Cardiac Magnetic Resonance Features of the Disruption-Prone and the Disrupted Carotid Plaque

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Stroke is a leading cause of long-term disability and is the third most common cause of death in many countries (1). Twenty percent of strokes are thought to be related to extracranial carotid atherosclerosis (2). A means to prevent such events is carotid endarterectomy and carotid stenting. However, the ACAS (Asymptomatic Carotid Atherosclerosis Study) (3) showed that carotid endarterectomy reduced risk for ipsilateral stroke by only 5.9% at 5 years when compared with best medical management. Therefore, additional criteria, other than the degree of stenosis, have been sought to better identify patients most at risk of complications from carotid disease.

Based on analysis of histological findings in carotid endarterectomy specimens, plaque disruption is believed to be a major factor in the etiology of carotid-territory ischemic events. Features such as intraplaque hemorrhage, large necrotic cores with thin overlying fibrous caps, plaque neovascularization, and inflammatory cell infiltrate (4–7) may predispose the atherosclerotic lesion to disruption. Hence, these features represent targets for imaging techniques aimed at identifying high-risk, disruption-prone plaque.

Equally important is the identification of luminal surface disruption, such as fibrous...
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cap rupture, ulceration, and calcified nodules. Retrospective studies have shown that these so-called culprit lesion features are associated with a prior history of recent transient ischemic attack or stroke (8). Furthermore, they may pose a persistent increased risk for secondary events, as suggested by findings from histology studies showing evidence of repeated cap ruptures (9), and by findings from the NASCET (North American Symptomatic Carotid Endarterectomy Trial). In a subgroup analysis comparing individuals with and without ulcerated carotid plaques, those with ulcers on angiography had a 1.2- to 3.4-fold increased risk for stroke (10).

As a noninvasive imaging modality with the capability to distinguish tissue characteristics, cardiac magnetic resonance (CMR) is an optimal method for characterizing the morphology and composition of atherosclerotic carotid plaques (8,11–28). Multiple centers have shown that CMR can reliably identify fibrous cap status, the lipid-rich necrotic core, intraplaque hemorrhage, and vascular wall inflammation, using histology as the gold standard (14–21,23,29–36). Additional advantages of CMR include image generation without ionizing radiation or the need for invasive procedures, which make it an ideal tool for serial, longitudinal study of plaque progression or regression.

Recently published clinical studies show the potential prognostic value of CMR in patients with moderate carotid stenosis. In a prospective study of 154 patients with 50% to 79% carotid stenosis who were asymptomatic at the time of enrollment, participants underwent baseline carotid CMR and were contacted every 3 months to identify symptoms of new-onset transient ischemic attack or stroke (25). Twelve cerebrovascular events occurred ipsilateral to the index carotid artery over a mean follow-up period of 38.2 months. Cox regression analysis showed significant associations between ischemic events and presence of a thin or ruptured fibrous cap (hazard ratio: 17.0; \( p < 0.001 \)), intraplaque hemorrhage (hazard ratio: 5.2; \( p = 0.005 \)), and larger mean necrotic core area (hazard ratio for 10 mm\(^2\) increase, 1.6; \( p = 0.01 \)) in the carotid plaque. In another prospective study of 64 recently symptomatic patients with 30% to 69% carotid stenosis, baseline carotid CMR scans were performed to identify intraplaque hemorrhage, and subjects were followed up for the development of subsequent transient ischemic attack or stroke (37). Thirty-nine (61%) of the ipsilateral arteries showed intraplaque hemorrhage on baseline CMR. Fourteen ipsilateral ischemic events were observed during follow-up. Thirteen of the 14 events occurred ipsilateral to carotid arteries with intraplaque hemorrhage (hazard ratio: 9.8, 95% confidence interval: 1.3 to 75.1, \( p = 0.03 \)). These studies suggest that carotid CMR may provide additional diagnostic criteria to identify patients with moderate carotid stenosis who are at increased risk for subsequent stroke. Although these initial results are highly promising, larger multicenter studies are needed to confirm the role of carotid plaque imaging in routine clinical practice.

This pictorial essay illustrates the capability of CMR for the identification of the disruption-prone and disrupted carotid plaque.

### Multicontrast-Weighted CMR

The greatest strength of CMR for characterizing atherosclerotic plaque is the availability of multicontrast-weighted protocols using bright- and black-blood techniques. In the past, most applications of carotid CMR have been limited to the evaluation of stenosis using bright-blood magnetic resonance angiography (MRA). These angiographic pulse sequences produce strong attenuation of signals from stationary tissues, limiting their usefulness for direct imaging of the atherosclerotic plaque. Nevertheless, bright-blood MRA using a 3-dimensional time-of-flight (TOF) technique presents specific contrast features that can be helpful in identifying certain plaque components when used in combination with black-blood imaging (16). Black-blood sequences rely on the elimination of the signal from flowing blood and represent a general approach for characterizing the vessel wall, where precise identification of the lumen–wall interface plays a critical role in assessment of morphology and tissue composition of the atherosclerotic plaque (26) (Table 1). Implementation of this multicontrast-weighted CMR protocol has been technically successful in 76% to 90% of cases. Most of the failures are attributable to poor image quality secondary to patient motion (15–18).

Numerous studies have shown that combined intensity information from different contrast weightings (Table 1) can be used to identify all major plaque components, including fibrous tissue, lipid-rich necrotic core, calcification, and intraplaque hemorrhage (8,11–28). Using established guidelines...
for the relative intensities of these features to guide manual outlining, studies have shown that quantitative characterization of plaque composition has excellent correlation with histological measurements (14,15,17,18,23) and is highly reproducible (15). Furthermore, robust automatic classifiers, such as the morphology-enhanced probabilistic plaque segmentations algorithm (27,28), have been developed that automate plaque compositional measurements and achieve similar levels of accuracy and reproducibility. This automated segmentation is based on the fact that various tissue contents, such as lipid, calcium, loose matrix, and fibrous tissue, have different signal characteristics on each magnetic resonance weighting (28). The system first determines the probability that each CMR/pixel belongs to each of the 4 tissue types (lipid, calcification, loose matrix, and fibrous tissue). Afterward, it uses competing active contours (38) to identify the boundaries of the high-probability regions for each tissue type.

### Identification of Disruption-Prone Plaque Features

**The thin fibrous cap and lipid-rich necrotic core.** Studies of advanced atherosclerotic lesions suggest that the thickness of the fibrous cap overlying the necrotic core distinguishes stable lesions from those at high risk for disruption and thromboembolic events (8,16). The combined use of bright- and black-blood techniques can aid in the identification of both the fibrous cap and vessel wall components such as the lipid-rich necrotic core (Fig. 1). Bright-blood TOF allows for the visualization of the vascular lumen and is capable of identifying the status of fibrous cap (16). After the administration of gadolinium diethylene triamine penta-acetic acid, contrast-enhanced T1 weighting differentiates the fibrous cap from the underlying lipid-rich necrotic core and thus allows for the quantification of both components (18,21–24). Accurate quantification of both fibrous cap and lipid-rich necrotic core is crucial for measurement of lesion progression. The spatial resolution of CMR is lower than the proposed histological definition of a thin cap (18). However, prospective studies suggest that when an intact fibrous cap is visualized by CMR (categorically defined as a thick cap), these individuals have a significantly lower risk for future transient ischemic attack or stroke compared with individuals in whom the cap is not visualized (categorized as a thin cap) or in whom the cap is disrupted (25). Thus, cap visualization by CMR may have prognostic value. Quantification of minimal cap thickness and identification of finer structures within the diseased arterial wall will require higher spatial resolution. Improvement in spatial resolution is possible with further technical development, such as a higher field strength scanner and the use of 8-element phased-array carotid coils (39,40).

**Intraplaque hemorrhage.** Intraplaque hemorrhage is frequently observed in carotid atherosclerosis and is

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**Table 1. High-Resolution Multicontrast-Weighted Magnetic Resonance Sequences on 3-T Philips Achieva Magnet**

<table>
<thead>
<tr>
<th>Technique</th>
<th>3-D TOF</th>
<th>PDW</th>
<th>T2W</th>
<th>Pre- and Post-CE T1W</th>
<th>Dynamic CE</th>
<th>IR TFE</th>
<th>BB MRA</th>
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<td>4,000/9</td>
<td>4,000/50</td>
<td>800/10</td>
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For technical details please refer to Yarnykh et al. (26).

3-D TOF = 3-dimensional time-of-flight; BB = black blood; CE = contrast-enhanced; DIR = double inversion recovery; FFE = fast field echo; FOV = field of view; IR = inversion recovery; MRA = magnetic resonance angiography; MS = multislice; NSA = number of signal average; PDW = proton density weighted; QIR = quadruple inversion recovery; T1W = T1-weighted; T2W = T2-weighted; TFE = turbo field echo; TR/TE = repetition time/echo time; TSE = turbo spin echo.

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**Chu et al.**

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purported to arise from plaque neovasculature (Fig. 2). Microvessels can be fragile because of a lack of support by smooth muscle cells and focal discontinuity of the endothelial lining (41). Kolodgie et al. (42) suggested that intraplaque hemorrhage may represent a potent atherogenic stimulus by contributing to the deposition of free cholesterol, macrophage infiltration, and enlargement of the necrotic core in a histopathological study of coronary artery specimens. Immunohistochemical staining with the

**Figure 1. Automated Segmentation of Bright- and Black-Blood, High-Spatial-Resolution, Multicontrast In Vivo CMR**

Quantification of in vivo cardiac magnetic resonance (CMR) (TOF, T1W, T2W, and CE T1W) using CASCADE to generate component outlines. The CASCADE fibrous cap (FC) is an additional algorithm that collects FC length, depth, and area. The automated map of the plaque is produced by the MEPPS algorithm. Loose matrix is shown in purple, lipid-rich necrotic core in yellow, and intraplaque hemorrhage in red on the MEPPS image. Histology from CEA confirms the components and the enhancing thick FC (arrow). In vivo CMR were acquired on a 3T Philips Achieva scanner (Best, the Netherlands) along with the use of an 8-element phased-array carotid coil. CASCADE = Computer-Aided System for Cardiovascular Disease Evaluation; CE = contrast-enhanced; CEA = carotid endarterectomy; MEPPS = morphology-enhanced probabilistic plaque segmentation; TOF = time-of-flight; T1W = T1-weighted; T2W = T2-weighted.

**Figure 2. Identification of Intraplaque Hemorrhage Using High-Spatial-Resolution, Multicontrast In Vivo CMR**

Intraplaque hemorrhage in the right common carotid artery is shown by hyperintense signals on the 3-D TOF, T1W, and IR TFE sequences. Hypointense signals in both the TOF and IR TFE images are characteristic of regions with dense calcification. The CMR were acquired on a 3T Philips Achieva scanner along with the use of an 8-element phased-array carotid coil. IR TFE = inversion recovery turbo field echo; other abbreviations as in Figure 1.
antibody to glycophorin A, a protein specific to erythrocyte membranes, was strongly associated with the size of the necrotic core and degree of macrophage infiltration. The group also noted that rabbit lesions with induced intramural hemorrhage had significantly greater lipid content than control lesions without hemorrhage. Other investigators have noted that erythrocyte membranes contain more free cholesterol than any other cell in the body (43).

High-resolution, multicontrast MRI can accurately detect the presence and age of carotid intraplaque hemorrhage (13,17,20). Prospective CMR studies have shown that hemorrhage into the carotid atherosclerotic plaque is associated with a rapid increase in plaque burden and lipid-rich

![Figure 3. Intraplaque Hemorrhage Is Associated With Rapid Progression of Carotid Atherosclerotic Plaque](image)

Representative T1W images show rapid progression of atherosclerosis with intraplaque hemorrhage in the right carotid artery. Each column presents matched cross-sectional locations in the carotid artery from baseline (A) and 18 months later (B). A marked decrease in lumen area and an increase in wall area are present in each location of the second examination. The CMR were acquired on a 1.5-T GE Signa scanner (Waukesha, Wisconsin) along with the use of a 4-element phased-array carotid coil. Reprinted, with permission, from Takaya et al. (13). Bif = bifurcation; CCA = common carotid artery; ECA = external carotid artery; ICA = internal carotid artery; other abbreviations as in Figure 1.

![Figure 4. Intraplaque Hemorrhage Precedes Carotid Plaque Rupture](image)

Matched baseline CMR show atherosclerotic plaque in a carotid artery with minimal luminal stenosis: (A) 3-D TOF, (B) T2W, (C) pre-contrast-enhanced, T1W and (D) post-contrast-enhanced, T1W. The smooth luminal contour (arrow, A) and the enhancing band on the post-contrast T1W image (D) are characteristic of an intact fibrous cap. Hyperintense signals on images A, B, and C suggest presence of intraplaque hemorrhage. Follow-up CMR E through G (corresponding to images A through C, respectively, in sequence and location) show ulceration into the plaque (open arrows). The ulcer manifests as a hyperintense signal on E, mixed hyper- and hypointense signal on F, hypointense signal on G, and contrast enhancement on H. This signal pattern suggests turbulent flow within the ulcer. The CMR were acquired on a 1.5-T GE Signa scanner along with the use of a 4-element phased-array carotid coil. Reprinted, with permission, from Chu et al. (44). Abbreviations as in Figure 1.
necrotic core size and a decrease in lumen volume (Fig. 3). Furthermore, lesions that had intraplaque hemorrhage at baseline had a greater probability of repeated intraplaque hemorrhage at 18 months (13). Rapid progression and the destabilization of plaque driven by the intraplaque hemorrhage can set the stage for subsequent plaque rupture (44) (Fig. 4).

Plaque inflammation. Inflammation has been described to be an important component of unstable atherosclerotic plaque, whether in the coronary or the carotid vessels, because it contributes to necrotic core size, plaque angiogenesis, and thinning of the fibrous cap through the release of potent matrix metalloproteinases (MMPs) (45,46). Regions of the plaque with high-density macrophages are reported to contain MMPs; some MMPs are co-localized with cleaved collagen (46,47). Therefore, recent emphasis has been placed on macrophages as the primary component of rupture.

Contrast enhancement in carotid atherosclerotic plaques has been observed in recent CMR investigations after the injection of a gadolinium-based contrast agent (18,21–24,36,48) (Fig. 5). Strong contrast enhancement suggests the presence of a vascular supply to the plaque and increased endothelial permeability that facilitates the entry of the contrast agent from the blood plasma. Because neovasculature growth into the plaque and increased endothelial permeability are associated with plaque inflammation, plaque enhancement has been considered a sign of inflammation (36). Because plaque inflammation may have multiple effects that weaken plaque structural integrity, contrast-enhanced CMR may be a tool for detecting plaque inflammation before fibrous cap disruption.

**Figure 5. Carotid Plaque Inflammation**

Plaque with histologically confirmed inflammation of the fibrous cap shows a strong enhancement pattern by dynamic contrast-enhanced cardiac magnetic resonance (DCE CMR), with high transfer constant (green) in the adventitial vasa vasorum (near the outer vessel wall boundary) and along the lumen boundary (inner boundary demarcating red lumen region). Movat pentachrome stains hemorrhage within the lipid-rich necrotic core red. Immunocytochemistry with HAM 56, an antibody to human macrophages, shows an abundance of macrophages in the fibrous cap. Arrows indicate the stenotic lumen of the internal carotid artery. DCE CMR was collected at 10 time points, with a separation interval of 15 s between time points. Gadolinium-based contrast agent (Omniscan, GE Medical Systems; 0.1 mmol/kg body weight) was injected at a rate of 2 ml/s. All CMR were acquired on a 1.5-T GE Signa scanner along with the use of a 4-element phased-array carotid coil.
Dynamic CMR of contrast enhancement permits quantitative analysis of contrast media kinetics. Uptake is characterized by a parameter for fractional plasma volume (\(v_p\)) and by a transfer constant (\(K^{\text{trans}}\)) that reflects blood supply, vessel permeability, and the extracellular space (23,36).

**Figure 6. High Shear Stress Associated With Subsequent Carotid Plaque Rupture**

(A) Matched T1W images with and without superimposed vessel wall segmentation at baseline (top) and 10-month follow-up (bottom). Baseline images show intraplaque hemorrhage (teal) within a large lipid-rich necrotic core (blue). Matched locations on 10-month follow-up CMR show the development of a large ulcer (purple) in the proximal internal carotid artery. The CMR were acquired on a 1.5-T GE Signa scanner along with the use of a 4-element phased-array carotid coil. (B) Baseline wall shear stress mapped at baseline 3-D lumen geometry of a carotid bifurcation including plaque segmentation (A). Plaque segmentation at 10-month follow-up, including the ulcer (B). The average WSS at baseline in the carotid bifurcation was 3.2 ± 2.0 Pa, and the site of ulceration was observed at the highest WSS. Co-localization of high WSS regions at baseline to the site of subsequent ulceration shows the utility of serial CMR in studies examining the relationship between hemodynamic variables, plaque composition, and future plaque disruption. Reprinted, with permission, from Groen et al. (54). WSS = wall shear stress; other abbreviations as in Figure 1.
Because these factors are all modulated by the inflammatory process, kinetic modeling has been suggested as a means for characterizing the effects of plaque inflammation. The integrated area under the enhancement versus time curve has also been used to characterize enhancement dynamics in plaque (49).

Studies involving dynamic CMR in carotid atherosclerotic plaques have shown that $v_p$ correlates strongly with histologically determined neovascular content (23), a key portal for the entry of inflammatory cells into the atherosclerotic plaques. In subsequent studies, $K_{\text{trans}}$ has emerged as the preferred marker of plaque inflammation. In a study of 27 patients with histologic results, $K_{\text{trans}}$ correlated with macrophage ($r = 0.75$, $p < 0.001$), neovascularure ($r = 0.71$, $p < 0.001$), and loose matrix ($r = 0.50$, $p = 0.01$) content (36). In another study of 20 subjects, $K_{\text{trans}}$ was associated with serum markers of inflammation and pro-inflammatory risk factors, including high levels of C-reactive protein, low high-density lipoprotein, and smoking (48).

However, there is overestimation of neovascularure as measured by dynamic CMR versus histology. This overestimation suggests the presence of interstitial volumes undergoing very rapid exchange of the contrast agent with the blood plasma. Any such regions that come to equilibrium within 1 time frame of the dynamic sequence will be indistinguishable from blood and therefore included in $v_p$. Development of advanced techniques that also quantify this rapid exchange may lend further insight into the kinetics of contrast agents in plaque.

Another contrast agent used in plaque imaging uses ultrasmall particles of superparamagnetic iron oxide, which have been shown to accumulate in macrophages within atherosclerotic plaques and to lead to characteristic losses in signal intensity on CMRs (50). Superparamagnetic iron oxides, however, require multiple imaging sessions over periods of 24 h or more (51).
High shear stress. It is well known that high shear stress can destabilize the vascular wall (Figs. 6A and 6B). Shear stress acting on the vessel wall plays an important role in many processes in the cardiovascular system primarily focused on the regulation of vessel lumen and wall dimensions. There is ample evidence that atherosclerotic plaques are generated at low shear stress regions in the cardiovascular system (52). In addition, plaque rupture has been more frequently observed at the proximal, upstream side of the site of maximal stenosis, which is exposed to higher wall shear stress (WSS) (53). It is also hypothesized that high WSS at the upstream side of the plaque has a biological effect on the fibrous cap by inducing antiproliferative activity by endothelial cells and therefore may enhance plaque vulnerability (52–55).

To quantify WSS, computational fluid dynamics models are built with realistic boundary conditions. Imaging techniques such as CMR provide information regarding vascular geometry and inflow conditions. Figure 7 illustrates a computational fluid dynamics model of an atherosclerotic internal carotid artery. After generating the computational mesh, the shear stress field acting on the luminal surface is computed from the velocity field. Although this approach is commonly accepted (54), it is still unclear how variability in geometry reconstruction and restricting assumptions on blood rheology or vessel wall compliance may affect the accuracy of the results.

Phase-contrast CMR, however, is a promising, noninvasive technique for determining various in vivo blood flow characteristics. The WSS of the carotid artery can be assessed semiautomatically with good to excellent reproducibility without interobserver or intraobserver variability using model-based segmentation of phase-contrast CMR by determination of flow volume and maximum flow velocity in cross-sections of these vessels (56). However, WSS is site specific, a finding that opposes the notion that physiological WSS values are maintained at a constant magnitude in all parts of the arterial system. Among the WSS values.
obtained at the same site by different investigators, there is qualitative agreement. However, differences exist in absolute values mainly because of the dependence on the method used to obtain WSS values from velocity data \((57,58)\). Large variations in absolute WSS levels are also reported within one species and between species \((59)\).

**Identification of Luminal Surface Disruption**

**Fibrous cap rupture and thrombus.** Rupture of the fibrous cap, with the resultant exposure of thrombogenic subendothelial plaque constituents, is believed to be the critical event that leads to thromboembolic complications in atherosclerotic carotid artery disease \((5–7)\). Histopathology studies have shown that the prevalence of carotid plaque rupture was significantly higher among patients with a past history of an ischemic neurological event \((5–7,60)\). Disrupted plaques in patients affected by stroke were characterized by the presence of a more severe inflammatory infiltrate compared with that observed in the transient ischemic attack and asymptomatic groups \((6)\).

High-resolution CMR with a 3-dimensional TOF protocol is capable of distinguishing intact, thick fibrous caps from intact thin and disrupted caps in atherosclerotic human carotid arteries in vivo \((8,16)\) (Fig. 8). With the addition of contrast-enhanced T1-weighted sequence, magnetic resonance can more readily identify the intact, thick fibrous cap \((18)\) (Figs. 1 and 4). Identification of fibrous cap rupture with CMR is highly associated with recent transient ischemic attack or stroke \((8)\).

**Ulceration and thrombus.** Studies of carotid endarterectomy specimens have shown that plaque ulceration and thrombosis are more prevalent in symptomatic patients \((5–7)\). Ulceration is more common in symptomatic patients regardless of side of carotid symptoms, whereas thrombus is associated with ipsilateral symptoms and plaque ulceration \((5)\). A thrombocytically active carotid plaque associated with high inflammatory infiltrate was observed in 71 of 96 (74.0%) patients with ipsilateral major stroke compared with 32 of 91 (35.2%) patients with transient ischemic attack \((p < 0.001)\) or 12 of 82 (14.6%) patients who were without symptoms in a study \((6)\).

Case studies have shown promise in the detection of carotid atherosclerotic ulceration using multisequence cross-sectional CMR \((44,61)\) (Figs. 9 and 10). Adding longitudinal black-blood MRA to multisequence high-spatial-resolution cross-sectional CMRs can increase the ability of CMR to identify carotid plaque ulceration \((62)\) (Fig. 11).

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**Figure 9. Carotid Artery Ulceration and Thrombus Formation**

A surface disruption is clearly visible on TOF and T1W images. The crescent-shaped high signal on the PDW, T2W, and CE T1W images is caused by the infiltration of blood and contrast into the thrombus filling the disruption. Gadolinium-based contrast agent (Omniscan, GE Medical Systems; 0.1 mmol/kg body weight) was injected for CE T1W image acquisition. Histology from the same location confirms the disruption and subsequent thrombus formation. All CMR are acquired on a 1.5-T GE Signa along with the use of a 4-element phased-array carotid coil. PDW = proton density-weighted; other abbreviations as in Figure 1.
Calcified nodules. Calcified nodules were first reported in acutely thrombosed coronary arteries, but have since been reported in carotid arteries. Surface nodules can have exposed thrombogenic surfaces or can be encapsulated (63). Transverse CMRs show the usefulness of TOF in the multisequence magnetic resonance protocol for detecting nodules that present with the same hypointense signal as the background blood in black-blood sequences (64) (Fig. 12).

Figure 10. Carotid Artery Plaque Ulceration

Although image quality is marginal, the large surface ulceration is clearly visible on all contrast weightings (T1W, PDW, T2W, and DCE CMR). Histology confirms the presence of a large ulceration into a fatty, cholesterol-laden necrotic core. All CMR were acquired on a 1.5-T GE Signa along with the use of a 4-element phased-array carotid coil. The DCE CMR was obtained after the injection of a gadolinium-based contrast agent (Omniscan, GE Medical Systems; 0.1 mmol/kg body weight injected at a rate of 2 ml/s using a power injector).

DCE = dynamic contrast-enhanced; H&E = hematoxylin and eosin; ICA = internal carotid artery; other abbreviations as in Figure 1.

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Figure 11. Progression of Carotid Plaque Ulceration

Cross-sectional CMR and oblique black-blood magnetic resonance angiography (BB MRA) collected at baseline and at 2 years show the irregular surface of the carotid artery at the bulb containing a penetrating ulcer (chevron). The internal carotid artery is indicated by the asterisk. The TOF image obtained 2 years later shows a marked increase in signal from baseline illustrating the enlargement of the ulceration (arrow). All CMR were acquired on a 1.5-T GE Signa along with the use of a 4-element phased-array carotid coil. Abbreviations as in Figure 1.
Summary

This review shows the value of high-spatial-resolution, multicontrast-weighted CMR techniques in the identification of the disruption-prone and disrupted carotid atherosclerotic plaque. Based on these histologically verified techniques, we highlight plaque features that are associated with rapid progression, surface disruption, and an increased risk for subsequent ischemic events. These features include the necrotic core, intraplaque hemorrhage, the thin and ruptured fibrous cap, ulceration, and calcified nodules. In addition, CMR lends itself to collecting information on WSS that may influence both rupture of the fibrous cap and plaque progression.

We expect that new and continuing advances in CMR technology, such as higher field strength, dedicated phased-array coils for higher signal-to-noise ratio and larger coverage of carotid artery, multislice motion-sensitized driven-equilibrium turbo spin-echo sequences to improve suppression of plaque-mimicking artifacts (65), and 3-dimensional isotropic sequences for better luminal surface and plaque delineation (66), will provide even more tools to better characterize the vulnerable atherosclerotic carotid plaque.

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