

Magnetic Resonance Imaging

Use of MRI in Carotid Artery Plaque Detection

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[Rev Cardiovasc Med. 2011;12(4):244-245 doi: 10.3909/ricm0627]

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MR Imaging of Carotid Plaque Composition During Lipid-Lowering Therapy: A Prospective Assessment of Effect and Time Course

Zhao XQ, Dong L, Hatsukami T, et al.

JACC Cardiovasc Imaging. 2011;4:977-985.

High-resolution magnetic resonance imaging (hs-MRI) with 3T magnets applied to atherosclerotic vascular disease represents a pivotal development. It enhances our ability to identify plaque with a greater degree of sensitivity than technologies such as

ultrasound; it also improves our ability to identify the biology of a given plaque in a given patient better than carotid intima-media thickness (cIMT), and follow the effects of interventions with a higher degree of accuracy. Dr. Zhao and colleagues have been pioneers in this field. These investigators designed and conducted the Carotid Plaque Composition by MRI During Lipid Lowering (CPC) trial to test the hypothesis that intensive lipid-lowering therapy will deplete plaque lipid content and that lowering low-density lipoprotein cholesterol (LDL-C) in addition to raising high-density lipoprotein cholesterol (HDL-C) compared with LDL-C lowering alone will lead to more plaque depletion. LDL-C reduction in the range of 50% and HDL-C increase in the range of 20% were achieved; the primary endpoint was carotid plaque lipid content.

A total of 123 subjects with documented coronary artery disease, apolipoprotein B levels > 120 mg/dL, and duration of lipid therapy < 1 year were studied. Bilateral carotid MRI scans were obtained at baseline, and at 1, 2, and 3 years. Two-dimensional time-of-flight, proton density, and T2- and T1-weighted images were acquired. A T1 scan was then performed 5 to 7 minutes after gadolinium infusion. Carotid plaque tissue composition was identified and quantified using accepted MRI criteria. Figure 3 provides an example of the images obtained in the carotid artery from the common carotid to the internal carotid artery.

This study demonstrated that significant lipid depletion can be achieved after 1 year of intensive lipid therapy,

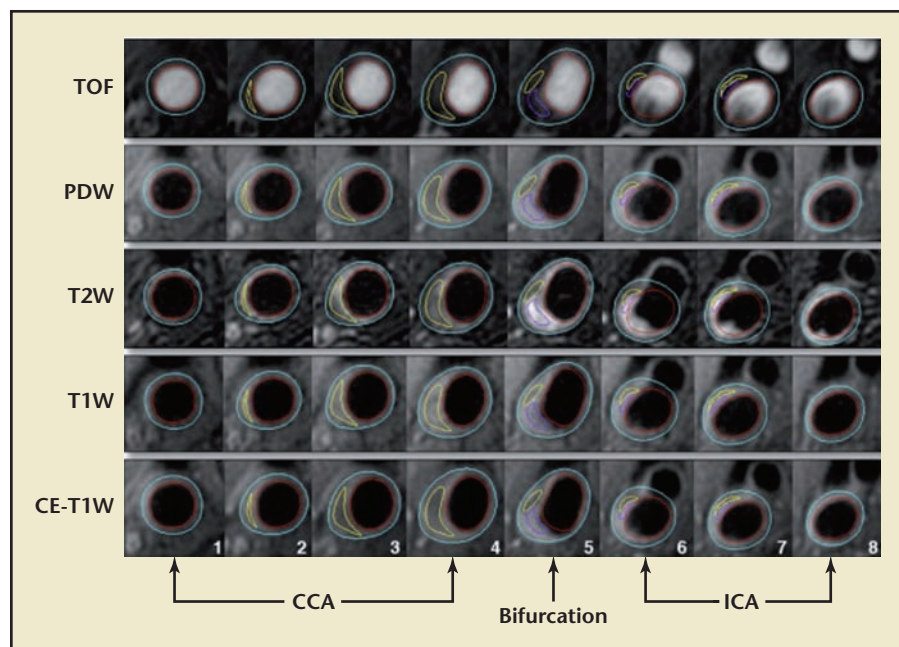


Figure 3. Example of carotid artery in multicontrast MRI. MRI example of eight consecutive images of the CCA through bifurcation to the ICA from five contrast weightings: TOF, PDW, T2W, T1W, and postcontrast T1W. The lumen in red and outer wall boundary in blue of the carotid artery are identified and outlined. Plaque tissue components within the arterial wall are identified using an established algorithm and quantified (loose matrix in purple and lipid content in yellow). For example, lipid content in yellow appears isointense on the TOF and pre-contrast T1W images and hypointense on PDW and T2W images, and has no contrast enhancement compared with the surrounding tissue on post-contrast T1W images. Total LRNC volume is calculated by multiplying the slice thickness (2 mm) by the sum of the areas circled in yellow color on the eight consecutive images. Percent LRNC (%LRNC) is the proportion of the wall occupied by the LRNC only in the lipid-containing slices, from slice #2 to #7. CCA, common carotid artery; ICA, internal carotid arteries; LRNC, lipid-rich necrotic core; MRI, magnetic resonance imaging; PDW, proton density-weighted; T1W, T1-weighted; T2W, T2-weighted; TOF, time of flight. Reprinted from Serruys PW et al. J Am Coll Cardiol. 2011;58:1578-1588; with permission from Cardiosource.

continued into the second year with a diminished reduction of effect at year 3, corresponding to the clinical benefits observed in lipid-lowering trials. In addition, overall plaque burden was noted to be reduced as well. It is the feeling of the authors that the decrease in plaque burden occurred primarily as a result of lipid depletion. It is intuitive to suggest that untreated patients with lipid-laden plaques would be more responsive to delipidation with LDL-C lowering. This could have led to the differences observed using cIMT in the Effect of Rosuvastatin on Progression of Carotid Intima Media Thickness in Low-Risk Individuals (METEOR) trial and the Simvastatin With or Without Ezetimibe in Familial Hypercholesterolemia (ENHANCE) trial. The patients enrolled in METEOR were relatively statin naïve with thickened cIMTs at baseline (1.16 mm), versus those in ENHANCE, who had been treated for an extended period of time and had baseline cIMTs of 0.68 mm. One would therefore expect a greater change in IMT with the greater abnormality at baseline in METEOR.

It is becoming increasingly clear that hsMRI is the gold standard for carotid artery atherosclerosis plaque detection and assessment of delipidation with therapy (Figure 4). Several interesting questions arise from this experience with carotid MRI:

- Is hsMRI-assessed delipidation of plaque in the carotid artery related to similar changes in the coronary bed and a reduction of cardiovascular events?
- If plaque delipidation reaches a peak after 3 years, can statin therapy be modified (ie, dose reduction) instead of continuing treatment, and is this safe?
- Are there differences in the approach to lipid reduction (weight loss/exercise, statins, niacin, ezetimibe, fibric acid derivatives, resin binders) that influence lipid content assessed by hsMRI?
- Is there a relationship between the extent of plaque regression and other biomarkers of atherosclerosis such as Lp-PLA₂, myeloperoxidase, or C-reactive protein? ■

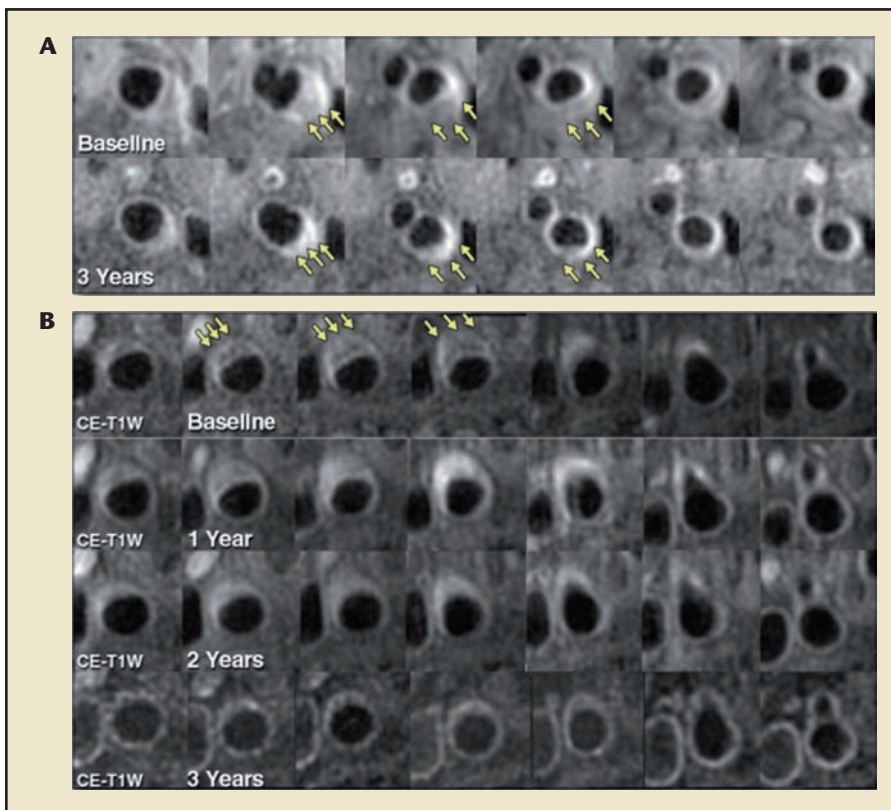


Figure 4. MRI examples of plaque lipid depletion and time course. **(A)** An example of significant lipid content reduction (yellow arrows) and plaque regression at 3 years compared with baseline in the left carotid artery. Overall, 11% of study subjects had completed plaque lipid depletion over 3 years. **(B)** MRI example of the plaque lipid depletion time course. Regression in LRNC size was notable between the baseline, 1-year, and 2-year MRI scans. Regression in plaque volume seemed to follow plaque lipid depletion and was most pronounced from years 1 to 3. CE, contrast enhanced; LRNC, lipid-rich necrotic core; MRI, magnetic resonance imaging; T1W, T1-weighted. Reprinted from Serruys PW et al. *J Am Coll Cardiol.* 2011;58:1578-1588; with permission from Cardiosource.