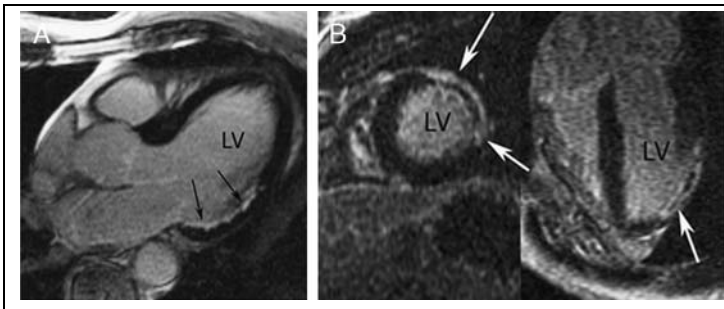
Ischemic Late Gadolinium Enhancement

Shinn-Huey S. Chou, MD, Christopher M. Walker, MD, and Jonathan H. Chung, MD



Ischemic late gadolinium enhancement

Subendocardial to transmural late gadolinium enhancement corresponding to a coronary arterial territory

Differential diagnosis

Acute myocardial infarction
Chronic myocardial infarct scar
Subendocardial late gadolinium enhancement mimickers
- Adjacent blood pool
- Myocardial cleft
- Septal perforator branch

Nonischemic late gadolinium enhancement
- Amyloidosis (diffuse crossing vascular territories)

FIGURE 1. A, LGE inversion-recovery MR image in left ventricular outflow tract view 15 minutes after intravenous injection of gadolinium chelate contrast agent in a patient with left circumflex coronary artery MI. Note subendocardial LGE (arrows) involving less than 50% of the lateral wall of the left ventricle (LV) indicating a high likelihood of segmental functional recovery following revascularization in the setting of hypokinesis and low ejection fraction. B, Composite image of a short axis (left) and radial (right) LGE inversion-recovery MR in a man with recent left anterior descending (LAD) coronary artery infarction shows transmural LGE (arrows) in the anteroseptal left ventricle (LV). There is a nonenhancing central core (black area) best visualized on the radial image indicating no reflow or microvascular obstruction. The degree of LGE measures greater than 50% of the wall thickness indicating a low likelihood of segmental functional recovery following revascularization.

Appearance: The classic appearance of ischemic myocardial late gadolinium enhancement (LGE) is subendocardial hyperenhancement in the distribution of a major coronary artery (Fig. 1). The thickness or transmurality of the LGE is variable and may extend across the entire myocardial wall. Occasionally, the LGE contains a dark nonenhancing core.

Explanation: Ischemic LGE is due to acute myocardial infarction (MI) or chronic scar from remote infarct and is seen on inversion recovery late gadolinium magnetic resonance (MR) imaging. New techniques to detect LGE with improved speed and quality have been developed.[1]

While research exploring the utility of intravascular gadolinium based contrast agents (GBCA), such as gadofosveset (Vasovist, ABLAVAR) is ongoing, cardiac MR is currently routinely performed with administration of standard extracellular GBCAs, e.g. Gd-DTPA (Magnevist) or Gd-BOPTA (Multihance). LGE is a phenomenon related to the distribution of the extracellular GBCAs. Upon intravenous injection of an extracellular GBCA, myocardium enhances when gadolinium chelate contrast material diffuses passively throughout the extracellular space.[2]

When a coronary artery occludes, myocardial cell death begins invariably in the subendocardium and progresses outward to involve the remainder of the myocardium.[3] Regions of infarcted myocardium have a significantly increased extracellular volume, requiring a longer time for the extracellular GBCA to wash-in and out compared to normal myocardium, thereby resulting in LGE.[1] Specifically, in chronic myocardial infarction, myocytes are replaced by an enlarged extracellular space of collagen matrix and fibrous tissue. Conversely, in acute myocardial infarction, there is a locally increased extracelluar collection of GBCA due to disruption of cellular membranes in edematous and necrotic myocardium.[4] A dark core may be present within the area of LGE in acute myocardial infarction because of microvascular obstruction, which prevents delivery of any gadolinium to the infarcted core.[4] Hypokinetic stunned or hibernating myocardium does not exhibit LGE.

Discussion: The high spatial resolution of MR provides a significant advantage over other noninvasive cardiac imaging modalities in the detection and characterization of MI. In animal models, ischemic LGE accurately correlates to an area of myonecrosis secondary to ischemic injury on histopathology.[5] LGE, however, is not specific for MI. For this reason, recognition of the subendocardial pattern confined to a coronary arterial territory is crucial in the diagnosis of ischemic LGE.

Mimickers such as high signal from the adjacent blood pool, myocardial cleft, or septal perforator branch can resemble subendocardial LGE.[6] Therefore, LGE should be seen on two consecutive short-axis images or be confirmed on orthogonal images and correlated with history and ancillary testing.[6] LGE images should also be interpreted alongside steady-state free precession (SSFP) cine images to detect hypokinesis/akinesis and thickness and morphology of the affected

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myocardium, which helps distinguish true LGE from normal blood pool in myocardium containing large trabeculations.[6] Reference to myocardial thickness on SSFP images also helps differentiate microvascular obstruction located within the myocardium from thrombus that resides in the adjacent ventricular cavity.

MR may differentiate ischemic LGE of acute MI from chronic scar. Acute MI is associated with myocardial edema that demonstrates high signal on T2-weighted imaging,[7,8] whereas chronic transmural infarct often results in myocardial thinning.[7] Many methods can quantify the infarct from the size of LGE.[3] Certain threshold measurements may overestimate the infarct size due to the presence of peripheral gray zones, an admixture of viable and nonviable myocytes which are a powerful predictor of all-cause post-MI mortality.[3,9] LGE quantification of infarct size as a percentage of the total LV volume is a strong predictor of future adverse cardiac events.[10] The transmural extent or infarct size is predictive of functional recovery of the affected myocardial segment after revascularization and thus helps guide therapeutic decisions.[3,7] A cutoff value of $<50\%$ LGE transmurality predicts reasonable functional segmental recovery supplied by the corresponding coronary artery, suggesting a potential benefit from revascularization.[5] The dark core of microvascular obstruction in acute MI portends a poor prognosis related to poor ventricular remodeling and increased complications.[4,7]

Stunned myocardium represents hypokinetic (but viable) myocardium in the setting of ischemia and subsequent reperfusion, which then recovers function after days or weeks.[2] Hibernating myocardium is similarly dysfunctional but viable myocardium which results from chronic coronary artery disease and local hypoperfusion.[2] Without the expanded extracellular volume from myonecrosis or fibrosis, these regions do not demonstrate ischemic LGE and suggest a good prognosis with intervention (e.g., angioplasty with or without stenting or coronary artery bypass graft surgery).[4] LGE of nonischemic etiologies, for instance, amyloidosis and vasculitides, may be subendocardial in distribution. Nonetheless, they are usually not restricted to specific coronary arterial distributions which are typical of ischemic LGE.

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