# Early diagnosis of cardiomyopathies by cardiac magnetic resonance. Overview of the main criteria

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## Abstract

Cardiomyopathies (CMPs) are diseases of the heart muscle. They include a variety of myocardial disorders that manifest with various structural and functional phenotypes and are frequently genetic. Myocardial disease caused by known cardiovascular causes (such as hypertension, ischemic heart disease, or valvular disease) should be distinguished from CMPs for classification and management purposes. Identification of various CMP phenotypes relies primarily upon echocardiographic evaluation. In selected cases, cardiac magnetic resonance imaging (CMR) or computed tomography may be useful to identify and localize fatty infiltration, inflammation, scar/fibrosis, focal hypertrophy, and better visualize the left ventricular apex and right ventricle. CMR imaging has emerged as a comprehensive tool for the diagnosis and follow-up of patients with CMPs. The accuracy and reproducibility in evaluating cardiac structures, the unique ability of non-invasive tissue characterization and the lack of ionizing radiation, make CMR very attractive as a potential "all-in-one technique". Indeed, it provides valuable data to confirm or establish the diagnosis, screen subclinical cases, identify aetiology, establish the prognosis. Additionally, it provides information for setting a risk stratification (based on evaluation of proved independent prognostic factors as ejection fraction, end-systolic-volume, myocardial fibrosis) and follow-up. Last, it helps to monitor the response to the therapy. In this review, the pivotal role of CMR in the comprehensive evaluation of patients with CMP is discussed, highlighting the key features guiding differential diagnosis and the assessment of prognosis.

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Key words: Cardiomyopathy; cardiac magnetic resonance; early diagnosis.

Contributions: All the authors made a substantive contribution, read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest: The authors declare that they have no competing interests, and all authors confirm accuracy.

Ethics approval and consent to participate: not applicable.

Received for publication: 18 November 2021.
Accepted for publication: 15 March 2022.


*Monaldi Archives for Chest Disease 2022; 92:2151*
*doi: 10.4081/monaldi.2022.2151*



## Introduction

The latest definition of cardiomyopathy (CMP) is a myocardial disorder in which the heart muscle is structurally and functionally abnormal in the absence of coronary artery disease, hypertension, valvular disease, and congenital heart disease sufficient to explain the observed myocardial abnormality [1,2]. They were originally defined as idiopathic disorders. CMPs include a variety of myocardial disorders that manifest with various structural and functional phenotypes and are frequently genetic. Current major society definitions of CMP exclude heart disease secondary to known cardiovascular disorders (hypertension, ischemic heart disease, or valvular disease). Nevertheless, in clinical practice, the terms «ischemic,» «valvular,» and «hypertensive CMP» have been used commonly, particularly in North America. The 1995 WHO/ISFC Task Force used the term «specific CMP» to reflect this reality and the fact that the genetic basis of the CMPs was being elucidated [3]. The 2008 European Society of Cardiology (ESC) proposal provided a clinical approach to diagnosing a patient who presents with symptoms, a family history of CMP, or electrocardiographic (ECG) and echocardiographic abnormalities that are otherwise unexplained [4]. Like the 2006 American Heart Association (AHA) proposal, it focuses on the established morphological types described by the 1995 World Heart Federation WHO/ISFC Task Force: i) hypertrophic CMP (HCM), ii) dilated CMP (DCM), iii) arrhythmogenic right ventricular (RV) CMP/dysplasia (ARVC/D), iv) restrictive right

ventricular (RV) CMP (RCM), v) unclassified CMP, including stress CMP and non-compaction disease [5]. The AHA/ESC and MOGE(S) classification systems then went on to define the familial and, if possible, genetic basis of disease. The MOGE(S) takes in account the morpho-functional phenotype (M), the organ/system involvement (O), the genetic inheritance pattern (G), the aetiology (E) and the stage (S) of each disease [1].

## How to diagnose CMPs

Identification of various CMP phenotypes relies primarily upon echocardiographic evaluation. Transthoracic echocardiography (TTE) can define the anatomic and functional characteristics of the heart, diagnostic for DCM, HCM, ACM, or RCM. It represents a widely available and not-expensive exam for the first-line diagnosis. In selected cases, CMR may be useful to identify aetiology through the localization of fat, iron, amyloid infiltration, inflammation, scar/fibrosis. Morphological features not easily described by TTE, as focal hypertrophy, left ventricular (LV) apical aneurysm, and RV structure and function are also evaluated.

## Role of CMR

CMR enables characterization of the range of myocardial diseases from ischemic to inflammatory and various types of CMP. It enables visualization and quantification of the size, volumes, mass, and global and regional function of the LV and RV cavities with high spatial and temporal resolution [6]. It has the unique ability of non-invasive tissue characterization for assessment of fibrosis, oedema, iron over overload; infiltrative deposition using gadolinium-contrast enhancement (GE) techniques and T1-T2 mapping [7,8]. Collaterally, the morphology and function of the cardiac valves, thoracic aorta, pulmonary arteries and pericardium are analysed without spatial limitation, therefore showing low inter-observer variability. Late-gadolinium enhancement study (LGE) helps in the differentiation between ischemic and non-ischemic etiopathology, with different pathognomonic pattern of the gadolinium uptake (Figure 1) [6].

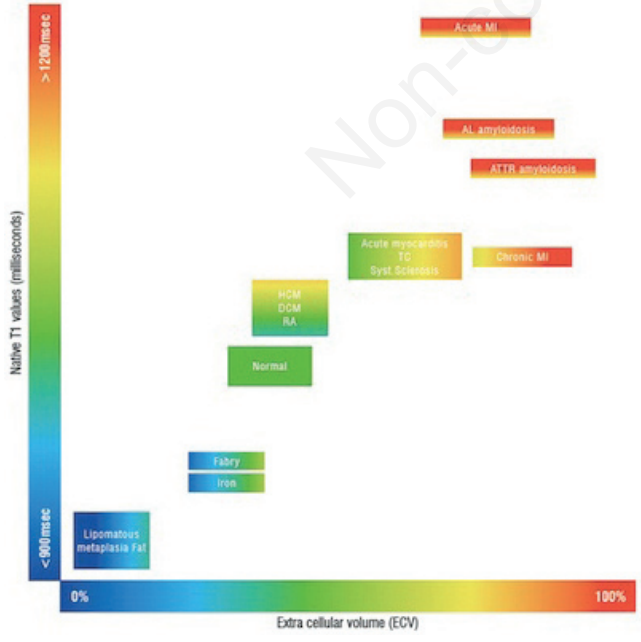


**Figure 1. Extra-cellular volume *versus* T1-mapping in normal myocardium and in different pathological conditions.**

T1-T2 mapping enable the quantification of the myocardial signal in T1, T2 and T2* relaxation time, with different values for different diseases [8]. For diseases with very low T1 value (e.g., iron overload, Anderson-Fabry disease) and very high T1 value (amyloidosis), CMR is very specific, without overlap of normal myocardium (Figure 2). Each aetiology of CMP has a typical CMR morphological feature, with occasionally the possible coexistence of two different morphologies (e.g., patient with asymmetrical HCM who present a non-compaction in lateral wall).

CMR is the preferred imaging examination for patients with LV hypertrophy to diagnose the underlying aetiology and assess myocardial morphology and function. Native T1, T2, T2*, extracellular volume fraction (ECV) imaging and LGE is often used [9)].

## Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a genetic disorder, caused by autosomal dominant mutations of genes encoding contractile sarcomeric proteins and myofilament elements.

Histology is represented by cellular disarray, hypertrophy and interstitial fibrosis. Clinically it is associated with the development of dyspnoea (related to LV outflow tract dynamic obstruction), atrial and ventricular arrhythmias, thromboembolic events, sudden cardiac death (SCD), and heart failure (HF). It represents the most common cause of SCD among young athletes together with arrhythmogenic CMP (ACM).

CMR enables the diagnosis of HCM, with accurate and reproducible assessment of the extent and location of hypertrophy [10]. It is useful in the differential diagnosis with other hypertrophic phenotypes, as in athlete's heart and hypertensive LV hypertrophic remodelling or infiltrative disease like amyloidosis. Figure 3 is showing a case of severe basal and medial anterior and inferior hypertrophy at SSFP sequence (Figure 3 A-A1), with increased T1 at the septum in short axis and 4-chamber view (Figure 3 B-B1) and intramyocardial captation of interventricular septum at LGE in short axis and 4 chamber-view (Figure 3 C-C1). Another case with severe hypertrophy and LGE enhancement in anterior and inferior wall is shown in Figure 4. TTE, the first-line method of assessment, may be inaccurate in detecting focal areas of increased wall thickness when confined to three specific "echo-blind" regions of the LV chamber, including the anterolateral wall, apex, and inferior septum. In 12% of patient who present a HCM phenotype on CMR, echocardiography was normal [11]. Therefore, CMR is mandatory for the accurate depiction of the hypertrophic phenotype, measurements of wall thickness and cardiac mass, which are all prognostic variables. In fact, massive hypertrophy, defined as a wall-thickness ≥30 mm, and cardiac mass are independent risk factors for SCD [12]. After gadolinium injection, up to 60% of HCM patients demonstrate focal replacement fibrosis, which is generally patchy, most commonly in the hypertrophied regions, and typically at the right ventricle insertion points. A special phenotype with mid ventricular obstruction and apical aneurysm can be associated with thromboembolic complications.

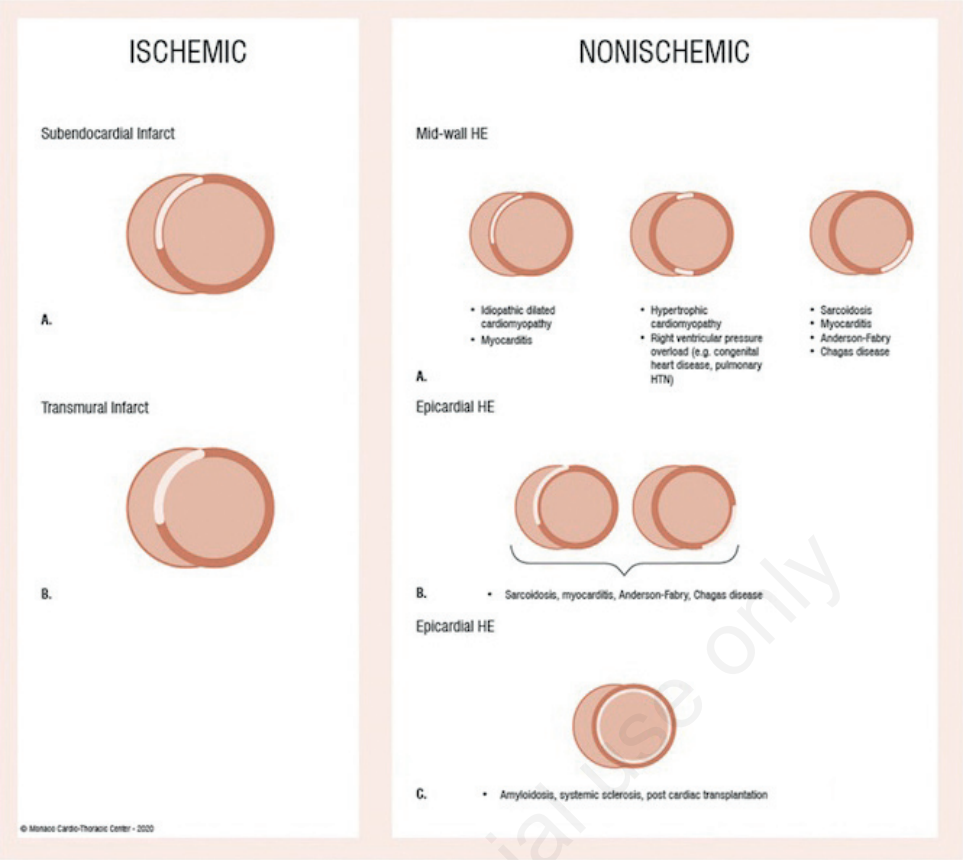


**Figure 2. Multiple late-gadolinium enhancement (LGE) patterns showing the different localizations in ischemic and non-ischemic CMP.**

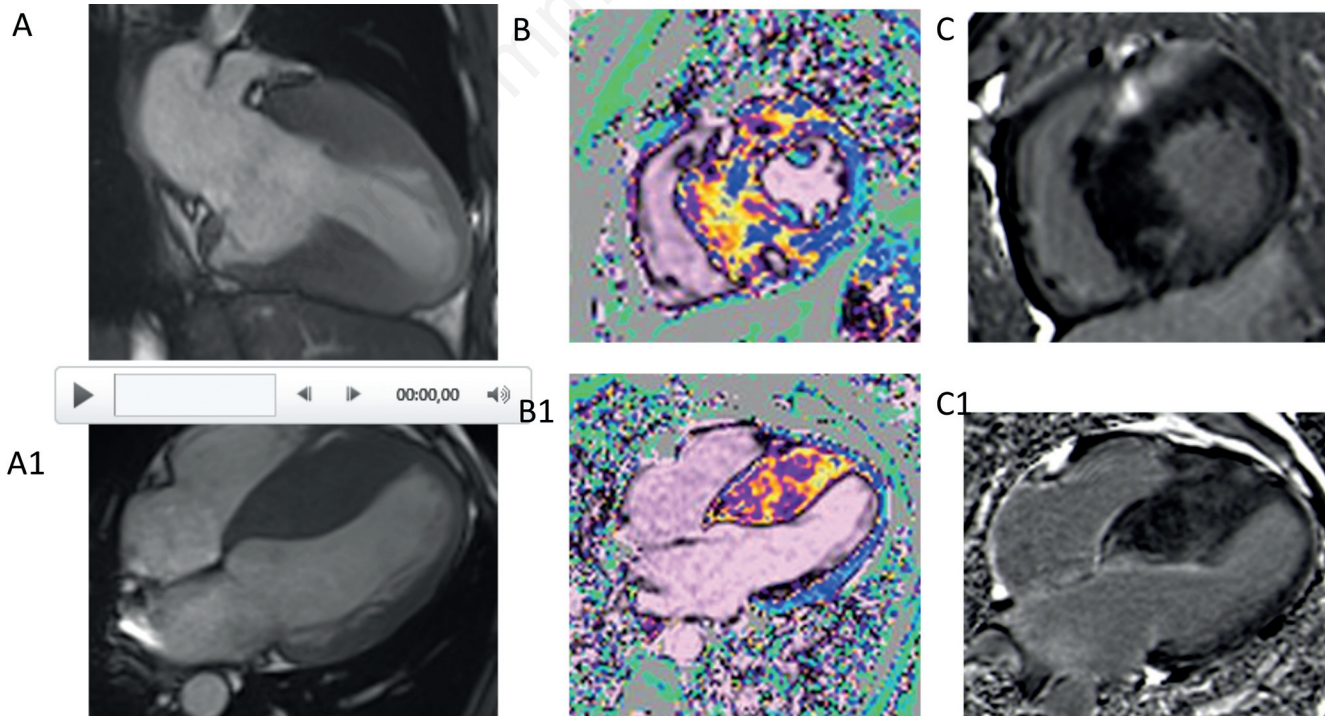


**Figure 3. A case of severe left ventricular hypertrophy (LVH). A-A1) Severe LVH cine SSFP sequence con LVH severe basal and median anterior and inferior in 4-chamber (4C) and 2-chamber (2C) view. B-B1) T1 mapping shows increased T1 of the hypertrophied septum in SAX and 4C view. C-C1) LGE with intramyocardial hypercaptation in SAX and 4C view.**

The presence of LGE is associated with a 3.4-fold increase in risk for SCD, a 1.8-fold increase in all-cause mortality, a 2.9-fold increase in cardiovascular mortality, and a trend to increase in the development of "end-stage" HCM and risk of HF death [13]. Moreover, LGE confers up to a 7-fold increased risk for ambulatory non-sustained ventricular tachyarrhythmia compared with HCM patients without LGE. Given the high prevalence of LGE and the average low risk of SCD in HCM (<1% per year), a cut-off value for LGE extension ≥15% has been proposed as a criterion in the evaluation for intra-ventricular cardioverter (ICD) implantation [14].

Native T1 and ECV mapping play an important role in the assessment of diffuse interstitial fibrosis in HCM, especially at very early stage of the disease, when LGE is not present yet. The early clinical experience in this field is confined in the differential diagnosis of HCM from LV hypertrophy "phenocopies" and in the evaluation of HCM family members, where promising results have been obtained [15].

LV fibrosis strongly affects prognosis and response to therapy. CMR offers the unique opportunity to non-invasively detect the two main histological patterns of fibrosis occurring in DCM, as in other CMPs: interstitial and replacement fibrosis. Diffuse interstitial fibrosis, defined as an increase in collagen volume fraction with the expansion of extracellular matrix, can be reversible. It is associated with adverse ventricular remodelling and increased all-cause mortality and SCD, acting as a substrate for re-entry arrhythmias [17]. T1 mapping with ECV measurement can detect diffuse interstitial fibrosis with an accuracy comparable to endomyocardial biopsy, even in early stages of DCM, when LV has only mild functional impairment and dilatation, and LGE is not present yet. It therefore represents an early marker of the disease. Replacement fibrosis, defined as focal irreversible 'reparative' scarring that follows myocyte injury or necrosis, occurs in approximately one-third of patients with DCM, typically in the mid-wall of interventricular septum, and is detectable by LGE (Figure 5 A-C) [18]. The presence of midwall replacement

## Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is the leading cause of cardiac transplantation and is associated with high morbidity and mortality, with increased risk of SCD and HF. DCM represents the final common phenotype of cardiac damage due to both genetic (non-modifiable) and environmental (potentially modifiable) insults. Morphological and structural details, together with tissue characterization, provided by CMR imaging, help the identification of aetiology, which may allow targeted disease-specific treatment, guide the need for family screening or inform on prognosis. Although LV ejection fraction (EF) is a pivotal parameter in guiding therapy and should be precisely calculated, other parameters of systolic function such as longitudinal strain, using myocardial feature tracking, have been shown to predict adverse outcomes, independently of the other predictors. CMR allows for an evaluation and quantification of the RV size and function. Reduced RV EF on CMR is an independent predictor of all-cause mortality and adverse HF outcomes [6]. Also left atrial (LA) enlargement has a prognostic value. Indexed LA volume calculated using CMR independently predicts cardiac transplant-free survival in DCM. A cut-off value of >72 mL/m$^2$ has been shown to predict a 3-fold increase in adverse outcomes in patients with DCM [16].

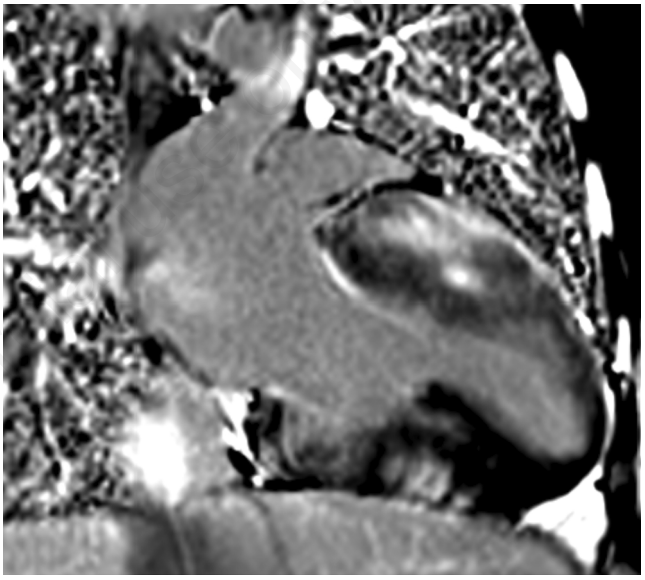

**Figure 4. Severe hypertrophy and LGE in anterior and inferior wall (2C view).**

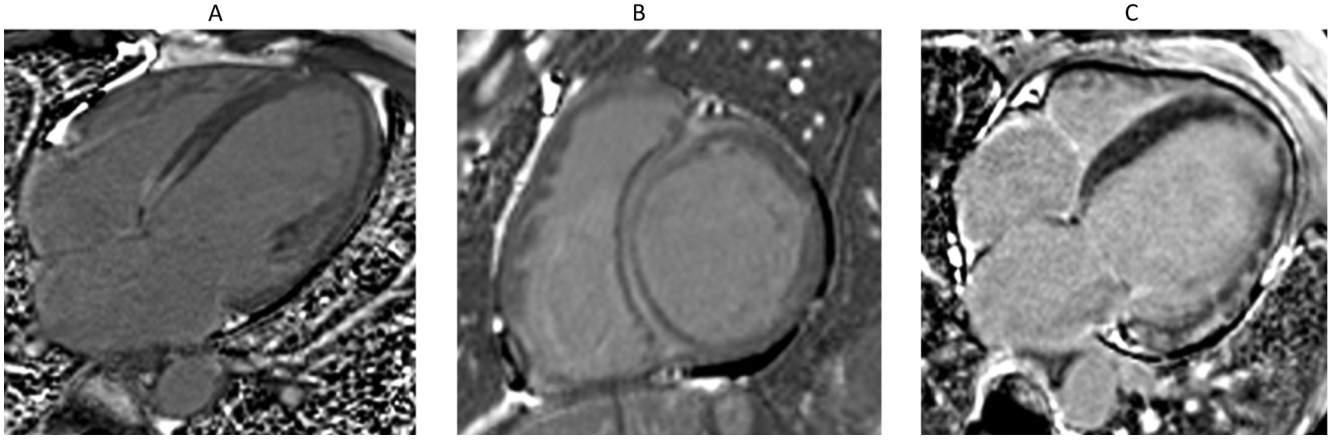


**Figure 5. Dilated cardiomyopathy. A,B) A typical case of DCM at the PSIR sequence LGE, which is positive in mid-wall of the inter-ventricular septum (4C and SAX view respectively). C) A case of DCM post-myocarditis presenting subepicardial LGE in 4C view.**

fibrosis is emerging as a strong independent prognostic factor of SCD and all-cause mortality, thus gaining a significant potential role especially in guiding patient selection for an implantable device [i.e., ICD or cardiac resynchronization therapy (CRT)]. Current evidence has shown that LVEF-based criteria for SCD risk stratification have suboptimal sensitivity and specificity, which could be improved by the addition of the analysis of midwall fibrosis at LGE [19].

Otherwise, all-causes mortality and SCD/aborted SCD rates are significantly higher in patients with midwall fibrosis compared with those without fibrosis (27% and 30% *versus* 11% and 7%, respectively). Moreover, the presence of LGE identifies subjects at higher risk of hospitalization for HF [20]. Therefore, new algorithms for the appropriate selection of candidates to ICD based on the combination of low LVEF and presence of fibrosis have been proposed and may be implemented in clinical practice in the near future. Similarly, LGE may be helpful for predicting response to biventricular pacemaker resynchronization therapy and for guiding LV lead deployment away from scarred myocardium. Compared with pacing non-scar, pacing scarred myocardium is associated non-only with a markedly reduced clinical response, but also with a higher risk of cardiovascular death, hospitalizations for HF and major cardiac events. Among patients with a class I indication for CRT, the absence of myocardial scar or a scar mass <10 g identifies CRT patients at low risk of ICD therapies/SCD during long-term follow-up. Moreover, scar characterization, both quantitatively and qualitatively, may provide additional information to select patients who would benefit the most from CRT-D [21]. Regardless of the ability of LGE to predict response to resynchronization therapy, midwall fibrosis remains an independent predictor of morbidity and mortality in patients with DCM undergoing CRT-D implantation.

## Dilated cardiomyopathy: differential aetiopathology

DCM are classified in idiopathic or familiar; other pathologies can lead to a DCM. In patients with recent onset of DCM, identification of myocarditis, which occurs in about 30% of cases, has important clinical implications due to the high potential for LV recovery. The traditional Lake-Louis Criteria for identification of myocardial inflammation consists in the detection of necrosis or scarring at LGE, in conjunction with oedema at T2-weighted sequences and hyperaemia at early GE [22]. CMR represents a powerful positive diagnostic test, with a multi parametric evaluation of the LV involvement. The new T1-T2 mapping techniques increases the capability of CMR in diagnosing myocarditis.

Anthracycline cardiotoxicity is another possible cause of DCM, which may not become clinically apparent until over 10 years after exposure [23]. Global longitudinal strain (GLS) represents a sensitive and early marker of ventricular dysfunction. Increased subclinical interstitial fibrosis, represented by a T1 mapping/ECV elevation, can be identified before LVEF reduction and predicts decreases in LVEF in adult cancer survivors 3 years after receiving anthracycline-based chemotherapy [24].

## Infiltrative cardiomyopathies

Infiltrative cardiomyopathies (ICM) can mimic HCM, since deposition of abnormal substances in cardiac interstitial space can increase wall thickness. ICM includes the following different subtypes:

### Amyloidosis

Cardiac amyloidosis (CA) consists of an abnormal cardiac deposit of amyloid proteins, which accumulate in the extracellular space of the myocardium, and lead to direct cell toxicity, cell death, and expansion of the extracellular space. Clinically, deposition of amyloid proteins in the heart, and consequent diastolic dysfunction, cause heart failure and sudden cardiac death. The majority of abnormal proteins frequently encountered are light chain immunoglobulin (AL), wild type transthyretin (wtTTR), and mutant transthyretin (mtTTR). Some features are typical on CMR examination. The infiltration of amyloid proteins involves all the 4-chambers, with consequent LV and RV hypertrophy, thickening of the atria, interatrial septum and atrio-ventricular valves. The left ventricle LV hypertrophy is generally concentric (Figure 6A). The EF is preserved or mildly reduced, with global hypokinesia; in contrast, longitudinal strain is reduced in the early stage. Global longitudinal strain is preserved in apical segment, with an "apical sparing" pattern, specific of amyloidosis. Post-contrast CMR images show a typical pattern: characteristic alterations in the inversion time with difficulty to nulling of the myocardium (rapid wash-out from the blood pool of gadolinium with its storage in amyloid deposits), global subendocardial LGE in a non-coronary artery territory distribution [25]. Transmural LGE can be found and is associated with a 5-fold increase in mortality compared with patients with CA without LGE [26,27]. LGE can be present in LV, RV and atria walls (Figure 6B).

The diagnosis of the types of amyloidosis, in particular the distinction between light chain (AL) and transthyretin (ATTR) amyloidosis, may be challenging. In most cases, ATTR have a more increased left ventricular (LV) mass, a thicker interventricular septum (IVS), larger atrial areas, smaller cavity volumes and a lower LV ejection fraction (LVEF) than AL amyloidosis [28]. Myocardial enhancement on LGE sequence is reported to be more intense in ATTR than in AL amyloidosis, with predominant transmural enhancement and frequent right ventricular involvement [29]. More recently, using parametric imaging, it was reported that ATTR amyloid deposits were larger than AL amyloid deposits suggesting a concomitant myocyte hypertrophy. AL amyloidosis was associated with a greater elevation of myocardial native T1 and a smaller ECV suggests myocardial oedema. Myocardial native T2 significantly is increased in CA, especially in AL patients in comparison to ATTR patients [30].

### Anderson-Fabry disease

It is a rare X-linked autosomal recessive disease, due to a mutation of lysosomal enzyme, alfa-galactosidase A, with

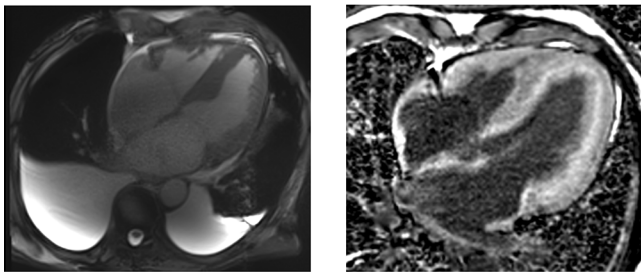


**Figure 6. A case of amyloidosis. A) SSFP sequence shows concentric LVH, pleural effusion, mild pericardial effusion. B) PSIR LGE sequence showing contrast uptake in all the walls in 4C view.**

consequent intra-cellular accumulation of glycosphingolipids. It occurs commonly in men during the third or fourth decade of life. The phenotype is characterized by LV concentric hypertrophy; some patients can present a different phenotype, with apical or asymmetric septal hypertrophy. There is a good correlation between LVH, native T1 and global longitudinal strain, the last two being an early marker of disease. GLS is a more sensitive functional marker than EF. As storage occurs early before the establishment of hypertrophy, native T1 mapping is an early marker of the disease. Typically, native T1 mapping is lower than in any other disease, together with iron overload. However, as the latter, ECV is normal in Anderson-Fabry disease, as it is caused by an intra-cellular storage, without increasing in ECV as shown in Figure 2. T1 mapping is crucial for differential diagnosis since it shows characteristically reduced native T1, unlike all other forms of HCM [31]. LGE is present in half of patients and shows a typical focal basal infero-lateral midwall or sub-epicardial uptake. Early detection of cardiac involvement in Anderson-Fabry disease is crucial because therapy may alter disease progression. In this context, CMR has been used to follow regression of LV hypertrophy with enzyme replacement therapy [32].

### Sarcoidosis

The cardiac involvement occurs approximately in 25% of cases in the United States and >50% in Japan [33]. It often consists of patchy granulomatous inflammation, LV dilatation with regional and global hypokinesia/dyskinesia and wall thinning. The presence of LGE, occurring in a wide variety of patterns, is associated with a 30-fold increased risk of death and aborted SCD and significant ventricular arrhythmia even in patients with preserved LVEF [34]. LGE imaging, together with T2-weighted sequences may also be used to assess the response to steroid therapy [35].

### Cardiac iron overload

Most frequently encountered in patients with transfusion dependent-thalassemia or hemochromatosis, it is associated with a long asymptomatic phase of progressive myocardial damage with sudden onset of either malignant ventricular arrhythmias or acute heart failure. However, intensive iron chelation therapy can completely restore cardiac function in most patients with asymptomatic cardiac dysfunction and even in patients with clinical HF. T2*-weighted sequences in CMR are particularly well suited in the detection and quantification of both myocardial and hepatic iron-overload and in the serial monitoring of patients necessitating chelation therapy. Iron overload is considered severe if T2* is <20 ms in the LV medial septal segment. It has been demonstrated that a T2*-guided chelation therapy has resulted in a significant decrease in cardiac morbidity and mortality in patients with thalassemia requiring frequent blood transfusions [36].

## Restrictive cardiomyopathy (RCM)

Endomyocardial fibrosis (EMF) is a cause of primary restrictive cardiomyopathy (RCM) with highest prevalence reported in sub-Saharan Africa, South Asia, and South America. The etiopathogenesis of EMF is not well known. Eosinophilia and parasitic infection can be a trigger to autoimmunity; also, malnutrition, ambient factors and genetic predisposition can play a role to the development of fibrosis of the endomyocardium. Morphological features are an apical obliteration of one or both ventricles, atrial enlargement, atrio-ventricular valve regurgitation and pleural/pericardial effusion. The fibrosis affects mainly the apex of the involved ventricle, and eventually extending to the inflow tract, sparing the outflow tract. CMR findings are a typical sub-endocardial LGE with a "double V" sign, consists of three layers (normal myocardium, thickened enhanced fibrotic endomyocardium, and apical thrombus. LGE quantification is a strong independent predictor of mortality (a volume of apical fibrous tissue deposition indexed to BSA of greater than 19 mL/m$^2$) [37]. Clinical presentation is a restrictive pattern with diastolic HF. CMR allows easy differential diagnosis with apical HCM, also known as Yamaguchi disease, in which intra myocardial rather than sub endocardial LGE is present, with no intraventricular thrombus. The prognosis is poor in the absence of surgical intervention.

## Arrhythmogenic cardiomyopathy

Arrhythmogenic cardiomyopathy (ACM) was previously defined as arrhythmogenic RV dysplasia, and it is now named ACM for the frequent LV involvement. The diagnosis is challenging. CMR can show RV/LV global and segmental dysfunction, fibrosis infiltration and LGE with a patchy non-ischemic pattern. While large RV/LV dysfunction can be easily detected by TTE, focal dysfunctions (as in sub-tricuspid region, typical localization of the "triangle of dysplasia") can be missed. Fat infiltration, prior considered pathognomonic of arrhythmogenic RV dysplasia, is no longer a criteria for the diagnosis according to the new guidelines (Figure 7 A,B) [38].

Major criteria for the RV are represented by: regional RV akinesia, dyskinesia, or bulging with the addition of either global RV dilatation (increase of RV EDV according to the imaging test specific nomograms), or global RV systolic dysfunction (reduction of RV EF according to the imaging test specific nomograms). The minor criteria are: regional RV akinesia, dyskinesia or aneurysm of RV free wall and transmural LGE (stria pattern) of ≥1 RV region(s) (inlet, outlet, and apex in 2 orthogonal views). The LV criteria are new diagnostic parameters described as major if LGE (stria pattern) of ≥1 bull's eye segment of the free wall (subepicardial or midmyocardial), septum, or both (excluding septal junctional LGE) are found [39]. They are defined minor when global LV dysfunction (as reduced LV EF or reduced global longitudinal strain), with or without LV dilatation is found, or when an increase of LV end-

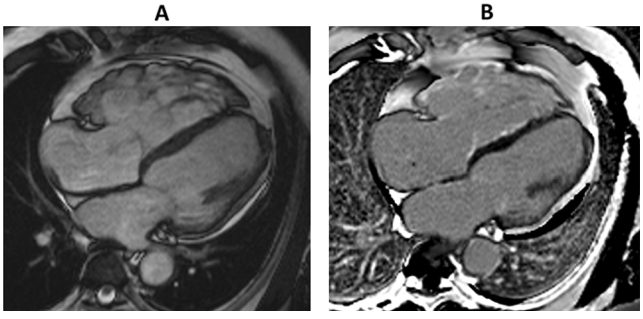


**Figure 7. A case of arrhythmogenic CMP (ACM). A) A case of RV dilatation with multiple dyskinetic aneurism of the free wall at cine SSFP sequence in 4C view. B) The same patient shows extensive Gadolinium contrast uptake in the free wall at LGE sequence.**

diastolic volume according to the imaging test specific nomograms for age, sex, and BSA is presented. Last if regional LV hypokinesia or akinesia of LV free wall, septum, or both are documented [40].

## Unclassified CMP

In this subgroup, the left ventricular non-compaction and stress CMP are included.

### Left ventricular non-compaction

TTE may lead to a false diagnosis of apical HCM in some cases of LV non-compaction (LVNC) due to the difficult visualization of apical segments. This is characterised by abnormal trabeculations, most commonly at the apex, and is often associated with ventricular myocardial thinning, dilatation or impairment of systolic and diastolic function [41]. Many CMPs may present a certain degree of trabeculations, particularly in presence of global LV dilatation, thus the correct diagnosis of LVNC is challenging. CMR criteria for LVNC are a non-compacted to compacted myocardium ratio of >2.3, presence of systolic or regional wall motion abnormalities, with reduced global longitudinal strain. The diagnosis of non-compaction is based on a criterion of trabeculated LV mass >20-35%. LGE may be seen in LVNC and is predictive of cardiac events, as well as LV dilatation and LV dysfunction, but not the extent of trabeculations (Figure 8) [42]. CMR criteria are sensitive but not highly specific, and need to be integrated with clinical criteria of symptomatic HF or LV dysfunction.

### Stress cardiomyopathy

Stress CMP (also called apical ballooning syndrome, Takotsubo CMP, broken heart syndrome) is a syndrome characterized by transient regional systolic dysfunction, principally, of the LV, mimicking myocardial infarction, but in the absence of angiographic evidence of obstructive coronary artery disease or acute plaque rupture. Stress cardiomyopathy is much more common in women than men and occurs predominantly in older adults. In the International Takotsubo Registry of 1750 patients with stress CMP, 89.9% were women and mean age was 66.4 years [43]. We use the following proposed Mayo Clinic diagnostic criteria, all four of which are required for the diagnosis: i) Transient LV dysfunction. The wall motion abnormalities are typically regional and extend beyond a single epicardial coronary distribution; rare exceptions are the focal (within one coronary distribution) and the global type. ii) Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture. iii) New ECG abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin. iv) Absence of pheochromocytoma or myocarditis. A serial assessment of LV function with identification of wall motion abnormalities is required and identify characteristic patterns - the apical type with systolic apical ballooning of the LV. Less common (atypical) variants are: the mid-ventricular type and basal type, focal type (with dysfunction of an isolated LV segment) and global type. Although reports of stress CMP have focused on transient dysfunction of the LV, there is evidence that approximately one-third of cases involve both right and left ventricle. CMR imaging may be helpful in the diagnosis and evaluation of stress CMP, particularly when the echocardiogram is technically suboptimal and/or there is coexistent coronary artery disease. CMR may assist in the differential diagnosis, delineate the full extent of ventricular abnormalities, and identify associated complications. Key CMR features of stress CMP are represented by absence of LGE, in contrast to MI in which it is intense transmural or subendocardial, and myocarditis (where is represented as patchy); evidence of myocardial oedema, as is also seen in acute myocardial infarction and myocarditis.

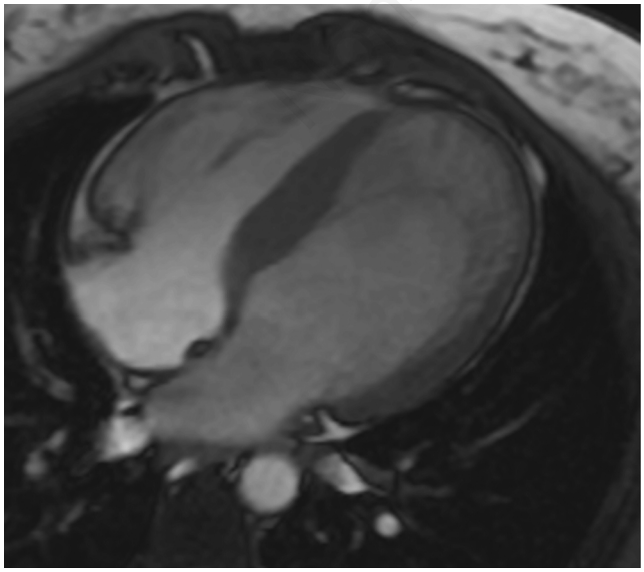

**Figure 8. A case of LV non compaction (LVNC) shown in SSFP sequence 4C view.**

## Conclusions

CMRI has revolutionized our understanding and management of CMPs. Because of its excellent ability to non-invasively characterise tissue, cardiac CMR has emerged as particularly useful in patients affected with CMPs. The classification of CMPs has changed over the years, being updated with knowledges. This review aims to define the features that can be highlighted by CMR in all the CMPs described by the current classification, excluding ischemic CMP. It results a description that might be useful to the clinician in the clinical practice.

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