

Cardiovascular Magnetic Resonance Imaging

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Magnetic resonance imaging (MRI) is a machine that consists of superconducting magnets with the use of hydrogen atoms. Electrocardiogram (ECG) is used to synchronize image acquisition with the cardiac cycle phases (gaiting) and images are usually acquired during breath-holding. A cardiac coil is used for an increased signal which relates to image quality. For infants, a head coil is more suitable due to the small body habitus. Absolute contraindications for MRI are patients with pacemakers, implanted cardioverter-defibrillators, neurostimulators, cochlear implants, and vascular clips. Many metallic surgical implants such as stent and occluder, cardiac clips are safe for a 1.5 Tesla(T) magnet. It is recommended to wait approximately 6 weeks from implantation before performing cardiac MR to prevent dislodgement.¹

Clinical applications of cardiovascular MR (CMR) are of wide spectrum including evaluation of ventricular function and mass, determination of ischemic heart disease (stress perfusion and stress dobutamine), myocardial infarction, nonischemic cardiomyopathy, congenital heart disease, cardiac and paracardiac masses, etc. Further development of coronary MR angiography and interventional magnetic resonance are emerging clinical applications.

Our experience at Ramathibodi hospital with 3T magnet CMR reveals no difference in clinical applications and the most common adult and congenital heart diseases referred for CMR are for evaluation for ischemic heart disease and repaired tetralogy of Fallot, respectively.

Pulse sequences²

Due to the development of cardiac software, recently many new sequences are developed for cardiac MR, both 1.5 Tesla and 3.0 Tesla magnets.

Steady-state free-precession (SSFP) is a group of gradient recall echo (GRE) used for contractile and valvular functional evaluation. The technique is less dependent on inflow enhancement with an intrinsic high signal-to-noise ratio and superb blood-myocardial contrast. It is a sequence of choice for evaluating ventricular function, both regional and global functions.

T1-weighted fast gradient echo (FGE) and fast gradient echo-echo train imaging (FGRET) are first pass imaging sequences which are used to determine myocar-

dial blood flow by using a gadolinium chelate contrast agent (0.05 mmol/kg body weight). The study can be accomplished within less than a minute. It requires a certain amount of patient co-operation, mainly breath-holding.

Myocardial delay enhancement (MDE) is based on delay enhancement by using a T1-weighted inversion recovery pulse sequence after administration of gadolinium (0.1-0.2 mmol/kg body weight). The degree of enhancement depends on the concentration of gadolinium in the extracellular space.

Velocity-encoding phase contrast cine sequence is a technique which the speed and direction of the flowing blood are encoded as phase shifts by applying two magnetic gradients. Care must be taken for the proper preset velocity encoded. It is useful to determine the shunt volume, regurgitation volume and severity of valvular or conduit stenosis.

Contrast enhanced magnetic resonance angiography (CE-MRA) is mainly used for evaluation of vascular anatomy. The technique needs the use of gadolinium contrast agent via the peripheral vein and rapid data acquisition in both arterial and delay phases. The dose of gadolinium is 0.1-0.2 mmol/kg body weight.

Applications:

Ventricular function and mass

CMR is the "gold standard" for quantifying ventricular volumes, ejection fraction and myocardial mass.³ Interobserver and intraobserver variation of CMR to determine ventricular volume and global function are significantly lower than echocardiography. Therefore, CMR is the best method for longitudinal follow-up after therapeutic intervention.⁴ The two main reasons are due to excellent contrast between the endocardial surface and the blood pool resulting in accurate delineation of the LV cavity with no requirement of geometric assumption (Fig 1). This is important in particular cases when the left ventricular shape deviates from the assumed geometric model such as dilated cardiomyopathy.

Left ventricular function can be accessed both qualitatively and quantitatively. Abnormal segments are characterized as hypokinetic (less than 40% systolic thickening); akinetic (less than 10% systolic thickening); dyskinetic (paradoxical systolic motion) and aneurismal.⁴

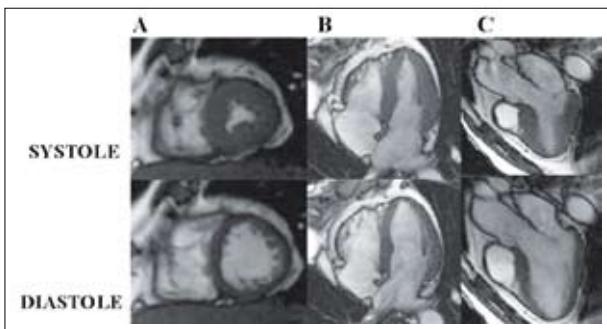


Fig 1. MR steady-state free precession images. (A) Short-axis, (B) four-chamber, and (C) three-chamber. All show superb contrast between blood pool and myocardium in both systole (top row) and diastole (bottom row).

Myocardial tissue strain can be measured by applying tag lines, however, it is not practical for routine clinical practice due to complicated mathematics and computation.

Ischemic heart disease

Two common ways for the diagnosis are stress perfusion and stress function.

Stress perfusion MR: The method is primary for evaluating blood flow to the myocardium, either by exercise or pharmacologic vasodilatation. In the resting stage, myocardial blood flow is not altered unless the luminal diameter stenosis exceeds 90% as a protective mechanism via the process of autoregulation.⁵ Pharmacologic vasodilators, either adenosine or dipyridamole, increase the resting coronary blood flow approximately four to eight times in the regions of normal perfusion in contrast to the myocardium distal to a stenotic coronary artery which shows diminished capacity to increase blood flow. Hence, the segment distal to a stenosis becomes hypoperfused relative to a normal segment during stress perfusion study.⁴

Adenosine (140 μ g/kg/min) is infused continuously for at least 4-6 minutes followed with the administration of gadolinium (0.05 mmol/kg body weight) and perfusion scan follows promptly. Pathological myocardial segments are demonstrated as diminished and delayed myocardial enhancement which follow the coronary territories, termed as “perfusion defect” (Fig 2). Myocardial ischemia is characterized by the presence of a myocardial perfusion defect during pharmacologic stress, but is normal at rest while myocardial infarction is characterized by the fixed perfusion defect in both rest and stress. However, delay enhancement technique may be used to substitute the rest perfusion study.

Jahnke C, et al. showed that the presence of ischemia detected by dipyridamole stress perfusion has



Fig 2. First-pass dipyridamole stress perfusion with contrast enhancement MR images in short axis. The hypoperfused regions (arrows) in the anteroseptal and anterior walls are demonstrated as regions of lower enhancement compare to the other myocardial regions.

an approximately twelve fold increased risk of future adverse cardiac events and negative results preclude the major cardiac events similar to nuclear medicine and can be used interchangeably for determine the low risk group.⁶ Presence of abnormal wall motion during dipyridamole stress is useful for predicting the outcome of patients with known or suspected coronary artery disease.⁷

First pass perfusion imaging can be analyzed in qualitative and quantitative ways, both of which give good results in detection of coronary artery disease. Two clinical limitations are: there is no consensus regarding imaging protocol and it is not feasible to do a quantitative approach for everyday clinical use.

Stress function MR: the test is for detection of myocardial contractility in response to an inotropic stimulating agent (dobutamine). High dose dobutamine stress (40 μ g/kg/min) augments myocardial contractility, and thus increases oxygen demand and produces ischemia in the areas perfused by a stenotic coronary artery.⁵ In a normal myocardium, there is an increased regional function myocardial oxygen demand with a corresponding increase in blood flow (Fig 3). A myocardial segment supplied by a stenotic coronary artery is demonstrated as normal wall motion at rest and further develops regional wall motion abnormality after inotropic stimulation.⁴ MR is better than echocardiography due to better image quality and lesser nondiagnostic images. The sensitivity and specificity for dobutamine stress MR in detecting significant coronary artery disease are 83%.⁸ Jahnke C, et al. showed that the presence of ischemia detected by dobutamine stress MR has an approximately five fold increased risk of an adverse future cardiac event.⁶ A normal stress dobutamine stress echocardiography results in a low annual cardiac event rate in the range of 1%-3%. Dobutamine stress MR showed the similar result in this study.

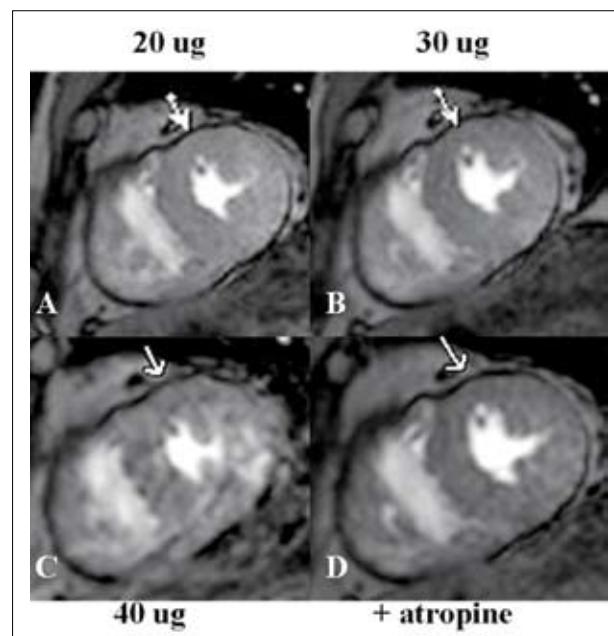


Fig 3. Dobutamine stress MR images in a patient presented with atypical chest pain and suspected anterior wall abnormality (arrow). (A) At 20 μ g/kg/min, (B) at 30 μ g/kg/min, (C) at 40 μ g/kg/min, and (D) at 40 μ g/kg/min with atropine reveals normal LV contractility (increase myocardial thickness) in all walls.

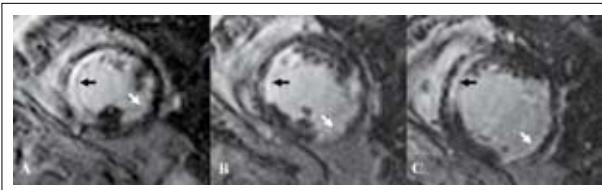


Fig 4. Short-axis myocardial delay enhancement (MDE) MR images from a patient with multivessel coronary disease. (A) At apex, (B) at mid, and (C) at basal show multiple segments of large transmural infarction (arrow). Also shown is a subendocardial infarction involving the inferolateral wall (white arrow in C).

Myocardial viability

Myocardial infarction (MI) is determined by the presence of myocardial necrosis as demonstrated by biochemical markers combined with chest pain, electrocardiographic changes and presence coronary stenosis.⁹ Viable dysfunctional myocardium, either in the form of stunned or hibernating myocardium, has a different clinical outcome after successful revascularization compared with myocardial infarction.

When imaged after Gd (0.1-0.2 mmol/kg body weight) administration and a delay for 10-15 minutes, the infarcted myocardium appears white in contrast to the normal myocardium which shows hyposignal (black) (Fig 4). Adjustment time for inversion recovery (TI) over time is required and is eased by using the Look-Locker pulse sequence (Fig 5). The technique is limited to determine the age of the infarction. Additional T2-weighted MR aids in differentiation acute from chronic infarction. Major advantages of MRI in detection MI compared with nuclear medicine imaging are higher spatial resolution that is suitable for detection of subendocardial infarction and small infarctions and the lack of tissue attenuation resulting in the better detection of inferior wall infarction.¹⁰

The extracellular space per unit of myocardium increases four fold following acute myocardial infarction and about two fold during the chronic myocardial insult compared with preinfarction.⁴ Although the imaging findings of both acute and chronic MI are the same, the mechanism of enhancement is different. In the acute stage, the accumulation of gadolinium is due to loss of membrane integrity due to myocyte death. Increased extracellular volume from tissue edema and increased capillary permeability result in overestimation of infarct size by 10% when compared with triphenyltetrazolium chloride staining of tissue samples and imaging with a necrosis-specific porphyrin agent.¹⁰ In the chronic stage, accumulation of gadolinium is due to the increased extracellular space from myocyte death and fibrosis tissue replacement.

Microvascular obstruction (no-reflow phenomenon), determined by the non-enhanced dark core within the acutely infarcted myocardium, signifies the persistent impairment of myocardial blood flow despite successful re-opening the insult coronary artery (Fig 6). The process is strongly associated with impaired functional recovery.¹⁰

The clinical significance of MDE in predicting functional recovery of wall motion were determined in many studies. Kim et al showed a good relationship between the lack of transmural enhancement and the likelihood of improvement of contractile function after

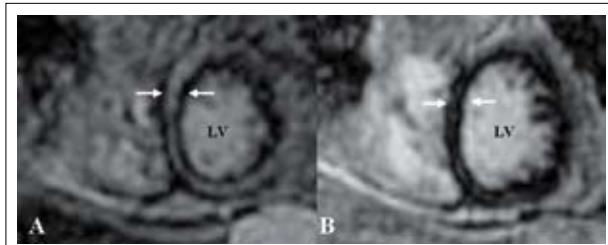


Fig 5. Short axis myocardial delay enhancement with 3T magnet at different TI time. (A) AT TI = 200 msec and (B) at TI = 250 msec. The proper TI time is at 250 msec due to completely null myocardium signal seen as black myocardium. LV = left ventricle.

revascularization.¹¹ The probability of death was six times higher in the large scar (more than 6 segments) than in those with the small scar group.¹⁰ Large infarct (> 30% of the left ventricle) posses a higher risk of cardiac death, heart failure, recurrent MI, unstable angina and stroke after 16 months follow-up.¹⁰ Future study with a large clinical trial to determine the adverse clinical outcome of small infarction detected only by MR, not by nuclear imaging, is an interesting issue.

Nonischemic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM): the disease is characterized by abnormal myocardial hypertrophy with normal left ventricular systolic function and impaired diastolic function. There are increased myocardial perfusion defect (PD) and presence of delay enhancement (DE) in the area of increased wall thickness (Fig 7). Both PD and DE are useful for evaluating the site and the extent of myocardial injury which corresponding with the areas of regional wall motion abnormality. The findings relate to other poor prognostic risks of sudden death, LV dilatation and heart failure.^{3,12}

Dilated cardiomyopathy (DCM): The gold standard method to differentiate between ischemic and non-ischemic cardiomyopathy is a coronary angiogram. Myocardial ischemic scar is present in less than 15% of patients with DCM which is explained by unrecognized MI or coronary embolism. The nonischemic enhancement in DCM is seen in about 28% of patients and is demonstrated as patchy or linear midwall enhancement which is correlated with a negative prognostic implication.³

Arrhythmogenic right ventricular dysplasia (ARVD): The disease is characterized by fibro-fatty replacement

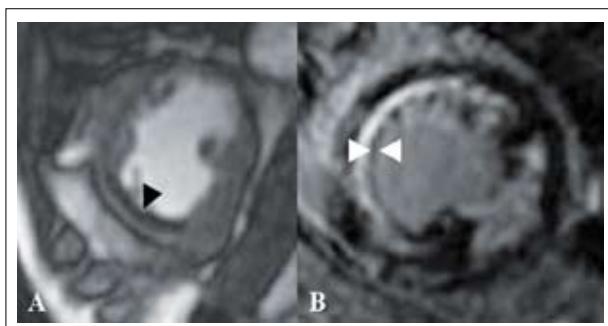


Fig 6. Short-axis rest perfusion MR (A) and short-axis delay enhancement (B) images show non-enhanced tissue at subendocardial side (arrow) of transmural infarction indicating presence of microvascular obstruction.

in the right ventricle with lesser common replacement in the left ventricle. The MR diagnostic criteria are the presence of regional wall motion abnormality corresponding to the area of fibrofatty replacement (Fig 8). However, the diagnosis should be based on established clinical and laboratory criteria.

Congenital heart disease (CHD)

Echocardiography is the initial imaging modality of choice in evaluation of CHD. CMR is mainly used for problem solving and to determine ventricular function, particular for the right sided chamber. Young children and infants require general anesthesia to protect airways and control respiration. The major vascular indication is the disease of aorta: particularly coarctation of the aorta. CMR is useful for long term follow-up repaired coarctation. Aside from the anatomical data, phase contrast can be used to determine disease severity by applying velocity encoding to the stenosis site, which can be converted to a pressure gradient via the Bernoulli equation. A step up aortic flow just at the distal to coarctation aorta compared with the level of diaphragm signifies the amount of collateral.¹³

Echocardiography is a mainstay in evaluation of left ventricular function, although, there is a limitation in evaluation of right ventricular (RV) function due to poor visualization. RV volume and ejection fraction are important in follow up patients who have been repaired of tetralogy of Fallot in correlation with their clinical context and ECG change to determine the proper time for pulmonic valve replacement for the purpose of preventing pump failure and arrhythmias (Fig 9). Currently, CMR is the modality of choice for serial follow-up of right ventricular function quantitatively.¹³

Valvular heart disease

Valvular regurgitation : Echocardiography is the mainstay for evaluation of left-heart valves. Valvular regurgitation can be evaluated with MR, both qualitatively and quantitatively. Qualitatively, regurgitation is seen as an area of signal loss (black) from the dysfunctional valve to the proximal cardiac chamber. The graded system is followed angiography. Quantitatively, this can be approached by indirect and direct methods. The indirect quantification method applies mainly for the single valvular dysfunction, particularly in the atrioventricular valves (mitral and tricuspid valves). Difference in stroke volume between right and left ventricles yields regurgitation volume. A direct quantification method is accomplished by using phase contrast to determine the forward and backward flow through the valve. Severe

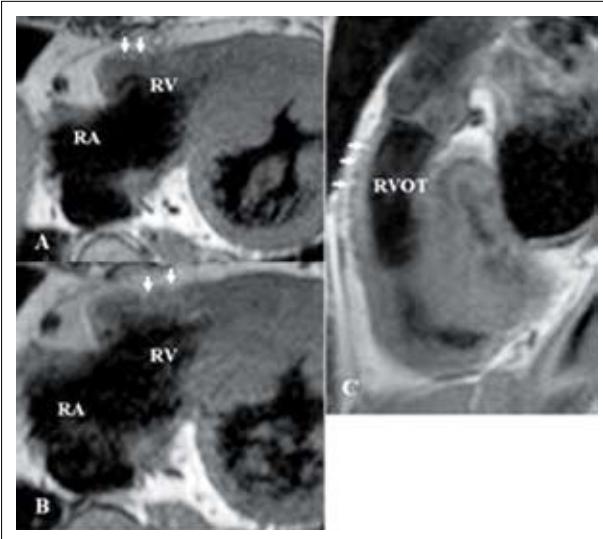


Fig 8. Arrhythmogenic right ventricular dysplasia (ARVD). Axial T1W at different level of right ventricular outflow region (A, B) and sagittal T1W (C) show multiple foci of fatty infiltration seen as white dots along the subepicardium and myocardium (arrows). RA= right atrium, RV = right ventricle, RVOT = right ventricular outflow tract.

aortic and pulmonic regurgitation is determined by the presence of regurgitation fraction $> 48\%$ and $\geq 40\%$, respectively. The leading role of MR in evaluation of pulmonic valvular regurgitation is well established (Fig 9).¹⁴

Valvular stenosis: MR in evaluation of valvular stenosis is based on the echocardiographic basis, for example the continuity equation to determine valve area. Through plane velocity mapping is used to determine the gradient across the valve via the Bernoulli equation. Although MR is useful, echocardiography is a mainstay in the evaluation of valvular disease.¹⁴

Cardiac masses

The main advantages of MR over echocardiography are a larger field of view and tissue characterization in certain lesions such as fat and fibrous tissue. MR is also useful in differentiation of thrombus from tumor, either benign or malignant and evaluation of tumor extent (Fig 10, 11).

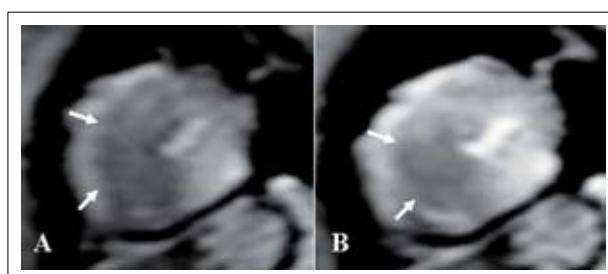


Fig 7. Short-axis rest perfusion MR images from a patient with hypertrophic cardiomyopathy. (A) At apex and (B) at mid ventricle show a dense perfusion defect (arrows) in the septum.

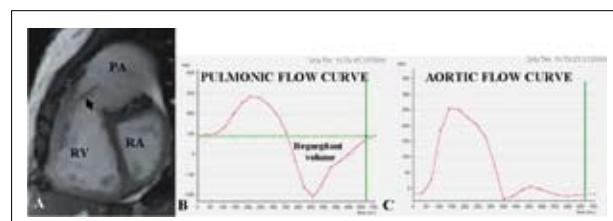


Fig 9. Pulmonic regurgitation. (A) steady-state free precession cine MR in sagittal plane of right ventricle shows pulmonic regurgitation seen as black jet line (arrow). (B) Main pulmonary artery flow curve and (C) aortic flow curve measured by velocity-encoded cine MR demonstrate the area below the baseline signify the regurgitation volume (B), comparing with the normal aortic flow curve (C). RA= right atrium, RV = right ventricle, PA = pulmonary artery.

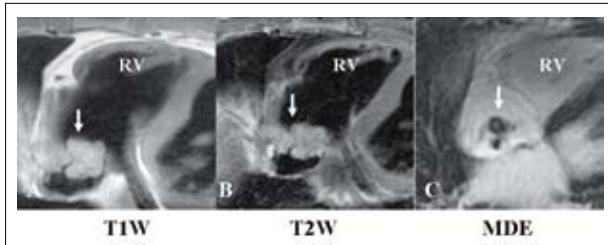


Fig 10. Transvenous extension of hepatocellular carcinoma into the right atrium (arrow) demonstrated as isosignal T1W (A), slight hypersignal T2W (B) and thick irregular enhancement in myocardial delay enhancement (C). RV = right ventricle.

CONCLUSION

Cardiovascular MR is a complex set up machine which requires special training for both technicians and a specialist cardiovascular MR interpreter. Its most important clinical applications are evaluation of LV function and coronary artery disease in patients with suboptimal echocardiographic examination, quantification of myocardial scar tissue for treatment planning and longitudinal follow-up, right ventricular function evaluation such as in tetralogy of Fallot and follow-up of congenital heart disease. Future directions with improvement of hardware and software will allow shorter examination time and improvement of coronary MR imaging. Interventional MR is a fantastic future direction that can obviate the radiation dose to patients.

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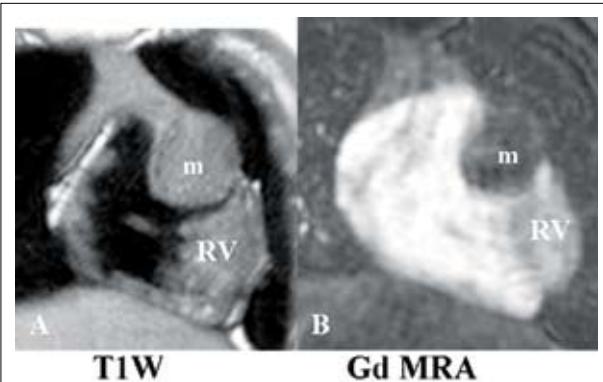


Fig 11. Rhabdomyoma in a child presented with arrhythmia. The mass (m) is demonstrated as isosignal T1W (A), lack of contrast enhancement in dynamic gadolinium MR angiography (B). RV = right ventricle.