Myocardial late gadolinium enhancement patterns made easy: non-ischaemic cardiac disease at a glance

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Learning objectives

The aims of this educational exhibit are:

• to review the spectrum of non-ischaemic diseases affecting myocardium
• to cover their appearance in late gadolinium enhancement sequences of cardiac magnetic resonance imaging in a comprehensive way.
Background

The term "cardiomyopathy" stands for a heterogeneous group of diseases that affect the cardiac muscle tissue, leading to a mechanical and/or electrical dysfunction. In many cases the underlying abnormality (the death of myocytes and their subsequent replacement by non-cardiac tissue) macroscopically will be seen as a hypertrophy or dilatation of the ventricular wall.

There are two major groups of cardiomyopathies to be distinguished according to their origin: primary and secondary. Primary cardiomyopathies (genetic, mixed or acquired) only affect the cardiac muscle while in case of a secondary cardiomyopathy the myocardial involvement is part of a multi-organic disorder [1].

Ischemic cardiomyopathy has been an ambiguous term, as the myocardial damage is this case is secondary to the occlusion of an epicardial artery. The extension of the injury will typically be segmental and limited to the epicardial artery territory. When this injury is extensive enough to impair the cardiac function, an ischaemic cardiomyopathy develops.

Cardiac magnetic resonance imaging has emerged as one of the most powerful techniques in evaluating the cardiac morphology and function. It provides high-quality images that allow the characterization of the myocardial structure (presence of oedema, haemorrhage, fibrosis, intra-myocardial deposits such as amyloid or iron) and cardiac function.

In the assessment of cardiomyopathies the evaluation of myocardial fibrosis is of special relevance. Considering the distribution pattern of myocardial fibrosis combined with other features such as cardiac hypertrophy or dilatation can aid for an accurate diagnosis.

The aim of this poster is to review the main radiologic patterns that allow the correct characterization of the principal types of cardiomyopathies.
Findings and procedure details

Technique

The evaluation of fibrosis by cardiac MRI is achieved by using contrast material enhanced sequences due to the property of gadolinium to shorten the T1-relaxation and increase the signal intensity on T1-weighted images.

The gadolinium chelates present a totally extracellular distribution, diffusing from intravascular to extravascular space without passing through the myocyte cell membranes. Normally the myocardial extracellular volume is small. Myocytes are densely packed within the myocardium and therefore intracellular space accounts for 85% of the myocardial volume (2).

In some pathological conditions the effective extracellular space increases, which leads to a retention of gadolinium within the myocardium. This can be seen in the setting of acute myocardial injury (acute infarction, myocarditis) or in chronic situations when fibrosis replaces the normal myocardium or when a deposit material (for example, amyloid) expands the extracellular space.

The most widely used sequence for the evaluation of myocardial damage is a two dimensional T1 weighted, ECG gated, inversion recovery (IR) spoiled gradient echo (SPGR) sequence (3) (The trade name varies depending on the manufacturer (4): FLASH for Siemens, SPGR for GE, and FFE for Philips and Toshiba).

The inversion recovery pulse is used to null the normal myocardium and optimize the contrast difference with the gadolinium retaining tissue.

The delay to achieve the optimal contrast between normal and abnormal myocardium is 10-30minutes. Imaging too early or too late results in a reduced image contrast (5).

The clearance of contrast material from normal myocardium depends on several factors and is very variable from one patient to another. In order to ensure the correct differentiation of normal and damaged tissue the inversion time (TI) is selected just before the imaging acquisition. For this purpose a "look-locker" or "TI scout" sequence of multiple low resolution images with different TI is obtained. Afterwards the TI that produces the best contrast between normal and abnormal tissue is chosen (5). An example of a "look-locker" sequence is given below, illustrating a case of amyloidosis (Fig.9).

Practical approach to cardiomyopathies
In most of cardiomyopathies the location (subendocardial, mid-wall (mesocardial), subepicardial) and distribution (homogeneous, patchy, diffuse, focal) of the abnormal myocardium follows a relatively specific pattern in each pathological condition. Besides, in combination with the assessment of wall thickness (hypertrophy or dilatation) an accurate differential diagnosis may be achieved. (Fig. 1).

Fig. 1: A scheme illustrating a practical approach to non-ischemic cardiomyopathies using late gadolinium enhancement and thickness of LV wall. 

References: Hospital de la Santa Creu i Sant Pau, Barcelona, Spain.

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) probably is the most frequent cardiomyopathy with a prevalence of 1:500 in general population. It is the most common cause of sudden cardiac death (SCD) in young adults (including athletes) and represents an important substrate for heart failure at any age (6).
Clinical manifestations are widely variable with a spectrum that ranges from asymptomatic patients to sudden cardiac death. When symptoms are present, chest pain, and dyspnoea on exertion or syncope are the most common.

The inheritance of the disease is autosomal dominant with more than 400 individual mutations of up to 11 mutant genes being able to produce the HCM phenotype. Of these the gene the b-myosin and myosin-binding protein C are the most commonly implicated (6). Such variability in the genetic abnormalities is likely to be related to the diversity of clinical presentation.

In gross examination it is common to observe a non-dilated left ventricle (LV) with thickened ventricular walls and increased cardiac mass. Microscopically myocyte hypertrophy with myocyte and myofibrilar disarray is the hallmark of HCM. Along with these key features focal interstitial fibrosis, replacement endocardial fibrosis and dysplasia of the small arteries are also seen (7,8).

However, it is important to note that the genetic defect does not necessarily have a clinical expression. Even in case of normal ventricular wall thickness a HCM mutant gene may be present.

Non-invasive diagnosis of HCM is based on the presence of LV wall thickness >15mm at the end diastole or septal-to-lateral wall thickness ratio higher than 1.3 in a non-dilated myocardium. Up to 7 different patterns of distribution of the hypertrophy have been reported (8). (See Table 1).
### Distribution patterns of HCM

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Description</th>
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| Asymmetric HCM with sigmoid septal contour ("septal HCM")              | - The most common morphology pattern (2/3 of the cases).  
- Hypertrophy of the antero-septal myocardium resulting in a sigmoid contour of the septum.  
- The bulging of the septum into the left ventricle outflow tract (LVOT) usually alters the blood hydrodynamics and leads to the anterior movement of the mitral valve during the systole (systolic anterior movement of mitral valve or SAM). That results in sub-aortic obstruction and concomitant mitral insufficiency. |
| Asymmetric HCM with sigmoid reversed septal contour                     | - The septum hypertrophies as a reversed S-shaped curve and does not cause LVOT obstruction.                                              |
| HCM with mid-ventricular obstruction                                    | - Hypertrophy seen in mid-ventricular wall that results in the so-called "dumb-bell" appearance with mid-cavity narrowing.               |
| Apical HCM                                                             | - This pattern is more frequent in Japanese population and is characterized by hypertrophy seen in the apex, obliteration the LV cavity and the "spade-like” configuration. |
| Symmetric (concentric) HCM                                             | - Hypertrophy of the LV wall without regional preferences in the absence of hypertension or aortic stenosis.                          |
| Focal HCM                                                              | - Focal mass-like thickening of the LV. First pass perfusion, LGE and tagging sequences are useful in differentiating neoplastic masses from hypertrophied myocardium. |
| Right Ventricle (RV) involvement                                       | - Up to 18% of patients. Usually the mid-to-apical portion is affected.                                                                     |

**Table 1**: Table 1. Summary of different morphologic patterns of HCM and its main features.

**References**: Hospital de la Santa Creu i Sant Pau, Barcelona, Spain.

LGE it is seen in up to 80% of patients with HCM (9). Its distribution is non-specific but usually involves the septum and manifests as small patchy mid-wall enhancement areas with predilection to the segments of greater hypertrophy or the LV/RV insertion points (8,9). In some cases fibrosis can be rather extensive. See Cases 1, 2, 3 (Fig. 2-8).
Fig. 3: Case 1. Hypertrophic cardiomyopathy. Long axis four chamber view image in end-systole.

References: Hospital de la Santa Creu i Sant Pau, Barcelona, Spain.
Fig. 5: Case 1. Hypertrophic cardiomyopathy. Long axis T1-weighted image, showing patchy late gadolinium myocardial enhancement pattern at left ventricle's apical level. References: Hospital de la Santa Creu i Sant Pau, Barcelona, Spain.
**Fig. 7**: Case 3. Hypertrophic cardiomyopathy. Short axis cine still image in end-systole. Note the hypertrophied basal septum.

**References**: Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT
**Fig. 8**: Case 3. Hypertrophic cardiomyopathy. Short axis T1-weighted image (same level as previous image), showing late gadolinium myocardial enhancement at superior LV/RV insertion point and mid-wall enhancement of basal septum and anterior wall.

**References**: Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT

The presence of LGE in patients with HCM has an important prognostic significance, since it has been reported as an increased risk for sudden cardiac death, systolic dysfunction and non-sustained ventricular tachycardia compared with patients without LGE in CMR (8). Therefore LGE sequence may have an important role in improving the risk assessment in borderline or controversial cases benefiting from ICD (9).
**Secondary left ventricle hypertrophy**

In situations where left ventricle chronically pumps against an increased back pressure it becomes hypertrophied, which is the base of a hypertensive cardiomyopathy. The most common cause of left ventricle hypertrophy (LVH) is chronic systemic hypertension and less commonly, aortic stenosis.

Hypertensive heart disease is linked to an increased cardiac mortality and cardiac events such as atrial fibrillation, heart failure or myocardial infarction (7).

On the gross examination homogeneously hypertrophied left ventricle is seen, without disproportionate septal thickening. Besides, the grade of hypertrophy usually is lesser than in HCM. Microscopically small areas of fibrosis may be observed in the left ventricle and also in the right ventricle or in both atria (7). In general the more severe the chronic hypertension, the more hypertrophy and fibrosis can be observed (7). No typical pattern of LGE has been described. When present, a focal involvement of non-subendocardial distribution is characteristic.

In case of left ventricle hypertrophy secondary to aortic stenosis, a patchy distribution affecting the mid-wall or subendocardium of basal segments may be present (9). The presence of LGE is associated with a greater ventricular mass and higher probability of adverse cardiac events (9).

**Anderson-Fabry disease**

Anderson-Fabry disease is a X-chromosome-linked disease of lysosomal metabolism resulting in the accumulation of sphingolipids with repercussions in multiple organs.

The clinical hallmark begins in the childhood with a broad variety of neurological, ocular and cutaneous symptoms. The most severe complications are related to progressive nephropathy, which typically progress to kidney failure, cardiac involvement and early stroke (less common) (10).

The cardiac manifestations consist of left ventricular hypertrophy (which is seen in up to 30-50% of the patients) development of fibrosis in the basal posterolateral segments and conduction abnormalities (10).

The hypertrophy is typically concentric although asymmetrical septal thickening may occur in sever cases, mimicking HCM. LGE it is present in 50% of the cases and
characteristically involves the mid-wall of the basal inferolateral region of the LV. This is a key point since the isolated involvement of inferolateral wall is rare in HCM (8,9).

**Amyloidosis**

Amyloidosis is a systemic glycoprotein storage process, which occurs due to the failure of normal protein folding and its subsequent deposition in the extracellular space. The disease affects various tissues and organs. Cardiac involvement can be associated with either amyloid light chain (AL) type amyloidosis, where the pathologic protein is produced by plasma cells, or the amyloid transthyretin type amyloidosis (ATTR). Transthyretin is a plasma protein, produced in the liver; in case of TTR gene mutations it expresses as familial amyloidosis (mostly in Afro-Caribbean populations), whereas non-mutant TTR amyloidosis occurs sporadically in older Caucasian people, causing the so-called senile systemic amyloidosis (11). In the heart the pathologic fibrils in the interstitial space cause thickening of the ventricular walls, diastolic dysfunction; atrial walls and septum thicken with subsequent dilatation due to end-diastolic pressures.

Clinically, amyloidosis appears as right-sided heart failure due to a restrictive process, arrhythmias and sudden cardiac death (7).

Abnormal protein within the extracellular space causes characteristic changes of the signal intensity in T1-weighted imaging, which results in the typical difficulty to "null" the myocardium in the inversion-recovery sequences because of shorter inversion times comparing with blood pool; this feature, commonly resulting in difficulties of acquiring correct inversion time, can give a clue about the diagnosis already in the TI scout ("look-locker") sequences (7). See Fig.9.
**Fig. 9**: Look-locker sequence. Note the difficulty to "null" myocardium with different inversion times.

**References**: Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT

On late gadolinium sequences, diffuse enhancement within the thickened walls can be observed; it is mostly subendocardial, but non-ischemic in distribution (7). See Case 4 (Fig. 10-11).
**Fig. 10:** Case 4. Confirmed amyloidosis. Long axis T1-weighted image. Apart from breathing artefact, note the difficulty to "null" myocardium. Enhancement in LV septum with extension to mid-wall as well as some subendocardial enhancement of RV septum is observed.

**References:** Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT
Fig. 11: Case 4. Confirmed amyloidosis. Short axis T1-weighted image. Note the difficulty to "null" myocardium. Enhancement at LV septum, anterior and inferolateral and inferior walls is observed.

References: Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT

The so-called "zebra" appearance is characteristic for amyloidosis - where subendocardial enhancement of right and left ventricle is seen, separated by low-signal normal myocardium of the septum (12). Affected right ventricle as well as more diffuse and transmural distribution of LGE can suggest ATTR type amyloidosis (11). The involvement of myocardium is a prognostic tool, which can aid for treatment (12).
Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is characterized by ventricular chamber enlargement and systolic dysfunction with normal or reduced left ventricular wall thickness, in a context of no identifiable coronary artery disease.

Its estimated prevalence is 1:2500 representing the most common cause of cardiac transplantation and the 3rd most common cause of heart failure (6). Approximately 50% of the cases are idiopathic, whereas the rest are due to familiar inheritance (20-35%) or secondary to a broad range of environmental etiologic factors. The most common of them is a viral infection (coxsackievirus, adenovirus, parvovirus, HIV). Other frequent causes include toxic agents (excessive consumption of alcohol, chemotherapeutic agents), autoimmune or systemic disorders and neuromuscular disorders (6).

DCM leads to a progressive impairment of ventricular contractility and conduction system abnormalities resulting in progressive heart failure or ventricular and supraventricular arrhythmias. Because of the stagnant blood flow secondary to the ventricular dilatation intracavitary thrombus may form, leading to embolic pathology. Death usually is related to malignant arrhythmias or heart failure.

Grossly cardiac hypertrophy (indexed weight of 50-150%) with ventricular dilatation is observed in the setting of normal or decreased wall thickening. In early stages only the left ventricle is affected with progressive involvement of right ventricle and subsequently dilatation of all chambers in final stages. Microscopically the findings are non-specific with myocyte hypertrophy and fibrosis, usually in the base and mid-ventricular regions (13).

In CMR approximately the 60% of the patients has no enhancement, probably reflecting the mostly microscopic distribution of the fibrosis. When LGE is seen the typical pattern is in form of stripes and patches in the basal to mid-ventricular regions (Figure 13). In a minority of patients with DCM subendocardial LGE may be seen. In such cases coronary artery disease with reanalysed coronary arteries should be considered (14).

When present, LGE has important prognostic implications. Independently of LVEF, the presence of fibrosis is related with an increased likelihood of all cause mortality and also of malignant arrhythmias, heart failure hospitalization or cardiac transplantation; therefore LGE may be a hopeful advancement in the risk stratification and management of patients with DCM (9). See Case 5 (Fig. 12).
Fig. 12: Case 5. Dilated cardiomyopathy. Short axis T1-weighted image, showing linear mid-wall myocardial enhancement in the septum as well enhancement at RV/LV insertion points.

**References:** Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT

**Sarcoidosis**

Sarcoidosis is a multi-systemic inflammatory disorder, characterised by non-caseating granulomas. Cardiac involvement is not uncommon; in the USA ¼ of sarcoidosis cases affect heart, which results in 13-25% of deaths, meanwhile in Japan cardiac involvement accounts for 80% of cases and reaches 50% of deaths. By the criteria of Japanese Ministry of Health and Welfare (JMHW), mortality at 5 years can reach up to ¼ of cases. (15)
Patients, referred to a CMR scan, both those with confirmed sarcoid process and not, can either be asymptomatic or symptomatic.

The revised Japanese Criteria (2006) among other modalities include gadolinium-enhanced MR imaging with late enhancement findings within myocardium as minor criteria.

CMR allows to evaluate cardiac morphology, function and to characterise tissue (inflammation and fibrosis). Sarcoidosis usually presents with mid-wall or subepicardial patchy late gadolinium enhancement pattern. However, subendocardial and transmural enhancement is not uncommon as well (16). Basal and mid wall regions are affected in most cases (17,18). Overall, the appearance of sarcoidosis in late enhancement sequences is variable; therefore it can sometimes mimic other conditions.

Late gadolinium enhancement seems to be useful for prognosis of outcome; with myocardial scar as the best independent prediction tool of death or potentially lethal events such as aborted sudden cardiac death and appropriate implantable cardioverter-defibrillator (ICD) discharge, ventricular tachycardia (VT) and non-sustained VT (19). See Case 6 (Fig 13, 14).

**Fig. 13:** Case 6. Sarcoidosis. Long axis T1-weighted IR image, showing extensive transmural enhancement in the lateral/inferolateral, subepicardial enhancement of anterior wall from basal-to-mid cavity levels, as well as subenndocardial enhancement basal anteroseptal level. There is late gadolinium enhancement in the basal level of RV anterior wall

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Myocarditis

Myocarditis is an inflammation of myocardium (acute or chronic), which is caused by a range of infectious, toxic and drug agents. Clinically, it can vary from fatigue, chest pain, raised troponin levels to arrhythmias and sudden cardiac death (6).
On cardiovascular magnetic resonance imaging, late gadolinium sequences in case of myocarditis provide a distinctive pattern of sub-epicardial and mid-wall (sparing subendocardial portion) enhancement with non-ischemic distribution. Lake Louise Consensus criteria include three CMR features for improving diagnostic accuracy:

- increased regional or global signal on T2-weighted images
- increased early gadolinium enhancement ratio (between myocardium and skeletal muscle)
- one or more focal lesions of late gadolinium enhancement without an ischemic distribution (20).

With two or more of these criteria positive, the diagnostic accuracy can reach up to 78% compared with endomyocardial biopsy. Moreover, the segmental distribution of LGE findings can aid for a successful subsequent endomyocardial biopsy. Case 7 (Figures 15, 16)
Fig. 15: Case 7. Myocarditis. Four chambers T1-weighted IR image, showing mid-wall enhancement of the septum at the mid-cavity level.

References: Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT
Fig. 16: Case 7. Myocarditis. Short axis T1-weighted IR image, showing mid-wall enhancement of the septum and inferior/inferolateral wall at the mid-cavity level.

References: Radiology, Hospital of Lithuanian University of Health Sciences - Kaunas/LT

Chagas disease (named after the Brazilian physician Carlos Chagas) is a tropical parasitic disease, also known as American trypanosomiasis. When cardiac muscle is involved, it presents as myocarditis and is one of the major factors of heart failure in Latin America. Its appearance on the late gadolinium enhancement sequences typically involves ischemic and non-ischemic LGE patterns, affecting mostly apical and mid-inferolateral walls of myocardium, within the areas of thinning and aneurysms of left ventricle (21). The amount of fibrosis may be a predictor of patient's prognosis (22). Case 8 (Figure 17).
**Fig. 17:** Case 8. Chronic Chagas disease. Long-axis T1-weighted IR images, showing linear late gadolinium enhancement in basal-to-mid cavity level with extension to apical lateral wall.

**References:** Hospital de la Santa Creu i Sant Pau, Barcelona, Spain.
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Fig. 6: Case 2. Hypertrophic cardiomyopathy. Short axis T1-weighted image, showing late gadolinium myocardial enhancement at LV/RV insertion points. Note LV wall thickness.

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Fig. 7: Case 3. Hypertrophic cardiomyopathy. Short axis cine still image in end-systole. Note the hypertrophied basal septum.

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Conclusion

Non-ischaemic myocardial disease stands for a broad and heterogeneous group of entities with morphologic or electrical repercussion over the heart. The diagnosis and risk stratification of these diseases may be difficult due to non-specific clinical, ultrasound and ECG findings.

CMR has emerged as a powerful diagnostic technique of these pathologies. Combining the accuracy in the evaluation of left ventricular thickness with the detection of myocardial fibrosis an accurate specific diagnosis and risk stratification may be done.