Article published online: 2021-10-04

Magnetic resonance imaging in the evaluation of congestive cardiac failure

Prabhakar Rajiah

Department of Radiology, Cardiothoracic Imaging Section, University Hospitals of Cleveland, Case Western Reserve University, Cleveland, Ohio USA

Correspondence: Dr. Prabhakar Rajiah, Radiology Department, University Hospitals Case Medical Center, 11100 Euclid Avenue, Cleveland OH 44106, United States, USA. E-mail: radprabhakar@gmail.com

Abstract

Congestive cardiac failure is the end-result of various cardiac disorders, and is a major contributor to morbidity, mortality, and financial burden throughout the world. Due to advances in the knowledge of the disease and scanner technology, magnetic resonance imaging (MRI) is playing an increasingly important role in the evaluation of cardiac failure, including in establishing diagnosis, problem solving, risk stratification, and monitoring of therapy. This review discusses and illustrates the role of MRI in the assessment of congestive cardiac failure.

Key words: Cardiac failure; ischemia; magnetic resonance imaging

Introduction

Congestive cardiac failure is the end result of various cardiac disorders [Table 1]. Due to an aging population and improved survival from coronary events, the prevalence of congestive cardiac failure has increased. It is a major cause of morbidity and mortality, and is an important cause of high healthcare cost.[1] Due to advances in knowledge about the disease and in scanner technology, magnetic resonance imaging (MRI) is playing an increasingly important role in the evaluation of various aspects of cardiac failure [Table 2]. A standardized protocol for the evaluation of cardiac failure is shown in Table 3. This review discusses and illustrates the role of MRI in the assessment of congestive cardiac failure.

Establishing the Diagnosis

The diagnosis of cardiac failure is typically based on clinical symptoms and signs and investigations, including echocardiography. However, MRI is occasionally used for establishing the diagnosis when the diagnosis is indeterminate, usually due to discrepant ejection fractions as measured by different imaging techniques. MRI has high accuracy and reproducibility in the measurement of ventricular systolic function.[2] Diastolic function can also be evaluated using flow curves or time–volume curves, although this is not routinely performed in clinical practice.

Table 1: Causes of cardiac failure

Ischemic cardiomyopathy
Dilated cardiomyopathy
Myocarditis
Hypertrophic cardiomyopathy
Amyloidosis
Sarcoidosis
Anderson–Fabry disease
ARVD
Iron-overload
Left ventricular non-compaction
Constrictive pericarditis
Chagas disease
Churg–Strauss syndrome
Endomyocardial fibrosis
Takotsubo cardiomyopathy
Masses
Valvular heart disease
Congenital heart disease

ARVD : Arrhythmogenic right ventricular dysplasia

Access this article online

Quick Response Code:

Website: www.ijri.org

DOI: 10.4103/0971-3026.107177

Table 2: Role of magnetic resonance imaging in the evaluation of congestive cardiac failure

Role	Comments
Establishing diagnosis	Useful for evaluation if there is discordant information from other investigations
Establishing etiology	Ischemic versus non-ischemic
	Enables tailoring of therapy
Quantification	Ventricular function, scar burden
Prognostic markers	Information on several prognostic markers, which enables risk stratification.
Prediction of response to therapy	Determines suitability for therapeutic procedures such as coronary revascularization and cardiac resynchronization
Monitoring of therapy	Accurate, reproducible, and ideal for serial follow-up and monitoring of therapy and disease progression
Structural information	Provides information on left ventricle, right ventricle, valves, pericardium, coronary arteries, great vessels
Others	Identifies shunts, masses, thrombus, myocardial edema

Table 3: Magnetic resonance imaging protocol suggested for evaluation of congestive cardiac failure

Sequences	Role
Ultra-fast spin echo	Evaluation of thorax
	Planning of subsequent views
Cine SSFP- Multiple planes	Functional evaluation; morphology; quantification
T2 FSE, T2 TSE STIR	Morphology; myocardial edema
Velocity-encoded phase contrast imaging	Flow quantification
Perfusion imaging	Myocardial ischemia
Delayed enhancement	Scar/interstitial fibrosis
Optional sequences	
Myocardial tagging	Regional function
T2* black-blood	Myocardial iron quantification
Early post-contrast T1 fast spin-echo	Acute inflammation of pericardium/ myocardium
Real-time imaging of septum	Pericardial constriction
T1 mapping	Quantification of myocardial fibrosis
Whole-heart 3D fat suppressed SSFP	Proximal coronary arteries

SSFP : Steady-state free-precession, FSE : Fast spin echo; STIR : Short tau inversion recovery

Establishing the Etiology

The principal utility of MRI in the evaluation of cardiac failure is its ability to characterize the underlying disease based on the pattern and location of scar/interstitial fibrosis using delayed enhancement imaging [Table 4].[3] Establishing the etiology enables tailoring of treatment according to the cause.[3] Ischemic cardiomyopathy is the most common cause of cardiac failure (62%).[4] Subendocardial pattern of delayed enhancement is seen in early infarct and a transmural pattern is seen in established infarct, both conforming to a vascular territorial distribution. In acute myocardial infarction (MI), T2-weighted images show myocardial edema in the affected vascular territory [Figure 1A]. In severe acute MI, a dark area can be seen within the enhanced scar [Figure 1B] due to microvascular obstruction. Non-ischemic patterns of enhancement are mid-myocardial (linear, patchy, or at right ventricular insertion points), subepicardial, and global subendocardial/transmural. Non-ischemic dilated cardiomyopathy is a diagnosis of exclusion, made when the left ventricle is dilated, with poor systolic function, but with normal coronary arteries. In 10-28% of these patients, a mid-myocardial pattern of enhancement is seen in the basal and mid-septum [Figure 2].[5] However, ischemic scar pattern is seen in 13% of clinically diagnosed non-ischemic cardiomyopathy. Myocarditis produces cardiac failure in severe cases. In addition to global or regional dysfunction, myocardial edema, and contrast enhancement (early and delayed) is seen in a mid-myocardial or subepicardial distribution. Typically, the enhancement decreases or disappears with time (in 88% of cases), but may persist occasionally.[6] Sarcoidosis involves the heart in 5-25% of patients and is associated with regional wall-motion abnormalities, myocardial edema, and thickening and mid-myocardial or subepicardial pattern of delayed enhancement [Figure 3].[7] The disease activity may be monitored with T2-weighted imaging and, typically, the areas of delayed enhancement decrease following steroid therapy.[8] Hypertrophic cardiomyopathy is characterized by various patterns of myocardial hypertrophy, which is typically asymmetric septal. Although in the early stages there is hyperdynamic systolic function, in the late/burn-out phases there is diminished function with chamber dilation and wall thinning. Papillary muscle abnormalities may be seen. Eighty-eight percent of these patients have delayed enhancement, which is of a patchy mid-myocardial pattern, more common at Right ventricle (RV) insertion sites [Figure 4].[9] Cardiac amyloidosis is characterized by thickened myocardium, atria, and interatrial septum, with diminished systolic function and bi-atrial enlargement.[10] Delayed enhancement is typically global subendocardial [Figure 5] progressing to transmural, but can also occasionally be patchy. A unique feature of cardiac amyloidosis is the alteration of T1 kinetics of gadolinium distribution, with nulling of the myocardium before the blood pool due to diffuse amyloid infiltration, resulting in higher gadolinium uptake and T1 shortening. T1 values can be mapped using Look-Locker or modified Look–Locker sequences (MOLLI).[11]

Pericardial constriction is characterized by impaired filling of the cardiac chambers due to a thick (>4 mm) or non-compliant pericardium.[12] Other morphological features include conical or tubular deformity of ventricles, bi-atrial enlargement, pleural effusion, superior vena cava and inferior vena cava dilation, and pulmonary artery dilation. Diastolic septal bounce and abrupt cessation of diastolic filling may also be seen. Real-time imaging of the ventricular septum shows septal flattening or bowing towards the left ventricle during inspiration [Figure 6].[13] MRI is a good modality for the evaluation of valvular heart disease, particularly in qualitative and quantitative estimation of valvular function [Figure 7].[14] MRI is the ideal technique for the evaluation of various congenital heart diseases, being particularly useful in the evaluation of morphology and ventricular and valvular function following treatment.[15] Cardiac masses can present with new-onset heart failure. In addition to detecting cardiac masses, MRI can also characterize these masses and detect involvement of adjacent structures and obstruction of valve or compression of ventricles.

Iron-overload cardiomyopathy is a major cause of cardiac failure in patients with hemolytic anemias and multiple blood transfusions. The T2* value of the myocardium can be detected using a single breath-hold, black-blood multi-echo sequence (Images obtained at various TEs), and this is directly related to myocardial iron

Table 4: Various patterns of delayed enhancement

- Subendocardial- vascular distribution
  - Ischemia
- Transmural- vascular distribution
  - Ischemia
- Global subendocardial
  - Amyloidosis
  - Systemic sclerosis
  - Cardiac transplant
  - Uremia
- Subepicardial
  - Myocarditis
  - Sarcoidosis
  - Fabry disease
  - Chagas disease
- Mid-myocardial
  - Linear
    - Dilated cardiomyopathy
  - Insertion points
    - Hypertrophic cardiomyopathy
    - Right ventricular pressure overload
    - Systemic sclerosis
  - Patchy
    - Myocarditis
    - Sarcoidosis
    - Fabry disease
    - Chagas disease

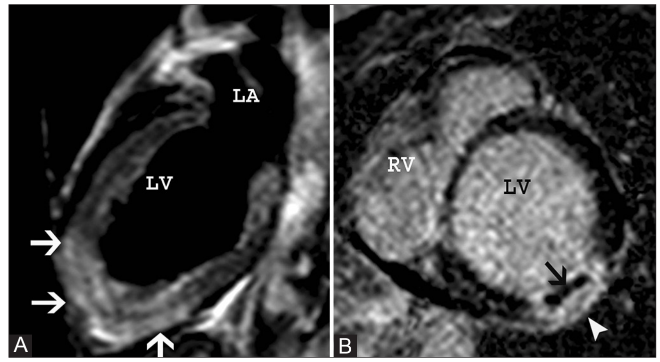

Figure 1 (A,B): Myocardial infarction. (A) Acute myocardial edema; the two-chamber T2-weighted black-blood image shows high signal in the apical anterior, apical inferior, and apical segments (arrows), consistent with myocardial edema in a patient with acute myocardial infarction. (B) Short-axis delayed-enhancement image shows a dark non-enhancing area (arrow) in the basal infero-lateral segment within a focal area of enhancing myocardial scar (arrowhead), consistent with microvascular obstruction within an acute myocardial infarction

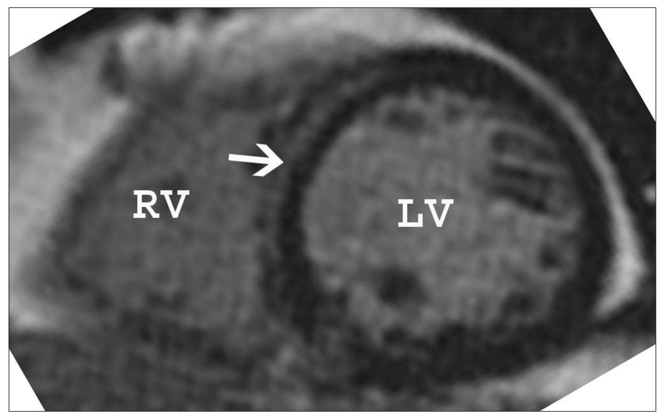

Figure 2: Non-ischemic dilated cardiomyopathy. Short-axis delayed-enhancement magnetic resonance imaging demonstrates a dilated left ventricle with linear mid-myocardial scarring in the basal septum (arrow); this is a characteristic pattern seen in non-ischemic dilated cardiomyopathy. The coronary arteries were normal in this patient

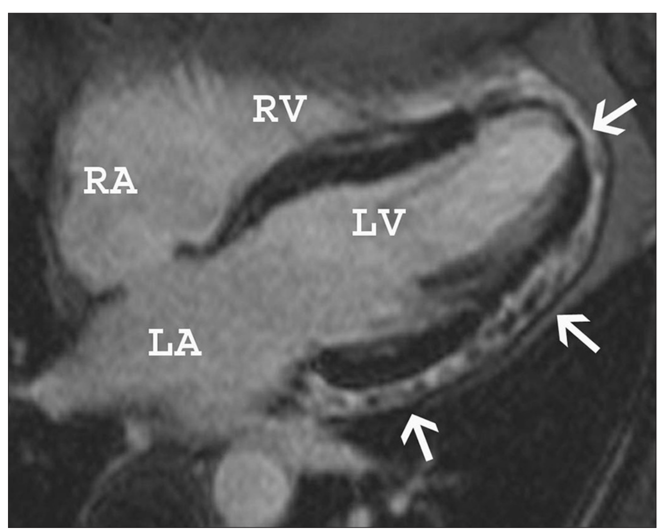

Figure 3: Cardiac sarcoidosis. Four-chamber delayed-enhancement magnetic resonance imaging in a patient with known sarcoidosis and heart block shows diffuse myoepicardial enhancement (arrows) in a pattern consistent with cardiac sarcoidosis

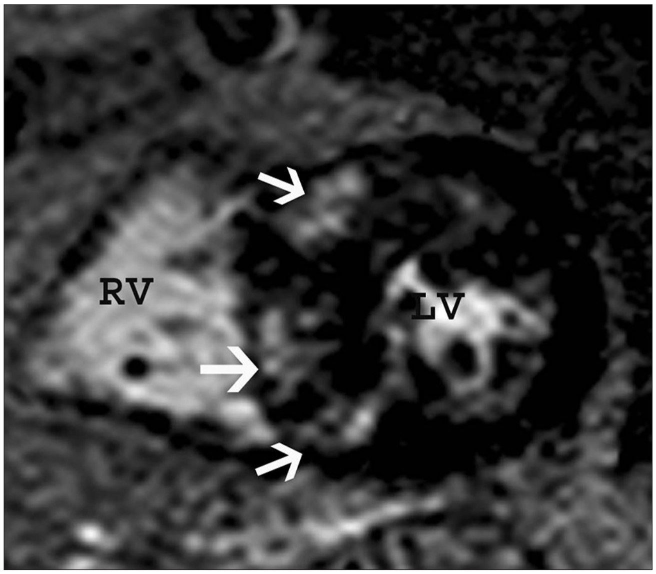

Figure 4: Hypertrophic cardiomyopathy. Short-axis delayed-enhancement image shows hypertrophied myocardium and patchy, sandy areas of delayed enhancement (arrows) in a pattern typical for hypertrophic cardiomyopathy

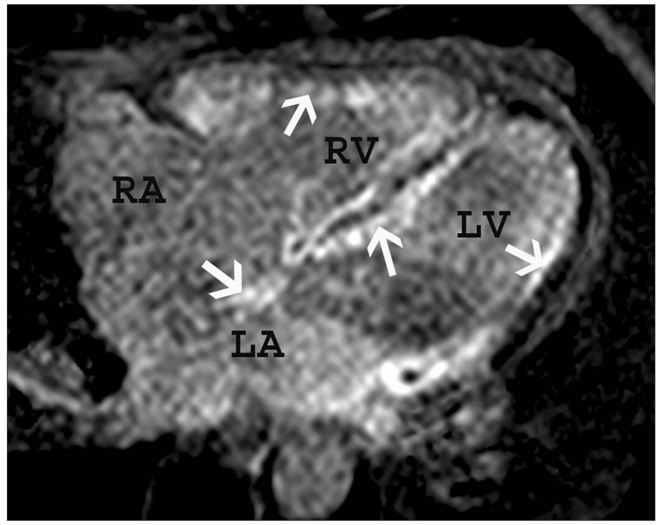

Figure 5: Cardiac amyloidosis. Four-chamber delayed-enhancement magnetic resonance imaging shows diffuse subendocardial enhancement (arrows) extending to the mid-myocardium, involving the entire left ventricle, right ventricle, interatrial septum, atrial walls, and valves, consistent with cardiac amyloidosis

Figure 6: Constrictive pericarditis. Real-time image of the ventricular septum obtained after inspiration shows a flattened interventricular septum (arrow), consistent with constrictive pericarditis

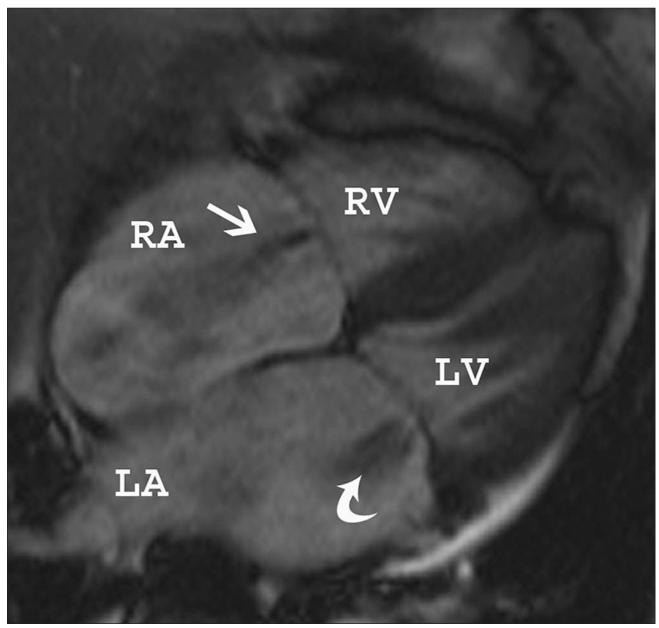

Figure 7: Valvular regurgitation. Four-chamber steady-state free-precession image shows severe tricuspid (straight arrow) and moderate mitral valvular (curved arrow) regurgitation

level [Figure 8]. Using T2* imaging, iron chelation therapy can be initiated before the onset of symptoms and the myocardial T2* and ejection fraction can be improved.[16] This approach has resulted in markedly improved survival in thalassemia major patients in the United Kingdom.[17] Arrhythmogenic right ventricular dysplasia (ARVD) is characterized by progressive fibrofatty replacement of the right ventricular myocardium, with fat demonstrated using black-blood images and fibrous tissue using delayed enhancement. Global systolic dysfunction, regional wall-motion abnormalities and aneurysms indicate the diagnosis, which is usually based on the Task Force criteria.[18] Left ventricular non-compaction is characterized by a ratio of non-compacted to compacted myocardium of >2.3:1 and an abrupt transition from thick compacted myocardium to a thinned myocardium [Figure 9]. Delayed enhancement may be seen in the non-compacted myocardium.[19] Takotsubo cardiomyopathy (stress-induced cardiomyopathy) is characterized by acute onset of left ventricular dysfunction, with akinesis of the apical segments and

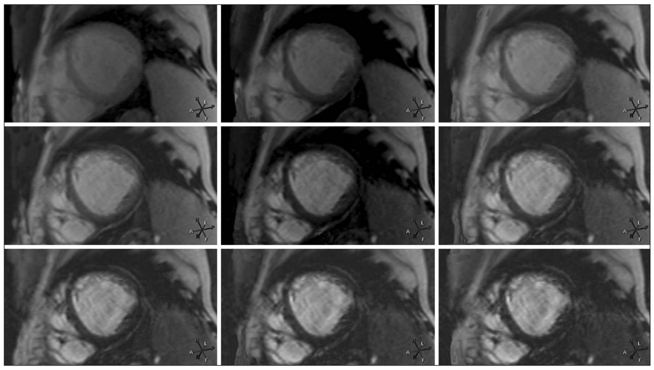

Figure 8: Iron-overload cardiomyopathy. Short-axis black-blood T2*-weighted images acquired with progressively increasing echo time (TE) (mentioned in the top left of each image) shows progressive darkening of the myocardium with increasing TE; this is due to iron deposition

hyperkinesis of the basal segments, myocardial edema, and no delayed enhancement.[20] The cardiac failure is usually reversible. Anderson–Fabry disease is a lysosomal disorder, presenting with concentric myocardial hypertrophy and increased ejection fraction in early stages and wall thinning and systolic dysfunction in later phases. Enhancement is seen in the mid-myocardial to epicardial layers, more commonly in the basal infero-lateral wall.[21]

Quantification

MRI has high accuracy and reproducibility in the measurement of ventricular function.[2] Global systolic function is evaluated using short-axis cine images whereas regional function can be evaluated visually or through myocardial tagging techniques. MRI is also highly accurate and reproducible in the measurement of scar.[22,23] Scar can be measured either by qualitative, semi-quantitative, or quantitative means. Summed scar score and transmurality index are used in qualitative estimation of scar.[24] In the semi-quantitative technique, the signal intensity of remote normal myocardium is measured and scar is defined as tissue with signal intensity above a threshold of 2-6 standard deviations above the mean signal intensity of normal myocardium [Figure 10]. In manual planimetry, the areas of enhancement can be manually contoured and expressed as grams or percentage of cardiac mass.

Prognostic Information

MRI provides prognostic information in the various disorders that cause cardiac failure [Table 5]. Delayed enhancement implies adverse prognosis in most of these diseases, as scar is a substrate for ventricular arrhythmia and is associated with adverse cardiovascular events and sudden cardiac death. Scar size by MRI (irrespective of the cause) [Figure 10] also predicts survival and all-cause mortality, independent of left ventricular ejection fraction.[22-24]

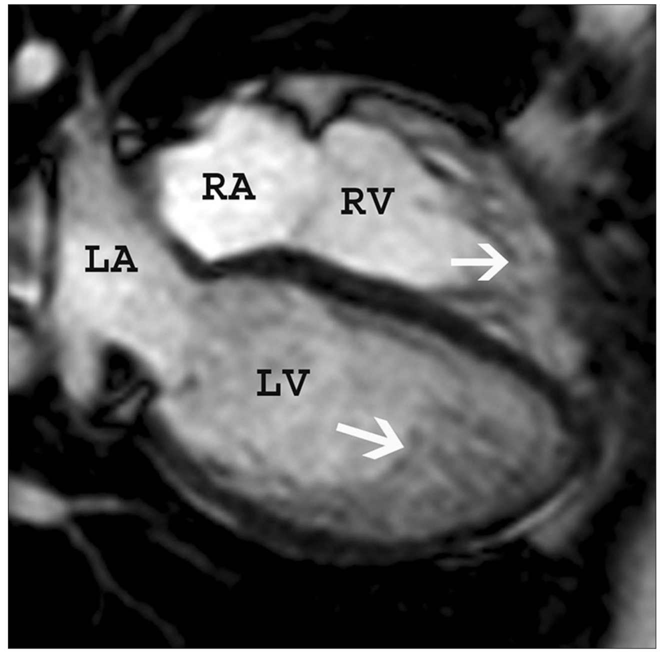

Figure 9: Left ventricle non-compaction. Four-chamber steady-state free-precession image in a 27-year-old man shows prominent trabeculations (arrows) in the mid- and apical regions of the left ventricle and thinning of compacted myocardium, consistent with left ventricular non-compaction

Ischemic cardiomyopathy

In acute MI, micro-vascular obstruction implies poor prognosis due to association with adverse cardiovascular events, and adverse remodeling[25] Hemorrhage within the core of infarct also implies adverse prognosis due to association with larger infarct size, adverse remodeling and increase of LV end-systolic volume.[26] Myocardial salvage index (Area of high signal in T2-weighted images − Area of delayed hypernenhancement/Area of high signal in T2-weighted images) has a prognostic value comparable to infarct size and microvascular obstruction.[27] After the acute stage, infarct size is the most important predictor of functional recovery, with transmural scar associated with poor recovery following revascularization procedures [Figure 11A].[28] Patients with silent MI have an increased (6- to 11-fold) risk for major cardiac events.[29] The presence of tiny amounts of scar, regardless of history of MI, is associated with higher risk of adverse events.[30] The infarct size is a better predictor of ventricular tachycardia (VT) than left ventricular ejection fraction or left ventricular volumes.[31] Higher infarct heterogeneity has a direct correlation with higher susceptibility to VT. Right ventricular function late after MI is also an important predictor of prognosis.[32] Peri-infarct ischemia is associated with higher incidence of cardiovascular events.[33]

Non-ischemic cardiomyopathy

As in ischemic disease, the presence of delayed enhancement generally implies adverse prognosis

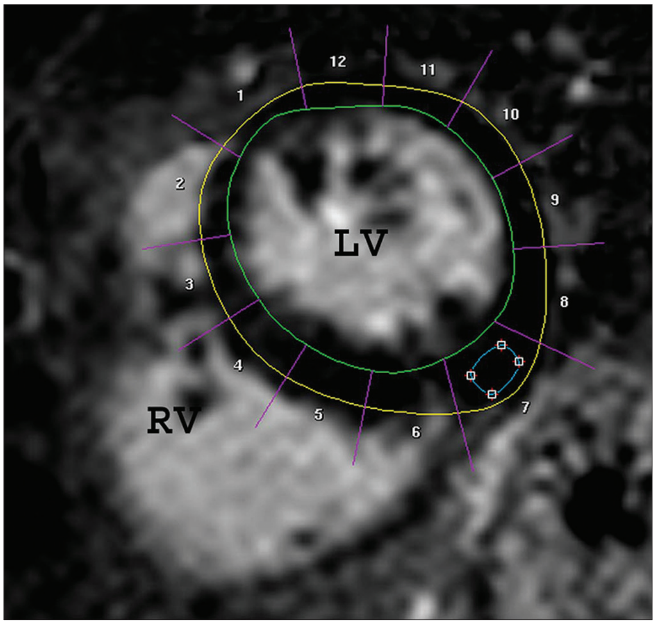

Figure 10: Scar quantification. The endocardial and epicardial contours are segmented. The normal myocardium is selected (blue), and based on this value a threshold for abnormal myocardium is selected. This helps in quantitative estimation of scarred areas

in non-ischemic disorders. Mid-myocardial scar in non-ischemic cardiomyopathy is associated with higher incidence of arrhythmias, adverse events, hospitalization, and mortality.[34] Parvovirus B19 myocarditis produces lateral wall enhancement and recovery within a few months, but herpesvirus six myocarditis produces septal enhancement and rapid progression to cardiac failure.[35] Severe hyperenhancement is associated with poor prognosis in sarcoidosis,[36] hypertrophic cardiomyopathy,[37] ARVD,[18] LV non-compaction,[38] Fabry's disease[39] and cardiac amyloidosis.[40] In cardiac amyloidosis, a 2 min post-contrast T1 difference between the subepicardium and the subendocardium of less than 23 ms decreased survival.[11] In iron-overload cardiomyopathy, myocardial T2* values of less than 20 ms indicate iron-overload and values less than 10 ms indicate severe iron-overload. Lower T2* values are generally associated with severe ventricular dysfunction,[41] Presence of non-compaction in the mid-ventricular level indicates the presence of relatively more severe disease and bad prognosis.[42]

Prediction of Response to Therapy

MRI plays an important role in selecting patients who would benefit from surgical or interventional procedures. There is an inverse relationship between the amount of scar and the recovery of contractile function, following coronary revascularization procedures.[43] While myocardial segments with wall motion abnormalities and no/mild (<25%) scar have good likelihood of functional recovery following revascularization, segments

Table 5: Adverse prognostic indicators in cardiac failure due to various causes

Disease	Adverse prognosis
Ischemic cardiomyopathy	Microvascular Obstruction
	Hemorrhage in the core
	Infarct size
	Peri-infarct zone
	Infarct heterogeneity
	Right ventricular ejection fraction
	Peri-infarct ischemia
Non-ischemic dilated cardiomyopathy	Mid-myocardial scar
Hypertrophic cardiomyopathy	Fibrosis
Amyloidosis	Delayed enhancement
	2 min post-contrast T1 difference between the subepicardium and the subendocardium of less than 23 ms
Thalassemia	T2*<10 ms
Myocarditis, sarcoidosis, ARVD, Fabry's disease, Non compaction	Delayed enhancement

with >75% hyperenhancement have been shown to have little or no potential for functional recovery following revascularization [Figure 11A] [43] In addition, cardiac resynchronization therapy (CRT) will not be effective if there is extensive scar in the lateral wall or septum that prevents electrical activation [Figure 11B].[44] Three-dimensional whole-heart MR-venography can be used to assess the venous anatomy since variations in venous anatomy, including absence of common veins, may result in failure of the procedure and therefore warrant surgical epicardial lead placement.

Monitoring of Therapy

Due to its high accuracy and reproducibility in the evaluation of systolic function, MRI is the ideal modality for serial follow-up to monitor response to various therapeutic interventions. MRI is also used for evaluating the efficacy of novel therapeutic strategies in reducing reperfusion injury and infarct size, increasing salvageable myocardium and altering prognostic indicators. The size of the scar in MI is a useful surrogate endpoint for new clinical trials on the efficacy of drugs in the treatment of MI.[32] A reduction of infarct size may alter ventricular remodeling and improve prognosis.

Conclusion

MRI plays a pivotal role in various aspects of cardiac failure. It is useful in establishing the diagnosis and etiology. It enables risk stratification, provides prognostic information, and determines suitability for surgical/interventional procedures. The presence of scar or fibrosis implies adverse prognosis in several conditions that cause cardiac failure.

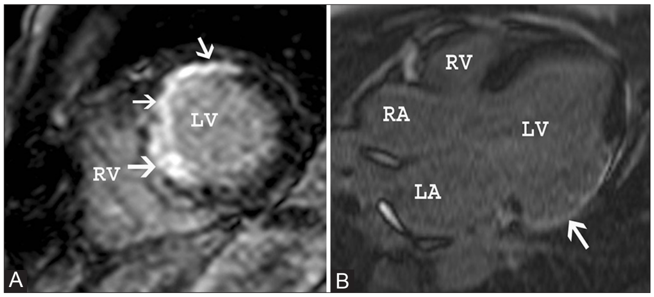

Figure 11 (A,B): Prognostic markers. (A) Short-axis delayed-enhancement image shows transmural scar in the anterior wall and anteroseptum (arrows) in the left anterior descending (LAD) distribution. Due to the extensive scar in this vascular territory, a revascularization procedure such as coronary artery bypass surgery is unlikely to be successful. (B) Three-chamber delayed-enhancement image shows an extensive transmural scar in the basal and mid-lateral wall (arrow), which indicates low probability of success with cardiac resynchronization therapy

References

1. Dayer M, Cowie MR. Heart failure: Diagnosis and healthcare burden. Clin Med 2004;4:13-8.
2. Grothues F, Smith GC, Moon JC, Bellenger NG, Collins P, Klein HU, *et al*. Comparison of interstudy reproducibility of cardiovascular magnetic resonance with two-dimensional echocardiography in normal subjects and in patients with heart failure or left ventricular hypertrophy. Am J Cardiol 2002;90:29-34.
3. Shah DJ, Judd RM, Kim J. Myocardial viability. In: Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, editors. Clinical Magnetic Resonance Imaging. 3rd ed.. New York, NY: Elsevier; 2006. p. 1030-49.
4. Gheorghiade M, Sopko G, De Luca L, Velazquez EJ, Parker JD, Binkley PF, *et al*. Navigating the crossroads of coronary artery disease and heart failure. Circulation 2006;114:1202-13.
5. McCrohon JA, Moon JC, Prasad SK, McKenna WJ, Lorenz CH, Coats AJ, *et al*. Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance. Circulation 2003;108:54-9.
6. De Cobelli F, Pieroni M, Esposito A, Chimenti C, Belloni E, Mellone R, *et al*. Delayed gadolinium-enhanced cardiac magnetic resonance in patients with chronic myocarditis presenting with heart failure or recurrent arrhythmias. J Am Coll Cardiol 2006;47:1649-54.
7. Vignaux O. Cardiac sarcoidosis: Spectrum of MRI features. AJR Am J Roentgenol 2005;184:249-54.
8. Vignaux O, Dhote R, Duboc D, Blanche P, Devaux JY, Weber S, *et al*. Detection of myocardial involvement in patients with sarcoidosis applying T2-weighted, contrast-enhanced, and cine magnetic resonance imaging: Initial results of a prospective study. J Comput Assist Tomogr 2002;26:762-7.
9. Moon JC, Reed E, Sheppard MN, Elkington AG, Ho SY, Burke M, *et al*. The histologic basis of late gadolinium enhancement cardiovascular magnetic resonance in hypertrophic cardiomyopathy. J Am Coll Cardiol 2004;43:2260-4.
10. Vogelsberg H, Mahrholdt H, Deluigi CC, Yilmaz A, Kispert EM, Greulich S, *et al*. Cardiovascular magnetic resonance in clinically suspected cardiac amyloidosis: Noninvasive imaging compared to endomyocardial biopsy. J Am Coll Cardiol 2008;51:1022-30.
11. Maceira AM, Prasad SK, Hawkins PN, Roughton M, Pennell DJ. Cardiovascular magnetic resonance and prognosis in cardiac amyloidosis. J Cardiovasc Magn Reson 2008;10:54.
12. Talreja DR, Nishimura RA, Oh JK, Holmes DR. Constrictive pericarditis in the modern era: Novel criteria for diagnosis in the cardiac catheterization laboratory. J Am Coll Cardiol 2008;51:315-9.
13. Francone M, Dymarkowski S, Kalantzi M, Bogaert J. Real-time cine MRI of ventricular septal motion: A novel approach to assess ventricular coupling. J Magn Reson Imaging 2005;21:305-9.
14. Cawley PJ, Maki JH, Otto CM. Cardiovascular magnetic resonance imaging for valvular heart disease: Technique and validation. Circulation 2009;119:468-78.
15. Gutiérrez FR, Ho ML, Siegel MJ. Practical applications of magnetic resonance in congenital heart disease. Magn Reson Imaging Clin N Am 2008;16:403-35.
16. Pennell DJ. T2* magnetic resonance and myocardial iron in thalassemia. Ann N Y Acad Sci 2005;1054:373-8.
17. Modell B, Khan M, Darlison M, Westwood MA, Ingram D, Pennell DJ. Improved survival of thalassaemia major in the UK and relation to T2* cardiovascular magnetic resonance. J Cardiovasc Magn Reson 2008;10:42.
18. Marcus FI, McKenna WJ, Sherrill D, Basso C, Bauce B, Bluemke DA, *et al*. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: Proposed modification of the task force criteria. Circulation 2010;121:1533-41.
19. Petersen SE, Selvanayagam JB, Wiesmann F, Robson MD, Francis JM, Anderson RH, *et al*. Left ventricular non-compaction: Insights from cardiovascular magnetic resonance imaging. J Am Coll Cardiol 2005;46:101-5.
20. Pilgrim TM, Wyss TR. Takotsubo cardiomyopathy or transient left ventricular apical ballooning syndrome: A systematic review. Int J Cardiol 2008;124:283-92.
21. De Cobelli F, Esposito A, Belloni E, Pieroni M, Perseghin G, Chimenti C, *et al*. Delayed-enhanced cardiac MRI for differentiation of Fabry's disease from symmetric hypertrophic cardiomyopathy. AJR Am J Roentgenol 2009;192:W97-102.
22. Cheong BY, Muthupillai R, Wilson JM, Sung A, Huber S, Amin S, *et al*. Prognostic significance of delayed-enhancement magnetic resonance imaging: Survival of 857 patients with and without left ventricular dysfunction. Circulation 2009;120:2069-76.
23. Roes SD, Kelle S, Kaandorp TA, Kokocinski T, Poldermans D, Lamb HJ, *et al*. Comparison of myocardial infarct size assessed with contrast-enhanced magnetic resonance imaging and left ventricular function and volumes to predict mortality in patients with healed myocardial infarction. Am J Cardiol 2007;100:930-6.
24. Kwon DH, Halley CM, Carrigan TP, Zysek V, Popovic ZB, Setser R, *et al*. Extent of left ventricular scar predicts outcomes in ischemic cardiomyopathy patients with significantly reduced systolic function: A delayed hyperenhancement cardiac magnetic resonance study. JACC Cardiovasc Imaging 2009;2:34-44.
25. Rogers WJ Jr, Kramer CM, Geskin G, Hu YL, Theobald TM, Vido DA, *et al*. Early contrast-enhanced MRI predicts late functional recovery after reperfused myocardial infarction. Circulation 1999;99:744-50.
26. Ganame J, Messalli G, Dymarkowski S, Rademakers FE, Desmet W, Van de Werf F, *et al*. Impact of myocardial haemorrhage on left ventricular function and remodelling in patients with reperfused acute myocardial infarction. Eur Heart J 2009;30:1440-9.
27. Eitel I, Desch S, Fuernau G, Hildebrand L, Gutberlet M, Schuler G, *et al*. Prognostic significance and determinants of myocardial salvage assessed by cardiovascular magnetic resonance in acute reperfused myocardial infarction. J Am Coll Cardiol 2010;55:2470-9.
28. Choi KM, Kim RJ, Gubernikoff G, Vargas JD, Parker M, Judd RM. Transmural extent of acute myocardial infarction predicts long-term improvement in contractile function. Circulation 2001;104:1101-7.
29. Kwong RY, Chan AK, Brown KA, Chan CW, Reynolds HG, Tsang S, *et al*. Impact of unrecognized myocardial scar detected by cardiac

magnetic resonance imaging on event-free survival in patients presenting with signs or symptoms of coronary artery disease. Circulation 2006;113:2733-43.

30. Beek AM, Kühl HP, Bondarenko O, Twisk JW, Hofman MB, van Dockum WG, *et al*. Delayed contrast-enhanced magnetic resonance imaging for the prediction of regional functional improvement after acute myocardial infarction. J Am Coll Cardiol 2003;42:895-901.
31. Bello D, Fieno DS, Kim RJ, Pereles FS, Passman R, Song G, *et al*. Infarct morphology identifies patients with substrate for sustained ventricular tachycardia. J Am Coll Cardiol 2005;45:1104-8.
32. Kim HW, Farzaneh-Far A, Kim RJ. Cardiovascular magnetic resonance in patients with myocardial infarction: Current and emerging applications. J Am Coll Cardiol 2009;55:1-16.
33. Tsukiji M, Nguyen P, Narayan G, Hellinger J, Chan F, Herfkens R, *et al*. Peri-infarct ischemia determined by cardiovascular magnetic resonance evaluation of myocardial viability and stress perfusion predicts future cardiovascular events in patients with severe ischemic cardiomyopathy. J Cardiovasc Magn Reson 2006;8:773-9.
34. Assomull RG, Prasad SK, Lyne J, Smith G, Burman ED, Khan M, *et al*. Cardiovascular magnetic resonance, fibrosis, and prognosis in dilated cardiomyopathy. J Am Coll Cardiol 2006;48:1977-85.
35. Mahrholdt H, Wagner A, Deluigi CC, Kispert E, Hager S, Meinhardt G, *et al*. Presentation, patterns of myocardial damage, and clinical course of viral myocarditis. Circulation 2006;114:1581-90.
36. Smedema JP, Snoep G, van Kroonenburgh MP, van Geuns RJ, Dassen WR, Gorgels AP, *et al*. Evaluation of the accuracy of gadolinium-enhanced cardiovascular magnetic resonance in the diagnosis of cardiac sarcoidosis. J Am Coll Cardiol 2005;45:1683-90.
37. Adabag AS, Maron BJ, Appelbaum E, Harrigan CJ, Buros JL, Gibson CM, *et al*. Occurrence and frequency of arrhythmias in hypertrophic cardiomyopathy in relation to delayed enhancement on cardiovascular magnetic resonance. J Am Coll Cardiol 2008;51:1369-74.
38. Dodd JD, Holmvang G, Hoffmann U, Ferencik M, Abbara S, Brady TJ, *et al*. Quantification of left ventricular noncompaction and trabecular delayed hyperenhancement with cardiac MRI: Correlation with clinical severity. AJR Am J Roentgenol 2007;189:974-80.
39. Moon JC, Sheppard M, Reed E, Lee P, Elliott PM, Pennell DJ. The histological basis of late gadolinium enhancement cardiovascular magnetic resonance in a patient with Anderson-Fabry disease. J Cardiovasc Magn Reson 2006;8:479-82.
40. Austin BA, Tang WH, Rodriguez ER, Tan C, Flamm SD, Taylor DO, *et al*. Delayed hyper-enhancement magnetic resonance imaging provides incremental diagnostic and prognostic utility in suspected cardiac amyloidosis. JACC Cardiovasc Imaging 2009;2:1369-77.
41. Kirk P, Roughton M, Porter JB, Walker JM, Tanner MA, Patel J, *et al*. Cardiac T2* magnetic resonance for prediction of cardiac complications in thalassemia major. J Cardiovasc Magn Reson 2009;11:2.
42. Nucifora G, Aquaro GD, Pingitore A, Masci PG, Lombardi M. Myocardial fibrosis in isolated left ventricular non-compaction and its relation to disease severity. Eur J Heart Fail 2011;13:170-6.
43. Kim RJ, Wu E, Rafael A, Chen EL, Parker MA, Simonetti O, *et al*. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. N Engl J Med 2000;343:1445-53.
44. Bleeker GB, Kaandorp TA, Lamb HJ, Boersma E, Steendijk P, de Roos A, *et al*. Effect of posterolateral scar tissue on clinical and echocardiographic improvement after cardiac resynchronization therapy. Circulation 2006;113:969-76.

Cite this article as: Rajiah P. Magnetic resonance imaging in the evaluation of congestive cardiac failure. Indian J Radiol Imaging 2012;22:170-7.

Source of Support: Nil, **Conflict of Interest:** None declared.