"Non-ischemic Delayed Enhancement"

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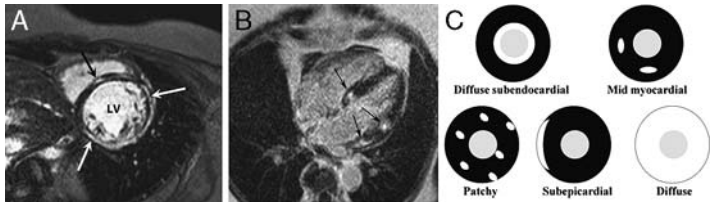

FIGURE 1. A, Delayed-enhancement inversion-recovery MR image in short axis view 15 minutes after intravenous injection of gadolinium chelate contrast agent in a 19-year-old woman with a non-ischemic dilated cardiomyopathy secondary to myocarditis. Note mid myocardial delayed enhancement (white and black arrows) involving the majority of the left ventricular myocardium. The subendocardium is completely spared, which essentially rules out ischemic heart disease. B, Four-chamber delayed-enhancement inversion-recovery MR image in a 44-year-old woman with cardiac sarcoidosis shows nodular enhancement in the basal interventricular septum and lateral left ventricular wall (arrows). C, Graphic shows various patterns of nonischemic delayed enhancement including diffuse subendocardial, patchy, mid myocardial, diffuse, and subepicardial. LV indicates left ventricle (to view a larger image, go to http://links.lww.com/JTI/A43).

Non-ischemic Delayed Enhancement

Pattern of delayed enhancement not corresponding to a vascular territory

Differential diagnosis with typical enhancement patterns

Myocarditis (Subepicardial or mid myocardium)

Idiopathic dilated cardiomyopathy (mid myocardium)

Amyloidosis (Diffuse subendocardial)

Sarcoidosis (Patchy or nodular; RV side of interventricular septum is highly specific)

Hypertrophic cardiomyopathy (Mid myocardial ventricular septum at junction with RV)

Churg-Strauss syndrome (Highly variable)

Fabry disease (Mid myocardium in the inferolateral basal segments)

RV indicates Right ventricle.

Appearance: Non-ischemic delayed enhancement (NIDE) is characterized by enhancement within abnormal myocardium that does not follow a vascular distribution (i.e., not subendocardial and not in the distribution of a major coronary artery). Patterns of NIDE include diffuse subendocardial, midwall, subepicardial, patchy, and diffuse (Fig. 1).

Explanation: Delayed enhancement MR imaging is an inversion-recovery, gradient-echo sequence. It is typically performed 10-15 minutes following the administration of intravenous gadolinium chelate contrast agent. The inversion time is different for each patient and is specifically chosen to null (i.e., make black) normal myocardium with the goal to increase conspicuity of the enhancing myocardium. The cause of nonischemic delayed myocardial enhancement varies depending on the underlying pathology.[1,2] (All references cited in this article can be found at http://links.lww.com/JTI/A40.) For instance, early myocarditis exhibits patchy delayed enhancement due to myocardial necrosis; whereas, chronic myocarditis enhances in regions of myocardial fibrosis with a mechanism similar to that seen in chronic myocardial infarction.[3] The mechanism of delayed enhancement in cardiac amyloidosis is controversial. Amyloid may deposit in and around small coronary vessels leading to distal myocardial fibrosis and prolonged retention of gadolinium in a subendocardial distribution.[4] A second theory states amyloid deposition alters gadolinium kinetics leading to delayed enhancement through expansion of the extracellular space.[2]

Discussion: A key question to answer when interpreting delayed enhancement imaging is whether the enhancement is secondary to myocardial infarction or a non-ischemic condition. This question can be answered by determining the pattern of delayed enhancement. Myocardial infarction leads to delayed enhancement in a vascular territory (e.g., left anterior descending, left circumflex, or right coronary arteries) and always involves the subendocardium with variable extension to the remainder of the ventricular wall (i.e., transmural enhancement).[1,2,5,6] In contrast, non-ischemic heart disease is characterized by patterns of delayed enhancement that do not follow a vascular territory and often spares the subendocardium while involving the mid myocardium or subepicardium.[1,2,6,7] When the pattern of enhancement in nonischemic heart disease is restricted to the subendocardial layer, the enhancement usually crosses vascular territories.[6] Amyloidosis is the most common non-ischemic condition to cause diffuse subendocardial delayed enhancement. It is differentiated from three vessel coronary artery disease by its lack of severe systolic dysfunction. Conversely, myocarditis and sarcoidosis often cause patchy or nodular delayed enhancement involving the subepicardium and mid myocardium. It has been proposed that certain viral infections may preferentially affect different segments of myocardium.[2] Hypertrophic cardiomyopathy is usually location specific, most often involving the mid myocardial ventricular septum at its junction with the right ventricle. The specific pattern of delayed enhancement is used to help narrow the differential diagnosis with the help of pertinent patient history, physical examination, and other imaging findings.

The authors declare no conflicts of interest.

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