Visualisation of Left Atrial Fibrosis by Delayed Enhancement MRI: Correlation to an Atypical Atrial Flutter Substrate During Endocardial Mapping and Ablation

PETER A. NOSEWORTHY, MD, GODTFRED HOLMVANG, MD, WILLIAM J. KOSTIS, PhD, MD, MICHAEL G. FRADLEY, MD, CONOR BARRETT, MD, STEPHAN DANIK, MD, KEVIN E. HEIST, MD, PhD, JEREMY N. RUSKIN, MD and MOUSSA Mansour, MD

Cardiac Arrhythmia Service and The Cardiology Division, Massachusetts General Hospital, Boston, MA

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Case report

A 73-year-old man with longstanding, drug-refractory persistent atrial fibrillation (AF) and atrial flutter was referred for catheter ablation. Preprocedure delayed enhancement magnetic resonance imaging (DE-MRI) was performed to map left atrial and pulmonary vein anatomy and to quantify the degree and distribution of left atrial scar/fibrosis. The MRI was obtained on a 3.0-tesla clinical scanner using a cardiac array coil. The scan was acquired 45 min following gadolinium contrast agent injection using a three-dimensional inversion recovery, respiration navigated, electrocardiogram-gated, gradient echo pulse sequence as previously described.1,2 The left atrial wall was segmented manually using volume-rendering software. The fibrosis within the left atrial wall was analyzed qualitatively using a multiple threshold-based approach in which the color thresholds were adjusted according to signal intensities in the left atrial appendage (assumed to have no fibrosis/scar, rendered in blue) and to the mitral annulus (a fibrous structure with prominent delayed enhancement, rendered in green) (Figure 1a).

Radiofrequency applications were delivered to the antral regions of all four pulmonary veins, resulting in isolation. Subsequently, the AF cycle length increased and the arrhythmia organized into an atypical flutter with a cycle length of 218 ms (Figure 1b). Activation mapping and entrainment maneuvers localized the clockwise flutter circuit to the anterior left atrial wall. A second flutter with a counterclockwise activation sequence was then observed (cycle length 248 ms, Figure 1c). Both atrial flutters exhibited an area of slow conduction in the anterior left atrium, as manifested by crowded isochrones on the activation map and low amplitude, fractionated signals on intracardiac electrograms (Figure 2a). Entrainment from this area yielded good postpacing intervals during the clockwise (Figure 2b) and counterclockwise (Figure 2c) left atrial flutter. Radiofrequency ablation was delivered to the anterior left atrial wall creating an anterior mitral isthmus line (Figure 1d), and the flutter terminated with ablation. Now 3 months post ablation, the patient feels well and has returned to his previous activity level.

References

Compared with paroxysmal AF, catheter ablation for persistent AF has lower success rates and a standardized approach to ablation has not been firmly established. Atrial scar imaging holds promise in defining the arrhythmogenic substrate and defining targets for ablation. DE-MRI has been used to evaluate the degree and distribution of fibrosis associated with AF, and this has been related to recurrence rates after catheter ablation. For this reason, many believe that DE-MRI may help guide clinicians in their approach to AF catheter ablation. Difficulty in standardizing DE-MRI protocols and obtaining readily available and interpretable images has been challenging, and this approach has not yet been widely adopted.

Here, we demonstrate an example of correlation of preprocedural DE-MRI scar to an arrhythmogenic substrate defined during electrophysiology study. In this case, DE-MRI was useful, not only in assessing the degree of fibrosis/scar, but in defining a discrete region of slow conduction responsible for two flutter circuits. Ablation of an isthmus of tissue within this scar was successful in terminating the arrhythmia. This case illustrates the potential of preprocedural DE-MRI to guide effective, targeted ablation for atrial arrhythmias.

Figure 1: (a) Delayed enhancement magnetic resonance imaging indicates a region of fibrosis in the anterior wall of the left atrium (red arrow), in the mitral annulus (red dotted line), and in the left superior pulmonary vein. There is no evidence of fibrosis or scar in the left atrial appendage. Isochronal activation maps demonstrate (b) an atypical left atrial flutter with a counterclockwise perimitral circuit and a zone of slow conduction in the anterior left atrium, and (c) a second perimitral flutter in the clockwise direction with an anterior zone of slow conduction. (d) A series of anterior mitral isthmus ablation lesions (red dots) created a line of block which broke and prevented reinduction of the flutter (white dot indicates the lesion that broke the flutter).
Figure 2: (a) During clockwise atrial flutter, an area of fractionated, low amplitude electrograms was identified on the anterior left atrium (red arrow). Entrainment from this area yielded good postpacing intervals during clockwise (b) and counterclockwise (c) left atrial flutter.
References
