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Classic Images in Cardiac Magnetic Resonance Imaging: A Case-based Atlas Highlighting Current Applications of Cardiac Magnetic Resonance Imaging

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Abstract

There have been tremendous technological advances in noninvasive cardiovascular imaging, offering the clinician unparalleled information from a variety of modalities. Cardiovascular magnetic resonance imaging (MRI) has the advantages of superior spatial resolution, detailed tissue characterization, and accurate quantitative assessment of cardiac structure and function, without the need for radiation exposure. Recent advances in image acquisition and postimage processing have led to clinically validated protocols for myocardial perfusion, late gadolinium enhancement, and coronary angiography. The following collection of images was selected to demonstrate the typical appearance of various cardiovascular conditions using MRI. There is, of course, much heterogeneity in both the phenotypic severity of a given condition as well as its appearance on MRI. This article, while not intended to be a comprehensive collection, aims to serve as an introduction to the current applications of cardiac MRI.

Case 1: Myocardial Ischemia

A 62-year-old woman with hypertension, diabetes, and chronic renal insufficiency was admitted with unstable angina. Given her inability to perform exercise, adenosine stress cardiac magnetic resonance imaging (MRI) was performed to evaluate for myocardial ischemia. Fig 1A and B are late gadolinium enhancement (LGE) images of the short-axis location at mid and distal levels, respectively, indicating no evidence of myocardial infarction. Fig 1C and D are perfusion images during adenosine stress matching in location to the late gadolinium images. These perfusion images demonstrated perfusion defects involving the entire inferior wall, mid-inferoseptal, and mid-inferolateral walls (white arrows; Fig 1C and D). There were no perfusion defects visualized during resting first-pass perfusion (not shown). These findings are consistent with significant reversible ischemia in the posterior descending artery and the left circumflex territory. The patient was subsequently referred for cardiac catheterization, which revealed a 95% thrombotic, mid-RCA occlusion and severe left circumflex stenosis, which was successfully treated with percutaneous intervention.

Case 2: Myocardial Infarction

A 49-year-old man with a history of myocardial infarction and prior percutaneous interventions was admitted with a ST elevation myocardial infarction. Cardiac MRI revealed significant late gadolinium enhancement in the anterolateral wall. Fig 2A indicates a postcontrast late gadolinium enhancement image in the mid short-axis location, where the white arrow points to the small anterolateral infarction. Fig 2B is a T2-weighted image for

myocardial edema. There was a corresponding increase in T2 intensity (Fig 2B; white arrows) consistent with residual transmural myocardial edema because of the recent ischemic event. In addition, there was another region of subendocardial infarction involving the anteroseptal wall that did not demonstrate increase in T2 signal, representing a chronic myocardial infarction (black arrow).

Case 3: Right Ventricular Myocardial Infarction

A 49-year-old man with no significant past medical history presented with new onset congestive heart failure in the setting of a possible viral syndrome. Echocardiography revealed significant left ventricle (LV) systolic dysfunction and his electrocardiogram was consistent with prior inferior myocardial infarction. Cardiac MRI was performed to determine the etiology of his cardiomyopathy and revealed severe systolic dysfunction (ejection fraction = 23%) with a thin, aneurysmal inferior wall. There was full-thickness late gadolinium enhancement of the entire inferior wall, basal to mid-inferoseptal and inferolateral walls, and right ventricle. These findings were consistent with a prior extensive inferior and right ventricular infarction. There was also an area of hypo-enhancement consistent with laminated thrombus involving the basal to mid inferior wall aneurysm. Fig 3A demonstrates cine function imaging (diastolic frame only). The inferior wall was thinned and akinetic. The black arrows demonstrate an aneurysm of the inferior, inferoseptal, and inferolateral walls. Fig 3B demonstrates the late enhancement image of the matching short-axis location. The white arrows illustrate extensive late enhancement of the right ventricle, consistent with right ventricular infarction. In addition, there is a laminated thrombus denoted by the asterisks.

Case 4: Left Ventricular Aneurysm

A 52-year-old man with a history of a large inferior ST elevation myocardial infarction was noted to have a rapidly enlarging LV on serial echocardiograms. He presented with intermittent nausea and there was uncertainty as to whether the distorted ventricular anatomy represented an enlarging aneurysm versus a pseudoaneurysm. Cardiac MRI revealed a large basal inferior and inferolateral dyskinetic region. This region demonstrated a “wide-neck” morphology (asterisks; Fig 4A and C) with evidence of full-thickness infarction on late gadolinium enhancement imaging (Fig 4D). These findings are diagnostic of a ventricular aneurysm secondary to a full-thickness myocardial infarction, rather than a pseudoaneurysm from contained ventricular rupture. In addition, cardiac MRI detected multiple thrombi within the aneurysm (arrow; Fig 4B) undetected by echocardiography. The patient underwent operative exploration, which confirmed a true left ventricular aneurysm with associated thrombus, and was treated with aneurysm resection and patch repair.

Case 5: Myocardial Viability

A 56-year-old man with a remote anterior myocardial infarction and significant LV dysfunction had an occluded left anterior descending artery (LAD) on recent cardiac catheterization. He was referred for consideration of coronary artery bypass graft (CABG) and aneurysmectomy. Cardiac MRI revealed evidence of a proximal LAD territory infarct with 50% thickness transmural infarction in the mid anterior wall and anteroseptum, > 75% thickness transmural infarction in the distal anterior and septal segments, and full-thickness infarction at the apex. These findings indicated nonviability of the mid-to-distal LAD territory. In addition there was evidence of a very small apical LV thrombus (black arrow; Fig 5B) and severe systolic dysfunction with left ventricular ejection fraction (LVEF) quantitated at 23%. Cardiac positron emission tomography (PET) with fluorodeoxyglucose demonstrated nonviable myocardium in the mid to distal anterior wall, mid to distal anteroseptum, and the entire apex.

Case 6: Arrhythmogenic Right Ventricular Cardiomyopathy

A 37-year-old man with no significant past medical history developed ventricular tachycardia that was initially managed with sotalol. Coronary angiography showed normal coronary arteries. He presented with recurrent ventricular tachycardia. Cardiac MRI cine imaging revealed a moderately dilated right ventricle (RV) with akinesis of the basal to mid RV free wall and evidence of fatty infiltration (arrows; Fig 6A and B). The patient underwent radiofrequency ablation of the right ventricle but developed recurrent ventricular tachycardia requiring placement of an automated internal cardiac defibrillator.

Case 7: Left Ventricular Noncompaction

A 58-year-old man with a history of a repaired atrial septal defect developed decompensated heart failure in the setting of atrial flutter. Echocardiography demonstrated mild LV hypertrophy, more prominent at the midventricle and apex. Cardiac MRI revealed normal left ventricular size and mild systolic dysfunction (LVEF quantitated at 53%). There was an extensive sponge-like appearance to the LV and RV myocardium involving the anterior, lateral, and apical walls, consistent with ventricular noncompaction (asterisks; Fig 7A and C). The ratio between the noncompacted and compacted myocardium is 9 for the apex and 3 for the lateral wall, values that meet diagnostic criteria for noncompaction.

Case 8: Cardiac Hemochromatosis

An 80-year-old man with recently diagnosed hemochromatosis presented with worsening lower extremity edema and dyspnea on exertion. Cardiac MRI revealed moderate biventricular dysfunction (LVEF = 32%) with biatrial enlargement.

T2* imaging is a method to quantify iron content in myocardium. Myocardial T2* is defined as the exponential rate of signal decay acquired during progressive lengthening of the echo time (TE). Illustrated in Fig 8A–D, as tissue iron content increases from iron-loading conditions, the signal intensities confined by the region of interests (indicated by the red circles) progressively decrease. As shown in Fig 8E, this rate of signal decay as the TE lengthens is quantified by the T2* value, which is a specific parameter of the myocardium. While the normal myocardial T2* value is at 48 ± 6 ms, it has been reported in patients with cardiomyopathy that a T2* value of < 20 ms is a strong indication of iron-overloading, as the etiology of the cardiomyopathy. Here the T2* value was measured at 2.56 ms. Fig 8F and G are cine images (diastolic and systolic frames, respectively) demonstrating moderate biventricular systolic dysfunction. The RV is markedly dilated leading with significant tricuspid regurgitation.

Case 9: Hypertrophic Cardiomyopathy

A 34-year-old woman with a history of a childhood murmur and left ventricular hypertrophy presented with palpitations associated and lightheadedness. Cardiac MRI revealed hyperdynamic left ventricular systolic function with a LVEF quantitated at 67%. The basal anteroseptum was markedly thickened and measured 21 mm; the basal inferolateral wall measured 13 mm (ratio = 1.6). There was extensive, heterogeneous epicardial and myocardial late gadolinium enhancement involving the anterior wall and the entire septum. There was no evidence of systolic anterior motion of the mitral valve leaflet and no evidence by phase-contrast of left ventricular outflow tract obstruction. These data are diagnostic of hypertrophic cardiomyopathy. Fig 9A shows a 4-chamber long-axis cine image in diastole frame demonstrating significant asymmetric septal hypertrophy (arrow). Fig 9B shows the matching postcontrast image demonstrating diffuse, heterogeneous late gadolinium enhancement of the septum (arrows). Fig 9C is a 3-chamber long-axis diastolic cine image

that shows that the hypertrophic segment also involves the basal anteroseptal wall (arrow). Fig 9D shows the matching postcontrast 3-chamber long-axis view, which demonstrates heterogeneous late gadolinium enhancement of the anteroseptal wall, consistent with myocardial fibrosis (arrows).

Case 10: Cardiac Sarcoidosis

A 43-year-old man with no prior past medical history presented with palpitations and a junctional rhythm with frequent premature ventricular contractions on a resting electrocardiogram. Cardiac MRI revealed mildly reduced LV systolic function with akinesis of the basal and mid inferior and inferolateral walls associated with midmyocardial and epicardial late gadolinium enhancement (arrows; Fig 10A–D). In Fig 10B, apart from the inferolateral midwall enhancement (bottom arrows), there was a second focus of late gadolinium enhancement seen in the basal septum (top arrow). This pattern of midmyocardial and epicardial late enhancement was highly suspicious for an infiltrative process such as sarcoidosis. A cardiac PET study confirmed increased fluorodeoxyglucose uptake in these territories, consistent with myocardial inflammation. Mediastinal lymph node biopsy confirmed granulomatous disease consistent with sarcoidosis.

Case 11: Cardiac Amyloidosis

An 82-year-old man with a history of hypertension, diabetes, and chronic renal insufficiency presented with a 10-year history of progressive dyspnea on exertion. Cardiac MRI revealed left ventricular hypertrophy with severe biventricular systolic dysfunction (Fig 11A; 4-chamber long-axis cine systolic image). Matching late gadolinium enhancement showed rapid washout of the contrast agent from the blood pool with diffuse intramyocardial enhancement of the septum, subendocardial enhancement of the inferior wall, and diffuse atrial enhancement (black arrows; Fig 11B). Fig 11C shows 2-chamber systolic long-axis cine image demonstrating reduced global left ventricular function. Matching postcontrast image demonstrates anterior and inferior late gadolinium enhancement, top and bottom arrow, respectively (Fig 11D).

Case 12: Right Atrial Thrombus

A 64-year-old woman with an indwelling Hickman catheter for total parenteral nutrition administration presented with bacteremia with an echocardiogram showing a right atrial mass after the catheter was removed. Cardiac MRI revealed an 18 × 12 mm ovoid mass within the right atrium adjacent to the confluence with the inferior vena cava. The mass did not show first-pass gadolinium perfusion, was hypointense on T2-weighted images consistent with lack of fluid content, and did not demonstrate late gadolinium enhancement. These findings are consistent with a thrombus (Fig 12).

Case 13: Metastatic Sarcoma

A 63-year-old man with a history of advanced gastrointestinal stromal cell sarcoma presented with 2 months of chest pain, palpitations, and shortness of breath. Cardiac MRI revealed a broad-based mass attached to the midinferior wall at the level of the posteromedial papillary muscle (asterisk; Fig 13A) with a large mobile component that prolapsed into the left ventricular outflow tract and across the aortic valve during systole (asterisk; Fig 13B). The mass was highly vascular as shown by the increased signal intensity after the administration of gadolinium contrast (number sign; Fig 13C, precontrast; Fig 13D, postcontrast). The patient underwent surgical resection of the mass, which was confirmed on pathology to be metastatic sarcoma.

Case 14: Metastatic Melanoma

A 68-year-old man with a history of Stage IV melanoma with pulmonary metastases presented with a suspicious lesion on a chest computed tomographic scan. Cardiac MRI revealed a 2.4×2.2 cm mass in the basal anterior wall of the left ventricle, which was isointense on T1-weighted images (Fig 14A), hyperintense on T2-weighted images (Fig 14B), with peripheral late gadolinium enhancement suggestive of tumor necrosis (Fig 14C). These findings are consistent with a focal metastasis of his melanoma to the LV.

Case 15: Common Origin of the Coronary Arteries

A 60-year-old man with a history of hypertension and hyperlipidemia presented with symptomatic exercise-induced ventricular tachycardia. Cardiac MRI revealed structurally normal left and right ventricle with no valvular heart disease. Free-breathing, navigation-guided, contrast-enhanced coronary magnetic resonance angiography (MRA) revealed a common origin of the left anterior descending, left circumflex, and right coronary arteries arising from the right coronary cusp. The LAD followed an anterior course relative to the pulmonary artery and the circumflex followed a posterior course to the aorta. Exercise testing with perfusion imaging demonstrated no evidence of ischemia. Invasive electrophysiologic testing mapped the ventricular tachycardia to the right ventricular outflow tract. Because of the proximity to the LAD, endocardial ablation was not performed and an automated internal cardiac defibrillator was implanted (Fig 15).

Case 16: Myocarditis

A 26-year-old man with no significant past medical history presented with chest pain, fever, and dyspnea on exertion. His laboratory evaluation was notable for elevated cardiac enzymes, elevated liver function tests, and pancytopenia. Cardiac MRI revealed normal LV size and systolic function, with a diffuse, patchy pattern of epicardial and midmyocardial late gadolinium enhancement (arrows; Fig 16A and B) sparing the basal anterior, basal anteroseptal, and basal lateral walls. These findings are consistent with myocarditis. The patient underwent a right ventricular endomyocardial biopsy that was inconclusive, a bone marrow biopsy that was normal, and a liver biopsy that showed evidence of hepatitis with PCR positive for Epstein-Barr virus. His clinical presentation was attributed to Epstein-Barr virus infection; he was managed with supportive care, and on subsequent follow-up had resolution of his symptoms.

Case 17: Pericarditis

A 58-year-old woman with a history of lupus presented with shortness of breath and chest pain. Cardiac MRI revealed a mild increase in pericardial thickness with diffuse late gadolinium enhancement indicative of active and diffuse pericardial inflammation (arrows; Fig 17A and C) without significant pericardial effusion (Fig 17B and D). There was normal left and right ventricular size and function (Fig 17B and D) with no evidence of myocardial involvement.

Case 18: Pericardial Effusion

A 24-year-old woman with Behçet's syndrome and myelodysplastic syndrome status post recent bone marrow transplant presented with dyspnea, fatigue, and lower extremity swelling. Cardiac MRI revealed normal ventricular function (Fig 18A demonstrates a diastolic frame of the cine long-axis view, and is compared to Fig 18B, which represents the systolic frame of the same cine long-axis view) and a large, circumferential pericardial effusion measuring 1.8 cm in its largest dimension (asterisks; Fig 18A and B). There was

mild, early, diastolic inversion of the right atrium suggestive of elevated intrapericardial pressure (arrow; Fig 18C) with no evidence of tamponade physiology (no evidence of diastolic collapse of the free wall of the right ventricle or early systolic compression of the right atrium). The homogenous appearance of the pericardial effusion (asterisks) and hypointense appearance on T1-weighted images (not shown) are consistent with a transudative effusion.

Case 19: Aortic Dissection

A 48-year-old woman with hypertension and a family history of aortic dissection presented with sudden onset of severe chest pain radiating to the back. The initial MRA revealed a Type B aortic dissection originating distal to the origin of the left subclavian artery and extending to the common iliac arteries. After several days of medical therapy in the intensive care unit, she developed recurrent back pain. A repeat MRA revealed rapid, aneurysmal dilation of the proximal descending thoracic aorta (Fig 19). The patient subsequently underwent replacement of the descending thoracic aorta with a synthetic graft.

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FIG 1. Myocardial ischemia is shown. (A) Short axis, mid-ventricle, LGE; demonstrating no evidence of prior infarction. (B) Short axis, distal ventricle, LGE. (C,D) First pass gadolinium perfusion during vasodilator stress demonstrating a perfusion defect involving the mid to distal inferior, mid-inferoseptal, and mid-inferolateral walls (arrows).

FIG 2.

Myocardial infarction is shown. (A) Short axis, mid-ventricle, LGE; anterolateral infarction (white arrow), subendocardial anteroseptal infarction (black arrow) (B) Short axis, mid-ventricle, T2 weighted fast spin echo imaging with fat suppression; increased T2 intensity consistent with myocardial edema (white arrow).

FIG 3.

Right ventricular myocardial infarction is shown. (A) short axis, cine steady-state free precession (SSFP); demonstrating an aneurysm of the inferior, inferoseptum and inferolateral walls (black arrows). (B) short axis, LGE; demonstrating extensive late enhancement of the right ventricle consistent with infarction (white arrows), asterisks represent a laminated thrombus.

FIG 4.

Left ventricular aneurysm is shown. (A) Short-axis, cine steady-state free precession (SSFP); demonstrating large aneurysm neck. (B) Short-axis, LGE, demonstrating full-thickness myocardial infarction with a small thrombus (arrow). (C) Two-chamber, cine SSFP; demonstrating basal inferior aneurysm with a wide neck (asterisks). (D) Two-chamber, LGE; demonstrating a full-thickness myocardial infarction with a rim of scar tissue appearing hyperintense, forming the wall of the aneurysm sac (arrow).

FIG 5. Myocardial viability is shown. (A) 3 chamber, LGE; demonstrating extent of infarction (white arrows). (B) 2 chamber, LGE; demonstrating apical LV thrombus (black arrow). (C) 4 chamber, LGE. (D) short axis, LGE.

FIG 6.

Arrhythmogenic right ventricular cardiomyopathy is shown. (A) 4 chamber, diastole, cine SSFP; demonstrating fatty infiltration of the RV free wall (white arrows). (B) 4 chamber, systole, cine SSFP; demonstrating akinesis of basal to mid RV free wall.

FIG 7. Left ventricular noncompaction is shown. (A) 3 chamber, diastole, cine SSFP. (B) 3 chamber, systole, cine SSFP. (C) 4 chamber, diastole, cine SSFP. (D) 4 chamber, systole, cine SSFP; sponge-like appearance of LV, RV (asterisks).

FIG 8.

Cardiac hemochromatosis is shown. (A–D) Measurement of myocardial T2* using gradient echo sequence with multiple TE times. A region of interest is defined. (E) Myocardial T2* value derived from exponential decay curve. (F) 4 chamber, diastole, cine SSFP; demonstrating biatrial and biventricular dilatation. (G) 4 chamber, systole, cine SSFP; demonstrating moderate biventricular systolic dysfunction, with significant tricuspid regurgitation.

FIG 9.

Hypertrophic cardiomyopathy is shown. (A) 4 chamber, diastole, cine SSFP; demonstrating significant asymmetric septal hypertrophy. (B) 4 chamber, diastole, LGE; demonstrating diffuse, heterogenous late gadolinium enhancement of the septum. (C) 3 chamber, diastole, cine SSFP. (D) 3 chamber, diastole, LGE; demonstrating heterogenous late gadolinium enhancement of the anteroseptum.

FIG 10.

Cardiac sarcoidosis is shown. (A) 4 chamber, LGE; mid-wall and epicardial late enhancement. (B) 3 chamber, LGE; inferolateral midwall enhancement (bottom arrows), basal septal enhancement. (C) 2 chamber, LGE; basal inferior mid-wall and epicardial enhancement. (D) short axis, LGE; mid inferoseptal, inferior, and inferolateral mid myocardial and epicardial late enhancement.

FIG 11.

Cardiac amyloidosis is shown. (A) 4 chamber, cine SSFP, systole; demonstrating severe biventricular systolic dysfunction. (B) 4 chamber, LGE; demonstrating diffuse atrial enhancement (black arrows), basal septal and lateral enhancement (white arrows). (C) 2 chamber, cine SSFP, systole. (D) 2 chamber, LGE; demonstrating diffuse basal and mid anterior late enhancement, basal inferior sub-endocardial late enhancement (white arrows).

FIG 12.

Right atrial thrombus is shown. (A) 4 chamber, cine SSFP; demonstrating a mass within the right atrium with no involvement of the tricuspid valve or interatrial septum. (B) 4 chamber, MDE; demonstrating no evidence of late gadolinium enhancement. (C) Axial, cine SSFP; demonstrating a mass within the right atrium adjacent to the entrance of the inferior vena cava. (D) Axial, LGE; demonstrating no evidence of late gadolinium enhancement of the mass.

FIG 13. Metastatic sarcoma is shown. (A) 3 chamber, diastole, gradient recalled echo (GRE). (B) 3 chamber, systole, GRE; demonstrating mobile component of mass prolapsing across the aortic valve (asterisk). (C) Short axis, cine SSFP, pre-contrast. (D) Short axis, cine SSFP, post-gadolinium contrast; demonstrating increased signal intensity of the mass (#).

FIG 14.

Metastatic melanoma is shown. (A) Short-axis, midventricle, T1-weighted double-inversion recovery fast spin-echo imaging; demonstrating isointense signal of the mass. (B) Short-axis, T2-weighted image with fat saturation; demonstrating hyperintense signal of the mass consistent with edema or inflammation. (C) Short-axis, LGE; demonstrating enhancement of the periphery of the mass suggestive of necrosis. The dark central core of the mass on delayed enhancement image is also suggestive of central hemorrhage. (D) Two-chamber, cine SSFP.

FIG 15. Common origin of the coronary arteries is shown. 3D volume reconstruction of gadolinium-enhanced coronary MRA. AO, aorta; RCA, right coronary artery; LA, left atrium; LCX, left circumflex.

FIG 16. Myocarditis is shown. (A) 4 chamber, MDE; arrows demonstrating patchy, diffuse epicardial and LGE; midmyocardial late gadolinium enhancement (white arrows). (B) 2 chamber, LGE.

FIG 17. Pericarditis is shown. (A) Short axis, LGE; demonstrating diffuse pericardial late gadolinium enhancement consistent with inflammation. (B) Short axis, cine SSFP. (C) 3 chamber, LGE; demonstrating diffuse pericardial enhancement. (D) 3 chamber, cine SSFP.

FIG 18.

Pericardial effusion is shown. (A) 3 chamber, diastole, cine SSFP; large pericardial effusion (asterisk). (B) 3 chamber, systole, cine SSFP. (C) 4 chamber, diastole, cine SSFP; demonstrating large effusion with early diastolic inversion of the right atrium (arrow). (D) 4 chamber, systole, cine SSFP.

FIG 19. Aortic dissection is shown. Gadolinium-enhanced MRA, sagittal oblique view. Type B dissection with a dissection flap (white arrow), focal descending thoracic aneurysm (gray arrow), and layering thrombus within the false lumen (black arrow).