Radiology

CATCH the Wave of Coronary Atherosclerotic Plaque MRI

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Cardiovascular disease is the leading cause of death in industrialized nations. Despite improved prevention, diagnosis, interventions, and medical therapy, close to 50% of these events can be attributed to coronary heart disease. An early manifestation of coronary atherosclerosis is arterial remodeling (enlargement of the artery) with atherosclerotic plaque formation in the coronary vessel wall. Thus, greater atheroma burden often precedes luminal narrowing. This early plaque does not result in coronary artery narrowing and is therefore not discernible at conventional invasive radiographic coronary angiography.

By using intravascular US (IVUS), atheroma volume can be measured as a quantitative end point to study plaque development. IVUS is an invasive procedure that provides useful morphologic information. However, more granular knowledge about plaque constituents linked with plaque rupture derived from IVUS may ultimately be of critical value to monitor the effects of individualized treatments, to be applied to a broader patient population, and to support clinical decision making. Therefore, there is a strong need for a noninvasive test to assess atheroma that can be broadly and safely applied to improve patient management. Clearly, these requirements put MRI into the spotlight: MRI is noninvasive, safe, and operates without ionizing radiation. Serial studies to monitor plaque progression or regression in response to therapy can easily be performed with MRI.

The use of MRI to help characterize coronary artery plaque is not new. In 2007, Yeon et al (1) investigated gadolinium chelate contrast enhancement of the coronary arteries in patients with risk factors for coronary artery disease. Patients underwent dark-blood plaque and bright-blood coronary MRI, coronary multisection CT. Quantitative coronary angiography was also performed. By using T1-weighted inversion recovery three-dimensional gradient-echo imaging in a relatively small cohort of patients, the authors successfully identified areas of gadolinium enhancement. This correlated with the severity of atherosclerosis at multisection CT and quantitative coronary angiography (1). Gadolinium enhancement occurred more often in calcified plaques where, intriguingly, native (noncontrast-enhanced) high T1 signal was occasionally observed. Yeon et al attributed the high T1 signal to atherosclerotic plaque hemorrhage or thrombus. However, an in-depth analysis to elucidate the origin of high T1 signal at noncontrast-enhanced MRI was not performed. Nevertheless, the observations of Yeon et al stimulated further investigations. This included a noncontrast-enhanced study by Kawasaki et al (2) that reported hyperintense coronary artery plaque at T1-weighted inversion recovery whole-heart three-dimensional MRI. This was linked with arterial remodeling, US attenuation, and lower Hounsfield units, all believed to be indicative of unstable plaque.

More recent investigations with a similar MRI approach (3,4) in approximately 600 patients with suspected or known coronary artery disease suggest that noncontrast-enhanced coronary vessel wall high signal was predictive for future coronary events in patients with stable coronary artery disease (3). Importantly, high T1 signal in the coronary artery wall was reduced after statin therapy (4). As part of these noncontrast-enhanced studies, Noguchi et al (3) carefully established a dichotomous optimal cutoff value of 1.4 or greater for plaque to myocardium signal intensity ratio to define high-intensity plaque (HIP) at MRI.

Taken together, these noncontrast-enhanced studies help advance the hypothesis that coronary plaque may be more vulnerable (ie, more likely to rupture) when a high-intensity signal exists at noncontrast-enhanced T1-weighted inversion-recovery MRI. Therefore, these investigations helped to advance research and discovery in the domain of coronary artery plaque MRI and provided a strong impetus to further elucidate its clinical utility.

Nevertheless, the MRI method used in these previous studies required two separate pulse sequences: a bright-blood coronary MR angiogram and a T1-weighted inversion-recovery scan for HIP visualization. These paired scans were mandatory to ensure precise and nonambiguous spatial colocalization of HIP and the underlying coronary anatomy. However, the need for these two separate sequences were potentially problematic for this powerful method. Limitations included co-registration of the two acquisitions for postprocessing and long MRI scan acquisition times.

Taken together, there is a need for technical developments aimed at time-efficient and easy-to-use, free-breathing,

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Conflicts of interest are listed at the end of this article.

See also the article by Sato and Matsumoto et al in this issue.

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whole-heart, three-dimensional, noncontrast-enhanced, T1-weighted, inversion-recovery coronary plaque imaging. Such developments are necessary to support more widespread dissemination of this technology and also for its continued and rigorous validation by using reference standard comparisons.

Coronary atherosclerosis T1-weighted characterization (CATCH) MRI was introduced in 2017 (5). In brief, during a single free-breathing three-dimensional whole-heart examination, an inversion-prepared acquisition for native HIP visualization is interleaved with a bright-blood image acquisition of the coronary anatomy so that there is precise spatial colocalization. Only a single MRI scan must be prescribed, whereas coregistration of the images for postprocessing is no longer needed. In the initial description, CATCH was applied before and after contrast enhancement at 3.0 T. Thirty patients with stable angina were studied; a subset also underwent invasive coronary angiography and invasive optical coherence tomography to identify high-risk plaque features. The authors concluded that CATCH provided time-efficient (about 10 minutes) whole-heart coronary plaque characterization. High T1 signal intensity regions with CATCH showed a positive association with high-risk plaque features in the invasive imaging studies. Consistent with earlier studies (1), high coronary T1 signal was not present in healthy individuals without coronary artery disease. However, the reason for HIP by CATCH MRI remained unknown: both intraplaque hemorrhage and regional lipid accumulation could potentially cause high T1 signal in the coronary artery wall.

To bridge this knowledge gap, in this issue of *Radiology*, Sato and Matsumoto et al (6) addressed the important topic of noninvasive characterization of coronary artery plaque constituents by using the CATCH MRI method in a retrospective study. The authors studied 117 patients with chronic coronary artery disease. The study cohort also underwent native noncontrast-enhanced CATCH MRI with near-infrared spectroscopy (NIRS) IVUS to study detailed lesion composition. Patient data were collected in two facilities at both 1.5 T and 3.0 T. Quantitative and qualitative end points and intra- and interobserver variability were studied with scientific rigor. In 95 patients, the authors found 205 atherosclerotic plaques. From these, 42 plaques were classified as HIPs by using the discussed threshold. These HIPs were associated with both echolucent zones at IVUS and a lipid core burden index at NIRS. By using a multivariable model, however, HIPs were then independently associated with echolucent zones but not with the lipid-rich plaque. Therefore, the authors were correct in pointing out that the study shows the link between HIP at MRI and coronary plaque characteristics at NIRS IVUS in patients with stable coronary artery disease.

The main finding was that the echolucent zone at IVUS had a strong link with HIPs. In 79% of the cases, these echolucent zones coincided with HIP in the same region. Simultaneously, lipid-rich plaque at NIRS was not independently associated with HIP in stable coronary artery disease. Among 42 HIPs at MRI, 25 were categorized as lipid rich and the remainder were categorized as nonlipid rich at NIRS. These results are intriguing.

The findings by Sato and Matsumoto et al advance the hypothesis that the predominant substrate for HIPs in stable coronary artery disease may be intraplaque hemorrhage. These findings are corroborated in a recent study by Kuroiwa et al (7). In that study, in vitro MRI in 37 human cadavers was performed and paired with immunohistochemistry. Consistent with the results from Sato and Matsumoto et al, these investigations revealed that most coronary HIPs at T1-weighted MRI reflect intraplaque hemorrhage and have biologically unstable and procoagulant potential. However, a study (8) conducted in 105 patients also with stable coronary artery disease showed that HIPs at T1-weighted inversion-recovery MRI were strongly associated with healed plaque rupture and a large lipid core as defined at optical coherence tomography. Overall, there seems to be considerable evidence that HIPs at T1-weighted MRI are associated with the propensity of a plaque to rupture, and that the magnitude of the regional coronary signal enhancement may be a valuable end point to help guide and monitor therapy.

The study by Sato and Matsumoto et al (6) had some limitations that were well described. The limitations were related to the inclusion of patients with significant coronary artery disease, the small sample size, and the lack of rigorous histopathologic validation. To address these limitations and harvest the full potential of MRI with reference standard comparisons, a time-efficient and easy-to-use technique is needed to conduct patient studies on a larger scale. This gap may be bridged by using CATCH MRI that could easily be implemented on multiple vendors' MRI systems for broader worldwide dissemination in the interest of better management of coronary heart disease.

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