

ORIGINAL RESEARCH

Carotid Plaque-RADS

A Novel Stroke Risk Classification System



Luca Saba, MD,^a Riccardo Cau, MD,^a Alessandro Murgia, MD,^a Andrew N. Nicolaides, MS, DSc,^{b,c,d} Max Wintermark, MD,^e Mauricio Castillo, MD,^f Daniel Staub, MD,^g Stavros K. Kakkos, MD, PhD,^h Qi Yang, MD, PhD,ⁱ Kosmas I. Paraskevas, MD,^j Chun Yuan, PhD,^k Myriam Edjlali, MD, PhD,^{l,m} Roberto Sanfilippo, MD,ⁿ Jeroen Hendrikse, MD, PhD,^o Elias Johansson, MD,^p Mahmud Mossa-Basha, MD,^k Niranjan Balu, MD, PhD,^q Martin Dichgans, MD,^{r,s} David Saloner, PhD,^t Daniel Bos, MD, PhD,^{u,v,w,x} H. Rolf Jager, MD,^y Ross Naylor, MD,^z Gavino Faa, MD,^{aa} Jasjit S. Suri, PhD,^{bb} Justin Costello, MD,^{cc} Dorothee P. Auer, MD,^{dd} J. Scott McNally, MD, PhD,^{ee} Leo H. Bonati, MD,^{ff} Valentina Nardi, MD,^{gg} Aad van der Lugt, MD,^u Maura Griffin, MSc, PhD,^b Bruce A. Wasserman, MD,^{hh} M. Eline Kooi, MD,ⁱⁱ Jonathan Gillard, MD,^{jj} Giuseppe Lanzino, MD,^{kk} Dimitri P. Mikhailidis, MD,^{ll} Daniel M. Mandell, MD, PhD,^{mm} John C. Benson, MD,ⁿⁿ Dianne H.K. van Dam-Nolen, MD,^u Anna Kopczak, MD,^{oo} Jae W. Song, MD,^{pp} Ajay Gupta, MD,^{qq} J. Kevin DeMarco, MD,^{rr} Seemant Chaturvedi, MD,^{ss} Renu Virmani, MD,^{tt} Thomas S. Hatsukami, MD,^q Martin Brown, MD,^{uu} Alan R. Moody, MD,^{vv} Peter Libby, MD,^{ww} Andreas Schindler, MD,^{xx,*} Tobias Saam, MD^{yy,zz,*}

ABSTRACT

BACKGROUND Carotid artery atherosclerosis is highly prevalent in the general population and is a well-established risk factor for acute ischemic stroke. Although the morphological characteristics of vulnerable plaques are well recognized, there is a lack of consensus in reporting and interpreting carotid plaque features.

OBJECTIVES The aim of this paper is to establish a consistent and comprehensive approach for imaging and reporting carotid plaque by introducing the Plaque-RADS (Reporting and Data System) score.

METHODS A panel of experts recognized the necessity to develop a classification system for carotid plaque and its defining characteristics. Using a multimodality analysis approach, the Plaque-RADS categories were established through consensus, drawing on existing published reports.

RESULTS The authors present a universal classification that is applicable to both researchers and clinicians. The Plaque-RADS score offers a morphological assessment in addition to the prevailing quantitative parameter of "stenosis." The Plaque-RADS score spans from grade 1 (indicating complete absence of plaque) to grade 4 (representing complicated plaque). Accompanying visual examples are included to facilitate a clear understanding of the Plaque-RADS categories.

CONCLUSIONS Plaque-RADS is a standardized and reliable system of reporting carotid plaque composition and morphology via different imaging modalities, such as ultrasound, computed tomography, and magnetic resonance imaging. This scoring system has the potential to help in the precise identification of patients who may benefit from exclusive medical intervention and those who require alternative treatments, thereby enhancing patient care. A standardized lexicon and structured reporting promise to enhance communication between radiologists, referring clinicians, and scientists. (J Am Coll Cardiol Img 2024;17:62-75) © 2024 by the American College of Cardiology Foundation.

From the ^aDepartment of Radiology, University of Cagliari, Cagliari, Italy; ^bVascular Screening and Diagnostic Centre, Nicosia, Cyprus; ^cUniversity of Nicosia Medical School, Nicosia, Cyprus; ^dDepartment of Vascular Surgery, Imperial College, London, United Kingdom; ^eDepartment of Neuroradiology, The University of Texas MD Anderson Center, Houston, Texas, USA; ^fDepartment of Radiology, University of North Carolina, Chapel Hill, North Carolina, USA; ^gVascular Medicine/Angiology, University Hospital Basel and University of Basel, Basel, Switzerland; ^hDepartment of Vascular Surgery, University of Patras Medical School, Patras, Greece; ⁱDepartment of Radiology, Xuanwu Hospital, Capital Medical University, Beijing, China; ^jDepartment of Vascular Surgery, Central Clinic of Athens, Athens, Greece; ^kDepartment of Radiology, University of Washington,

Noninvasive carotid imaging modalities have demonstrated their ability to characterize plaque features as predictors of future events, offering a significant contribution to risk stratification and patient management.¹ Translation of the present knowledge on plaque vulnerability into routine clinical practice requires a standardized reporting system.

The 2017 European Society of Cardiology clinical practice guidelines have recognized these developments and recommend evaluating the presence of plaque imaging characteristics that may indicate an increased risk of ipsilateral stroke additionally to the degree of carotid stenosis in asymptomatic individuals.² These include, among others, intraplaque hemorrhage (IPH) or lipid-rich necrotic core (LRNC) on magnetic resonance imaging (MRI) and large or echolucent plaques or increased juxtaluminal black (hypoechoic) areas on

carotid ultrasound.² Similarly, the European Society for Vascular Surgery clinical practice guidelines emphasize the importance of plaque vulnerability assessment.³ Although scoring systems for singular modalities have been suggested (eg, American Heart Association [AHA] lesion-types, modified AHA lesion-types for MRI, carotid plaque score for ultrasound, etc), there is still no universal classification system for various imaging modalities that scores the severity of an atherosclerotic lesion based on plaque morphology and composition.⁴⁻⁶ The proposed Plaque-Reporting and Data System (RADS) score aims to create an intuitive, accurate, reliable, and standardized scoring system that can be used with various imaging modalities to provide risk estimates for first-time or recurrent large artery cerebrovascular events.

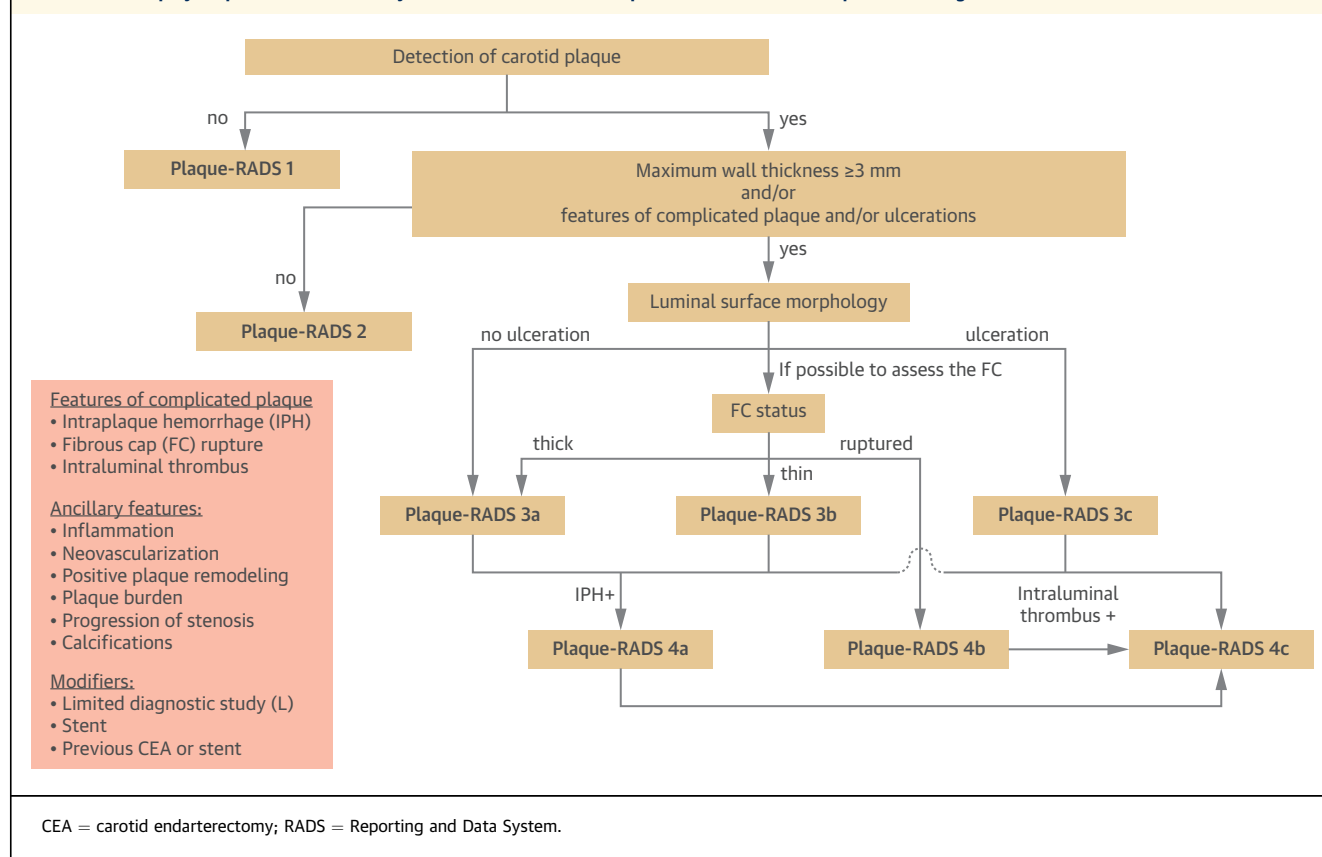
ABBREVIATIONS AND ACRONYMS

CEA	= carotid endarterectomy
CTA	= computed tomography angiography
FC	= fibrous cap
IPH	= intraplaque hemorrhage
LRNC	= lipid-rich necrotic core
MRI	= magnetic resonance imaging
MWT	= maximum wall thickness
RADS	= reporting and data system

Seattle, Washington, USA; ¹Multimodal Biomedical Imaging Laboratory (BioMaps), Paris-Saclay University, CEA, CNRS, Inserm, Frédéric Joliot Hospital Department, Orsay, France; ²Department of Radiology, APHP, Paris, France; ³Department of Vascular Surgery, University of Cagliari, Cagliari, Italy; ⁴University Medical Center Utrecht, Utrecht, the Netherlands; ⁵Clinical Science, Umeå University, Neurosciences, Umeå, Sweden; ⁶Department of Surgery, University of Washington, Seattle, WA, USA; ⁷Institute for Stroke and Dementia Research, University Hospital, LMU Munich, Munich, Germany; ⁸Munich Cluster for Systems Neurology (SyNergy), Munich, Germany; ⁹German Center for Neurodegenerative Diseases (DZNE), Munich, Germany; ¹⁰Department of Radiology and Biomedical Imaging, University of California-San Francisco, San Francisco, California, USA; ¹¹Department of Radiology and Nuclear Medicine, Erasmus MC Rotterdam, University Medical Center Rotterdam, Rotterdam, the Netherlands; ¹²Department of Epidemiology, Erasmus MC, Rotterdam, the Netherlands; ¹³Department of Cardiovascular Sciences, KU Leuven, Leuven, Belgium; ¹⁴Department of Clinical Epidemiology, Harvard School of Public Health, Boston, Massachusetts, USA; ¹⁵Lysholm Department of Neuroradiology and the Neuroradiological Academic Unit, Department of Brain Repair and Rehabilitation, UCL Institute of Neurology, London, United Kingdom; ¹⁶The Leicester Vascular Institute, Glenfield Hospital, Leicester, United Kingdom; ¹⁷Department of Pathology, University of Cagliari, Cagliari, Italy; ¹⁸Stroke Monitoring and Diagnostic Division, AtheroPoin, Roseville, California, USA; ¹⁹Department of Neuroradiology, Walter Reed National Military Medical Center and Uniformed Services University of Health Sciences, Bethesda, Maryland, USA; ²⁰Radiological Sciences, Division of Clinical Neuroscience, and NIHR Nottingham Biomedical Research Centre, University of Nottingham, Nottingham, United Kingdom; ²¹Department of Radiology, University of Utah, Salt Lake City, Utah, USA; ²²Department of Neurology and Stroke Center, Department of Clinical Research, University Hospital Basel, University of Basel, Basel, Switzerland; ²³Department of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota, USA; ²⁴Department of Radiology, University of Maryland School of Medicine and Johns Hopkins University School of Medicine, Baltimore, Maryland, USA; ²⁵Department of Radiology and Nuclear Medicine, Cardiovascular Research Institute Maastricht (CARIM), Maastricht University Medical Center, Maastricht, the Netherlands; ²⁶Christ's College, Cambridge, United Kingdom; ²⁷Department of Neurosurgery, Mayo Clinic, Rochester, Minnesota, USA; ²⁸Department of Clinical Biochemistry, Royal Free Hospital Campus, University College London School, University College London, London, United Kingdom; ²⁹Joint Department of Medical Imaging, University Health Network, Toronto, Ontario, Canada; ³⁰Department of Radiology Mayo Clinic, Rochester, Minnesota, USA; ³¹Institute for Stroke and Dementia Research, University Hospital, LMU Munich, Munich, Germany; ³²Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania, USA; ³³Department of Radiology Weill Cornell Medical College, New York, New York, USA; ³⁴Walter Reed National Military Medical Center and Uniformed Services University of the Health Sciences, Bethesda, Maryland, USA; ³⁵Department of Neurology, University of Maryland Medical Center, Baltimore, Maryland, USA; ³⁶Department of Cardiovascular Pathology, CVPath Institute, Gaithersburg, Maryland, USA; ³⁷Department of Neurology and Neurosurgery, University College London Hospitals, London, United Kingdom; ³⁸Department of Medical Imaging, University of Toronto, Toronto, Ontario, Canada; ³⁹Division of Cardiovascular Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts, USA; ⁴⁰Institute of Neuroradiology, University Hospital, LMU Munich, Munich, Germany; ⁴¹Department of Radiology, University Hospital, LMU Munich, Munich, Germany; and the ⁴²Die Radiologie, Rosenheim, Germany. *Drs Schindler and Saam contributed equally to this paper as co-senior authors.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

FIGURE 1 Step-by-Step Flowchart to Classify Carotid Atherosclerotic Plaques Into the Different Plaque-RADS Categories



THE RATIONALE OF NEW IMAGE-BASED CLASSIFICATION

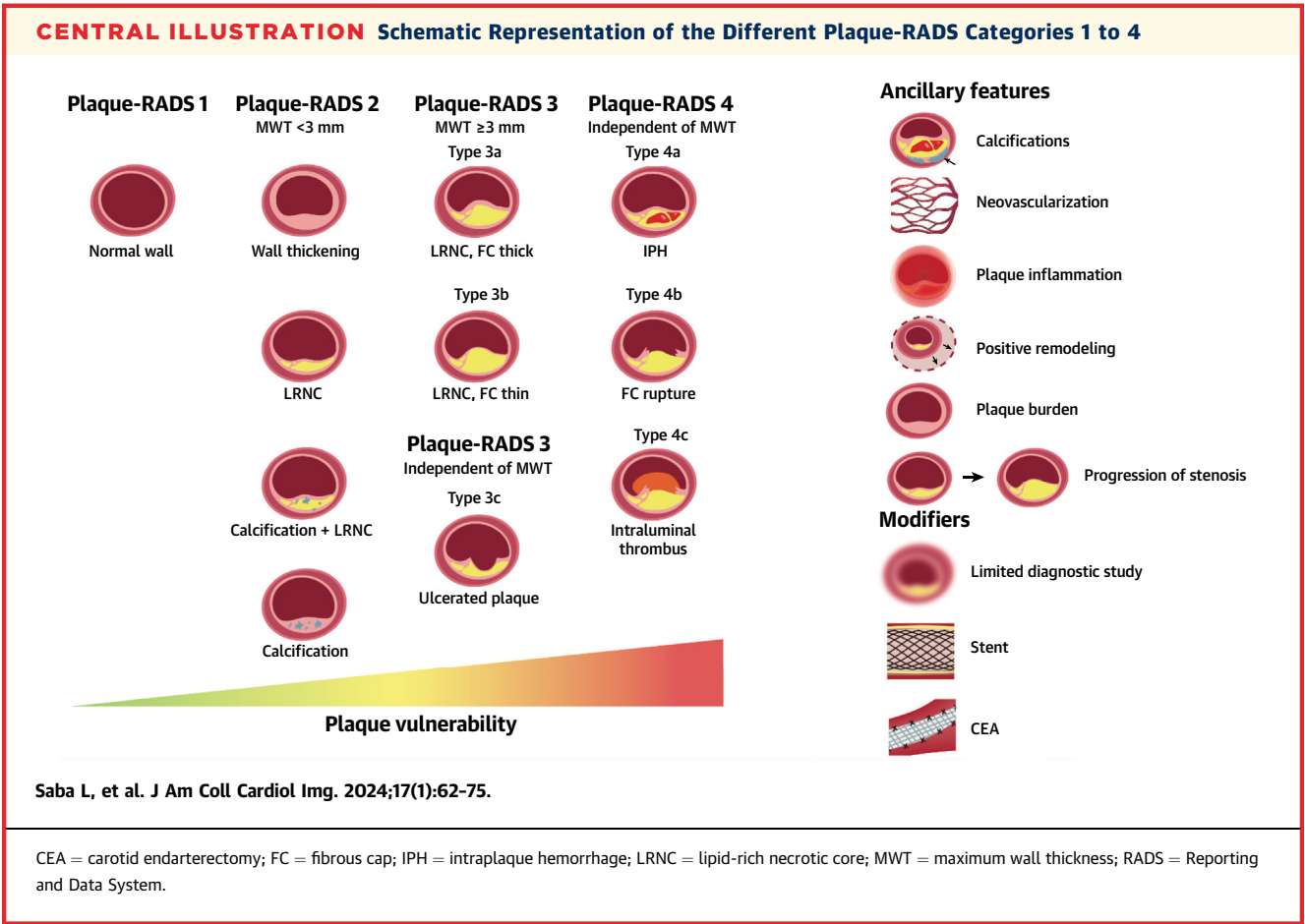
As shown with previous scores, such as the Lung-RADS, BI-RADS, PI-RADS, LI-RADS, and CAD-RADS score for lung, breast, prostate, liver, and coronary artery imaging, respectively, the use of a standardized reporting system improves communication and patient selection by reducing differences in terminology, harmonizing classification formats between different institutions, and facilitating the exchange of clear and systematic information between imaging and referring physicians and researchers.⁷⁻¹¹

To date, there is no such system for a standardized classification of atherosclerotic carotid plaque. Instead, most clinical reports of computed tomography angiography mention the degree of carotid stenosis, but despite their increasingly recognized value, specific plaque features are accounted for in only a minority of cases.¹² This lack of reporting may be at least partly due to gaps in knowledge of high-risk plaque features and their associated risk and possible therapeutic consequences.

Consequently, the introduction of a standardized classification system for carotid atherosclerotic plaque (Plaque-RADS): 1) will level the differences across the various institutions regarding the use of terminology and patient evaluation criteria, serving as a reference format in everyday clinical practice; 2) facilitates data mining and allows researchers across different institutions to collect information in a more homogenous and synergistic way; eg, in the course of time stratified prognostic data could be collected for each Plaque-RADS category and help clinicians design agreed-upon treatment flowcharts; and 3) draws attention to imaging findings representative of plaque morphology and composition beyond the mere degree of stenosis underscoring a paradigm shift.

PLAQUE-RADS REPORTING SYSTEM

Plaque-RADS categories are based on specific imaging features of plaque composition and other characteristics. The score is applied on a per-vessel basis and can be established by ultrasound, computed tomography angiography, and MRI. **Figure 1**



and the **Central Illustration** provide a flowchart and schematic overview of the Plaque-RADS categories. Categories range from Plaque-RADS 1 (absence of atherosclerosis) to Plaque-RADS 4 (plaque with

features of complicated plaque) and should represent the clinically most relevant finding per vessel. Further subspecifications (a, b, c) can be provided for Plaque-RADS categories 3 and 4. Not all imaging modalities

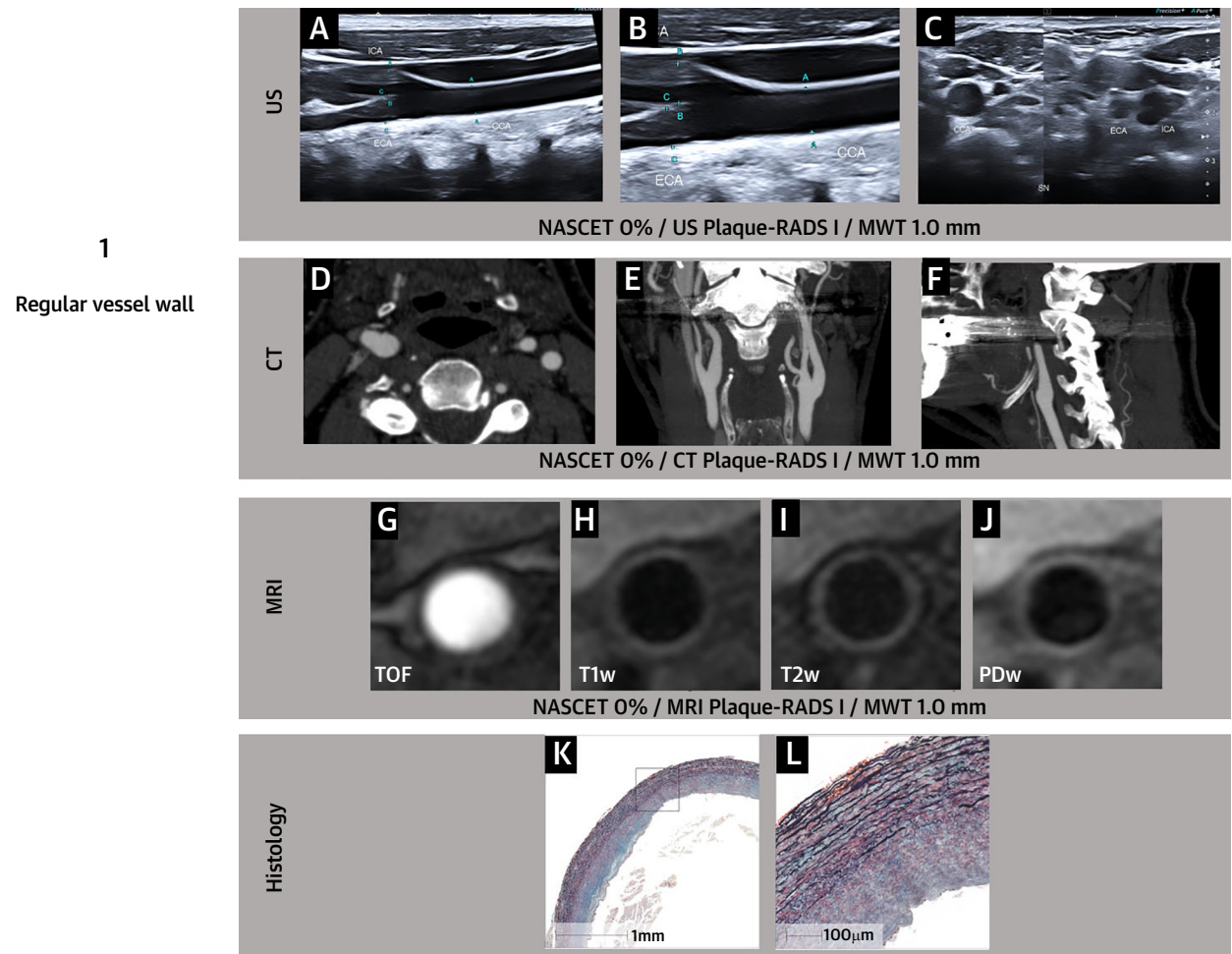
TABLE 1 Summary of Plaque-RADS Categories Based on Imaging Findings and the Attributable Risk of Developing Symptoms

Plaque-RADS Score	Attributable Risk of Ipsilateral Cerebrovascular Events	Imaging Findings
1	Absent	Normal vessel wall
2	Low	MWT <3 mm
3	Moderate	MWT ≥3 mm or Healed ulcerated plaque
3a	Moderate	LRNC with intact thick FC (MWT ≥3 mm)
3b	Moderate	LRNC with thin FC (MWT ≥3 mm)
3c	Moderate	Healed ulcerated plaque
4	High	Complicated plaque (irrespective of MWT)
4a	High	IPH
4b	High	Ruptured FC
4c	High	Intraluminal thrombus

Ancillary features: inflammation, neovascularization, positive plaque remodeling, plaque progression, calcifications. Modifiers: limited diagnostic study ("L"), presence of a stent ("Stent"), previous carotid endarterectomy ("CEA").

FC = fibrous cap; IPH = intraplaque hemorrhage; LRNC = lipid-rich necrotic core; MWT = maximum wall thickness; RADS = reporting and data system.

FIGURE 2 Plaque-RADS 1



Ultrasound (US) shows regular wall in the common carotid artery (CCA) and bifurcation. (A to C) The vessel wall in US is homogenous and thin. Computed tomography (CT) shows regular wall in the CCA and bifurcation on axial (D), coronal (E), and sagittal (F) reconstructions. (G to J) Magnetic resonance imaging (MRI) shows regular wall of the CCA. (K and L) Histology shows normal vessel wall. High magnification of the boxed area (L) shows tunica media and intima with mild intimal thickening, which cannot be visualized with current in vivo imaging modalities. Abbreviation as in [Figure 1](#).

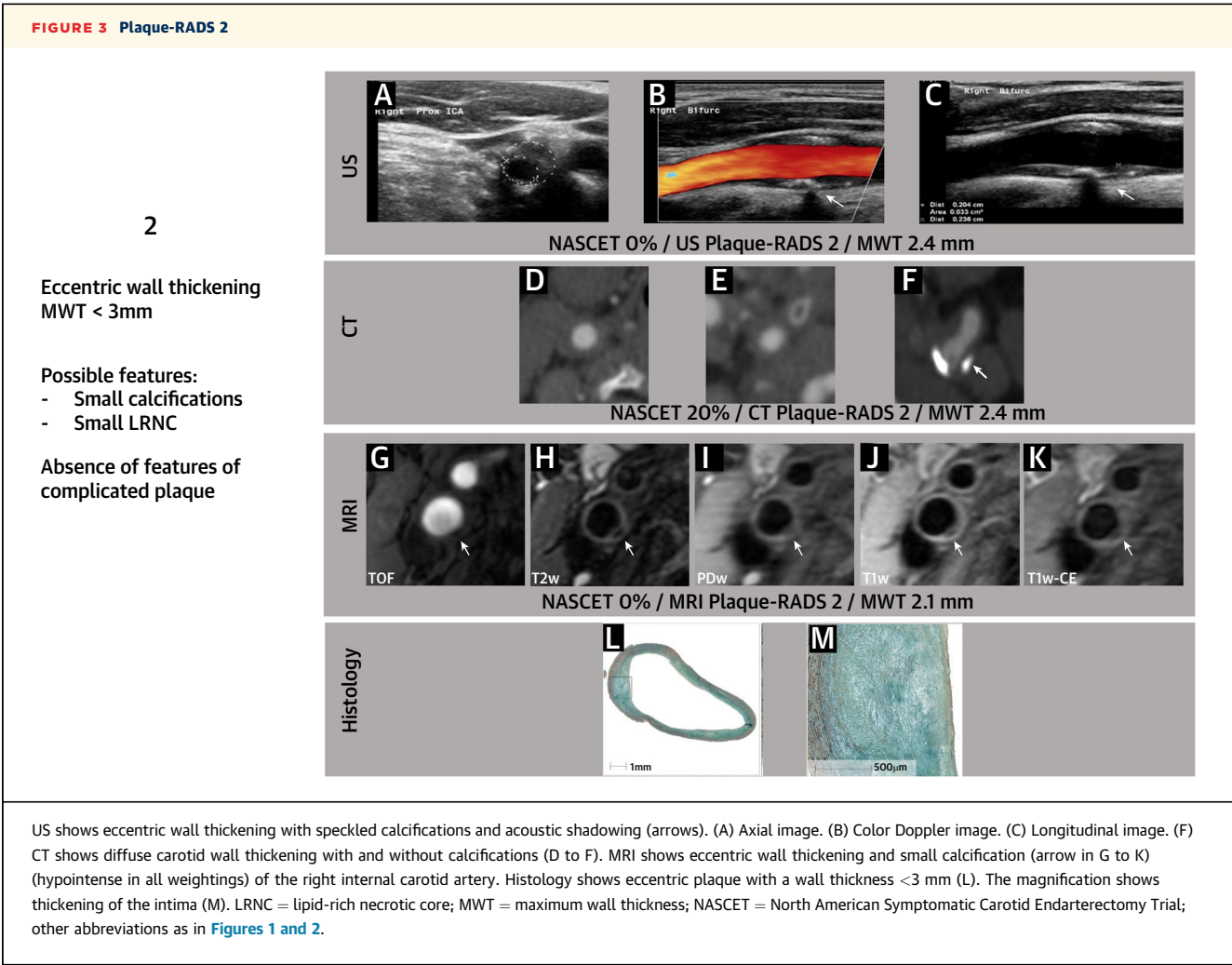
are currently equally suited to identify the individual categories. Therefore, the modality used to obtain the score should always be provided.

In addition, the Plaque-RADS categories may be supplemented by “ancillary features” of carotid plaque vulnerability, namely, plaque inflammation and neovascularization, positive carotid artery remodeling, plaque burden, progression of stenosis, and carotid plaque calcifications (see [Supplemental Methods](#), [Supplemental Table 1](#), [Supplemental Figures 1 to 4](#)).

Table 1 summarizes the characteristic imaging features of the Plaque-RADS categories and the attributable risk of developing symptoms.

PLAQUE-RADS CATEGORIES

PLAQUE-RADS 1. This category represents the normal vessel wall with no evidence of localized atherosclerotic plaque ([Figure 2](#)). Population-based cohort studies including the Rotterdam Study, the Tromsø Study, and the MESA (Multi-Ethnic Study of



Atherosclerosis) study have shown that patients without carotid plaque are not at risk of atherosclerosis-related cardiovascular or cerebrovascular events.¹³⁻¹⁶ Vessels of this category are consistent with AHA lesion-type I/II plaques.

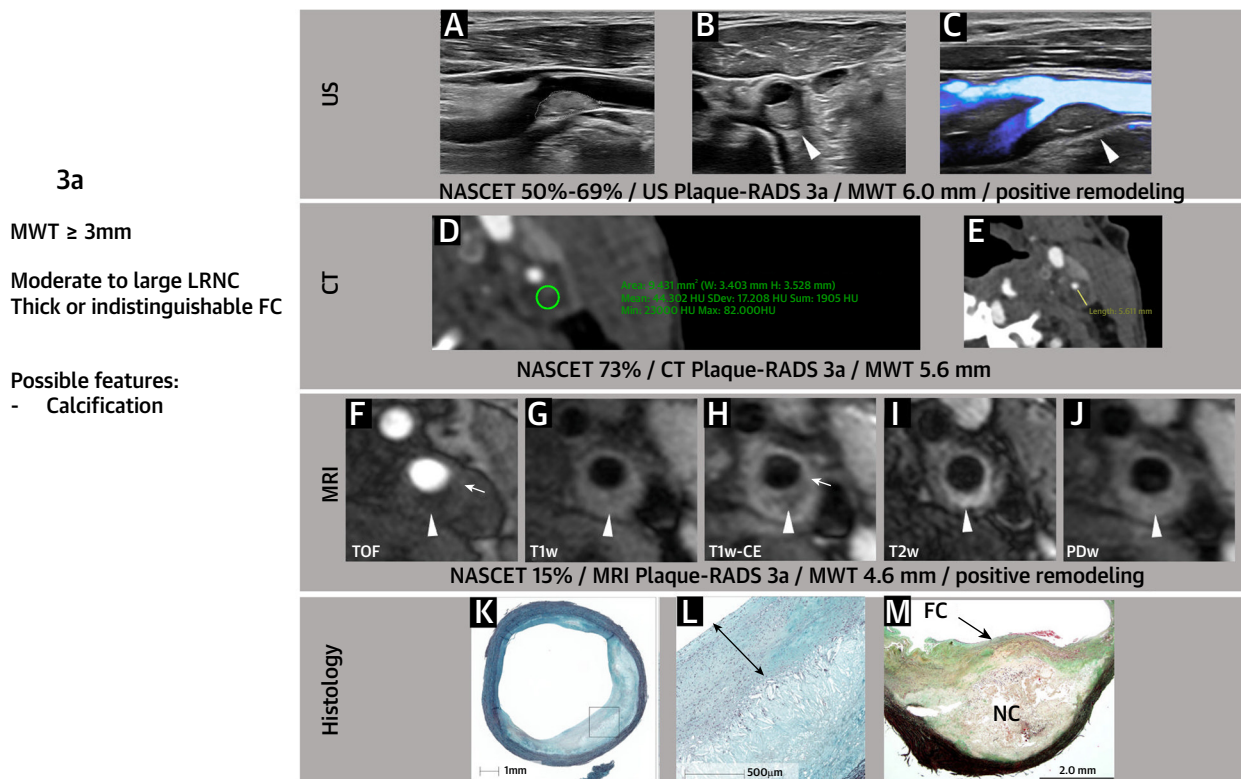
PLAQUE-RADS 2. This category is defined by an eccentric plaque with a maximum wall thickness (MWT) <3 mm and the absence of complicated plaque features such as IPH, fibrous cap (FC) rupture, and intraluminal thrombus ([Figure 3](#)).

Plaques in this category may consist mainly of fibrous tissue, small lipid pools, a small LRNC, calcifications, or a combination of these tissue types.

These plaque features are hallmarks of relatively stable plaques, although they are also potential precursors of more advanced lesions. The presence of these features results in an increase in wall thickness that has been shown to be associated with

increased cerebrovascular and cardiovascular risk, but less than that associated with complicated plaque features.¹⁷ In this regard, total plaque thickness, as determined by ultrasound, has been shown to improve the prediction of future atherosclerotic cardiovascular events over and above that provided by traditional risk factors alone.^{18,19} The risk of Plaque-RADS 2 lesions is higher than Plaque-RADS 1 lesions, but it is still relatively low. This category contains plaques of AHA-lesion types III, IV/V (small), VII, and VIII. The rationale of choosing a cutoff of MWT <3 mm is discussed in the [Supplemental Methods](#).

PLAQUE-RADS 3. This category represents a carotid plaque with an MWT of ≥ 3 mm which may consist of a moderate to large LRNC, calcifications, healed ulcerations, and fibrous tissue. Complicated plaque features, such as IPH, thrombus, and plaque

FIGURE 4 Plaque-RADS 3a

US shows large plaque of the carotid bifurcation with uniform isoechoic echogenicity on B-mode imaging (arrowhead) consistent with LRNC and thick FC. Longitudinal (A) and transverse (B) views. (C) Microflow imaging. (D and E) CT shows low-attenuating plaque with a mean HU value of 44 HU in the right internal carotid artery (ICA) resembling an LRNC. The status of the FC cannot be assessed with CT. MRI shows nonstenosing plaque of the left ICA. A large LRNC (arrowhead in F to J) appears isointense in time-of-flight (TOF) images (hypointense in the T1w-CE images, and isointense to hyperintense in T1w pre-contrast, PDw, and T2w images). A thick and intact FC (arrow in F and H) (hyperintense in T1w-CE and hypointense in TOF-imaging) separates the LRNC from the lumen. (K and L) Histology shows intimal thickening consistent with a thick FC over a LRNC. (M) A magnified view of a thick FC overlying the LRNC. (M) Image is reproduced with permission from Kolodgie et al.⁴⁰ FC = fibrous cap; NC = necrotic core; other abbreviations as in [Figures 1 to 3](#).

rupture are absent. Further subclassification may be undertaken with dedicated imaging. This category contains plaques of AHA lesion-types IV/V, VII, and VIII.

PLAQUE-RADS 3A. This subcategory represents a carotid plaque with a moderate to large LRNC, a thick FC, and an MWT of ≥ 3 mm in the absence of complicated plaque features ([Figure 4](#)).

Currently, data on the risk of LRNC is limited. Nonetheless, a meta-analysis by Gupta et al²⁰ showed an increased risk for future ipsilateral cerebrovascular events when LRNC is present with an HR of 3.00 (95% CI: 1.511-5.945; $P = 0.002$).² Besides an increased downstream cerebrovascular risk, the presence of an LRNC is also associated with an increase in cardiovascular risk.²¹

PLAQUE-RADS 3B. This subcategory contains carotid plaque with an MWT ≥ 3 mm with a moderate to large LRNC with thin and intact FC ([Figure 5](#)).

It must be emphasized that the capability of contemporary imaging to accurately assess thin FCs lacks evidence. Thus, for assigning a score 3b in the Plaque-RADS classification system, the thin FC may be either directly visualized (if the spatial resolution of the modality in use allows that) or inferred by the presence of an LRNC without visualization of a thick and intact FC. Most importantly, what distinguishes this class from higher-risk class 4 is the absence of complicated plaque features.

Regarding FC integrity, several studies have emphasized its determinant role in plaque stability.^{20,21} A thick FC is associated with a low risk of

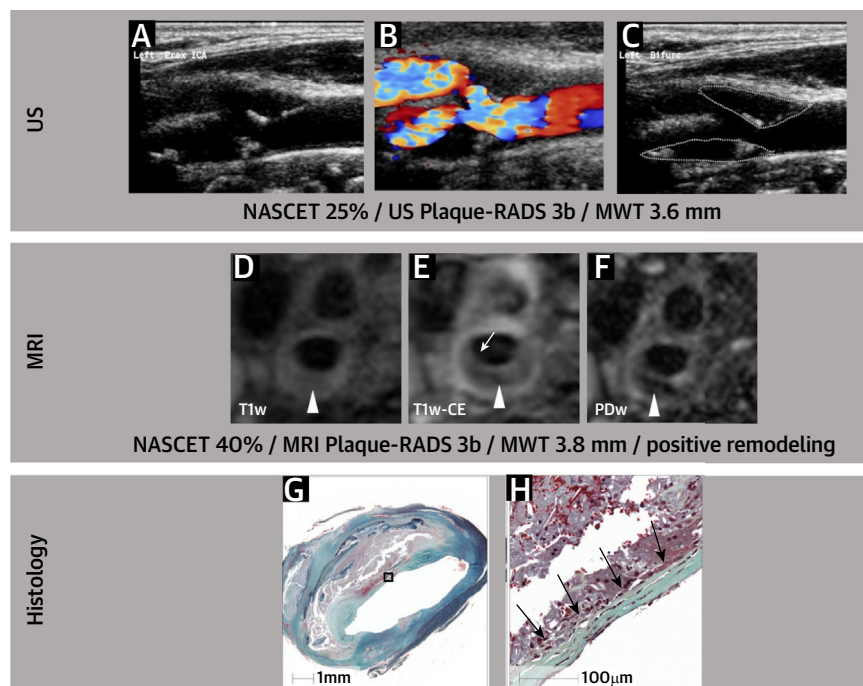
FIGURE 5 Plaque-RADS 3b

3b

MWT ≥ 3 mm

Moderate to large LRNC
Thin FC

Possible features:
- Calcification



US shows complex plaque with presence of juxtaluminal black areas (JBAs) in both the anterior and posterior component of the plaque with 2 discrete white areas (DWAs) in the far wall component of the plaque consistent with a large LRNC or IPH at the origin of the left carotid bifurcation. Large sections of the plaque outline do not have a visible (ie, thin) FC. (A, C) B-mode images. (B) Color flow. (C) Outline of the anterior and posterior plaque components. MRI shows mildly stenosing plaque in the right ICA with a large LRNC (arrowheads in D, E, and F) (hypointense in contrast enhanced T1w-CE). The FC is thin and not in its entity delineated (arrow in E). Histology shows thin FC (arrows in magnified image from G) overlying a large LRNC (G, H). IPH = intraplaque hemorrhage; other abbreviations as in [Figures 1 to 3](#).

plaque rupture, whereas the risk of rupture increases for a thin FC.^{20,23}

PLAQUE-RADS 3C. The defining feature of this category is plaque ulceration regardless of plaque thickness in the absence of IPH, FC-disruption, or intraluminal thrombus ([Figure 6](#)).

Thus, for what pertains to the designation of score 3c in the Plaque-RADS classification system, the term ulceration must be intended as ulceration not associated with the presence of IPH (score 4a), visible FC disruption (score 4b) or intraluminal thrombus (score 4c); rather, the term ulceration in this context refers to a surface cavity most likely secondary to previous extrusion of atheromatous material in the context of a healed plaque rupture.

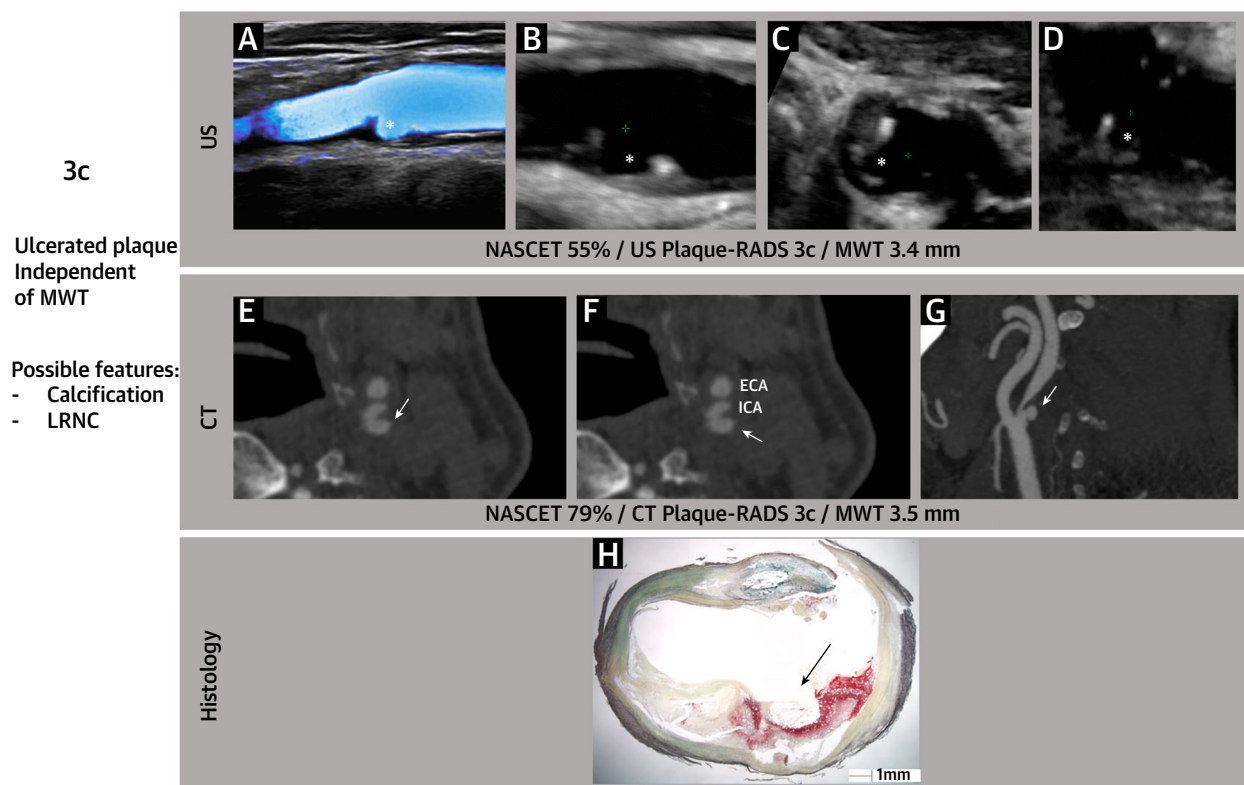
PLAQUE-RADS 4. Plaque-RADS score 4 is assigned in the presence of at least one of the following findings independent of plaque thickness: IPH, a ruptured FC, or an intraluminal thrombus. When feasible, a further subclassification can be used, differentiating IPH, ruptured FC, and intraluminal thrombi into classes

4a, 4b, and 4c, respectively. Subclasses may provide important information in future studies to better understand statistical correlations between such specific entities and clinical events. This category contains plaques of AHA lesion-type VI.

PLAQUE-RADS 4A. The defining feature of this category is IPH ([Figure 7](#)).

In the CAPIAS (Carotid Plaque Imaging in Acute Stroke) study, IPH was the most common feature of complicated plaques and present in 89% of all complicated plaques ipsilateral to acute ischemic stroke.²⁴ In the recent prospective PARISK (Plaque At RISK) study of 244 patients with a recent symptomatic mild-to-moderate carotid stenosis during a mean follow-up period of 5.1 years, the presence of IPH was associated with recurrent cerebrovascular events (HR: 2.12; 95% CI: 1.02-4.44).²⁵ Along the same lines, pooled individual patient data from 7 cohort studies of 560 patients with symptomatic and 136 patients with asymptomatic carotid stenosis found MRI-detected IPH in 51.6% of the symptomatic and

FIGURE 6 Plaque-RADS 3c



US shows mixed hyperechogenic and hypoechoic plaque at the carotid bulb on B-mode imaging with ulceration (asterisk) on microflow imaging (A), and B-mode 3-dimensional-US with longitudinal (B), axial (C), and coronal (D) views. CT shows axial and sagittal views of an ulcerated plaque in the left ICA, visible as contrast outpouching (≥ 1 mm) into the plaque (arrows in E to G). High-grade stenosis. Histology shows ulcerated plaque. (H) An arrow indicates the site of ulceration. (H) Image reproduced with permission from Peeters et al.⁴¹ Abbreviations as in [Figures 1, 2, and 4](#).

29.4% of the asymptomatic patients. Multivariate analysis identified IPH (HR: 11.0; 95% CI: 4.8-25.1) and severity of stenosis (HR: 3.3; 95% CI: 1.4-7.8) as independent predictors of recurrent ipsilateral stroke. The presence of IPH increased the risk for first-time stroke in asymptomatic patients with carotid stenosis by almost 8-fold (HR: 7.9; 95% CI: 1.3-47.6).²⁶

PLAQUE-RADS 4B. The defining feature of this category is a ruptured FC, usually accompanied by juxtaluminal plaque hemorrhage ([Figure 8](#)).²⁷

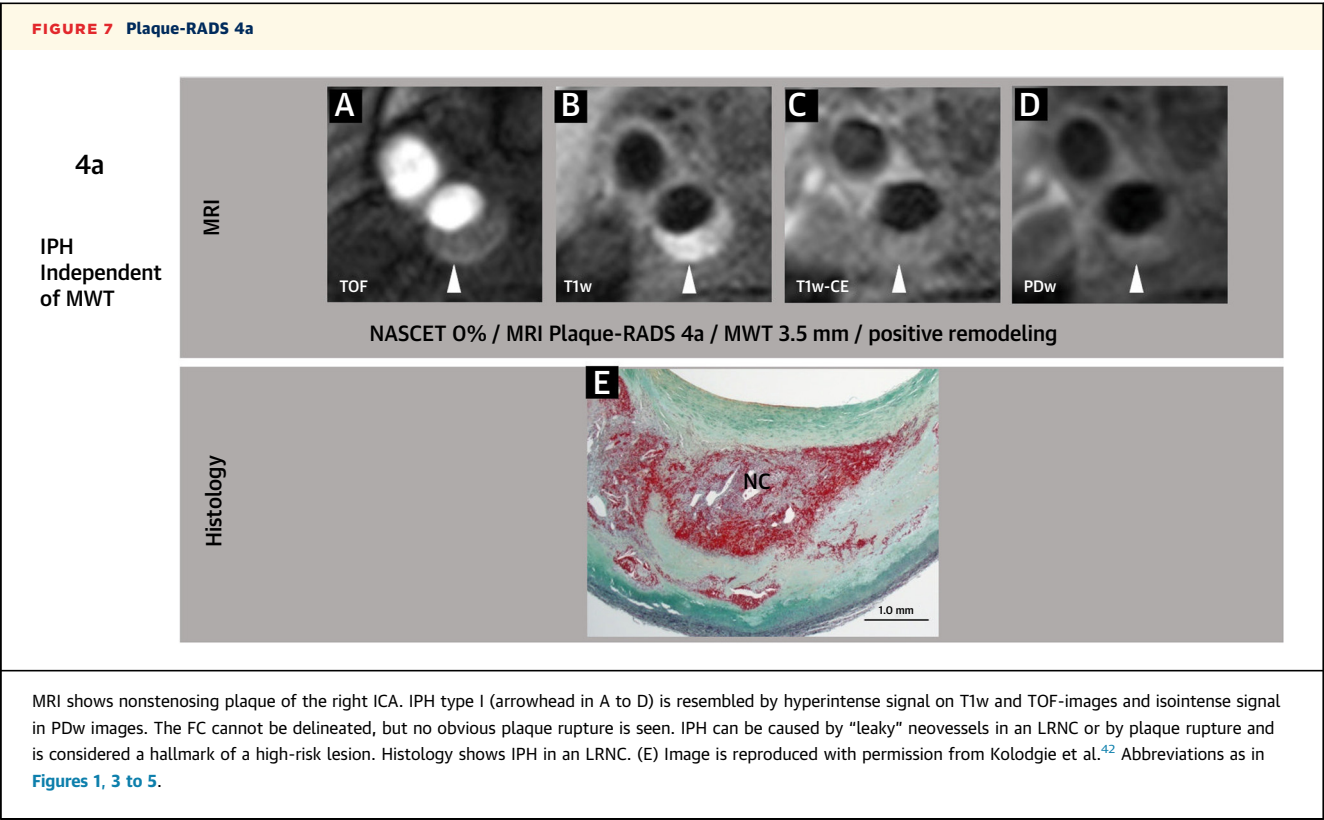
Disruption of the FC, with the resultant exposure of thrombogenic subendothelial plaque constituents, can precipitate thromboembolic complications both in the carotid and coronary vascular bed.²⁸ It appears that plaque ruptures represent a dynamic process of rupture, thrombus formation, healing, and remodeling of the plaque.²⁹ A meta-analysis of 363 carotid arteries from asymptomatic and symptomatic patients showed that a thin or ruptured FC (HR: 5.93

[95% CI: 2.65-13.29]; $P < 0.01$) is associated with future cerebrovascular events.²⁰

PLAQUE-RADS 4C. This category is characterized by carotid plaque with an intraluminal thrombus ([Figure 9](#)). Other features such as IPH or FC rupture may also be present.

Intraluminal carotid artery thrombi are associated with neurologic symptoms in up to 92% of cases and are recognized predictors of stroke of carotid origin.^{17,30-33} McNally et al¹⁷ conducted a retrospective cross-sectional study of 726 carotid-brain MRI examinations in patients undergoing stroke work-up. After the exclusion of noncarotid-plaque stroke, occlusions, and near-occlusions, the strongest predictor of carotid-source stroke was intraluminal thrombus (odds ratio: 103.6 [95% CI: 8.64-710.8]; $P < 0.001$).¹⁷

[Supplemental Table 2](#) provides an overview of previous studies examining carotid plaque



characteristics according to Plaque-RADS categories and attributable risk for symptom development.

REPORTING THE PLAQUE-RADS SCORE

For the structured reporting of a Plaque-RADS score, we recommend using the following syntax, which will be further detailed in the paragraphs below: side of carotid: stenosis degree/imaging modality Plaque-RADS score/MWT/ancillary features/modifiers.

STENOSIS DEGREE. The Plaque-RADS score is not meant to replace the measurement of stenosis but rather to integrate synergistically with it. The independent association between the degree of carotid stenosis in both symptomatic and asymptomatic patients is well known.³⁴⁻³⁸ The degree of luminal stenosis should be reported using the NASCET (North American Symptomatic Carotid Endarterectomy Trial) protocol as it is widely used and already harmonized across modalities: stenosis [%] = (diameter of the normal distal internal carotid artery – narrowest ICA diameter in the stenotic segment) / diameter of the normal distal ICA, where ICA represents the internal carotid artery.

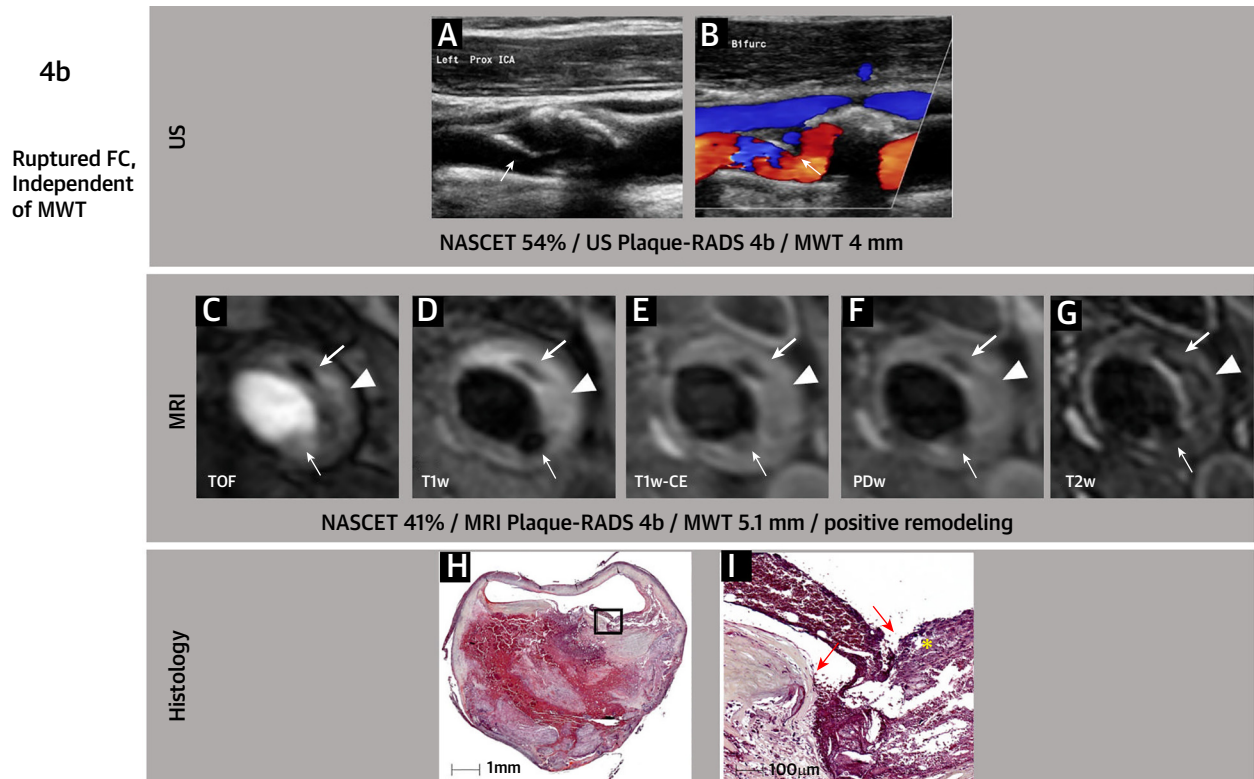
IMAGING MODALITY. The imaging modality used to obtain the Plaque-RADS score should be indicated. In the final evaluation, all modalities used should be listed, with the one leading to the highest score mentioned first. The [Supplemental Methods](#) contains suggestions regarding which imaging modalities should be used for optimal assessment of each Plaque-RADS category. [Supplemental Table 3](#) summarizes key plaque features across different imaging modalities.

However, a detailed discussion of ideal imaging practice of the atherosclerotic plaque is beyond the purpose of this paper but can be found in the consensus document by the American Society of Neuroradiology Vessel Wall Imaging Study group.¹

MWT. The MWT (mm) is derived via a linear measurement of the greatest thickness of the vessel wall as measured on axial images perpendicular to the vessel's long axis and includes the arterial vessel wall and both calcified and noncalcified components of the plaque.

ANCILLARY FEATURES. To accommodate the variety of other imaging vulnerability markers that have been well studied and validated in the

FIGURE 8 Plaque-RADS 4b



US shows ruptured plaque in the left carotid bulb. Calcified area is observed on the anterior wall producing an acoustic shadow (white arrow in A). A free flap is visible in the lumen attached to the anterior wall on the left (white arrow in B). LRNC is not visible, presumably discharged, with color flow including flow reversal (blue area above the flap in B) between the flap and the near wall of the artery. This is a high-risk plaque. MRI shows complex plaque in the left carotid bulb. Ulceration with rupture of the FC at the posterior end is observed (solid arrow in C to G). The signal intensity of the ulcer is the same as that of the lumen. Large IPH in almost the entire plaque is seen as hyperintense on T1w, and TOF images and isointense in PDw and T2w images (arrowhead in C to G) are suggestive of fresh plaque hemorrhage. Speckled calcification appears as hypointense signal in all MRI sequences (open arrow in C to G). This is a high-risk plaque. Histology shows ruptured FC (H and I). Magnification shows the area of FC rupture (red arrows in I) and adherent thrombus (asterisk in I). Abbreviations as in Figures 1, 2, and 5.

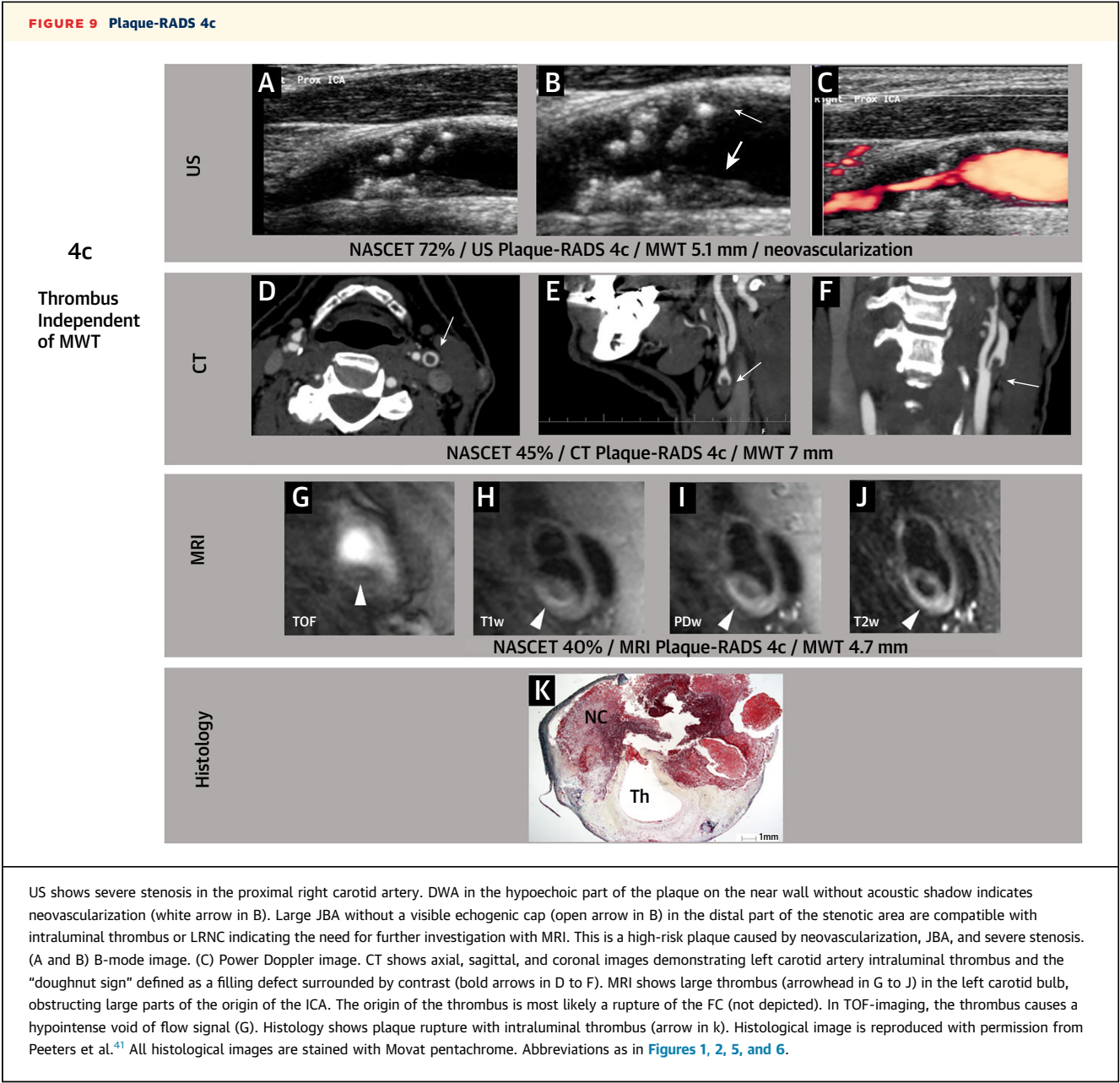
scientific published reports, and with an open mind for future advancements, we propose an optional subclassifier of Plaque-RADS: AnFe. We suggested reporting each assessed individual AnFe in the final score.

MODIFIERS. Similar to CAD-RADS, categories can be complemented by modifiers including limited-diagnostic study (“L”), the presence of a stent (“Stent”), and previous carotid endarterectomy (CEA).¹¹ The Modifier L can be applied if the study is not fully diagnostic, eg, in case of blooming artifacts on computed tomography (CT) or motion artifacts or metal-induced artifacts on CT or MRI. Overestimation of restenosis using noninvasive imaging is a potential risk in stented carotid arteries and assessment of plaque morphology is limited in stented vessels.

Therefore, the application of the modifier “Stent” may be useful in clinical practice.

Following the logic described earlier, a plaque in a symptomatic patient with ipsilateral 50% stenosis with IPH with positive remodeling would be classified as “Right carotid: 50%/MRI Plaque-RADS 4a/MWT = 5 mm/Positive Remodeling.” AnFe do not determine the main Plaque-RADS score. Therefore, the assessment of the AnFe is not mandatory in the Plaque-RADS score but rather serves as a complementary tool when available and is also for research purposes.

Finally, it is fundamental to consider the appropriateness of the modality used for each Plaque-RADS score. Whenever practitioners find that the study could not definitively exclude the possibility of a relevant score upgrade, further investigation should



be considered. By means of example, the identification of a Plaque-RADS score 3a on CT may require further investigation on MRI to rule out the presence of IPH (which would upgrade to score 4a). Rather than adding a classification category dedicated to the imaging modality, we suggest that “consider MRI examination” is reported in the score and further information is provided in the impressions; in this case, a plaque in an asymptomatic patient with 70% carotid stenosis and an MWT of 5 mm, a positive rim sign, and positive remodeling would read as “Left carotid:

70%/CT Plaque-RADS 3a/MWT = 5 mm/Positive rim sign AND Positive Remodeling/Consider MRI examination.”

INTEROBSERVER AGREEMENT

A score is only helpful if it is straightforward and reliable. As confirmation of applicability and reliability, Plaque-RADS categories were assigned by blinded experts in the field of plaque imaging to 100 vessels on ultrasound, CT, and MRI, each. The

interobserver agreement was retrospectively assessed based on Cohen κ test to investigate the reproducibility of Plaque-RADS categories (0.00 = poor, 0.00-0.20 = slight, 0.21-0.40 = fair, 0.41-0.60 = moderate, 0.61-0.80 = substantial, and 0.81-1.00 = almost perfect).³⁹ The analysis was based on data from previously published studies approved by the institutional review boards, and informed consent was waived because of its retrospective nature. Interobserver agreement for ultrasound, CT, and MRI images was excellent ($\kappa = 0.804$; $P < 0.001$; $\kappa = 0.868$; $P < 0.001$; and $\kappa = 0.876$; $P < 0.001$; respectively). Additionally, the overall inter-reader agreement among the readers across different modalities was excellent ($\kappa = 0.856$; $P < 0.001$). The results are presented in more detail in [Supplemental Table 4](#).

CONCLUSIONS

Plaque-RADS is a standardized cross-modality system for reporting carotid plaque composition and morphology. This structured system aims to provide in-depth insight into carotid imaging markers of vulnerability to better evaluate carotid artery disease and predict the risk of cerebrovascular events. The main purposes of the Plaque-RADS score are to create a standardized lexicon and structured reporting for carotid artery disease and to improve communication between those interpreting images, referring clinicians, and researchers by providing a clear and reproducible, personalized risk stratification of patients.

ACKNOWLEDGMENT The authors thank Antonia Weingart for the pictorial presentation of the different Plaque-RADS categories.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The views expressed in this paper are those of the authors and do not reflect the official policy of the Department of Army/Navy/Air Force, Department of Defense, or U.S. Government. The identification of specific products or scientific instrumentation does not constitute endorsement or implied endorsement on the part of the author, Department of Defense, or any component agency. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Luca Saba, University of Cagliari-Azienda Ospedaliero Universitaria di Cagliari-polo di Monserrato, via Tola 7, Provincia di Cagliari, Sardegna 09128, Italy. E-mail: lucasaba@tiscali.it.

PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE:

Plaque-RADS is an intuitive and reliably assessable tool which enables standardized description of a given atherosclerotic carotid lesion in symptomatic and asymptomatic patients. The system, which can be applied by readers of any experience, helps to detect critical hallmarks of atherosclerotic plaques and translates the findings into the attributable risk. This facilitates interpretation of findings in both clinical routine and research. This may allow improvement in diseases risk stratification and adequate therapy.

TRANSLATIONAL OUTLOOK: The introduction of the Plaque-RADS classification and its broad application will facilitate communication in clinical routine and may serve as basis for studies, which advance the management of carotid atherosclerotic disease.

REFERENCES

1. Saba L, Yuan C, Hatsukami TS, et al. Carotid artery wall imaging: perspective and guidelines from the ASNR Vessel Wall Imaging Study Group and expert consensus recommendations of the American Society of Neuroradiology. *Am J Neuroradiol*. 2018;39(2):E9-E31. <https://doi.org/10.3174/ajnr.A5488>
2. Aboyans V, Ricco J-B, Bartelink M-LEL, et al. 2017 ESC guidelines on the diagnosis and treatment of peripheral arterial diseases, in collaboration with the European Society for Vascular Surgery (ESVS): document covering atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal. *Eur Heart J*. 2018;39(9):763-816. <https://doi.org/10.1093/eurheartj/ehx095>
3. Naylor R, Rantner B, Ancetti S, et al. Editor's choice - European Society for Vascular Surgery (ESVS) 2023 clinical practice guidelines on the management of atherosclerotic carotid and vertebral artery disease. *Eur J Vasc Endovasc Surg*. 2023;65(1):7-111. <https://doi.org/10.1016/j.ejvs.2022.04.011>
4. Stary HC, Chandler AB, Dinsmore RE, et al. A definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis. A report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. *Circulation*. 1995;92(5):1355-1374. <https://doi.org/10.1161/01.cir.92.5.1355>
5. Cai JM, Hatsukami TS, Ferguson MS, Small R, Polissar NL, Yuan C. Classification of human carotid atherosclerotic lesions with in vivo multi-contrast magnetic resonance imaging. *Circulation*. 2002;106(11):1368-1373. <https://doi.org/10.1161/01.CIR.0000028591.44554.F9>
6. Hollander M, Bots ML, del Sol AI, et al. Carotid plaques increase the risk of stroke and subtypes of cerebral infarction in asymptomatic elderly. *Circulation*. 2002;105(24):2872-2877. <https://doi.org/10.1161/01.CIR.0000018650.58984.75>
7. Version 1.1. American College of Radiology. Published 2019. Accessed September 5, 2019. <https://www.acr.org/-/media/ACR/Files/RADS/Lung-RADS/LungRADSAssessmentCategoriesv1-1.pdf?la=en>
8. American College of Radiology. ACR BI-RADS atlas: breast imaging reporting and data system; mammography, ultrasound, magnetic resonance imaging, follow-up and outcome monitoring,

data dictionary. Accessed December 15, 2023. <https://www.acr.org/Clinical-Resources/Reporting-and-Data-Systems/LI-RADS>

9. Weinreb JC, Barentsz JO, Choyke PL, et al. PI-RADS Prostate Imaging - Reporting and Data System: 2015, Version 2. *Eur Urol*. 2016;69(1):16-40.

10. American College of Radiology. CT/MRI LI-RADS® v2017. Accessed January 1, 2018. <https://www.acr.org/Clinical-Resources/Reporting-and-Data-Systems/LI-RADS/LI-RADS-CT-MRI-v2017>

11. Cury RC, Abbara S, Achenbach S, et al. CAD-RADSTM Coronary Artery Disease—Reporting and Data System. An expert consensus document of the Society of Cardiovascular Computed Tomography (SCCT), the American College of Radiology (ACR) and the North American Society for Cardiovascular Imaging (NASCI). *J Cardiovasc Comput Tomogr*. 2016;10(4):269-281. <https://doi.org/10.1016/j.jcct.2016.04.005>

12. Baradaran H, Foster T, Harrie P, et al. Carotid artery plaque characteristics: current reporting practices on CT angiography. *Neuroradiology*. 2021;63(7):1013-1018. <https://doi.org/10.1007/s00234-020-02610-w>

13. Gepner AD, Young R, Delaney JA, et al. Comparison of coronary artery calcium presence, carotid plaque presence, and carotid intima-media thickness for cardiovascular disease prediction in the Multi-Ethnic Study of Atherosclerosis. *Circ Cardiovasc Imaging*. 2015;8(1):e002262. <https://doi.org/10.1161/CIRCIMAGING.114.002262>

14. Zavodni AEH, Wasserman BA, McClelland RL, et al. Carotid artery plaque morphology and composition in relation to incident cardiovascular events: the Multi-Ethnic Study of Atherosclerosis (MESA). *Radiology*. 2014;271(2):381-389. <https://doi.org/10.1148/radiol.14131020>

15. Mathiesen EB, Johnsen SH, Wilsgaard T, Børnaa KH, Løchen M-L, Njølstad I. Carotid plaque area and intima-media thickness in prediction of first-ever ischemic stroke. *Stroke*. 2011;42(4):972-978. <https://doi.org/10.1161/STROKEAHA.110.589754>

16. Van Den Bouwhuisen QJA, Bos D, Ikram MA, et al. Coexistence of calcification, intraplaque hemorrhage and lipid core within the asymptomatic atherosclerotic carotid plaque: the Rotterdam study. *Cerebrovasc Dis*. 2015;39(5-6):319-324. <https://doi.org/10.1159/000381138>

17. McNally JS, McLaughlin MS, Hinckley PJ, et al. Intraluminal thrombus, intraplaque hemorrhage, plaque thickness, and current smoking optimally predict carotid stroke. *Stroke*. 2015;46(1):84-90. <https://doi.org/10.1161/STROKEAHA.114.006286>

18. Nicolaidis AN, Panayiotou AG, Griffin M, et al. Arterial ultrasound testing to predict atherosclerotic cardiovascular events. *J Am Coll Cardiol*. 2022;79(20):1969-1982. <https://doi.org/10.1016/j.jacc.2022.03.352>

19. Selwaness M, Bos D, van den Bouwhuisen Q, et al. Carotid atherosclerotic plaque characteristics on magnetic resonance imaging relate with history of stroke and coronary heart disease. *Stroke*. 2016;47(6):1542-1547. <https://doi.org/10.1161/STROKEAHA.116.012923>

20. Gupta A, Baradaran H, Schweitzer AD, et al. Carotid plaque MRI and stroke risk: a systematic review and meta-analysis. *Stroke*. 2013;44(11):3071-3077. <https://doi.org/10.1161/STROKEAHA.113.002551>

21. Brunner G, Virani SS, Sun W, et al. Associations between carotid artery plaque burden, plaque characteristics, and cardiovascular events: the ARIC carotid magnetic resonance imaging study. *JAMA Cardiol*. 2021;6(1):79-86. <https://doi.org/10.1001/jamacardio.2020.5573>

22. van Dijk AC, Truijman MTB, Hussain B, et al. Intraplaque hemorrhage and the plaque surface in carotid atherosclerosis: the Plaque At RISK Study (PARISK). *AJNR Am J Neuroradiol*. 2015;36(11):2127-2133. <https://doi.org/10.3174/ajnr.A4414>

23. Sun J, Zhao X-Q, Balu N, et al. Carotid plaque lipid content and fibrous cap status predict systemic CV outcomes: the MRI substudy in AIM-HIGH. *J Am Coll Cardiol Img*. 2017;10(3):241-249. <https://doi.org/10.1016/j.jcmg.2016.06.017>

24. Kopczak A, Schindler A, Bayer-Karpinska A, et al. Complicated carotid artery plaques as a cause of cryptogenic stroke. *J Am Coll Cardiol*. 2020;76(19):2212-2222. <https://doi.org/10.1016/j.jacc.2020.09.532>

25. Kopczak A, Schindler A, Sepp D, et al. Complicated carotid artery plaques and risk of recurrent ischemic stroke or TIA. *J Am Coll Cardiol*. 2022;79(22):2189-2199. <https://doi.org/10.1016/j.jacc.2022.03.376>

26. Schindler A, Schinner R, Altat Nishaf, et al. Prediction of stroke risk by detection of hemorrhage in carotid plaques. *J Am Coll Cardiol Img*. 2020;13(2 Part 1):395-406. <https://doi.org/10.1016/j.jcmg.2019.03.028>

27. Kampschulte A, Ferguson MS, Kerwin WS, et al. Differentiation of intraplaque versus juxta-luminal hemorrhage/thrombus in advanced human carotid atherosclerotic lesions by in vivo magnetic resonance imaging. *Circulation*. 2004;110(20):3239-3244. <https://doi.org/10.1161/01.CIR.0000147287.23741.9A>

28. Cademartiri F, Balestrieri A, Cau R, et al. Insight from imaging on plaque vulnerability: similarities and differences between coronary and carotid arteries — implications for systemic therapies. *Cardiovasc Diagn Ther*. 2020;10(4):1150-1162. <https://doi.org/10.21037/cdt-20-528>

29. Carr S, Farb A, Pearce WH, Virmani R, Yao JST. Atherosclerotic plaque rupture in symptomatic carotid artery stenosis. *J Vasc Surg*. 1996;23(5):755-766. [https://doi.org/10.1016/S0741-5214\(96\)70237-9](https://doi.org/10.1016/S0741-5214(96)70237-9)

30. Bhatti AF, Leon LRJ, Labropoulos N, et al. Free-floating thrombus of the carotid artery: literature review and case reports. *J Vasc Surg*. 2007;45(1):199-205. <https://doi.org/10.1016/j.jvs.2006.09.057>

31. Eesa M, Hill MD, Al-Khatthami A, et al. Role of CT angiographic plaque morphologic characteristics in addition to stenosis in predicting the symptomatic side in carotid artery disease. *Am J Neuroradiol*. 2010;31(7):1254 LP-1260. <https://doi.org/10.3174/ajnr.A2078>

32. Paraskevas KI, Veith FJ, Spence JD. How to identify which patients with asymptomatic carotid stenosis could benefit from endarterectomy or stenting. *Stroke Vasc Neurol*. 2018;3(2):92-100. <https://doi.org/10.1136/svn-2017-000129>

33. Markus HS, King A, Shipley M, et al. Asymptomatic embolisation for prediction of stroke in the Asymptomatic Carotid Emboli Study (ACES): a prospective observational study. *Lancet Neurol*. 2010;9(7):663-671. [https://doi.org/10.1016/S1474-4422\(10\)70120-4](https://doi.org/10.1016/S1474-4422(10)70120-4)

34. Messas E, Goudot G, Halliday A, et al. Management of carotid stenosis for primary and secondary prevention of stroke: state-of-the-art 2020: a critical review. *Eur Heart J Suppl*. 2020;22(suppl M):M35-M42. <https://doi.org/10.1093/eurheartj/suaa162>

35. Strömberg S, Nordanstig A, Bentzel T, Österberg K, Bergström GML. Risk of early recurrent stroke in symptomatic carotid stenosis. *J Vasc Surg*. 2015;61(2):570. <https://doi.org/10.1016/j.jvs.2014.12.017>

36. Nadareishvili ZG, Rothwell PM, Beletsky V, Pagniello A, Norris JW. Long-term risk of stroke and other vascular events in patients with asymptomatic carotid artery stenosis. *Arch Neurol*. 2002;59(7):1162-1166. <https://doi.org/10.1001/archneur.59.7.1162>

37. Chang RW, Tucker L-Y, Rothenberg KA, et al. Incidence of ischemic stroke in patients with asymptomatic severe carotid stenosis without surgical intervention. *JAMA*. 2022;327(20):1974-1982. <https://doi.org/10.1001/jama.2022.4835>

38. Howard DPJ, Gaziano L, Rothwell PM. Risk of stroke in relation to degree of asymptomatic carotid stenosis: a population-based cohort study, systematic review, and meta-analysis. *Lancet Neurol*. 2021;20(3):193-202. [https://doi.org/10.1016/S1474-4422\(20\)30484-1](https://doi.org/10.1016/S1474-4422(20)30484-1)

39. McHugh ML. Interrater reliability: the kappa statistic. *Biochem Medica*. 2012;22(3):276-282.

40. Kolodgie FD, Nakawaza G, Sangiorgio G, Ladich E, Burke AP, Virmani R. Pathology of atherosclerosis and stenting. *Neuroimaging Clin N Am*. 2007;17(3):285-301.

41. Peeters W, Hellings WH, de Kleijn DPV, et al. Carotid atherosclerosis plaques stabilize after stroke: insights into the natural process of atherosclerotic plaque stabilization. *Arterioscler Thromb Vasc Biol*. 2009;29(1):128-133.

42. Kolodgie FD, Yahagi K, Mori H, et al. High-risk carotid plaque: lessons learned from histopathology. *Semin Vasc Surg*. 2017;30(1):31-43.

KEY WORDS atherosclerosis, carotid plaque, carotid stenosis, complicated plaque, plaque imaging, Plaque-RADS, reporting and data system, stroke

APPENDIX For an expanded Methods section as well as supplemental figures, tables, and references, please see the online version of this paper.