58. Ischemic Heart Disease

In recent years cardiac MRI has gained more and more importance for risk stratification and decision making in the management of ischemic heart disease. Typically cine images are obtained to evaluate local wall motion, global cardiac function and morphology, followed by first pass perfusion studies (under rest and/or stress), then T1-weighed IR sequences to evaluate myocardial viability. MR stress testing exploits the fact that extravascular, extracellular gadolinium chelates pass through the myocardial interstitium within minutes of bolus injection, allowing detection of perfusion differences. Within 10 minutes, washout occurs, and a stress-inducing agent and additional contrast may be given. Stress-inducing agents include dobutamine, dipyridamole, or adenosine. The latter two are coronary vasodilators that are contraindicated in obstructive lung disease and necessitate aminophylline administration should angina be induced. In the presence of vascular stenosis, normal vessels are dilated by these agents, such dilatation stealing flow from the distribution of the stenotic vessel. Areas lacking normal perfusion (enhancement) are characterized as fixed (present on both acquisitions) or reversible (present on stress only). Reduced subendocardial perfusion may also represent reversible microvascular disease, severe three-vessel disease with poor reserve, or even artifact. The latter is favored by a deficit of inconsistent size and SI, not localized to a coronary arterial distribution or present only on rest images. Dobutamine increases heart rate and contractility allowing MR evaluation of wall motion with higher spatial resolution, scan quality, and reproducibility than echo. For wall motion evaluations, cine images are obtained and myocardial segments (usually based on the 17-segment model of the American Heart Association) graded before and after dobutamine in terms of normal wall thickening during systole. Grade 1 constitutes normal wall motion, whereas grades 2, 3, and 4 correlate with hypokinesis, akinesis, and dyskinesis, respectively.

The two chamber long axis viability study in Fig. 58.1A was obtained 10-15 minutes following contrast bolus with IR (set to attenuate normal myocardial SI) T1WI in a patient with a history suggestive of acute ischemia. Viability studies exploit the fact that extravascular, extracellular gadolinium chelates only enter cells when membrane damage is present, resulting in persistent enhancement of infarcted, nonviable myocardium. Cardiac MR helps to distinguish stunned (or hibernating) and thus potentially salvable from truly infarcted myocardium—the former predicting a positive outcome with revascularization. While contractility, as visualized with cine sequences, is variably impaired for both, viable and infarcted myocardium, stunned or hibernating myocardium consistently demonstrates normal, early enhancement with brisk washout on viability studies, whereas enhancement

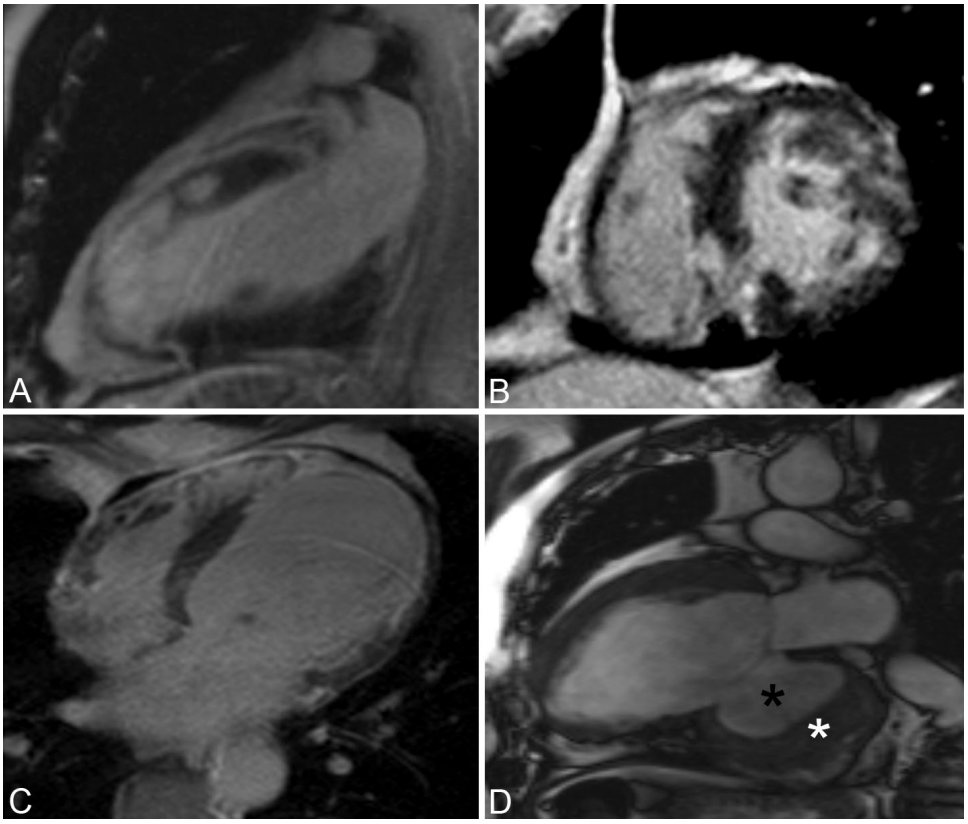

Fig. 58.1

of nonviable, infarcted tissue is early and persists. In Fig. 58.1A, myocardial enhancement of the mid anterior left ventricular wall to the LV apex confirmed the presence of acute myocardial infarction within the distribution of the left anterior descending artery. No reflow phenomenon is occasionally present on MR viability studies, with the characteristic appearance of an area of low SI in the center of brightly enhancing myocardium. No perfusion or subsequent contrast penetration occurs in the low SI region secondary to microvascular occlusions, whereas the larger, enhancing area corresponds to a region of infarction reperfused (and contrast delivered by) a patent larger artery. In contrast to normal myocardium, diminished enhancement within an area of no reflow also exhibits progressive enhancement over time and persists as low SI even as the timing of the IR pulse (usually set to suppress normal myocardial SI) is varied.

Enhancement within a specific arterial territory and subendocardial to transmural predominance, as seen in Fig. 58.1A, implicates the delayed enhancement of ischemia rather than that of myocarditis. This pattern is not always reliable, as seen in the patchy, less predominant enhancement in the short axis IR CE T1WI image in Fig. 58.1B—a case of ischemia secondary to embolic phenomenon in a patient with coronary artery ectasia (a condition associated with coronary aneurysm formation). Delayed enhancement may also

occur with myocardial scarring—the end product of myocyte damage—in which contrast is retained within the widened interstitium between collagen fibers. Wall thickness may help distinguish enhancement of infarction from that of fibrosis: a thin enhancing wall—as seen in the left ventricular wall in the four chamber view of the IR CE T1WI in Fig. 58.1C—implies chronic scarring, whereas a thick, enhancing wall signifies acute ischemia. Complications of myocardial infarction are also readily visible on MR. The two chamber cine image in Fig. 58.1D demonstrates an aneurysm of the inferior left ventricular wall. While flow is present within portions of the aneurysm (black asterisk), low SI clot is also present (white asterisk). Unlike regions of scarring or ischemia, clot does not exhibit delayed enhancement on CE IR T1WI. Pericardium surrounding a true aneurysm (Fig. 58.1D) is less likely to enhance than that surrounding a false aneurysm. The latter consists of ruptured myocardium, its contents are enclosed by thickened pericardial adhesions.
MR cardiac imaging techniques continue to improve, with technologic advances, making image acquisition easier and more robust. BioMatrix sensors integrated into the MR coils used for signal reception capture respiratory and cardiac motion, information that can be used to improve image quality, and obviate the use of prior techniques requiring additional hardware and setup such as ECG monitoring. Furthermore, the implementation of compressed sensing enables real-time imaging during free-breathing, an approach employed for aquisition of the cine images in Fig. 58.2.

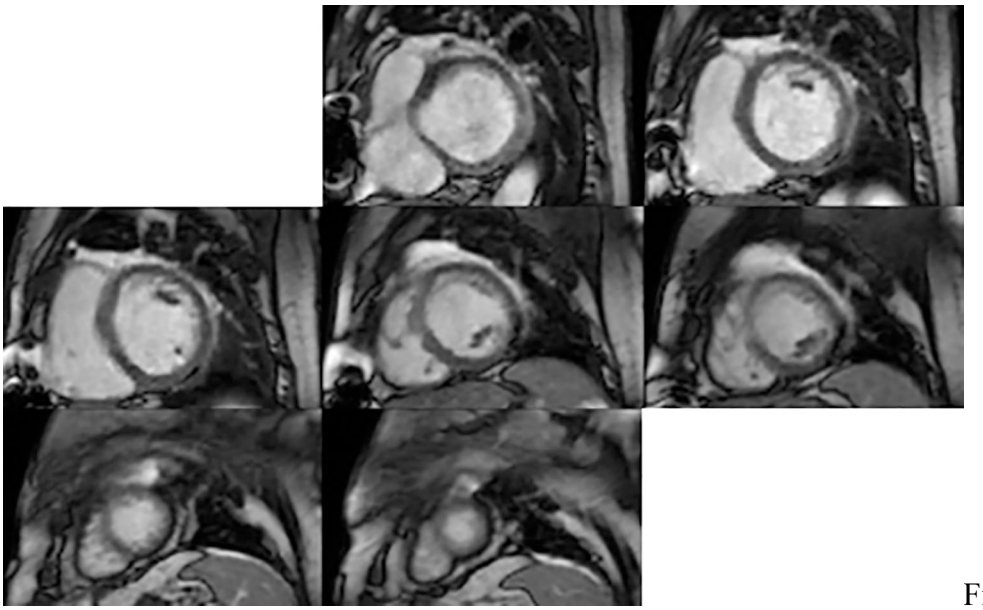

Fig. 58.2

Courtesy of Ulrike Attenberger