Conclusions—Measures of plaque burden do not substantially improve disease assessment compared to stenosis. The finding of IPH in all categories of stenosis and plaque burden suggests that direct characterization of plaque composition and surface status is necessary to fully discriminate disease severity. 

Key Words: atherosclerosis ■ carotid artery ■ magnetic resonance imaging ■ plaque burden ■ stenosis

Carotid artery atherosclerosis is a major etiology of cerebrovascular events such as transient ischemia attack and stroke.1 Because of the well-defined association between severity of luminal stenosis and clinical events, carotid stenosis currently represents the principal criterion for ischemic risk stratification and surgical intervention.2 However, the occurrence of stroke in patients with mild to moderate (<70%) carotid stenosis suggests that stenosis may not be the strongest classifier of atherosclerotic disease severity.3 In addition, high-risk features of atherosclerosis, such as intraplaque hemorrhage (IPH) and surface disruption, have been reported across all stenotic categories (0%–99%).4,5 As such, alternate or complementary strategies for evaluating carotid atherosclerotic disease have become increasingly desirable.

The arterial remodeling hypothesis,6 defined as the preservation of the lumen via outward expansion of the arterial wall in response to atherosclerosis, has led to the development of strategies for evaluating the arterial wall. In the coronary artery, measures of plaque burden have been used to characterize disease severity and occur after response to therapy. In particular, Nissen et al7 have proposed percent atheroma volume (100% × atheroma volume/total vessel volume) during invasive intravascular ultrasound investigations as a parameter that captures plaque burden while accounting for inherent differences in arterial size. An analogous measurement in the carotid artery obtained noninvasively via high-resolution black-blood MRI is percent wall volume (PWV = 100% × wall volume/total vessel volume). As an alternative to PWV, arterial wall thickness might also repre-
sent a viable measure of local disease severity. B-mode ultrasound has been shown to effectively measure the intima-media thickness of the arterial wall. Multiple long-term, prospective investigations have shown that carotid intima-media thickness is associated with stroke. Recently, a strong association has been shown between intima-media thickness measured by B-mode ultrasound and mean wall thickness (MWT) measured by carotid MRI.

We hypothesized that measures of plaque burden (ie, PWV and MWT) would be stronger classifiers of high-risk features, specifically IPH and surface disruption, than stenosis. This study sought to determine: (1) associations between stenosis, PWV, MWT, and the compositional features of carotid atherosclerotic disease; and (2) strength of plaque burden in classifying carotid high-risk features. Identification of a parameter that accurately classifies the presence or absence of these features may prove valuable in advancing the accurate assessment of carotid disease severity.

Materials and Methods

Study Sample
Carotid MRI data were pooled from participants recruited at a single academic medical center either for >50% stenosis by duplex ultrasound in at least 1 carotid artery or for coronary CTA for suspected coronary artery disease attributable to chest pain. Carotid duplex ultrasound examinations were performed by a singular operator with 3 years of experience. Utilization of images from the cohort with suspected coronary artery disease has been previously shown to yield arteries with normal to moderate carotid atherosclerotic disease, thus ensuring a broad distribution of carotid atherosclerosis for the sample population as a whole. For each cohort, the artery selected for imaging with carotid MRI, termed the index artery, was determined by recruitment strategy. In subjects with >50% stenosis in at least 1 carotid artery, the artery with the greatest amount of stenosis on duplex ultrasound was assigned as the index artery. For subjects recruited from coronary CTA, the index artery was randomly assigned. Clinical information was obtained through chart review. The Institutional Review Board approved the research protocol before study initiation and informed consent was obtained from all participants.

Carotid MRI Protocol
All images were acquired on a 3.0-T whole-body scanner (Signa Excite; General Electric Medical Systems) utilizing a 4-channel, phased-array carotid surface coil. A standardized protocol adapted for imaging at 3.0 T was used to obtain 4 contrast-weighted images of the index carotid artery in the transverse plane: T1-weighted, proton-density weighted, T2-weighted, and 3-dimensional time-of-flight MRA. The scan was longitudinally centered on the bifurcation of the index artery. MRI parameters are listed in the Supplemental Table (available online at http://stroke.ahajournals.org). Postcontrast T1-weighted images were acquired for subjects with >50% carotid stenosis on duplex ultrasound. Gadodiamide (Omniscan; GE Healthcare) was administered intravenously with a dose of 0.1 mmol/kg at a rate of 1 mL/sec 5 minutes before contrast-enhanced T1-weighted image acquisition. For patients with carotid >50% stenosis, the carotid MRI was performed within 2 weeks after carotid ultrasound examinations. For patients with suspected coronary artery disease, the carotid arteries were imaged by MR within 1 week after coronary CTA.

Image Analysis
Two trained reviewers with 2 years of experience in reading carotid MR images, blinded to clinical information and stenosis measurements, evaluated image quality and interpreted the multi-contrast images of the index carotid artery via consensus opinion. For each index artery, an image quality rating was assigned using a 4-point scale (1=poor, 4=excellent). Arteries with image quality <2 were excluded from analysis. For the remaining interpretable arteries, a software package (CASCADE, Seattle, WA) was used to measure the lumen area, wall area, total vessel area, and mean wall thickness at each axial location. The PWV was subsequently determined from the wall volume and total vessel volume for each index artery. The average MWT across all axial locations defined the MWT for each index artery. The presence or absence of carotid compositional features, including calcification, lipid-rich necrotic core (LRNC), IPH, and surface disruption (ulceration or fibrous cap rupture), was determined using previously published multi-contrast criteria validated with histology. Using CASCADE, the intrareader and inter-reader coefficients of variation were from 3.0% to 7.2% in quantifying the carotid lumen and wall area, and the intraclass correlation coefficient was from 0.73 to 0.95 in quantitative evaluation of carotid plaque compositions.

Maximum intensity projection images were reconstructed from the 3-dimensional time-of-flight MRA images using GE software (GE Medical System Advantage Workstation 4.3). Using the same GE software package, a single reviewer with 3 years of experience in cardiovascular radiology, blinded to images and results from multi-contrast carotid MRI, measured luminal stenosis using NASCET criteria (percent stenosis=100%×[1−luminal diameter at the point of maximal narrowing/the diameter of the normal distal internal carotid artery]).

Data Analysis
The stenosis, PWV, and MWT between index arteries with and without each carotid compositional feature, such as calcification, LRNC, IPH, and surface disruption, were compared using the independent t test. Logistic regression analysis was used to calculate the OR and corresponding 95% CI for stenosis, PWV, and MWT. Receiver-operating characteristics analysis was used to determine the strength of classification for stenosis, PWV, and MWT for determining the presence vs absence of each compositional feature. The area under the curve and corresponding 95% CI are reported. Prevalence of carotid compositional features for different categories of stenosis, PWV, and MWT are reported. Stenosis and PWV were categorized by 10% intervals. MWT was partitioned into the following 5 categories: <1.0 mm, 1.0 to 1.5 mm, 1.5 to 2.0 mm, 2.0 to 2.5 mm, and >2.5 mm. Pearson correlation coefficient, r, was used to determine associations between stenosis and PWV. All analyses were performed with SPSS for Windows (version 12.0; SPSS). Statistical significance was defined as a value of P<0.05.

Results
From October 2005 to February 2009, 190 participants (N=67, duplex stenosis >50%; N=123, coronary CTA) underwent carotid MRI. Of the 190 participants, 9 (4.7%) were excluded because of total occlusion in the index carotid artery. All of the remaining 181 index arteries had sufficient image quality for comprehensive plaque interpretation. The demographics and arterial characteristics for this cohort are reported in Table 1. The mean±SD of stenosis, PWV, and MWT for this population were 21.3%±27.5%, 43.7%±14.5%, and 1.3±0.6 mm, respectively.

Compared to carotid arteries without each compositional feature, those arteries with the corresponding compositional feature exhibited significantly greater stenosis, PWV, and MWT (all P<0.001; Table 2). In addition, logistic regression analysis found that stenosis, PWV, and MWT were significant predictors of all carotid compositional features (OR, 1.42–4.51; all P<0.001; Table 2). Receiver-operating characteristic analysis found that measurements of stenosis, PWV, and MWT were strong classifiers of carotid composi-
Table 2. Association of Carotid Plaque Features With Stenosis and Plaque Burden

<table>
<thead>
<tr>
<th>Plaque Feature</th>
<th>Presence Mean±SD</th>
<th>Absence Mean±SD</th>
<th>P*</th>
<th>OR (95% CI)</th>
<th>Area Under the Curve (95% CI)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Calcification</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis (%)</td>
<td>33.1±28.7</td>
<td>8.2±19.0</td>
<td>&lt;0.001</td>
<td>1.57 (1.33–1.86)</td>
<td>&lt;0.001</td>
<td>0.78 (0.71–0.83)</td>
</tr>
<tr>
<td>PWV (%)</td>
<td>50.6±13.7</td>
<td>36.1±11.0</td>
<td>&lt;0.001</td>
<td>2.49 (1.85–3.35)</td>
<td>&lt;0.001</td>
<td>0.81 (0.75–0.87)</td>
</tr>
<tr>
<td>MWT (mm)</td>
<td>1.6±0.6</td>
<td>1.1±0.6</td>
<td>&lt;0.001</td>
<td>1.42 (1.24–1.64)</td>
<td>&lt;0.001</td>
<td>0.84 (0.78–0.89)</td>
</tr>
<tr>
<td><strong>Lipid-rich necrotic core</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis (%)</td>
<td>27.6±28.3</td>
<td>4.4±15.5</td>
<td>&lt;0.001</td>
<td>1.83 (1.38–2.43)</td>
<td>&lt;0.001</td>
<td>0.78 (0.71–0.83)</td>
</tr>
<tr>
<td>PWV (%)</td>
<td>47.9±14.3</td>
<td>32.5±6.7</td>
<td>&lt;0.001</td>
<td>4.03 (2.37–6.86)</td>
<td>&lt;0.001</td>
<td>0.84 (0.78–0.89)</td>
</tr>
<tr>
<td>MWT (mm)</td>
<td>1.5±0.7</td>
<td>0.9±0.2</td>
<td>&lt;0.001</td>
<td>3.58 (2.07–6.19)</td>
<td>&lt;0.001</td>
<td>0.89 (0.83–0.93)</td>
</tr>
<tr>
<td><strong>Intraplaque hemorrhage</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis (%)</td>
<td>46.4±27.0</td>
<td>14.4±23.3</td>
<td>&lt;0.001</td>
<td>1.51 (1.31–1.74)</td>
<td>&lt;0.001</td>
<td>0.82 (0.76–0.87)</td>
</tr>
<tr>
<td>PWV (%)</td>
<td>59.5±11.5</td>
<td>39.4±12.0</td>
<td>&lt;0.001</td>
<td>3.09 (2.17–4.37)</td>
<td>&lt;0.001</td>
<td>0.88 (0.82–0.92)</td>
</tr>
<tr>
<td>MWT (mm)</td>
<td>2.0±0.7</td>
<td>1.1±0.5</td>
<td>&lt;0.001</td>
<td>1.59 (1.37–1.84)</td>
<td>&lt;0.001</td>
<td>0.88 (0.82–0.92)</td>
</tr>
<tr>
<td><strong>Surface disruption</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis (%)</td>
<td>53.5±23.7</td>
<td>15.6±24.0</td>
<td>&lt;0.001</td>
<td>1.58 (1.34–1.86)</td>
<td>&lt;0.001</td>
<td>0.87 (0.81–0.92)</td>
</tr>
<tr>
<td>PWV (%)</td>
<td>64.1±8.7</td>
<td>40.1±12.1</td>
<td>&lt;0.001</td>
<td>4.51 (2.68–7.60)</td>
<td>&lt;0.001</td>
<td>0.93 (0.88–0.96)</td>
</tr>
<tr>
<td>MWT (mm)</td>
<td>2.3±0.6</td>
<td>1.2±0.5</td>
<td>&lt;0.001</td>
<td>1.96 (1.56–2.48)</td>
<td>&lt;0.001</td>
<td>0.94 (0.90–0.97)</td>
</tr>
</tbody>
</table>

MWT indicates mean wall thickness; PWV, percent wall volume.
*P is calculated by independent t test.
†P is calculated by logistic regression with 10% increment for both stenosis and PWV and 0.2-mm increment for MWT, respectively.
‡P is calculated by receiver-operating characteristic analysis.

There was a significant correlation between PWV and stenosis (r=0.80; P<0.001; Figure 2). Scatter plots (Figure 2)
demonstrate a general linear trend, but a wide distribution of PWV for all levels of stenosis was evident. Points labeled according to the presence or absence of calcification (Figure 2A), LRNC (Figure 2B), IPH (Figure 2C), or surface disruption (Figure 2D) indicate that each plaque feature occurred across a range of both PWV and stenosis. Calcification, LRNC, and IPH were present in nearly the full range of stenosis and PWV, whereas surface disruption started to occur in arteries with greater stenosis and PWV than other compositional features. Figure 3 shows a general increasing prevalence of carotid compositional features with increasing categories of stenosis (Figure 3A), PWV (Figure 3B), and MWT (Figure 3C). Of note, IPH was identified in lesions with 0% stenosis, with PWV <40%, and in the lowest MWT category (<1.0 mm; Figure 4). In contrast, surface disruption occurred in arteries with stenosis ≥17.4%, PWV ≥48.0%, or MWT ≥1.29 mm in this cohort.

**Discussion**

This study is one of the first to our knowledge to directly compare carotid luminal stenosis and plaque burden measurements in classifying presence or absence of carotid compositional features. In this study, we found that greater plaque burden, as measured by PWV and MWT, and increased luminal stenosis were significantly associated with the presence of calcification, LRNC, IPH, and surface disruption. In addition, plaque burden and stenosis were strong classifiers for the presence of each of these compositional features. However, high-risk features (ie, IPH and surface disruption) were identified across a wide range of plaque burden parameters and stenosis. Moreover, IPH was present not only in lesions with 0% stenosis but also within the plaque burden categories indicative of the earliest disease. Although unexpected, these findings offer compelling evidence for direct evaluation of plaque composition and surface status to adequately differentiate severity of disease rather than the continued reliance on conventional utilization of indirect markers (eg, stenosis and plaque burden measurements).

The identification of IPH and surface disruption across the full range of stenosis has been previously reported. Saam et al\(^4\) found that complicated plaques, which included lesions with either IPH or surface disruption, occurred in 21.7% of arteries with 16% to 49% stenosis and in 8.1% of arteries with 1% to 15% stenosis by duplex ultrasound. Recently, Dong et al\(^5\) reported that in individuals with 0% stenosis on contrast-enhanced MRA, the prevalence of IPH and surface disruption were 8.7% and 4.3%, respectively. For IPH, our findings are in strong agreement with these previous studies.\(^4,5\) For surface disruption, we found a lower prevalence in lesions with 0% stenosis compared to that in the study by Dong et al.\(^5\) Differences may be consequent of the disparity in the sample populations. Dong et al\(^5\) recruited patients with >50% stenosis in 1 carotid artery and used the contralateral artery for their analysis. The presence of advanced carotid disease in the contralateral artery may have predisposed the sample population to have a higher prevalence of surface
disruption in the index artery, despite no measurable luminal encroachment. In the study reported herein, the artery with greater stenosis was utilized in patients with known preexisting carotid atherosclerosis. We observed the development of stenosis and increased plaque burden before surface disruption in our cohort. These findings are consistent with the development of a sufficiently large LRNC before disruption, which recently has been reported as the strongest predictor of future surface disruptions. However, in consideration of the findings by Dong et al., advanced disease in 1 carotid artery may predispose plaque instability on the contralateral side, regardless of the measure of disease severity. Longitudinal studies that follow bilateral carotid disease are necessary to better-appreciate the relationships or, perhaps, independence of carotid disease within the same individual.

The role of IPH in carotid atherosclerotic disease has become increasingly prominent. Prospective studies have demonstrated a significant association between IPH and ischemic events. In addition, IPH has been reported to accelerate plaque progression in carotid arteries and predict recurrent cerebrovascular events. Notably, Underhill et al. recently reported that the presence of IPH in lesions with 16% to 49% stenosis at baseline not only accelerated plaque growth but also was associated with inward remodeling. Moreover, they observed that IPH may mitigate the positive effects of statin therapy. Collectively, these studies indicate that identification of IPH may be a necessary and critical aspect in differentiating disease severity. Although there was a higher prevalence of IPH in lesions with more stenosis and increased plaque burden, the presence of IPH spanned the entire range of these parameters. Accordingly, patients with negative traditional imaging findings but with a history of ischemic events or an increased clinical risk of ischemic events may warrant imaging for the detection of IPH. Sequences developed specifically for IPH detection in the carotid artery have been developed and validated at 3.0 T. The utilization of a single contrast weighting for IPH detection may prove cost-effective in the management of susceptible individuals for the prevention of stroke.

In this study, we used PWV as a plaque burden measurement in the carotid artery. Beyond the correspondence to a well-established parameter in the coronary artery, there are additional factors that motivate the selection of PWV for assessment of disease severity in the carotid artery. First, PWV has been shown to have the highest measure of reproducibility across all measures of carotid plaque morphology with MRL. Second, PWV fully exploits the 3-dimensional data acquisition inherent to
carotid MRI. Finally, high levels of in vivo plaque burden, as measured by PWV, have been identified across the full spectrum of stenosis in the carotid artery. Importantly, the latter factor suggests that PWV may provide unique information compared to stenosis measurements.

Similar to Babiarz et al, we found a significant association between PWV and stenosis. However, at each level of stenosis there was a wide spread of PWV. Glagov et al originally observed from observations of postmortem coronary arteries that luminal narrowing occurred after the atherosclerotic lesion occupied >40% of the internal elastic lamina area. Our data suggest that arterial remodeling may occur differently in the carotid artery and/or may be dependent on additional factors beyond plaque burden. Underhill et al hypothesized that the presence of IPH or alternative change in composition may alter the biological behavior of the lesion. Such a phenomenon previously has been described in balloon-injured rabbits. Other factors beyond plaque composition may also govern arterial remodeling, such as shear stress. Accordingly, large prospective studies that investigate a variety of demographic, serological, and imaging parameters (e.g., inflammation, neovasculature) are necessary to identify factors that contribute to differential remodeling patterns.

In this study, the carotid black-blood MRI technique was utilized to determine the compositional features of atherosclerotic plaque. This histologically validated technique provided unique plaque compositional information related to plaque vulnerability beyond luminal stenosis. However, there were several study limitations. First, 2 groups of patients with heterogeneous demographics were recruited. Second, slightly different imaging protocols were used in these 2 groups of subjects. A better-designed future study may include a larger sample size, homogenous inclusion criteria and imaging protocol, multiple races, and fair gender distribution. Additionally, the new developed imaging techniques for detecting IPH and thrombus might be helpful in characterizing vulnerable plaques in future studies.

**Conclusion**

In conclusion, luminal stenosis and measures of plaque burden are associated with the composition of carotid plaques. However, high-risk features such as IPH and surface disruption occur across a wide range of both parameters. In addition, measures of plaque burden do not substantially improve assessment of carotid disease severity compared to the traditional criterion of luminal stenosis. As such, direct imaging assessment for the detection of IPH and surface disruption appears necessary to adequately assess disease severity. Additionally, patients with negative imaging findings by traditional criteria but with a history of ischemic events or strong clinical risk factors for ischemic events may warrant vessel wall imaging.
Sources of Funding
This study is supported in part by a grant from GE Healthcare and the National Institutes of Health (RO1 HL 56874).

Disclosure
None.

References
Discriminating Carotid Atherosclerotic Lesion Severity by Luminal Stenosis and Plaque Burden: A Comparison Utilizing High-Resolution Magnetic Resonance Imaging at 3.0 Tesla

Xihai Zhao, Hunter R. Underhill, Qian Zhao, Jianming Cai, Feiyu Li, Minako Oikawa, Li Dong, Hideki Ota, Thomas S. Hatsukami, Baocheng Chu and Chun Yuan

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### SUPPLEMENTAL MATERIAL

Supplemental Table. Carotid MR imaging parameters.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Black-blood PDW</th>
<th>Black-blood T2W</th>
<th>Black-blood T1W</th>
<th>TOF MRA</th>
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<tr>
<td>Contrast</td>
<td>No</td>
<td>No</td>
<td>No/Yes</td>
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<td>FSE</td>
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<tr>
<td>Blood suppression(^b)</td>
<td>MDIR</td>
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<td>QIR</td>
<td>Saturation-veins</td>
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<td>2D</td>
<td>3D</td>
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<td>3000</td>
<td>800</td>
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<tr>
<td>TE, msec</td>
<td>10-13.1(^c)</td>
<td>60-66(^c)</td>
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<tr>
<td>FOV, cm</td>
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<tr>
<td>Matrix size</td>
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<tr>
<td>Spatial Resolution, mm(^2)</td>
<td>0.55x0.55</td>
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<td>Slice thickness, mm</td>
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<td>Slice number</td>
<td>12-16</td>
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<td>12-16</td>
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<tr>
<td>Number of excitations</td>
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<td>1</td>
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<td>Special parameters</td>
<td>Echo train 12;</td>
<td>Echo train 12;</td>
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<tr>
<td></td>
<td>8 slices/TR</td>
<td>8 slices/TR</td>
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<tr>
<td>Fat suppression</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

\(^a\) GE acronyms. \(^b\) Special blood-suppression techniques are multislice double inversion-recovery (MDIR) and quadruple inversion-recovery (QIR) performed with custom-designed software. \(^c\) PDW and T2W sequences were acquired using a shared echo technique (double-echo imaging).
Abstract

Discriminating Carotid Atherosclerotic Lesion Severity by Luminal Stenosis and Plaque Burden
— A Comparison Utilizing High-Resolution Magnetic Resonance Imaging at 3.0 Tesla

Xihai Zhao, MD, PhD1,2; Hunter R. Underhill, MD3; Qian Zhao, MD, PhD; Jianming Cai, MD, PhD; Feiyu Li, MD, PhD; Mirako Oikawa, MD, PhD; Li Deng, MD; Hideki Ota, MD, PhD; Thomas S. Hatushikami, MD; Baosheng Chiu, MD, PhD3; Chun Yuan, PhD1,2

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Stoke 2011; 42: 347-353

図3: 石灰化、PLA、IIPH・ブラスト面の破壊

図4: 左内脳動脈 (*) にブラスト内出血を持つ動脈硬化性ブラスト (DAS-BBB) が認められた。この症例はブラスト量が少なく、MRA (磁場重量状態) に異常を認めなかった。