

Utility of magnetic resonance imaging in the evaluation of patients with ST segment elevation on an electrocardiogram

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Abstract

ST segment elevation is an important electrocardiographic (ECG) change that is typically found in acute myocardial infarction, but may also be seen in a variety of other conditions. MRI plays an important role in the evaluation of these patients. MRI not only establishes the diagnosis, which is essential for appropriate management, but also helps in the assessment of other factors that are important for risk stratification. In this review, we discuss the common and uncommon causes of ST segment elevation and the role of MRI in the evaluation of these disease processes.

Key words: Cardiomyopathies; magnetic resonance imaging; scar; viability

Introduction

ST segment elevation is an important electrocardiographic (ECG) finding that, in a patient with acute chest pain and elevated cardiac enzymes, typically indicates acute myocardial infarction (MI). However, ST segment elevation can also be caused by a variety of other conditions [Table 1]. Of the patients who present with acute chest pain and ST segment elevation, 51–85% have causes other than MI. Mild ST segment elevation can also be a normal finding, particularly in males of age group 17–24 years.^[1-3]

Since the treatment strategies for these various conditions are different, it is important to make an accurate diagnosis based on clinical examination and various diagnostic tests.

Acute ST segment elevation MI (STEMI) warrants coronary angiography and immediate reperfusion therapy, which is not indicated in other diseases. Analysis of the shape of the ST segment elevation, the ECG leads involved, associated ECG features, and the clinical features may enable narrowing of the differential diagnosis. MRI is playing an increasingly important role in the assessment of these patients. In a recent study, MRI provided a new diagnosis in 65% of patients with chest pain, elevated cardiac enzymes, and unobstructed coronary arteries and also excluded significant pathologies in the remaining patients. Common causes for ST elevation in this study were myocarditis (31%), Takotsubo cardiomyopathy (31%), and STEMI without an angiographic lesion (29%).^[4]

MRI has the advantages of having good spatial, temporal, and contrast resolutions; wide field of view; and multiplanar reconstruction capabilities, besides being noninvasive. There is no radiation involved with MRI. However, MRI cannot be performed in patients who are anxious and claustrophobic or in patients with contraindications such as implanted metallic or electronic devices. Intravenous gadolinium is not advisable in patients with severe renal dysfunction due to the risk of nephrogenic systemic fibrosis.^[5]

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Table 1: Causes of ST segment elevation in electrocardiography

Acute myocardial infarction
Acute myocarditis
Acute pericarditis
Takotsubo cardiomyopathy
Left ventricular hypertrophy
Left ventricular aneurysm
Sarcoidosis
Acute aortic dissection
Pulmonary embolism
Arrhythmogenic right ventricular dysplasia
Brugada syndrome
Left bundle branch block
Hyperkalemia
Post electrical cardioversion
Ventricular paced rhythm
Prinzmetal's angina
Benign early repolarization (normal variant)
Osborn wave hypothermia
Acute cerebral hemorrhage
Normal variant

MRI Protocol

A combination of various MRI sequences is used in the evaluation of patients with chest pain and ST segment elevation. A standardized protocol for the evaluation of such patients is listed in Table 2. Sequences can be added or modified depending on the clinical scenario and the individual patient characteristics (ability to breath-hold, presence of arrhythmia, presence of implanted metal device, etc.).

- Scout images:* These are acquired in the axial, coronal, and sagittal planes. In addition to their value in planning for subsequent views, scout images are also used to ensure that the heart is positioned in the isocenter of the coil.
- Axial fast spin-echo with half-Fourier acquisition (HASTE):* This is performed with ECG gating but without breath-hold. This sequence is used for planning subsequent cardiac views as well as for detecting gross abnormalities in the lung and mediastinum.
- Cine imaging:* Steady-state free-precession (SSFP) sequence is the workhorse of cardiac MRI due to its high signal-to-noise and contrast-to-noise ratios. In addition to allowing qualitative assessment of morphology and ventricular contractility, quantitative measurement of ventricular volumes, mass, and ejection fraction can also be done. Regional function can be estimated either visually or by using more accurate myocardial tagging techniques, such as spatial modulation of magnetization (SPAMM) or complementary spatial modulation of magnetization (CSPAMM).
- T2W black blood imaging:* T2W black blood images are

Table 2: MRI imaging protocol in the evaluation of acute chest pain

Sequence	Planes	Utility
Scouts	Axial, sagittal, coronal	Localizer Coil position
HASTE/SSFSE black blood	Axial stacks	Overview Planning for cardiac views
Cine SSFP	VLA, four-chamber, three-chamber, short-axis stacks	Morphology Volumes Function (global, regional)
T2W imaging T2 FSE/ T2 FSE STIR/ T2 prep SSFP	VLA, four-chamber, three-chamber, short-axis	Morphology Myocardial edema Pericardial effusion/ thickening
Flow quantification	Ascending aorta Pulmonary artery	Quantification of regurgitation/stenosis Estimation of shunt volume
Delayed enhancement 2D/ 3D/ PSIR/ Single shot	VLA, three-chamber, four-chamber, short-axis stacks	Diagnosis of MI and size Myocardial viability Scar pattern in nonischemic Thrombus Pericardial enhancement
Perfusion imaging Stress Rest	Three short-axis slices	Identification of ischemia/ infarct Post-MI risk stratification

HASTE: Half-Fourier acquisition in steady state, SSFSE: Single-shot fast spin-echo, VLA: Vertical long axis, HLA: Horizontal long axis, FSE: Fast spin-echo, SSFP: Steady-state free precession, PSIR: Phase-sensitive inversion-recovery, STIR: Short tau inversion recovery

- Perfusion imaging:* Myocardial perfusion imaging using MRI is a sensitive and accurate technique for detecting myocardial ischemia,^[7] which is important in risk stratification. Multislice T1W 2D sequences, either SSFP, fast low-angle shot (FLASH), or gradient-recalled echo echoplanar imaging (GRE EPI) are acquired during the first pass of gadolinium through the heart after intravenous administration at high flow rates (4–6 ml/s), both at rest and after pharmacological stress with adenosine or dipyridamole.^[8] Due to the T1 shortening induced by contrast, normally perfused myocardium appears bright and hypoperfused areas appear dark. Whereas in ischemia, the perfusion defect is seen only at stress, in infarct and microvascular obstruction, it is seen both at rest and stress. Perfusion imaging

without intravenous contrast can be performed using endogenous contrast with techniques such as blood oxygen level-dependent (BOLD) or arterial spin labeling (ASL).^[9]

F. *Delayed contrast enhancement*: Delayed enhancement sequence (DE-MRI) is the most accurate method for the diagnosis and characterization of scar, both in ischemic and non-ischemic cardiomyopathies. It is acquired 10–20 min after administration of gadolinium chelate (0.1–0.2 mmol/kg), using a segmented *k*-space inversion-recovery gradient-echo pulse sequence, where the inversion time is set to null signal from normal myocardium. The gadolinium chelate is biologically inert and passively diffuses into the myocardial extracellular space and has a half-life of approximately 20 min. Hyper-enhancement is seen in the absence of viable myocytes that exclude gadolinium, which results in increased volume of distribution of gadolinium and rapid wash-in and wash-out kinetics. In acute myocardial injury, this happens due to rupture of cells with resulting intracellular entry of contrast, whereas in chronic myocardial diseases, this happens due to expansion of extracellular volume in scar or fibrosis.

With optimal nulling time, the hyperenhanced area stands out against the dark signal of the nulled normal myocardium.^[10] The optimal inversion time can be selected by using a Look-Locker sequence, which is a T1 scout performed at different inversion times. Delayed enhancement images can be acquired using 2D, 3D, single-shot, or phase-sensitive inversion-recovery (PSIR) techniques. In the 2D technique, each slice is acquired during a single breath-hold, with data acquired at either every heartbeat or at every other heartbeat. In the 3D technique, the entire left ventricle can be covered in a single breath-hold or during free breathing, using navigator gating of the diaphragmatic movements. Single-shot sequences acquire each image slice within a single heart beat, which is useful in patients with arrhythmia or breathing difficulties. The PSIR sequence eliminates the need for selecting the optimal inversion time.

G. *Velocity-encoded imaging*: Velocity-encoded phase contrast images are acquired perpendicular to the level of the mid-ascending aorta and the pulmonary artery. This enables quantification of forward and reverse flow in these arteries, from which aortic and pulmonary regurgitation volume and fraction and cardiac output can be calculated. Cardiac shunts can also be quantified.

Optional Sequences

- a. *MRI angiography*: MRI angiography (MRA) following contrast administration is performed if there is suspicion of acute aortic dissection or pulmonary embolism. Three-dimensional whole-heart SSFP can be used if there is any contraindication to gadolinium.
- b. *Early post-contrast imaging*: T1W fast spin-echo sequence approximately 15 s after the intravenous injection of gadolinium can be used for detection of early enhancement in the myocardium or pericardium, which is seen in acute myocarditis and pericarditis, respectively.
- c. *Real-time imaging*: Real-time imaging of the ventricular septum in the short axis is used to demonstrate exaggerated respiratory variations of the septal movements in patients with constrictive pericarditis.

Acute Myocardial Infarction

Acute myocardial infarction (MI) is myocardial cell death caused by inadequate supply of oxygenated blood due to thrombotic occlusion of coronary arteries, superimposed on atherosclerotic disease. Occasionally, it can be caused by coronary spasm, coronary embolism, or thrombosis in nonatherosclerotic vessels. In a patient with ischemic symptoms, MI is diagnosed when there are typical rise and fall of cardiac enzymes (troponin or creatine kinase MB fraction) and ECG abnormalities (ST segment elevation or depression, Q waves). Imaging criteria for diagnosis of MI include development of a new regional wall motion abnormality and loss of viable myocardium.^[11]

In MRI, cine imaging shows regional wall motion abnormalities in the involved vascular territory along with global systolic dysfunction. T2W images show wall thickening and high signal due to myocardial edema [Figure 1A]. If the damage is irreversible, delayed enhancement is seen in the myocardium of the affected vascular territory due to increased volume of distribution of gadolinium into the cells as a result of membrane rupture [Figure 1B]. In addition, capillary plugging in the infarct results in altered wash-in and wash-out kinetics, with slow uptake and prolonged retention. Since the infarct progresses like a wave front, subendocardial enhancement is seen initially, which then progresses outward to become transmural in severe cases. Microvascular obstruction is caused by severe sludging and occlusion of end arteries and capillaries and is seen as a dark nonenhancing area within the bright scar [Figure 2]. Perfusion defects are seen on rest perfusion scans [Figure 3]. Stress scans are not typically performed in suspected acute coronary syndrome.

MRI is not routinely used in the evaluation of MI, but comes into play in various scenarios, either after or before coronary angiography has been performed. Ten percent of patients with suspected STEMI do not have a culprit lesion in coronary angiography and 9.5% do not have any significant coronary artery disease. One explanation for the absence of a culprit lesion is that the symptoms are caused by noncoronary artery disease. In this scenario, MRI can be used to establish the alternative diagnosis (see below). A culprit lesion could also be absent if there has

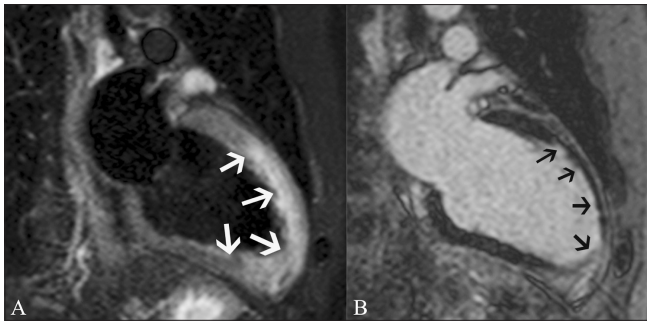


Figure 1 (A, B): Acute myocardial infarction. Two-chamber T2W STIR image (A) in a 34-year-old male with acute chest pain, elevated cardiac enzymes, and ST segment elevation, shows high signal in the mid and apical anterior segments of the left ventricle (arrows) consistent with myocardial edema. Two-chamber phase-sensitive inversion-recovery (PSIR) image (B) in the same patient shows subendocardial scar in the mid and apical anterior segments (arrows). Note that the scarred area is less than the edematous area and this difference is the “area at risk/salvageable myocardium”

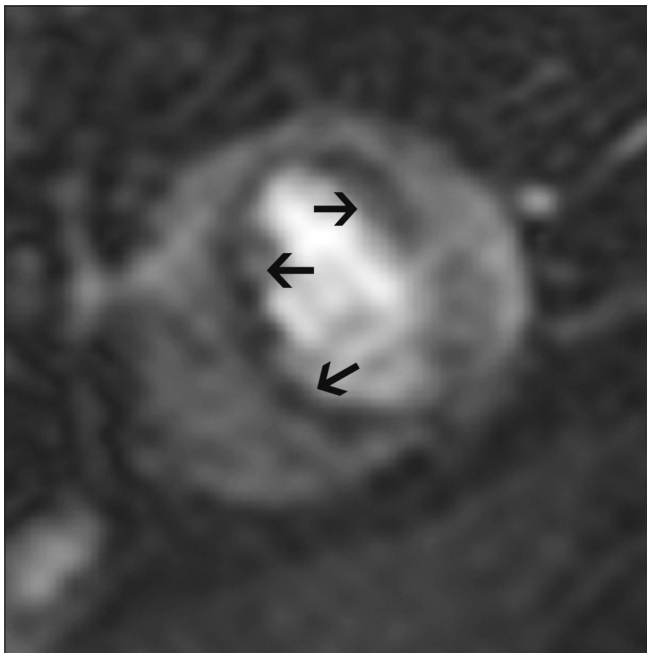


Figure 3: Perfusion defect in myocardial infarction. Short-axis SSFP first-pass perfusion imaging at rest in a 43-year-old male with chest pain shows a perfusion defect (arrows) in the apical anterior, septal, and inferior segments (arrows), consistent with myocardial infarction in left anterior descending (LAD) artery territory

been recanalization of the occlusion by spontaneous lysis or if the occlusion was caused by an embolus or by coronary vasospasm. In this scenario, if MRI detects an ischemic scar, it establishes MI as the cause of symptoms and identifies the involved vessel. Occasionally, multiple vessels are found diseased on coronary angiography and identification of the culprit lesion may be difficult. In this scenario, the vessel supplying the scarred or edematous area in MRI would be the culprit lesion and treatment can be targeted toward this.



Figure 2: Microvascular obstruction. Short-axis PSIR image in a 47-year-old male with acute chest pain and ST segment elevation reveals dark nonenhancing tissue (straight arrow) located within bright enhancing scar (curved arrow), consistent with microvascular obstruction within an area of myocardial infarction in the left circumflex artery territory

MRI can also be used in the diagnosis of MI before performing angiography since it is very sensitive for the detection of small and subendocardial scars.^[11] MRI has been shown to add diagnostic value over and above the standard tests used in acute coronary syndrome (ECG and enzymes), increasing the sensitivity and specificity of diagnosis to 84%.^[12] MRI can be used as a quadruple rule-out study, excluding acute coronary syndrome, acute aortic dissection, acute pulmonary embolism, and acute myocarditis. MRI can also diagnose acute MI even when the first troponin measurement is negative since T2W imaging shows myocardial edema as early as 26 min after occlusion.^[13] MRI is also useful in patients with acute chest pain but with negative ECG or cardiac enzymes.

Various factors determined by MRI enable risk stratification of patients with MI. In the acute stage, the area of myocardial edema is higher than the area of irreversible necrosis shown by delayed enhancement, and the difference is the “area at risk/salvageable area”, i.e., the area that can be salvaged by revascularization. There is an inverse relationship between the transmural extent of MI and recovery of segmental contractile function after revascularization.^[11] Microvascular obstruction indicates severe ischemic disease and is associated with poor prognosis, adverse cardiovascular events, and adverse remodeling. Hemorrhage within the core of the infarct is also associated with adverse left ventricular

remodeling, large infarct size, increased left ventricular end systolic volume, and no improvement of ejection fraction at follow-up. MRI predicts those patients who will benefit from a revascularization procedure by estimating how much of the myocardium is scarred. Myocardial segments with enhancement <25% have good potential for functional recovery after revascularization, segments with >75% enhancement have no potential for functional recovery, and segments with 26–75% enhancement have an intermediate potential for functional recovery. MRI can also help in determining which patients will benefit from cardiac resynchronization therapy, since extensive scar in the posterolateral wall is associated with a poor response. Complications such as free wall rupture, ventricular septal rupture, aneurysm, pericarditis, thrombosis, and mitral regurgitation can be evaluated with MRI.^[11]

Acute Myocarditis

Acute myocarditis is inflammation of the myocardium and may be secondary to viral infection, post-viral immune-mediated reaction, toxins, ischemia, mechanical injury, drugs, transplant rejection, or other immune reactions. It presents with chest pain, occasionally mimicking acute MI or heart failure. Diffuse ST segment elevation, T wave changes, Q waves, atrioventricular and bundle branch block can be seen in the ECG. Cardiac enzymes may be increased. In MRI, global or regional functional abnormality may be seen. Patchy areas of edema can be seen in the T2W images, typically in the subepicardial/mid-myocardial regions [Figure 4A]; less commonly, it may be global. Early contrast enhancement may be seen due to hyperemia and capillary leak. Delayed enhancement is typically seen in the subepicardial or mid-myocardial layers, often in the inferolateral and lateral walls [Figure 4B] and, less commonly, in the septum.^[14] Parvovirus infection typically affects the lateral wall, with recovery in few months, but herpes virus 6 infection affects the septum and progresses toward chronic heart failure.^[15] Pericardial enhancement and pericardial effusion are seen in patients suspected to have acute myocarditis, indicating that myopericarditis is a common presentation.

Acute Pericarditis

Acute pericarditis is acute inflammation of the pericardium, which can be idiopathic or caused by viral infection, trauma, MI, systemic disease, radiation, metastasis, or uremia. It presents with acute chest pain and a pericardial friction rub. Diffuse upright concave ST segment elevation and PR segment depression is seen in all the precordial leads. On MRI, the pericardium is thickened (>4 mm) and effusion may be seen [Figure 5A]. High signal is seen in T2W STIR images due to edema or effusion. Early and delayed contrast enhancement of the pericardium is seen due to inflammation [Figure 5B]. Contrast enhancement

may extend into the epicardial fat and myocardium. Occasionally, features of pericardial constriction are seen transiently. This includes diastolic restraint, abrupt cessation of diastolic filling, tubular deformity of the ventricles, and exaggerated ventricular interdependence, which manifests as exaggerated diastolic septal flattening with inspiration. It can progress to a chronic stage, which could be inflammatory or fibrotic.^[16]

Takotsubo Cardiomyopathy

Takotsubo cardiomyopathy (stress-induced cardiomyopathy, apical ballooning syndrome) is characterized by transient wall motion abnormalities in the apical and mid portions of the left ventricle, with no evidence of significant obstructive coronary disease. It is more commonly seen in postmenopausal women, typically following a stressful event. It is seen in 0.7–2.5% of all patients presenting with acute coronary syndrome. Chest pain and dyspnea following a stress trigger are the common clinical symptoms. ST segment elevation, T wave inversion, and Q waves are seen in the ECG. Mild elevation of cardiac enzymes and catecholamines may be seen. Myocardial edema may be seen in the affected segments [Figure 6A-C], but delayed enhancement is not seen. Akinesis/dyskinesis of the left ventricular apex and mid-ventricular segments (not corresponding to any vascular territory) is seen, along with hyperkinetic basal regions [Figure 6D]. Global left ventricular function is decreased, but recovers after treatment in 95% of cases. In the inverted type, basal and mid-ventricular akinesia, with or without apical hyperkinesis, is seen. In the mid-ventricular type, mid-ventricular akinesia with preservation or hyperkinesis of the basal and apical segments is seen.^[17]

Left Ventricular Hypertrophy

Left ventricular hypertrophy could be a primary genetic process or it may be secondary to hypertension or obstructive lesions such as aortic stenosis. Presence of ST segment changes in left ventricular hypertrophy is an indicator of subendocardial ischemia. Hypertrophic cardiomyopathy (HCM) is a genetic disorder of the cardiac sarcomere, with heterogeneous phenotypical expression. Asymmetric hypertrophy of the basal ventricular septum is the most common type [Figure 7A]; apical, mid-ventricular, symmetrical, helical, mass-like and burnt-out are the less common types. Occasionally, HCM can present acutely with chest pain, dyspnea, and abnormal ECG changes. MRI shows the ventricular hypertrophy, with normal left ventricular volumes and normal or elevated systolic left ventricular function. Systolic gradients in the left ventricular outflow tract (LVOT), systolic anterior motion of the mitral valve, and mitral regurgitation can be seen. Patchy, sand-like, delayed enhancement can be seen in the hypertrophied areas [Figure 7B], particularly in the right

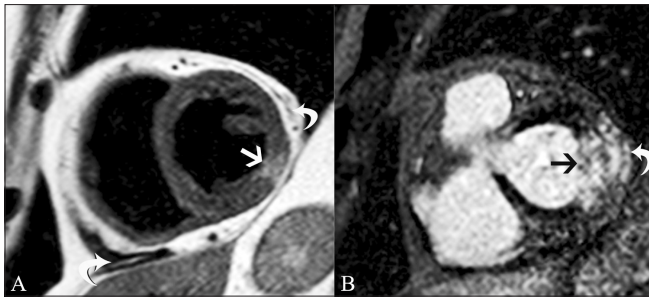


Figure 4 (A, B): Acute myopericarditis. Short-axis T2W double inversion-recovery black blood image (A) in a 43-year-old male with acute chest pain and ST segment elevation shows pericardial thickening on the inferior and lateral aspects (curved arrows) and high signal in the basal lateral myocardium (straight arrow). Short-axis PSIR image (B) shows delayed enhancement in the basal lateral myocardium (straight arrow) and the surrounding pericardium (curved arrow). These findings are consistent with myopericarditis

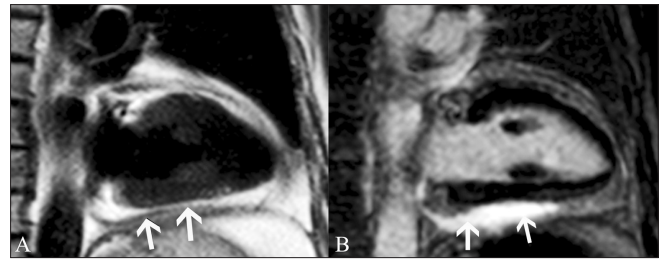


Figure 5 (A, B): Acute pericarditis. Two-chamber T2W image (A) in a 38-year-old female who presented with acute chest pain and ST segment elevation shows thickening of pericardium, which is more prominent in the inferior aspect (arrows). Two-chamber PSIR image (B) in the same patient shows intense enhancement (arrows) of the thickened pericardium adjacent to the left ventricular inferior wall

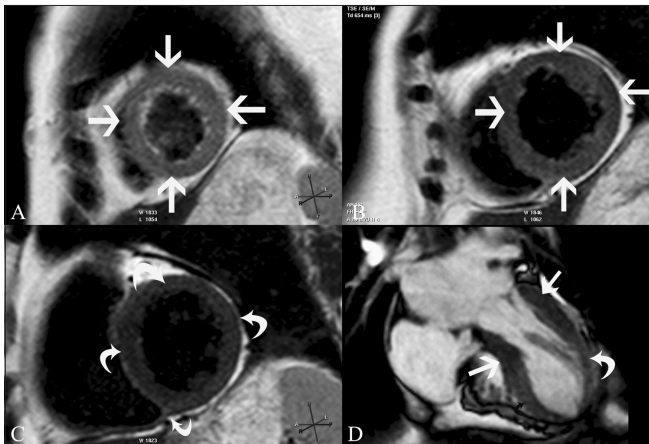


Figure 6 (A-D): Takotsubo cardiomyopathy. Short-axis T2W images (A,B,C) in a 67-year old female patient who presented with acute chest pain and ST segment elevation shows diffuse edema in the mid and apical myocardial segments (straight arrows), but no edema in the basal segments (curved arrows). PSIR images (not shown here) did not show any delayed enhancement. Four-chamber SSFP cine image (D) in the same patient during systole shows normal contraction of the basal segments (straight arrows), but there is no thickening of the mid and apical segments (curved arrows). Coronary angiography was normal in this patient. This is a classic case of Takotsubo cardiomyopathy

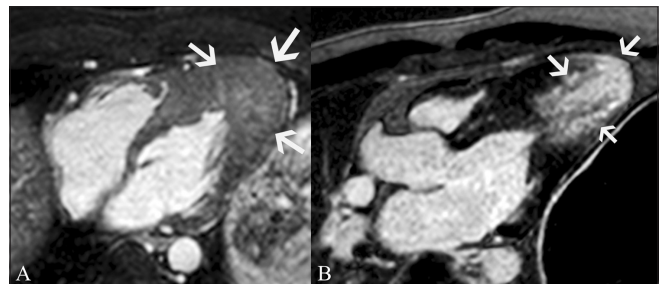


Figure 7 (A, B): Hypertrophic cardiomyopathy. Axial SSFP image (A) in a 33-year-old female with acute chest pain, dyspnea, and ST segment elevation shows severe hypertrophy of the apical and mid-ventricular segments (arrows). Three-chamber PSIR image (B) shows extensive, patchy, mid-myocardial delayed enhancement (arrows) in the apical and mid-ventricular segments, suggestive of interstitial fibrosis in a pattern typical for hypertrophic cardiomyopathy

ventricular insertion sites due to replacement interstitial fibrosis, myofibrillar disarray, abundant connective tissue, or ischemia-related necrosis. Scar is an important prognostic marker since it is a substrate for arrhythmia and is associated with sudden cardiac death and cardiac failure.^[18,19]

Left Ventricular Aneurysm

Left ventricular aneurysm is a cause of ST segment elevation, particularly when the elevation is persistent. Left ventricular aneurysm can be either true or false. A *true aneurysm* is a saccular protrusion of the left ventricular wall due to mechanical weakness, resulting in deficient or absent myocardium with subendocardial

or transmural fibrosis, typically caused by an anterior or apical myocardial infarct, but also by infections, arteritis, Loeffler endocarditis, myocarditis, trauma, or anomalous origin of a coronary artery from the pulmonary artery. In MRI, a true aneurysm has a wide mouth (larger than the size of the aneurysm) and is akinetic or dyskinetic in systole [Figure 8]. Scar tissue produces delayed enhancement and thrombus may be seen. Persistent ST segment elevation is related to a relatively larger extent of transmural necrosis and persistent microvascular damage as seen on MRI.^[20] A *pseudoaneurysm* can be caused by MI, trauma, or infection, which results in myocardial rupture that is contained by the pericardium or scar tissue. It is more common in the inferior and inferolateral walls and has a narrow neck (less than the actual size of the aneurysm). It is dyskinetic and there is a high risk of rupture. Mural thrombus and marked pericardial enhancement are associated findings.^[21]

Sarcoidosis

Sarcoidosis is a multisystemic inflammatory disorder of unknown etiology, characterized pathologically by the presence of noncaseating granulomas. Clinical cardiac involvement is seen in 5% of these patients and typically

implies a poor prognosis. Occasionally it presents with clinical and ECG features simulating acute MI. T2W images show myocardial thickening and edema. Patchy mid-myocardial or subepicardial delayed enhancement due to scarring is seen [Figure 9]. MRI helps in early detection. Transmural enhancement may be seen in the burnt-out phase. Scar implies a poor prognosis. Detection of scar by delayed enhancement MRI is also useful as a guide for endomyocardial biopsy.^[22]

Acute Aortic Dissection

Acute aortic dissection is characterized by separation of the layers of the aorta, which is initiated by a tear in the intimal layer with subsequent entry of blood into the intima-media space. The formation of an exit tear results in true and false lumens. Common causes are hypertension, aneurysm, connective tissue disorders, iatrogenic, aortic valve anomalies, and vasculitis. The Stanford classification divides aortic dissection into "type A", when the ascending aorta is involved, and "type B", when the ascending aorta is not involved. The classic presentation is with a "tearing" type

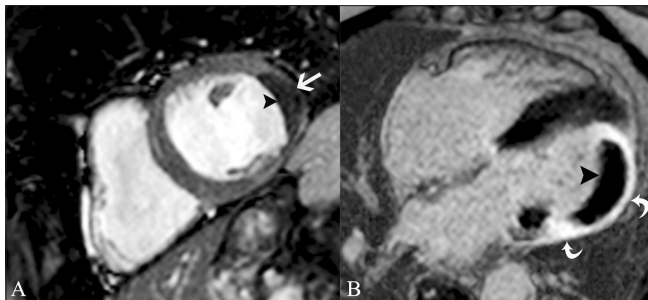


Figure 8 (A, B): Left ventricular aneurysm. Short-axis SSFP image (A) in a 74-year-old patient with acute chest pain and ST segment elevation shows a wide-necked true aneurysm in the lateral wall of the left ventricular (arrow); it contains a dark thrombus (arrowhead). Four-chamber PSIR image (B) of the same patient shows the aneurysm, with severely scarred myocardium of the lateral wall (curved arrow) and a large dark thrombus (arrowhead)

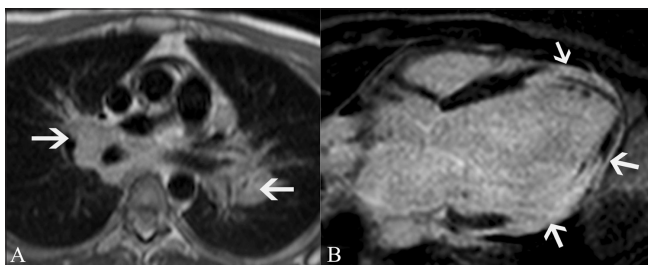


Figure 9 (A, B): Cardiac sarcoidosis. Axial T2 black blood image (A) in a 69-year-old male with history of biopsy-proven systemic sarcoidosis, who presented with acute dyspnea, chest pain, and ST segment elevation; bilateral enlarged hilar lymph nodes (arrows) can be seen. Three-chamber PSIR image (B) in the same patient shows a diffuse near-transmural myocardial scar (arrows) with some sparing of the subendocardial segment. The findings are suggestive of extensive cardiac sarcoidosis, and this was confirmed by endomyocardial biopsy

of acute chest pain that radiates to the back. Occasionally ST segment elevation can be seen in type A dissection due to the involvement or compression of the coronary ostia. Type A dissection requires emergency surgery. Computed tomography (CT) angiography is the most commonly used imaging technique in dissection. MRI has a sensitivity of 98% and a specificity of 100% in the diagnosis of dissection, with the flap being demonstrable in SSFP images and MRA.^[23] Blood products or thrombosis can be seen in the false lumen [Figure 10]. In acute type A dissection, MRI can identify and quantify aortic regurgitation which may determine the type of surgery performed (with or without aortic valve replacement). Involvement of the aortic valve and coronary arteries implies a bad prognosis. MRA is also useful in the evaluation of the branch vessels.^[24]

Acute Pulmonary Embolism

Acute pulmonary embolism is characterized by obstruction of the pulmonary artery or its branches by material (thrombus, tumor, fat, or air) that has originated elsewhere in the body. Pleuritic chest pain and dyspnea are the clinical findings. T wave inversion, S1Q3T3 pattern, right bundle branch block, and sinus tachycardia are the common ECG findings. ST segment elevation can be seen in the anteroseptal and inferior leads due to right ventricular ischemia, dilatation, or overload.^[1] CT pulmonary angiography is the most effective and commonly used diagnostic modality. In MRI, emboli are seen as hypointense filling defects within the pulmonary arteries in MRA or in SSFP images [Figure 11]. Pulmonary MRA has a sensitivity of 78% and specificity of 99%, while a combined pulmonary MRA and extremity MRI venography has a sensitivity of 92% and specificity of 96% in detecting pulmonary embolism in patients with technically adequate images.^[25] Perfusion defects can be seen in the parenchymal phase of the MRA. Features of right ventricular strain such as right ventricular hypertrophy and systolic septal bowing can also be demonstrated on the SSFP images.

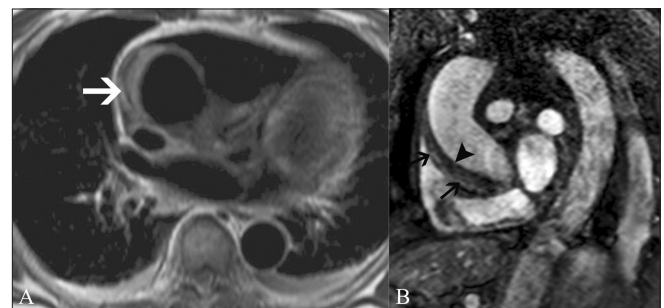


Figure 10 (A, B): Acute aortic dissection. Axial T2W black blood image (A) in a 63-year-old male with acute chest pain and mild ST segment elevation shows a type A ascending aortic dissection, with intermediate-signal-intensity blood products within the false lumen (arrows). Coronal SSFP image (B) in the same patient shows the flap (arrowheads) with blood products in the false lumen (arrows)

Arrhythmogenic Right Ventricular Dysplasia

Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C), characterized by fibrofatty replacement of the right ventricular myocardium, is mostly familial. It presents with ventricular tachycardia, with sudden death being seen in 20% of cases. Diagnosis is established based on the Task Force criteria.^[26,27] Rarely, it may present with acute chest pain and ST segment elevation in the right precordial leads. It is more commonly seen in the anterobasal right ventricular wall and the right ventricular outflow tract. Fat infiltration of the right ventricular myocardium is seen on black blood images, and delayed enhancement is seen if there is scar [Figure 12]. Cine images demonstrate right ventricular dilation, focal aneurysms, and global and regional wall motion abnormalities. Scar correlates with induction of ventricular tachycardia, decreased right ventricular function, and increased right ventricular volume.^[28]

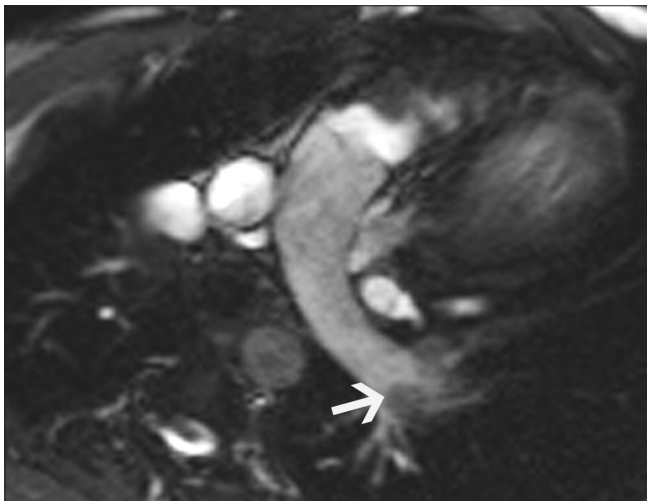


Figure 11: Pulmonary embolism. Axial SSFP image in a 38-year-old female with chest pain, dyspnea, and ST segment elevation shows a hypointense filling defect (arrow) in the left pulmonary artery

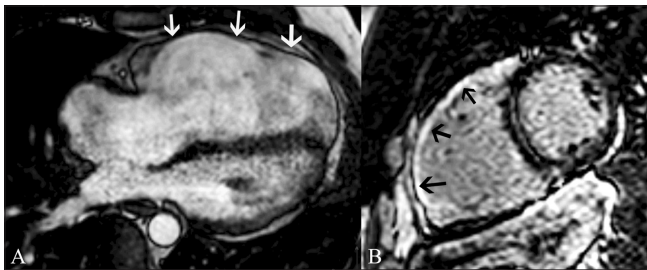


Figure 12 (A, B): Arrhythmogenic right ventricular dysplasia. Horizontal long-axis SSFP image (A) in a 31-year-old female with chest pain, ventricular arrhythmia, and ST segment elevation shows severely dilated right ventricle (arrows). Short-axis PSIR image (B) in the same patient shows diffuse delayed enhancement of the right ventricular free wall (arrows). The wall motion abnormalities along with the scar pattern are consistent with ARVD

Brugada Syndrome

Brugada syndrome is a genetic disease characterized by mutations of the cardiac sodium channel gene, resulting in typical ECG changes and sudden cardiac death due to ventricular fibrillation. It is seen in young adults, more commonly in Southeast Asia, where it underlies the sudden unexpected death syndrome.^[29] Characteristic ECG changes include right bundle branch block with ST segment elevation in the right precordial leads and a normal QT interval in the absence of any structural heart disease. However, recent studies in Brugada syndrome have shown the presence of some abnormalities, including right ventricular dilatation, right ventricular dysfunction, regional wall motion abnormalities, aneurysms [Figure 13], and fatty/fibrofatty replacement of the right ventricular free wall, similar to ARVD, although the genes are different. The morphological changes may be secondary to electrical abnormalities.^[30]

Left Bundle Branch Block

Left bundle branch block (LBBB) is a cardiac conduction abnormality that results in delayed activation and contraction of the left ventricle; it is often secondary to coronary artery disease, acute MI, aortic stenosis, dilated cardiomyopathy, hypertension, and conduction system abnormalities. In the presence of LBBB, the diagnosis of acute MI can be difficult since LBBB causes ST segment elevation or depression on its own, which may mimic or



Figure 13: Brugada syndrome. Horizontal long-axis SSFP image in 27-year-old male patient with acute chest pain, arrhythmias, and ST elevation shows focal out-pouching in the right ventricular outflow tract (arrow). No scar was seen in delayed enhancement images (not shown here). The right ventricular ejection fraction was 45%. This is an example of Brugada syndrome

mask MI, respectively.^[1] CT coronary angiography can be performed for the evaluation of underlying coronary artery disease. MRI shows paradoxical septal motion, where the septum, instead of moving toward the free wall in systole, moves away from the free wall. High temporal resolution cine MRI has been used to demonstrate increased delay between time to peak contraction (interval between R wave and peak of contraction) of the left ventricular lateral wall and ventricular septum and between the left ventricular lateral wall and the right ventricular lateral wall. This technique can be potentially used to distinguish patients with mechanical asynchrony from those without. Regional and global function can also be evaluated, along with the detection of a left ventricular scar.^[31]

Conclusion

ST segment elevation is an important ECG finding that is typically caused by acute myocardial infarction; however, it may also be seen in a variety of other disease processes. Although the differential diagnosis can be narrowed down by meticulous analysis of the clinical and ECG findings, MRI can play an important role in the evaluation of these patients. MRI not only establishes the diagnosis, which is essential for appropriate management, but also helps in assessment of other factors that are important for risk stratification.

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I, **Dr. Bhavin Jankharia**, hereby declare that the particulars given above are true to the best of my knowledge and belief.

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Dr. Bhavin Jankharia