
The Top Ten Cases in Cardiac MRI and the Most Important Differential Diagnoses

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Additional information is available at the end of the chapter

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1. Introduction

Noninvasive imaging plays a central role in the diagnosis of heart failure, assessment of prognosis, and monitoring of therapy. Cardiovascular magnetic resonance (CMR) offers a comprehensive assessment of heart failure patients and is now the gold standard imaging technique to assess myocardial anatomy, regional and global function, and viability. Furthermore, it allows assessment of perfusion and acute tissue injury (edema and necrosis), whereas in nonischemic heart failure, fibrosis, infiltration, and iron overload can be detected [1]. The American College of Cardiology/American Heart Association have recently updated their guidelines for imaging techniques used to assess patients with HF [2]. According to it, two-dimensional echocardiography is currently the imaging modality most commonly used in clinical practice to meet the ACCF/AHA requirements. It provides a good general assessment of LV function but is limited in patients with poor acoustic windows, it requires geometric assumptions in quantifying global LV systolic function, and its ability to provide specific tissue characterization is modest. CMR is the gold standard to LV and RV global and regional dysfunction, dilation, viability and what is the underlying etiology of HF. CMR makes it particularly well suited to studying the RV, which is difficult to assess with echocardiography.

The CE-MARC study [3,4] defines the role of Cardiac MR: 752 patients with suspected heart disease were recruited and scheduled for MR and SPECT as well as for an X-ray coronary angiogram used as the reference standard. Angiography identified 39% of the patients with CAD. Cardiac MR had a sensitivity and specificity of 86.5% and 83.4%, respectively. In comparison, SPECT delivered a sensitivity and specificity of 66.5% and 82.6 %, respectively. Viability/post-myocardial infarction scarring can be described with unprecedented resolution,

and highly specific patterns of fibrosis and scarring have been described for many nonischemic cardiomyopathic processes. DE-CMR appears to offer advantages in detecting small or subendocardial infarcts with high accuracy and is well validated. Late studies showed that the sensitivity of DE-CMR increased with increasing gadolinium dose, reaching 99% and 94% in acute and chronic MI, respectively. These surveys have shown that unrecognized MIs are common, comprising as many as 40% to 60% of all MIs.

Nearly one-half of HF patients have abnormalities in diastolic function with preserved ejection fraction. CMR can assess diastolic function in several ways. In an analogous manner to echocardiography, MR tagging is a sophisticated method for quantitative analysis of regional systolic and diastolic function.

2. Background

Cardiac MR is increasingly becoming the modality of choice in the diagnosis of coronary and cardiac disease. The aim of this guide is to cover the most frequent conditions encountered, highlight technical issues, differential diagnoses, and the most common pitfalls found when diagnosing patients.

Cardiovascular disease is the most frequent cause of mortality in the developed world. Although diverse, many techniques exist for diagnosing cardiac diseases, and it is frequently necessary to request several tests to reach a conclusive diagnosis. Magnetic resonance (MR) is a well tolerated and safe technique, which is currently available in a majority of hospitals. This technique makes it possible in a single exploration to study the anatomy of the heart, allowing qualitative, semi-quantitative, and quantitative assessments to be made of the parameters of cardiac anatomy and function. It provides information of cardiac and vascular anatomy and function in complex congenital and acquired cardiopathies. With the administration of intravenous contrast, it also enables us to visualize the extent of ischemic cardiopathies and assess myocardial viability. As such, cardiac magnetic resonance is emerging as one of the most promising techniques for the study of congenital and acquired cardiac pathology.

According to the American College of Cardiology [1], the proposed indications for CMR Imaging in patients with Heart Failure are:

- Serial assessment of biventricular structure, size, and function (anatomy, LV/RV volumes, global and regional systolic function, mass)
- Evaluation of native and prosthetic cardiac valves (planimetry of stenotic disease, estimation of peak stenotic velocities and gradients, quantification of regurgitant disease)
- Evaluation of cardiac masses, differentiation between tumour and thrombus
- Evaluation of great vessels and pulmonary veins

- Determination of the location and extent of acute (including no-reflow regions) and chronic myocardial necrosis
- Viability assessment before revascularization (LGE or low-dose dobutamine)
- Determination of the area at risk in patients with acute myocardial infarction and the salvageable area post-revascularization with percutaneous coronary intervention
- Identification of the presence and quantification of the extent of inducible ischemia (vasodilator perfusion or high-dose dobutamine stress CMR)
- Evaluation of suspected anomalous coronary origins (MR coronary angiography)
- Differentiation of ischemic versus non-ischemic cardiomyopathy
- Evaluation of myocarditis
- Evaluation of specific cardiomyopathies (in vivo tissue characterization)
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Arrhythmogenic right ventricular cardiomyopathy
- Cardiac amyloidosis
- Cardiac sarcoidosis
- Anderson-Fabry disease
- Iron overload cardiomyopathy
- Left ventricular noncompaction
- Other less common diseases (e.g., Chagas disease, endomyocardial fibrosis, Churg-Strauss syndrome, and so on)
- Assessment of mechanical dyssynchrony before resynchronization therapy
- Patients with technically limited images from echocardiogram
- Discordant information that is clinically significant from prior tests

3. A survival guide to cardiac MRI

3.1. Protocol

- a. Sequences: SE (black blood), gradient ECHO: CineMRI, perfusion, viability, flow quantification, motion tagging, and others.

- b. Planes: SA, 2CH, 3CH, 4CH, inflow and outflow.
- c. How to review:
 - Structure (size, thickness)
 - Function (motility, volumes, EF, cardiac output)
 - Valves (morphology, flow)
 - Ischemia: T2 STIR, perfusion first pass and stress, viability (DHE)

3.2. Sequences

BLACK BLOOD: SE (Spin echo) is the most suitable sequence to analyze the morphology of the heart and great vessels. The blood is black and fat is white. Variations are fast (or turbo). Spin echo (FSE or TSE) allows the acquisition of an entire image in a single heartbeat.

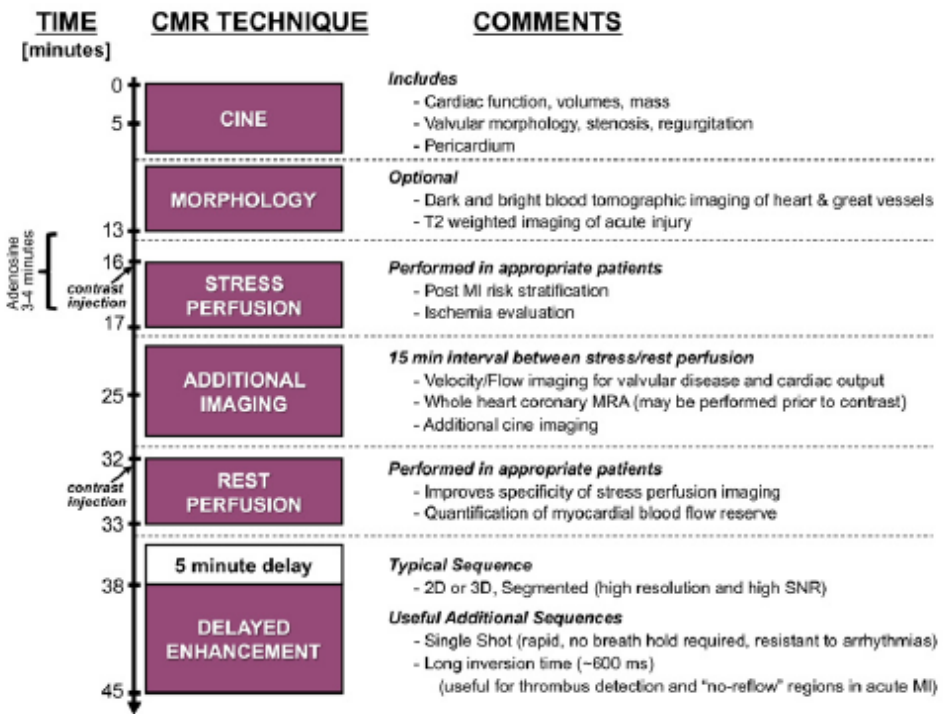


Figure 1. Protocol cardiac MR. Timeline and potential components of a multitechnique CMR examination for Cardiac imaging. 2D=2-dimensional; 3D= 3-dimensional; CMR= cardiovascular magnetic resonance; MI= myocardial infarction; MRA= magnetic resonance angiography; SNR= signal-to-noise ratio.

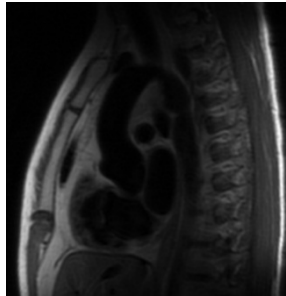


Figure 2. T1 Black Blood image using SENSE aortic outflow plane: coarctation of the aorta.

WHITE BLOOD: GE (Gradient echo). Blood and fat appear white. Variations: b-SSFP (balanced Steady-State Free Precession Imaging), FLASH and velocity mapping. b-SSFP cine sequence can also be applied 2D (single shot), as a real time technique (does not require breath holding or ECG triggering) and 3D volume scan.

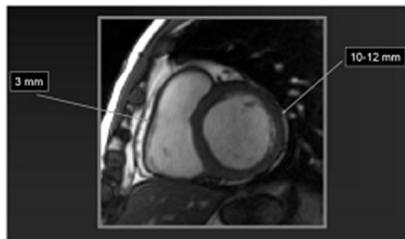


Figure 3. b-SSFP: 2 CH short axis: measure thickness RV and LV. Normal thickness RV 3mm LV 10-12mm.

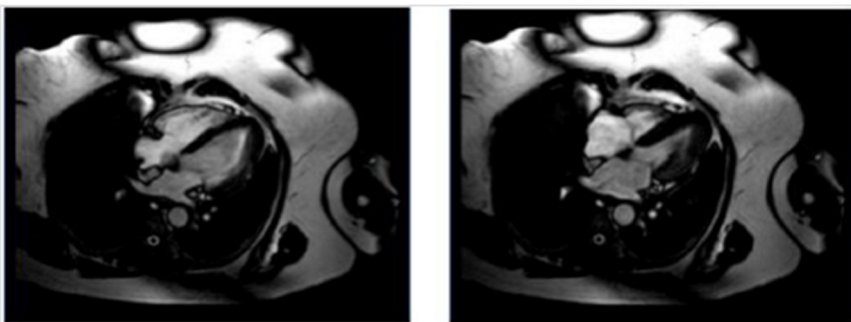


Figure 4. b-SSFP cine (diastole and systole): Global and segment analysis of movement and thickness.

It is used to analyze global and regional cardiac function, detecting abnormal flow, such as jets from stenoses of the valves or insufficiency, to study flows and quantify the degree of stenosis and regurgitation, to quantify $Q_p:Q_s$ between pulmonary and systemic flow, and is also useful in detecting congenital malformations. CineMR: To analyze the contractility of the heart wall. Analysis is performed for each segment (hypokinesia, akinesia, dyskinesia): There must also be evidence of wall thickening.

We can see jets (turbulent flow) in the valves or from the outflow of left or right ventricles.

- AV valves:

- Insufficiency or regurgitation, if the flow of the jet is directed into the ventricles.
- Stenosis: the flow of the jet originates from inside the ventricles.

- Aortic and pulmonary valves:

- Insufficiency, the flow of the jet is directed into the ventricles
- Stenosis: the origin of the jet comes from outside the ventricles from the vessels.

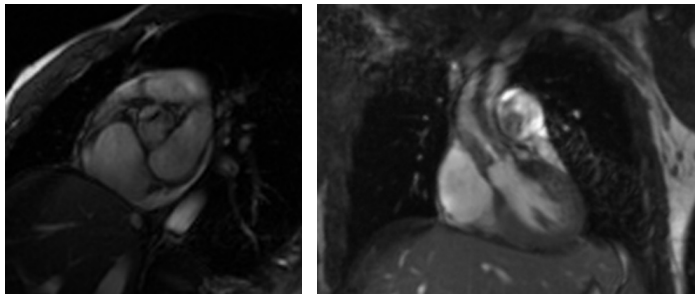


Figure 5. Plane aortic valve (bicuspid aortic valve) and plane outflow LV with jet of aortic insufficiency

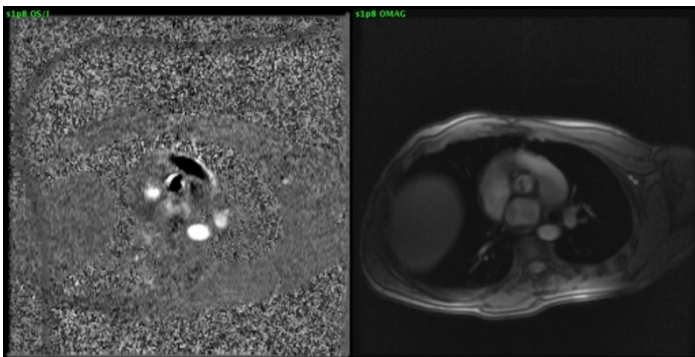


Figure 6. Q-Flow magnitude and phase encoding (velocity mapping): bicuspid aortic valve with regurgitation

VELOCITY MAPPING (Flow velocity mapping):

- Detect and quantify peak velocity in valvular stenosis.
- Calculate overflow in a mayor vessel through the cardiac cycle
- Quantification in regurgitation or valvular incompetence.
- Confirm abnormal chamber communication and the ratio of pulmonary to systemic flow in shunts, such as septal defects.
- Determine the average velocity: The operator selects the plane and sets a maximal encoding velocity (VENC), the initial VENC used is an approximation upper of the velocity expected.

INVERSION RECOVERY TECHNIQUE AND CONTRAST ENHANCEMENT MR (CE-MR):
Uses a pre-pulse to create high T1 tissue contrast to visualize infarct imaging (black myocardium), compound of gadolinium (Gd-DTPA) to detect insufficient perfusion in first pass, followed by a delayed DHE to detect scar or inflammatory changes (white myocardium in infarct, myocarditis and infiltrative diseases.) Stress MR can be obtained using adenosine or dobutamine as chemical stressors.

Furthermore, this can be used to evaluate cardiovascular physiology and anatomy, characterize tissues, and vascular angiography.

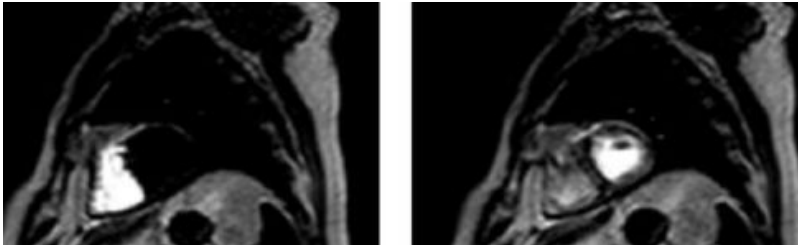


Figure 7. Early Enhancement Perfusion Short Axis SENSE (TR: 2, TE: 1)

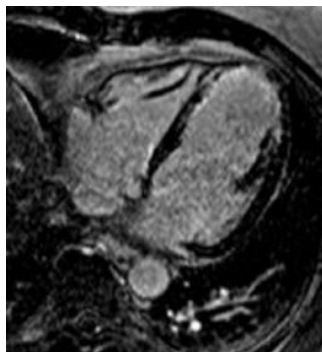


Figure 8. DHE Late contrast enhancement 8 minutes after injection of Gadolinium. Apical enhancement: scar of inferior infarction.

OTHERS:

- Contrast-enhanced MR angiography: study of aortic aneurism and dissection, congenital anomalies of great vessels, vasculitis evaluation, central thoracic veins, pulmonary artery anatomy, postsurgical follow-up and contraindications to computed tomography
- TAGGING IMAGING: Measurement the contractility of myocardium; T2 STIR: In edema, and T2* for quantification of haemochromatosis; Sequences fat suppression (SPIR), etc

3.3. Planes in cardiac imaging

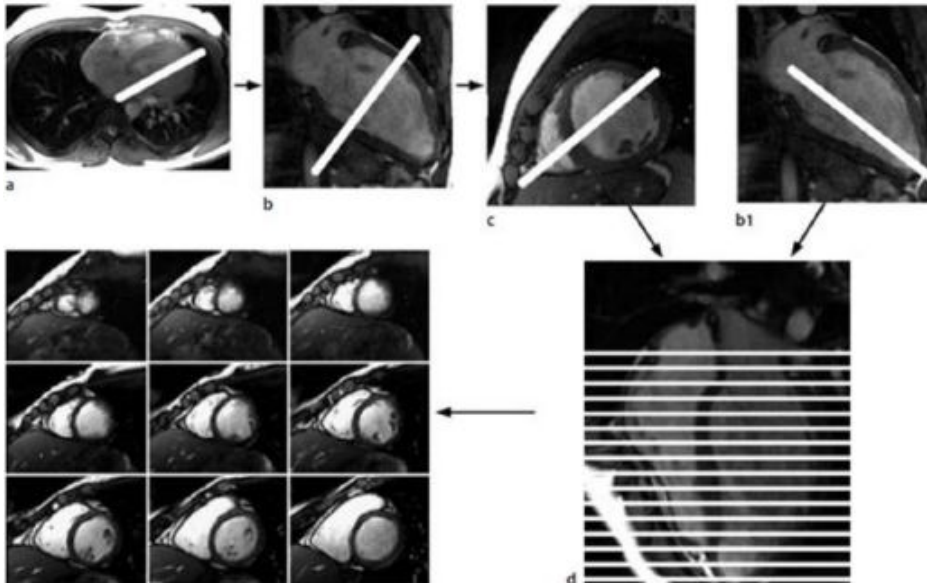


Figure 9. Protocols for cardiac imaging: a,b,c. Rapid pilot scans in the 3 orthogonal planes (axial, coronal and sagittal) in a breath-hold, FSE 1 imaging per cardiac cycle; d. Anatomical coverage from the diaphragm up to the thoracic inlet into the 3 orthogonal planes.

3.3.1. Specific standard cardiac planes (intrinsic) with GE cines and SE sequences

From the axial plane:

- 2CH (2 chamber) and LA (ventricular long axis) to acquire HA (horizontal long axis), inflow (mitral plane) and outflow (aortic plane)
- SA (Short Axis): inflow HA and 4CH (4 chambers).
- Real 4CH: from SA (short axis) between papillary muscles and inflow LA (mitral valve)
- 3CH (3 chambers): from LA and perpendicular aortic valve.

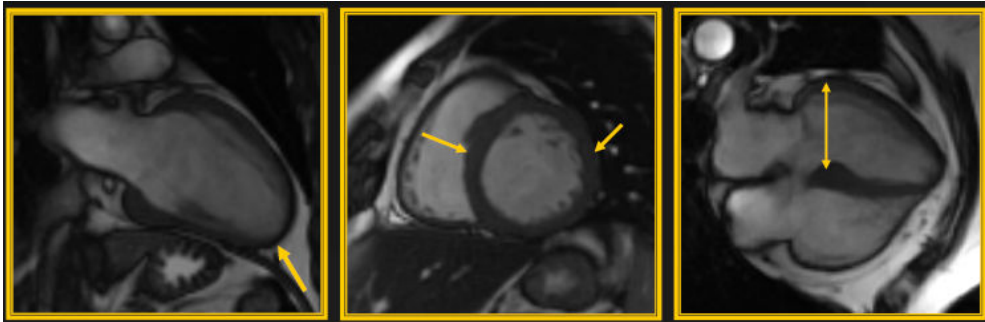


Figure 10. Ventricles size and wall thickness measurement procedure. First and second images: 2CH LA apex 3mm, SA lateral wall and septum 10-12 mm; Third image: 4CH LV size 50mm

3.3.2. Target CMR with additional planes and sequences, such inversion recovery

Sagittal, coronal or oblique planes, LV and RV outflow, valves plane, etc.

3.4. Topics to review

3.4.1. Structure

Size is measured in 4CH in systole and in diastole. Size of chambers in systole and diastole (ant-post): LV 50mm, RV 32mm and LA (left atrium) inferior a 40mm.

The thickness of the wall is measured (end of diastole) in SA (basal and middle) and LA (apical). Left ventricle 10-12mm (< 15mm hypertrophy) and right ventricle > 6mm.

3.4.2. Function

CINE SA and LA:

REGIONAL FUNCION:

Visual assessment of wall motion patterns using cardiac short axis b-SSFP cine: End- diastolic, mid- diastolic and end-systolic.

- Normokinesia: normal wall motion.
- Hypokinesia: Decreased wall motion.
- Akinesia: absent wall motion.
- Dyskinesia: wall motion in the opposite direction.
- Hyperkinesia: increased wall motion.

GLOBAL FUNCION:

CINE MRI SA and LA using b-SSFP is probably the best technique to quantify ventricular volumes, function, and mass. The most commonplace is the Simpson method: drawing epicardial and endocardial borders of LV in systole and diastole, this can also be used for the RV.

The following items are to be analyzed:

- LV undergoes a circumferential and longitudinal ventricular shortening and an extensive wall thickening during systole. The sequence allows the obtention of the ejection fraction ($EDV - ESV = SV$ (mL); $SV/EDV = EF$ (%).
- Ventricular mass to be measured in diastole.

| | Male | | Female | |
|--------------------------------|--------|--------|--------|------|
| | LV | RV | LV | RV |
| EDV (end diastolic volume) | 77-195 | 88-227 | 52-141 | |
| ESV (end systolic volume) | 19-72 | 23-103 | 12-51 | |
| SV (systolic volume) | 51-133 | 52-138 | 33-97 | |
| EF (%) (ejection fraction) | 56-78 | 47-74 | 56-87 | |
| Mass index (g/m ²) | < 113 | < 36 | < 95 | < 33 |

Table 1. Parameters of ventricular function in the population.

3.4.3. Study valves

CINE: jet and direction. Stenosis: through chamber and regurgitation through proximal chamber.

VELOCITY MAPPING: Imaging plane perpendicular to the vessel.

- Quantification volume in both ventricles in SA. N 1:1
- Quantitative velocity mapping: Flow measurement, LV and RV outputs quantification and comparison with ascending aorta and pulmonary trucks.
- Ventricular function EF ejection fraction.

3.4.4. Study ischemia

- T2 STIR: Hyperintensity in edema. It could appear in acute infarct or myocarditis.

- PERFUSION:

- FIRST PASS or EGE (early Ga enhancement): ischemic areas no enhancement. Stress perfusion with adenosine or dobutamine: Induced ischemia (revascularizable)
- VIABILITY or DHE (delayed enhancement) or LGE (late Ga enhancements) after 10-20 min Ga in interstitial space. This is present in an acute infarct because of edema and rup-

ture of the membrane, and in chronic infarcts due to scarring of the tissue. Necrosis (“The White is dead”) is typically ischemic, and occurs within the endocardium or can be transmural, and extend across the entire wall.

- INVERSION RECOVERY TECHNIQUE AND CONTRAST ENHANCEMENT MR (CE-MR). This uses a pre-pulse to create high T1 tissue contrast to visualize infarct imaging (black myocardium) and chelates of gadolinium (Gd-DTPA) to detect damage in PERFUSION in first pass and delayed at 10 mins DHE to detect scarring or inflammatory changes (white myocardium in infarct, myocarditis, infiltrative diseases...). Also be can be used with Stress MR using adenosine or dobutamine.

| CMR imaging technique | Morphological correlates | Clinical application |
|---|--------------------------------|--|
| <ul style="list-style-type: none"> ▪ Cine imaging ▪ Rest/stress (LDD) | contractile function | <ul style="list-style-type: none"> ▪ LV function ▪ ischemia / viability |
| T2/weighted | myocardial oedema | <ul style="list-style-type: none"> ▪ tissue at risk ▪ inflammation |
| First pass perfusion rest/stress | regional myocardial blood flow | <ul style="list-style-type: none"> ▪ myocardial ischemia ▪ microvascular obstruction |
| Early Gadolinium enhancement | microvascular integrity | <ul style="list-style-type: none"> ▪ No reflow ▪ MVO |
| Late Gadolinium Enhancement | Myocardial necrosis / fibrosis | <ul style="list-style-type: none"> ▪ Infarct size ▪ Viability |

Table 2. Evaluation of CAD (Coronary artery disease) with cardiac MRI

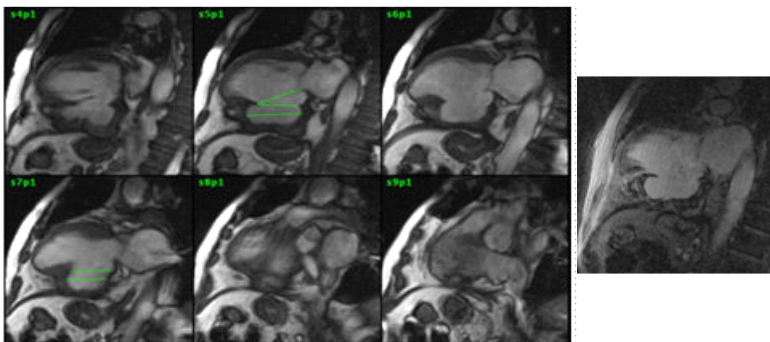


Figure 11. SSFP Cine: enormous aneurism with thrombus in the wall. Huge dilatated LV 163ml/m². Systolic dysfunction FE 20%. On the right: GDHE apical and inferior aneurism (no enhancement) with apical thrombus. Transmural inferior myocardium.enhancement suggest microvascular obstruction

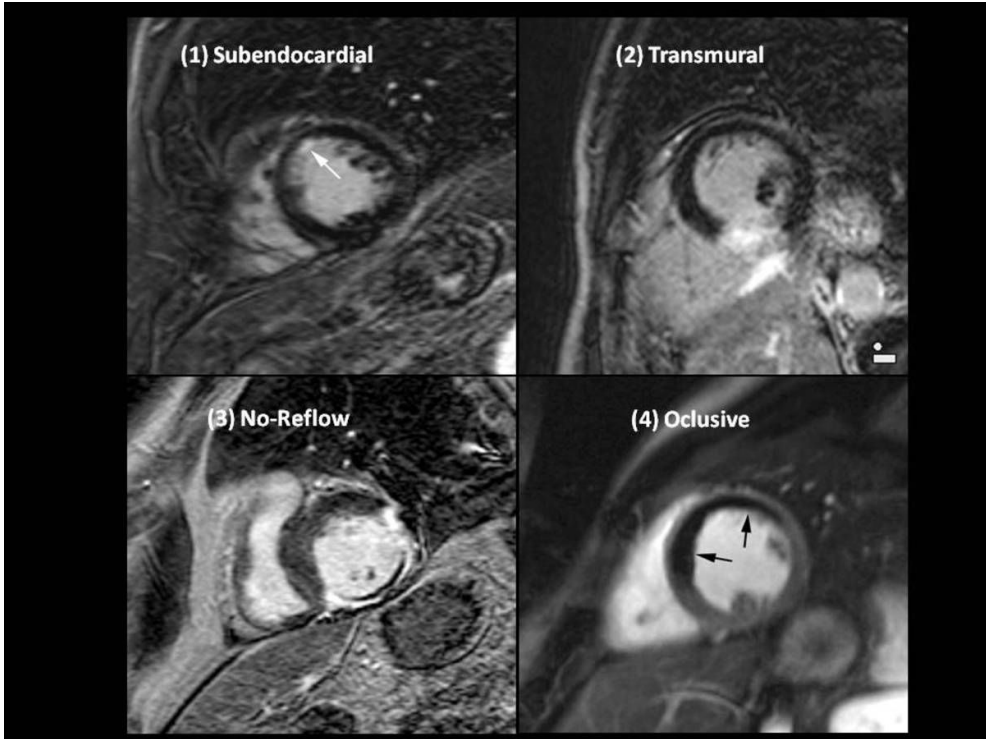


Figure 12. Types of DHE in ischemic cardiopathy: (1) Subendocardial infarction: enhancement only in endocardium and thinness of wall; (2) Transmural infarction: enhancement all the thickness of the inferior wall; (3) No-reflow; (4) Occlusive infarction.

“The no reflow phenomenon”: Early DHE 5-7mins could be transient: This pattern represents a transmural infarction, in which the reperfusion was only partially successful, with a residual lack of reperfusion at the tissue level. This can show evidence of important edema or necrosis with microvascular damage, and predicts against a functional recovery.

Non-reperused occlusive infarcts: DHE Peripheral enhancement surrounding a dark core of non-perfused myocardium.

4. The top 10 cases

4.1. Ischemic cardiopathy

In patients with known or suspected myocardial infarction (MI), cardiovascular magnetic resonance (CMR) provides a comprehensive, multifaceted view of the heart.

Recent multicenter clinical trial indicates that delayed-enhancement cardiac magnetic resonance imaging (DE-CMR) is a well-validated, robust technique that can be easily implemented on scanners that are commonly available worldwide with an effectiveness that clearly rivals the best available imaging techniques for the detection and assessment of acute and chronic MI. When patients present with symptoms outside the usual diagnostic window of cardiac troponins, DE-CMR may be especially useful. Moreover, because DE-CMR can uniquely differentiate between ischemic and various nonischemic forms of myocardial injury, it may be helpful in cases of diagnostic uncertainty, such as in patients with classical features of MI, in whom coronary angiography does not show a culprit lesion. Even once a diagnosis of MI has been made, CMR can also provide clinically relevant information such as identifying residual viability, microvascular damage, stunning, and right ventricular infarction. In addition, post-MI complications, including left ventricular thrombus and pericarditis, are easily identified. Given that quantification of infarct size by DE-CMR is highly reproducible, this technique may provide a useful surrogate end point for clinical trials with appreciable reductions in sample size compared with alternative methods. [5]

Study of ischemia sequences

- *T2 weighted STIR*: edema-weighted imaging. Hyper intensity could appear in acute infarct or myocarditis.
- CINE b-SSFP: motility: Hypokinesia dyskinesia of ischemic areas
- Perfusion: first pass of EGE (early Ga enhancement): ischemic areas no enhancement. Stress perfusion with adenosine and dobutamine: Induced ischemia.
- Viability DHE (delayed enhancement) or LGE (late Ga enhancement) after 10-20 mins. Gadolinium in interstitial space in acute infarct because of edema and rupture of membrane and in chronic infarct due to scarring, showing the extension of the scar.

Scarring less than 25% of the thickness of the wall (subendocardial), 25-50%, greater than 50% (transmural). This usually associates with thinning of the wall and is a prognosis factor that predicts functional recovery after revascularization.

Patterns of ischemic enhancement

The distribution of DHE from ischemia is typically subendocardial to transmural. Differential diagnosis with myocarditis: diffuse multifocal enhancement, often epicardial The morphology of an area of DHE is also relative to the longitudinal distribution of the blood supply of the coronary artery.

- Inferior infarctions: area of enhancement basal and mid-ventricular myocardium. May extend towards the infero-lateral RV right ventricular wall
- Lateral wall infarctions: mid ventricular and apical portions
- Anterior infarctions: antero-basal wall and may include the apex

| Imaging Technique cardio RM | Morphologic correlation | Clinical applications | Normal | Acute IAM | Chronic IAM | Induced Ischemia |
|--|----------------------------------|--|--------------|--|--|-----------------------------------|
| Cine Rest/stress (LDD) | Contractile Function | LV Function Ischemia/ viability | Normal | Normal/ decrease contractile function | Normal/ decrease contractile function | Normal decrease with stress |
| Weighted T2 | Myocardial edema | Tissue in risk Inflammation (swelling) | Black circle | White circle | Black circle | Black circle |
| Perfusion first pass: Rest and stress | Regional myocardial flow | Myocardial ischemia Microvascular obstruction | White circle | N. I. | White circle | White circle |
| Early enhancement EHG | Normal microvascular | No reflow. Microvascular obstruction | White circle | Black circle | White circle | White circle |
| Late enhancement DHE | Myocardial necrosis/ fibrosis | Infarct size Viability | Black circle | White circle | White circle | Black circle |

Table 3. Ischemic cardiomyopathy differential diagnosis between normal, acute or chronic infarct and induced ischemia (black circle: hypointensive; White circle: hyperintensive; N. I.: not indicated)

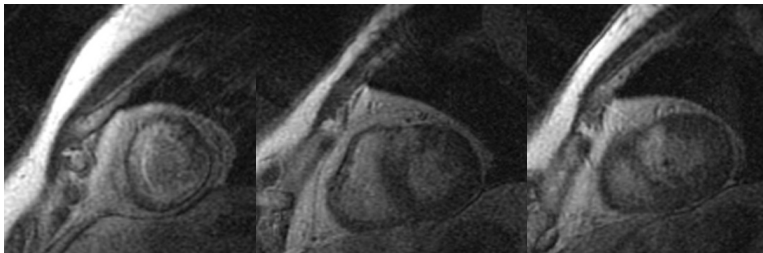


Figure 13. Ischemic cardiomyopathy. DHE. Enhancement inferior infarction: area of enhancement basal and mid-ventricular myocardium.

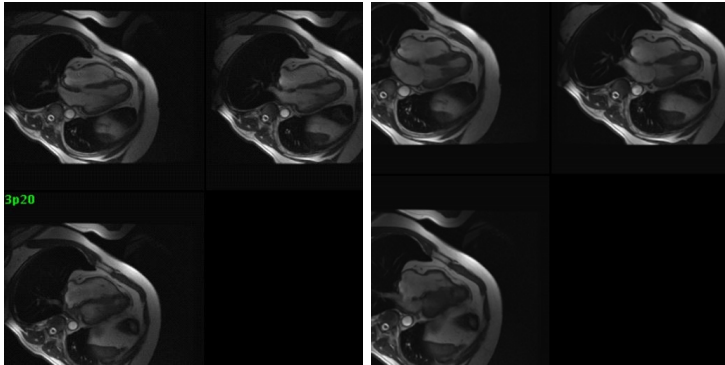


Figure 14. Cine b-SSFP Diastole and systole: apical aneurism (diskinetic area)

4.2. Dilated cardiopathy

The main differential diagnosis is between ischemic and non-ischemic dilated cardiopathies.

Non-ischemic: idiopathic, amyloidosis, haemochromatosis end stage, metabolic toxic alcohol

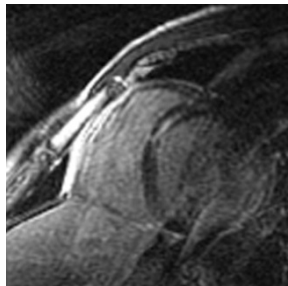


Figure 15. DHE: Chagas Disease. American trypanosomiasis. Severe LV dysfunction, heart failure and tachycardia MR Ga: high signal of inflammation in infero-lateral wall of the LV.

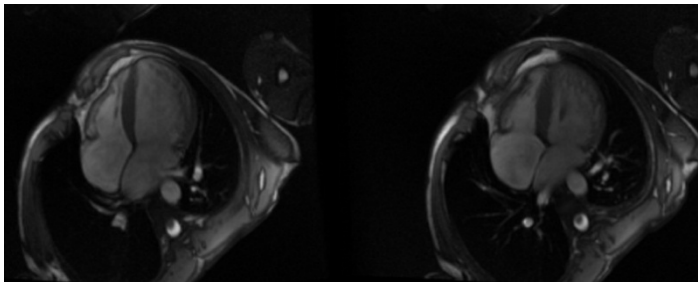


Figure 16. SSFP systole: dilated four chamber hypokinesia

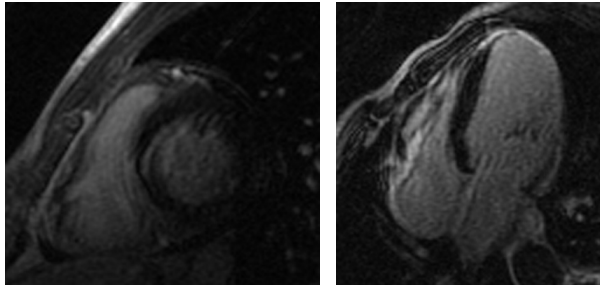


Figure 17. Ischemic dilated cardiomyopathy: 2CH SA and 4CH Dilated chambers and DHE inf basal because of an infarct.

4.3. Hypertrophic MC

Criteria:

Morphological: measure diastolic myocardial thickness LV > 15mm, RV > 5mm in absence of Hypertension, aortic stenosis, amyloidosis or pulmonary hypertension. Measure left chambers.

MR is used when a cardio echogram cannot determinate the severity and risk.

Functional: mitral valvulopathy, obstruction left ventricle outflow and motility dysfunction.

DHE: detect fibrosis and perfusion disorders as a prognosis factor. Non-enhanced is good prognosis.

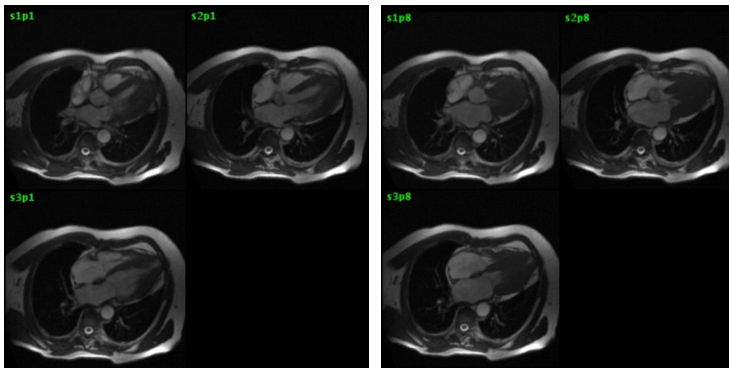


Figure 18. Cine b-SSFP Diastole and systole: Hypertrophic myocardopathy. Measure diastolic myocardial thickness LV > 15mm

4.4. RVAD (Right ventricle arrhythmogenic dysplasia)

Etiology: inherence diseases (1:5000). A. D. Transmission variability. The diagnosis criteria proposed by RVAD Task Force in 1994 are based on the presence of major and minor criteria

encompassing structural, histologic, electrocardiographic, arrhythmic and genetic factors: 2 major criteria, 1 major plus 2 minor, or 4 minor. MR allows multiplanar evaluation of the right ventricle (RV), enabling accurate morphologic and functional assessment.

- RV and outflow dilated
- Aneurisms
- Global systolic dysfunction
- Regional dysfunction contraction
- Fibro fatty transformation

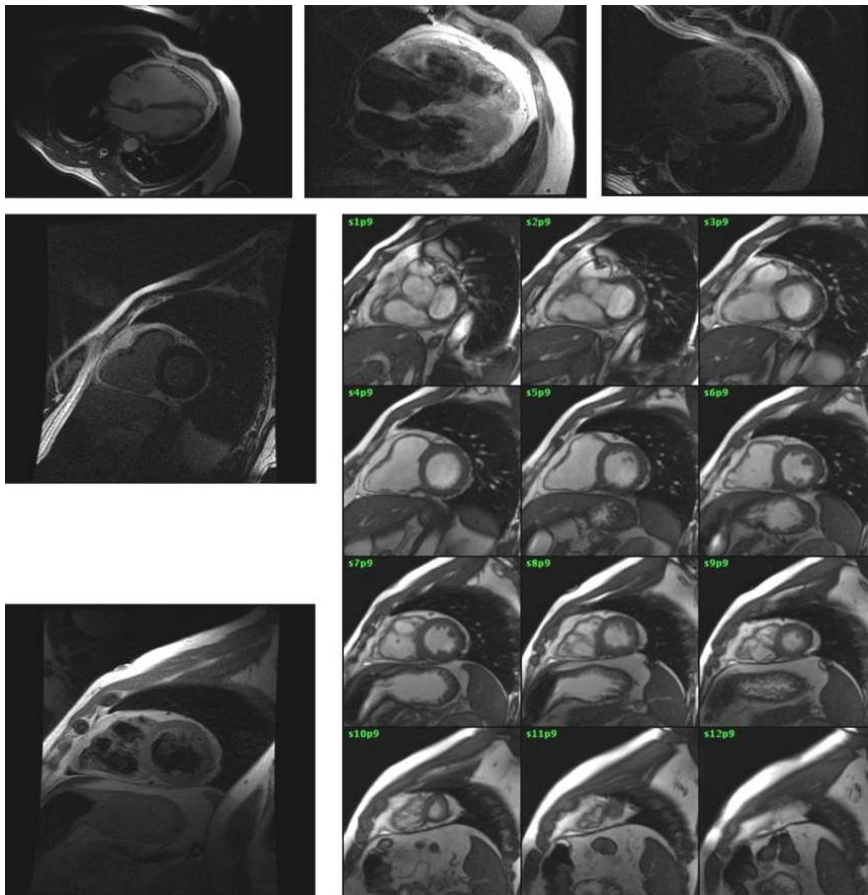


Figure 19. b-SSFP sequences fat suppression, cine and DHE of a ADRV. Criteria: RV 129 ml/m² global dysfunction of RV (FE RV 29%), dyskinesia, saccular granulations, fibro fatty transformation and late enhancement of gadolinium (DHE).

4.5. Restricted cardiopathy

Restrictive cardiomyopathies constitute a heterogeneous group of heart muscle conditions that all present with the symptoms of heart failure, showing diastolic dysfunction with preserved systolic function.

The majority of restrictive cardiomyopathies are secondary to a systemic disorder such as amyloidosis, sarcoidosis, scleroderma, haemochromatosis, eosinophilic heart disease, or as a result of radiation treatment. The more rare diagnosis of idiopathic restrictive cardiomyopathy is supported only by the absence of specific pathology or endomyocardial biopsies.

The classic anatomical features of a restrictive cardiomyopathy are: small left ventricle (not dilated) with marked atrial dilatation and normal systolic function in the absence of pericardial disease.

MRI can demonstrate the underlying anatomical lesion:

- Pericardial thickening, though the presence of a pericardium or normal thickening does not entirely exclude the possibility of constriction. The main differential diagnosis is with constrictive pericarditis.
- Additional imaging features such as abnormal right ventricular shape, vena cava dilatation, and paradoxical movement of the intraventricular septum, during operator guided deep respiration.
- Characteristic tissues, especially the demonstration of interstitial or nodular fibrosis based on the underlying etiology. In the presence of constrictive pericarditis from pericardial inflammation, fibrosis or calcifications, diastolic expansion is impaired resulting in poor diastolic ventricular filling, resulting in a characteristic type of diastolic impairment.
- Amyloidosis: Homogenous increased thickness of ventricular and atrial walls. The ventricular cavities are normal or reduced in size. Severe concentric hypertrophy of both normal sized ventricles in absence of hypertension or valvular heart disease is suggestive of amyloidosis. The atria are usually enlarged owing to the diastolic dysfunction and /or valvular dysfunction due to amyloid deposition. Atrial septum usually > 6mm. Pleural and epicardial effusions are frequent. Inhomogeneous enhancement, patchy, subepicardial or subendocardial, and irrespective of coronary territories.

The differential diagnosis from constrictive pericarditis may be necessary.

MR imaging can serve as a noninvasive examination for the definitive diagnosis of constrictive pericarditis and can help distinguish between constrictive pericarditis and restrictive cardiomyopathy on the basis of pericardial thickness. Mean thickness 1mm.

- The most frequent site of pericardial thickening is over the right ventricle. In Constrictive pericarditis, the signal intensity of the thickened pericardium is similar or decreased compared with that of the myocardium.

- Indirect findings of impaired right ventricular diastolic filling (e.g., dilatation of the inferior vena cava and right atrium) identified in constrictive pericarditis and restrictive cardiomyopathy.

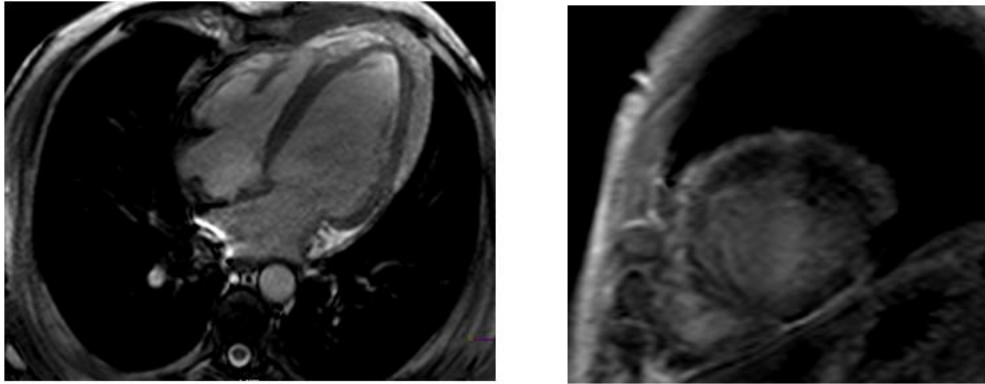


Figure 20. b-SSFP and DHE in Amyloidosis: patchy, subepicardial or subendocardial enhancement irrespective of coronary territories.

Miocardopathies

Etiology: Primary or secondary to systemic diseases. [6]

Cardiac MR is very useful in myocardopathies (MC):

- Genetics: hypertrophic MC (MCH), RVDA arrhythmogenic dysplasia of the RV, non-compacted MC.
- Mixed: dilated MC (MCD), restrictive MC.
- Acquired: inflammatory MC (myocarditis), stress MC (apical ballooning: Tako-Tsubo): transient apical dilatation and dysfunction.

4.6. Pericarditis and myopericarditis

Pericardial inflammation can be caused by infectious diseases viral/bacterial/tuberculosis/fungal); can be manifestation of various systemic diseases (e.g. Rheumatoid arthritis, lupus scleroderma, in patients with uremia, following an acute myocardial infarction, as a result of radiation exposure, or idiopathic.

- Acute may result in diastolic heart failure
- Chronic: fibrosing pericardium ending in stiffening of the pericardium constricting the heart.

Constrictive pericarditis is a thickening (greater than 4 mm), fibrotic and/or calcified pericardium constricting heart.

Normal pericardial thickness is 2mm or less.

Myopericarditis is frequently associated with pericarditis. With a third of cases developing into dilated cardiomyopathy. Global dysfunction of myocardium with non coronary distribution.

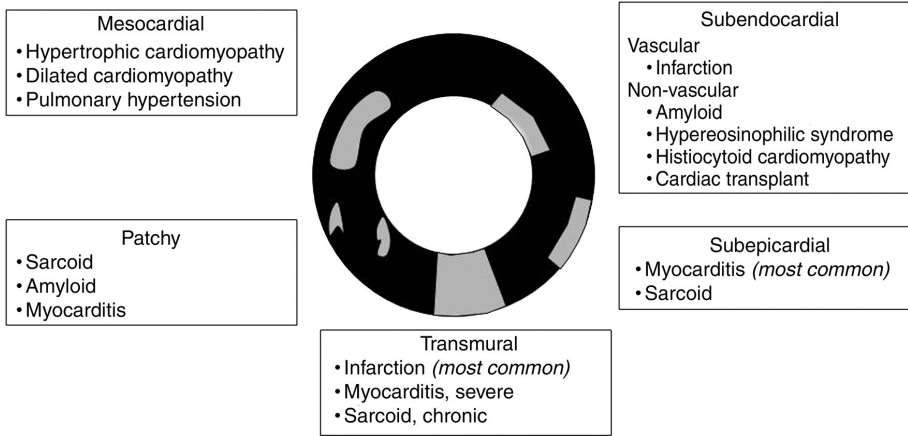


Figure 21. A pattern-based approach to assessment of delayed enhancement in nonischemic cardiomyopathy using MR imaging. [7]

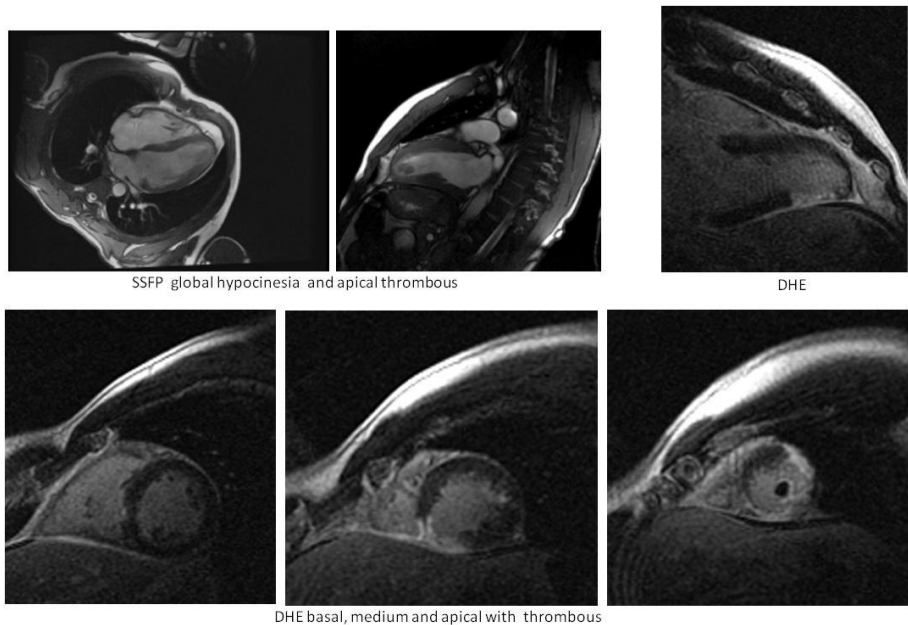


Figure 22. Myocarditis: SSFP apical hypokinesia and thrombus. DHE basal, medium and apical

4.7. Valvular diseases

- Cardiac MR provides good functional information about both valvular stenosis and regurgitation, and allows accurate assessment of ventricular function and relevant cardiac and vascular anatomy. Cardiovascular MR is the gold standard for non invasive imaging of regurgitation: can image the regurgitant volume in any plane, and thus 3D appreciation of the jets can be acquired. Furthermore, it can quantify the regurgitant volume and regurgitant fraction, as well as ventricular function. Transthoracic echocardiography remains the most important and accessible, and easily performed, quantification of valvular heart disease, measuring valvular stenosis and valve area. However this technique is less accurate in quantifying valvular regurgitation with a semi quantitative assessment and only provides an estimate of ventricular function. In addition, imaging planes may be restricted with this technique. X-ray-Angiography (AGF) has been regarded the gold standard but the assessment of valvular regurgitation is both imprecise, inaccurate, and invasive.
- Qualitative assessment of signal loss on cine MR images
- GE technique: For regurgitant lesions signal loss can be grades in a similar way to X-ray AGF: grade 1 = signal loss close to the valve; grade 2 = signal loss extending into the proximal chamber; grade 3 = signal loss filling the whole of the proximal chamber; grade 4 = signal loss in the receiving chamber throughout the relevant half of the cardiac cycle.

Quantitative assessment by measurement of ventricular volumes. MR is the gold standard technique. Using a set of SA cuts covering the length of the ventricles, in combination with Simpson's rule, the stroke volumes of both right and left ventricle can be measured. There is a 1:1 relationship between these stroke volumes; any discrepancy between the ventricular volumes in a patient with regurgitation will identify the regurgitant volume.

- Quantitative assessment by phase-contrast velocity mapping can be applied in any direction for flow quantification of RV and LV outputs can be compared from the proximal ascending aorta and pulmonary trunk. The severity of the regurgitation fraction: 15-20% mild, 20-40% moderate and >40% severe regurgitation.
- Valvular stenosis can be identified by signal loss in cine-MR. Velocity mapping is used to establish an accurate peak velocity across the valve and to quantify the severity of the stenosis. The mean velocity across caval veins and the mitral valve can be used to describe the inflow curves for the A-V valves.

4.8. Cardiac masses

Cardiac mass may be a neoplasm or non-neoplastic swelling, such as a thrombus

Cardiac tumors may be secondary with direct extension, venous, with lymphatic extension, malignant primary tumors, or benign, such as mixoma. The most frequent mass found in clinical practice is due to a thrombus, while the second most frequent is due to mixoma.

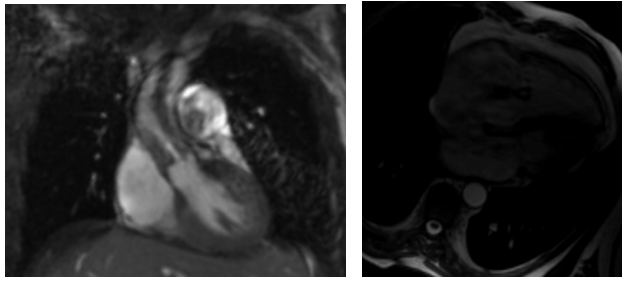


Figure 23. Aortic stenosis and tricuspid regurgitation.

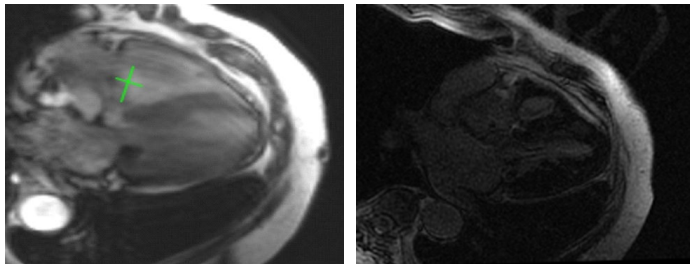


Figure 24. Mixoma: b-SSFP pedunculated mass in LA with heterogeneous DHEg

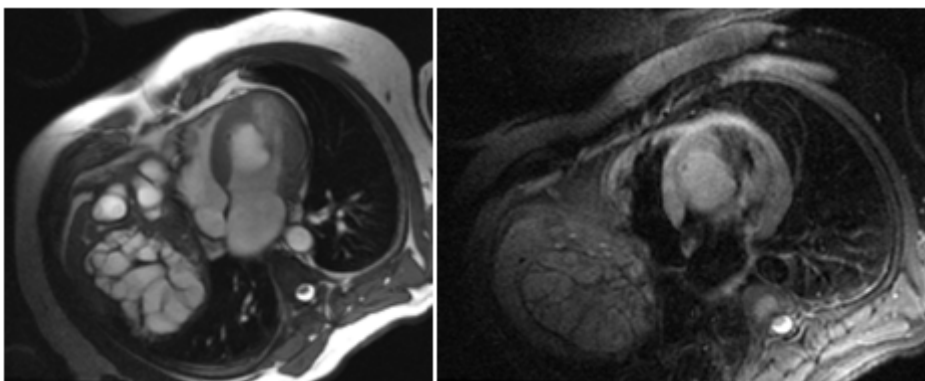


Figure 25. b-SSFP and DHE hidatidic cyst in myocardium and liver.

| LOCATION | TUMOR | MR FINDINGS | CHARACTERISTICS |
|---------------------|--|--|---|
| INTRACAVITARY | Myxoma | Oval, mobile, LA (left atrium) heterogeneous enhancement | Fosa ovalis |
| | Thrombus | LA, orejuela, ventricles in IAM | Low signal in DHE |
| | Valvular vegetations | Irregular masses valvular or perivalvular | Signal similar to thrombus. |
| | Metastases | Transvenous spread | Continued spread Primary tumor |
| INTRAMURAL CHILDREN | Rabdomioma | Several masses similar signal to muscles | Children with E.T |
| | Fibroma | Solitary mass disturbing anatomy. Ventricles. Intramural | Low signal T2 |
| INTRAMURAL ADULTS | Metastases | Pericardial effusion mass with enhancement | Melanoma high signal T1 |
| | Lipomatous hypertrophy of the interatrial septum | Septum >2cm, high signal in T1 | Loose signal in fat suppression sequences |
| | Lipoma | Epicardial or intramural high signal in T1 | Loose signal in fat suppression sequences |
| | Paraganglioma | Well defined in atrial walls or septum | Light-bulb in T2 |
| Epi or PERICARDIAL | Metastases | Pericardial effusion | Direct spread of tumour |
| | Pericardial cyst | Well defined cyst no enhancement | Signal liquid in all sequences |
| | Hemangioma | Multiple cysts with enhancement | High signal in T1 y T2 |

Table 4. Differential diagnosis of cardiac masses by location [8]

4.9. Aortic diseases and great vessels

- MR is the non invasive method of choice for the assessing the great vessels of the thorax.
- MR angiography provides exquisite 3D imaging.
- Assessment of ventricular function and intracardiac anatomy may be important.
- Aortic aneurism: sinus of valsalva > 3.3cm; ascending aorta: mid > 3cm and distal > 2.4cm.

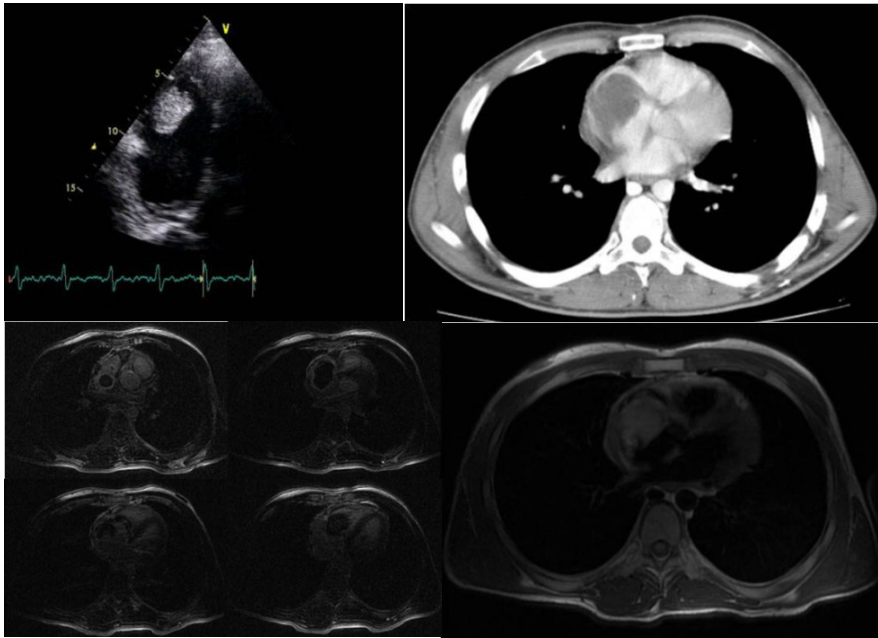
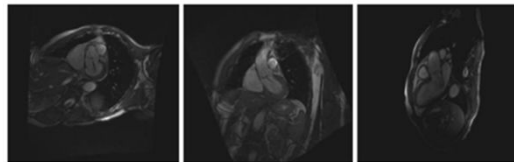


Figure 26. Metastases of germinal tumor: eco and TC pediculated mass in RA. MR: SE and DHE with enhancement of a large mass.



b-SSFP jet of aortic regurgitation in 2CH, coronal and outflow

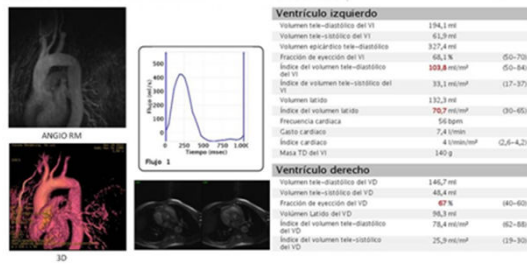


Figure 27. b-SSFP, cine SSFP, RM angiography and QFlow: Dilated ascending aortic with moderate aortic regurgitation. EF and % regurgitation

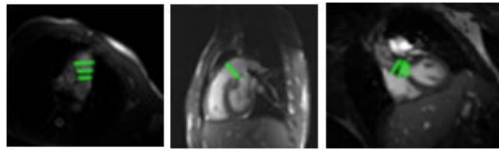


Figure 28. Aortic aneurysm: sinus of valsalva > 3.3cm, mid ascending aorta > 3cm and distal ascending aorta > 2.4cm

- Aortic coarctation: area of narrowing of the thoracic aorta in the region of insertion of the arterial duct with or without additional abnormalities.

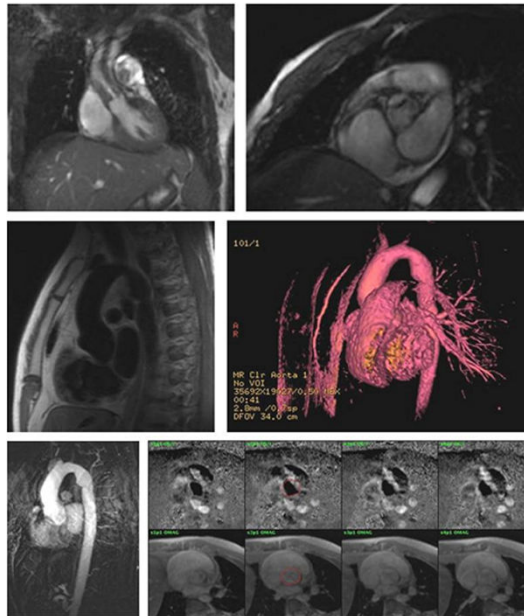


Figure 29. SE blackblood coarctation. Volume-rendered 3D. AGF-MR: collateral vessel. Magnitude and velocity map: Bicuspid aortic valve (jet of aortic stenosis) and aortic coarctation after surgery

- Others: Aortic arch anomalies, interrupted aortic arch, vascular rings, aortic dissection, ulceration and intramural hematoma, Marfan.
- Anomalous pulmonary veins (PV): anomalous pulmonary venous return or systemic venous abnormalities.

4.10. Congenital diseases

Cardiac MR is increasingly becoming an important tool for the diagnosis and follow-up of children and adult patients with congenital heart disease. Its main role is as an adjunct to

echocardiography. MR can provide an accurate, non-invasive method of imaging for assessment of form (3D assessment of anatomy) and function, and is the best method for quantification of ventricular function and vascular flow. The use of (VENC) velocity-encoded-phase-contrast MR allows accurate non invasive quantification of blood flow and pressure gradients, Qp:Qs, and assessment of myocardial perfusion and coronary artery anatomy. Best method for ventricular volumetry (in particular of the RV).

SEQUENTIAL SEGMENTAL ANALYSIS: the nomenclature of complex congenital diseases is based in segmental analysis. Require description of atrial situs, atrio-ventricular connections, and other associated lesions. Increasing ability to assess myocardial perfusion and coronary artery anatomy.

- ASD (Atrial septal defect) are the most common congenital heart defects detected in adults. Cause left-to-right shunting at the atrial level. There are three types: Ostium secundum (80% of ASD), Ostium primum and sinus venous defect.
- AVSD atrio-ventricular septal defect. Partial with a defect of the atrial septum or complete with defect of atrial and ventricular septum there are also abnormalities of the AV valves.

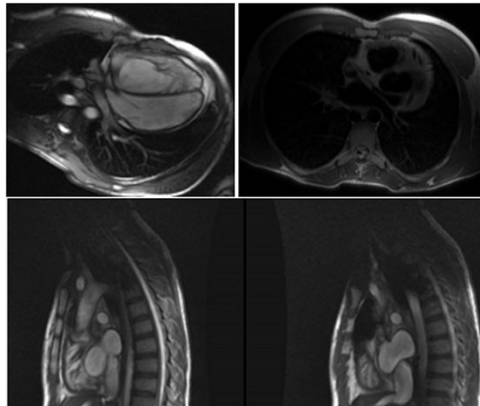


Figure 30. TGV: transposition great vessels after the Mustard procedure. SE black blood LA left atrium connection after surgery. SSFP: RV anatomic (left functionally) dilated 145ml/m², hypertrophic FE 60%. Anatomic LV dilated FE 50%

- TGV is the second-commonest cyanotic congenital heart diseases in the first year of life. It is defined as ventriculoarterial discordance with an anterior aorta arising from the RV, and the pulmonary artery arising from the LV. 40% have a VSD and 30% subpulmonary stenosis
- VSD ventricular septal defect depends of the shunt volume the left sided heart failure and pulmonary vascular disease. Quantification of left-to-right shunts using VENC MR as a non invasive method. Perimembranous lesions make up 80% of VSD. 20% muscular septum.

- Congenital aortic supra-ventricular stenosis: area of narrowing of the thoracic aorta in the region of insertion of the arterial duct. There are three types; hourglass, membranous and diffuse coarctation.

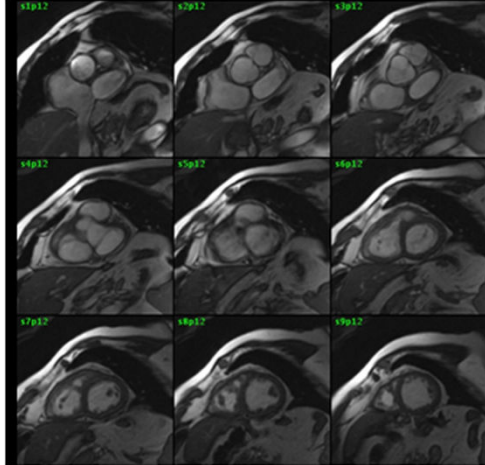


Figure 31. b-SSFP SA outflow. Jet of tricuspid regurgitation Ventricular septal defect and hypertrophy of RV. Tetralogy of Fallot: is the most common cyanotic congenital heart defect, caused by malalignment of the infundibular septum, which leads to right ventricular outflow (RVOT) obstruction, a subaortic VSD (ventricular septal defect) with aortic override, and right ventricular hypertrophy. The role of MR is assessment of postoperative complications besides accurate diagnosis.

Others: pulmonary atresia, double outlet RV, common arterial trunk, anomalous coronary artery, etc.

5. Conclusion

Cardiovascular MR has become a revolutionary technique for the management of cardiac disease. With new sequences the development of this technique has become very important in the diagnosis and management of multiples cardiac diseases and conditions, complementing echocardiography and cardiac angiography.

It has become one of the most accurate techniques (the gold standard) in quantification volumes, mass, study of the right ventricle, and the most complete in the diagnosis of ischemic diseases and congenital diseases. The development of the science in this area has widened the indications and reduced exploration times. Cardiologists and Radiologists must work closely together, and have a widespread knowledge of the most frequent cases and possible differential diagnoses, with a clear understanding of MR sequences and their uses and limitations of this technique.

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