Acute Myocardial Infarction and Postinfarct Remodeling

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Magnetic Resonance Assessment of Left Ventricular Size and Function Postinfarction

In the wake of an acute myocardial infarction (AMI), accurate assessment of the left ventricle (LV) is of paramount importance as functional impairment or chamber dilatation predicts increased mortality (1–3). In a study of 866 postinfarct patients, a resting LV ejection fraction (EF) less than 0.40 predicted higher 1-year mortality than did an LVEF greater than 0.40 (1). Others have demonstrated that, following reperfusion therapy, the relationship between resting LVEF and all-cause mortality persists (4). Although LVEF is a powerful predictor of postinfarct survival, end-systolic volume (ESV) is superior as a prognostic parameter. This was demonstrated by White et al., who enrolled 605 patients following AMI and followed them for an average of 78 months. Ultimately, ESV was the best predictor of survival, and neither the LVEF nor end-diastolic volume (EDV) added prognostic value.

Radionuclide imaging and transthoracic echocardiography are widely available and well-validated noninvasive techniques employed for measuring LVEF and cardiac volumes. Although studies have generally demonstrated good correlation between these modalities, both inter- and intraobserver agreement is suboptimal, and most studies have only assessed patients with normal LV dimensions and function (5). Moreover, in patients following AMI, distortion of LV chamber size and geometry can exacerbate measurement error.

In contrast, cardiovascular magnetic resonance (CMR) generates a complete three-dimensional (3D) data set from apex to base with high temporal and spatial resolution (Fig. 1). Measurements made with these data set do not require geometric assumptions.
and are therefore less prone to error in ventricles deformed by infarction. Furthermore, the large field of view and excellent contrast generated between the myocardium and blood pool facilitate accurate assessment of chamber dimensions and both regional and global function (6). Consequently, CMR is increasingly employed for prognostication and to guide therapeutic management in patients postinfarct.

**Contrast-Enhanced CMR**

Histopathologic studies of AMI have consistently revealed the complex, heterogeneous, and dynamic character of infarct zones (7). This heterogeneity appears to be a consequence of many factors, including the presence and opening of collaterals during and after injury, magnitude of the ischemic insult, and impact of therapeutic interventions (8). Furthermore, it has been demonstrated that infarcts evolve over time, generally passing through an early, necrotic phase followed by both a fibrotic and remodeling phase that is dominated by the laying down of new collagen and infarct involution (9,10). In fact, infarct scar remains biologically active long after ischemic injury, populated by cells involved in collagen turnover and scar tissue contraction, and may therefore never truly reach a “stable” configuration (7).

In the acute phase, at the very core of infarcts and typically within the subendocardium, there is often microvascular obstruction (MO). MO may result from embolization and platelet activation, although the exact mechanisms are unclear (11). These areas are generally devoid of blood flow and have also been described as “no-reflow zones” with the extent of no reflow related to total infarct size (12). Surrounding areas of no reflow are often comprised of myocytes subjected to significant, but varying, degrees of ischemic injury. Many undergo necrosis with loss of membrane integrity and depletion of cellular energy stores (13). In this acute phase, tissue edema, hemorrhage, and inflammation can increase infarct volume by as much as 25% (8). Beyond these necrotic regions, dysfunctional, nonnecrotic tissue coexists that has the potential for functional recovery (14).