

SOCIETY FOR HEART FAILURE AND TRANSPLANTATION'S

THE RECOVAL Promoting Academics to Improve Clinical Outcomes.

### ISSUE : 11 | December, 2021

Society Reg.No.: EKM/TC/186/2013, Society secretariat: Address 36/117, 2nd Floor, Lisie Hospital Road, Kottecanal Junction, Kochi 682018. Ph.: 025750048, Email: <u>infoshft@gmail.com</u>, <u>www.sfhft.org</u>\_

# EDITOR'S NOTE



### Dr. Manoj Durairaj

Heart Transplant Surgeon, MS, MCh. (AIIMS, New Delhi), FACC.

Director, Marian Cardiac Centre and Research Foundation.

Program Director, Department of Heart and Lung Transplantation, Sahyadri Hospitals, Pune.

#### Dear Colleagues,

Greetings from the Editorial desk. It is my pleasure to present to you this compelling article on Cardiac MRI in Heart Failure by Dr Onkar Auti, Cardiac Imaging Specialist at the Ruby Hall Clinic, Pune. Dr Auti is a Level 3 Certified (EACVI) in CMR. Over the last few years Dr Auti, has been giving us impeccable guidance in the management of our End stage heart failure patients by accurate diagnosis on aetiology, quantification of heart failure and helping us prognosticate our patients precisely. CMR is the gold standard for assessment of ventricular function especially the right ventricle. Multi-planar imaging technique involved in CMR is without any geometric assumptions, hence functional assessment is more accurate especially in abnormally shaped LV in heart failure. CMR myocardial viability results are at par with PET imaging. CMR has shown a promising role in Heart failure with preserved ejection fraction (HFpEF). The diastolic dysfunction is diagnosed by Left atrial dilatation, LV hypertrophy, mitral inflow pattern, LV filling curve. CMR provides accurate estimation of all these parameters. CMR- FT (feature tracking) can calculate Global longitudinal strain and is a promising modality in assessment of HFpEF.

On behalf of the Editorial team, I thank Dr Auti for accepting to be the guest author of this December 2021 issue of "The Revival". I take the opportunity to wish our dear readers a Happy New Year 2022 and a "Happy Reading".

### - Dr Manoj Durairaj Editor "The Revival"

# **SUB EDITOR**



# Dr. Talha Meeran

MBBS, MD, FACC, Consultant Cardiologist, Dept of Advanced Cardiac Sciences and Cardiac Transplant, Sir HN Reliance Foundation Hospital, Mumbai.

### Dear Colleagues,

Cardiac MRI has helped tremendously to improve our understanding, diagnosis and monitoring of numerous cardiac pathologies. In recent years, cardiac MRI has also rapidly expanded its footprint in various cities across India hence making it an accessible technology.

For this edition of the REVIVAL, Dr Auti succinctly elaborates on the utility of cardiac MRI in the heart failure landscape. The excellent case illustrations help to put more meaning into the texts. Given its easy flow and read, the article has equal appeal for all, be it the cardiologist or the cardiac surgeon. This would certainly stimulate even more interest in cardiac MRI and promote further learning on this subject.

Sincerely, Dr Talha Meeran Sub Editor "The Revival"

# PRESIDENTIAL MESSAGE



December issue of ' The Revival ' deals with Cardiac MRI in heart failure. Cardiac MRI plays a major role in the functional assessment of myocardium. At present it is quite an underutilized imaging modality which in fact can provide lot of information with respect to heart failure and also about the viability and stress myocardial perfusion.

Dr Onkar Auti has covered the entire

topic in detail, but at the same time in a simplified manner. I am sure this article will be highly useful for Cardiologists and Cardiac Surgeons alike in the management of patients with cardiac failure ischaemic & non ischaemic.

Best wishes for a Happy and Prosperous New Year free from Covid -19

- Prof. (Dr) V. Nandakumar President

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Special thanks to Dr Onkar Auti

for authoring this month's article.

Designed by Maithili Kulkarni

# **CMR IN HEART FAILURE**



Dr Onkar B Auti DNB, Fellowship in Cardiac imaging, Level 3 certified in CMR (EACVI)

Dr Onkar Auti, is a Consultant Radiologist and Cardiac Imaging Specialist, Ruby Hall clinic, Pune.

- Specialist in Cross Sectional Cardiac imaging (MRI and CT) in adults and children
- Published 10 articles in peer reviewed journals
- · Author of multiple textbook chapters related to cardiac imaging
- Multiple national and few international invited talks
- Involved in regular academic activities at institute level
- Life member of Indian Radiological & Imaging Association (IRIA)
- Executive committee member of Indian Association of Cardiac Imaging (IACI)
- Member of European Society of Cardiology (ESC)

Cardiac Magnetic Resonance (CMR) imaging is a powerful and versatile imaging modality that provides additional information in patients with suspected or known cases of heart failure (HF) [1]. CMR is the gold standard in ventricular function [2]. Moreover it provides excellent information regarding segment wise myocardial wall motion, myocardial morphology and characterisation, stress myocardial perfusion and viability [3-5]. Newer CMR techniques like T1 mapping and T2\* mapping provide additional inputs into the diagnosis and prognosis of various causes of heart failure like amyloidosis [6,7]. Abbasi et al [8] assessed impact of CMR in heart failure patients in term of management and clinical decision making. They found CMR has significant additive impact on management, clinical decision making and diagnosis in 65% patients with heart failure. This impact was observed despite universal use of previous echocardiography in heart failure patients.

### A] Assessment of function:

Accurate quantification of right and left ventricular volumes and function is mainstay in management and prognosis of patients with heart failure. CMR is gold standard in assessment of ventricular function [2]. Gradient echo images are used to assess wall motion and ventricular function. Breath hold, retrospective VCG (vectorcardiogram) gated, cine balanced Steady State Free Precession (bSSFP) images are used for this purpose in multi-planar views with standard views being short axis, 2 chamber (2ch) and 4 chamber (4ch) views. Continuous cine stacks in short axis covering entire ventricles (from base to apex) with no slice gap are preferred for ventricular functional assessment. CMR offers excellent myocardium to blood contrast which enables to observe wall motion in great detail. Multi-planar imaging technique involved in CMR is without any geometrical assumptions, hence functional assessment is more accurate even in abnormally shaped LV which occurs in heart failure [1].

Endocardial and epicardial borders are traced and ventricular functional parameters including end diastolic volume (EDV), end systolic volume (ESV), ejection fraction (EF) and LV myocardial mass are calculated. CMR shows excellent reproducibility in quantification of these functional parameters which is helpful in serial assessment and therapeutic monitoring [9]. With newer techniques, good quality free-breathing cine images are possible even in patients with poor breath hold [10]. In patients with arrhythmia, diagnostic cine images can be obtained using prospective gating, arrhythmia rejection protocols or real time imaging [11].

### B] Role of CMR in ischemic heart disease:

Apart from accurate estimation of LV and RV function which is crucial in management of ischemic heart disease (IHD), CMR plays a key role due to its excellent myocardial characterisation. In CMR, Late gadolinium enhancement (LGE) sequences have higher sensitivity in detection of infarction, extent of infarction and assessment of scar burden [12-14]. Predominant role of CMR in IHD is to assess viability and stress inducible ischemia.

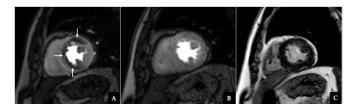
Viability assessment is performed by evaluating regional

wall motion abnormalities and extent of myocardial infarction. Subendocardial layer is the first affected part of myocardium in infarction, where scar formation begins. Depending on severity and duration of impaired blood flow, the scar extends transmurally towards epicardium. Thickness of myocardium infarcted is inversely proportional to degree of functional recovery of myocardium. Hypokinesia with myocardial infarction involving more than 50% of thickness has low probability of functional recovery and subendocardial infarction involving less than 25% thickness has great potential for functional recovery. It is also shown that myocardium with transmural infarction involving >75% thickness is unlikely to improve after revascularisation [15] **(figure 1).** 



CMR myocardial viability results show excellent comparison and is at par with PET (positron emission tomography) findings [16]. Also CMR is proven to have higher sensitivity and specificity in detection of infarcted myocardium compared to SPECT (single photon emission computed tomography) [17].

CMR perfusion imaging in stress and rest environment allows detection of stress inducible ischemia and peri-infarct ischemia. Pharmacological vasodilator stress agents like Adenosine is commonly used to assess stress inducible ischemia. First pass perfusion technique is used with administration of gadolinium based contrast agent. First pass perfusion is assessed after stress induction and also in rest conditions. Presence of perfusion defects on stress imaging with normal rest perfusion (reversible defect) indicates stress inducible ischemia *(figure 2).* 



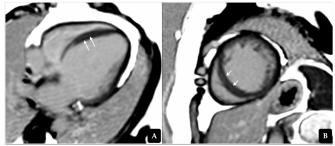
Infarcted tissue shows perfusion defect in rest as well as stress imaging, referred as fixed defect. Ischemia burden more than 10-15% is considered as a significant coronary artery disease and revascularisation will benefit the patient [18,19]. Stress perfusion imaging are further correlated with LGE images to assess viability as well as peri-infarct ischemia. Detection of myocardial ischemia on stress CMR is similar or is at least not inferior to other non-invasive modalities like positron emission tomography (PET) or single photon emission computed tomography (SPECT) [20-22]. Moreover, single stress CMR study along with LGE images provides comprehensive assessment of myocardium evaluating viability as well as stress inducible ischemia and/or periinfarct ischemia.

CMR is also an excellent modality for detecting LV thrombus and reperfusion injuries. LV thrombus is common phenomenon in chronic myocardial infarction especially with akinetic or dyskinetic segments. Thrombus is seen as filling defect in the LV cavity on bSSFP cine images with no enhancement on LGE images. Reperfusion injuries include mainly microvascular obstruction (MVO) and intra-myocardial hematoma (IMH). MVOs are areas within infarction where complete lack of perfusion exists even after restoration of blood flow, also known as 'no reflow' phenomenon [23,24]. Early gadolinium enhancement (EGE) at 3-5 minutes with high nulling inversion time and LGE images should acquire where MVO appear dark areas within hyper-enhancing infarction [25]. IMH can be easily identified as hypo intense areas within the infarction on T2\* imaging. T2\* mapping is more sensitive to IMH than other CMR sequences [26]. Presence of MVO or IMH are poor prognostic markers in IHD.

### C] CMR in non-ischemic cardiomyopathy:

Determination of cardiac structure and myocardial involvement is essential in classifying the heart failure and also to determine underlying aetiology like, dilated cardiomyopathy, hypertrophic cardiomyopathy, infiltrative cardiomyopathy, myocarditis etc.

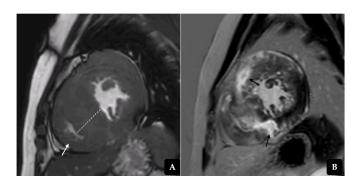
Dilated cardiomyopathy (DCM) is characterised by dilatation of LV with systolic dysfunction in absence of significant coronary artery disease or any other obvious underlying cause [27]. CMR helps to rule out other causes of dilated LV like myocarditis, infarction or infiltration. Primary DCM is diagnosis of exclusion. CMR provides accurate estimation of LV function which is essential in management of DCM. Also it determines RV failure and accurate quantification of RV function. Involvement of RV in DCM is associated with poor prognosis in DCM [28]. Replacement fibrosis in DCM is seen as mid myocardial enhancement on LGE images (*figure 3*).



This mid wall fibrosis is independent prognostic fibrosis

in DCM which is seen in almost 30% patients of DCM [29]. Presence of septal mid wall fibrosis on LGE image is associated with higher risk for sudden cardiac death (SCD) in DCM patients. SCD risk is highest with concomitant septal and free-wall LGE [30]. Newer T1 mapping and ECV calculation methods in CMR offers risk stratification in cases of DCM. ECV is is found to increase in approximately 58% of DCM patients therefore, ECV estimation offers prognostication in heart failure outcomes incremental to LGE and T1 mapping [31].

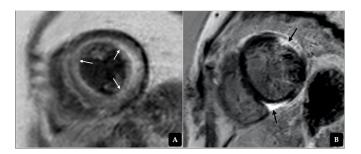
Hypertrophic cardiomyopathy (HCM) is most common genetic cardiomyopathy and is characterised by asymmetrical hypertrophy of LV, myocyte disarray and interstitial plus replacement fibrosis. CMR is excellent modality to assess suspected or diagnosed cases of HCM. CMR can effectively differentiate between different types of HCM like asymmetrical septal HCM, apical HCM and focal HCM [32]. CMR also can effectively assess systolic motion of mitral valve (SAM), LVOT obstruction or mid cavity obstruction. It also allows to detect RV hypertrophy which is present in about 1/3rd of HCM cases [33]. Interstitial and replacement fibrosis observed in HCM very distinct and characterised by patchy mid wall enhancement on LGE images. Percentage of fibrosis can be quantified on LGE sequences as a proportion of fibrosis to the myocardial mass. There is significant association between LGE and cardiovascular all cause mortality in HCM and also a trend towards increased SCD was observed [34]. T1 mapping shows increased native T1 values in about 30% HCM patients with absent LGE [35]. Combined use of LGE, T1 mappings and global ECV calculation can improve selection of candidates for implantable cardioverter defibrillator (ICD) placement [36]. CMR also helps in prognostication in HCM patients where poor prognostic indicators include LV hypertrophy measuring >30 mm in thickness, extensive patchy fibrosis in hypertrophied segments, presence of SAM and severe LVOT obstruction and mid ventricular obstruction with apical aneurysm (figure 4).



CMR also differentiate SHCM from other causes of LV hypertrophy like hypertensive cardiomyopathy, infiltrative cardiomyopathy like amyloidosis and Fabry's disease.

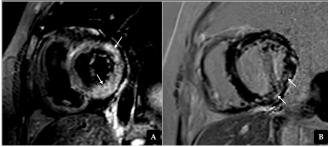
Infiltrative cardiomyopathy is a broad spectrum abnormality consisting amyloidosis, sarcoidosis, Fabry's disease etc.

CMR provides differentiation between types of infiltrative cardiomyopathy due to excellent myocardial characterisation *(figure 5).* 



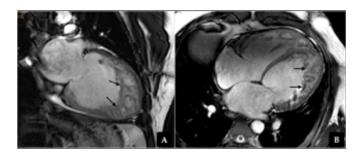
Amyloidosis is characterised by fibril deposition in the myocardium. CMR shows concentric symmetrical hypertrophy of LV with biatrial dilatation and inter-atrial septal thickening. Hypokinesia of LV is noted with pronounced diastolic dysfunction. LGE shows characteristic pattern of concentric subendocardial LGE with dark blood pool giving rise to 'zebra pattern' [37]. CMR also can differentiate between light chain amyloid (AL) and transthyretin related amyloidosis (ATTR), where transmural pattern of LGE is common in ATTR type [38]. CMR also allows to assess involvement of RV in amyloidosis. Noncontrast T1 mapping has high diagnostic accuracy for detecting cardiac AL amyloidosis, which correlates well with markers of systolic and diastolic dysfunction, and is potentially more sensitive for detecting early disease than LGE imaging [39]. Fabry's disease is x linked disorder of sphingolipid metabolism causing LV hypertrophy. CMR shows concentric LV hypertrophy with district and characteristic lateral wall enhancement in LGE images [1]. Sarcoidosis is an idiopathic granulomatous disease which can involve any organ system including heart. CMR features shows patchy of diffuse LGE predominantly in lateral wall [1]. Patients with the presence of LGE are at increased risk of death from any cause and arrhythmogenic events, even if their cardiac function is normal or near normal [40].

Myocarditis is the acute inflammation of the myocardium. Patients with acute heart failure and normal coronaries, myocarditis is suspected. STIR (short tau inversion recovery) sequence on CMR helps to detect acute myocardial edema/ inflammation as bright signals within the myocardium. LGE may or may nor show any enhancement corresponding to acute myocardial edema (*figure 6*).



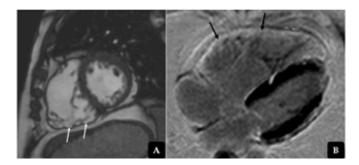
CMR helps in diagnosis and guiding biopsy increasing yield of invasive procedures [41].

LV non-compaction (LVNC) is a specific cardiomyopathy with hypertrabeculation of LV forming a layer of non-competed myocardium at endocardial surface of LV cavity. It has been demonstrated that CMR can reliably detect LV trabeculations more clearly than echocardiography [42]. There are many studies demonstrating different cut off for non-competed to compacted myocardium (NC:C) ratios in end diastole and end systole phases [43]. The criteria for the diagnosis by CMR - NC:C ratio greater than 2.3 during the diastole has a sensitivity of 86% and specificity of 99% [44] *(figure 7).* 



Another method to diagnose this entity is a trabeculated left ventricular mass above 20% of total mass with a sensitivity of 91.6% and a specificity of 86.5% is predictive of LVNC [45].

Arrhythmogenic right ventricular dysplasia (ARVD) is idiopathic cardiomyopathy of RV and is a prominent cause of SCD. It is characterised by RV dilatation and RV dyskinesia. RV is difficult to visualise on echocardiography due to near field signal drop out and crescentic structure if RV. On the other hand CMR can provide three dimensional and multi-planar imaging of RV with accurate quantification of RV volumes and ejection fraction (EF). CMR features of ARVD shows dilatation of LV associated with RV wall dyskinesia and/or aneurysmal outpouchings. LGE shows diffuse or patchy enhancement of RV free wall (*figure 8*).



According to revised task force criteria for diagnosis of ARVD, CMR can fulfil one major or one minor criterion [46]. One major criteria on CMR include RV dyskinesia/akinesia/ dyssynchronous RV contraction with indexed RV EDV > 110 ml/m2 (male) and > 100 ml/m2 (female) or RVEF < 40%. CMR also detects contiguous involvement of LV myocardium. Iron overload cardiomyopathy is manifestation of repetitive blood transfusions in patients with hemoglobinopathies or in primary hemochromatosis. CMR is approved as gold standard to quantify myocardial iron overload. T2\* imaging technique is used in qualification of iron overload which is ECG gated, single breath hold sequence [47]. Accurate quantification of myocardial iron provides excellent inputs to start or modify chelation therapy in patients with hemoglobinopathies [48].

# D] Heart failure with preserved ejection fraction (HFpEF):

HFpEF consists of approximately 50% of cases of heart failure and contains diverse clinical heterogeneity [49]. These cases usually present with diastolic dysfunction. Echocardiography is traditional modality to diagnose and grade diastolic dysfunction in these cases. However, in last few years CMR showed promising role in HEpEF. Diastolic dysfunction is usually diagnosed by left atrial (LA) dilatation, LV hypertrophy, mitral inflow patten, LV filling curve etc. CMR allows accurate estimation of LA volume with the help of 2ch and 4ch cine views. LA volume is estimated using biplane area length (BAL) technique where LA volume (ml) =  $(0.85 \times A2C \times A4C)/L$  (A2C and A4C are the LA areas on the 2ch and 4 ch views, and L is the shorter length of the LA, from either the 2ch or 4ch). It is shown that CMR BAL technique is equally accurate with CMR Simpson's volumetric assessment and echocardiography BAL technique is less accurate as compared to CMR volumetric assessment especially in cases of atrial fibrillation [50]. LA size more than 32 ml/m2 is associated with increased rate of heart failure independent of age, LV hypertrophy, diabetes, hypertension, myocardial infarction of mitral inflow velocities [51]. LV hypertrophy is most common structural abnormality associated with HFpEF. CMR findings are more reproducible than M mode and 2D echocardiography in assessing LV hypertrophy [52]. Phase contrast imaging of CMR is used to calculate transmitral flow pattern with E and A velocities and E/A ratio which can be used in classifying different grade of diastolic dysfunction [53]. Other parameters like flow assessment across pulmonary veins and LV time-volume filling curves on CMR provides further assessment in HFpEF. CMR feature tracking (CMR-FT) is newer technique for evaluation of LV strain. CMR-FT are anatomic elements typically identified along the cavity-myocardial interface due to the high contrast resolution between blood pool and myocardium [54]. Bloodmyocardium interface is traced in end diastolic cine images and CMR-FT software automatically tracks it throughout cardiac cycle. Global longitudinal strain is calculated on long axis cine images while global circumferential strain and global radial strains are calculated on short axis cine images [55,56]. CMR-FT is evolving and promising modality in assessment of HFpEF.

Around 10-15% cases of HFpEF belong to restrictive cardiomyopathy (RCM) or constrictive pericarditis (CP) [57].

RCM and CP share similar clinical presentation and many common features in diagnostic imaging tests however, the management of both conditions is extremely different as CP can be treated by pericardiectomy. CMR is excellent modality to differentiate between RCM and CP due to its multi-planar 3 dimensional imaging capabilities. Endomyocardial fibrosis (EMF) is most common cause of RCM [58]. EMF on CMR is characterised by obliterative apical fibrosis and thrombus. Apical fibrosis is seen as diffuse circumferential enhancement and thrombus is seen as non-enhancing filling defect in the apex on LGE images affecting single or both ventricles [59]. Other causes of RCM like amyloidosis, Fabry disease and iron overload cardiomyopathy are described above in the article. On the other hand, CP is associated with thickened pericardium causing abnormal pericardial compliance which affects relaxation of ventricles. CMR shows diffuse or focal thickening of pericardium with or without post contrast enhancement. Dynamic imaging with CMR can detect the exaggerated respiratory interdependence of the ventricles

and abnormal septal bounce [60]. Treatment includes sodium restriction and diuretic agents to reduce edema and hepatic congestion; however, definitive therapy will require surgical pericardiectomy.

### E] Cardiac resynchronisation therapy (CRT):

Patients with significant LV dysfunction, a challenge related to management involves determining which patients will benefit from CRT. Echocardiographic measurements are not sufficient to identify those individuals that will benefit from CRT. Cine CMR helps to detect areas of global or regional LV dyssynchrony. With use of LGE images of CMR, identification of scarred myocardium provides important information as to whether myocardial segments will improve contractility or develop synchronous contraction when a pacing lead is placed in close juxtaposition. As, if pacing leads are placed in regions with scarred myocardium, it will not facilitate the development of synchronous contraction [61].

## **Conclusions:**

CMR is excellent modality in evaluation of suspected or known cases of heart failure. CMR provides valuable information in ischemic heart disease in terms of viability and detection of stress inducible ischemia. CMR is also unique in differentiating between the various causes of LV dysfunction. CMR information helps in assessing prognosis and also deciding management in various cardiomyopathies. CMR also allows to assess risk stratification in patients undergoing cardiac resynchronisation therapy. Newer imaging techniques like CMR-FT helps to study strain patterns in different cardiac diseases.

### **References :**

1. Assomull R, Pennell D, Prasad S. Cardiovascular magnetic resonance in the evaluation of heart failure. Heart. 2007;93(8):985-992.

2. Pennell DJ. Cardiovascular magnetic resonance: twenty-first century solutions in cardiology. Clin Med 2003;3:273–8.

3. Auti O, Bandekar K, Kamat N, Raj V. Cardiac magnetic resonance techniques: Our experience on wide bore 3 tesla magnetic resonance system. Indian Journal of Radiology and Imaging. 2017;27(4):404.

4. Salerno M, Kramer C. Advances in cardiovascular MRI for diagnostics: applications in coronary artery disease and cardiomyopathies. Expert Opinion on Medical Diagnostics. 2009;3(6):673-687.

5. Franco A, Javidi S, Ruehm S. Delayed Myocardial Enhancement in Cardiac Magnetic Resonance Imaging. Journal of Radiology Case Reports. 2015;9(6).

 Burt J, Zimmerman S, Kamel I, Halushka M, Bluemke D. Myocardial T1 Mapping: Techniques and Potential Applications. RadioGraphics. 2014;34(2):377-395.

7. Kim P, Hong Y, Im D, Suh Y, Park C, Kim J et al. Myocardial T1 and T2 Mapping: Techniques and Clinical Applications. Korean Journal of Radiology. 2017;18(1):113.

8. Abbasi S, Ertel A, Shah R, Dandekar V, Chung J, Bhat G et al. Impact of cardiovascular magnetic resonance on management and clinical decision-making in heart failure patients. Journal of Cardiovascular Magnetic Resonance. 2013;15(1):89.

9. Grothues F, Smith G C, Moon J C.et al Comparison of interstudy reproducibility of cardiovascular magnetic resonance with twodimensional echocardiography in normal subjects and in patients with heart failure or left ventricular hypertrophy. Am J Cardiol 20029029–34.

10. Moghari M, Komarlu R, Annese D, Geva T, Powell A. Freebreathing steady-state free precession cine cardiac magnetic resonance with respiratory navigator gating. Magnetic Resonance in Medicine. 2014;73(4):1555-1561.

11. Wage, R., Hanlon, R.O. Comprehensive cmr imaging in patients with arrhythmia. J Cardiovasc Magn Reson 12, T9 (2010).

12. Achenbach S, Barkhausen J, Beer M, Beerbaum P, Dill T, Eichhorn J, et al. [Consensus recommendations of the German Radiology Society (DRG), the German Cardiac Society (DGK) and the German Society for Pediatric Cardiology (DGPK) on the use of cardiac imaging with computed tomography and magnetic resonance imaging]. Rofo. 2012;184(4):345-68.

13. Krumm P, Zitzelsberger T, Weinmann M, Mangold S, Rath D, Nikolaou K, et al. Cardiac MRI left ventricular global function index and quantitative late gadolinium enhancement in unrecognized myocardial infarction. Eur J Radiol. 2017;92:11-6.

14. Hunold P, Jakob H, Erbel R, Barkhausen J, Heilmaier C. Accuracy of myocardial viability imaging by cardiac MRI and PET depending on left ventricular function. World J Cardiol. 2018;10(9):110-8.

15. Kim R J, Wu E, Rafael A.et al The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. N Engl J Med 20003431445–1453.

16. Klein C, Nekolla S G, Bengel F M.et al Assessment of myocardial viability with contrast-enhanced magnetic resonance imaging: comparison with positron emission tomography. Circulation 2002105162–167.

17. Wagner A, Mahrholdt H, Holly T A.et al Contrast-enhanced MRI and routine single photon emission computed tomography (SPECT) perfusion imaging for detection of subendocardial myocardial infarcts: an imaging study. Lancet 2003361374–379.

18. Shaw LJ, Berman DS, Picard MH, Friedrich MG, Kwong RY, Stone GW, et al. Comparative definitions for moderate-severe ischemia in stress nuclear, echocardiography, and magnetic resonance imaging. JACC Cardiovasc Imaging. 2014;7(6):593-604.

19. Sammut EC, Villa ADM, Di Giovine G, Dancy L, Bosio F, Gibbs T, et al. Prognostic Value of Quantitative Stress Perfusion Cardiac Magnetic Resonance. JACC Cardiovasc Imaging. 2018;11(5):686-94.

20. Greenwood JP, Maredia N, Younger JF, Brown JM, Nixon J, Everett CC, et al. Cardiovascular magnetic resonance and single-photon emission computed tomography for diagnosis of coronary heart disease (CE-MARC): a prospective trial. Lancet. 2012;379(9814):453-60.

21. Schwitter J, Wacker CM, Wilke N, Al-Saadi N, Sauer E, Huettle K, et al. Superior diagnostic performance of perfusion-cardiovascular magnetic resonance versus SPECT to detect coronary artery disease: The secondary endpoints of the multicenter multivendor MR-IMPACT II (Magnetic Resonance Imaging for Myocardial Perfusion Assessment in Coronary Artery Disease Trial). J Cardiovasc Magn Reson. 2012;14:61.

22. Schwitter J, Wacker CM, Wilke N, Al-Saadi N, Sauer E, Huettle K, et al. MR-IMPACT II: Magnetic Resonance Imaging for Myocardial Perfusion Assessment in Coronary artery disease Trial: perfusion-cardiac magnetic resonance vs. single-photon emission computed tomography for the detection of coronary artery disease: a comparative multicentre, multivendor trial. Eur Heart J. 2013;34(10):775-81.

23. Yellon DM, Hausenloy DJ. Myocardial reperfusion injury. N Engl J Med. 2007;357(11):1121-35.

24. Niccoli G, Burzotta F, Galiuto L, Crea F. Myocardial no-reflow in humans. J Am Coll Cardiol. 2009;54(4):281-92.

25. Bogaert J, Kalantzi M, Rademakers FE, Dymarkowski S, Janssens S. Determinants and impact of microvascular obstruction in successfully reperfused ST-segment elevation myocardial infarction. Assessment by magnetic resonance imaging. Eur Radiol. 2007;17(10):2572-80.

26. Bulluck H, Rosmini S, Abdel-Gadir A, Bhuva A, Treibel T, Fontana M et al. Diagnostic performance ofT1andT2mapping to detect intramyocardial hemorrhage in reperfused ST-segment elevation myocardial infarction (STEMI) patients. Journal of Magnetic Resonance Imaging. 2017;46(3):877-886.

27. Japp AG, Gulati A, Cook SA, Cowie MR, Prasad SK. The diagnosis and evaluation of dilated cardiomyopathy. J Am Coll Cardiol 2016;67: 2996–3010.

28. Gulati A, Ismail T, Jabbour A, Alpendurada F, Guha K, Ismail N et al. The Prevalence and Prognostic Significance of Right Ventricular Systolic Dysfunction in Nonischemic Dilated Cardiomyopathy. Circulation. 2013;128(15):1623-1633.

29. J.A. McCrohon, J.C. Moon, S.K. Prasad, et al. Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance. Circulation, 108 (2003), pp. 54-59.

30. Halliday B, Baksi A, Gulati A, Ali A, Newsome S, Izgi C et al. Outcome in Dilated Cardiomyopathy Related to the Extent, Location, and Pattern of Late Gadolinium Enhancement. JACC: Cardiovascular Imaging. 2019;12(8):1645-1655.

31. Vita T, Gräni C, Abbasi S, Neilan T, Rowin E, Kaneko K et al. Comparing CMR Mapping Methods and Myocardial Patterns Toward Heart Failure Outcomes in Nonischemic Dilated Cardiomyopathy. JACC: Cardiovascular Imaging. 2019;12(8):1659-1669.

32. Brenes J, Doltra A, Prat S. Cardiac magnetic resonance imaging in the evaluation of patients with hypertrophic cardiomyopathy. Global Cardiology Science and Practice. 2018;2018(3).

33. Keeling AN, Carr JC, Choudhury L. Right ventricular hypertrophy and scarring in mutation positive hypertrophic cardiomyopathy. Eur Heart J. 2010 Feb; 31(3):381.

34. Green JJ, Berger JS, Kramer CM, Salerno M. Prognostic value of late gadolinium enhancement in clinical outcomes for hypertrophic cardiomyopathy. JACC Cardiovasc Imaging. 2012 Apr; 5(4):370-7.

35. Swoboda et al. (2017) Swoboda PP, McDiarmid AK, Erhayiem B, et al. Effect of cellular and extracellular pathology assessed by T1 mapping on regional contractile function in hypertrophic cardiomyopathy. J. Cardiovasc. Magn. Reson. 2017;19:16.

36. Avanesov et al. (2017) Avanesov M, Münch J, Weinrich J, et al. Prediction of the estimated 5-year risk of sudden cardiac death and syncope or non-sustained ventricular tachycardia in patients with hypertrophic cardiomyopathy using late gadolinium enhancement and extracellular volume CMR. Eur. Radiol. 2017;27:5136–5145.

37. Smedema J P, Snoep G, van Kroonenburgh M P.et al The additional value of gadolinium-enhanced MRI to standard assessment for cardiac involvement in patients with pulmonary sarcoidosis. Chest 20051281629–1637.

 Dungu J, Valencia O, Pinney J, Gibbs S, Rowczenio D, Gilbertson J et al. CMR-Based Differentiation of AL and ATTR Cardiac Amyloidosis. JACC: Cardiovascular Imaging. 2014;7(2):133-142.

39. Karamitsos T, Piechnik S, Banypersad S, Fontana M, Ntusi N, Ferreira V et al. Noncontrast T1 Mapping for the Diagnosis of Cardiac Amyloidosis. JACC: Cardiovascular Imaging. 2013;6(4):488-497.

40. Coleman G, Shaw P, Balfour P, Gonzalez J, Kramer C, Patel A et al. Prognostic Value of Myocardial Scarring on CMR in Patients With Cardiac Sarcoidosis. JACC: Cardiovascular Imaging. 2017;10(4):411-420.

41. Mahrholdt H, Goedecke C, Wagner A.et al Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. Circulation 20041091250–1258.

42. Alhabshan F, Smallhorn JF, Golding F, Musewe N, Freedom RM, Yoo SJ. Extent of myocardial non-compaction: comparison between MRI and echocardiographic evaluation. Pediatr Radiol. 2005 Nov; 35(11):1147-51.

 Stacey RB, Andersen MM, St Clair M, Hundley WG, Thohan
Comparison of systolic and diastolic criteria for isolated LV noncompaction in CMR. JACC Cardiovasc Imaging. 2013 Sep; 6(9):931-40.

44. Petersen SE, Selvanayagam JB, Wiesmann F et al., "Left ventricular non-compaction: insights from cardiovascular magnetic resonance imaging," Journal of the American College of Cardiology, vol. 46, no. 1, pp. 101–105, 2005.

45. Jacquier A, Thuny F, Jop B et al., "Measurement of trabeculated left ventricular mass using cardiac magnetic resonance imaging in the diagnosis of left ventricular non-compaction," European Heart Journal, vol. 31, no. 9, pp. 1098–1104, 2010.

46. Marcus F, McKenna W, Sherrill D, Basso C, Bauce B, Bluemke D et al. Diagnosis of Arrhythmogenic Right Ventricular Cardiomyopathy/ Dysplasia. Circulation. 2010;121(13):1533-1541.

47. Hankins JS, McCarville MB, Loeffler RB, Smeltzer MP, Onciu M, Hoffer FA, et al. R2\* magnetic resonance imaging of the liver in patients with iron overload. Blood 2009;113:4853–5.

48. Tanner M A, Galanello R, Dessi C.et al A randomized, placebocontrolled, double-blind trial of the effect of combined therapy with deferoxamine and deferiprone on myocardial iron in thalassemia major using cardiovascular magnetic resonance. Circulation 20071151876–1884.

49. Komajda M, Lam CS. Heart failure with preserved ejection fraction: a clinical dilemma. Eur Heart J. 2014;35(16):1022–32.

50. Vassiliou VS, Patel HC, Rosen SD, Auger D, Hayward C, Alpendurada F, Lyon AR, Pennell DJ, di Mario C, Prasad SK. Left atrial dilation in patients with heart failure and preserved ejection fraction: insights from cardiovascular magnetic resonance. Int J Cardiol. 2016;210:158–160. doi: 10.1016/j.ijcard.2016.02.101.

51. Takemoto Y, Barnes ME, Seward JB, Lester SJ, Appleton CA, Gersh BJ, Bailey KR, Tsang TSM. Usefulness of left atrial volume in predicting first congestive heart failure in patients > or = 65 years of age with well-preserved left ventricular systolic function. Am J Cardiol. 2005;96(6):832–6.

52. Mavrogeni S, Katsi V, Vartela V, Noutsias M, Markousis-Mavrogenis G, Kolovou G, Manolis A. The emerging role of cardiovascular magnetic resonance in the evaluation of hypertensive heart disease. BMC Cardiovasc Disord. 2017;17(1):132.

53. Nagueh SF, Appleton CP, Gillebert TC, Marino PN, JK O, Smiseth OA, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. Eur J Echocardiogr. 2009;10(2):165–193.

54. Hor KN, Baumann R, Pedrizzetti G, Tonti G, Gottliebson WM, Taylor M, Benson DW, Mazur W. Magnetic resonance derived myocardial strain assessment using feature tracking. J Vis Exp. 2011;pii:2356.

55. Claus P, Omar AMS, Pedrizzetti G, Sengupta PP, Nagel E. Tissue Tracking Technology for Assessing Cardiac Mechanics: Principles, Normal Values, and Clinical Applications. JACC Cardiovasc Imaging. 2015;8:1444–1460.

56. Schuster A, Stahnke VC, Unterberg-Buchwald C, Kowallick JT, Lamata P, Steinmetz M, Kutty S, Fasshauer M, Staab W, Sohns JM, et al. Cardiovascular magnetic resonance feature-tracking assessment of myocardial mechanics: Intervendor agreement and considerations regarding reproducibility. Clin Radiol. 2015;70:989–998.

57. Richardson P., McKenna W., Bristow M., et al. (1996) Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of Cardiomyopathies. Circulation 93:841–842.

58. Sliwa K., Damasceno A., Mayosi B.M. Epidemiology and etiology of cardiomyopathy in Africa. Circulation. 2005;112(23):3577–3583.

59. Gulati, G.S., Sharma, A.K., Paliwal, S. et al. Cardiac magnetic resonance in tropical endomyocardial fibrosis. J Cardiovasc Magn Reson 13, P270 (2011). https://doi.org/10.1186/1532-429X-13-S1-P270

60. Giorgi B, Mollet NRA, Dymarkowski S, Rademakers FA, Bogaert J: Assessment of ventricular septal motion in patients clinically suspected of constrictive pericarditis, using magnetic resonance imaging. Radiology. 2003, 228: 417-424.

61. Leyva F, Taylor RJ, Foley PW, Umar F, Mulligan LJ, Patel K, et al. Left ventricular midwall fibrosis as a predictor of mortality and morbidity after cardiac resynchronization therapy in patients with nonischemic cardiomyopathy. J Am Coll Cardiol. 2012;60(17):1659–67.

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