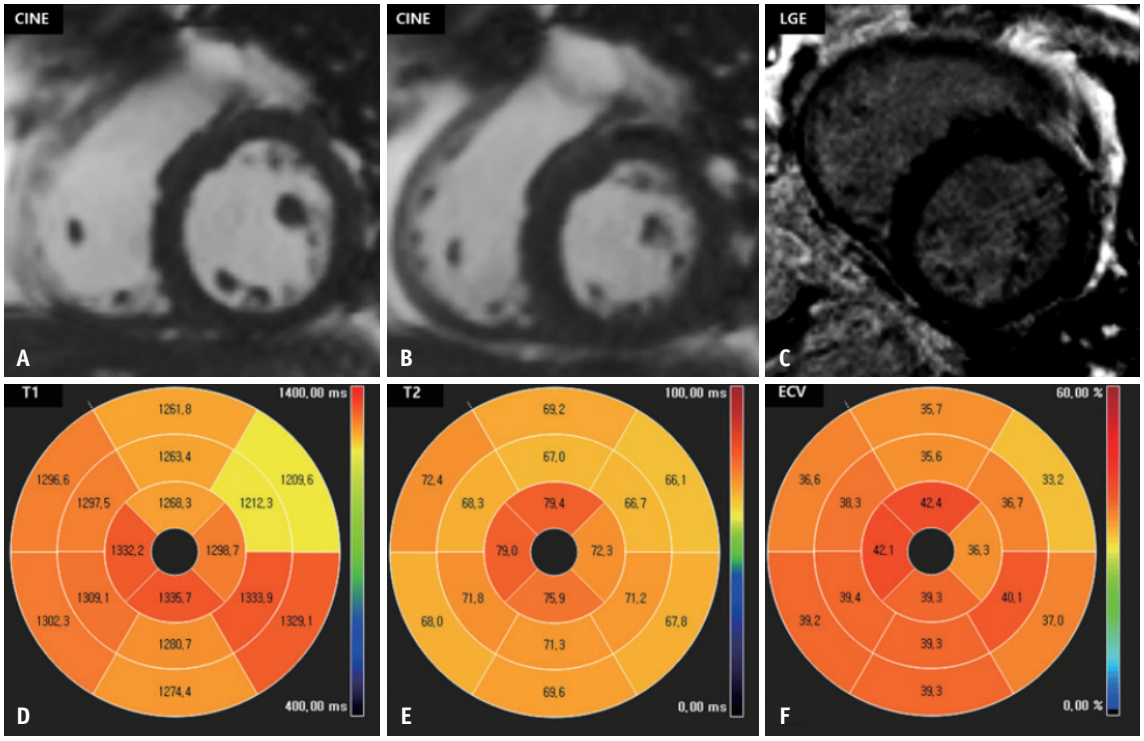

Fig. 2. Alcohol-induced dilated cardiomyopathy. A 68-year-old heavy alcoholic patient was admitted with general edema, and CMR was performed using 1.5T MRI. On cine images (**A:** short-axis image of the end-diastolic phase, **B:** short-axis image of the end-systolic phase), the LV shows diffuse hypokinesia. The LV end-diastolic diameter is 6.2 cm, and its ejection fraction is 26%. **C:** LGE scans reveal no focal hyperenhancements in the LV. However, T1 **(D)**, T2 **(E)**, and ECV **(F)** maps reveal diffusely increased myocardial T1 and T2 values and ECV fractions in the bull's eye diagrams. CMR = cardiac magnetic resonance, MRI = magnetic resonance imaging, LV = left ventricle, LGE = late gadolinium enhancement, ECV = extracellular volume

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CTRCD has poor prognosis and can compromise oncological care by interrupting therapy or causing a switch to potentially less effective treatments, which lead to decreased patient survival rates [49]. Therefore, early recognition and careful monitoring are crucial. A personalized approach is recommended by various regulatory bodies for the pretreatment risk evaluation of CTRCD [48]. The recommended cardiotoxicity risk was stratified into low, medium, and high according to the baseline cardiovascular profile, risk factors, pre-existing cardiovascular disease, and cancer therapy type and dose [48].

Considering the lack of widespread accessibility and high cost, the routine use of CMR for CTRCD surveillance is not feasible. However, because it has minor temporal variability and provides the most accurate LVEF measurements among the available imaging modalities, LVEF measurements using CMR is recommended, particularly in patients with poor-quality echocardiographic images or if there is a discrepancy between imaging modalities [50]. CMR provides crucial information regarding prior myocardial infarction scars, myocardial perfusion, diffuse fibrosis, and intracellular/interstitial edema [51].

GLS is the most commonly used parameter in strain imaging and has emerged as a new marker of subclinical ventricular dysfunction in CTRCD [52]. Recent studies have demonstrated that LV strain parameters obtained from CMR-FT also allow the detection of early LV dysfunction and myocardial fibrosis during and after potentially cardiotoxic cancer therapy (Fig. 3) [53]. However, further research on the diagnostic and prognostic performance of CMR strains for CTRCD is needed.

Infection

Myocarditis has diverse and regionally varying etiologies, and viral infections are the most common cause of DCM due to myocarditis. Chagas disease, post-streptococcal rheumatic heart disease, and human immunodeficiency virus infections are important causes of DCM in specific regions [54]. Myocarditis may be reversible if the acute inflammatory process heals and the cause resolves [55]. Most patients with acute myocarditis recover without any clinically relevant sequelae; however, some progress to chronic

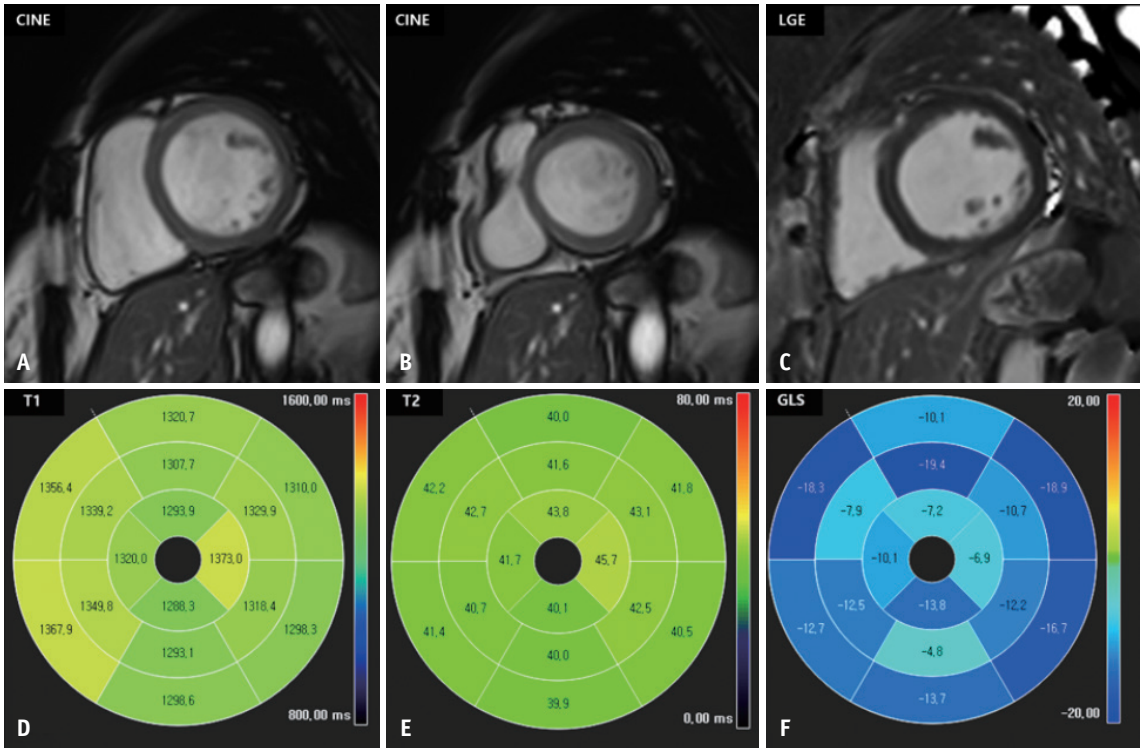

Fig. 3. Chemotherapy-induced dilated cardiomyopathy. A 64-year-old woman who had undergone partial mastectomy and 6 chemotherapy sessions was admitted with dyspnea on exertion. Short-axis cine images of the **(A)** end-diastolic and **(B)** end-systolic phases show severe hypokinesia in the LV. The LV end-diastolic diameter is 8.1 cm, and its ejection fraction is 17%. **C:** The LGE scans reveal no focal hyperenhancement in the LV. However, the T1 **(D)** and T2 **(E)** maps show diffusely increased myocardial T1 (up to 1373 ms) and T2 values (up to 45.7 ms). **F:** A bull's eye polar map of the GLS measured on the cine images shows decreased absolute strain values in each myocardial segment. LV = left ventricle, LGE = late gadolinium enhancement, GLS = global longitudinal strain

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DCM. Among histologically confirmed cases, the reported incidence of DCM, which evolves from myocarditis, ranges from 14% to 52% [56]. The mechanism of progression to DCM is unknown, but the inflammatory response starts with the activation of a proinflammatory cascade of cytokines followed by an immune response that may eventually lead to LV dysfunction and dilatation [57]. It is still unclear why some patients recover without residual myocardial injury, whereas others develop DCM [58].

CMR plays an essential role in diagnosing myocarditis and enabling tissue characterization, such as myocardial edema, hyperemia/capillary leak, and necrosis/fibrosis [59]. The Lake Louise criteria were proposed in 2009 for CMR-based myocarditis diagnosis and include T2-weighted imaging, early gadolinium enhancement, and LGE imaging [60]. Quantitative T1 and T2 mappings were included in the revised 2018 Lake Louise criteria. Multiparametric CMR data have been reported to yield higher diagnostic performance in patients who are clinically suspected of acute myocarditis [61]. Cases of combined myocarditis and cardiac dysfunction were classified as inflammatory cardiomyopathies. CMR has less sensitivity for the diagnosis of chronic inflammatory cardiomyopathy than for the diagnosis of acute myocarditis [62]. Consequently, endomyocardial biopsy combined with immunohistochemistry and viral genetic analysis may provide pathway-specific information.

Metabolic Diseases

Cushing Syndrome

Hypercortisolism can induce various myocardial changes, including hypertrophy, remodeling, fibrosis, and diastolic/systolic dysfunctions [63]. Patients with Cushing syndrome can show decreased LV function, increased LV mass, and rarely, DCM. However, few studies have been conducted on Cushing syndrome–related DCM [64]. The morpho-functional characteristics of Cushing syndrome–related DCM were similar to those of DCM with other causes; however, most Cushing syndrome–related DCM cases showed functional recovery after treatment and cortisol normalization [65].

Hypothyroidism and Hyperthyroidism

Thyroid disease can cause structural and functional changes in the heart because triiodothyronine (T3) has an important role in the inotropic modulation of the myocardium [66]. Hyperthyroidism increases heart rate, cardiac output, and blood volume and decreases systemic vascular resistance, whereas hypothyroidism has the opposite effect. Hyperthyroidism and hypothyroidism can result in cardiac contractility and heart failure. Rarely, DCM can occur because of hypothyroidism or hyperthyroidism, and such patients show functional recovery after treatment for an underlying thyroid disease [67].

Mitochondrial Disorders

Mitochondria produce adenosine triphosphate, which plays an essential role in intracellular energy production and cellular metabolism [68]. Mitochondrial DNA mutations cause various multi-systemic mitochondrial diseases [69]. Considering that the heart continuously pumps blood and consumes large amounts of energy, it is susceptible to mitochondrial diseases, and various cardiac complications can occur, including heart failure and arrhythmia [70]. Mitochondrial myopathy has a broad range of phenotypes, ranging from DCM to HCM. The DCM phenotype is less frequent (prevalence of 2%–22% according to the type of mutation) than HCM in patients with mitochondrial myopathy [71]. CMR is helpful for ruling out cardiac involvement or other types of cardiomyopathy in patients with mitochondrial disorders. Mitochondrial myopathy often shows non-coronary-type LGEs, and increased myocardial T2 signals or T2 values indicate intracellular vacuolar changes or increased water content [72]. The early diagnosis of mitochondrial cardiomyopathy is helpful for preventing SCD and improving patient prognosis.

Inflammatory/Infiltrative Diseases

Hemochromatosis

Chronically increased intestinal iron uptake during genetic hemochromatosis may cause organ failure, and excess iron deposition may cause DCM [73]. The His63Asp (*H63D*) mutation is significantly associated with DCM development [74]. Myocardial iron overload occurs when the myocardial T2 value is < 20 ms [75], and heart failure usually occurs when the myocardial T2 value is < 10 ms [76]. In a study of 31 genetically confirmed hemochromatosis cases, myocardial siderosis was present in 33% of patients with ferritin levels > 1000 μg/L and was the most common cause of reduced LVEF [77]. Therefore, CMR is useful for detecting myocardial siderosis and monitoring responses to therapy.

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Eosinophilic Granulomatosis with Polyangiitis (Churg–Strauss Syndrome)

Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare multi-systemic vasculitis disorder, and DCM may develop as an unusual form of cardiac involvement during the course of EGPA and may be reversible with appropriate treatment [78]. CMR is essential for the diagnosis of cardiac involvement in EGPA and may replace invasive cardiac biopsy. Typical CMR findings include multifocal LGE in the subendocardial area, which suggests multifocal myocarditis due to small-vessel vasculitis [79].

Systemic Lupus Erythematosus

Pericarditis is the most common cardiac complication of systemic lupus erythematosus (SLE), followed by myocarditis or fibrosis [80]. Lupus myocarditis may develop into DCM and heart failure, thus leading to cardiac remodeling and ventricular dilatation with normal or LV wall thinning [81]. CMR is required for the early diagnosis of lupus myocarditis [82]. According to T1 mapping, the development of subtle myocardial inflammation in SLE may lead to diffuse fibrosis [82].

Neuromuscular Disorders

Muscular dystrophy (MD) is a group of diseases characterized by genetic mutations that result in progressive muscular wasting and weakness. Cardiac involvement can occur in patients with MD, and cardiac disease may be the predominant manifestation of the underlying genetic myopathy. Duchenne MD (DMD) and Becker MD (BMD) are X-linked diseases associated with mutations in recessive genes that encode dystrophin proteins, which provide structural support to myocytes [83]. Dystrophin is important for stabilizing the cell membrane, and its absence causes sarcolemmal fragility and muscle cell degeneration [84].

The early detection of MD-associated cardiomyopathy is important because optimal cardioprotective management may slow adverse remodeling [85]. CMR can serve as a sensitive modality for the detection of early cardiac involvement in MD. Segmental LGE has been demonstrated in individuals with DMD and BMD before the onset of LV or right ventricular systolic dysfunction and may indicate early disease [86]. The characteristic pattern of LGE is subepicardial fibrosis of the inferolateral wall [87], which may be similar to that observed in viral myocarditis. However, Giglio et al. [88] reported that myocardial edema was not observed in patients with MD. Furthermore, elevated ECV and native T1 values have been documented in patients with DMD and normal LV systolic function (including some without LGE) [89]. In some regions of fibrosis where fatty metaplasia develops, native T1 values may be low [90]. Reduced circumferential strain, which is an early indicator of disease involvement, has been observed in patients with normal LV size and function [91]. In progressive disease, global LV hypokinesia, dilatation, thinning, and regional wall-motion abnormalities corresponded to the LGE area (Fig. 4).

Others

Tachycardia-induced DCM

Long-standing tachycardia, such as supraventricular or ventricular tachyarrhythmia, can induce LV dysfunction and DCM [92] because tachycardia results in extracellular matrix remodeling, cellular remodeling, and contractile dysfunction [93]. It is important to differentiate tachycardia-induced DCM from DCM of other causes or idiopathic DCM because LV dysfunction can improve after the proper management of the underlying arrhythmia. However, patients with DCM may show various types of arrhythmias, including tachyarrhythmia, and whether arrhythmia is the cause or consequence of DCM can sometimes be arbitrary [94]. The presence of tachycardia (> 100 beats/min) suggests the possibility of tachycardia-induced DCM after excluding other causes, and CMR can help differentiate challenging cases. Hasdemir et al. [95] reported that LGE was rare in 27 patients with tachycardia-induced cardiomyopathy and that the absence of LGE indicated a good response to arrhythmia treatment (Fig. 5). However, the pathognomonic CMR findings of tachycardia-induced cardiomyopathy have not yet been established.

Peripartum Cardiomyopathy

Peripartum cardiomyopathy (PPCM) is a rare type of DCM. It is an idiopathic cardiomyopathy presenting with heart failure secondary to LV systolic dysfunction toward the end of pregnancy or in the months following delivery where no other cause of heart failure is found [96]. The diagnostic definition is LV systolic dysfunction and LVEF < 45% during the last trimester of pregnancy or within a few months following delivery in previously healthy women without pre-existing cardiac dysfunction or a determinable cause of cardiomyopathy [97]. The incidence of PPCM can be as high as 1 in 100–300 deliveries or as low as 1 in 20000 deliveries and varies according to country [98]. Pregnant women with more risk factors are more likely to develop PPCM than those with fewer risk factors [99].

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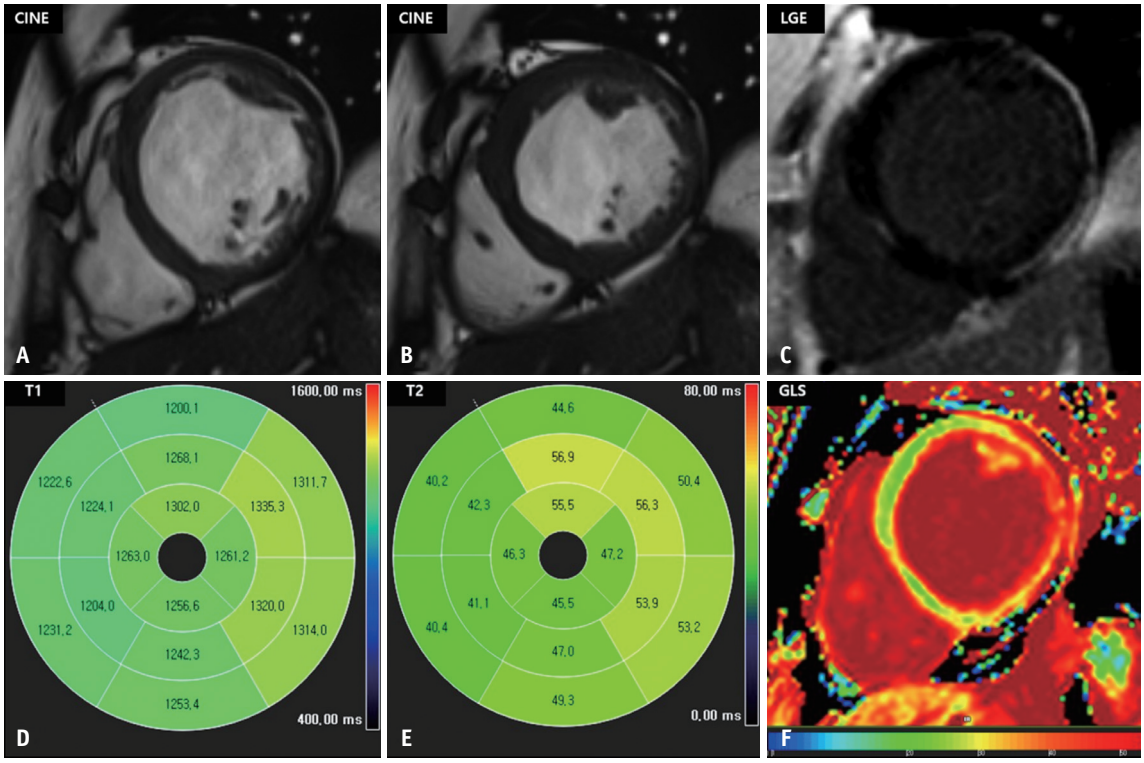

Fig. 4. Muscular dystrophy–associated cardiomyopathy. A 20-year-old male with Duchenne muscular dystrophy underwent 3T cardiac MRI because of dyspnea. On the short-axis cine images of the **(A)** end-diastolic and **(B)** end-systolic phases, the LV shows irregular wall thickness and diffuse hypokinesia. The LV end-diastolic diameter is 7.7 cm, and its ejection fraction is 38%. **C:** A LGE image reveals focal epicardial hyperenhancement in the septum of the mid-LV and heterogeneous hyperenhancement in the lateral free wall of the LV. The T1 **(D)** and T2 **(E)** maps show heterogeneously increased myocardial T1 (up to 1335.3 ms) and T2 values (up to 56.9 ms), particularly in the lateral free-wall area of the LV. **F:** ECV maps show a heterogeneously increased myocardial ECV fraction of up to 43%, particularly in the lateral free-wall area of the LV. MRI = magnetic resonance imaging, LV = left ventricle, LGE = late gadolinium enhancement, ECV = extracellular volume, GLS = global longitudinal strain

Prolactin conversion causes vascular and myocardial dysfunction [100]. Several genes associated with DCM (*TTN*, *MYBPC3*) have been detected in cases of PPCM, and approximately 20% of PPCM cases have an identified pathogenic mutation with *TTN* truncations, which are most commonly implicated in cardiomyopathy genetic screening [101].

Echocardiography is the primary modality for the initial imaging diagnosis of PPCM; however, CMR has significant added value because it accurately assesses LVEF and identifies myocardial edema and LGE [102]. LGE distribution was noted in 40%–70% of patients and was more likely to be present in follow-up scans than in acute-phase scans performed within seven days from diagnosis [103]. Mid-wall LGE was observed on the initial scan, and both mid-wall and subepicardial LGE were observed on the follow-up scan. LGE has a relatively unfavorable clinical course [104]. Patients with PPCM have significantly higher native T1, ECV, and T2 values than healthy controls [105]. The native T1, ECV, and T2 values were associated with recovery from ejection fraction. In addition, the ECV can independently predict the recovery of LV function in patients with PPCM. Right ventricular dysfunction and dilatation were observed in 35% of patients with PPCM at the time of diagnosis and were associated with unfavorable outcomes (Fig. 6) [103].

Differential Diagnosis of DCM and Its Phenocopies Based on CMR Findings

In cases of LV dilatation and dysfunction, conditions with phenotypic overlap must be excluded before establishing a diagnosis of DCM. CMR can be used to differentiate between DCM and its various phenotypes (Table 3).

Ischemic Cardiomyopathy

Ischemic cardiomyopathy may share key features with DCM, including a dilated LV and impaired LV systolic function, and should be considered a possible differential

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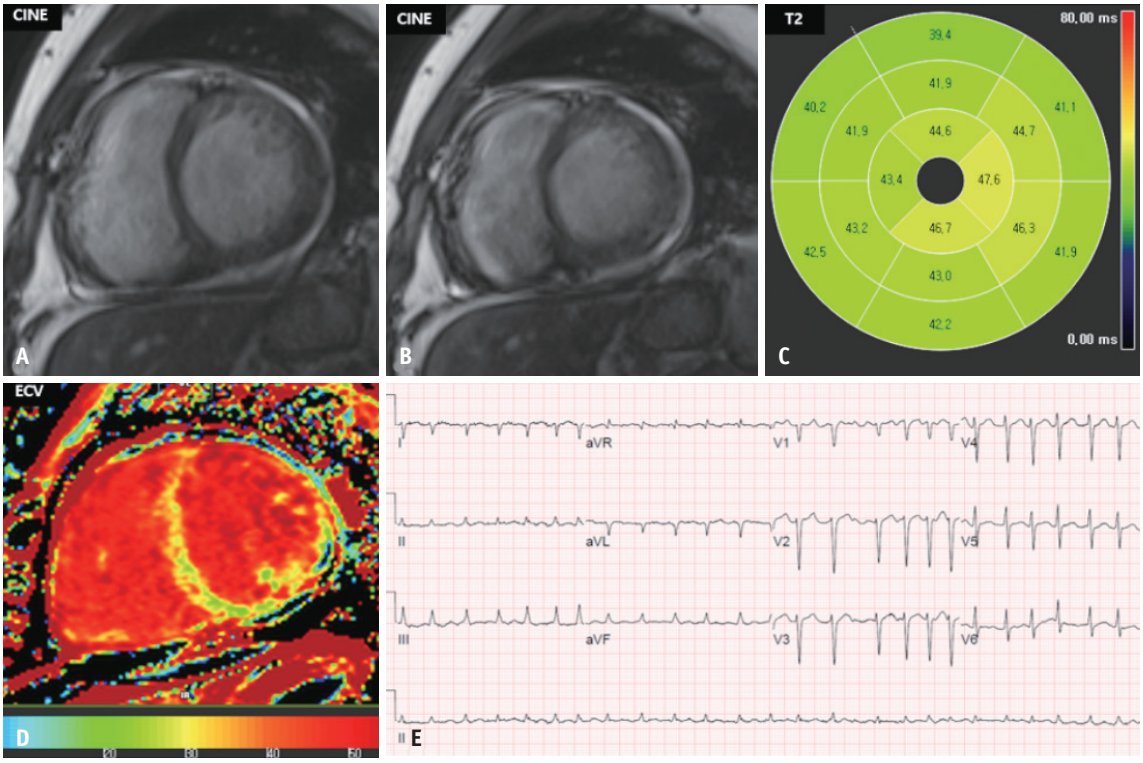

Fig. 5. Tachycardia-induced dilated cardiomyopathy. A 45-year-old man with atrial fibrillation was admitted with dyspnea on exertion. CMR was performed using a 3T MRI. The quality of the MR images is poor because of motion artifacts. On cine images (**A:** short-axis image of the end-diastolic phase, **B:** short-axis image of the end-systolic phase), the LV shows diffuse hypokinesia and dilatation. The LV end-diastolic diameter is 8.0 cm, and its ejection fraction is 15%. **C:** The T2 map shows increased T2 values, thus suggesting myocardial edema. The LGE images revealed no focal hyperenhancement of the LV (not shown). **D:** The ECV map reveals a diffusely increased myocardial ECV fraction of up to 38%. **E:** An ECG test shows atrial fibrillation with a rapid ventricular response. CMR = cardiac magnetic resonance, MRI = magnetic resonance imaging, MR = magnetic resonance, LV = left ventricle, LGE = late gadolinium enhancement, ECV = extracellular volume, ECG = electrocardiogram

diagnosis [106]. One method of differentiation is non-invasive coronary artery evaluation such as coronary computed tomography angiography (CCTA) or magnetic resonance (MR) coronary angiography. However, CCTA has issues related to radiation exposure, use of iodine contrast materials, and limitations in myocardial assessments. MR coronary angiography can replace CCTA but has technical complexity and accuracy issues. Resting/stress perfusion CMR imaging could be helpful in ruling out stress-induced myocardial ischemia and wall-motion abnormalities [107]. Most importantly, LGE can provide beneficial information in a non-invasive manner for differentiating between ischemic and non-ischemic cardiomyopathies [20]. Subendocardial or transmural LGE along the epicardial coronary artery territory is usually considered an ischemic LGE pattern. In contrast, DCM could show mid-wall LGE or non-coronary-type LGEs (Fig. 7).

"Burned-out" Phase HCM

HCM can develop at different stages of the disease progression [108]. Stage IV presents with severe LV functional deterioration, LVEF < 50%, and extensive fibrosis [109]. This end-stage or "burned-out" phase HCM demonstrates dilated hypokinetic evolution and may have a morpho-functional phenotype similar to that of DCM [109]. As the wall thickness regresses and the LV cavity dilates, proper differential diagnosis of DCM without prior imaging data becomes challenging. However, the careful evaluation of CMR may provide valuable information for differentiation. Dilated hypokinetic evolution in HCM results in residual hypertrophy and shows an uneven asymmetric wall thickness through the LV or right ventricle (RV). Extensive LGE was more prominent along the thin myocardium than along the thick myocardium. A young age and a family history of HCM also supported end-stage HCM rather than DCM (Fig. 8) [110].

Arrhythmogenic Cardiomyopathy

Arrhythmogenic cardiomyopathy (ACM) is a rare familial disorder characterized by the progressive fibro-

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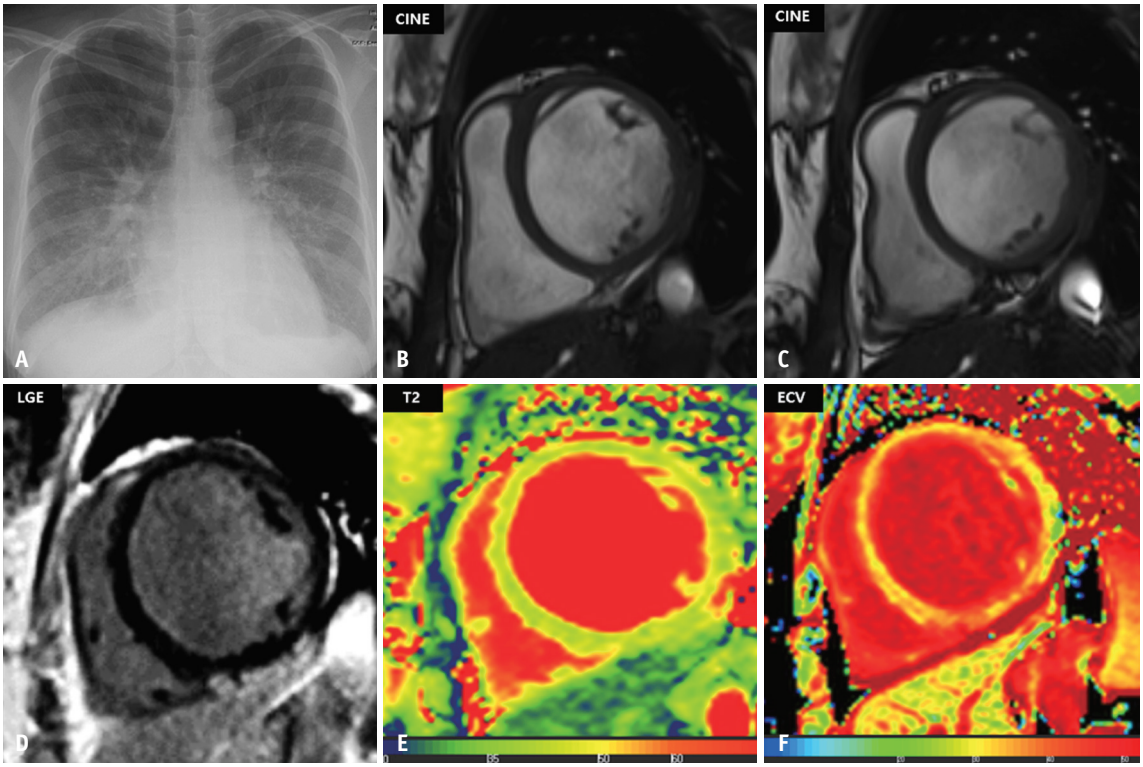

Fig. 6. Peripartum cardiomyopathy. A 34-year-old female patient was admitted with dyspnea after a cesarean section. **A:** Initial chest radiography reveals cardiomegaly, pulmonary edema, and bilateral pleural effusion. On cine images (**B:** short-axis image of the end-diastolic phase, **C:** short-axis image of the end-systolic phase), the LV shows diffuse hypokinesia. The LV end-diastolic diameter is 7.4 cm, and the LV ejection fraction is 20.7%. **D:** The LGE images show no focal hyperenhancement in the LV. However, on the T2 map **(E)**, the T2 value of the LV is diffusely increased. **F:** The ECV map reveals a diffusely increased myocardial ECV fraction of up to 36%. LV = left ventricle, LGE = late gadolinium enhancement, ECV = extracellular volume

Table 3. Differential diagnosis of DCM

Phenocopies of DCM	Differential CMR findings
Ischemic cardiomyopathy	Subendocardial or transmural LGE along the epicardial coronary artery territory Stress-induced myocardial ischemia or infarction on MR perfusion Abnormality on MR coronary angiography
End-stage sarcoidosis	Extensive LGE + epicardial/transmural layer ± RV involvement The relatively distinct border between LGE area and non-LGE area
Burned-out phase HCM	Uneven, asymmetric wall thickness through LV or RV Extensive LGE, which is more prominent in the thin myocardium
Arrhythmogenic cardiomyopathy	Morpho-functional abnormality on RV Stria pattern LGE involving a subepicardial layer Fat infiltration
Non-compaction	The ratio of non-compacted to compacted layer > 2.3 at the end-diastolic phase

DCM = dilated cardiomyopathy, CMR = cardiac magnetic resonance, LGE = late gadolinium enhancement, MR = magnetic resonance, RV = right ventricle, HCM = hypertrophic cardiomyopathy, LV = left ventricle

fatty replacement of the myocardium [111]. Previously, it was considered a disease of the RV; however, after histopathological examination (autopsy), the LV was found to be involved. Recently, three types of arrhythmogenic cardiomyopathies were defined: right dominant, biventricular, and left dominant [112]. Owing to LV involvement, ACM can be overlooked and misdiagnosed as other diseases, such as DCM. According to the Padua criteria, ACM involving the LV (biventricular variant or left-dominant variant) should be considered in cases with global LV systolic dysfunction (minor

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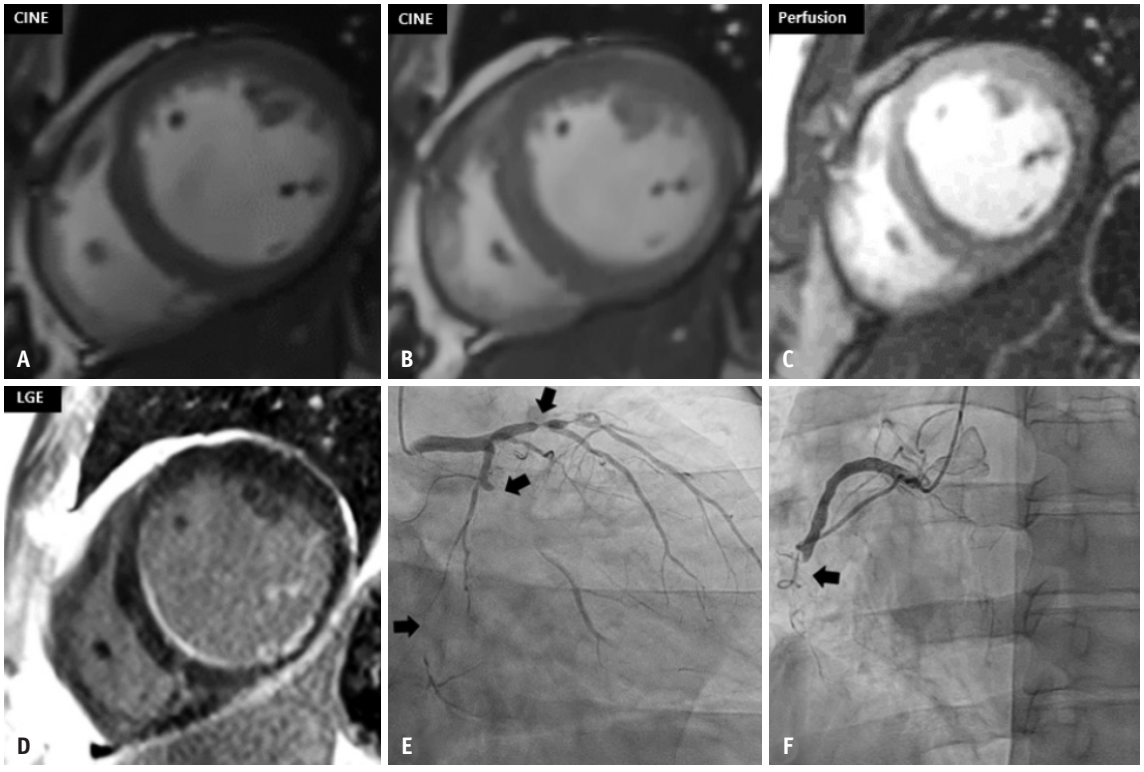

Fig. 7. Ischemic cardiomyopathy. A 29-year-old man was admitted with chest tightness and dyspnea. He was diagnosed with uncontrolled diabetes mellitus with a fasting serum glucose level of 162 mg/dL. On cine images (**A:** short-axis image of the end-diastolic phase, **B:** short-axis image of the end-systolic phase), the LV shows global hypokinesia. The LV end-diastolic diameter is 6.8 cm, and its ejection fraction is 25.0%. **C:** On the resting perfusion image, a subendocardial perfusion defect is observed at the mid-LV anteroseptal, inferoseptal, inferior, and inferolateral walls. **D:** The LGE image shows subendocardial hyperenhancement in the corresponding area. Coronary angiography shows the total occlusion of the distal LCx, obtuse marginal and subtotal occlusion of the proximal LAD (arrows) **(E)**, and total occlusion of the midRCA (arrow) **(F)**. LV = left ventricle, LGE = late gadolinium enhancement, LCx = left circumflex artery, LAD = left anterior descending artery, midRCA = mid-right coronary artery

criterion of morpho-functional ventricular abnormality) or LGE distribution in the LV of > 1 bull's eye segment (major criterion of structural myocardial abnormality) [113]. However, these criteria overlap with those for DCM, and careful evaluation is required to avoid overlooking ACM. ACM with biventricular involvement is more common than an isolated LV phenotype. Therefore, RV abnormalities should be carefully examined. Although there are no abnormalities in the RV, the predominance of LGE in the subepicardial layers of the LV with a stria pattern could suggest ACM rather than DCM [114]. In such cases, genetic evaluation should be considered when diagnosing the left-dominant variant of ACM (Fig. 9).

Myocardial Non-compaction

Myocardial non-compaction is characterized by an extensive non-compacted endocardial layer with prominent trabeculations. LV non-compaction genetically and morphologically overlaps with DCM, and LV non-compaction with decreased LV function can be misdiagnosed as DCM. Even though the ratio of non-compacted to compacted layers of > 2.3 at the end-diastolic phase could suggest LV non-compaction on MR imaging, prominent LV trabeculation is not rare in DCM [115]. Gregor et al. [116] reported that LV non-compaction resulted in higher trabeculated and papillary muscle mass and lower apical LV strain than DCM. LGE imaging can reveal delayed trabecular hyperenhancement in LV non-compaction, which is related to LVEF [117]. Careful evaluation is needed because of the morphological similarities between DCM and LV non-compaction (Fig. 10).

CONCLUSION

CMR has various roles in the diagnosis and investigation of DCM, such as the precise evaluation of wall motion and function, and tissue characterization [118]. The two main

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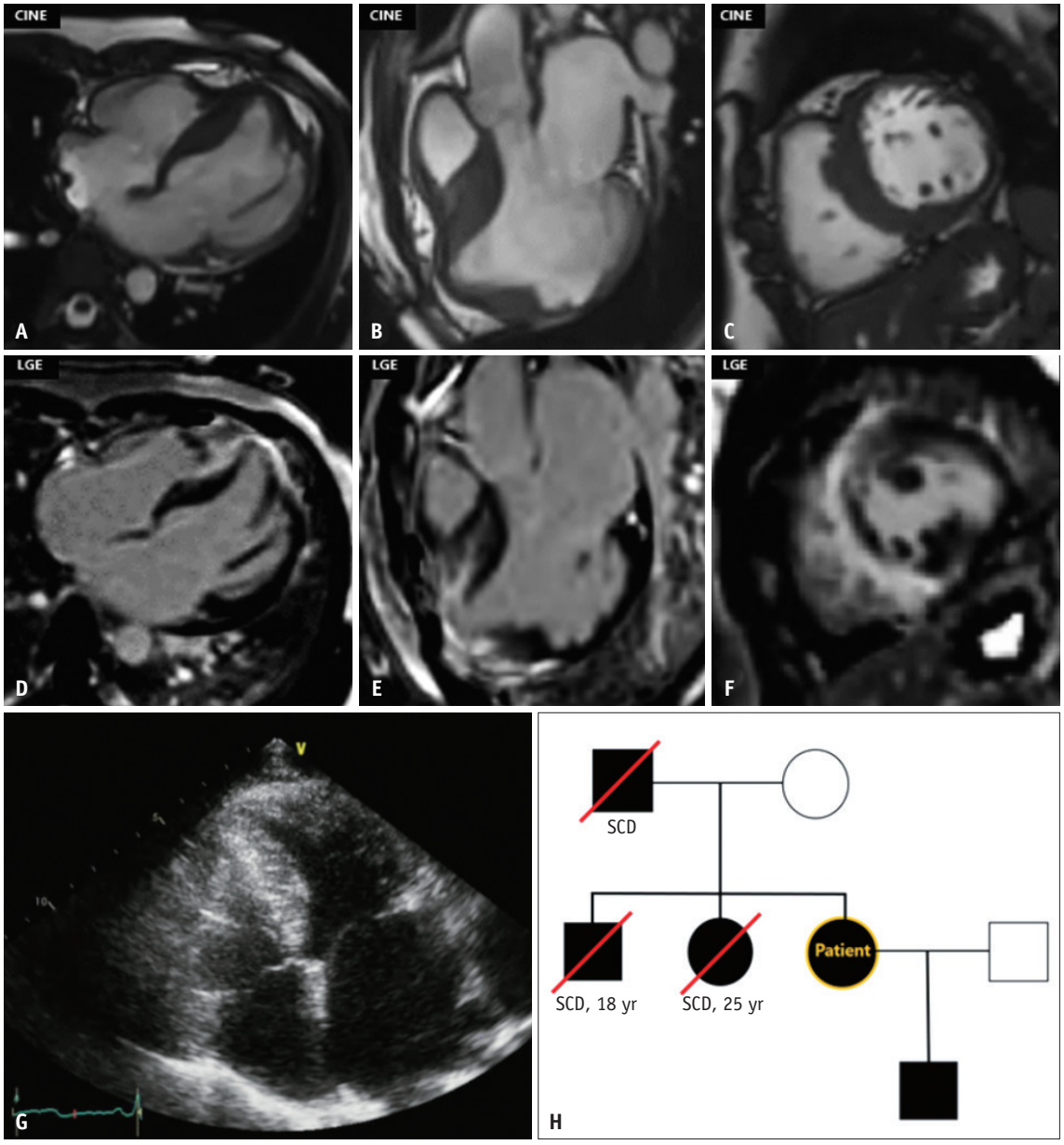

Fig. 8. Burned-out phase hypertrophic cardiomyopathy. A 49-year-old woman presented with exertional chest pain and dyspnea. On the four-chamber **(A)**, three-chamber **(B)**, and short-axis cine images **(C)** of the end-diastolic phase, uneven myocardial thickness ranging from 15 mm at the LV septal wall to 4 mm at the LV lateral wall is observed. Similarly, RV apical wall thickening is also observed. The LV ejection fraction is 47%. On the four-chamber **(D)**, three-chamber **(E)**, and short-axis LGE images **(F)**, epicardial or transmural delayed hyperenhancements at the LV septum and apex are observed. **G:** Echocardiography performed four years ago shows an evenly thick LV myocardium with a preserved LV ejection fraction of 67%. **H:** A pedigree shows the history of SCD in the father, brother, and sister. '□' means 'unaffected male', '■' means 'affected male', '○' means 'unaffected female', and '●' means 'affected female'. LV = left ventricle, RV = right ventricle, LGE = late gadolinium enhancement, SCD = sudden cardiac death

characteristics of DCM, namely, LV dilatation and dysfunction, have various phenotypes that hamper prompt differential diagnosis. Although CMR has limitations in evaluating the etiologies of DCM, it is useful for early diagnosis, risk stratification, treatment planning, prognostic evaluation, and follow-up. A comprehensive CMR analysis of DCM with various etiologies will improve our understanding of DCM.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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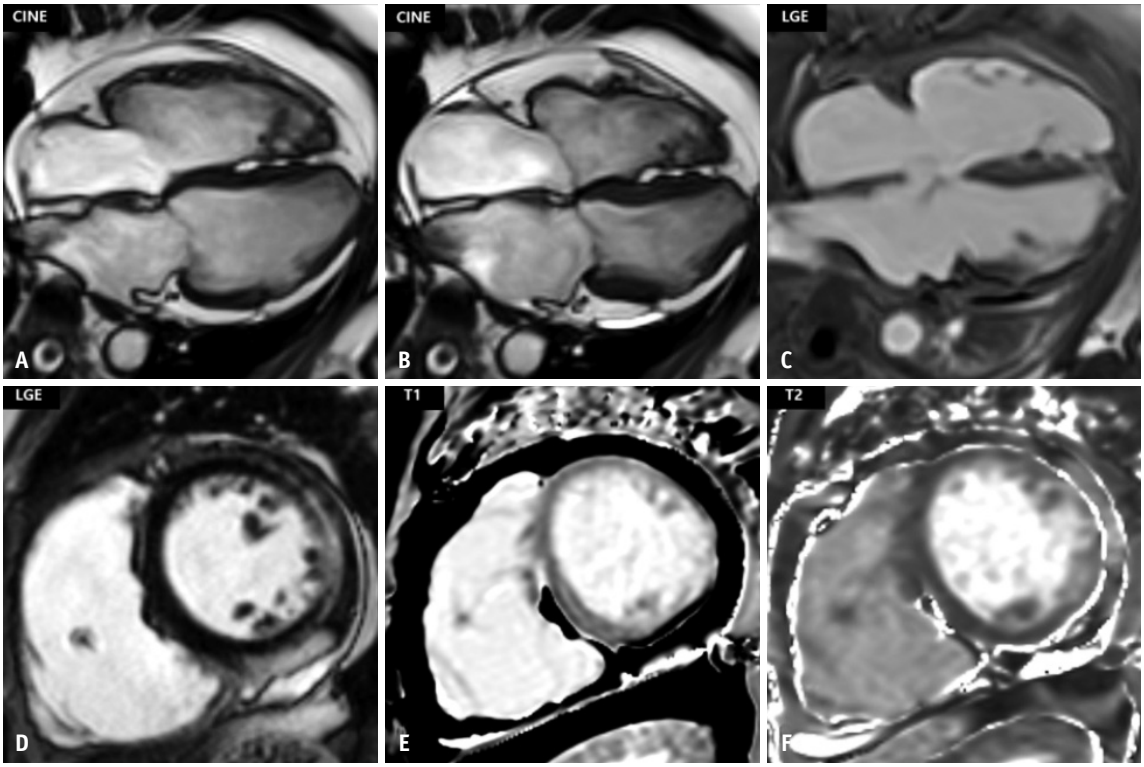

Fig. 9. Arrhythmogenic cardiomyopathy. A 41-year-old woman was admitted with dyspnea. On the long-axis cine images (**A:** end-diastolic phase, **B:** end-systolic phase), the RV shows regional dyskinesia of the free wall and is dilated (RV end-diastolic volume: 126 mL/m^2); the RV ejection fraction is 33.4%. Irregular wall thinning is also observed in the lateral wall of the mid-LV. **C, D:** The four-chamber and short-axis LGE images show abnormal LGE in the RV free wall, epicardial LGE in the lateral wall of the basal LV, and epicardial LGE in the inferior wall of the apical to mid-LV. **E:** The T1 map shows fat infiltration in the interventricular septum and biventricular wall. **F:** The T2 map shows mild T2 elevation in the lateral and inferior walls of LV. RV = right ventricle, LV = left ventricle, LGE = late gadolinium enhancement

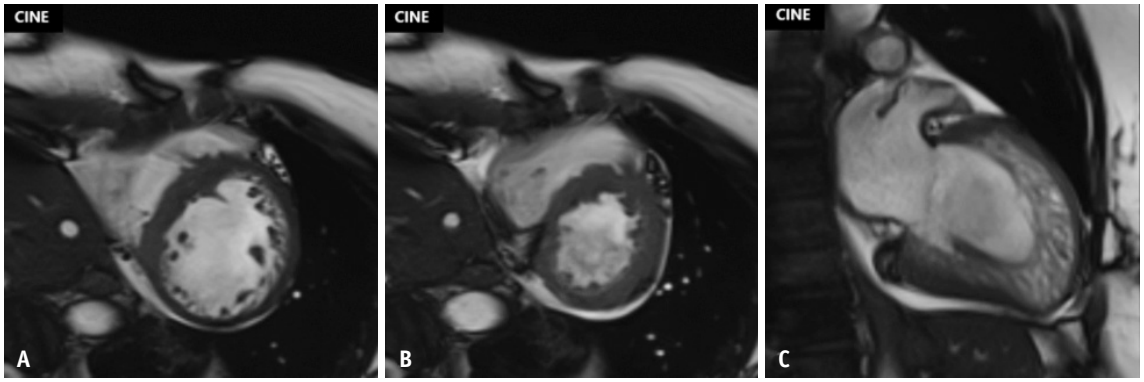

Fig. 10. Myocardial non-compaction. On the short-axis cine images of the end-diastolic **(A)** and end-systolic phases **(B)**, the LV shows global hypokinesia and dilatation. **C:** The non-compacted endocardial layer is prominent and shows trabeculation on the long-axis two-chamber view. The ratio of the non-compacted to the compacted layer (> 2.3) at the end-diastolic phase on MRI suggests the presence of LV non-compaction. LV = left ventricle, MRI = magnetic resonance imaging

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