Delayed Myocardial Enhancement

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Case Presentation

A 50-year-old woman with a 40 pack-year smoking history suffered a large myocardial infarction and underwent 2-vessel percutaneous coronary intervention with placement of bare metal stents. Follow-up transthoracic echocardiogram showed depressed left-ventricular ejection fraction (estimated 25-30%) with anterior wall motion abnormalities. However, the patient denied symptoms of congestive heart failure. Physical examination showed no jugular venous distension or peripheral edema. Heart rate and rhythm were normal without murmurs. Lungs were clear to auscultation. Patient was referred for cardiac MRI to measure ejection fraction, assess myocardial viability and scar, and further characterize wall motion abnormalities. Cardiac MRI showed a large area of full-thickness delayed enhancement in the lateral wall of the left ventricle with some involvement of the anterolateral and inferolateral walls. (Fig.) Left ventricular ejection fraction measured 46%.

Figure. Delayed enhancement cardiac MRI demonstrates full-thickness enhancement of the anterolateral wall (yellow arrow).
Key clinical finding

Wall motion abnormalities and diminished LVEF on echocardiogram.

Key imaging finding

Delayed myocardial enhancement

Differential diagnoses

- Myocardial infarction
- Myocarditis
- Non-ischemic cardiomyopathies

Discussion

Delayed gadolinium-enhanced magnetic resonance imaging can be used to evaluate pathologic processes that affect the myocardium. Gadolinium enhancement follows a predictable course through the cardiac blood pool, coronary circulation, and myocardial interstitium. Many pathologic processes of the myocardium will cause delayed washout of the gadolinium from the myocardium. Using a pulse sequence with an initial saturation pulse that nulls the signal of normal myocardium will yield sensitive evaluation for delayed myocardial enhancement. Dynamic fast gradient echo sequences can also be used for evaluation of left ventricular function and ejection fraction.

1. Myocardial infarction.

Myocardial infarction is caused by compromised coronary blood flow due to in situ thrombus, vasospasm, or hypoperfusion. In acute myocardial infarction, there may be hypoenhancement due to persistence of compromised blood flow within the infarct core on first-pass gadolinium enhanced images. However, if there has been reperfusion, the infarct core may not always be visualized. On delayed imaging, there will be hyperenhancement corresponding to edema and myocyte necrosis. In the later stages of healing, the myocardial tissue within the infarction will be replaced by fibrosis, which will show delayed enhancement. The pattern of the delayed enhancement will reflect the vascular distribution of the affected vessel. The location of delayed enhancement within the myocardium will show either subendocardial or transmural enhancement.

The role of MR in myocardial infarction is to assess the size and vascular territory of the infarction scar. Dynamic fast gradient echo imaging can also be used to assess wall motion abnormalities. This modality also provides information about the viability of the myocardium for planning revascularization therapy. Viable myocardium (stunned or hibernating) will show wall motion abnormalities or decreased ventricular function with normal enhancement kinetics, and carries a favorable prognosis for revascularization therapy. Infarction scar that involves greater than 50% of the myocardial thickness is unlikely to show return of function with revascularization.


Infection is the most common etiology of myocarditis. In the US, viral infection accounts for most cases of infectious myocarditis, particularly infection with Coxsackie B virus. Other infectious etiologies include bacterial, HIV, Lyme disease, and Chagas disease. Non-infectious etiologies include drug hypersensitivity and radiation. Clinical presentation may range from mild non-specific chest pain to severe chest pain simulating acute myocardial infarction. On cardiac MR imaging, the myocardium will show delayed enhancement in a predominantly subepicardial location. Delayed enhancement may also progress to transmural involvement. The regions of enhancement will typically not be confined within a singular vascular territory. Additionally, the areas of involvement will show edema manifesting as intense T2 bright signal.


Infiltrative/Inflammatory Cardiomyopathies

Many systemic conditions have been associated with cardiomyopathy secondary to deposition or infiltration of the myocardium. The two most common systemic conditions in this category are amyloidosis and sarcoidosis. Amyloidosis may be either primary or secondary to multiple myeloma or chronic inflammatory states. Both primary and secondary
amyloidosis may lead to deposition in the myocardial interstitium, thereby expanding the extracellular space of the myocardium. The pattern of delayed enhancement will be patchy early in the disease process and will usually progress to a pattern of subendocardial delayed enhancement. Because it is a systemic process, there may be involvement of all four chambers of the heart. Clinically, this manifests as a restrictive cardiomyopathy. Sarcoidosis is a systemic granulomatous disease that may affect any organ system. When sarcoid infiltrates the myocardium, it will typically manifest early as increased edema in the subepicardial and mesocardial locations with later appearance of patchy or nodular delayed enhancement. Clinical manifestations of sarcoïd cardiomyopathy include ventricular arrhythmias and restrictive cardiomyopathy.3,6,7

Other non-ischemic cardiomyopathies

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant congenital cardiomyopathy that causes myocardial thickening without left ventricular dilatation. The myocardial thickening may be diffuse or predominantly within the interventricular septum. Mid-myocardial enhancement may be seen within areas of fibrosis, and this finding has been associated with an increased risk of arrhythmia and sudden cardiac death.3,6,8

Dilated cardiomyopathy is an acquired cardiomyopathy that is often idiopathic. Other etiologies include drug toxicity, chronic alcohol abuse, or myocarditis. The predominant findings include left ventricular chamber enlargement and decreased ejection fraction. Similar to HCM, foci of mid-myocardial delayed enhancement correspond to foci of fibrosis and may be associated with increased risk of arrhythmia and sudden cardiac death.7

Peripartum cardiomyopathy is a condition in which congestive heart failure develops in the peripartum period, with approximately half of patients recovering full left ventricular function. On imaging, there is often delayed mid-myocardial enhancement in the anterior and anterolateral left ventricular walls. The loss of delayed enhancement often corresponds to recovery of left ventricular function.7

Diagnosis

Myocardial infarction

Summary

Cardiac MRI can be a useful modality in the workup of many ischemic and non-ischemic cardiac diseases. Delayed gadolinium enhancement of the myocardium may be seen with many conditions affecting the myocardium. Using clinical context as well as the pattern and location of myocardial enhancement, a specific diagnosis or focused differential diagnosis can be made.

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References